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Does autism merit belief? Developing an account of scientific realism for psychiatry

Ph.D. Philosophy

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Abstract

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The PhD outlines criteria under which a psychiatric classification merits belief and, as a case study, establishes that autism merits belief. Three chapters respond to anti-realist arguments, three chapters establish conditions under which psychiatric classifications merit belief.

Chapter one addresses the pessimistic meta-induction. I historically analyse autism to show there has been sufficient historical continuity to avoid the pessimistic meta induction.

Chapter two considers arguments from underdetermination. I consider the strongest candidate for an alternative to autism, classificatory changes which occurred between 1980 and 1985. I argue this does not constitute underdetermination because those changes were methodologically and evidentially flawed.

Chapter three considers theory-ladenness. I consider the two strongest candidates for background theories which might have a negative epistemic effect (cognitive psychology and psychoanalysis). I show these have little influence on what symptoms are formulated or how symptoms are grouped together.

Chapter four argues against psychiatric classifications as natural kinds and against notions that inductive knowledge of psychiatric classifications requires robust causes. I outline psychiatric classifications as scientific laws. They are high level idealised models which guide construction of lower level, more specific, models. This opens alternative routes to belief for psychiatric classification lacking robust causes.

Chapter five shows that psychiatric classifications can set relevant populations for deriving statistically significant symptoms. The same behaviour can count as statistically significant for one psychiatric classification but not another. I argue this process strengthens psychiatric classifications inductively, thus contributing to belief.

Chapter six bases belief on theoretical virtues. Unifications and causation are

the two main theoretical virtues. Autism strongly exhibits unifications, stringently covering a wide range of otherwise unrelated symptoms. Additionally, emphasising causation may reduce unifications and thus reduce belief. Attributing unifications is reliable because autism is accessible without employing extremely complicated experimental processes and relies upon secure background theories.

Contents

Publications and Submission Statement

I declare that this thesis is my own work, and has not been submitted in substantially the same form for the award of a higher degree elsewhere**.**

Significant elements of sections of this thesis will be published in a forthcoming article: Fellowes, Sam. (2016). A reappraisal of Kendell and Jablensky's account of validity, *Journal of Evaluation in Clinical Practise.* These sections are *4.3.1 Physical Systems and phenomena, 6.5.1.5 Balancing IBEs, 6.4.4 Validity.*

Significant elements of sections of this thesis formed the essay, RDoC should not always see symptoms as independent of psychiatric categories, which was one of two winners of the 2016 Jaspers Award from the Association for the Advancement of Philosophy and Psychiatry. These sections are **5.2 Classifications and symptoms,** *5.2.1 Classifications, 5.2.2 Symptoms,* **5.3 Systematicity,** *5.3.1 Constructing symptoms, 5.3.2 Employing classifications to systematise symptoms.*

Acronyms

IBE: Inference to the best explanation, inferring one explanation as better than another.

PMI: Pessimistic meta induction, scientists historically believed in theories in the past yet the theories turned out false, suggesting belief over existing theories is undeserved.

NMA: No miracles argument, the only explanation of the success of science is miracles or the truth, since miracles are not allowed in philosophy then truth is the only explanation.

RDoC: Research Domain Criteria, a project started in 2009 by the National Institute for Mental Health. This is part of the National Institute of Health, an agency of the United States Department of Health and Human Services. Rather than link classifications to causes as typically occurs in DSM based casual research RDoC researchers intend to base causal investigation on directly linking causes to symptoms. The hope is that causal investigation will no longer be held back by the potential falsity of currently employed psychiatric classifications.

0.0 Introduction

0.1 The Problem

"Under what circumstances should we chuck the whole thing out and start over?" (Kendler 2012a, p.xiv). Kendler asks this question of psychiatric classifications and, despite being one of the less pessimistic philosophers of psychiatry, his best reason for keeping existing classifications is a lack of alternatives rather than the inherent virtues of existing classifications (Kendler & First 2010, p.265). Scepticism over psychiatric classifications is widespread. Attending an autistic advocacy conference, I observed one autistic speaker criticise many different institutions and services associated with autism before turning on the classification itself. "What is it actually *doing*?", she asked in an exasperated manner, questioning what the diagnosis actually contributed. Similarly, many mental health workers take an "everyone is different" approach, preferring to treat people rather than labels. Most philosophers of psychiatry and psychiatrists accept psychiatric classifications cannot be fully dispensed with but many are concerned about current classifications. Some see most psychiatric classifications as arbitrary (Horwitz 2002, p.5; Kendell & Jablensky 2003, p.7) or not meriting belief (Cooper 2005, p.150; Murphy 2006, p.10). Psychiatry is currently facing a crisis of confidence because over thirty years of investigation has not established causes or corroborative factors for most psychiatric classifications, leaving them unvalidated (Zachar & Jablensky 2014, p.9). Major figures involved in formulating DSM-5 wished "to transcend the limitations of the current DSM paradigm and encourage a research agenda that goes beyond our current ways of thinking" (Kupfer, First & Regier 2002, p.xix).[1] Many other sources could be drawn upon to highlight such concerns.

1 DSM refers to the Diagnostic and Statistical Manual. It is published by the American Psychiatric Association, and is currently in its 5th edition. DSM lists many individual psychiatric classifications, providing diagnostic criteria and describing other information like treatment responses, course and family studies. It is employed in many different countries and the World Health Organisation diagnostic manual, the International Classification of Diseases, generally reflects DSM.

0.2 Importance of the Problem

Questions of belief in scientific concepts are especially important for psychiatry compared to most sciences. Anti-realist philosophers of science may disbelieve in electrons but do not argue notions of electrons need immediate replacement, taking electrons as adequate provisional mathematical models accounting for phenomena. In contrast, psychiatric classifications not meriting belief could have huge ramifications. A psychiatric classification can influence decisions over treatment, over termination of foetuses and over sectioning an individual. Being diagnosed can influence an individual's self-perception and perception of how they relate to others. Psychiatric classifications being somehow false or untrue would raise serious questions over how much they should influence decision making. More is at stake when worries are raised regarding psychiatric classifications compared to concerns over the reality of electrons.

Many advocate the replacement of existing psychiatric classifications with new and superior ones. This has a long history (see Aragona (2014)) but is most prominent today with RDoC [Research Domain Criteria]. RDoC fears falsity of existing psychiatric classifications negatively influences causal investigation. Consequently, RDoC seeks to circumnavigate existing classifications by directly investigating connections between causes and symptoms, with the long term intent of producing new and superior classifications (Cuthbert & Insel 2013, p.4). RDoC is run by the National Institute for Mental Health. This is part of the National Institute of Health, an agency of the United States Department of Health and Human Services. Thus, a major governmental organisation of the country which typically sets worldwide trends in psychiatry is funding a project specifically intending to replace existing psychiatric classifications. It is reorienting research grants away from DSM based research to RDoC based research (Casey et al 2013, p.813). The major assumptions driving RDoC's methodology is that existing psychiatric classifications are deeply flawed. Scepticism over psychiatric classifications has major implications for policy relating to psychiatric research.

0.3 Major aim of thesis

I will establish criteria under which an individual psychiatric classification (rather than a classificatory scheme like DSM) merits belief and show autism meets those criteria. I frame my discussion in terms of scientific realism, aiming to establish criteria under which we should consider a psychiatric classification as true (or approximately true) and hence worthy of belief. I primarily focus upon one psychiatric classification, autism, as a case study of where belief is justified under the approach I develop.

I employ notions of scientific realism rather than validity. Generally, notions of validity are not defined. In rare instances where definitions are provided they are typically based around notions of causation (for example, Kendell & Jablensky (2003, p.7) and Murphy (2014a, p.62)). This is problematic because those advocating such definitions typically do not explain why causation is important for belief. In contrast, the scientific realism I employ sees causation as important, but not all important, for belief. Additionally, which notions of causation are employed is not typically discussed by those offering such definitions but, I will argue, this has important consequences for what is considered real. The particular type of causation and notions of scientific realism I endorse will be fully outlined; nothing is gained by framing my argument in terms of validity since validation is both imprecise and implicitly seems to make claims which I do not commit to.

I will explore arguments that suggest we should be sceptical about psychiatric classifications and qualify them, describing situations under which those arguments are inapplicable. I will employ autism as a case study to show an instance where sceptical arguments are inapplicable. Doing this only counters scepticism rather than actually establishing grounds for belief. Consequently, I shall develop new criteria under which a psychiatric classification would merit belief, under which it should be taken as true. I will then show these criteria are applicable to autism. I do not claim my criteria for belief will be met by all psychiatric classifications and do not claim they will only be met by autism. The criteria will justify belief for some existing psychiatric classifications and not others, equally, it will likely justify belief over some future yet to be developed psychiatric classifications but not for others. It is a new route to belief and shown to be applicable to autism; its applicability elsewhere will need investigation outside these pages.

0.4 Initial hunch and initial development

I first contemplated that autism merits belief after reading *The Siege* by Clara Clarebourne Park (1967). Park's daughter was diagnosed with autism in 1961 and *The Siege* recounts Park's attempts to cure her. Park developed the theory that autism was a severe psychological inability to cope with disappointment. By never trying anything new the daughter would never face disappointment, so she lived in her own world and the family conducted their siege to break her out of that world. Describing events of 50 years ago when the concept of autism was relatively new, written by someone with no scientific or medical training and by someone who holds clearly false beliefs about autism, intuitively, one would consider *The Siege* a historical curiosity rather than credible scientific text. Despite this, I was deeply struck by how recognisable the symptoms mentioned were to the modern eye. Park appeared to be describing the modern symptoms associated with autism despite having discredited beliefs about autism. I drew parallels with 1700s theories about electricity. Scientists in Russia and Britain endorsed a Newtonian understanding of electricity whilst the European continent largely endorsed a radically ontologically different Cartesian understanding of electricity. Despite having radically different explanations of what electricity was, what made up electricity and how electricity worked, they all came up with the same experimental results (Christie 2006). I wondered if autism was similarly epistemically robust, the psychiatric classification not being influenced by the changing explanations of autism which come and go with each generation. Was this the case then, I wondered, might this be because the concept of autism is not theoretical in the manner which speculative theories like psychoanalysis and cognitive psychology are?

Unfortunately, a strict demarcation between theory and non-theory is untenable; we cannot take the concept of autism as a-theoretical and therefore epistemically superior to theoretical explanations like psychoanalysis. The operationalism of the DSM-III (1980) famously aimed for a theory neutral psychiatry, aiming to base psychiatry on logical positivism (though operationalism was severely misunderstood (Aragona 2014, p.30; Green 1992, p.308; Zachar & Jablensky 2014, p.5)). The logical positivists argued only observations and statements purely derived

from observations were meaningful whilst all other claims were literally nonsense and did not merit belief (Carnap 1932). However, it has been long established that scientific claims cannot be directly derived from observation; scientific claims are first derived from theory rather than a-theoretical observation (Hempel 1973, p.70; Popper 1959, p.106; Quine 1951). Also well established is that observations themselves are theoretical so a divide between a-theoretical observations and theoretical claims fails (Feyerabend 1975, p.212; Kuhn 1996, p.4). The impossibility of a theory neutral psychiatry is almost universally agreed upon by philosophers of psychiatry (Cooper 2005, p.101; Murphy 2006, p.226). Given the failure of the observation – theory distinction, the symptoms of autism are theoretical. Claiming autism is epistemically superior to Park's belief in her daughter's fear of disappointment requires alternative grounds than presence or absence of theory. I was deeply acquainted with the impossibility of a theory-neutral science yet still felt committed to the notion that autism somehow had a different theoretical status to many scientific theories.

The solution came from exploring scientific realism. Rather than trying to avoid or discount theories, scientific realists argue belief in science is justified if scientific claims are based upon sufficiently good theories (Psillos 1999, p.189). Rather than see autism as less theoretical, I wondered if instead we should see autism as a good theory. Of course, critics of psychiatry would deny psychiatry has good theories. Even if Park's theories were especially weak few would claim modern theories about autism are as good as theories in mature sciences like physics. If belief in science requires good theories then intuitively physics would pass and psychiatry would fail. Many philosophers, psychiatrists, psychologists, scientists and members of the general public would endorse this precise view: psychiatry does not generally merit belief because it is based upon unscientific claims or bad scientific claims. The only way autism would merit scientific realism is if most such criticisms are inapplicable to autism. Symptoms and classification of autism need be based on good theories to merit belief, including escaping various sceptical arguments.

0.5 Identifying Arguments

Let’s formally identify the main arguments against belief in psychiatric classifications

that I need respond to. These take two interrelated forms. Firstly, arguments against belief in psychiatric classifications. Secondly, the failure of arguments which aim to establish belief in psychiatric classifications.

0.5.1 Three arguments against belief in psychiatric classifications

There are three main anti-realist arguments in philosophy of science. They are intended, where applicable, to show scientific realism (belief in scientific concepts) is not justified.

The first argument against belief is the pessimistic meta induction. Past and potential future changes suggest modern psychiatric classifications are on shaky ground and are likely to change. This likelihood of being abandoned undermines reliable belief in them. Throughout history scientists have believed in theories, often on extremely good evidence, which turned out false, suggesting good evidence is insufficient for belief and our current theories will turn out false (Laudan 1981, p.33). Related arguments in philosophy of psychiatry are common, with regular complaints that psychiatric classification keeps changing. Historians of psychiatry have outlined numerous historical changes to classifications. For example, historical study of schizophrenia suggests psychiatric classifications are often initially conceptualized on weak grounds (Bentall 1992a, p.49; Boyle 1990, p.169) and undergo many changes (Bentall 2004, p.39; Boyle 1990, p.169). Such changes are likely to continue.

> "There is no reason to suppose that this process of grouping and re-grouping phenomena, of enriching, abandoning and adding constructs [classifications], will not continue indefinitely... The twenty-second century's medical constructs will no doubt have as varied conceptual bases as have ours" (Boyle 1990, p.79).

Schizophrenia may be abandoned like phlogiston or the aether (Bentall 1992b, p.287) and Bentall suggests changing notions of schizophrenia reduces confidence that psychiatrists are getting things right (2004, p.39). Berrios suggests the changing history of psychiatric classifications raises questions about them being natural kinds

(2015, p.31). Advances in genetics and neuroscience may mean we abandon schizophrenia and manic depression (Schaffner 2012, p.181). Current categorical approaches of DSM may not last due to gene research and changes in socio-economic environment (Horwitz 2002, p.228). Past and potential future changes are often taken to suggest current psychiatric classification do not merit belief.

The second argument against belief is underdetermination. This is where two different theories can equally well explain the evidence; therefore there is no justification for assigning belief to one theory rather than the other (Stanford 2006, p.8). If more than one theory is equally compatible with the evidence then scientists cannot employ evidence to decide which theory is correct. Related arguments in philosophy of psychiatry are common, with the suggestion that current psychiatric classifications are just one way of classifying mentally ill people. Murphy writes, "what evidence do we have that current practice in psychiatry produces good outcomes, outcomes that couldn't be matched by alternative nosologies that start from a completely different set of premises?" (Murphy 2006, p.10). Giere notes that underdetermination fits the social constructivist argument well (Giere, 1999 p.237) and philosophers of psychiatry have indeed often suggested psychiatric classifications are arbitrary constructs, formulated on personal opinion rather than clear scientific evidence. Pointing to the multiple possible approaches to schizophrenia, Bentall writes that "[i]f schizophrenia is a valid scientific concept, then at the very least psychiatrists will need to answer the question. Which set of criteria [symptoms forming a classification] are the right ones?" (1992a, p.28). However, there is little means of determining which classification is correct (Bentall 2004, p.65). The possibility of constructing alternative psychiatric classifications is taken to suggest we lack reason to believe current psychiatric classifications are the correct ones.

The third argument against belief is theory-laden nature of evidence. This is where evidence employed to construct scientific theories itself partly depends upon scientific theories, potentially mistaken ones, which can result in substantial negative epistemic consequences (Bogen 2013). Note that it is generally accepted most or perhaps all science is theory-laden. This is only concerning when the theories involved are weak or when the process of interpreting evidence from (good or bad) theories is unreliable. This also has parallels in philosophy of psychiatry, it being commonly argued that psychiatric classifications are overly reliant upon false theories.

Paul Hoff describes psychiatry as historically a series of "repeated collapses into "single message mythologies" " (Fulford et al 2013, p.2). There are many competing theoretical approaches to mental illness, such as psychoanalysis, cognitive psychology, neuroscience and genetics. Kendler asks, if psychiatry is going to be based upon a theory then which of the above theories do we go with (2012, p.xv; also Bentall 1992a, p.28). Not only are we unsure which theory is correct, potentially all of them are false. Cooper believes "it is likely that much current psychiatric theory is mistaken" (2005, p.77). Most psychiatric theories have major flaws, making them highly limited or completely false. Alternatively, more than one theory may be correct but this situation would create immense challenges: very few psychiatric classifications have a single, clear theoretical explanation (Kendler 2005a, p.434). Instead, we must employ multiple theories, being sure each theory is applied only to its relevant domain alongside needing understand how phenomena described by different theories interact, the "difficulty of which is hard to overestimate" (Kendler 2005b, p.438). The worrying consequence is that false or inadequate theories might cause psychiatrists to make false observations, theory influencing and thus distorting the observations used to form classifications. "[P]sychiatrists' false beliefs can be expected to distort their observations of their patients and prevent them from seeing the true similarities and differences between types of mental disorder" (Cooper 2005, p.77). For example, Bentall thinks the multiplicity of theories about schizophrenia suggests we should not be confident over notions of schizophrenia (Bentall 2004, p.xiv). Reliance upon false theories undermines belief in existing psychiatric classifications.

0.5.2 The inapplicability of arguments for belief in psychiatric classifications

I will now consider two arguments employed by psychiatrists and philosophers of psychiatry which suggest conditions under which psychiatric classifications merit belief. As is recognised by those who employ such arguments, these conditions are rarely met in psychiatry. Hence the absence of their success can be taken as reason to disbelieve in (or at least remain agnostic about) psychiatric classifications.

The first, and most popular, argument for belief is that psychiatric

classifications might be based on causal knowledge. Murphy thinks "nosology must be based on causal explanations of what is being classified" (2006, p.5). Claiming psychiatry currently is unsuccessful, he says

> "my solution [to the lack of success], in essence, is to borrow from the neighboring science[s]... my basic project assumes that they [other sciences] are in good enough shape for us to conclude that if psychiatry, which studies the same subject matter (human beings), aims for consilience with the sciences of the mind/brain, and adopts their best practices, it will be on a sounder footing" (Murphy 2006, p.11).

Modern psychiatrists often also believe weaknesses with classifications mean we should base psychiatry on explanations, such as genetics (Andreasen 1995, p. 162; Pies 2008, p.49; Smolik 1999 p.188). Psychologists like Baron-Cohen (1997, p.137) and Happé (1994, p.98) believe autism should be formulated on causes. A related argument is taking corroborations between psychiatric classifications and other factors, such as from treatment responses and family studies, as a means to belief (Kincaird 2014, p.151; Meehl 1995, p.273). The greater the responses to specific treatment and the greater co-occurrence in families, the more likelihood diagnosed individuals have shared underlying causes. However, almost all who endorse this causal or corroborative argument believe adequately strong causes or corroborative evidence have not been sufficiently found to establish belief over most existing psychiatric classifications.

The second argument is that psychiatric classifications which take a particular form merit belief. For Cooper, psychiatric classifications ideally group patients based upon similar symptoms and sometimes

> "these similarities will be theoretically important, and in some cases patients who are grouped together will be alike in fundamental ways… If we take cases of mental disorders as our domain and plot them onto a multidimensional quality space (as in cluster analysis) then we will find clusters of similar cases. If we focus on the right properties, then the clusters that such a process generates will be inductively powerful"

Psychiatric classifications like this she calls natural kinds and considers these as meriting belief. Additionally, we can believe something is a natural kind without identifying its causes. Psychiatric classifications which are natural kinds mean individuals with the psychiatric classification (when properly diagnosed) have some fundamental similarities, grounding further inductive and explanatory claims. Cooper takes biological species to be paradigmatic examples of natural kinds; biological species are real because members of a species have theoretically important similarity due to shared causes. She similarly links kinds with being real in psychiatry (Cooper 2012b, p.38); psychiatric classification resembling biological species will be getting something right about the world so merit belief. However, Cooper believes weaknesses in psychiatric practice means most psychiatric classifications do not describe natural kinds. Related ideas are expressed by Kendell and Jablensky. They see psychiatric classification with a zone of rarity – where the symptom pattern of one psychiatric classification has little overlap with other psychiatric classifications – as meriting belief (Kendell & Jablensky 2003, p.8). A zone of rarity allows an inference that the underlying biology of the psychiatric classification is unique, not present in other psychiatric classifications, which legitimises belief. However, very few psychiatric classifications have zones of rarity (Kendell & Jablensky 2003, p.8).

0.5.3 Suitability of autism as an example of a contested psychiatric diagnosis

This thesis takes autism as a case study for exploring questions of scientific realism in psychiatry. DSM-V takes autism as a disorder with symptoms present from early development which impair areas of functioning of the child or adult. The symptoms of autism are broken down into two main areas. Firstly, deficient social communication and interaction, including both verbal and non-verbal communications. Secondly, repetitive behaviour, interests and activities, including rigid thinking and hyperactive or hyporeactive sensory input. The symptoms must be not better accounted for by intellectual disability though intellectual disability can co-occur with autism (APA 2013, p.50-51).

Autism is a major psychiatric classification and, as is well known, autism does

not have one single identified cause. For example, it is currently being linked to hundreds of genes (Devlin & Scherer 2012, p.233). Recent commentators have expressed disbelief over autism on similar grounds to those listed above. For example, the first article of a recent collection of papers entitled *The Philosophy of Autism,* written by an editor of the book, highlights this clearly. Cushing points out that not all autistic people have all the symptoms of autism and some non-autistic people have some symptoms of autism (Cushing 2013, p.19). Therefore, the symptoms alone are not enough to determine who is autistic and it is an assumption that all people labeled autistic share the same syndrome (Cushing 2013, p.22). Cushing suggests there needs to be something deeper to autism than just behaviour (2013, p.23) but he shows that psychology, genetics and neuroscience all fail to provide something deeper (2013, p.25-38). Lacking any other candidate for what autism might be, he sees the DSM-5 notion of autism as "either arbitrary or solely politically/economically motivated" (2013, p.38). On this basis,

> "there is no such thing as "autism" if we are to understand it on the model of something like Down syndrome. At best there are several conditions that can occur independently of each other but seem to co-occur, that can vary significantly in severity, and that seem to cluster in families, but not necessarily always" (Cushing 2013, p.38-39).

From this he claims that "progress can only come from abandoning it [the classification of autism] and starting from scratch" (Cushing 2013, p.41). Cushing

> "propose[s] that we focus instead on specific deficiencies, like sensory processing disorders, communication difficulties or food sensitivities and stop trying to cluster them together as something called "autism" " (2013, p.39).

Similar ideas are expressed in Sammi Timimi's recent book *The Myth of Autism.* The lack of a causal basis for autism (Timimi et al 2011, p.139) means "the field of autism rests on ideological assumptions, *not* scientific evidence" (Timimi et al 2011, p.4, emphasis original). Instead,

> "the autism spectrum has become a catch-all metaphor for focusing on a disparate range of behaviours that suggest a lack of the type of social and emotional competences thought to be necessary for the functioning of society dominated by neo-liberal economic and political foundations" (Timimi et al 2011, p.7)

Again, the absent causal basis means some extra-scientific factors are used to formulate autism, leaving autism illegitimate science. Autism thus serves well as a case study for my project; it's an important psychiatric classification and its legitimacy is contested.

0.6 Alternative starting point and argument

My argument will require a novel account of what symptoms are. I draw upon an important late 20th century development in philosophy of science, one not yet applied to philosophy of psychiatry. Traditionally, philosophy of science demarcates sharply between data and theory. Data is the product of experiments and traditionally theories were portrayed as inductive summaries of that data (even if they often or always also included elements beyond the data). An influential 1988 paper by Bogen & Woodward argued this picture missed out an important step. Bogen & Woodward claim theories do not summarise data but instead describe something else which they call phenomena. Typically, data is the product of many different causal factors which are specific to that moment. For example,

> "the outcome of any given application of a thermometer to a lead sample depends not only on the melting point of lead, but also on its purity, on the workings of the thermometer, on the way it applied and read, on interactions between the initial temperature of the thermometer and that of the sample, and a variety of other background conditions" (Bogen & Woodward 1988, p.309).

When building theories we desire a more general figure than is provided by specific experiments, one not tied to specific causal factors present in specific instances of the experiment. Bogen & Woodward suggest the traditional picture of data and theories needs to be supplemented by phenomena, an additional step lying between them. This notion of phenomena is a more abstracted, more generalised notion than is given by individual experiments. Thus whilst different experiments might provide results of 3.28, 3.21, 3.33, etc., scientists might agree upon phenomena as 3.27, a general figure abstracted away from specific experiments. Theories account for this more generalised notion of phenomena rather than specific instances of data.

Following numerous philosophers of science (Psillos 2011a, p.7; Teller 2010, p.825) I link this picture of data, phenomena and theories with Giere's account of theories as high level models (Giere 1994, p.293). According to Giere, scientific theories (a term which includes scientific laws) provide very abstract probabilistic descriptions of phenomena, detailing how under various conditions probabilities of which and to what magnitude phenomena will occur. This abstract model of phenomena can be applied to specific situations which reduces the generality and narrows down the probabilities of what phenomena will occur and to what magnitude. Thus phenomena is a link in a chain of levels of abstraction. Data is tied to specific instances thus lacks abstractness, phenomena is a generalised summary which lacks the specificity of data and theories are more generalised probabilistic statements about phenomena.

Phenomena can be derived from a data driven bottom up approach or a theory driven top down approach. Many different individual instances of data can be analysed statistically to derive an abstracted and generalised phenomena. Alternatively, one can use theory to derive phenomena. For example, theories about molecules suggest that, when arranged into the configuration we call lead, the intermolecular bonds will require 327.5 degrees to break them.

I apply all this to psychiatry. People exhibit instances of behaviour, we should consider these as data. However, these instances of data are not identical to symptoms. Different specific situations will cause low social skills to manifest and each manifestation will involve different words being spoken. In contrast, the symptom low social skills has an abstracted generality not present in specific behavioural manifestations of low social skills. We should therefore see symptoms as phenomena,

an abstracted generalised notion lacking the specific details of behavioural instances. I then argue we should understand psychiatric classifications on Giere's account of scientific theories, high level abstract models with abstract probabilistic statements about symptoms. Formulating symptoms can be bottom up behaviour driven through statistical analysis of behaviour. This is the typical understanding of how we formulate symptoms. However, I also argue symptoms can be formulated through a top down classification driven approach. I show classifications can set populations from which statistically significant behaviour is derived. Different behaviour can reach statistical significance when employing different classifications, thereby entailing different symptoms. I argue this approach produces symptoms of greater relevance and therefore increases their inductiveness. This strengthens psychiatric classifications inductively, making them more theoretically virtuous and therefore more greatly meriting belief.

This alternative picture of what symptoms are and how they can be formulated shows symptoms can be inductive without strong underlying causes. This removes the need for psychiatric classifications to have strong underlying causes to be scientific models of significant worth. This opens up the possibility of psychiatric classifications meriting belief in the absence of strong underlying causes.

I re-orientate questions of the grounds of belief away from existing approaches found in psychiatry and philosophy of psychiatry. Existing approaches often see causes (directly found or inferred) as the sole means to legitimise belief over psychiatric classifications. This is a cause first approach, whereby belief primarily depends upon establishing what has been caused by the causal structure of the world. I offer an alternative notion of reality. I see reality as a series of regularities (see Psillos 2002, p.293) . These regularities have probabilistic relationships between them. Scientific theories are worthy of belief to the degree they maximise information about these regularities. Information is maximised by theoretical virtues. There are two main theoretical virtues, unifications and causation (see Psillos 2002, p.149 & p.281-283) which encapsulate other theoretical virtues like simplicity, mechanisms and coherence with background beliefs. In some situations either or both of these can deliver belief over a scientific theory. Thus philosophers of psychiatry have been right to believe causes are a route to belief. They are wrong, however, to believe they are the sole route to belief. Unifications, whereby as few theories as possible cover as much

phenomena as possible as stringently as possible, are also routes to belief. Unifications take seemingly otherwise dissimilar phenomena and show

> "connections, common patterns, in what initially appeared to be different situations... *Science advances our understanding of nature by showing us how to derive descriptions of many phenomena, using the same patterns of derivation again and again, and, in demonstrating this, it teaches us how to reduce the number of types of facts we have to accept as ultimate (or brute* [unexplained]*)*" (Kitcher 1989, p.432 emphasis original).

By using theories to link together phenomena we reduce the number of unconnected facts, increasing "systemitization of our beliefs" (Kitcher 1989, p.476; also Psillos 2002, p.11). Systemitization via unifications is increased by explaining as much as possible by as few theories as possible; theories significantly contributing to high systematisation merit belief (providing our attribution of the unification is epistemologically reliable, as discussed below). Additionally, sometimes unifications and causation often conflict with one another. This means focusing heavily on causation can actually reduce the strength of the theoretical virtues exhibited, thus reducing belief. I will support these claims by discussing various metaphysical positions. I suggest much discussion over belief in psychiatric classifications has adopted an implicit neo-Aristoleanism whereas the notion of belief I discuss is compatible with neo-Humeanism, neo-Kantianism and pragmatism. Also, I shall show that modern scientific evidence does not usually support notions of psychiatric classifications as having strong, unique causes. I claim an unification approach fits much better with the nature of reality in the domain of psychiatry.

Existing approaches have also focused more on metaphysical questions rather than epistemic questions. I show the importance of epistemic questions, showing how inferences are only justified when made under conditions that generally produce reliable beliefs. Establishing this situation requires two things. Firstly, we must be sure certain problems will not arise, via combating the three main anti-realist arguments (these are the pessimistic meta induction, underdetermination and (where weak background theories are involved) theory laden-nature of evidence). We also must be

sure certain conditions are present, namely epistemologically reliable ones. Typically, realists employ the no miracles argument as evidence that these conditions are present. This argues that there are only two explanations of miracle like predictions, either the theory is true or a miracle has occurred. Since miracles are not allowed in philosophy, the theory must be true. Given the rarity of miracle like predictions in psychiatry the argument has little applicability in philosophy of psychiatry. Instead, I consider the specific conditions under which inferences in psychiatry are made and show they often carry relatively little epistemic risk.

Philosophers disagree about the status of theoretical virtues, disagreeing over whether theoretical virtues are testable and, if so, whether they relate to the mind-independent structure of the world. These issues relate to whether a neo-Humean, neo-Kantian or pragmatist perspective is adopted; I remain neutral on this question and so my argument can fit all three perspectives. Rather, I merely argue we have good epistemological reasons to believe the theoretical virtues are present. Scientists can be mistaken about the presence of theoretical virtues. For example, they might believe a theory is simple only because they failed to gather evidence or disregarded evidence which a simple theory could not accommodate. This problem can be the case regardless of whether one takes a neo-Humean, neo-Kantian or pragmatist approach to theoretical virtues (though this is not necessarily true of neo-Aristoleanism). Beyond providing alternative approaches to the currently popular implicit neo-Aristoleanism in philosophy of psychiatry I do not need establish the metaphysical status of theoretical virtues to provide argument that theoretical virtues are present rather than merely apparent.

I argue modern autism merits belief because (we have good reason to believe) it strongly exhibits the theoretical virtue unification. The psychiatric classification autism substantially reduces the number of symptoms, and probabilistic relationships between symptoms, which would otherwise remain unconnected by modern psychiatry. Autism strongly exhibits the theoretical virtue unification because it unifies together so many symptoms which significantly probabilistically co-occur and does so through a single classification. It increases the systematisation of psychiatry by unifying many symptoms, and their probabilistic relationships, whilst only adding a single additional classification. Also, since theoretical virtues can conflict, modifying the classification to greater increase the strength of causes may greater

decrease strength of unifications, resulting in a classification or classifications which are overall less theoretically virtuous. I do not, however, claim modern autism is the ultimate best possible unification. The highest truth is given by the theory with the best possible balance of theoretical virtues. Modern autism has approximate truth to the degree to which its unification is part of the best possible balance of theoretical virtues. By modern autism I mean both DSM-IV autism (an overarching classification plus specific subtypes like Asperger's syndrome and childhood disintegrative disorder) and DSM-5 autism (a single classification without subtypes). Both DSM-IV and DSM-5 autism merit belief because both attain significant approximate truth through unifications; establishing which has higher approximate truth will require far more clinical and scientific studies of DSM-5 (only published in 2013). The DSM-IV and DSM-5 approaches respectively resemble two earlier approaches commonly employed between the late 1940s to the late 1970s. Kanner's approach resembles DSM-IV and Bender's resembles DSM-5. Both these also merit belief, to an extent close to that merited by modern autism. I will suggest their approximate truth is slightly lower than modern autism but more scientific evidence needs gathering to firmly establish this. None of this compromises belief over DSM-5 autism; assuming Kanner's approach, Bender's approach or DSM-IV autism has higher approximate truth than DSM-5 autism then this just entails that DSM-5 has slightly lower approximate truth than a very similar alternative approach.

To summarise, reality is a series of regularities and the probabilistic relationships between them. Scientific theories deserve greater belief the greater they exhibit theoretical virtues which maximise information about those regularities and their probabilistic relationships. We also need suitable epistemic conditions to reliably believe we have accurately detected those probabilistic relationships. I will apply these arguments to autism. I show autism strongly exhibits unifications, covering a wide range of symptoms in a stringent way. I will show we have good epistemological reasons to believe autism is informative of those regularities. I also show it successfully avoids arguments which threaten epistemic doubt, specifically showing it avoids the pessimistic meta-induction, underdetermination and negative effect of theory laden nature of evidence. Reformulating autism on causes would increase the strength of the theoretical virtue causation but may, depending on the causes established, reduce the strength of the theoretical virtue unification by splitting autism

into numerous classification, potentially an epistemologically worse situation. Autism shows how a psychiatric classification can merit belief in the absence of causes.

0.7 Chapter summaries

Chapter one analyses the history of autism, from the mid-1930s to the late 1970s, to establish if sufficient historical continuity is present to avoid the pessimistic meta induction. From around the late 1940s clusters of symptoms are described which look highly recognisable from a modern perspective. These clustered in both less severe and more severe forms, the less severe form was associated with social deficits and the more severe was associated with low intellect. Many psychiatrists at the time also envisaged these as lying on a continuum, notions analogous to an autistic spectrum. The two most important figures, Kanner and Bender, employed classificatory approaches which respectively had great similarity to DSM-IV autism and DSM-V. This leaves the pessimistic meta induction largely inapplicable.

Chapter two covers the late 1970s to the mid-1980s and uses material from that era to show that avoids the underdetermination argument. During this period DSM-III autism was formulated and implemented. This approach to autism is very different to notions employed before hand and very different to notions employed from DSM-III-R onwards, potentially raising an underdetermination worry. Underdetermination requires two theories which are equally successful empirically and theoretically. Showing flaws in the development of DSM-III autism and showing its inadequacies as a classification means it is not an instance of underdetermination. Additionally, I briefly describe why these historical changes do not entail the pessimistic meta induction.

Chapter three addresses the theory-laden nature of evidence by comparing the symptoms and classifications employed under psychoanalytical and cognitive psychological accounts of autism. These are the two best candidates for a classic theory-laden account where changing theoretical views modify observations. I argue most symptoms of autism were described and quite similar approaches to the classification were employed by both psychoanalysts and cognitive psychologists. This means we lack reason to believe either the symptoms or the classification is

theory laden by either theory. In contrast, many causal claims are theory-laden by one of the theories, leaving those causal claims resting on potentially flawed theories.

Chapter four outlines an alternative metaphysical picture. Metaphysical accounts influence epistemic accounts. Some philosophers employ metaphysical accounts which emphasise causes and consequently demand epistemological arguments showing psychiatric classifications have high causal unity. Such epistemological arguments are largely inapplicable to most psychiatric classification. I provide an alternative metaphysical picture of how things exist, taking regularities as real regardless of whether those regularities have high causal unity. By not demanding high causal unity metaphysically there is also no requirement to demand it epistemologically. Consequently, psychiatric classifications can merit belief despite lacking a strong causal basis. I draw upon accounts of scientific laws whereby laws describe regularities without having to account for causes. Similarly, I portray psychiatric classifications as a form of scientific law, whereby abstract high level laws guide assigning the regularities of less abstract lower level laws, each more detailed law sharpening descriptions of probabilistic relationships between symptoms. Note that I do not employ laws as exceptionless regularities true in all possible worlds. My alternative metaphysical picture of reality as regularities gives psychiatric classification the role of scientific laws and opens up new epistemic arguments.

Chapter fives highlights a previously unnoticed role that psychiatric classifications can play. Generally, it is believed psychiatric symptoms can be known independently of psychiatric classifications. Psychiatric classification group symptoms but we can formulate symptoms without employing psychiatric classifications. I show this is not always the case, how identical behaviour can be formulated as different symptoms depending upon which classifications an individual has. Additionally, I argue this process strengthens psychiatric classifications inductively, thereby making psychiatric classifications stronger laws. It also makes attempts to circumnavigate psychiatric classifications by just focusing on symptoms problematic.

Chapter six supplies the epistemological arguments which legitimate belief over autism. I suggest the current focus on causes is based in an implicit neo-Aristotelianism, whereas my focus on regularities allows realism through neo-Humeanism, neo-Kantian and pragmatism. I outline three steps to belief, firstly an inference to the best explanation, secondly, the inference to the best explanation being

made under reliable conditions and thirdly not exceeding a personal limit on epistemic risk. The strength of unifications means autism passes the inference to the best explanation. Also, since unifications and causal knowledge can conflict, strengthening causal knowledge may actually decrease the strength of unifications, meaning focusing on causes may leave a psychiatric classification no longer the best explanation. Inferences to the best explanation are justified when made under reliable conditions. The level of access psychiatrists have to autism, the scale of the inferences involved and the level of idealisation present are much less than often occurs in other sciences. We can take the inference to the best explanation as reliable because it is made on safe grounds, involving good access and unproblematic inferential approaches. I outline a plausible, moderate stance on epistemic risk, one between an optimist and a pessimist, and argue autism does not exceed that level of epistemic risk. This means autism merits belief.

1.0 Chapter 1 – Historical Continuities of Autism

1.1 Introduction

The pessimistic meta-induction (PMI) argues scientists historically believed in their theories, often on extremely good evidence, yet those theories turned out false. Inductively, this suggests modern theories will also turn out to be false. In this chapter I show autism has sufficient historical continuity to make the pessimistic meta induction inapplicable.

This chapter will need counter many recent histories of autism which argue for conceptual discontinuity across the history of thinking about autism (Evans 2013, p.15; Eyal et al 2010, p.236; Jacobson 2010, p.442; Verheoff 2013, p.452). They correctly show that ideas of autism from the 1940s to the 1970s involved many concepts now abandoned, specifically causal and theoretical claims. Also, they have shown major changes to the socio-medical environment. None of this I challenge. Rather, I focus upon their account of changing clinical pictures (descriptions of symptoms and descriptions of how symptoms cluster). These authors argue that autism was re-conceptualised in the 1980s; DSM-III (1980) linked autism with mental retardation and the DSM-III-R (1987) conceptualised high functioning autism as social impairment. Also, both low and high functioning autism came to be linked together by the idea of an autistic spectrum where autism varied in severity. Some historians argue these three concepts, still very central to notions of autism today, were absent or far from central in thinking about autism from the 1940s to the 1970s. By contrast, I will argue autism as social impairment, autism coexisting with mental retardation and autism as varying massively in severity are concepts with deeper historical roots. I will show these three concepts were widely employed and very important from the early 1950s, at least two decades earlier than historians typically describe, and pre-dating many of the theoretical, classificatory and socio-economic changes of the late 1970s or early 1980s that historians often claim produced or

heavily influenced 1980s concepts of autism.

The chapter looks at two historical periods. Firstly, 1925 to 1943. I consider the classifications employed prior to autism being conceptualised in 1943 and highlight how autism filled a gap within the existing classificatory schemes. The introduction of autism made it possible to better diagnose some patients. Secondly, 1943 to 1978. Here autism, alongside childhood schizophrenia, underwent development and from around the early 1950s both classifications have substantial continuities with modern autism. This period is especially important because many recent histories argue for discontinuity by comparing Leo Kanner's 1943 account of autism with modern autism. I argue these historians have overlooked important evidence when analysing Kanner. While historians correctly highlight substantial differences between Kanner's 1943 initial account of autism and modern autism, Kanner's account of autism changed and his 1943 paper is not representative of his later writings which are largely unexplored by recent histories of autism. These link autism with social impairment and mental retardation. Additionally, childhood schizophrenia was diagnosed far more than autism and provided clinical pictures remarkably similar to modern autism, something also largely unexplored by recent histories of autism. Finally, both autism and childhood schizophrenia were widely recognized as varying greatly in severity and some child psychiatrists explicitly conceptualised autism and childhood schizophrenia as lying on a spectrum. Thus many concepts typically considered to only become central in the 1980s were present and widely employed by Kanner and others during the 1950s to 1970s. This matters because the PMI requires there to be discontinuities in mature scientific theories. Though the classifications from the 1950s to late 1970s are not identical with modern autism, they have sufficient similarity to escape PMI.

I start by outlining the pessimistic meta induction, conceptual continuity and discontinuity. I then take both historical phases in turn. I discuss how the PMI relates to each historical period. I then clarify PMI to show it is a limited threat. Finally, I consider the implications of Hacking's looping affect for a PMI over autism.

1.2 Discontinuity and the Pessimistic Meta Induction

1.2.1 Discontinuity and history of autism

Whilst historians have convincingly argued for discontinuity over many aspects of the history of autism I aim to show discontinuity arguments fail for three central concepts of autism in the 1980s.[2] Autism as social disability, autism coexisting with mental retardation and autism varying greatly in severity are central to modern autism and all three have been recently targeted by historians making discontinuity arguments. In contrast, I argue all three were all present, widely employed and very important from the early 1950s. These were not revolutionary *additions* in the 1980s, rather, they were present and very important from the 1950s and gained additional importance in the 1980s through the *subtractions* of many now abandoned notions. Additionally, 1950s to 1970s childhood schizophrenia – which was closely associated with autism – covered a significantly broader clinical picture than modern autism but within that diversity often occurred clinical pictures very similar to modern low and high functioning autism. Naive continuity over all aspects of autism adopted by many scientific textbooks is certainly untenable. Nonetheless, the importance of those three concepts from the early 1950s means they are not discontinuous with earlier decades in spite of the other changes which occurred in the 1980s.

Historians often employ discontinuity accounts to question current approaches to autism. Verheoff takes discontinuity accounts as showing "the historicity, proportionality and plurality of knowledge and truth about autism... [acknowledging this] creates space for other possible perspectives and conceptualizations of autism in the present and future" (2013, p.455). This approach also seems present, if stated less directly, in the other recent histories of autism, specifically Evans (2013, p.24), Jacobsen (2010, p.447) and Eyal et al (2010, p.263). This is especially relevant if current concepts were (consciously or unconsciously) chosen due to changes in wider theoretical, classificatory or socio-economic trends rather than firm evidence. This scenario is argued for by many recent histories of autism (Evans 2013, p.15; Eyal et al 2010, p.236; Jacobson 2010, p.442; Nadesan 2005, p.184; Verheoff 2013, p.452).

2 See Hollin (2014) for development of cognitive psychological theories, Raz (2014) for influence of notions of sensory deprivations; Evans (2013) for changing notions about abnormal thinking; Verheoff (2013) for changing views on perceptual and cognitive differences; see Verhoeff (2014), Silverman (2012), Jacobson (2010), Evans (2013) Feinstein (2010), Gil el at (2010), Nadesan (2005) for both changing views on causal factors and for wider changes to the socio-cultural-political medical setting.

Social constructivists have made such arguments about psychiatric classifications more generally (Bentall 2004, p.39; Boyle 1990, p.169). Whilst such discontinuity arguments are certainly applicable to many concepts of autism, my history shows that such discontinuity arguments are much harder to apply to the three central concepts I focus upon. This is not to suggest wider theoretical, classificatory or socio-economic trends have not been influential, only that the influence has not prevented continuity occurring over some parts of autism. If changes to, for example, theoretical beliefs over what causes autism or changes to funding of diagnostic services did not result in changes to symptoms descriptions then those changes would not be relevant for historical continuity over symptoms.

1.2.2 Pessimistic Meta Induction

Let's consider this philosophically. Laudan writes that

> "what the history of science offers us is a plethora of theories which were both successful and (so far as we can judge) non-referential with respect to many of their central explanatory concepts" (1981, p.33).

He provides many examples, including the crystalline spheres, phlogiston, caloric theory of heat, electromagnetic aether and optical aether. There exist many instances where scientists believed in extremely successful theories yet those theories turned out wrong, suggesting inductively successful theories we believe in will also turn out false (a pessimistic meta-induction (PMI)), therefore modern theories do not merit belief.

There are some common responses to PMI. Firstly, deny it works inductively by reducing the inductive base. Laudan says his infamous list of false theories "could be extended *ad nauseam*" (Laudan 1981, p.33 emphasis original) whereas Psillos claims "theoretical discontinuities in theory-change were neither as widespread nor as radical as Laudan has suggested" (Psillos 1999, p.103). If few theories were mistaken historically then the induction fails. Unfortunately, the ratio between historically false theories and existing successful theories is unknown, leaving the size of the inductive basis unknown (Magnus & Callendar 2004, p.331). PMI remains an active threat

which cannot be countered by simply denying it has a strong inductive base. More promising is limiting the inductive base by demanding only mature scientific theories count, accepting scientists make errors as they develop theories (Psillos 1999, p.105). Secondly, we can move from considering the inductive base of science to considering the history of specific theories, no longer wielding PMI as applying to all science. Scientists often produce theories with a limited life span but some theories have extended well beyond that life span without refutation whereas others are yet to reach the life span. If an individual theory has a long history which shows large historical continuity then PMI fails over that theory. Additionally, we can look at individual theories and establish which aspects account for phenomena and which are idle theoretical baggage. If we limit belief only to parts playing an important role and if only the idle theoretical baggage were mistaken historically then PMI fails (Psillos 1999, p.109). One might only defend a specific type of claim, such as only defending scientific laws or causal claims.

We need consider the history of autism to establish its level of historical continuity. We will only need historical continuity once a theory has become mature. Additionally, we only defend the symptoms and classification of autism but do not defend causal claims. Discussed in more detail in later chapters, there have been many different causal claims associated with autism over the decades and they have a tendency of being replaced. This makes defending causal claims over autism against PMI difficult. I specifically address this point in section 6.5.2.4. For the purposes of this chapter there is no need to establish belief or doubt over causal claims to establish historical continuity or discontinuity over symptoms.

A distinction needs making between two types of historical continuity. Firstly, there is continuity of clinical pictures. This is where the descriptions assigned to individual autistic people were very similar both historically and today. This means similar symptoms and similar clustering of symptoms. As I show, this is largely defensible for both autism and childhood schizophrenia. Secondly, continuity of the classifications itself. This is where the classification has not changed over time. However, historically there was childhood schizophrenia and its numerous subtypes (including autism) whereas today there is an autistic spectrum. The diagnosis has undergone changes. Since we require belief in the psychiatric classification autism, rather than the symptom patterns, this could be problematic. I describe why this is not

problematic at the end of this chapter and then more fully in chapter six. Briefly, PMI is only applicable when previous scientists were mistaken. PMI is not applicable simply because a theory undergoes modifications. I will show the classifications I discuss were, from the late 1940s onwards, not mistaken. They exhibit the theoretical virtue of unification, the same standards which delivers belief over autism in later chapters.

1.3 1925 to 1943

1.3.1 Leo Kanner

Leo Kanner was born in 1896 in what was Austria (currently Ukraine), studied and practised medicine in Berlin before emigrating to America in 1924. In 1930 Leo Kanner founded the first child psychiatry centre in America. In 1935 he published *Child Psychiatry*, the first English language textbook on child psychiatry. This textbook "formed the foundation on which child and adolescent psychiatry was based, not only in the United States but worldwide" (Neumärker 2003, p.216) and would be republished in later editions in 1948, 1957 and 1972. He is generally credited with providing the first English language description of autism. He also founded and became the editor of the *Journal of Autism and Childhood Schizophrenia* in 1971, the only journal devoted specifically to both disorders. To describe Kanner as being hugely important to the history of autism would be an understatement.

1.3.2 Kanner's autism and discontinuity

In 1943 Kanner published what is typically considered the first description of autism. The bulk of the paper described eleven case studies. Kanner suggested these children would better fit a new classification rather than the classifications of mental retardation or childhood schizophrenia which most these children had previously been diagnosed with (Kanner 1943, p.247-248). Kanner outlined what he called infantile autism, "an *extreme autistic aloneness*, that, whenever possible, disregards, ignores,

shuts out anything that comes to the child from the outside" (Kanner 1943, p.242, emphasis original). Kanner described intelligent children who actively shun human contact and strongly dislike intrusion into their own world. This is a considerably more severe clinical picture than that typical of modern high functioning children, who mainly struggle with social information, often avoiding social situations but rarely avoid all possible human contact and outside interference. Additionally, Kanner believed autistic children have normal intelligence (Kanner 1943, p.247), their aloneness and desire for sameness is not due to low intelligence unlike modern low functioning children. Although Kanner's 1943 paper does not provide strict diagnostic criteria, these two symptoms of aloneness and desire for sameness featured heavily and he would later consider them essential to receive a diagnosis of autism (Kanner & Eisenberg 1955, p.227). Discontinuity arguments typically refer to these symptoms or normal intelligence when discussing Kanner.

1.3.3 The addition of autism to the diagnostic field

Autism and childhood schizophrenia were first conceptualised in the 1930s and 1940s, soon after the establishment of child psychiatry. Prior to the 1920s there was no separate child psychiatry (Kanner 1959, p.582) with children diagnosed instead according to adult classifications (Bradley 1941, p.19). A major development within the growing field of child psychiatry was the hugely influential first English language textbook, Leo Kanner's *Child Psychiatry* in 1935. This pre-dated the concept of autism and the section on childhood schizophrenia only discusses adult symptoms (Kanner 1935, p.484-492) and has no symptoms specific to children. Kanner notes children with onset of schizophrenia prior to ten years show much greater variation in symptoms than those with onset before ten years (Kanner 1935, p.484). However, since no symptoms are given for this variation a description of how these younger schizophrenic children differ from adult schizophrenia is lacking.

Whilst Kanner was writing his textbook a new notion of childhood schizophrenia with symptoms different to adult symptoms was being developed. Whilst absent from Kanner's 1935 textbook, Kanner later identified Potter (1933) as the first to formulate a new notion of childhood schizophrenia that soon became a

major theory in child psychiatry (Kanner 1971a, p.17). Within a decade the first major literature review of childhood schizophrenia was published, Bradley's hugely influential *Childhood Schizophrenia* (Bradley 1941). This classificatory situation was what Kanner worked among when he introduced the new classification of autism in 1943 (see Fellowes 2015, p.2275; Kanner 1965, p.419 and Kanner 1973a, p.94 for details). Let's consider the main relevant classifications used at that time in more detail and then what autism added.

Childhood schizophrenia covered communication, emotional and thought disorders and had a wide range of symptoms. In this regard it was quite similar to modern autism. However, childhood schizophrenia in this period had a specific course (Bradley 1941, p.84) that would largely exclude autistic children. Schizophrenic children were born normal then declined, an onset which excludes any child with symptoms present from birth from being diagnosed childhood schizophrenic. Kanner says autistic children have symptoms from birth, claiming "schizophrenic children emphasize a more or less gradual change in the patients' behaviour. The [autistic] children of our group have all shown their extreme aloneness from the very beginning of life" (Kanner 1943, p.48). Present from birth demarcated autism from childhood schizophrenia.

The main alternative diagnosis for individuals who were impaired to levels equivalent to childhood schizophrenia was mental retardation. The main symptom for mental retardation was low intelligence (Kanner 1935, p.58), which would exclude Kanner's notion of autism but not modern notions of autism. Kanner demarcates autism from mental retardation on intelligence, writing that "though most of these [autistic] children were at one time or another looked upon as feebleminded, they are all unquestionably endowed with good cognitive potentialities" (Kanner 1943, p.47). Requirements of low intelligence should have excluded Kanner's autistic children from a diagnosis of mental retardation.

Speech disorders would have fit the communication difficulties of autism and unlike the other disorders listed above nothing in speech disorders should have prevented diagnosis. However, speech disorders would have only covered one of the myriad symptoms of autism.

The classifications of pre-1943 psychiatry had enough constraints and restrictions to leave considerable room for the new classification of autism to slot into.

The biggest factor to determine between the two most common diagnoses, childhood schizophrenia and mental retardation, was time of onset. Symptoms present from birth meant the child was intellectually disabled. If symptoms occurred with a decline after a period of normality then the child had childhood schizophrenia. Bradley recognised that onset of illness played this role. Schizophrenic children sometimes declined to intellectual levels comparable with mental retardation (Bradley 1943, p.54-55) whilst mental retardation often had some symptoms similar to childhood schizophrenia (Bradley 1943, p.94), therefore onset of illness is key to differentiating mental retardation and childhood schizophrenia (Bradley 1943, p.81). Since Kanner described autism as present from birth, the age of onset of autistic children should have diagnosed them as mentally retarded and excluded them from childhood schizophrenia. However, low intelligence was required for mental retardation whereas Kanner believed autistic children had normal or above average intelligence, excluding them also from mental retardation (I later argue some autistic children were mentally retarded but not all were). Therefore, pre-1943 psychiatry lacked categories for children with symptoms present from birth yet had normal or superior intelligence. Some single symptom disorders like speech disorder were compatible with symptoms present from birth and normal intelligence. However, single symptom disorders would be wholly inadequate to describe the myriad of difficulties afflicting Kanner's children.

Autism filled this gap, showing a new set of statistical relationships between symptoms. Now classifications existed that could diagnose those individuals not fitting any other category. Without making claims about epistemic status, we can see the formulation of autism is not arbitrary in any obvious sense. There was a gap within the diagnostic system which patients fitted, thus legitimizing creating a new classification.

1.3.4 Kanner and Bleuler

Many scholars interpret Kanner's 1943 autism paper in light of Bleuler's earlier notion of autism. Verheoff writes, “if we want to retrieve the specific meaning of this new disorder [Kanner's autism], it might be better to start with the well-known Swiss

psychiatrist Eugen Bleuler" (2013 p.446; also Eyal et al, 2010, p.213; Feinstein, 2010, p.26; Jacobsen, 2010, p.437; Nadesan, 2005, p.11; Silverman, 2012, p.33). Bleuler's early 1900s pioneering work on schizophrenia outlined the concept of autism as a primary symptom of schizophrenia.

> "The most severe schizophrenics, who have no more contact with the outside world, live in a world of their own. They have encased themselves with their desires and wishes (which they considered fulfilled) or occupy themselves with the trials and tribulations of their persecutory ideas; they have cut themselves off as much as possible from any contact with the external world. This detachment from reality, together with the relative and absolute predominance of

Verheoff claims there is "a fundamental aloofness that relates the two [Kanner's and Bleuler's] autisms" (Verheoff 2013, p.447) and many historians see Bleuler's emphasis on withdrawal as influencing Kanner's concept of autism.

This interpretation has limitations. Kanner explicitly links autism with Bleuler in two papers (1965; 1973a). Kanner says his autism "does not seem to fit in with Bleuler's criteria for autism" (Kanner 1973a, p.95). There are similarities since autistic children "start out in a state which, in a way, resembles the end results of later-life withdrawal [of Bleuler's autism]" (1973a, p.95) but exactly what 'in a way' covers is left unstated beyond both autisms having a "remoteness" (Kanner 1973a, p.95). Historians linking Bleuler with Kanner need a clearer picture of which aspects of Bleuler's autism Kanner had in mind. Parnas writes that Bleuler

> "described a rich variety of clinical manifestations under the heading of autism: poor ability to enter into contact with others, withdrawal and/or inaccessibility, negativistic tendencies, indifference, rigid attitudes and behaviours, private hierarchy of values and goals, inappropriate expression and behaviour, idiosyncratic logic and thinking, and a propensity to delusion formation" (Parnas 2011, p.1122).

Kanner never stated which of these symptoms remoteness covers, meaning

establishing exactly which aspects Kanner had in mind will require a careful analysis. The case studies I describe below suggest that by the 1950s, Kanner linked autism to few of those clinical manifestations. Kanner said the "designation "early infantile autism" was suggested by... self-centered and, at least in the beginning, often impenetrable aloneness" (Kanner & Lesser 1958, p.711). Of the symptoms Parnas assigns to Bleuler's autism this plausibly covers poor ability to enter into contact with others, withdrawal and/or inaccessibility and, more speculatively, private hierarchy of values and goals. Kanner chose the name autism to convey only parts of Bleuler's autism.

Kanner may not have outlined the exact relationship between his autism and Bleuler's autism because Kanner was probably unsure himself and ultimately did not consider the question particularly important. Kanner's methodology involves a cautious approach to theoretical questions. His preface to Despert's collected papers on childhood schizophrenia highlights this.

> "What impresses one more than anything else is the persistent emphasis on factual data, on the absence of dogmatism, on a truly scientific study of perceived phenomena and their correlations... She does not claim to know all the answers" (Kanner in Despert 1968, p.v).

He described many theories employed in child psychiatry as

> "huge hypothetical skyscrapers... evaluated one-sidedly and not too critically... [Kanner recommends] a pluralistic inclusion and evaluation of all the factors involved will keep his [the child psychiatrist's] vision unrestrained by theoretical blinders." (Kanner 1935, p.191).

Kanner probably associated autism with Bleuler's theories but this does not entail that he placed any substantial importance on that association or that he held it with much confidence.

I suggest Bleuler's autism is less central to Kanner's account than some historians have portrayed. Kanner wrote too little on the topic for historians to reach firm conclusions. What little he did write suggests he only associated autism with

parts of Bleuler's autism and he held that association with great uncertainty. Some historians only link Kanner with Bleuler in passing but Verheoff relies heavily on interpreting Kanner in light of Bleuler when arguing Kanner's autism is radically different to modern autism. Historians are likely correct to suggest Bleuler's notion of autism influenced Kanner's notions but this should not be overemphasised.

Treating Kanner's 1943 autism as only tentatively and partially related to Bleuler's autism makes it easier to accept that Kanner would describe notions of autism even less similar to Bleuler's autism from the 1950s onwards.

1.4 1943 to 1978

1.4.1 Kanner's changing notion of autism

Kanner's autism has undergone radical changes in symptomatology but I argue these changes were well underway by the early 1950s, much earlier than the various dates given by many other historians of autism. Also, historians typically see the radical changes to symptomatology occurring after autism was a major diagnostic category. However, "early infantile autism did not enter the public arena until about 1950" (Kanner 1973b, p.207) and little was published on autism until 1949 (Eisenberg 1957, p.72). The major changes were occurring whilst autism was still in early stages of scientific development and occurred as it started being widely employed. This matters because the alterations to Kanner's theory occurred while autism was still under development, making these changes of little epistemic significance with respect to PMI.

Verheoff argues "Kanner's first description of autism as a diagnostic entity, characterized by extreme emotional withdrawal and tenacious insistence on sameness, remained largely unchallenged for approximately the first two decades after its introduction" (2013, p.449). Eyal et al and Jacobsen also mention no changes to symptomatology Kanner assigns to autism. However, the clinical picture described by Kanner in 1943 was not fully born out in the 1950s. By 1956 Kanner noted autistic children diverge around age five, producing clinical pictures with great similarities to modern high and low functioning autism. "On clinical grounds, it is now useful to

differentiate between the [autistic] children who have learned to speak by the age of five and those who have no useful language function by that age" (Kanner & Eisenberg 1956, p.559). Half the children who can speak "have made some sort of scholastic adjustment and participate in a limited way in the social life of the community" (Kanner & Eisenberg 1956, p.559) whereas those without language typically end up in institutions and are "functionally severely retarded" (Kanner & Eisenberg 1956, p.559). The importance of speech developing was noted by other child psychiatrists (Alderton 1966, p.280; Bender 1959a, p.85; Eveloff 1960, p.103; Fish & Shapiro 1965, p.42; Havelkova 1968, p.853; Ornitz & Ritvo 1968, p.84; Rimland 1971, p.163). I will now describe this change, arguing that Kanner from the 1950s onwards primarily, though not exclusively, described socially impaired autistic people who do not actively shun all human contact and non-verbal autistic people with limited intellect. Both of these largely make up modern autism, highlighting the large overlaps between 1950s to 1970s autism and modern autism. Doing so shows that whilst Kanner's 1943 autism is discontinuous with modern autism his account from the early 1950s onwards is much less so.

1.4.2 Autism as social impairment

Lorna Wing's notions of Asperger's syndrome was popularised in the mid-1980s and soon most autistic people were considered high functioning, their condition primarily conceptualised as a disorder of social understanding. The majority of autistic symptoms fit within Wing's triad of impairment, covering social communication, social interaction and social imagination (Wing 2005, p.198). From around the 1990s, the stereotypical person with autism is an odd individual with low social skills, obsessively interested in socially abnormal topics like trains. Historians deny the importance of social deficits in descriptions prior to the 1980s. Verheoff writes,

> "what is considered essential in autism has gone through major changes, from profound affective withdrawal and aloofness [in the 1940s and 1950s]... to deficits in social cognition and intuition [in the 1980s]" (2013, p.454).

Evans writes,

> "studies [in the 1980s] increasingly regarded autism as a problem of 'social' interaction, rather than a problem of emotional relationships with others. The 'autism' employed in these studies was not... Kanner's 'autistic disturbance of affective contact'" (2013, p.21).

Eyal et al write "the aloofness and flat affect noted by Kanner were possible but no longer necessary components [of 1980s autism]" (2010, p.207), and "Autism in the DSM-III-R was no longer the same thing as what Kanner saw and described" (2010, p209). Although they provide much less detail, similar concerns are raised by Silverman (2012, p.130) and Feinstein (2011, p.265). I will now show that social deficiencies were present and very important to Kanner's autism from the early 1950s.

Kanner's clinical picture from the 1950s involved many children who did not actively shun all human contact.

> "The major pathology remains in the area of inability to relate in the ordinary fashion to other human beings. Even the relatively "successful" children exhibited a lack of social perceptiveness, perhaps best characterized as a lack of *savoir faire*" (Kanner & Eisenberg 1956, p.558-559).

The major aspect of autism is here described as an inability to relate ordinarily, something quite different to actively shunning all human contact. Additionally, in sharp contrast to how many historians describe Kanner's account, this inability to relate is best characterised by social imperceptiveness. Unfortunately, it is unclear who exactly Kanner's quote refers to. Is the inability to relate normally best characterised as impaired social perception for all autistic people or just those who are relatively successful? If the former then Kanner's 1950s autism was primarily conceptualised as weak social perceptiveness, if the latter then at a minimum the relatively successful were conceptualised as primarily socially impaired. Eisenberg, who was Kanner's long-time collaborator, takes the former interpretation, writing that

“the primary psychopathologic mechanism in infantile autism might be described as a disturbance in social perception” (Eisenberg 1956, p.23). Kanner and Eisenberg had close ties, co-authoring papers on autism in both 1955 and 1956 and Eisenberg's 1956 paper containing that quote was actually a follow-up study of the children in Kanner's 1943 paper.[3] This increases the likelihood that Kanner considered all autistic people as primarily characterised by impaired social perceptiveness, rather than just the successful ones. Unfortunately, Kanner never made a clearer statement on this, probably because demarcating symptoms as either primary or secondary was too theoretical for his tastes. By 1956 Kanner's 1943 picture of autism as purposeful isolation from human contact had already undergone a substantial shift towards a disorder of social impairment for some, perhaps all, autistic people.

Exactly how comparable Kanner's and Wing's 'social' are is not clear: Kanner only highlights social imperceptiveness through case studies whereas Wing conceptualises a triad of impairment covering social communication, social interaction and social imagination (Wing 2005, p.198). Best characterised as social imperceptiveness seems less pervading than Wing conceptualising most symptoms as stemming from abnormal social understanding but social imperceptiveness is clearly very important to Kanner.

Setting aside debates over the relationship between Kanner's and Wing's 'social', the clinical picture of these successful autistic individuals looks quite similar to modern stereotypes of high functioning autism. “They relate well to books and blackboards but have few, if any, real friends, and have retained some of the earlier obsessive-compulsive qualities” (Kanner & Eisenberg 1955, p.236). They are “isolated, strange persons... and still maintain a somewhat tenuous contact with reality” (Kanner & Lesser 1958, p.727). Academic, few friends, obsessive, strange, this bares obvious parallels with high functioning autism. The case studies in Kanner's 1950s and later publications show children, adolescents and adults trying to engage in the social world, if often making social failures today stereotypically associated with high functioning autism.

3 In 1953 Eisenberg “took two years of Fellowship in Child Psychiatry with Dr. Leo Kanner at the Johns Hopkins Hospital, where he has [since then] remained on the faculty... Within five years he became Psychiatrist-in-Charge of the Children's Psychiatric Service, on Dr. Kanner's retirement” (Lourie 1962, p.757). Also, Eisenberg's views are highly significant in their own right, he became the editor of the American Journal of Orthopsychiatry in 1962, perhaps the major journal in child psychiatry during this era.

> "Attending a football rally of his junior college and called upon to speak, he shocked the assembly by stating that he thought the team was likely to lose – a prediction that was correct but unthinkable in the setting. The ensuing round of booing dismayed this young man who was totally unable to comprehend why the truth should be so unwelcome" (Kanner & Eisenberg 1956 p.559).

A nineteen year old woman

> "took part in routine activities, though she made few, if any, real friendships. She is a serious, rather literal-minded young woman who augurs to do reasonably well in an occupation in which no demand is made on give-and-take relationship with other people" (Kanner & Lesser 1958, p.713).

Another shows literal interpretation and severe social misjudgement.

> "Having been told that 20 minutes were the usual time for breastfeeding, she [a nurse] entered the room at the exact moment and took the babies away without saying a word; there were many complaints from the mothers" (Kanner 1973b, p.194).

Another "obsessively tried to make social contacts [but largely failed]... as a hobby, collects time tables to maintain his interests in trains" (Kanner 1973b, p.198). Eyal et al deny such higher functioning individuals were described prior to the 1980s, giving the example of how autistic adult Temple Grandin

> "approximated the prototype of Kanner's infantile autism enough to become associated with the label, but she grew into something that was previously unthinkable to many people, an independent living, self-reflexive, highly articulate if idiosyncratic autistic adult" (Eyal et al

2010, p.228).[4]

Such higher functioning individuals were clearly not unthinkable to Kanner.

Exactly when Kanner modified his notion of autism is difficult to judge. From 1947 to 1954 Kanner published four papers specifically on autism. Only one of these contained case studies, focusing solely on clinical accounts of desire for sameness (Kanner 1951). This leaves a large window where we lack general case studies. His most concrete statement can be found in a lengthy case study by Darr & Warden in 1951. They described an adult who they believed was autistic. She had an active social life, she was not withdrawn, but she "never developed any intuitive social sense and made repeated *faux pas*" (Darr & Warden 1951 p.564, emphasis original), the paper mentioning various social failures. Kanner, in the comments section of their paper, agrees she is autistic. Kanner would later reference this autistic adult as an example of social imperceptiveness (Kanner & Eisenberg 1956, p.559). By 1951 Kanner is associating autism with someone who is not withdrawn and is described as lacking social intuition but he believes such cases are in the minority. He writes that of the 80 autistic children he has encountered, the

> "majority of autistic children have settled in their withdrawal to the extent that no emergence seemed possible. Some few who were followed therapeutically were able to make sufficient compromise with reality to attend school and relate themselves well to blackboard and books, to convert functionally useless obsessiveness to socially more acceptable routine activities, and to have a degree of more or less mechanized contact with people" (Kanner in Darr & Warden 1951, p.570).

Here the majority of autistic children are withdrawn, presumably in his 1943 sense, but a few have taken a different course. By the mid 1950's Kanner is describing half of all children who develop language as having taken that alternative course, these children being able to "participate in a limited way in the social life of the community" (Kanner & Eisenberg 1956, p.559). Kanner sees all autistic children as

4 Temple Grandin is an individual with autism who has written books about her experiences.

withdrawn in 1943, by 1951 a majority are withdrawn but a few are not, whereas by 1956 a third of all autistic people are engaging socially and are not withdrawn in his 1943 sense.

From the 1950s onwards Kanner wrote little about those children who develop language but do not engage socially.

> "Most of them, even at low ebb to which they have receded, still show remnants which distinguish them from the demented or pseudo-demented level of the mute autistic children... They have given up much of their earlier ritualism, and the typical features of autism shown in their childhood are much less in evidence" (Kanner & Eisenberg

This suggests they are not as severely impaired as non-verbal autistic people but unfortunately Kanner offers little more information. Kanner & Eisenberg have far fewer case studies on these children compared with autistic people who engage socially. There is certainly a possibility that these autistic people have a symptom profile rarely or even never described in modern autism (thus suggesting a discontinuity), but with such little written about this manifestation of autism conclusions are difficult to draw.

1.4.3 Autism and low intellect

I now discuss those autistic children who did not develop language.[5] Kanner's 1943 paper claimed "though most of these children [his case studies] were at one time or another looked upon as feebleminded, they are all unquestionably endowed with good cognitive *potentialities*" (Kanner 1943, p.247, emphasis original). Jacobsen takes this as evidence that Kanner saw children almost never now described, what he calls "high functioning isolates" (Jacobsen 2010, p.442) in the sense of Temple Grandin and Donna Williams.[6] This is highly impaired but intelligent people, whereas modern autism sees highly impaired autistic people as having severe intellectual limitations

5 Not developing language means being unable to communicate meaningfully rather than complete absence of speech (Kanner & Eisenberg 1956, p.4).

6 Donna Williams is an individual with autism who has written books about her experiences.

(Jacobsen 2010, p.439).[7] Jacobsen applies this to all children Kanner diagnosed and therefore the non-verbal autistic people discussed here. Jacobsen specifically denies these children were intellectually retarded (Jacobsen 2010, p.444). However, Kanner's 1940s claims about the non-verbal are more moderate than Jacobsen believes and became even more so after the mid-1950s.

Kanner says non-verbal children are typically institutionalised and resemble mentally retarded children but are distinguishable by "residual oases of planned mental activity" (Kanner 1949, p.417) and "the preservation of isolated areas of unusual intellectual functioning" (Kanner & Eisenberg 1956, p.559). His earlier claims of "good intelligence" (Kanner 1943, p.247) for non-verbal children are not repeated. Jacobsen seems to take good cognitive potentialities to mean they had an underlying, largely untestable intelligence, denying they were retarded (Jacobsen 2010, p.444). I argue Kanner's account of non-autistic feebleminded people suggests an alternative interpretation, applicable to the late 1940s onwards. Kanner believed some feebleminded people cannot develop intelligence whereas the pseudo-feebleminded are prevented from developing intelligence by environmental factors (Kanner 1948a, p.374), typically a lack of educational support. Autistic children with their good cognitive potentialities would seemingly fall into this latter category. Pseudo-feeblemindedness is treated by removing "those handicaps which have prevented existing potentialities from coming to full fruition" (Kanner 1948a, p.393). Unfortunately, treatment of autistic people had been, in Kanner's views, remarkably unsuccessful (Kanner in Darr & Warden 1951, p.370). This suggests they do not develop normal intelligence. Although autistic children are born with good cognitive *potentialities*, their withdrawal renders education near impossible. Consequently, rather than developing normally across all areas of intellect, their limited contact with the world results in *isolated* areas of intellect. Eisenberg's position supports my interpretation. He writes that autistic children "must undergo irreversible intellectual deterioration when opportunities for growth are [severely] barred by the exclusion of normal experience" (1956, p.21). I suggest Kanner's claims about intellect are quite different to Jacobsen's picture of high functioning isolates who cannot communicate their underlying intelligence.

Although Kanner does not believe these children have normal intellect, his

7 Note that Jacobsen and Eyal et al characterise Temple Grandin in quite different ways.

claims about good cognitive potentialities is still quite different to the presumed unalterable brain damage of modern low functioning autism. However, Kanner's views change from the mid-1950s onwards. Whereas in 1943 he claimed that all autistic children have normal intelligence, in the 1950s Kanner admits that his assessment that non-verbal autistic children have good cognitive potential is made "with less confidence" (Kanner & Eisenberg 1956, p.557) than his judgement with regard to verbal children and that only "a number... are still distinguishable from their fellow [feebleminded] patients" (Kanner & Eisenberg 1956, p.559). None of Kanner's papers on autism after the 1950s actually claim non-verbal children have good cognitive potentialities, merely maintaining autism and mental retardation are distinct (Kanner 1965, p.413; Kanner, 1968, p.140). Kanner admitted to substantial overlap of symptoms between mental deficiency and autism (Kanner 1969, p.6) and admitted there was often uncertainty over which classification applied, writing that "[m]oderate or severe mental retardation poses more of a problem in differential diagnosis" (Kanner & Lesser 1958, p.728; also Kanner 1969, p.6). His 1971 follow-up study of his 1943 cohort has five adults who are non-verbal. One is currently functioning fairly well, doing chores. The others he described in 1943 as having good intelligence whereas now he notes that those capable of performing IQ tests score extremely low (Kanner 1971b, p.143) and he omits any claims about isolated intellectual functioning. He never provided a clear statement about the intellectual level of non-verbal autistic people after 1956 but certainly became much less optimistic about intelligence in the decades following 1943.

Kanner's picture of mental retardation was quite compatible with modern notions of low functioning autism.

> "Because some autistic-like symptoms are found in innately retarded and in brain-injured children, the differential diagnosis, which admittedly does on occasions present difficulties, must depend on clear evidence of the essential features of extreme aloneness and the desire for the maintenance of sameness" (Kanner 1958, p.110).

Kanner is using the primary symptoms of autism, rather than IQ, to demarcate autism from mental retardation. In Kanner's views they are not autistic because they lack the

primary symptoms but they can show other symptoms of autism such as obsessiveness, echolalia, skilled manipulation of objects etc. This is similar to modern low functioning autism where low intellect coexists with other autistic symptoms without requiring aloneness or maintenance of the same to be present.

Other child psychiatrists emphasised the difficulty of distinguishing between autism without language from mental deficiency (Eveloff 1960, p.92; O'Gorman 1954, p.943). Some doubted if non-verbal autistic children were actually intelligent (Bender 1959a, p.82; Cappon 1953 p.47; Hingtgen & Bryson 1972, p.16; Ornitz & Ritvo 1968, p.84). The notion that autism can be present with low intelligence is present in 1950s to 1970s child psychiatry.

1.4.4 Kanner on other classifications

I have previously described how Kanner required aloneness and desire for sameness for a diagnosis of autism. Some historians correctly claim these diagnostic criteria are much narrower than modern autism (Eyal et al 2010, p.207; Feinstein 2010, p.265; Silverman 2012, p.130; Verheoff 2013, p.454), Kanner requiring both aloneness and desire for sameness to be apparent before the age of two (1949, p.419). I have already argued that most autistic people Kanner diagnosed using those narrow diagnostic criteria would grow into adolescents and adults who look remarkably like modern high and low functioning autism. Nonetheless, given how few children would have been eligible for Kanner's autism compared with the more relaxed diagnostic criteria of the 1987 DSM-III-R a substantial discontinuity would still have taken place unless there was another related classification which was not as restrictive as Kanner's autism and could also produce similar clinical pictures to autism. This was the case with childhood schizophrenia and some of its subclassifications, which described clinical pictures resembling modern autism whilst lacking Kanner's strict diagnostic criteria.

Even though childhood schizophrenia was diagnosed far more than autism (Eyal et al 2010, p.128; Silverman 2012, p.39-40), historians typically focus more on autism. Verhoeff (2013) and Jacobson (2010) make almost no mention of childhood schizophrenia, making their discontinuity claims highly limited in scope. Kanner's views on the relationship between autism and other classifications are complex and

sometimes misrepresented. Kanner often strongly objected to other child psychiatrists modifying the diagnostic criteria of autism (1965, p.413), preferring to keep the diagnostic criteria very narrow. Grinker writes that "Kanner would probably object to how inclusive a category autism has become" (2007, p.63). Grinker's conjecture may well be true – it is certainly consistent with Kanner's general stance – but this quote conceals that Kanner also believed in childhood schizophrenia which greatly overlaps with modern autism. Also, Kanner held no strong views on their relationship, writing that "it matters little whether autism be regarded as a form of [childhood] schizophrenia or looked upon as a disease *sui generis* … The issue is more one of semantics" (Kanner 1968, p.25 emphasis original). Kanner certainly preferred precise classifications to wide ranging classifications (1965, p.420) but Kanner would still diagnose the wide ranging classification of childhood schizophrenia even as late as the 1970s (Kanner 1973c, p.253-263). I will now discuss childhood schizophrenia, children with circumscribed interests and symbiotic psychosis, these latter two (plus autism) typically considered the three subtypes of childhood schizophrenia (Kanner 1969, p.3).

- *1.4.4.1 Childhood Schizophrenia*

Childhood schizophrenia was conceptualised in many different ways, such as severe and less severe, present from birth and delayed onset, gradual onset and sudden onset (Kanner 1957, p.732-733).[8] Most child psychiatrists thought childhood schizophrenia could manifest in all these ways. Also, some childhood psychiatrists associated childhood schizophrenia with auditory hallucinations (Bettelheim 1967, p.116; Lourie, Pacella & Piotrowski 1943, p.544; O'Gorman 1954, p.935), visual hallucinations or delusion but most did not. Rather, many child psychiatrists thought they had hallucinatory thinking in a sense related to Bleuler's notion of an abnormal contact with reality (Evans 2013, p.11). Whilst childhood schizophrenia covered a broader clinical picture than modern autism, many children diagnosed with childhood schizophrenia did have considerable resemblance to modern autistic people.

Whilst childhood schizophrenia covered a very broad clinical picture, common

8 Note that decline from normality was considered necessary until the mid 1940s but after that it was believed some schizophrenic children could exhibit symptoms from birth.

among clinical pictures of childhood schizophrenia were individuals who exhibited less severe manifestations of symptoms present in more severe form in Kanner's autism.

> "The various forms of childhood schizophrenia share with early infantile autism the loss of affective contact and autistic thinking. However, in other forms of childhood schizophrenia there is usually a later onset and a period of normal development preceding it. Communication and affective perceptions are not usually as deeply disturbed as in autistic children. In the broader schizophrenic group there may also be a wider variety of symptoms" (Kanner & Lesser 1958, p.728).

This shows schizophrenic children can have many symptoms of Kanner's autism but need not exhibit aloneness and desire for sameness by age two. Schizophrenic children have

> "withdrawal from affective contact with people, a progressive loss of interest in play, an increasing tendency to brood. Speech becomes more and more autistic and less and less communicative. Thinking loses its normal plasticity. Range of contents is narrowed to matters of immediate personal concern. At the same time there is excessive preoccupation with abstract concepts which are not ordinarily a part of infantile interests. It takes the form of obsessively repetitious ruminations about calendar dates, positions of the planets, arrangements of numbers, or various measurements. Some of the children are fascinated by names, especially of animals and plants. One of our patients "specialised" in names of snakes and deers; the distinguishing characteristics were of no importance to him, only the appellations mattered. Eventually, the picture becomes complicated by aggressiveness and destructiveness, which are impulsive rather than deliberate. Despert emphasised compulsive possessiveness "characterised by an intense drive for storing objects of no concrete

value or significance" [quoting Despert]". (Kanner 1948b, p.713).

Lauretta Bender, the main proponent of childhood schizophrenia, described how many schizophrenic children have less impaired symptoms, such as, preferring structured environments, intelligent, enjoying academic study, disliking new situations, rigid in approach, little awareness of self, fearing social situations, restricted range of actions and being obsessive (Bender 1959b, p.506).[9] Social impairment was also emphasised, Mahler writing that

> "no other single feature of child schizophrenia represents the essence of its psychopathology as pertinently as the gravely disturbed preverbal and verbal intercommunication between the child and his environment… [childhood] *schizophrenia represents, more than anything else, a grave disturbance of social intercommunication"*

All these symptoms look more like modern autism than any other DSM classification.

Childhood schizophrenia also overlapped with modern low functioning autism. "When schizophrenia occurs in the first few years or before language is well established, there is usually more or less retardation, inhibition or blocking, often with complete mutism" (Bender 1947, p.47). Childhood schizophrenia can involve mental retardation but unlike Kanner's autism, aloneness or desire for sameness is not required, meaning they have similarities to modern low functioning autism where mental retardation co-existed with other symptoms of autism.

Childhood schizophrenia had no specific diagnostic criteria, rather, diagnosis was typically based upon multiple pervasive abnormalities, Bender writing that "typical symptomatology must pervade in every area of functioning" (1953, p.673). This meant childhood schizophrenia covered a wide variety of children, including children with symptoms of Kanner's autism but who did not meet Kanner's strict diagnostic criteria for autism of aloneness and desire for sameness present at two years of age. Bender writes that "one can often make an unusually good contact with schizophrenic children. There is generally a searching, penetrating, even aggressive clinging dependence" (Bender 1947, p.53). These are not children who actively shun

9 These are symptoms of pseudo-neurotics and pseudo-psychopathics, who are discussed in section 1.4.5.

all human contact and outside interference. Also, this quote shows that many schizophrenic children exhibited symptoms quite different to Bleuler's sense of withdrawal.

- 1.4.4.2 Acute childhood schizophrenia

There were two types of childhood schizophrenia, insidious and acute. Above I described what Kanner called insidious childhood schizophrenia; here I describe what Kanner calls acute childhood schizophrenia.

> "[W]ithin a short period, there is a marked drop in scholastic efficiency. The ability to concentrate decreases. There may be complaints of headache or other physical discomfort. This prodromal stage is followed by a turbulent psychotic condition with acute anxiety, sleep disorder, motor restlessness, disturbances of speech, occasional hallucinations, general perplexity, bizarre bodily sensations, and loss of contact with people in the environment. The episode, which is often precipitated by physical illness, operation, or a major emotional upset, lasts a few days or weeks and tapers off gradually though occasionally it acts as a "catastrophic reaction" from which the patient never recovers. In most instances, there are remissions during which functioning on a simpler level may be re-established. Sometimes there is a return to the pre-psychotic mode of living but very often the remission represents merely a "recovery with defect" and is followed by other acute episodes, each of which leaves the patients with a further

Additionally, some acute schizophrenic children did have hallucinations, thought only in older children (Bender 1947, p.55; Clardy 1951, p.12; Despert 1938, p.369; Freedman 1953, p.490; Kratter 1959, p.416; Symonds & Herman 1957, p.523). Acute childhood schizophrenia appears to cover what are considered today two distinct clinical pictures. Acute childhood schizophrenia partly covered childhood disintegrative disorder, where a child is normal until around age four and then decline

to a severe state. Though a very different clinical picture to typical notions of high and low functioning autism, they are heavily associated in recent DSM and childhood disintegrative disorder is merged into autism DSM-5 (see section 2.5.2. for details). Acute childhood schizophrenia also partly cover early onset schizophrenia, rare cases where schizophrenia starts in late childhood and early adolescence. Both these clinical pictures of acute childhood schizophrenia were distinct from autism because autism was deemed to be present from birth. Both were distinct from insidious childhood schizophrenia because the childhood disintegrative pattern was much more severe than insidious childhood schizophrenia whilst insidious schizophrenic children did not hallucinate whereas the early adult schizophrenia pattern did hallucinate. In essence, insidious childhood schizophrenia looks much more like modern low and high functioning autism than it looks like either symptom pattern of acute childhood schizophrenia. Let's now consider other subtypes of childhood schizophrenia to see how they related to Kanner's autism.

- *1.4.4.3 Circumscribed interests*

Franklin Robinson and Louis Vitale worked at the Children's Service Centre in Wyoming Valley, Pennsylvania. They "presented three cases of children with circumscribed interest patterns and 'a limited establishment of interpersonal relationships'" (Robinson & Vitale 1954, p.755). Verhoeff states that such children would today fit Asperger's syndrome or high functioning autism (2013, p.448). "These children were all introvert, had average or above average intelligence, good language skills and circumscribed interests in rather unusual topics" (Verheoff 2014, p.448), such as chemistry, finances, calendars, maps, astronomy. However, as Verhoeff shows, Robinson & Vitale describe demarcation criteria between autism and these children and Kanner agreed with those demarcation criteria (Kanner in Robinson & Vitale 1954, p.765). Verheoff takes this as evidence that Kanner was describing something qualitatively different to modern high functioning autism (Verhoeff 2013, p.448). I argue Kanner thought the difference was between different subtypes of childhood schizophrenia, sharing many symptoms but having different diagnostic criteria.

Robinson's & Vitale's demarcation criteria between children with

circumscribed interests and autism do not support Verheoff's claims of qualitative differences between Kanner's autism and modern autism. Children with circumscribed interests "present a lesser degree of "withdrawal from contact with people" and a lesser measure of the "obsessive desire for the preservation of sameness" than is encountered in children who have been autistic from early infancy" (Robinson & Vitale 1954, p.760). Whilst these symptoms are quantitatively weaker than Kanner's autism they are not qualitatively different. Unmentioned by Verheoff is that Kanner considered both autism and children with circumscribed interests subtypes of childhood schizophrenia, much like how Asperger's syndrome could be considered a subtype found on the autistic spectrum. Also, Robinson & Vitale claimed children with circumscribed interests do not "utilize the favoured neologistic or meaning-restricted language forms which have special meaning for the autistic children" (1954, p.760). However, such idiosyncratic language is extremely common in Asperger's syndrome (Mayes, Calhoun & Crites 2001, p.268). Either Robinson & Vitale were mistaken to claim these children, unlike autistic children, lacked meaning restricted language or Verheoff cannot claim these children resembled Asperger's syndrome. Finally, "these children have not presented the early infantile [autism] incapacity for emotional responsiveness" (Robinson & Vitale 1954, p.760). Robinson & Vitale say they generally appear emotionless but they may briefly display emotion when discussing their favourite interests.

> "They, however, do reveal flashes of good emotional reactive capacity, especially in the first few interviews... Such fleeting reactions indicate that there is a good quality of emotional responsiveness beneath the fixed and usually serious expression he customarily retains" (Robinson & Vitale 1954, p.761).

Measured by symptoms expressed the difference is an absence of emotional responsiveness compared to fleeting flashes of emotional responses. If this counts as a qualitative difference it is one which manifests extremely rarely.

All this is fully compatible with children with circumscribed interest exhibiting similar, though less severe, symptoms to Kanner's autism. Measured by older autistic individuals, the case studies in Kanner & Eisenberg (1955) and Kanner & Eisenberg

(1956) look remarkably similar.[10] The major difference is autistic children need show aloneness and desire for sameness by age two, children with circumscribed interests need not show these symptoms and were generally diagnosed later, between eight to eleven years (Robinson & Vitale 1954, p.760). Like childhood schizophrenia, children with circumscribed interests covered many symptoms of Kanner's autism but lacked the stringent diagnostic criteria.

-1.4.4.4 Symbiotic psychosis

Manhattan based Hungarian psychoanalyst Margaret Mahler conceptualised symbiotic psychosis in the late 1940s. Mahler thought these children did not pass beyond mother infancy dual unity, unable to conceptualise themselves separately from their mother (Mahler 1952, p.289). Mahler does not provide a clear list of symptoms or differential diagnostic criteria beyond the psychoanalytical cause.

Those diagnosed with symbiotic psychosis generally have late onset, at around three or four years (Mahler 1952, p.292). During this time "symbiotic psychosis candidates are characterized by an abnormally low tolerance for frustration and later by a more or less evident lack of emotional separation or differentiation from the mother" (Mahler 1952, p.297). Autism and symbiotic-psychosis can

> "in many cases be clearly differentiated in the beginning. Later the pictures tend to overlap... When we meet cases of child psychosis at a later stage, it seems that pure cases of autistic child psychosis as well as pure cases of symbiotic-parasitic psychosis are rather rare, whereas mixed cases are frequent" (Mahler 1952, p.301).

Kanner describes Mahler's subdivisions as "excellent" (1953, p.527). Symbiotic psychosis included the clinical picture of Kanner's autism but without requiring Kanner's strict diagnostic criteria of aloneness and desire for sameness apparent from two years.

Childhood schizophrenia and symbiotic psychosis covered a substantially

10 Additionally, unmentioned by Verheoff, Robinson later changed his mind, seeing autism and children with circumscribed interests as on a continuum (1961, p.548).

wider set of clinical descriptions than modern autism. Within that set there existed clinical descriptions which heavily overlap with modern autism. Also, children with circumscribed interests are very similar to modern high functioning autism. Historians correctly claim that Kanner's diagnostic criteria for autism are much narrower than modern autism. This, however, does not prevent the much more widely diagnosed childhood schizophrenia and its less diagnosed subtypes – all of which (except autism) lacked those strict diagnostic criteria – covering similar clinical material to modern autism.

1.4.5 Autism as a spectrum

Wing's landmark 1979 epidemiological study of autism maintained there was a "continuum of severity" (Wing & Gould 1979, p.26). Although not employed in the 1980 DSM-III, such ideas made their impact on the 1987 DSM-III-R. Autism in the DSM-III-R is placed within an umbrella category of pervasive developmental disorders. This has two subtypes, autism and pervasive developmental disorders not otherwise specified (PDD-NOS), both covering almost identical symptoms but autism being more severe and PDD-NOS being less severe (APA 1987, p.34). The less severe notion of autism was generally referred to as high functioning autism or Asperger's syndrome. Jacobson (2010, p.438), Eyal et al (2010, p.130) and Verheoff (2013, p.452) emphasise that Kanner's autism was much more specific than the "nebulousness" (Jacobsen 2010, p.438) of the autistic spectrum, and claim that Kanner only described highly impaired children. Since the notion of an autistic spectrum is typically undefined and can have many meanings (Verheoff 2014, p.75) I shall only draw comparisons with its most central claim, showing autism and childhood schizophrenia were described as conditions that could manifest with varying levels of severity.

Severity played a central role in differentiating different types of childhood schizophrenia. Bender believed childhood schizophrenia went through stages, the first being pseudo-defective which is present from birth, the second being pseudo-neurotic which is present from mid-childhood, and the final stage being pseudo-psychopathic which has onset in late childhood (1959b, p.492). Onset could occur and could halt at

any stage, a child might start pseudo-defective and halt there, might start pseudo-neurotic and develop into pseudo-psychopathic, or might go through all three stages etc. Pseudo-defective children were severely impaired whereas pseudo-neurotics and pseudopsychopaths were both less impaired than pseudo-defectives, with those with onset prior to five worse off (Bender 1947, p.54; Clardy 1951, p.83-84; Richards, 1951, p.308) as are those who do not develop speech (Fish & Shaprio 1965, p.42). Autism and symbiotic-psychosis were often included in this relationship between onset and changing levels of severity. Some took autism as being early childhood schizophrenia, the autistic child developing into childhood schizophrenia (Alderton 1966, p.279; Despert; 1971, p.367; Fish & Shaprio, 1965, p.42; Havelkova, 1968, p.851; Kratter, 1959, p.416) whilst Ornitz & Rivto believe autism and childhood schizophrenia tend to merge at age 5 or 6 (1968, p.84). Additionally, some considered symbiotic psychosis as late onset autism (Eaton & Menolascino 1966, p.526) or believed autistic children could develop into symbiotic psychosis (Alderton 1966, p.281). Some considered symbiotic psychosis as equivalent to pseudo-neurotics or a less severe version of Kanner's autism and Bender's pseudo-defectives (Fish & Shapiro, 1965, p.42) or could occur during a transition phase from Kanner's autism to pseudo-neurotics (Havelkova 1968, p.852). All this bares relationship to Kanner's observation that autistic children change at age five. To exhibit identifiable symptoms before three years meant most autistic children were initially quite impaired, then either became more socially able (often interpreted as turning into pseudo-neurotics or symbiotic psychosis) or started to resemble mental retardation (often interpreted as remaining as pseudo-defectives). Less impaired children would have shown symptoms too late to qualify as autistic, typically being diagnosed as pseudo-neurotics or symbiotic-psychosis from three years onwards. Here we effectively see a low functioning child turn into a high functioning child, something which is part of modern autism where “changes occur over the years and a child who was appropriately diagnosed with Kanner’s autism can grow into an adolescent who fits Asperger’s descriptions” (Wing, Gould & Gillberg 2011, p.771). Concepts of differing levels of severity played an important role in the thinking of child psychiatrists.

A large number of child psychiatrists explicitly favoured some form of spectrum, covering autism and other classifications. Numerous child psychiatrists conceptualised various disorders as lying on a “continuum” (Goldfarb 1961, p.29;

Robinson, 1961, p.548; Smolen, 1965, p.444; Ward, 1970, p.353), others employed the word “spectrum” (Cappon 1953, p.45; Szurek, 1956, p.522), others focused on a “graduated series” (O'Gorman 1954, p.939) or “gradients” (Esman 1960, p.395). Generally, the spectrum covered at least autism and childhood schizophrenia with Kanner's autism generally considered close to the most severe end.

Kanner may have contemplated explicit notions of a spectrum of severity. Noting the diversity of their outcome as adults in the follow-up study of his 1943 paper, he ponders that “any illness may appear in different degrees of severity, all the way from so-called *forme fruste* [translation: atypical or incomplete] to the most fulminate manifestations. Does this possibly apply also to early infantile autism?” (Kanner 1971b, p.145). Much earlier, he had noted “differences in the intensity of autistic aloneness and fragmentation” (Kanner & Eisenberg 1955, p.232). Also, describing the parents of autistic children, Kanner wonders “do not the personalities of the parents indicate that there are milder degrees of detachment and obsessiveness which enable a person to function and even gain a certain type of success in a nonpsychotic existence?” (1949, p.426). The parents' “aloofness has not reached the gross preportions [proportions] of a psychotic illness. One is tempted to think of them as successfully autistic adults” (Kanner 1954, 334-384). These quotes bare obvious relationship to the modern notion that autism varies substantially in severity.

Child psychiatrists conceptualised both autism and childhood schizophrenia as varying in severity, many thought that severe autistic children might turn into less severe schizophrenic children, and some explicitly conceptualised a spectrum. Varying levels of severity played an important role in differentiating children and continued doing so today with high and low functioning autism.

1.5 Pessimistic Meta Induction

Does autism avoid the pessimistic meta induction? Broadly, yes. Let’s consider what has been established, firstly considering prior to the late 1940s and then considering after the late 1940s.

Prior to the late 1940s the diagnostic scheme has substantial dissimilarities with modern autism. This does not entail PMI given the maturity clause, that we only

judge for PMI once a theory has had time to develop. Maturity should be measured by level of evidence gathering and level of theoretical development. After the theory has been formulated we should allow further empirical evidence to be gathered, for that empirical evidence to be reflected upon and for the theory to be modified accordingly. This in principle gives a scientific community an opportunity to gather evidence about and potentially modify key tenets of a theory before they get locked in. At minimum, only once a significant number of scientists feel a significant level of commitment to a theory could it be considered mature. In terms of psychiatric classifications, I suggest that maturity will typically come through clinical use. A psychiatrist formulates a diagnosis (be it from observing patients or from a theory) and starts to diagnose it. By spending more time around those he or she initially diagnoses and diagnosing others the psychiatrist has only encountered after formulating the psychiatric classification, the psychiatrists should soon have more information about individuals they have diagnosed. This will become especially true if other psychiatrists start employing it and it starts being written about in scientific journals. All this should bring in much more empirical information than was present when the diagnosis was initially formulated. The diagnosis may then undergo changes in response to new evidence. I suggest a psychiatric diagnosis in this initial phase should be considered not mature and therefore modifications made in this initial stage should not be counted when measuring historical continuity.

Exactly when this stage has passed is difficult to judge and plausibly involves a level of arbitrary selection. Rather than specifically argue for one particular date as passing this threshold of maturity I shall instead suggest multiple possible dates and suggest that whichever one is chosen means the pessimistic meta-induction fails to be particularly strong. We need set two different dates. First, we must decide when the period of maturity starts and thus we can start to measure for continuity and discontinuity. Second, we then compare this with the period when autism actually starts being continuous. The longer the gap between these two times, the stronger the pessimistic meta-induction becomes.

Child psychiatry started to be distinct from adult psychiatry around 1925 (Bradley 1941, p.19; Kanner 1959, p.582). Relatively soon after this notions which modern autism can be traced back to are formulated. Potter introduced childhood schizophrenia in 1933 and Kanner introduced autism in 1943. Though potential

candidates for maturity, neither date allows much time for evidence gathering or theoretical developments. A fairer earliest date would be somewhere in the late 1940s because “early infantile autism did not enter the public arena until about 1950” (Kanner, 1973b, pp.207) and little was published on autism until 1949 (Eisenberg, 1957, pp.72). Additionally, childhood schizophrenia was also undergoing significant changes in the late 1930s and early 1940s (Fellowes 2015, p.2). I suggest 1948 as a better date for maturity, the publication of Kanner's second volume of child psychiatry, which included both childhood schizophrenia and autism, both absent from his 1935 edition. We then need establish how long the period was between maturity being obtained and historical autism starting being continuous. Childhood schizophrenia starts becoming recognisable from a modern perspective in Bender (1947) and Kanner (1948b) whilst modern autism starts becoming recognisable in Kanner's commentary in Darr & Warden's (1951) paper. These are all plausible dates but a more conservative choice would be 1955. Kanner & Eisenberg's 1956 paper I referenced extensively was from a 1955 symposium on childhood schizophrenia, so let us pick 1955 as a conservative date for continuity. We could, highly unfairly, start the clock from 1925, taking discontinuity to last thirty years until 1955 then having sixty years of continuity, meaning 33.3% to 66.6%.[11] Slightly better is 1933, though here we ignore the maturity clause, then discontinuity lasts twenty two years and continuity sixty, meaning 26.8% to 73.2%. If counting from Kanner's autism in 1943 then discontinuity is twelve years and continuity sixty, 16.6% to 83.3%. If we pick 1948 as a starting point for maturity then we have seven years of discontinuity from 1948 to 1955 and sixty years of continuity, 10% versus 90%. The level of discontinuity falls significantly if historical autism were to be considered continuous from an earlier date than 1955, say at 1948 or 1951. Ultimately, whichever is taken, levels of discontinuity are not massively high. Whether they are sufficiently high to entail the PMI depends upon a stance towards epistemic risk, as discussed in chapter five, but we should be encouraged.

Psychiatry from the late 1940s often assigned individuals clusters of symptoms with striking similarity to modern autism. This alone, however, does not avoid PMI. We seek to establish belief in the psychiatric classification, not just over symptom clusters. However, the psychiatric classification has taken multiple forms across its

11 Measured by 2015 when this was written.

history. Kanner diagnosed autism only for individuals who exhibited aloneness and desire for sameness by two years, whilst diagnosing childhood schizophrenia for individuals exhibiting similar symptoms to autism but lacked those two symptoms. Bender considered children exhibiting aloneness and desire for sameness by two years as pseudo-defectives but so were children who did not exhibit those symptoms. Also, Kanner employed specific subtypes of autism, children with circumscribe interests and symbiotic psychosis, whereas Bender just considered all these as schizophrenic children rather than additionally considering them those subtype diagnosis. Kanner's and Bender's diagnostic scheme were not identical to one another or to modern autism.

This does not entail PMI, however, to fully understand why requires an understanding of what psychiatric classifications are (as discussed in chapter four) and the exact role PMI plays in the scientific realist debate (as discussed in chapter six). Briefly, scientific realism is based on theoretical virtues. The two main virtues are causation and unification. I will argue autism merits belief due to unification. This is where a set of phenomena is accurately described by as few theories as possible. We can portray Kanner's and Bender's approaches as disagreeing over how many classifications are required to accurately maximise information; employing childhood schizophrenia and three subtypes (Kanner) or just employing childhood schizophrenia (Bender). Bender's approach advantageously employs fewer classifications than Kanner's approach of adding specific subtypes to childhood schizophrenia. Kanner's approach advantageously provides more specific clinical pictures but at the cost of employing more classifications. These are effectively two different ways to unify clusters of symptoms. We need demarcate between historical disagreements over balancing theoretical virtues and scientists being mistaken historically about the presence of theoretical virtues. Balancing theoretical virtues involves a subjective judgement. Scientists rarely, if ever, find the absolute best balance. Theories have greater approximate truth the closer they are to that best balance. Multiple theories can have good balances, each having high degrees of approximate truth. Thus multiple theories having different levels of theoretical virtues do not entail PMI. It is entailed only when earlier theories lacked theoretical virtues. If scientists believed in earlier theories but those theories were lacking theoretical virtues then the scientists were mistaken to assign belief. Though PMI is generally applied to causes, where scientists

believed in false causal claims despite good evidence, this is also applicable to unifications (see Psillos for discussion of non-causal unification based realism (2010, p.957)). Since neither Bender nor Kanner were mistaken, both employing classifications which attained some approximate truth via the theoretical virtue unification, PMI is not applicable.

1.6 Hacking and the Pessimistic Meta Induction

Let's consider Hacking's looping effect. Hacking claims people sometimes act in new ways after a psychiatric classification is introduced. If this were so, this might raise worries symptoms would change over time as people react to new or changing classifications. This may result in historical changes which might entail PMI. Hacking describes

> "a looping or feedback effect involving the introduction of classifications of people. New sorting and theorizing induces changes in self-conception and in behaviour of the people classified. Those changes demand revisions of the classification and theories, the causal connections, and the expectations. Kinds are modified, revised classifications are formed, and the classified change again, loop upon loop" (Hacking 1995, p.370).

Hacking makes the distinction between two different kinds of things. There are interactive kinds, which "are affected by the ways in which being [for example] female or having a disability are conceived, described, ordained by ourselves and the network of milieus in which we live" (Hacking 1999, p.104). These interactive kinds are contrasted with indifferent kinds which do not act differently upon being classified, such as how quarks may be treated differently by scientists once classified but the quark is not aware of this and does not modify itself based upon that classification (Hacking 1999, p.105). Only interactive kinds are subject to the looping effect and may change themselves over time in response to being classified.

Since autism is an interactive kind, new behaviour might have occurred when

autism was first introduced and when it was later modified. Hacking argues this is the case when he applies the effect to autism. Psychoanalysis was once dominant, casting the parent as causally responsible for the illness. Later on, cognitive psychology gave no such causal role to the parents. Hacking suggests the parents modified their behaviour, which in turn caused the children to modify their behaviour. Classification resulted in behavioural modifications. Unfortunately, Hacking provides almost no evidence for this, simply stating that "[m]ost of the behaviours described by Kanner seem not to exist any more" (1999, p.115). This claim does almost nothing to support Hacking's argument. Kanner's autism was a very narrowly defined diagnosis, whereas modern autism is an extremely widely defined diagnosis. A much fairer comparison would be with childhood schizophrenia. The only specific symptoms Hacking mentions is that absent anticipatory posture (where a baby puts out its arms to be picked up) and feeding problems are no longer discussed (1995, p.377). However, these are commonly described, such as Ledford & Gast's (2006) review on feeding problems in autism and Cliffard, Young & Williamson's (2007) analysis of pre-diagnosis individuals which identifies absence of anticipatory posture as more common in children who do subsequently develop autism. Hacking is mistaken on these accounts.

Hacking does describe some changes to autistic behaviours which better fit his looping claims (though not specifically discussing them in the context of looping). He describes how the formulation of an autistic community offers new notions of what autism means (Hacking 2009a, p.506) and how the internet allows new forms of communication which better suit the autistic (Hacking 2009b, p.56). Autistic behaviour changing does not necessarily entail PMI. The world often changes and science needs reflect this. For example, models of climate change need updating as the world heats up. Earlier models were not mistaken, the world actually changed. The earlier model may have been true at an earlier time, the current model might be true today. Similarly, as Hacking points out, some indifferent kinds undergo change, such as microbes mutating (Hacking 1999, p.105). Where interactive or indifferent kind change then scientists should change their descriptions, this process does not imply that scientists were mistaken and therefore does not entail PMI. Thus changing behaviour of autistic individuals over time does not entail PMI. It does entail a second problem, needing the correct explanation of why behaviour changed. Hacking has

offered plausible reasons accounting for minor changes in behaviour. In the absence of unexplained large changes to behaviour Hacking's looping effect is not epistemologically worrying.

1.7 Conclusion

The classification of autism needs have high historical continuation to avoid the pessimistic meta-induction. Autism as originally formulated in 1943 is not historically continuous with modern autism but is from around the early 1950s. By the mid-1950s Kanner identifies a third of all autistic people as those who engage socially in the community. These people look very similar to modern high functioning autism, they are conceptualised as having a disorder of social perceptiveness, something quite different to Bleuler's notion of autism. Kanner identifies a third of all autistic people as non-verbal. He expresses doubts about earlier claims that all these have normal intelligence and most child psychiatrists endorsed these doubts. I have argued many, perhaps most, of these autistic people also had mental retardation in a manner similar to modern low functioning autism.

Childhood schizophrenia could also manifest in ways with great overlap with modern high and low functioning autism, being associated with both social impairment and low intellect. Childhood schizophrenia certainly covered much broader clinical material than modern autism but central concepts of modern autism were present. Finally, child psychiatrists were very aware that both autism and childhood schizophrenia varied greatly in level of impairment, giving this a central role in differentiating various types of childhood schizophrenia. All this shows that three central concepts of modern and current autism – autism as social impairment, autism co-existing with mental retardation, autism as varying in severity – were present, widely employed and important between the 1950s and 1970s.

Autism is quite resilient to the PMI. Early discontinuity does not entail the pessimistic meta induction because of the maturity clause. A theory is allowed to develop before we are concerned by major changes. Kanner's autism and childhood schizophrenia started to effectively become something resembling modern autism between around 1948 to 1955. We discount somewhere between the first fifteen to

twenty years and then accept continuity has been present for over six decades. This alone does not defeat PMI but goes some way to weaken concerns. However, Kanner's and Bender's diagnostic scheme differ from one another and differ from modern autism. For now, we have established similar symptoms and symptoms clusters were describe both historically and today. In later chapters I will suggest Kanner's and Bender's approaches (alongside the approach of modern autism) all merit belief because they are all theoretically virtuous ways to accommodate the symptoms and the probabilistic relationships between symptoms. They are all good ways to accommodate the clinical pictures, therefore all deserve belief. PMI is only threatening if Kanner's, Bender's or the modern approaches lacked theoretical virtues, but they did not.

2.0 Chapter 2 – The challenge of underdetermination

2.1 Introduction

Philosophers of science sometimes question the truth of scientific concepts by pointing to alternative concepts that could be used to describe the same phenomena. This is the argument from underdetermination. Seemingly following this reasoning, philosopher of psychiatry Dominic Murphy raises concerns about psychiatric classification by asking “what evidence do we have that current practice in psychiatry produces good outcomes, outcomes that couldn't be matched by alternative nosologies that start from a completely different set of premises?” (Murphy 2006, p.10). The thought is that if there are alternative ways to describe any given scientific phenomena then why believe one scientific concept rather than an alternative is truthful. This chapter explores changes made to autism between the late 1970s to mid-1980s. This is effectively an alternative, competing classification to the notion of autism I defend throughout all the chapters, raising the prospect of an underdetermination argument. In this chapter I show major flaws in the process of abandoning late 1940s to 1970s autism in favour of the DSM-III autism, showing major problems with this alternative autism and show how it was quickly abandoned with psychiatry largely readopting late 1940s to 1970s autism.

The alternative notion of autism is that developed by Kolvin in the early 1970s, advocated by Rutter and then adopted by 1980 DSM-III. I call this competing alternative 'DSM-III autism'. This competes with what I call 'historically continuous autism', roughly late 1940s autism and childhood schizophrenia alongside autism from the mid-1980s onward. Kolvin's (1971) study is generally taken as proving that childhood schizophrenia is a separate disorder to autism but a close reading shows that Kolvin fails to establish this. Kolvin separates children previously diagnosed as either autistic or childhood schizophrenic into two categories, autism which occurs within the first three years of life or schizophrenia in childhood which is the early

manifestations of adult schizophrenia. The concept of childhood schizophrenia employed since 1933 was now portrayed as early manifestations of adult schizophrenia despite these children not having hallucinations. Kolvin instead could have merged childhood schizophrenia with autism by dropping the age requirements of autism or Kolvin could have retained a third category for childhood schizophrenia. There is no good reason to believe that Kolvin proved that childhood schizophrenia and autism were separate disorders. Kolvin's approach was largely adopted by DSM-III and upon doing so the inadequacies soon became apparent. Many clinical pictures present in autism and childhood schizophrenia from the late 1940s, especially those higher functioning types described in chapter one, no longer found a place in DSM-III. The diagnosis of autism and childhood onset pervasive developmental disorders had very little room for higher functioning individuals whilst the diagnosis of schizoid of childhood and schizotypal personality disorder were inadequate to cover many clinical pictures of childhood schizophrenia (plus subtypes) described from around 1950 onwards. Consequently, for good reason, the diagnostic scheme of DSM-III was often ignored (Eyal et al 2010, p.235). By 1987 DSM-III-R, the changes of DSM-III were partially reversed, reverting back to something with substantial similarities to late 1940s onwards autism.

Highlighting flaws with DSM-III autism and highlighting benefits of the DSM-III-R reversal shows that DSM-III autism is not a strong challenger to historically continuous autism, and this, in turn, provides a counter to any general argument based on underdetermination. Consequently, the key aim of this chapter is to show substantial inadequacies with the alternative notion of autism embraced in DSM-III. However, ultimately showing why underdetermination fails requires a broader understanding of what scientific theories are (as discussed in chapter four) and what theoretical virtues are (as discussed in chapter six). For now, we only seek to show the main alternative to autism had major scientific short comings and was soon replaced with good reason. Additionally, I briefly address how the changes introduced by DSM-III autism, which disrupt historical continuity, does not entail PMI, providing more detail in chapter six.

I start by outlining the underdetermination argument. Then I discuss the changes Kolvin introduced, showing major flaws. I then show Wing effectively reversed many of the changes Kolvin introduced. I then suggest this type of

underdetermination does not undermine belief in modern autism. Finally, I discuss how this relates to PMI.

2.2 Underdetermination

Underdetermination is

> "concerned with the possible existence of alternatives to our best scientific theories that share some or all of their empirical implications – that is, quite different accounts of the entities and/or processes inhabiting some inaccessible domains of nature that nonetheless make the same confirmed predictions about what we should expect to find in the world and recommend the same successful strategies for intervening in it that our own theories do. No matter how impressive a theory's practical achievements in guiding predictions and interventions are, those achievements do not favour the theory over any alternative that would ground those same predictions and interventions and therefore enjoy just the same degree of empirical success" (Stanford 2006, p.8).

No matter how much evidence is obtained, more than one theory can fit the evidence. Therefore, the evidence is insufficient to determine which theory is correct. Something more is needed to decide which theory is true.

Underdetermination suits the social constructivist agenda well. As Giere notes, social constructivists often argue that what singles out a theory as supposedly true is just social convention (Giere 1999 p.237). Social convention could here be interchanged with concerns over medicalisation or negative influence of ethical and political values. For example, Bentall thinks that for a psychiatric classification to be "a valid scientific concept, then at the very least psychiatrists will need to answer the question. Which set of criteria [classifications] are the right ones?" (Bentall 1992a, p.28). However, there are few means for determining which classification is correct (Bentall 2004, p.65). He sees psychiatry as a contingent dynamic process dependent on personal opinion, with modern psychiatric classifications as one of many possible

classifications that will likely be replaced with an alternative classification or a non-classificatory approach in the future (Bentall 1992a, p.50; also Boyle 1990, p.79).

The underdetermination argument needs qualifying since it is too sceptical, being applicable to everything. For example, induction is underdetermined. Any number of instances of the sun rising is compatible with it not rising the next day. Unless one is an inductive sceptic then something more is required for underdetermination to be acceptable. Similarly, we might modify our theory about electrons to claim they morph into protons once a minute at undetectable speeds. This seems unconvincing. Scientists often struggle greatly to establish a single working theory, let alone two, so fair underdeterminations need be constrained to actual working scientific theories (Kitcher 1993a, p.247; Stanford 2006, p.18). Candidates for underdetermination must accommodate a challenging range of empirical evidence, must have compatibility with a wider body of theories and must exhibit theoretical virtues. By only considering genuine scientific theories we narrow the range of possible underdeterminations.

Though limiting the range of possible underdeterminations is wise, this risks the possibility that no competing alternative theories are produced because one theory has unfairly been focused upon. One theory may receive the majority or all the attention, in the form of funding, experiments, publications and media attention. Perhaps possible competitors would have made similar empirical accomplishments had they been given an equal chance. Conveniently for our purposes we do have a competitor which for a time attracted most of the research efforts. Let’s demarcate the notion of autism I defend and its competitor. From around the late 1940s onwards a number of related diagnostic schemes were developed. Broadly, they involved a notion of childhood schizophrenia which was described either in terms of a continuum or had specific subtypes (or both simultaneously). Chapter one drew similarities between these and modern autism. Both covered many clinical pictures with remarkable similarity. The autism I defend is this diagnostic scheme which runs between these eras, what I call historically continuous autism. The competitor is the changes started in the late 1970s and which formulate DSM-III. Thus we have 'historically continuous autism' which I defend and we have 'DSM-III' autism which is the competitor. Let’s investigate what DSM-III autism was and how it briefly replaced historically continuous autism.

2.3 The demise of childhood schizophrenia

As we saw in chapter one, from 1943 autism and childhood schizophrenia co-existed as psychiatric classifications. The boundaries and relationship between both these classifications and other classifications was debated and discussed. Despite this, these approaches were still able to diagnose a wide variety of individuals with a broad range of related symptoms. They were applicable to higher and lower functioning individuals, individuals with and without mental retardation, and to individuals who did or did not exhibit Kanner's stringent diagnostic criteria of aloneness and desire for sameness (by two years old). Some thought all this was covered by a continuum. Some thought that subtypes were present. All this changed within a short period of time, starting in the late 1970s and fully implemented by 1980 DSM-III, when childhood schizophrenia was abandoned in favour of a narrowly-defined autism.

Kanner started the first journal dedicated to research on autism and childhood schizophrenia in 1971 (*The Journal of Autism and Childhood Schizophrenia)*. The journal reflected the disunity of the field, with the editorial board consisting of "psychiatrists, psychologists, and psychoanalysts, and the articles that they included in the journal reflected their often sharply divergent approaches to the disorder" (Silverman 2012, p.39). Indeed, the main proponent of childhood schizophrenia, Lauretta Bender, was on the editorial board despite disagreeing with Kanner over the nature of autism. However, this all changed in 1978 and 1979. Eric Schopler, who replaced Kanner as editor in 1974, wrote a provocative editorial in the 1978 edition. "[S]ince Leo Kanner founded this journal in 1971, many variations of his criteria for infantile autism have been used" (Schopler 1978, p.137) but now there are to be "guidelines for reducing confusion [of diagnostic criteria]. It is hoped these will be used for the research published in this journal" (Schopler 1978, p.138). This brief editorial is followed by the new guidelines in an article by Michael Rutter (1978) that sets out new diagnostic criteria for autism. These criteria were largely based on Kanner's 1943 paper (Blacher & Christiansen 2011, p.182; Mandy & Skuse 2008, p.39; Silverman 2012, p.49) except for an emphasis on mental retardation. Though many historians have focused too heavily on Kanner's 1943 notion, portraying it as

more significant than it was from the 1950s to 1970s, it now became very important. Also, notions of childhood schizophrenia were abandoned. This process was completed a year later when the journal was given its modern name of *Journal of Autism and Developmental Disorders*. The only journal specifically dedicated to autistic and schizophrenic children had officially come firmly down upon one side of a thirty five year long debate about their relationship and effectively told future contributors they must also take this side to get published.

The enormously influential DSM-III (APA 1980) followed these changes when published two years later. The *Diagnostic and Statistical Manual-III* was a guidebook for psychiatrists containing standardised criteria for diagnosis. The DSM-III was important because diagnosis based on DSM-III was required for insurance companies to pay out medical insurance, drug trials were only considered valid if based upon DSM-III and many journals only accepted research papers employing DSM-III classifications (Cooper 2005, p.146). Earlier editions of the DSM-I and DSM-II had entries for childhood schizophrenia (APA 1952, p.28; APA 1968, p.35) whilst autism was included only as a symptom of childhood schizophrenia. In DSM-III autism was now included as a classification (APA 1980, p.87) whilst childhood schizophrenia was removed (APA 1980, p.375). The notion of autism was based on Michael Rutter's late 1970s work (Blacher & Christensen 2011, p.182-183; Silverman 2012, p.49), the author who set the new guidelines for *The Journal of Autism and Childhood Schizophrenia*. Within a few years psychiatrists who employed notions of childhood schizophrenia popular for the previous five decades faced substantial barriers to publication and needed use other diagnostic categories for their patients to receive medical insurance.

This was a remarkable reversal of fortune. Autism went in a few years from being secondary to childhood schizophrenia to nearly completely replacing childhood schizophrenia. Even a proponent of childhood schizophrenia like Sanua admits by 1983 that “while childhood schizophrenia which has been a popular concept for almost five decades has lost its importance, infantile autism remains as a major diagnostic category” (Sanua 1983, p.1648). This sort of exercising of authority is commonly taken as harmful to scientific investigation. Historians and sociologists like Schaffer (1989) and Collins (Collin & Pinch 1998) often claim decisions in science are based less on scientific evidence than on power struggles between competing

scientists. Each side claims their own theory is legitimate science whilst their rival's theory is illegitimate, victory often going to who has greatest social influence and authority. More moderately, there should be an equal distribution of research effects among competing theories providing each theory is making empirical accomplishments (Solomon 2001, p.149). The changes which brought about DSM-III autism appear partly due to key sources of authority favouring a particular interpretation of autism whilst not giving equal support to notions of childhood schizophrenia popular in earlier decades. Below I show many of these changes were insufficient to justify the changes to autism and childhood schizophrenia made by DSM-III.

2.4 Kolvin's separation of autism and childhood schizophrenia

2.4.1 Kolvin's study

Kolvin (1971) is taken as primarily responsible for separating childhood schizophrenia from autism. Historians of autism will usually only cite Kolvin when discussing this separation. For example, Wolff writes "Kolvin's comparative studies distinguishing early childhood autism from childhood schizophrenia, by age of onset, phenomenology, family history and associated symptoms, are now rightly regarded as classics in the history of autism" (Wolff 2004, p.204). Also, "[t]he confusion between autism and schizophrenia occurring in childhood continued to affect the field until, early in the 1970s, Kolvin (1971) and his colleagues carried out a study comparing the two groups of conditions and listed the many differences" (Wing 1997, p.18 ; also Evans 2013, p.18; Feinstein 2010, p.143). They may also cite Rutter's 1972 or 1978 article, but his 1972 paper only cites Kolvin (Rutter 1972, p.320) when discussing separating childhood schizophrenia from autism whilst Rutter's 1978 only cites Kolvin plus a study from Japan and Russia (Rutter 1978, p.151). Papers on childhood schizophrenia from the 1980s almost always cite Kolvin (plus possibly Rutter and colleagues) and employ the notion of childhood schizophrenia Kolvin suggests (I provide evidence of this in section 2.4.2).

Kolvin's work is based upon a study beginning in 1962 in Oxford and

Newcastle of 80 children: 47 autistic and 33 schizophrenic children (Kolvin et al 1971, p.385).[12] The study was published in a series of six articles in the April 1971 edition of the *British Journal of Psychiatry*, and the papers were written by Kolvin and seven co-authors. The study aimed to test if psychotic disorders are dependent upon age of onset, if age of onset determines different symptoms and aetiology (Kolvin 1971, p.383). The autistic children are defined on Kanner's 1943 article (Kolvin 1971, p.381) whilst the schizophrenic children are defined upon criteria from the third edition of Kanner's *Child Psychiatry* (Kolvin 1971, p.382) who gives diagnostic criteria based upon Potter, Bradley and Bender (Kanner 1957, p.711).

Kolvin concludes that children with early onset disorder (prior to 3 years) have symptoms of autism as described by Kanner whilst children with late onset disorder (5 years and after) have symptoms corresponding to adult schizophrenia (Kolvin 1971, p.384). The early onset group had gaze avoidance (Kolvin et al 1971, p.387), abnormal pre-occupations, resistance to change and stereotyped repetitive movements (Kolvin et al 1971, p.390-391) all largely absent in late onset group. Both those in early and late onset groups had problems with social relationships but these were worse in early onset group (Kolvin et al 1971, p.387). Both had delayed speech but these were worse in early onset group (Kolvin et al 1971, p.388). Both had ritualistic and perseverative behaviour (Kolvin et al 1971, p.390) and both had behaviour problems (Kolvin et al 1971, p.391). The late onset group had thought disorder (Kolvin et al 1971, p.389) and the majority, though not all, had hallucinations (Kolvin et al 1971, p.390). Both of these were absent in the early onset group. Crucially though, those in the early onset group never developed hallucinations in later life but could develop thought disorders (Kolvin 1971, p.394). Kolvin effectively says early onset disorders is autistic whilst late onset are an early manifestation of adult schizophrenia, separating autism from childhood schizophrenia.

Kolvin also briefly mentioned children aged three to five years that are normal at birth who undergo a sudden regression to very low functionality (Kolvin 1971, p.381). Rutter says less is known about these disintegrative disorders except that they include a high proportion of abnormal brain disorders and are extremely rare (Rutter 1978, p.6). These rare cases were documented between 1940 and 1970s (De Heller in Hulse 1954, p.472-477). They are dissimilar to those with autism or insidious

12 Note that Kolvin (1971) and Kolvin et al (1971) are just different papers on the same study, in the same issue of the journal.

childhood schizophrenia, being demarcated from both disorders by the brain abnormalities, demarcated from autism by clear regression from normality, and demarcated from insidious childhood schizophrenia by regression to near zero functionality.

I will now criticise Kolvin's study, alongside showing how Rutter also made similar claims to Kolvin. The primary problem is that Kolvin's study is not sufficiently representative of the previous decades of US child psychiatry for it to form a basis from which autism can be separated from childhood schizophrenia.

2.4.2 Relationship between Kolvin and prior decades of childhood schizophrenia research

I will show Kolvin does not adequately show schizophrenic children should be related to schizophrenia rather than autism. Whilst Kolvin adequately shows children with symptoms of autism do not become schizophrenic adults, this does not show all children with symptoms of childhood schizophrenia become schizophrenic. Hallucinations do not occur in early onset disorders, they only ever occur in late onset disorders, however, not all late onset children have hallucinations.[13] These non-hallucinating schizophrenic children do not obviously fit either of the classifications proposed by Kolvin. Since these non-hallucinating schizophrenic children have late onset after a period of normality they do not fit early onset autism well and since they do not hallucinate they do not fit Kolvin's category of schizophrenia in childhood very well, it being basically early adult schizophrenia. Kolvin faced a choice between demarcating autism from schizophrenia in childhood on either age of onset or presence of hallucinations. Alternatively, Kolvin could have created a third category, for children with late onset but who did not hallucinate.

All these three choices were compatible with the child psychiatry of the previous decades. Kolvin claims age of onset separates autism from childhood schizophrenia but psychiatrists of the previous decades were fully aware of the importance of age of onset. "In the classification of childhood schizophrenia, the most

13 Note that Kolvin is aware that hallucinations are more difficult to detect in younger children (Kolvin & Edgell 1972, p.285) but there is no indication that his 1971 paper (or later work) took this problem into account. Evans correctly highlights how problematic this is (2013, p.16).

important factor is the age of onset of the illness" (Bender 1947, p.53; also, Clardy 1951, p.82; Creak 1951, p.548; DeMyer et al 1971, p.178; Despert 1938, p.366; Eaton & Mesnolascino 1966, p.526; Eisenberg 1957, p.72; Eveloff 1960, p.92; Havelkova 1968, p.849; Hingtgen & Bryson 1972, p.9; Kanner 1943, p.248; Kratter 1959, p.416; Mahler 1965, p.651; O'Gorman 1954, p.937; Ornitz 1973, p.24; Richards 1951, p.305; Szurek 1956, p.541; Ward 1970, p.351). We saw how many employed notions similar to a spectrum (Cappon 1953, p.45; Esman 1960, p.395; Goldfarb 1961, p.29; O'Gorman 1954, p.939; Robinson 1961, p.548; Smolen 1965, p.444; Szurek 1956, p.522; Ward 1970, p.353). We also saw how many thought autism could turn into childhood schizophrenia (Alderton 1966, p.279; Despert 1971, p.367; Fish & Shaprio 1965, p.42; Havelkova 1968, p.851; Kratter 1959, p.416). Whether autism and childhood schizophrenia were considered the same thing or separate, child psychiatrists recognised relationships between various symptom patterns and age of onset. As discussed in chapter one, Bender's pseudo-defectives, pseudo-neurotics and pseudo-psychopathics were differentiated on age of onset, similarly Kanner recognised age of onset as important for autism and symbiotic psychosis. Other child psychiatrists recognised age of onset produced different outcomes but considered this a matter of degrees, generally not feeling inclined to firmly differentiate psychiatric classification. Child psychiatrists of the 1950s to 1970s placed much less emphasis on hallucinations than Kolvin does. The vast majority of child psychiatrists of earlier decades believed schizophrenic children either rarely or never hallucinated (Alderton 1966, p.282; Bender 1947, p.50; Bender 1959b, p.501; Clardy 1951, p.82; Colbert & Koegler 1958, p.215; Despert 1940, p.190; Eisenberg 1956, p.21; Ornitz & Ritvo 1968, p.78; Ornitz 1973, p.33; Richards 1951, p.303; Robinson 1961, p.544). Those who recognised rare instances of childhood hallucinations thought, like Kolvin, they only occurred in older children (Bender 1947, p.55; Clardy 1951, p.12; Despert 1938, p.369; Freedman 1953, p.490; Kratter 1959, p.416; Symonds & Herman 1957, p.523). 1940s to 1970s child psychiatrists noticed different outcomes substantially related to age of onset but did not link this with hallucinations, except when the children were old. In contrast, Kolvin thinks age of onset is important primarily because late onset psychosis is linked to hallucinations, unlike early onset psychosis. I will now discuss why Kolvin obtained different results to those of earlier decades.

2.4.3 Unrepresentativeness

I will argue that there is reason to think there were methodological problems with Kolvin's study. I will suggest his sample was unrepresentative for drawing conclusions about US notions of childhood schizophrenia. A 1974 review article of childhood schizophrenia by Miller (1974) also claims Kolvin's study is unrepresentative, a claim I shall expand upon.

Kolvin's 1971 paper endorses Anthony's demarcation between three types of infantile psychosis (Kolvin et al 1971, p.381). James Anthony, a medical doctor based at the Maudsley Hospital in London, was a major figure in British child psychiatry (Evans 2014, p. 262). Anthony's first group was early onset and covered Kanner's autism and Bender's pseudo-defectives; his second group "at three to five years with an acute course followed by regression, included Heller's disease, De Sanctis and Weygant's dementias, Bender's pseudoneurotic, Despert's 'acute onset' type and Mahler's symbiotic psychosis" (Kolvin et al 1971, p.381); his third group was late onset which covered Bender's pseudo-psychopathics. Anthony's scheme is representative of the main approaches used in the previous decades.

However, the diagnostic criteria Kolvin employs are more restrictive than Anthony's. Kolvin's criteria for early onset are substantially (though not entirely) based on Kanner's autism. As Miller remarks, "an implication of the position maintained by Rutter and Kolvin is that there is only one syndrome of psychosis with onset under age three – namely, infantile autism" (1974, p.7). We saw in chapter one that Kanner's autism was a quite specific manifestation whereas Bender's pseudo-defectives were broader. Also, 55 percent of Kolvin's autistic children did not use speech meaningfully (Kolvin 1971, p.388), whereas Kanner thought only a third did not, suggesting Kolvin's criteria may be lower functioning than Kanner's. Also, as mentioned above, schizophrenic children described in the earlier decades rarely hallucinated, whereas most late onset individuals in Kolvin's study did have hallucinations, making them an unrepresentative sample of childhood schizophrenia of the previous decades. Though claiming to employ Anthony's first and third categories,

Kolvin's criteria for his study is more restrictive than earlier notions of childhood psychosis, indeed, "[i]t took Kolvin many years to collect a series of 33 [children meeting his criteria]" (Rutter 1972, p.321).

The biggest problem with Kolvin's study is that higher functioning non-hallucinating individuals appear to be largely excluded by Kolvin's diagnostic criteria.[14] He states most shy, introverted children with schizoid personalities in the general population do not develop late onset psychosis (Kolvin 1971, p.385) and Kolvin excluded children "with less clear-cut symptoms" (Kolvin 1971, p.384), whereas US notions of childhood schizophrenia were often applied to a wide variety of children. Kolvin's 1971 paper is not fully clear as to why he excludes such higher functioning individuals, but this is primarily because Kolvin's 1971 sample only included individuals from Anthony's first and third group whereas individuals from Anthony's second group are not included. Their omission is not explicitly stated in his 1971 paper but it is explicitly acknowledged in a later paper (Kolvin 1985, p.148). A 1985 article provides a problematic justification for this exclusion. "The second group, those with an onset between three and five years, has been described as 'disintegrative psychosis' (Rutter, 1972) and is very rare (Makita, 1966; Kolvin et al, 1971e)... These children have mostly been found to be suffering from clear-cut organic disorders" (Kolvin 1985, p.148). Here Kolvin suggests Anthony's second group is disintegrative and very rare. However, Anthony's second group includes Bender's pseudo-neurotics but the clinical picture Kolvin describes here is very different from Bender's pseudo-neurotics. They were generally relatively high functioning and common among childhood psychotics. Bender considered pseudo-neurotics to have symptoms like preferring structured environments, intelligent, enjoying academic study, disliking new situations, rigid in approach, little awareness of self, fearing social situations, restricted range of actions and being obsessive (Bender 1959b, p.506). Additionally, Kolvin considers this second group acute onset (1971, p.381), whereas Bender's pseudo-neurotics could have acute or insidious onset. Finally, the cause of most pseudo-neurotics was unknown (and this is true of high functioning autistic individuals today whom pseudo-neurotics resemble), whereas

14 Another study which supposedly separated schizophrenic children from autistic children, referenced far less frequently than Kolvin's study, also suffered from being unrepresentative. Egger's study specifically intended his notion of schizophrenic children to be different to prior notions of childhood schizophrenia (1975, p.22).

Kolvin considers most in the second group to have an organic disorder. Note that Rutter also endorses Anthony's groupings (Rutter & Lockyer 1968, p.1172) whilst most participants (58 of 63) in his influential late 1960s study have age of onset below that associated with Bender's pseudo-neurotics (Rutter & Lockyer 1968, p.1172). Miller correctly remarks that "there is some evidence that psychosis with onset between three and six years of age is not so rare as reported by Rutter (1972) and Kolvin and co-workers (1971) and is not necessarily associated with organic processes" (1974, p.9). Let's consider why Kolvin would largely exclude higher functioning individuals from his study.

Kolvin employed a different notion of schizophrenia from that used in most of US child psychiatry. In 1972, he suggests US and UK child psychiatrists may be employing different notions of psychosis (Kolvin & Edgell 1972, p.285). He is clearest on this point in 1990.

> "Traditionally, diagnostic criteria have reflected various combinations of Schneider's (1959) approach, where there is more emphasis on positive delusions and hallucinations, with that of Bleuler (1950) where the emphasis is on the more negative symptoms of withdrawal, loosening of association, and blunted affect... In the original research of Kolvin and his coworkers [referencing his 1971 study]... there was an attempt to sharpen and limit the concept and definition of schizophrenia in school-aged children and diagnosis was achieved by using the rank criteria (Schneider, 1959)" (Kolvin, Berney & Yoeli 1990, p.102).

Kolvin's 1971 paper references Schneider when outlining diagnostic criteria for late onset children (Kolvin 1971, p.384). Kolvin appears to think many individuals diagnosed in the US are not schizophrenic, or at least schizophrenic in a very difference sense, to those in the UK. Similarly, he claims that "overdiagnosis is a greater problem in the United States than in the United Kingdom. In the former there is a tendency to use broader definitions and hence a wider concept of psychosis, so that the severe adolescent crisis is often perceived as being a schizophrenic state" (Kolvin 1972, p.816). Note also that Rutter's 1972 article arguing for the abandonment of childhood schizophrenia states that of "Bender['s] (1953) [childhood

schizophrenia], only a few were autistic by Kanner's criteria, and her use of the word schizophrenia is wider than WHO [World Health Organisation] definition followed here" (1972, p.329). Kolvin's and Rutter's approach, based in UK notions, are not particularly relevant for drawing conclusions about US child psychiatry of earlier decades.

Kolvin's later publications consider Asperger's syndrome to be different to his 1971 notions of early onset autism and late onset psychosis. "Asperger's syndrome (1944) which presents clinically as a schizoid-type personality disorder of childhood. Some authorities (Wing, 1981) regard it as a mild variation of autism, while others agree with Asperger that it is a personality trait (Kolvin and Goodyer, 1981)" (Kolvin 1985 p.150). Similarly, "[t]he deviance, patterning, and severity of features are insufficient for it [Asperger's syndrome] to be considered as psychosis but rather as a personality variant" (Kolvin, Berney & Yoeli 1990, p.108) and Kolvin thinks Asperger's syndrome is not separate from schizoid personality disorder (Kolvin, Berney & Yoeli 1990, p.108). Rutter's 1978 paper also considers this a possibility, Asperger's syndrome "thought to be a personality trait" (Rutter, 1978, p.145) but "it remains uncertain whether they constitute a distinct syndrome different from mild childhood autism" (Rutter, 1978, p.145). Kolvin appears to think, and Rutter considered it a possibility in 1978, that such abnormalities are just substantial variations on personality rather than distinct syndrome. I conclude that Kolvin's study missed out a population that had previously been considered to suffer disorders in the autism-childhood schizophrenia family by US child psychiatrists. Kolvin excluded high-functioning children suffering from a late-onset, disorder who did not hallucinate from his study. Excluding this population made it look like autism and childhood schizophrenia are clearly distinguishable conditions, whereas if this population had been included in the study I suggest the results would not have been so clear-cut.

2.4.4 Additional problems

Let's consider other flaws with Kolvin's study beyond his sample being unrepresentative of previous decades. Kolvin notes that some with late onset seemed closer to the early onset group than to adult schizophrenia, that "there are some

overlapping cases in which the margins between the two conditions might be blurred" (Kolvin 1971, p.394). Some late onset children without hallucinations had symptoms resembling those of the early onset group (Kolvin 1971, p.394). Equally, some with early onset disorders had all the symptoms of late onset disorders except hallucinations: "some of the older [early onset] cases with their suggestion of thought disorder and affective rigidity or poverty were almost indistinguishable in terms of behavioural features alone from certain of the [late onset] cases" (Kolvin 1971, p.394). Kolvin seems aware that, except for restrictions over age of onset, some of the late onset cases could fit the classification of early onset autism.

Furthermore, Kolvin's notion of late onset actually allowed for prior abnormalities. Kolvin considered a child late onset only if specific symptoms of childhood schizophrenia were not present from birth. 87 percent of the late onset children had pre-onset conditions. They were considered odd by their parents and they had "shyness, diffidence, withdrawal, timidity and sensitivity" (Kolvin 1971, p. 385). This creates problems deciding which abnormal behaviour should be considered pre-onset and late onset, with Kolvin admitting to difficulties "trying to disentangle pre-morbid personality factors and pre-existing behaviour disorders from features of psychotic onset" (Kolvin 1971, p.386). Without clear symptoms only associated with late onset, non-hallucinating schizophrenic children have no exclusive symptoms absent in autism. Kolvin's study supports child psychiatrists who thought that some schizophrenic children had pre-onset abnormalities prior to full illness (Alderton 1966, p.281; Bender and Freedman 1959, p.564; Bradley 1941, p.49; Creak 1951, p.550; Richards 1951, p.300). Kolvin demarcates autism from childhood schizophrenia on age of onset despite most children with late onset having pre-onset conditions. This questions age of onset as best means of dividing autism from childhood schizophrenia.

Some of the schizophrenic children who overlapped with autistic children resemble modern notions of high functioning autism or Asperger's syndrome. Kolvin describes one such child

> "who starts off with poor speech development and combines this with some personality difficulties, behaviour disorder and poor intellectual endowment, is perhaps made worse by teasing in early school years,

> and drifts almost imperceptibly and insidiously around puberty into a simple schizophrenic state" (Kolvin et al, 1971, p.395).

Schizophrenic children who overlap with autism in Kolvin's study parallel children described by child psychiatrists in 1950s to 1970s and bare similarities to high functioning autism of today.

2.5 Reactions to Kolvin

2.5.1 Scientific Reactions to Kolvin

I argue that Kolvin cannot be said to have proven schizophrenic children of the previous decades should be considered early adult onset schizophrenic rather than autistic or have their own category. As shown earlier, some historians consider Kolvin as accomplishing this when they claim Kolvin separated autism from childhood schizophrenia. Let's now consider what the 1980s and 1990s made of Kolvin's findings. Consider the following references, which either refers to Kolvin and colleagues or Rutter and colleagues.

> "[The] age and developmental stage were important criteria for classification... Their importance was demonstrated by several empirical studies (Rutter & Lockyer 1967; Rutter et al. 1967; Kolvin 1971; Kolvin et al. 1971. 1971a, 1971b, 1971c, 1971d, 1971e). Finally, these studies confirmed the notion of Kanner (1943, 1957), who subdivided childhood psychoses into three groups: early infantile autism, childhood schizophrenia, and disintegrative psychosis of childhood" (Remschmidt et al 1994, p.728).

Remschmidt and colleagues are correct to claim Kanner did indeed employ autism and childhood schizophrenia whilst acute childhood schizophrenia covered childhood disintegrative disorder and hallucinating acute onset children.[15] However, for Kanner,

15 Though Remschmit and colleagues characterisation of Kanner does not fully account for Kanner's position since he employed other diagnosis (children and circumscribed interests and symbiotic

autism was rare and had very specific diagnostic criteria, whilst childhood schizophrenia was much more common, could be present from birth onwards and only involved hallucinations very rarely. More importantly, Kanner recognised autistic children and schizophrenic children had great similarities from around five years, differing over specific symptoms being present or absent from birth. For Kanner childhood schizophrenia is completely different to early adult onset schizophrenia, meaning Remschmidt et al are deeply mistaken to consider Kolvin's conclusions as relevant for the notion of childhood schizophrenia Kanner employed. Let's consider 1980s papers. "Subsequent data clarified important features of the autistic syndrome... its lack of association with other disorders such as schizophrenia (Kolvin, 1971; Rutter, 1972)" (Cohen, Volkmar & Paul 1986, p.158). Also,

> "[f]or a number of years, there were some who held that early infantile autism, or other forms of severe developmental disorder, might be such an expression [of early adult schizophrenia]... more recent epidemiological studies and retrospective chart reviews (Kolvin et al., 1971; Rutter 1972) have tended to discredit this idea" (Tanguay & Cantor 1986, p.591).

Additionally, see Cantor et al (1982, p.758), Kydd & Werry (1982, p.344) and Volkmar et al (1988, p.191). When claiming Kovlin separated autism from childhood schizophrenia these papers cite child psychiatrists like Bender, Kanner, Eisenberg, Despert, Mahler, Rank, Szurek, Bradley, Fish and Potter but, as I argued, Kolvin's work has little relevance to the notion of childhood schizophrenia these child psychiatrists employ.

2.5.2 DSM and Kolvin

Kolvin's conclusions were initially largely adopted by DSM-III then slowly largely reversed in later editions.

DSM-I and DSM-II included childhood schizophrenia, but not autism, as a

psychosis) and considered these, alongside autism, as subtypes of a childhood schizophrenia which covered both insidious and acute onset.

separate diagnosis. DSM-I lists no additional symptoms for childhood schizophrenia than those listed for schizophrenic children (APA 1952, p.28) except "[p]sychotic reactions in children, manifesting primarily autism" (APA 1952, p.28). The DSM-II added psychoanalytic concepts to childhood schizophrenia whilst autism is still a symptom (APA 1968, p.35). The DSM-I and the DSM-II seemingly take autism and childhood schizophrenia as the same disorder.

The DSM-III explicitly splits childhood schizophrenia into three categories, autism, childhood onset pervasive developmental disorders (COPDD) and schizophrenia occurring in childhood (APA 1980, p.375). The symptoms of autism are similar to those described by Kanner. The symptoms of COPDD are social difficulties and odd behaviour but "the full syndrome of Infantile Autism is not present" (APA 1980, p.89). The symptoms of schizophrenia occurring in childhood are the same as adult schizophrenia (APA 1980, p.35) including hallucinations and delusions (APA 1980, p.89). Pre-adolescent onset is said to be rare (APA 1980, p.184). Onset for autism is prior to 30 months (APA 1980, p.88) whilst the onset of COPDD is two and a half to twelve years (APA 1980, p.90). Autistic individuals do not hallucinate as children or adults (APA 1980, p.375), children with COPDD do not hallucinate as children (APA 1980, p.91) but it is unknown if they will hallucinate as adults (APA 1980, p.375) and children with schizophrenia occurring in childhood do typically hallucinate (APA 1980, p.89). Autism is "very rare (2-4 cases per 10,000) (APA 1980, p89) and COPDD is "extremely rare" (APA 1980, p.91) whilst schizophrenia occurring in childhood is even rarer. COPDD appears to cover both the children between 3-5 who decline to almost no functionality and the non-hallucinating schizophrenic children. On this basis, it had the potential to cover higher functioning individuals. However, COPDD does not have all the symptoms of autism which schizophrenic children used to have. Also, whereas before autism was a rare subtype of childhood schizophrenia, now autism is still rare but more common than COPDD.[16] DSM-III does have Infantile Autism, Residual State for individuals who once met the criteria for infantile autism but no longer do (APA 1980, p.90), potentially covering individuals who met Kanner's stringent diagnostic criteria before three years but whose symptoms then changed. There is also Atypical Pervasive Developmental

16 Whilst DSM prevalence figures are sometimes unreliable, especially when new categories are introduced, research in 1988 still considered DSM-III COPDD as rarer than DSM-III autism (Volkmar et al 1988, p.198).

Disorders, “for children with distortions in the development of multiple basic psychological functions that are involved in the development of social skills and language that cannot be classified as either Infantile Autism or Childhood Onset Pervasive Developmental Disorders” (APA 1980, p.92). This lacks the rich descriptive clinical picture provided by childhood schizophrenia in earlier decades. I shall continue my comparison of DSM editions before considering DSM-III schizoid and schizotypal personality disorder.

The DSM-III-R replaces autism and COPDD with pervasive developmental disorders (PDD). PDDs come in two forms, a severe version named autism plus a less severe version named PDD-NOS (pervasive developmental disorders not otherwise specified) (APA 1987, p.34). The children aged 4 to 5 who decline to almost no functionality can be diagnosed as autistic or PDD-NOS (APA 1987, p.36). DSM-III-R has two important relevant differences to DSM-III. Firstly, DSM-III COPDD could cover high-functioning individuals but COPDD was rarer than autism. In contrast, DSM-III-R considers PDD-NOS as three times more common than autism, meaning higher functioning individuals are now more common than lower functioning individuals (APA 1987, p.37). The second important difference is that higher functionality is not tied to age of onset. Most PDD (covering autism and PDD-NOS) have an age of onset prior to three (APA 1987, p.36) and onset after five or six years is very rare (APA 1987, p.36). In the DSM-III onset prior to three was slightly more common than onset after three whereas in DSM-III-R onset prior to three is far more common. DSM-III-R now has a basic notion of modern high and low functioning autism, with autism as lower functioning and PDD-NOS as slightly higher functioning. However, late onset non-hallucinating children have become even rarer. This partially reinstates Kanner's approach of having an overarching classification plus specific subtypes. Early pre-adolescent schizophrenia is still placed with the schizophrenia section (APA 1987, p.187) and is said to be rare (APA 1987, p.190).

Adopting an approach even closer to Kanner's, DSM-IV expands PDD and loosens age of onset as a requirement for a diagnosis. PDD now also includes childhood disintegrative disorder which describes the children aged 3 to 5 which Kolvin described who decline to near zero functionality (though onset in DSM-IV can be two to ten), Asperger’s Syndrome and Rett's disorder. Asperger's syndrome is now listed separately and is similar to autism except that such children do not have delayed

language development (APA 1994, p.69). Also, “Asperger's Disorder appears to have a somewhat later onset than Autistic Disorder, or at least to be recognized somewhat later” (APA 1994, p.76). Meanwhile, the addition of childhood disintegrative disorder allows for a diagnosis with substantial similarities to autism for children who decline around three or four years of age (APA 1994, p.74). The addition of Asperger’s Syndrome which does not have to present before three years of age and occasional cases similar to autism occurring after three years mean that the DSM-IV is far more compatible with late onset non-hallucinating children than DSM-III or DSM-III-R. Schizophrenic pre-adolescents are still placed within the schizophrenia category and are said to be rare (APA 1994, p.281).

After no changes in DSM-IV-TR (2000), DSM-5 made some substantial changes. All the subtypes were removed and subsumed into an autistic spectrum.

> “Autism spectrum disorder encompasses disorders previously referred to as early infantile autism, childhood autism, Kanner's autism, high-functioning autism, atypical autism, pervasive developmental disorder not otherwise specified, childhood disintegrative disorder, and Asperger's disorder” (APA 2013, p.53).

Now there is no longer any distinction between autism and late onset. All symptoms can be present from birth or after a period of normality. This classification is now closer to Bender's childhood schizophrenia than the previous three DSM, an overarching classification which covered high and low functioning autism and covered present from birth and delayed onset (though onset must be earlier than some of Bender's schizophrenic children since “[s]ymptoms must be present in the early development period” (APA 2013, p.50)). Early adult schizophrenic individuals are still considered schizophrenic and are still rare (APA 2013, p.102).

The changes in the DSM have been quite substantial. From the 1940s to the 1970s higher functioning individuals were considered more common than lower functioning individuals and late onset childhood schizophrenia was more common than early onset autism. DSM-III put early onset prior to three as more common than onset after two and a half years, whilst considering the lower functioning clinical picture as more common than the higher functioning clinical picture. The DSM-III-R

put early onset as much more common than late onset and higher functioning as more common than lower functioning, a situation retained by later editions. By including focus on higher functioning individuals, DSM-III-R and subsequent editions once again covered clinical pictures employed prior to DSM-III. The DSM-III changes described here were largely reversed by later editions; I discuss areas which were not reversed in section 2.7.1.

2.5.3 Schizoid and schizotypal personality disorder

An alternative location where autism might be located is schizoid and schizotypal personality disorder. Here I will discuss DSM-III and DSM-III-R schizoid and schizotypal personality disorder, alongside Sula Wolff's views.

DSM-III schizoid disorder of childhood and adolescence does overlap with some of the phenomenology of modern autism. Such individuals “often appear aloof, reserved, withdrawn, and seclusive” (APA 1980, p.60), they are “sensitive to criticism” (APA 1980, p.61) and “often are vague about their goals, indecisive, absentminded, and detached from their environment (“not with it” or “in a fog”). They often appear self-absorbed and engage in excessive day dreaming. They tend to pursue solitary interests and hobbies” (APA 1980, p.61). It can be seen as early as age five (APA 1980, p.61), just after when Bender's pseudo-neurotics could emerge. Also, it is more common in boys than girls (APA 1980, p.61). All this provides some compatibility with modern autism. However, the repetitive activities of modern autism are absent, differential diagnosis from pervasive developmental disorders being on no “marked defects in multiple areas of functioning” (APA 1980, p.61). Also, they “are often preoccupied with esoteric topics, such as violence or supernatural phenomena” (APA 1980, p.61), covering additional symptoms to modern autism. The clinical picture in DSM-III-R is largely similar and DSM-IV differentiates milder autism and Asperger's syndrome from schizoid by “more impaired social interaction and stereotyped behaviours and interests [in the latter]” (APA 1994, p.640).[17]

DSM-III schizotypal has some similarity to modern autism, though less than

17 Note that DSM-III has schizoid disorder of childhood and adolescence alongside schizoid personality disorder (which is for adults), whereas DSM-IV removes the childhood specific diagnosis but schizoid personality disorder affect both children and adults.

schizoid. In schizotypal “speech shows marked peculiarities: concepts may be expressed unclearly or oddly or words deviantly” (APA 1980, p.312), “odd speech... e.g., speech that is digressive, vague, overelaborate, circumstantial, metaphorical” (APA 1980, p.313). Also, “[u]sually some interference with social or occupational functioning occurs” (APA 1980, p.312). This seems weaker than modern autism which emphasises communication difficulties and both DSM-III and DSM-III-R schizoid which emphasise solitariness. However, DSM-III-R places more emphasis on social impairment (APA 1987, p.341). Schizotypal also covers “disturbance in the content of thought [which] may include magical thinking (or in children, bizarre fantasies or preoccupations), ideas of reference, or paranoid ideation. Perceptual disturbances may include recurrent illusions, depersonalization, or derealization” (APA 1987, p.312). These slightly overlap with modern autism, though symptoms not associated with modern autism are present such as the disturbed thinking and magical thinking. Also, as with schizoid, the repetitive routines are not covered and these are used for differential diagnosis from milder autism and Asperger's syndrome in DSM-IV (APA 1994, p.644).

It is briefly worth considering the work of Sula Wolff, a British based psychiatrist working in Edinburgh who pioneered notions of schizoid personality in childhood. Though publishing in the late 1970s and early 1980s, her notions did not influence DSM-III notions of schizoid disorder of childhood and adolescence or schizotypal personality disorder. However, considering Wolff provides some context to the work of British based Kolvin and Rutter. Additionally, the workbook describing the process of formulating DSM-IV considered Wolff's schizoid children as PDD-NOS) (Szatmari 1997a, p.45). Whilst DSM-III was not historically continuous with earlier decades it is still important to consider if non-DSM notions deliver historical continuity.

Wolff describes schizoid personality disorder children as exhibiting

> “(1) emotional detachment and solitariness, present in all subjects; (2) rigidity (or lack of adaptability), at times assuming obsessional proportions and often expressed in the form of long-lasting, circumscribed interests or preoccupations; (3) sensitivity with occasional suspiciousness and paranoid ideation; (4) lack of empathy for the

> feelings of others, at times amounting to callousness; and (5) odd ideation, often with metaphorical use of language and marked lack of guardedness. Although resembling autistic children in some respects they never exhibited the 3 cardinal features of this syndrome (absent or impaired language development with echolalia; lack of emotional responsiveness with gaze avoidance; ritualistic and compulsive behaviour), all beginning under the age of 2 years" (Wolff & Chick 1980, p.89).

This 1980s description, covering the DSM-III era, describes many aspects of modern autism by including both communicative and repetitive behaviour abnormalities. It is broader than modern autism because communication abnormalities can take the form of being too communicative and because of focusing on thought disorders, though both these were also true of childhood schizophrenia of earlier decades.

Despite this, Wolff's notion of schizoid does not adequately cover the missing clinical pictures absent from DSM-III which were described in earlier decades. Wolff writes that "[o]ur concept of schizoid personality has most in common [of DSM-III diagnoses] with the DSM-III definition of schizotypal personality disorder" (Wolff & Cull 1986, p.678), whereas DSM-III schizoid personality disorder seems closer to modern autism than DSM-III schizotypal. She did, however, later link her notion of schizoid to both DSM-III and DSM-III-R schizoid and schizotypal (Wolff 1991, p.619). Wolff considered her schizoid personality disorder as identical to Asperger's syndrome (Wolff & Chick 1980, p.88) as described by Asperger but different to Asperger's syndrome as described by Wing. She considered schizoid personality disorder as higher functioning than and much more common than Wing's Asperger's syndrome, which she considers rare (Wolff 1991 p.616-617). She maintained this stance over DSM-IV Asperger's syndrome. For Wolff's schizoid children, a diagnosis of "Asperger syndrome is inappropriate unless its criteria both in DSM-IV and ICD-10 are modified to omit the exclusion of significant delays in speech and language and of schizoid and schizotypal disorders; to specify the less severe social impairments and more sophisticated all-absorbing interests in comparison with autism; and to include a criterion for unusual fantasy" (Wolff 1995, p7). Wolff's schizoids are higher functioning than pervasive developmental disorders in DSM-III-R and DSM-IV. If

Wolff's schizoids were added to DSM-III then lower functioning individuals and much higher functioning individuals would be covered but this still leaves a gap (individuals not as impaired as lower functioning but more impaired than very high functioning) which DSM-III-R PDD-NOS and DSM-IV Asperger's syndrome filled.

2.6 Lorna Wing, Asperger's syndrome and an autistic spectrum

The 1980s DSM removed childhood schizophrenia and reformulated autism into something very narrow, leaving 1980s autism historically discontinuous with before and afterwards, notions similar to high functioning autism being absent during this period. However, this was partly reversed in DSM-III-R which has a distinction between high and low functioning autism, then near fully reversed with DSM-IV which introduced Asperger's syndrome. These changes can be traced back to Lorna Wing's work.

Wing's (& Gould's) 1979 epidemiological study was a major step in formulating modern autism, ultimately introducing the autistic spectrum, the triad of impairments and high functioning autism. Wing's study took a selection of children known to a local health service and subdivided them into two categories, a socially impaired category and a sociable but severe mentally retarded category. She analyzed how various symptoms inter-related and checked the results against earlier classifications.

Wing found most children in the socially impaired category also exhibited other symptoms rarely present in the sociable but severely retarded group.

> “[M]uteness or echolalia, absence or marked repetitiveness of symbolic activities, and an interest pattern consisting entirely or partly of repetitive activities occurred in virtually all of the socially impaired group, but these items could also have been seen in a very significantly smaller proportion of the sociable severely retarded children. In the latter, absence of symbolic activities and an interest pattern dominated by repetitive behaviours were found only in children with language comprehension ages below 20 months” (Wing & Gould 1979, p.20).

Wing defined social impairment based upon mental age, social impairment being where social skills were lower than mental age (Wing & Gould 1979, p.15). Some children had normal intelligence with lower comparative social skills, some had low mental age but with higher comparative social skills and some had low mental age with social skills equivalent to that mental age. Irrespective of intelligence, the above pattern was found only in children with weak social skills.

Wing checked her results against historically employed classifications, including Kanner's 1943 autism and Asperger's syndrome. The only classification that appeared present within her results was Kanner's autism (Wing & Gould 1979, p.19) yet Wing advocates abandoning Kanner's autism. His autism became statistically insignificant within Wing's wider symptom pattern.

> "The social impairment subgroups did not differ significantly on the three speech and behavioural abnormalities associated with typical autism, but were significantly differentiated on all other cognitive and behavioural variables that were measured" (Wing & Gould 1979, p.25).

Kanner's classification was present within the wider group but insufficiently present, as measured by statistical significance, whilst other factors were more significant when measured by how symptoms cluster.

> "Of the two independent methods of subclassifying the socially impaired children, the system based on severity of social impairment gave more statistically significant associations with behavioural, psychological, and medical variables than that based on the presence or absence of a history of typical autism" (Wing & Gould 1979, p.25).

Let's consider what these were.

> "Mutism, echolalia, absence of or repetitive symbolic activities, and an

> interest pattern consisting entirely or partly of stereotyped activities did not differentiate those with and without a history of typical autism. On the other hand, they were very significantly associated with the degree of social impairment. Mutism and stereotyped repetitive activities characterized the aloof groups while the passive and the odd children were more likely to have repetitive speech and repetitive symbolic activities, but some constructive pursuits as well" (Wing & Gould 1979, p.22).

Wing argues Kanner's pattern is much less significant than level of socialising. Level of socialising was associated with other symptoms and this held for the entire group, whereas children who met Kanner's specific pattern did not correlate interestingly with the wider pattern. She later wrote that "Wing and Gould found that there were many more children who also had the triad but who did not precisely fit Kanner's descriptions of his syndrome" (Wing 1993, p.70; also Wing 1981, p.37). Autism for Wing was the triad of impairment co-occurring.

Wing found within her grouping one overall class and three subclasses which varied on level of functioning, thus she favoured an autistic spectrum, subsuming Kanner's autism alongside much else. However, DSM-III largely did not cover higher functioning autism except for the rare childhood onset pervasive developmental disorders. During this period Lorna Wing advocated for Asperger's syndrome and developed the autistic spectrum. She became interested in the more specific and richer classification of Asperger's syndrome because DSM-III was too restrictive (Happé 1994, p.84). Asperger's syndrome soon gained in popularity, filling the gap Kolvin and Rutter created. Autism was again linked with weak social skills, with mental retardation and with variations in severity. From around 1950 there was Kanner's autism, covering substantial variations in severity by age five. Also, schizophrenic children could start and remain as lower functioning pseudo-defectives, or start as higher functioning pseudo-neurotics, or move from pseudo-defectives to pseudo-neurotics. Now there is low and high functioning autism with potential movement from low to high. Unlike under Kolvin and Rutter, these three symptom patterns were covered again.

2.7 Underdetermination and DSM-III autism

Kolvin's study suggested three classificatory options. He could place non-hallucinating childhood schizophrenia with autistic children, with the early adult onset schizophrenia or provide them their own category. It is not obvious from his sample why one would choose the middle option. However, much more problematic is how unrepresentative his sample was for comparisons with previous decades. Including so many hallucinating individuals means the population in his study has very little resemblance to previous decades. However, there are only six non-hallucinating schizophrenic children in a study of 33 individuals despite being much more common during the previous decades than autism or hallucinating schizophrenic children (Alderton 1966, p.282; Bender 1947, p.50; Bender 1959b, p.501; Clardy 1951, p.82; Clobert & Koegler 1958, p.215; Despert 1940, p.190; Eisenberg 1956, p.21; Ornitz & Ritvo 1968, p.78; Ornitz 1973, p.33; Richards 1951, p.303; Robinson 1961, p.544). Additionally, non-hallucinating schizophrenic children exhibited a wide variety of symptoms. There was pseudo-defectives, pseudo-neurotics and pseudo-psychopathics (Bender 1959b, p.492), children with circumscribed interest (Robinson & Vitale 1954, p.755) and symbiotic psychosis (Mahler 1952, p.289). Adequately covering all these variations with six children is impossible. Though he had reasons for not including Bender's pseudo-neurotics, their absence means Kolvin's study has little relevance for notions of US childhood psychosis of the 1940s to 1970s – though scientists in the 1980s onwards believed that it did.

How does all this relate to the underdetermination argument? As chapter six will describe, whether there are grounds for belief partly depends upon whether a theory has theoretical virtues. One major theoretical virtue is unification, which Kitcher specifically appeals to for resolving underdeterminations (Kitcher 1993, p.255). This is where as few theories as possible are employed to adequately account for the phenomena of a domain. Ideally, scientists would employ one theory to cover an entire domain. However, this theory might struggle to cover the entire domain with much accuracy, being too general. Accuracy could be increased through creating an alternative theory which achieves greater precision by being narrower but covers fewer phenomena. Greater accuracy leaves some phenomena not accounted for.

Alternatively, we might replace the original theory with two theories, each being less general which allows more accurate description of phenomena. Of these two options, the second is generally significantly preferable. What the two theory approach loses in unification (employing two, rather than one, theory) is likely made up for in strength (covering more phenomena), making it superior to the single theory which leaves many phenomena unaccounted for.

Kolvin's and Rutter's modification of childhood schizophrenia similarly leaves phenomena unaccounted for. Previously, a scientific domain consisting of clusters of symptoms was covered by both autism and childhood schizophrenia. Upon implementing Kolvin's and Rutter's approach DSM-III radically narrowed the domain described. Those higher functioning autistic and schizophrenic children of the late 1940s to late 1970s did not fit DSM-III. Some may have received no diagnosis. Some were likely diagnosed with an alternative diagnosis than autism (such as schizoid or schizotypal) meaning some now had a diagnosis which did not adequately account for many symptom profiles whereas earlier notions of childhood schizophrenia and its subtypes would have. Alternatively, some were effectively misdiagnosed with autism because autism was the closest diagnosis. Eyal and colleagues al write that "by 1987 they [DSM-III-R committee] had evidence that clinicians were not, in fact, adhering to the DSM-III criteria" (2010, p.235). DSM-III-R then substantially reversed the changes. DSM-III autism has flaws which late 1940 to late 1970s childhood schizophrenia and DSM-III-R autism do not have. Underdetermination is acceptable when two theories make similar empirical and theoretical accomplishment but DSM-III autism is far worse on these measurements than historically continuous autism. Therefore, DSM-III autism is insufficient to support an underdetermination argument against historically continuous autism.

2.7.1 Pessimistic Meta Induction

Chapter one argued autism is resistant to pessimistic meta induction (PMI) because it has been historically continuous, yet this chapter offers a clear instance of historical change. This does not necessarily automatically entail PMI, but to understand why we need wait until chapter six. Briefly, PMI creates epistemic risk, our inferences

carrying more risk the stronger PMI is. If that risk exceeds acceptable levels of epistemic risk then belief is no longer justified. Additionally, PMI can have some strength but this may only undermine parts of a theory it applies to without undermining those parts it does not apply to.

DSM-III autism gives PMI some, though not massive, strength. DSM-III autism was adopted due to the influence of key sources of authority rather than ideal scientific debate. Diagnosticians then did not adhere to DSM-III autism. Within seven years the DSM-III changes were substantially reversed. The levels of historical discontinuity are not large. Consequently, this is not a classic PMI where large sections of a scientific community believe in false claims for many years, but a short term change implemented by key figures which was initially partially ignored and soon largely reversed. All this strengthens PMI but not in my judgement to excessive levels.

More worrying for belief in modern autism is that parts of autism from 1987 onwards are missing parts of late 1940s to late 1970s autism and childhood schizophrenia. There were two major differences of symptomatology, which I now consider.

From the 1930s (see Potter 1933, p.1267; Despert 1938, p.366) child psychiatrists believed many, perhaps the majority, of children who exhibited symptoms we identify with autism underwent a decline from normality. Most today would assume those child psychiatrists were largely mistaken. Declines from apparent normality do occasionally occur, a modern psychologist writing that "in some [autistic] children problems do not appear to be apparent early in development but then appear later, leading to a late diagnosis" (Kaland 2011, p.985). Some regression is quite common for autistic children, whereby they have some normal and some abnormal development, then the normal development declines. Previously normal children fully declining are rarer. Studies disagree upon prevalence, some reporting this in a third of cases. However, all these declines are usually before three years, with regression from normality after three years extremely rare (Stefanatos 2008, p309; Rogers 2004, p.141). Perhaps child psychiatrists of earlier decades were mistaken about age of onset, usually just mistakenly taking the age symptoms were noticed for the age symptoms first appeared. Plausibly, this was due to the influence of psychoanalysis, which suggested childhood schizophrenia partially involved a

reaction to the environment so could not be present from birth. However, PMI suggests our modern beliefs about age of onset are unreliable. It is not inconceivable that late-onset has greater prevalence than typically believed. Following PMI, claims over age of onset needs treating with great caution, potentially not meriting belief. Therefore, one aspect of modern autism may not merit belief, though this only entail doubts over what age symptoms are exhibited rather than if they are exhibited. PMI has some applicability but only to age of onset rather than over symptoms.

More worrying is that child psychiatrists, especially when discussing schizophrenic children, focused more on abnormal thinking than they do today. Evans (2013) has insightfully charted how wider changes to theoretical beliefs moved focus away from the inner psychic life of autism and childhood schizophrenia towards social and cognitive approaches. Whilst child psychiatrists of the 1950s to 1970s may have emphasised this too much, especially those employing highly theoretical psychoanalysis, modern psychiatry may place too little emphasis on this. It might be that abnormal thinking occurs in autism-related disorders but is currently under-investigated. PMI is applicable here, limiting the approximate truth of autism.

Some philosophers are untroubled by false past theories given how much more empirical evidence there is today (Fahrbach 2011, p.1283). With age of onset it is important to note that modern psychologists have much more information about the early life of individuals later diagnosed with autism through extensive screening programs and home video recordings, information which was far less present from the 1940s to late 1970s. Therefore, modern psychiatry goes some way to meet Fahrbach's condition of greater empirical evidence. In contrast, investigation of the inner psychic life is studied far less today than between the 1940s to 1970s. Whilst both are potential causes for worry, missed abnormal thinking is the greater concern and likely limits to a greater degree the approximate truth of modern autism.

2.8 Conclusion

The underdetermination argument is a threat to scientific realism, suggesting alternative scientific theories may be as good as currently employed ones. It is undermined by assessing strengths and weaknesses of various alternative, competing

theories. DSM-III autism is an alternative to historically continuous autism but this does not entail underdetermination because DSM-III autism has major flaws which were soon largely reversed.

DSM-III autism is a genuine alternative to historical variations of an autistic spectrum. Plausibly, negative extra-scientific factors unfairly worked in favour of DSM-III autism and against historically continuous autism. Despite this, diagnosticians often ignored this DSM-III approach due to its inadequacies. It was soon abandoned and replaced with a basic notion of an autistic spectrum in DSM-III-R. Reconstruction of Kolvin's approach shows substantial flaws. It is unclear that Kolvin choose the best way to classify non-hallucinating schizophrenic children given his data. Additional, the sample from which data was gathered was unrepresentative of previous decades. Finally, DSM-III autism did not accommodate many clinical pictures which were accommodated pre-DSM-III. Assessed by theoretical virtues, discussed in chapter six, DSM-III autism does weaker on unifications than historically continuous autism. DSM-III autism does not provide a successful underdetermination against historically continuous autism.

This chapter does give some limited strength to the pessimistic meta induction but not sufficient to carry substantial epistemic risk. Additionally, some significant concerns are raised about modern approaches to thought disorders as not meriting belief; thought disorders may be present and modern psychiatry would have higher approximate truth if it described them. Some weaker concerns have been flagged up about age of onset. Chapter six covers issues over belief, approximate truth and acceptable levels of epistemic risk. For now, neither PMI nor underdetermination offer strong reason to disbelieve in autism.

3.0 Chapter 3 – The Theoretical Robustness of Autism

3.1 Introduction

Having covered pessimistic meta induction and underdetermination let's now consider the third sceptical argument, theory laden nature of evidence. The worry is that what observations are made depends upon what theories the scientist holds.

> "Theory-ladenness implies that when an observer has a false theory their observations will be distorted by their false beliefs. Unfortunately it is likely that much current psychiatric theory is mistaken... psychiatrists' false beliefs can be expected to distort their observations of their patients and prevent them from seeing the true similarities and differences between types of mental disorder" (Cooper 2005, p.77).

If theories are epistemologically insecure then observations dependent upon those theories will also be epistemologically insecure. We would lack justification for believing in any aspects of autism dependent upon such theories.

I show autism is theory-laden but only in an unproblematic manner. To do this, I provide an historical analysis comparing descriptions of autism under psychoanalysis and cognitive psychology. Both offer radically different accounts of the causes and nature of autism. Though I do not specifically show either is false, I am not willing to claim either merits belief. This means any aspect of autism theory-laden by psychoanalysis or cognitive psychology is in turn epistemologically insecure. Autism merits belief to the degree to which belief is justified over the symptoms and how those symptoms are grouped together into a classification. Belief would be undermined if the symptoms or classification is dependent upon cognitive psychology or psychoanalysis unless I first showed those theories merit belief – something which I do not commit to. I can undermine such concerns by identifying parts of autism

which were described by both psychoanalysts and cognitive psychologists. My history shows most symptoms were described by both psychoanalysts and cognitive psychologists whilst the classification was formulated in fairly similar ways. Most symptoms and the classification are thus not dependent upon either theory. Assuming psychoanalysis or cognitive psychology (or both) turn out mistaken, this does not entail those symptoms or the classification will also turn out wrong.

The chapter first outlines theory-laden nature of evidence. I then discuss cognitive psychology and psychoanalysis. I show that most symptoms and classification of autism described by proponents of these theories are broadly similar to modern autism. Where there are differences I discuss them in detail. Finally, I compare my findings to different types of theory-ladeness.

3.2 Theory-laden nature of evidence

The classic text on the theory-laden nature of evidence is Kuhn (1996 [1962]) but Kuhn make many diverse claims which need separating out; Bogen (2013) divides Kuhn's claims into three types, ones which Cooper roughly also employs (2005, p.80).

The strongest version of theory-laden evidence is perceptual loading (Bogen 2013). Scientists with different theories see the world differently, they have different visual experiences depending upon what theories they hold. If the account of theory-laden evidence most applicable to autism is perceptual loading then it would appear that the symptoms of autism actually depend upon highly contingent theories, suggesting those symptoms would have weak epistemological justification.

The next strongest level of theory-laden evidence is semantic theory loading (Bogen 2013). According to this, “theoretical commitments exert a strong influence on observation descriptions, and what they are understood to mean” (Bogen 2013). If the 'observation descriptions' of autistic people are heavily influenced by theory then this also suggests the symptoms formulated are dependent upon theories, potentially ones with negative epistemic consequences.

The weakest level of theory-laden evidence is salience (Bogen 2013). This is where scientists look at or attend to different things based upon the theories they hold. If autism best fits this version of theory-laden evidence the strength of this

epistemological concern depends upon the extent to which theories narrow the attention of scientists.

Due to the perceptual nature of perceptual loading it is very difficult to establish instances of it from subjective reports of scientists. However, we do not have to find instances of perceptual loading to be sceptical about the symptoms or explanations of autism since semantic theory loading would be adequate for scepticism. If semantic theory loading does not apply then neither will perceptual loading, therefore establishing that the symptoms of autism avoid semantic theory loading also shows they avoid perceptual loading. Since semantic theory loading is much easier to detect this chapter will focus on semantic theory loading over perceptual loading. Salience will also be investigated, though this is less epistemologically worrying than semantic theory loading.

The historical study below intends to show that the observation description aspect of semantic theory loading largely does not apply to symptoms or classifications. This also rules out perceptual loading. By contrast, what is thought to cause symptoms is heavily theory laden and therefore we should not believe in meanings or explanations assigned to them. Salience does apply in a limited manner but not to the majority of symptoms of autism or the classification.

3.3 Psychoanalysis, cognitive psychology and autism

Kuhn outlines major changes in theoretical conceptions of the world, such as Newtonian to relativistic physics (Kuhn 1996, p.101-102) or the affinity theory of chemistry to Dalton's atomic theory of chemistry (Kuhn 1996, p. 130-133). Kuhn argues these involved a compete shift in perspective, the world appearing in a new way after theory change. Psychoanalysis and cognitive psychology are two very different ways to conceptualise autism. I examine descriptions of autism provided by proponents of each type of theory to see if they see autism differently.

I do not argue psychoanalysis and cognitive psychology are false but here I mention possible reasons for doubt. Psychoanalysis supplied explanations of autism that were once popular whereas now most popular explanation of autism comes from cognitive psychology. Employing the pessimistic meta induction, instances of shifting

theoretical explanations suggests theoretical explanation may change again, undermining belief in psychoanalysis and cognitive psychology. Secondly, both are potentially competing theories, providing potentially contradictory explanations of the same phenomena. Employing underdetermination, we cannot work out which one is correct.

There are four possible relationships between these theories and inferences of symptoms and classifications. Firstly, a psychoanalyst (for example) might explicitly employ psychoanalysis when describing symptoms and classifications. Secondly, a psychoanalyst might unconsciously employ psychoanalysis when describing symptoms and classifications. They may believe symptoms and classifications have been observed or inferred on some other theory, but actually the inference was primarily or entirely unconsciously due to their psychoanalytical theory. Thirdly, the psychoanalyst did not consciously or unconsciously use psychoanalysis when inferring symptoms and classifications. They fully endorse psychoanalysis, potentially attempting to employ psychoanalysis where possible, but symptoms and classification are inferred using theories other than psychoanalysis. Fourthly, the psychoanalyst consciously or unconsciously employed psychoanalysis but the inference process mostly rested upon other theories, psychoanalysis having little influence on the symptoms and classifications formulated compared to other theories. The first and second options undermine belief in symptoms and classification. The third and fourth possibility does not undermine inferences over symptoms and classification.

3.3.1 Modern Symptoms

Below are a list of symptoms taken from two of the most well-known texts on autism, Uta Frith's *Autism: Explaining the Enigma* (1989 1st edition, 2003 2nd edition) and Francesa Happe's *Autism: Introduction to Psychological Theory* (1994). The symptoms listed below provide a bench mark from which a comparison with psychoanalytical notions of autism could be measured by.

Happe	Frith
Social	Social
Pester with questions and monologues	Too talkative (p.3)

(p.19)	
Stereotyped and repetitive use of language (p.36)	Repetitive language (p.3)
Not reciprocal social interactions (p.83)	Not understand social concept (p.3)
Impaired recognition of affect (p.36), lacking empathy (p.83)	Difficulty interpreting emotion (p.109)
Odd language (p.19), idiosyncratic use of words, abnormal pitch, stress, intonation (p.37), stereotyped speech (p.83)	Idiosyncratic word use, peculiar phrases (p.121), peculiar voice (p.3)
Echolalia (p.19)	Echolalia (p.121)
Monotone voice (p.83)	Monotone voice (p.103)
Inability to share and direct attention (p.35), failure to respond to other's speech (p.36)	Respond less to name being called (p.103)
Possible delay or lack of development of speech (p.36)	Delayed or absent language (p.103)
Prominal reversal (p.37)	Substituting I and you (p.124)
Seem cut off from others (p.19)	Not look up when name called (p.2)
Little face expressions (p.83)	Fewer social smiles (p.103)
Failure to initiate or sustain conversation (p.37)	
Inappropriate gestures (p.83)	
Semantic conceptual difficulties (p.37)	
Fairly normal phonology and grammer (p.37)	
Abnormal non-verbal communication (p.37)	
	Little interest when spoken to (p.2)
	Social praise and disapproval difficult to apply (p.103)
	Dislike and not understand criticism (p.103)
	Literal (p.3)
	Less eye contact (p.103)
	Looks through people (p.103)
	Poor language (p.3)
Repetitive	Repetitive
Obsessions (p.37), repetitive activities (p.37), enjoys repetitive activities (p.83), stereotyped movements (p.83),	Absorbed in repetitive activities (p.2), stereotypies, obsessions, compulsions (p.173)

circumscribed special interests (p.83)	
Spinning wheels (p.19)	Spinning wheels (p.2)
Preservation of sameness (p.19)	Require specific order (p.2)
Resistant to change (p.83)	
Dislike routines being disrupted (p.37)	
	Hyperstimulous (p.169)
	Lower sensitivity to sensations (p.170)
	Prefers certain foods and clothes (170)
Other	Other
Good rote memory (p.83)	Good rote memory (p.145)
Clumsy, gait and posture odd, gross movements (p.83)	Ungainly gait (p.3), stiff posture (p.110)
Lack of pretend and symbolic play (p.37)	
No interest in fiction (p.19)	
Not creative and do not show pretend play, with inappropriate thoughts (p.84)	
	No anticipatory posture (p.2)
	Sensitive to certain sounds (p.2)
	Did not point to things or look at things when pointed at (p.2)
	Flapping hand (p.2)
	Good at geometry (p.3)
	Difficulty remembering faces (p.104)
	Naïve (p.3)
	Some good at reading and maths (p.142)
	Self absorbed (p.1), obstinate (p.3)
	Seeing everything as black and white (p.127)
	Difficulty with irony (p.128)
	No sense of humour (p.128)
	Islets of ability (p.143)

3.3.2 Modern Classifications

There are three popular approaches to subdividing modern autism. I discuss these then later see how these subdivisions relate to the three cognitive psychological theories. Firstly, DSM-IV subdivides Pervasive Developmental Disorders into autism, Asperger's syndrome and pervasive developmental disorders not otherwise specified

(PDD-NOS).[18] This is usually interpreted as each category reflecting severity of symptoms (though some rare PDD-NOS take a very severe late onset disintegrative form). Secondly, Wing subdivides autism into aloof children, passive children and active but odd children (Wing & Gould 1979). Thirdly, many endorse level of IQ for subdivision (Borden & Ollendick 1994, p.33; Prior et al 1998, p.900; Witwer & Lecavalier 2008, p.1621).

All these three subdivisions appear related. Wing's three categories are linked to severity of symptoms, aloof children being most impaired whilst active but odd are least impaired (Borden & Ollendick 1994, p.26). Similarly, aloof children are more autistic whilst higher functioning children usually fit active but odd (Belinger & Smith 2001, p.417). Caselloe & Dawson (1993, p.235) also found aloof children more autistic. So two subdivisions, Wing's categories and level of functioning, are related. Additionally, Wing's subdivisions relate to IQ. Aloof have the lowest IQ whilst active but odd children are highest (Belinger & Smith 2001, p.413; Castelloe & Dawson 1993, p.235; Joseph et al 2002, p.818). Finally, level of functioning and level of IQ also relate, higher functioning individuals usually, though not always, having higher intellect than lower functioning individuals. This suggests all three methods of subdividing autism are linked. The DSM classifies on level of functioning which fits Wing's classifications which also have different levels of functioning. Subdivisions also occur on level of IQ and this matches Wing's classifications which also differ on IQ, as do DSM subdivisions over levels of functioning.

3.4 Cognitive Psychology

There are three main cognitive psychological accounts of autism: problems with theory of mind, weak central coherence, or executive functioning. Here I establish if autism is theory-laden by any of these cognitive psychological theories by seeing if each describes symptoms or classifications which the others do not (I consider psychoanalysis later).

The account of autism that sees it as stemming from difficulties with theory of

18 I mention DSM-IV here because it covers the period in which most cognitive psychological texts I discuss were published. DSM-5 removes Asperger's syndrome but notions of a spectrum varying in severity are present (APA 2013, p.53) and arguably stronger than in DSM-IV.

mind reasoning claims non-autistic people have a theory of mind which autistic people either lack or are deficient in. Theory of mind is

> "being able to infer the full range of mental states (beliefs, desires, intentions, imagination, emotions, etc.) that cause action. In brief, having a theory of mind is to be able to reflect on the contents of one's own and other's mind… Difficulty in understanding other minds is a core cognitive feature of autism spectrum conditions" (Baron-Cohen 2001, p.3).

Not appreciating fully that other people are thinking beings, autistic people fail to understand the perspective of others. This causes autistic people social difficulties and accounts for their lack of imagination.

The account of autism that sees it as stemming from weak central coherence claims that non-autistic people form meanings from perceptual information whilst autistic people instead focus on individual pieces of information without placing a wider meaning upon it. Normal people

> process incoming information for meaning and gestalt (global) form, often at the expense of attention to or memory for details and surface structure. The tendency, referred to by Bartlett (1932) as "drive for meaning", was termed "central coherence" by Frith. Individuals with ASD were hypothesised to show "weak central coherence"; a processing bias for featural and local information and relative failure to gist or "see the big picture' in everyday life" (Happe & Frith 2006, p.5-6).

The focus on local information means that autistic people focus on detail, accounting for obsessive interests and their difficulties seeing the perspectives of others.

The final account suggests that non-autistic people use executive functioning for planning and decision making but autistic people may be deficient in it.

> " 'Executive functioning' is traditionally used as an umbrella term for

> functioning such as planning, working memory, impulse control, inhibition, and shifting set, as well as initiation and monitoring of action… Historically these functions have been linked to frontal structures of the brain, and to prefrontal cortex in particular. These functions share the need to disengage from the immediate environment in order to guide actions" (Hill 2004, p.191).

Difficulties with planning and decision making accounts for the focused and often impractical approach autistic people take to life.

Each cognitive psychological theory largely complements one another, rather than competes. Each theory broadly accepts the main findings of the other theories, in that each describes sets of behaviour which are generally accepted by cognitive psychologists. Weak central coherence occurs across the spectrum (Happe & Frith 2006, p.18; Pellicano et al 2006, p.91). Many autistic people pass basic theory of mind tests, though most struggle excessively with more complicated theory of mind tests (Baron-Cohen 2001, p.19). Rajendran & Mitchell (2007, p.233) think it is unclear if executive functioning deficits are universal in autism whilst Hill (2004, p.224) thinks executive functioning deficits are not universal. For example, an advocate of executive functioning is not sceptical about mentalizing deficits and does not think they can be explained away by executive functioning (Russel 1997, p.1). Disagreements are not about what symptoms are exhibited; rather disagreements mainly relate to the level of theoretical explanations, to whether one explanation reduces down to another or if one explanation causes another. Weak central coherence may be linked to executive functioning, "failure to process information globally might be argued to follow from problems in shifting between local and global levels" (Happe & Frith 2006, p.19) though it does not appear reducible to executive functioning (Rajendran & Mitchell 2007, p.243). Executive functioning may cause theory of mind deficits, presence of executive functioning being predictive of theory of mind deficits whereas the reverse is not true (Hill 2004, p.221). Theory of mind may actually be reducible to executive functioning (Rajendran & Mitchell 2007, p.237). Despite this overlap between all three theories, they currently are independent because no single explanation accounts for all three (Volkmar et al 2004, p.141).

Each theory broadly accepts modern autism; they do not each propose a

different account of the symptoms or provide different classifications. They are not mentioned within DSM. They are not mentioned in the DSM-IV sourcebook (which discussed reasoning for proposed changes from DSM-III-R to DSM-IV) for either autism (Szatmari 1997b) or PDD-NOS (which primarily discussed Asperger's syndrome) (Szatmari 1997a). Arguably, amongst the three most well-known books on autism are Baron-Cohen (1997), Frith (2003) and Happe (1994), and these are often employed on undergraduate courses. Frith (2003) and Happe (1994) discuss all three cognitive psychological theories. They believe all three cognitive psychological defects can occur in the same person, therefore, each defect is not specific to a subtype of autism. Baron-Cohen (1997) only discusses theory of mind but his account of the symptoms and classification of autism is similar to Frith (2003) and Happe (1994). Also, Frith (2003), Happe (1994) and Baron-Cohen (1997) define autism in terms of DSM symptoms, suggesting all three theories are compatible with DSM. A review article extensively covering autism (Volkmar et al 2004) discusses all three cognitive psychological theories but does not subdivide autism on them or associate each with different symptoms. Rajendran & Mitchell (2007) review all three theories whilst Baron-Cohen (2001) reviews theory of mind, Frith & Happe (2006) review weak central coherence and Hill (2004) review executive functioning but none discuss subdivisions of autism on cognitive psychological theories. Cognitive psychological theories seek to explain DSM accounts of autism rather than modify it.

All this suggests each cognitive psychological theory is not, when measured against one another, theory-laden in a problematic way. That is, if cognitive psychology were adopted as a standpoint, there appears no further problematic theory-ladeness in relation to autism from each theory. Whichever theory is used a similar account of autism is formulated, both at the level of symptoms and classification. On this basis neither perceptual loading nor semantic theory-ladeness seems applicable. However, salience is highly applicable. For example, an advocate of executive functioning remarks that

> "research – especially *recent* research – in autism has neglected the existence of behavioural rigidity in its various forms (resistance to change, obsession with regularity, stereotypes, lack of spontaneity)" (Russell 1997, p.1)

Russell describes how Theory of Mind dominated autism research and how this made psychologists focus on particular aspects of autism at the cost of others. Inhibiting research in this manner is clearly unwise. Similarly, Russel suggests treatment can be influenced by which cognitive psychological theory is employed. If a theory of mind module never developed, it cannot be remedied by medication, so treatment usually focuses on training programs for teaching other means of assigning mental states to others. In contrast, problems with executive functioning could be caused by different parts of the brain not communicating because chemical neurotransmitters are not activating, something which drugs might potentially assist with (Russel 1997, p.15). Additionally, modifying the environment might be more suitable than training programs for weak executive functioning (Russel 1997, p.15). Salience is important but it does not raise substantial problems for scientific realism. It just means some parts of autism are emphasised more or less than other symptoms; it does not entail these parts are real or not real.

3.5 Psychoanalysis

Psychoanalysts do not clearly outline specific classifications. Consequently, psychoanalytical texts need detailed investigation to establish what symptoms they associated with autism and what classifications they employ. The texts chosen are Bettelheim (1967) because it is the most famous psychoanalytical text on autism, Tustin (1981) because it is often credited as the most sophisticated and Rhode & Klauber (2004a) because is covers relatively recent notions of autism, basing its notion of autism on the DSM-IV.[19]

3.5.1 Psychoanalytical explanations of autism

Historically, many psychoanalysts believed autism is the rejection of or lack of a notion of external reality, caused by an inability to accept an aspect of external reality.

19 The book describes itself as the only book to analyses Asperger's syndrome from a psychoanalytical perspective.

According to some psychoanalysts all children initially lack a concept of the external world, only being aware of themselves, making everyone initially autistic (Bettelheim 1967, p.4; Tustin 1981, p.18).[20] As they age children gain awareness of the external world, however, some encounter aspects of reality they cannot deal with, consequently rejecting external reality and remaining autistic. Bettelheim thinks autism is caused by the child firstly rejecting the mother which then causes the mother to consciously or unconsciously reject the child. This means the child does not gain an adequate response from their environment. "[A]utism is a state of mind that develops in reaction to feeling oneself in an extreme situation entirely without hope" (Bettelheim 1967, p68). The child believes they cannot influence the world, consequently retreating into their own world (Bettelheim 1967, p.46). Tustin thinks the realisation there exists an external world where desires might go unsatisfied is a massive stress (Tustin 1981, p.5). Children usually build up capacities to handle this stress prior to awareness of the external world, however, sometimes the realisation occurs before the capacities develop and sometimes the capacities never develop (Tustin 1981, p.5). Unable to accept external reality they remain autistic. "[A]utism is a system of *protective manoeuvres*, the function of which is to prevent or to massively diminish contact with the 'not-me' outside world" (Tustin 1981, p.173 emphasis original).

Rejecting the external world causes abnormal development. Bettelheim thinks that in the autistic child the personality does not develop properly, writing that "while they probably do not experience the world as the infant does, what they experience is not mediated through a complex personality, or at least not as complex as in normal children their age" (Bettelheim 1967, p.5). Emotional and intellectual level remains at or returns to the autism stage, focusing on the self and not the outside world. Tustin thinks rejecting the external world and a focus on the self results in abnormal notions of reality. She writes "the 'reality' of a psychotic child are [sic] different from ours because the apparatus which makes the construction [of someone's notion of reality] is in a different state of formation" (Tustin 1981, p.216). Both Bettelheim and Tustin think that autistic children develop abnormally.

Bettelheim and Tustin think unusual development gives autistic children an abnormal understanding of the world, including developing abnormal concepts. These abnormal concepts then influence how the child interacts with the world. Bettelheim

20 Tustin later abandoned this claim (1991, p.585) and the modern text on Asperger's syndrome specifically rejects it (Rhode 2004a, p.13)

thinks they fear disappointment so they refuse to hope by assuming the future cannot change, assuming the future will always be the same as the present. Believing there will be no changes means "[i]n the autistic child's world the chain of events is not conditioned by the causality we [non-autistic people] know" (Bettelheim 1967, p.84). Rejecting typical notions of causation also means they reject typical notions of time (Bettelheim 1967, p.84). Tustin thinks autistic children, having rejected external reality, focus on immediate sensation and conceive of the world in terms of sensation. "[F]or the infant, "being" is a stream of sensations. Put in another way, in earliest days, the infant *is* the stream of sensations from which constructs gradually emerge as nameless entities" (Tustin 1969, p.31 emphasis original). Thus the world for autistic children consists of "bodily rhythms and crude sensations" (Tustin 1981, p.254) and they interpret the world in light of these bodily sensations unlike most people who interpret external things socially. "[T]hese auto-generated, auto-sensual protective reactions appear in external behaviour as what are usually called 'stereotypes (rocking, hand flapping, finger flicking, twirling, object twirling, toe walking, etc.)" (Tustin 1991, p.588). Bettelheim and Tustin believe abnormal understanding causes many of their symptoms.

3.5.2 Bettelheim

- 3.5.2.1 Symptoms

Most of the symptoms described by Bettelheim in his book *The Empty Fortress* are also associated with modern autism, as covered in the table above.

Symptoms similar to modern autism	Symptoms not similar to modern autism
Routines (p.53)	Initially developed speech and then gave up (p.31)[21]
Rituals and terrible reaction if ritual fails (p.54)	"unremitting fear for their lives" (p.63)
Fantasy world (p.55)	"seem convinced that death is immanent" (p.63)
Mutism (p.56)	Disorganised thinking (p.80)

21 Whilst a small minority of mute autistic children do initially talk then lose the ability, this is extremely rare whereas Bettelheim claims this always occurs.

Withdrawn (p.56)	Hallucinations (p.116)
Talk to self (p.57)	Limp and non-reactive (p.101)
Self-created language (p.57)	
Insensitive to pain (p.57)	
Poor coordination (p.59)	
Insistence on sameness (p.62)	
Total non-action (p.62)	
Hostility (p.62)	
Rote memory, listing name or places (p.67)	
Anxiety (p.74)	
No practical skills (p.75)	
Immaturity (p.75)	
Desire for sameness (p.83)	
Limited diet (p.89)	
Echolalia (p.162)	
Pronoun reversal (p.462)	
Makes geometric patterns typical of autism (p.145)	
Non-social play (p.219)	
Intellectual difficulties (p.230)	
Likes spinning stuff (p.233)	
Repetitive activities, narrow interests (p.243)	
Obsessions (p.245)	
Stereotyped movements (p.243)	
Uses third person to talk to others (p.244)	
Likes making long lists (p.429)	

This list covered many symptoms of modern autism. On this basis there is no immediate large threat from theory-ladeness over symptoms. I will discuss dissimilar symptoms later.

- 3.5.2.2 Classification

Bettelheim mentions two different classifications of psychotic children but provides few specific symptoms for each classification. Schizophrenic children have the least autistic withdrawal. Autistic children insist on maintenance of the same and have more autistic withdrawal. Also there are mute children who are the most autistic withdrawn

(Bettelheim 1967, p.75-76). No other symptoms are specifically assigned to any classification and Bettelheim never elaborates upon the relationship between these disorders except to describe autism as a more severe subgroup of childhood schizophrenia (Bettelheim 1967, p.6). Though he does not explicitly state this, occasional comments suggest that all claims about schizophrenia also apply to autism but autistic individuals have additional characteristics (for example, see Bettelheim (1967, p.67)).

Bettelheim provides no firm figures for age of onset. Autistic children are normal until 18 to 24 months and "no obvious deviation or dramatization [is evident] at the earliest age, though it may have occurred" (Bettelheim 1967, p.31). This suggests Bettelheim believes autistic children decline from normality but at ages substantially lower than typical accounts of childhood schizophrenia. Bender, for example, thought pseudo-neurotics decline from around age four. Contradictory, he says autism is present from one year of age (Bettelheim 1967, p.393). The only figure given for age of onset for childhood schizophrenia is a specific child who declined at age four (Bettelheim 1967, p.118). Whilst all these figures are compatible with autism as early onset and childhood schizophrenia as late onset, more clarity from Bettelheim would be required for this conclusion.

Bettelheim believes schizophrenic children have better prognosis than autism. Schizophrenic children usually recover after a few years of intense therapy (Bettelheim 1967, p.409) but most still have problems. They still have difficulties with empathy (Bettelheim 1967, p.416) and about half who recover are still schizoid or borderline (Bettelheim 1967, p.415). This parallels Kanner's autism where some individuals became much less impaired as they aged and Bender's schizophrenic children where heavily impaired pseudo-defectives could grow into less impaired pseudo-neurotics and even less impaired pseudo-psychopathics. Although no symptoms are given for schizoid and borderline, children who lack empathy and have sufficient symptoms (the post-recovery abnormalities) to warrant a new diagnosis possibly suggests modern high functioning autism. If recovery is interpreted as evidence of higher functioning autism then the lower rates of recovery for autistic children suggests lower functioning. Bettelheim can be interpreted as having both a high and low functioning autism, corresponding to his notions of childhood schizophrenia and his notion of autism.

3.5.3 Tustin

- 3.5.3.1 Symptoms

To understand the symptoms Tustin assigns I must first briefly outline her classification. Tustin bases her classification on psychoanalytical theory. Children might fail to accept the distinction between themselves and the external world in two different ways. Those who retreat into their own world, rejecting external reality, are encapsulators; they encapsulate themselves in their own world and are "scarcely aware of the outside world" (Tustin 1981, p.39). Tustin believes her classification of encapsulators is "virtually identical" (Tustin 1981, p.34) to autism (she does not specify which diagnosis of autism she refers to). Alternatively, children who fail to make the distinction between themselves and the external world are confusional; they confuse the internal world and the external world. Discussing confusional children she writes that "some of whom have features in common with adult schizophrenics... but not all Confusional children are Childhood Schizophrenics" (Tustin 1981, p.34). Her psychoanalytical theory partially matches classifications employed between 1943 and 1978.

The below table lists symptoms associated and not associated with modern autism for both encapsulators and confusional children.

Autistic symptoms of both Encapsulators and Confusional	
Clumsy (p.21)	
Fascinated by mechanism (p.21)	
Spin objects obsessively (p.21)	
Hard rather than soft cuddly toys (p.21)	
Fussy eaters (p.21)	
Use other people's limbs to open doors (p.21)	
Often mute (p.21)	
Avoid looking at people (p.21)	
Intelligence seems likely (but untestable) (p.21)	
Preservation of sameness (p.37)	
Weak symbolising capacities (p.123)	
Sometimes strong sensory powers (p.138)	
Autistic symptoms of Encapsulator	Autistic symptoms of Confusional
Avert their eyes (lack eye contact) (p.35)	Some hyperactive (p.36)
Shy and withdrawn (p.35)	Play in unhealthy way (p.39)

Quiet and gentle (p.36)	May use pronoun reversal (p.40)
Unusual memory (p.37)	
Do not play (p.39)	
Rarely talk and when do talk use echolalia and pronoun reversal (p.40)	
Do not body mould (p.35)	

Non-autistic symptoms of Encapsulator	Non-autistic symptoms of Confusional
Difficulties with contrasts like hot and cold, light and dark (p.133)	Difficulties with contrasts like hot and cold, light and dark (p.133)
Some can spontaneously recover (p.21)	Some can spontaneously recover (p.21)
Not clumsy (p.35)	Soft and flaccid muscle tones (p.35)
Hard muscular bodies (p.35)	Do not avert eyes (they give eye contact) but might have unfocused and bleary eyes (p.35)
Aware of the shapes but not insides of objects (p.125)	Not shy and withdrawn (p.35)
	Bad health (p.36)
	Some limp and placid (p.36)
	Hallucinations (p.37)
	Heavy fantasy life but not psychologically sophisticated (p.40)

Encapsulators are reasonably similar to modern autism. The difficulty encapsulators have conceptualising contrasts and their unawareness of the insides of objects are symptoms not associated with modern autism. The claims that these children have hard muscular bodies is not incompatible with modern autism though if true it is puzzling that this symptom is absent from modern autism since it should be relatively easy to detect. It is possible she means hard and muscular in contrast to confusional children who have limp bodies. The claim that encapsulators can spontaneously recover is also incompatible though this only applies to very few encapsulators, suggesting the minority of encapsulators who do recover were not low functioning autistic. Whilst not identical, Tustin's account of encapsulators is largely compatible with modern autism.

Confusional children are more difficult to interpret as autistic. The least worrying symptoms are having a heavy but not psychologically sophisticated fantasy life. Autistic people are known to live in their own world, though Tustin describes this in far more detail than modern psychologists typically do. Having difficulty

conceptualising contrasts like light and dark is not associated with modern autism. The notion that confusional children do not lack eye contact is extremely worrying since this is relatively easy to detect in autistic people, are very common in autistic people and are considered a sign of autism by modern psychologists. Soft and flaccid muscle tones, bad health and being limp and placid have been linked to autism (Filiano et al 2002, p.438) though this is rare. Tustin says confusional children usually have this body type, meaning it is not rare. Confusional children being not shy and withdrawn could have some compatibility with modern autism, the low social skills of some autistic people causes them to be overly friendly, overly talkative, invading the space of others. It is unclear if 'not shy and withdrawn' means these confusional children are normal in this regard or are overly friendly, the latter making them compatible with autism. The most problematic are the notions that confusional children have hallucinations and that a small minority recover spontaneously. None of these are associated with modern autism. Although many symptoms of confusional children are associated with modern autism, a substantial number are not.

- *3.5.3.2 Classification*

Tustin divides both encapsulators and confusional children into primary and secondary types. Encapsulators are either globally encapsulated primary shell types (Tustin 1981, p.22) or secondary segmented types whose impairment "is not total as in the shell-type children" (Tustin 1981, p.23). Secondary segmented children are defined psychoanalytically, Tustin writing that "threatening 'not self' objects are broken up into segments until they can be brought together in familiar 'me' terms" (Tustin 1981, p.30), suggesting these children can partially understand the external world, unlike presumably primary shell types whose global impairment means the external world cannot be conceptualised in terms of the self and thus never accepted. The only symptom demarcating each group is shell-types are usually mute whereas segmented-types are usually not (Tustin 1981, p.23). A primary shell-type case study looks like low functioning autism (she presents no secondary segmented type case studies). Tustin appears to name confusional children with childhood schizophrenia as primary confusional children whilst confusional children who are not childhood

schizophrenic are secondary fragmented confusional children (Tustin 1981, p.70). Unfortunately, rather than list specific symptoms to demarcate primary and secondary confusional children she instead only provides a case study of each disorder (Tustin 1981, p.45-48) without any analysis. Both resembled low functioning autism and it was unclear how primary and secondary confusional children differ beyond having different psychoanalytical connotations.

Tustin claims that confusional children are more difficult to treat than encapsulators but it is unclear if this is related to severity of illness. This would initially appear to suggest that confusional children have a more severe illness than encapsulators in contrast to my claims in earlier chapters that suggested many schizophrenic children were higher functioning. However, Tustin's claims over difficulty treating each category appear based in psychoanalytical theory and might not be indicative of severity of illness. She writes that “treatment situation with regard to Confusionals is more difficult than with the Encapsulated Childhood Autistic because it is complicated by this pathological entanglement with the ‘not-self’, and because fragments of the ‘self’ are felt to be dispersed and scattered, so that ‘self’ and ‘not-self’ are inextricably confused” (Tustin 1981, p.34). Tustin appears to consider confusional children more difficult to treat because of their specific psychoanalytical problems. She makes similar claims about the sub-types of encapsulators and confusional children, writing that “secondary pathological autism of the segmented Encapsulated and the fragmented Confusional children has become intensified as the result of the disturbance, and this increases the difficulties in treating them” (Tustin 1981, p.55). This claim appears to only make sense if there is little connection between treatment and severity of symptoms. Primary shell-type encapsulators are mute and so probably would be considered very low functioning yet Tustin claims secondary segmented encapsulators are harder to treat, suggesting no link between Tustin's claims about treatment potential and level of functioning.

Tustin appears to endorse notions of age of onset employed from the 1950s to 1970. Encapsulators have symptoms present from birth or just after (Tustin 1981, p.36-37) whereas confusional start normal and then decline by before six to seven years of age (Tustin 1981, p.61). Tustin later changed her mind about age of onset and childhood schizophrenia. Writing in 1994, she no longer considered schizophrenic children to be in an autistic state (Tustin 1994, p.111). She also believes that children

with Asperger's syndrome had a late onset (Tusin 1994, p.144). Asperger's syndrome is not discussed in her 1981 book, however, due to their late onset they would be incompatible with encapsulators and have to fit confusional. This would heavily support the notion that at least some in Tustin's 1981 book would today fit Asperger's syndrome.

Tustin does not appear to link age of onset to specific outcome. Tustin states that both encapsulators and confusional children can become adult schizophrenic (Tustin 1981, p.62). This is problematic since neither autism nor Asperger's syndrome is today associated with schizophrenia. If Tustin's observations are accurate then clearly at least some encapsulators and some confusional children were not on the autistic spectrum. However, as discussed above, it looks like only primary confusional children resemble schizophrenic children, secondary confusional children do not resemble schizophrenic children. It would therefore be possible that only the secondary confusional children become adult schizophrenic whereas primary confusional children do not, meaning primary confusional children would still be compatible with Asperger's syndrome.

Rhode & Klauber, psychoanalysts I discuss below, suggest Tustin's classifications can be demarcated by presence or absence of theory of mind. "[C]onfusional entangled children (as opposed to shell-type Kanner's children) blur the difference between themselves and others by treating other people as though they represented aspects of themselves" (Klauber & Rhode 2004b, p.262). These are contrasted with shell-type children who "relate to others in terms of surface mimicry, if at all – as though they had no idea of the inner, mental life of other people" (Klauber & Rhode 2004b, p263). These two groups differ in respect to theory of mind. Confusional children appear aware that other people have mental states. By contrast, shell-type children do not understand that other people have mental states. No mention is made of the segmented children, who are the other type of encapsulator, so it is unclear if they have a theory of mind. Tustin makes no claims about confusional children and mental states but appears to agree encapsulators are not aware of the mental life of others, writing that encapsulators "have treated another person as if he had no life and identity of his [their] own" (Tustin 1981, p.33). Klauber's & Rhode's claim that confusional children have a theory of mind whereas encapsulators do not may be compatible with Tustin's classification. Klauber & Rhode then link

functionality to presence or absence of theory of mind, linking confusional children is Asperger's syndrome (Rhode 2004a, p.14) whilst shell-type children are linked to Kanner's autism (Klauber & Rhode 2004b, p.262) which is usually associated with low functioning autism. Children with low functioning autism can become children with Asperger's syndrome as treatment progresses since they start seeing people as more than surface (Klauber & Rhode 2004b, p.264). There is no mention in Tustin of encapsulators becoming confusional children but shell-type encapsulators can become segmented-type encapsulators as treatment progresses (Tustin 1981, p.31). If Klauber & Rhode describe the same process as Tustin here then segmented-type encapsulators do develop a theory of mind and so would fit the description of Asperger's syndrome under Klauber & Rhode's scheme.

Interpreting Tustin's classification to fit modern autism faces a number of difficulties. By modern standards some encapsulators were low functioning (because they were associated with autism which historically would more likely have meant low functioning, because some lacked a theory of mind, because some presumably had more severe symptoms), some were high functioning (because some may have had a theory of mind, because encapsulators may have differed in level of severity) and some were not on the autistic spectrum (because some spontaneously recovered and some became adult schizophrenic). Similarly, by modern standards some confusional children were low functioning (because the case studies look low functioning), some were high functioning (because some may have had a theory of mind, because they had late onset and so were likely Asperger's syndrome with a misinterpreted late onset, because they were presumably less severe) and some were not on the autistic spectrum (because they became adult schizophrenic and because many of the symptoms were different). All this suggests Tustin's analysis does not vastly reflect modern autism. As I explain below, this results from Tustin employing psychoanalytical theory to demarcate classifications.

3.5.4 Rhode & Klauber

Rhode & Klauber present two subdivisions of Asperger's syndrome. The first subdivision has “major problems relating to other people – which included theory of

mind capabilities – and in dealing with their own aggressive impulses, but they relied predominantly on controlling others by means of "bossy" behaviour and on retreating to their own obsessive "interests" (Klauber & Rhode 2004b, p.262). The second subdivision is "characterised by florid, confusing, and seemingly bizarre behaviour and utterances and often by reliance on physical action. They frequently seemed to be unable to distinguish between their own fantasies and external reality, or between themselves and other people, to whom they attributed many of their own feelings" (Klauber & Rhode 2004b, p.262). These children have all of the other symptoms of Asperger's Syndrome.[22] No evidence is offered for these subdivisions beyond the children in case studies supposedly falling into one category or the other (Klauber & Rhode 2004b, p.262) though some children mix both types (Klauber & Rhode 2004b, p.262). Both subdivisions seem compatible with modern autism, merely adding a few symptoms.

These two subdivisions appear to be based on abnormal theory of mind. The first group struggle to relate to others due to theory of mind difficulties and tries to control others or avoid others by retreating into obsessive interests. Whilst not stated, this is presumably because their theory of mind difficulties means they struggle to appreciate that other people are not objects. Though not explicitly described in theory of mind terms, the second group had problems which appear similar to theory of mind difficulties whereby they sometimes failed to understand other people did not have a separate theory of mind to themselves and consequently did not fully see the distinction between themselves and other people. Klauber & Rhode present two variations of higher functioning individuals, each with a different type of abnormally functioning theory of mind (though Klauber & Rhode's notions are slightly broader than theory of mind, covering other types of abnormal thinking which I will discuss later). Modern psychologists disagree about the degree to which both high and low functioning autistic children lack a theory of mind. Some psychologists argue higher functioning individuals who lack a theory of mind pass theory of mind tests through some other means than theory of mind, so called hacking out the answer through experience and intellect rather than by using theory of mind (Frith 1994, p.119; Happé

22 All children "recognizably fit Asperger's original description and the present-day criteria of ICD-10 and DSM-IV" which are Social isolation, "oddness", special interests verging on obsession, the eccentric and often pedantic use of language, physical clumsiness, and unusual sensory experiences" (Rhode 2004a, p.2).

1995, p.853). Others disagree, believing partial failure of theory of mind is possible (see Kaland et al for a study highlighting how higher functioning autistic people do not simply have stronger theory of mind but have strengths and weakness in various different aspects of theory of mind (2008)). Klauber's & Rhode's account of Asperger's syndrome fits the later approach, allowing for partial failure of theory of mind and subdividing two groups of higher functioning individuals based upon different areas of failure. Klauber's & Rhode's account is therefore compatible with some interpretations of modern autism.

3.6 Similarities and Differences of symptoms

3.6.1 The alternative symptoms of psychoanalysis

Having outlined the history, let's compare psychoanalytical accounts of autism with modern cognitive psychological accounts. Many descriptions of autistic people made by psychoanalysts were different to modern accounts but the epistemological consequences of this vary considerably.

Sometimes psychoanalysts saw symptoms never associated with modern autism, other times psychoanalysts noted symptoms today associated with modern autism but dismissed the symptoms as illusions and sometimes psychoanalysts observed symptoms today associated with autism but gave those symptoms a different interpretation to the modern interpretation. Examples of psychoanalysts seeing different symptoms or seeing symptoms but discounting them as false suggests psychoanalytical theory influences what symptoms are seen. Such instances are epistemologically worrying, suggesting epistemically insecure theories influence their observations. Examples of observing the same symptom but giving a different causal interpretation suggest psychoanalytical theory played little role in which symptoms are seen. These need not epistemologically worry us since we argue for belief in symptoms and classifications but not causes. I show below most symptoms which psychoanalysts reported as looking superficially different to modern autism are symptoms being given a different causal interpretation rather than being additional symptoms or discounted symptoms.

One of the difficulties with Bettelheim (1967) and Tustin (1981) is that they also incorporate schizophrenic children into their books on autism. Childhood schizophrenia was largely composed of children similar to modern autism but did include children with substantially different symptoms, ones who would today be considered to have childhood-disintegrative disorder and early adult onset schizophrenia. This means we cannot expect Bettelheim (1967) or Tustin (1981) to fully conform to the DSM notion of autism and therefore some differences reported can be discounted on these grounds.

3.6.2 Symptoms not associated with modern autism

All three psychoanalytical text studies stated some children they studied hallucinated, a claim very contrary to modern autism. However, very little evidence is given to support this claim. Bettelheim on occasions mentions hallucinations but rarely provides any evidence. The most detailed mention - the only one with evidence offered - states hallucinations were evident from "the way she then looked up into space, preferably up to the ceiling, entirely preoccupied with something going on in her mind, and totally oblivious to what was going on around her" (Bettelheim 1967, p.116). More broadly, he mentions "vague hallucinations and daydreams of more or less limited and stereotyped content" (Bettelheim 1967, p.206) but does not state what type of hallucinations they were or what evidence there was for them. Also, Bettelheim's psychoanalytical descriptions are extremely unclear if the children actually make direct statements about how they perceive their surroundings or if Bettelheim is just inferring these perceptions from the child's behaviour. If the latter then the same may be true about hallucination, Bettelheim inferring hallucinations from external behaviour rather than verbal reports. However, it is certainly possible that some of Bettelheim's children did hallucinate since a small minority of schizophrenic children did hallucinate. Tustin only makes one mention of hallucinations. When summarising confusional children she states confusional children have hallucinations but encapsulators do not, beyond this she makes no mention of hallucination let alone offers evidence for them or examples of them. One paper in the 2004 volume on autism and psychoanalysis states some children show

"psychotic phenomena, particularly hallucinations both positive and negative" (Simpson 2004, p.34). No evidence is offered for this beyond citing a paper published in 1955. Although all three texts mention hallucinations, mentions are rare, none provide any discussion of hallucinations and none provide any evidence for hallucinations or case studies covering hallucinations except Bettelheim's single example. Remember also that a very small number of schizophrenic children did hallucinate. This suggests hallucinations are not excessively theory-dependent on psychoanalysis.

All three psychoanalytical texts thought these children had a distorted understanding of the world and many bizarre beliefs. Abnormal thinking and understanding to this degree, what I call thought disorders, are also absent in modern autism. Bettelheim describes Laurie, a mute and near inactive autistic girl. Bettelheim thought she lacked an ego, describing "her central symptomatology: her inability to take in (and hence give out) or to relate to the world" (Bettelheim 1967, p.141) because she did not recognise the external world. Through psychotherapy her ego started to grow, recognised the external world and thus she abandoned playing with her faeces, no longer conceiving them as part of herself. He also describes Marcia who had obsession with the weather. Bettelheim thought this was because she feared her mother would eat her, the weather symbolising the phrase we/eat/her. Bettelheim also describes Joey who believed he was a machine that had to be constantly plugged in with imaginary wires to remain alive. All three texts make general statements about thought disorders not associated with modern autism. For example, Bettelheim claims that autistic children "fear constantly for their lives, they seem convinced that death is immanent..." (Bettelheim 1967, p.63). Tustin claims – "[c]hildren in autistic states invariably feel that cupboards and drawers are stomachs" (Tustin 1981, p.217). Rhode & Klauber claim a common theme described is "extreme infantile helplessness… strikingly represented in the image of a baby – or baby like animal - with arms hanging loosely, unable to grasp and hold on. A related image concerns that of a dead, brain-damaged, misshapen, or ugly (Meltzer, 1988) baby or foetus" (2004b, p.265). Psychoanalysts emphasised thought disorder in a way which has similarities to that employed from the 1940s to the late 1970s but which is absent in modern autism. These are certainly likely to be theory-laden though below I suggest modern psychologists perhaps do not observe this because cognitive psychology directs

observations away from them.

3.6.3 Symptoms observed but discounted

There were occasional cases where Bettelheim made an observation associated with modern autism but used psychoanalytical theory to discount the observation. Bettelheim disagrees with Kanner's claims that autistic children cannot relate to people but can relate to objects. Bettelheim disagrees, saying if autistic children could relate to objects then this would "constitute a self-chosen positive attachment and this would soon permit the child to escape his exclusive relatedness to one set of negative principles" (Bettelheim 1967, p.91). Bettelheim here claims the child only relates to the world negatively, the child interpreting the world as hostile. However, if the child could relate to objects then the child would be able to accept the world does have positive elements and stop rejecting the world. Since this has not happened, they clearly cannot relate to objects. Bettelheim seems to discount the evidence because he realises his psychoanalytical theory is contradicted by the evidence, resolving the conflict between theory and evidence by favouring the theory and discounting the evidence.

Bettelheim appears to discount instances of autistic children associating specific actions to specific situation. Some autistic children associate specific skills or words only with specific situation and struggle greatly to generalise those skills and words. Bettelheim cites Kanner who describes a child who associated the word 'yes' with being asked if he wished to be put on his father's shoulders and it took many years to use the word yes more generally. Bettelheim thinks it evidence of how the child reacts negatively to the world, unwilling to positively engage by not using the word yes to requests from others (Bettelheim 1967, p.426). Having rejected the external world, requests from others are always answered with a no and rarely a yes so the child does not have to do anything. Here Bettelheim accepts some autistic children use skills only in specific situation but uses psychoanalytic theory to interpret this as only occurring in relation to rejecting requests from others to engage in the world by refusing to use the word yes.

3.6.4 Alternative interpretations of symptoms

There are many cases where psychoanalysts discussed symptoms today associated with autism but gave different causal interpretations not employed within mainstream modern autism. These examples show instances where psychoanalytic theory provides an alternative causal explanation but does not influence what symptoms are seen.

Bettelheim spends many pages arguing that Kanner and other authors do not understand pronominal reversal, the phenomena when autistic people replace the word 'I' with 'you'. Bettelheim argues autistic children do not misunderstand the word I but avoid using it because of their anxiety about themselves and their unwillingness to commit to any course of action (Bettelheim 1967, p.427). Bettelheim claims autistic children like learning lists because it means they do not have to make a personal connection with other people (Bettelheim 1967, p.429). Bettelheim thinks that autistic children's dislike of unexpected changes reduces anxiety caused by their supposed inability to affect the external world (Bettelheim 1967, p.68). Rituals of autistic children are interpreted to serve the same purpose, ensuring certain outcomes occur to prevent anxiety stemming from unexpected outcomes (Bettelheim 1967, p.54), whereas some modern psychologists think repetitive behaviour stems from both desire to prevent anxiety alongside deeper psychological or biological causes.

Tustin thinks both hypersensitivity of some autistic children and muted inaction of other autistic children are explained by their failure to adequately symbolise the world. This causes an uncontrollable sea of waves of sensation causing "keyed-up state of hypersensitive awareness or in a muted state of inaction in order to fend off these threats to their bodily survival" (Tustin 1981, p.142). She describes non-social play such as autistic children playing with toy trains by continually spinning wheels or treating it merely as a rectangle, failing to recognize the toy symbolizes another object. Such items are autistic objects, Tustin writing, "[t]he main purpose of Autistic Objects (that is, objects used as part of the body to give reassuring and diverting sensations) is to shut out menaces which threaten bodily attack and ultimate annihilation. Hardness helps the soft and vulnerable child to feel safe in a world which seems fraught with unspeakable dangers, and about which he feel unutterable terror" (Tustin 1981, p.100). Similarly, using language non-meaningfully is also effectively

an autistic object, employed to block communication with others (Tustin 1981, p.130). Finally, repetitive behaviour is employed to prevent catastrophic breakdowns (Tustin 1981, p.133).

Klauber & Rhode similarly gave interpretations to symptoms today associated with autism. Obsessions were taken as attempts to focus the mind on elsewhere than primitive fears supposedly causing the psychosis (Klauber 2004, p.59). Obsessively collecting information intends to keep feelings down, to prevent themselves feeling emotions (Klauber 2004, p.63). Discussing difficulties with communication, autistic people "desire contact with another person's mental processes but cannot sustain genuine communication because differences remain overwhelmingly threatening" (Klauber & Rhode 2004b, p.263). Difficulties with communication and using obsessions to block feelings are linked, the children fear "relinquishing the alleged safety of their narrow and detailed interests [to] allows access to [the] subjectively distorted and frightening contents of their mind" (Rhode 2004b, p.84-85).

The above historical analysis shows many symptoms of autism described by cognitive psychologists and psychoanalysts were broadly similar. Although both saw some symptoms which the other did not these cases were quite rare. Plausibly, these rare symptoms may have been dependent upon a single theory, therefore have high epistemic risk if that theory has high epistemic risk. By contrast, most symptoms were described by both theories so are not dependent upon a single theory. Therefore, there is no specific reason to believe these would be at risk if theories changed. For such symptoms there is no evidence Bogen's (2013) notion of perceptual loading is applicable. Bogen's notion of semantic theory loading is partly applicable to symptoms shared by both theories. The symptom descriptions are generally the same under both theories, as shown in my discussion. However, symptoms had different causal accounts, psychoanalysis and cognitive psychology having very different causal approaches. For descriptions of symptoms semantic theory loading generally is not worrying; for causal explanations of symptoms semantic theory loading is deeply worrying. Finally, notions of salience appeared applicable to both symptoms and explanations. Cognitive psychologists and psychoanalysts clearly focused on different aspects of their patients depending upon theories used. Cognitive psychology focuses on cognition and perception; psychoanalysis focuses on emotions and thoughts. Cognitive psychologists see symptoms of autism but focus on cognition and

perception whilst ignoring or idealising specific aspects of emotion and thoughts. Psychoanalysts focus on emotions and thoughts over cognition and perception. Although they focus on some specific symptoms at the cost of other symptoms, this did not prevent those psychiatrists seeing many symptoms shared between both theories. There are epistemological concerns over theory laden nature of evidence for explanations of autism and symptoms only observed by one theory but not specific epistemological concerns from theory laden nature of evidence for symptoms present in both theories.We have no reason to believe most symptoms are dependent upon either theory. Consequently, if either theory is false this would not mean those symptoms are also false.

3.6.5 Classifications

Let's now consider if the psychiatric classification of autism is theory-laden. Here we compare psychoanalytical accounts with cognitive psychological accounts, establishing if a similar classification occurs under either theory. We shall consider Bettelheim and then Rhode & Klauber first, and then discuss Tustin separately because she raises different challenges.

Bettelheim is broadly compatible with modern autism except for a few of the symptoms he lists, mentioning many symptoms associated with modern autism and appearing to effectively endorse something analogous to high and low functioning autism. Rather, he demarcates on severity, considering autistic individuals as severe and schizophrenic children as less severe. This has obvious similarity with typical demarcations made by cognitive psychologists, namely a spectrum where individuals vary on severity.

Klauber & Rhode look largely compatible with modern autism. They identify themselves with modern autism, writing, "[a]ll of these children and young people [in the case studies in the book] recognizably fit Asperger's original description and the present-day criteria of ICD-10 and DSM-IV" (2004b, p.261). They contribute two additional classifications, describing two variations of how a child with Asperger's syndrome struggles with theory of mind. This only contradicts those accounts of autism that see children with Asperger's syndrome as lacking a theory of mind.

Before discussing Tustin, let's consider what my study epistemologically entails. Later chapters will argue modern autism merits belief because it exhibits the theoretical virtue of unification. Doubt would be cast on this if autism were theory-laden by bad theories. Autism might be falsely inferred as exhibiting unifications when actually it does not. I have sought to show this is not generally the case. Undeniably, neither Bettelheim's nor Rhode & Klauber's classification are identical with classifications employed in modern autism. Those psychoanalysts included symptoms not present in modern autism and it is unclear their demarcation between high and low functioning is identical to that employed in modern autism. Though their classifications are not identical to modern autism, they still involve unifications which are quite similar to those of modern autism. Bettelheim and Rhode & Klauber unifying together a large number of seemingly unrelated symptoms, including notions of higher and lower functioning, thus are also compatible with autism exhibiting the theoretical virtue unification. Though the classifications are not identical, the theoretical virtue of unification occurred under both cognitive psychology and psychoanalysis, suggesting inferring the unification is not dependent upon either theory. Belief in autism, offered in chapter six, will require our attribution of the theoretical virtue unification to autism to be reliable. If the unification was dependent upon either psychoanalysis or cognitive psychology then (without first defending those theories) its reliability would be undermined. Being present in both theories means the unification seems not to be theory-laden by either theory. If either theory turns out mistaken we can still believe in the unification.

Tustin is much more problematic. Tustin could potentially fit modern autism but this depends on what interpretation of Tustin is taken. She identifies encapsulators as equivalent to autism and generally encapsulators do resemble low functioning autism. However, confusional children can, with difficulty, be interpreted as resembling high functioning autism but also as being low functioning whilst some seem unrelated to autism. So the clinical pictures are far identical to modern autism. More importantly, the classification takes a very different form. She covers a vast array of clinical pictures and demarcates them on grounds other than functioning. Her approach only weakly unifies, covering far too many symptoms, thus highly lacking in stringency. This is despite employing many different subclassifications. Tustin loses simplicity by employing so many classifications but without gaining in stringency.

Her classificatory system would have had some level of unification if her subtypes provided very specific clinical pictures but each subtype is less stringent than, for example, the subtypes of childhood schizophrenia which Kanner employed. More classifications and worse stringency means Tustin does not unify the symptoms well. There is a danger psychoanalysis influenced Tustin's classification, the background theory influencing the bad classification she produced. We cannot rule out the possibility that a classification, one covering individuals that today we would identify as autistic (alongside many others), was negatively effected by theory-laden evidence.

This need not worry us if given the earlier demarcation between firstly, consciously using a theory to infer classifications, second, unconsciously doing so, thirdly, holding a theory but not using it when inferring classifications or, fourthly, the theory having very little effect. Of all texts employed, Tustin is the only text which specifically formulated its classification on psychoanalysis or cognitive psychology, whereas the other text studied involved psychoanalysts or cognitive psychologists constructing classifications without specifically formulating classifications on their theory. She consciously employed psychoanalysis when formulating her classification. In contrast, Bettelheim and Rhode & Klauber did not consciously or unconsciously use psychoanalysis when formulating their classifications (or if they did the effects were very minimal). Tustin's situation is only problematic because she chooses to employ psychoanalysis when inferring symptoms. She did not have to make this choice. Without first establishing their epistemological reliability I strongly advise against employing either psychoanalysis or cognitive psychology for inferring classifications. They are only epistemologically concerning when we actively choose to employ them for inferring classifications, an option we need not take.

3.6 Conclusion

Theory-laden nature of evidence, when involving bad theories, could undermine belief in both symptoms and classifications. Without actually showing psychoanalysis or cognitive psychology are bad theories, in the absence of showing they merit belief they must be considered epistemologically insecure, therefore potentially rendering any symptoms and classifications dependent upon them as epistemologically insecure.

My historical study largely suggests most symptoms of modern autism and the classification are not reliant upon such theories.

With a few exceptions, most symptoms reported by psychoanalysts did not conflict with modern notions of autism. Hallucinations were mentioned by all three psychoanalytical sources but very little evidence was presented for them. This might be psychoanalyst's willingness to make interpretations on minimal evidence whilst some schizophrenic children did have hallucinations. Abnormal thinking was also commonly observed, far in excess of modern notions of autism. Historian of autism Evans has argued psychoanalysts were correct to describe abnormal thinking, modern psychiatrist failing to observe them because of different theoretical beliefs (2013, p.18). Both hallucinations and abnormal thinking provide some evidence of psychoanalysis playing a small role in false interpretation of symptoms. There were a few cases where Bettelheim appeared to see symptoms associated with modern autism but claimed these symptoms must not be present because they contradicted his psychoanalytical theory but even here theory was not dictating what symptoms were observed, merely instead which observations he discounted. Psychoanalysts and cognitive psychologists disagree upon causal explanation of symptoms but still observe the same symptoms. In these cases theory did not determine what was observed. Overall, the symptoms observed are similar enough between autism as perceived by psychoanalysts and by cognitive psychologists to conclude most symptoms are not dependent upon either theory.

Bettelheim's and Rhode & Klauber's classifications have substantial similarities to the classification of modern autism. Though not identical, they make the same type of unification, covering many related symptoms to modern autism and having a high and low functioning autism. The unification, which I will later argue grants belief to autism, is not dependent upon psychoanalysis or cognitive psychology. The only psychologist or psychoanalysts studied with a classification very different to modern autism was Tustin. Of all psychologists and psychoanalysts described only Tustin specifically bases classification on psychoanalysis of cognitive psychology. My history suggests psychoanalysis or cognitive psychology would only be concerning if actively employed to formulate classifications. This conclusion does not concern me because I argue we should not use such theories for that role. This should concern Baron-Cohen (1997), Happe (1995) and Murphy (2006) who desire autism classified

upon causal theories. We so far have no reason to believe the theories used for interpreting symptoms and classification of autism are bad ones; chapters five and six will show they are good ones.

Plausibly many psychiatric theories are epistemologically insecure. However, only those symptoms and classifications which are interpreted by false theories are threatened. The two theories applicable to autism which most fit classic theory-laden notion of changing theories have relatively little influence on symptoms and classification of modern autism. Modern autism has a level of theoretical robustness from such theories.

4.0 Chapter 4 – A scientific law approach to psychiatric classifications

4.1 Introduction

By responding to the pessimistic meta induction, to underdetermination and theory-laden nature of evidence, we have challenged various arguments against belief. The next three chapters make the argument that autism merits belief. In this chapter I suggest a new metaphysical picture of the nature of existence in psychiatry, one which opens up new epistemic arguments which may be applicable to some psychiatric classifications. In chapter five I explore, given this new metaphysical picture, how psychiatric classifications operate, highlighting a previously unnoticed role for psychiatric classifications. Having built up a clearer picture of how psychiatric classifications could be real and how they operate, chapter six then shows the new epistemic arguments which have been opened up are applicable to autism.

Belief in scientific theories has two components: metaphysical realism (what exists) and epistemological realism (justified belief over what exists) (Psillos 2009a, p.4). Scientific realists debates usually focus on epistemological realism, establishing if we are justified to believe scientific concepts truthfully describe metaphysically real things. Epistemic anti-realists deny we are justified, accepting metaphysical realism but denying epistemic realism is justified. Scientific realism needs both metaphysical realism and epistemic realism.[23]

The introduction outlined numerous concerns which led many to disbelieve in current psychiatric classifications and call for their replacement. I argue many base disbelief on untenably restrictive metaphysical views of how things are real in psychiatry. This then restricts epistemological arguments over psychiatric

23 Belief also requires semantic realism. This is where scientific terms are taken as literally construed, intended to literally refer to the world rather than being simply predictive instruments (Psillos 2009a, p.4). This positions is roughly instrumentalism, relating to the semantic status of theories rather then epistemological questions. This is generally not held in modern philosophy of science. "Semantic realism is not contested anymore" (Psillos 2009a, p.5) with even anti-realists being semantic realists (Lauden 1984, p.105; Stanford 2006, p.193; Van Fraassen 1980, p.10). I assume science should aim to describe real things and not discuss semantic realism again.

classifications. Here I show how epistemological arguments rest on overly restrictive views of reality which unnecessarily demands high causal unity for psychiatric classification to merit belief. By showing how psychiatric classifications can be inductive despite lacking strong causes, we show how psychiatric classification can be informative of regularities in the world. I portray psychiatric classifications as scientific laws understood as idealised models for describing regularities (note that my account of laws is not one of exceptionless regularities true in all possible worlds). My alternative picture of how psychiatric classifications account for reality leaves many current epistemological arguments as unrequired; popular accounts which base belief on establishing high causal unity are still potential means to belief but, contrary to common arguments, my new picture shows failure to establish causal unity does not then entail disbelief. My new picture opens room for new epistemological arguments which come in chapter six.

I start by outlining then criticising Cooper's metaphysical realism. Then I show how Murphy's metaphysical realism is broadly correct but his epistemic realism presupposes a Cooper style metaphysical realism. Both Cooper's and Murphy's epistemic arguments mistakenly disallow causal instability. Consequently, I show how causal instability occurs in science more generally, showing how scientists handle it by outlining Bogen and Woodward's notion of phenomena. Once we see symptoms as phenomena we can now understand exactly what it means for symptoms to cluster. I show that clusters of symptoms potentially allow psychiatric classifications to be causes, explanations or scientific laws. The metaphysics I adopt allows psychiatric classifications which are laws to merit belief. Laws do not require causes so we can have psychiatric classification which merit belief in the absence of the causes Cooper and Murphy consider as required for scientific realism.

4.2 Psychiatry, natural kinds and the causal structure of the world

Many accounts of psychiatric classifications involve talk of natural kinds (Beebee & Sabbarton-Leary 2010, p.23; Cooper 2005, p.55; Haslam 2014, p.18; Kendler, Zachar & Craver 2011, p.1146; Meehl 2001, p.509; Murphy 2014b, p.111).[24] Also, a natural

24 Murphy's 2014b article appears to provide a different metaphysical account to his 2006 book, here I only refer to his 2014b article.

kind approach is adopted implicitly by DSM (Cooper 2007, p.46), psychiatrists (Horwitz 2002, p.5) and the general public (Haslam 2000, p.1043). Boyd's account of natural kinds is among the most influential accounts. Boyd believes "successful induction and explanation always require that we accommodate our categories [natural kinds] to the causal structure of the world" (Boyd 1991, p.139). Some demand natural kinds have essential properties, such as fundamental particles (Ellis 1998, p.32). Most believe natural kinds need similar, not identical properties (Boyd 1991, p.142; Dupré 1993, p.5; Psillos 1999, p.289). Classic examples of these natural kinds are biological species, collections of clustering properties classified according to our interests (Dupré 1993 p.51) or by nature (Boyd 1991, p.14). Notions of natural kinds fit into a scientific realist vision since properties are produced by the causal structure of the world. From this general principle we need both understand how natural kinds manifest in psychiatry, which I now discuss. Also we need a deeper understanding of causation and explanation, which I discuss later in this chapter.

4.2.1 Biological species approach to psychiatric classifications

- 4.2.1.1 Cooper's Position

Cooper endorses natural kinds, writing that

> "if we consider individual cases of mental disorder some can be seen to be similar to each other. Furthermore some of these similarities will be theoretically important, and in some cases patients who are grouped together will be alike in fundamental ways… If we take cases of mental disorders as our domain and plot them onto a multidimensional quality space (as in cluster analysis) then we will find clusters of similar cases. If we focus on the right properties, then the clusters that such a process generates will be inductively powerful" (2012, p.62).

Properties arise from determining properties where

> "members of a natural kind all have similar determining properties, and the determining properties determine the other properties of the entities, [and this means] that we can predict that all members of a natural kind will behave similarly" (2005, p.53).

Cooper does not explicitly define determining properties but says "[t]hese important properties are important because they determine many of the other properties possessed by members of the kind" (2005, p.51). An example of an underlying property is the defective gene in Huntingdon's which produces characteristic symptoms (Cooper 2007, p.63). It appears that determining properties cause (or strongly causally influence) many other properties an entity has.

Cooper takes a biological species approach (2007, p.50), relying heavily on Dupré's biological species as natural kinds approach. Dupré believes "there is no more to the discovery of a kind than the discovery of the correlations of properties characteristic of the members of a kind" (1993, p.61). Correlations can be grouped in multiple ways based upon "goal underlying the intent to classify the object" (Dupré 1993, p.5). Evolutionary linage or a functional analysis of organisms could group animals (Dupré 1993, p.51). Cooper supposes properties exist mind-independently, there existing correlating properties we might group into schizophrenia. Just like dogs and horses exist as correlations of properties, so too would autism and schizophrenia (assuming they were natural kinds). We might subdivide dogs, similarly we might subdivide schizophrenia. We might merge dogs and wolves, similarly so we might merge schizophrenia and autism. Providing the psychiatric classifications describe real properties then the classifications merits belief. Cooper's example of evidence entailing reality of a disorder has similarities to that employed for biological species.

> "[By saying] Down syndrome is a real condition I mean the following: there are people with Down syndrome who form a kind. In important respects people with Down syndrome are similar to each other. They tend to have characteristic physical appearances, and to have intellectual disabilities. In addition there are characteristic genetic abnormalities that cause Down syndrome" (Cooper 2012, p.38).

Consider the similarities. Firstly, genes are immensely powerful for delineating biological species. Secondly, Down syndrome has clear physical characteristics, just like animals do. Thirdly, Down syndrome is associated with quite prominent behavioural characteristics, intellectual difficulties, just like how animals have prominent behavioural characteristics.

Cooper, however, suggests existing classifications likely do not describe natural kinds. Flaws with psychiatric theory and interference by various organisations (drug companies, insurance companies) weaken psychiatry epistemologically sufficiently that Cooper doubts most DSM classifications are natural kinds (Cooper 2005, p.150). Mental illnesses exist as biological species but our current classificatory system does not generally describe them. This makes Cooper a metaphysical realist, since mental illnesses exist, but generally an epistemic anti-realist, since we have not located those real mental illnesses. Few current mental disorders have genetic associations as strong as Down syndrome, few have associated physical characteristics and few have symptoms as clear cut as intellectual deficiency. Mental illnesses existing metaphysically as biological species mean evidence for epistemic realism are out there but not yet found. Consequently, we must be epistemic anti-realists.

Cooper's position is popular, many philosophers believing mental illnesses exist as natural kinds but doubt existing classifications identify natural kinds (Kendell & Jablensky 2003, p.5; Meehl 1995, p.266; Meehl 2001, p.509; Murphy 2014b, p.120), or classifications only meet much weaker criteria of natural kinds than Boyd's (Haslam 2014, p.23) or remain silent over reality of current classifications (Beebee & Sabbarton-Leary 2010).[25] I certainly accept most psychiatric classifications lack evidence entailing epistemic realism over metaphysical realism as biological species. Consequently, I shall undermine Cooper's argument by providing an alternative to her metaphysical picture of mental illnesses as biological species.

- 4.2.1.2 Problems with Cooper

I now discuss problems with approaches that seek to model psychiatric classifications

25 Although Kendell & Jablensky (2003) do not mention natural kinds, they adopt a cutting nature at its joints approach (Murphy 2014a, p.64), a classic example of psychiatric classifications as natural kind in all but name.

on biological species (though remember biological species approach works for rare instances like Down's syndrome).[26]

Psychiatric classifications typically have higher causal instability than biological organisms, typically being the product of INUS conditions (Meehl 2001, p.511; Schaffner 2008, p.75; Rodrigues & Banzato 2014, p.53). INUS stands for *insufficient* but *nonredundant* part of an *unnecessary* but *sufficient* condition for an effect. This is where numerous causes can produce a result but none guarantee it (Mackie 1974, p.63; Psillos 2002, p.87; Salmon 1998, p.22). Psillos gives the example of a house fire, whereby a fire is caused by the causal combination of a short circuit, oxygen and combustible materials. All these three causes are required to cause the house fire, since the presence of only two of these do not result in a house fire. So we cannot say short circuits always cause house fires, since we might lack flammable material. Additionally, we can have house fires without short circuits because we can instead have petrol, flame, oxygen and combustible materials. So short circuits can, in the correct situations, cause house fires, but they do not guarantee house fires and neither are they the only route to house fires. In contrast, biological organisms are complex systems where genes are required alongside other causes (Gannet 1999, p.359). We can say genes ceteris paribus cause particular traits (Gannet 1999, p.353) whereas generally most disorders lack identified genes that even ceteris paribus produces the disorder. There are exceptions like Down's syndrome (Gannett 1999, p.366) but these are rare. Some philosophers consider genes probabilistic causes rather than deterministic (Kitcher 1989, p.456) but in psychiatry generally statistical correlations between genes and classifications are low (Kendler 2005b, p.1246; Murphy 2006, p.239). Most psychiatric classifications have higher causal instability compared to biological species.

Psychiatric classifications can be more heavily influenced by environmental factors than instances of biological species. Historical and cultural factors affect psychiatric disorders, making them “messier” (Kendler 2009, p.1940) than most scientific concepts. Some mental disorders heavily depend on environments, requiring specific historical, social and cultural conditions (Kendler & Zachar 2008, p.378-381). Environments also affect animals but generally only long term.

26 Some ideas below have been briefly mentioned in Haslam's recent paper (2014, p.11).

> "Large effects from the environment are far more likely to produce a creature that is unviable than one that is very different. Genes thus may be thought of as having a homeostatic effect on traits, keeping them relatively constant from generation to generation" (Garvey 2007, p.22).

Such strong homeostatic mechanisms seem lacking for most psychiatric classifications.

Individuals often change psychiatric classifications in a manner highly unlike biological species. An individual might have depression then anxiety then OCD, the symptoms they exhibit changing (Zachar 2014, p.176). The diagnosis given to a patient can change radically, even for schizophrenia (Baca-Garcia et al 2007, p.214). In contrast, Labradors do not turn into Dobermans. Interbreeding and evolution over time can produce vagueness over which species a particular animal belongs to but this is little barrier to understanding the biological and behavioural characteristics of intermediate animals.

Psychiatric theories employed to cluster symptoms are highly contestable compared to biology. Biological species are primarily clustered by evolution, a unifying and all-encompassing theory (Garvey 2007, p.134; Kitcher 1981, p.519) and nothing similar exists in psychiatry.

People manifest psychiatric classifications often much more loosely than animals manifest species. A diagnosis of depression requires five of nine symptoms, whereas we do not think something is a dog only if it has five of nine characteristics of dogs. Consequently, we often get individuals with depression that only have five symptoms (55%) of the information we consider relevant for establishing if someone has depression, whereas we rarely find dogs that only have 55% of the characteristics relevant to identify them as dogs. People diagnosed with psychiatric classifications seem more loosely connected to the classification than most animals do to species.

Cooper's biological species approach seems considerably misplaced. She might respond this merely shows epistemological weakness rather than metaphysical differences: ceteris paribus deterministic genes exist but are not identified, or theories as powerful as evolution will eventually be developed. A stronger hypothesis is most psychiatric classifications have levels of causal disunity untypical of biological species. There is plenty of evidence for this. Certainly some mental illnesses do

resemble biological species, such as Down's syndrome, Parkinson's and Huntingdon's. In chapter six I suggest these psychiatric classifications deserve belief because of this. However, the evidence suggests such determining properties are rare, meaning it is misleading to believe many psychiatric classifications exist in the manner of undiscovered biological species. With rare exceptions, psychiatric classifications as currently formulated almost never have identified causes, rather, "our genes seem neither to have read DSM-IV nor to particularly respect the diagnostic boundaries it established" (Kendler 2010, p.1291). Neither should we believe such causes are still out there waiting to be found.

> "We have hunted for big, simple neuropathological explanations for psychiatric disorders and have not found them. We have hunted for big, simple neurochemical explanations for psychiatric disorders and have not found them. We have hunted for big, simple genetic explanations for psychiatric disorders and have not found them" (Kendler 2005a, p.434-435).

Rare instances of identifying big, simple causes are unlikely to be replicated (Kendler 2005a, p.434) because stronger causes are much easier to find (Kendler 2005b, p.1247), also because causation in psychiatry is "inherently multifactorial" (Kendler & First 2010, p.264) and because similar biological lesions can produce very different symptoms in different individuals (Kendler & First 2010, p.264). Certainly evidence suggests autism is very much multi-causal (Happé, Ronald & Plomin 2006, p.3; Kendler 2010, p.1292-1293). Consequently, we need an alternative model of metaphysical realism in psychiatry than one that thinks of psychiatric disorders as being like biological species. Before we consider this, we need consider the nature of causes in psychiatry.

4.2.2 Rejecting biological species and the search for causes

- *4.2.2.1 Murphy's Position*

Murphy has written extensively arguing for the idea that psychiatric classifications should seek to reflect underlying causes. We shall consider his position before I discuss the nature of causes.

Murphy bases his realism on identifying the causes of psychiatric classification. He believes the aim of psychiatric "classification [should be] to track genuine structure in nature" (Murphy 2006, p.224). Murphy outlines psychiatric classifications as exemplars, "an idealised representation of the symptoms and course of a mental illness, and [we should] try to uncover the idealized causal relationships that accounts for that idealised picture" (Murphy 2006, p.202). An exemplar is a prototypical account of a patient with a classification, basically the image that a psychiatrist has in their head when they hear the word 'schizophrenic' (Murphy 2006, p.202). Exemplars are idealisations, patients exhibiting varying degrees of symptoms of the exemplar and potentially no patients have ever exhibited the full exemplar (Murphy 2006, p.202). Classifications for Murphy should be explained exemplars, an idealised causal story of the exemplar (Murphy 2006, p.202).

Murphy has similarities and dissimilarities to Cooper. The similarities are that Cooper's natural kinds provide information over what things tend to do (Cooper 2007, p.45) and manifestations of the natural kind differ in degrees from one another (Cooper 2005, p.52). There are two differences. Firstly, Murphy favours finding causes behind classifications whereas Cooper's realism focuses on identifying the products of causes without requiring the causes responsible being identified. Secondly, Murphy places less emphasis on natural kinds. Some mental illnesses have sufficient causal basis to be natural kinds (Murphy 2006, p.341) but some do not yet Murphy still allows realism over these (Murphy 2006, p.341). Cooper's realism requires a level of causal unity that Murphy's realism can have but does not require. For Murphy finding causes is important, not causal unity.[27]

Murphy's focus on causes is metaphysically realist but he largely adopts epistemic anti-realism, admitting the causes he seeks have largely not been found. Adopting Murphy's approach leaves antirealism largely justified. Problems with searching for causes are well known and need only summarising. Robins and Guze hoped laboratory tests would establish causes (1970, p.107) but these failed and much more complicated models are required (Andreasen 1995, p.161). Even an optimistic

27 This is the general idea of his 2006 book. His 2014b looks much closer to Cooper's position.

psychiatrist like Pies admits there are no smoking guns but only biomarkers which are only “frequently identified” (Pies 2008, p.49) and can only be described as “promising” (Pies 2008, p.50). Such optimism may be misplaced. First writes that “despite 30 years of intensive effort, the field has been unable to find a single biological or genetic marker that is specifically associated with a DSM category” (First 2012, p.13). Indeed, Bolton believes

> “there is not much prospect that the science of the etiology of psychiatric conditions will deliver a single, optimal classification scheme – the reason being that the last few decades of research has uncovered systemic complexity, rather than reductionist simplicity” (2012, p.6).

Murphy is aware of such limitations, admitting cases with identified genes like Huntingdon’s are rare (Murphy 2006, p.133). Rather, Murphy hopes psychiatry is “becoming steadily metaphysically committed as the science develops and driven by a commitment to uncovering the causal structure of mental illness” (Murphy 2006, p.204). Murphy's hopes for epistemic realism are based upon a future hypothetical psychiatry.

The future, as Murphy recognizes, will likely not be ideal either. Down's syndrome seems near perfect for Murphy's realism (and for Cooper's) because the cause is so strong, occurring in almost all people with Down's syndrome and almost no one else, allowing tests to have a 99% plus sensitivity (accurately establishing who has Down's syndrome) and specificity (accurately establishing who does not have Down's syndrome) (Hyett 2014, p.52). Ideally, we will find many such genes to reformulate psychiatric classifications on. However, as mentioned above, Kendler argues that high probability genes are much easier to find than low probability genes, therefore, we likely already have found them and all that remain are low probability genes (2005b, p.1247). So employing genes will require reformulating psychiatric classification on low probability genes. Recognizing this problem, Murphy suggests that generally “the best we can hope for is the development of explanations that trace the major symptom-types of each disorder to the pathological processes that give rise to them” (Murphy 2006, p.203). He suggests biological processes and psychological

models (Murphy 2006, p.215) as examples of causes that might be used to restructure psychiatric classification.

- *4.2.2.2 Problems with Murphy*

Murphy's metaphysical picture sits badly with his epistemic arguments. Assuming we found them, consider how these causes relate to existing classifications. They may largely match existing classifications, suggesting existing classifications are roughly correct and merit epistemic realism, leaving Murphy's pessimism largely misplaced. Of course, this possibility is only known once we find those causes. Consider the alternative, our classifications are radically mistaken, the causes found have no connection to our existing classifications and they need radically reformulating. Let's consider two problems with this second situation that undermine Murphy's optimism.

If existing classifications are fundamentally mistaken then causal investigation may be deeply flawed (Cuthbert & Insel 2013, p.3; Jablensky 1999, p.142; Poland 2014, p.46; Sullivan 2014, p.257), undermining Murphy's epistemic realism about the future. Deeply mistaken classifications might significantly constrain how scientist's conduct their search for biological causes and negatively influence what they take causes to be. Also, having found causes, false classifications may influence judgments over which causes are or are not responsible for classifications. A cause may appear to fit a psychiatric classification, however, assume that psychiatric classification does not reflect reality. If the psychiatric classification was improved, made more realistic, then the gene may no longer fit. This is especially likely for classifications produced by many causes of low probabilistic effects. Scientists might emphasize the presence of those genes in a psychiatric classification, consider their discovery a major scientific advance. Yet many of those low effect genes may also be present in many other symptom clusters (and potentially non-pathological populations). Scientists might have found some of those genes have a higher effect size within symptom clusters which better reflect the behaviour of individuals but this may go largely unnoticed because those other symptom clusters are not official DSM diagnoses and thus never tested. Where low effect genes are present in many different symptom clusters (even in non-pathological populations) our ability to establish causes and judge how they

result in psychiatric classifications might be substantially reduced compared to biological investigation of causes of animals.

Murphy could sidestep this problem by ignoring existing classifications like the RDoC attempts but this creates additional problems. Here we do not have false classifications holding back biological investigation but we are now in largely uncharted territory. The history of medicine and biology shows few instances of scientists searching for causes in complete independence of existing classifications. Biological evidence can have high levels of independence, evidence produced in support of one theoretical claim might end up being employed to support some other unrelated, non-competing theory (Leonelli 2009, p.747). So although evidence is not constrained to the theory which it was investigated for, at some point it needs relating to theory. In biology, it can be related to biological categories, which are usually based on good science. In contrast, in psychiatry it either needs relating to (supposedly) bad psychiatric classifications or relating to hypothetical psychiatric classifications which have not yet been constructed. RDoC attempts something relatively unique, requiring biological science to accomplish more when applied to psychiatric classification than is generally required for biological classification.

Additionally, whether employing existing classifications or not, linking causes in psychiatry to classifications faces more challenges than in biology. The failure of psychiatry to find causes of significant effect has led to much philosophical discussion about multi-level explanations (see the recent book Kendler & Parnas (2008) edited). Also much discussed is the importance of factoring in multiple types of evidence, such as clinical and biological, when validating psychiatric classifications (see the recent book Zachar, Stoyanov, Aragona & Jablensky (2014) edited). Poland writes that "any given pattern of behaviour and other clinically identified features will mask a wide range of distinct causal processes and a wide range of distinct features at all levels of analysis (Poland 2014, p.43). Murphy endorses this picture, different methods and different disciplines describing different levels (Murphy 2006, p.121) with interactions across levels spanning biological, psychological and environmental (Murphy 2006, p.141). Integrating these disciplines is immensely challenging, the "difficulty of which is hard to overestimate" (Kendler 2005b, p.438). Tracing such multi-level explanations, such as gene to disorder, is immensely complicated and cannot currently be done (Kendler 2005a, p.1249). For example, Sun et al (2010, p.5) found 24

possible causal pathways which can lead to schizophrenia. Multi-level explanations occur in biology but biologist can identify a cause, say a gene, and then use idealised multi-level explanations to link that gene to the end physical or behavioural characteristic. In psychiatry we rare have that initial starting point of highly probabilistically important genes, instead employing the multi-level explanation itself as the cause for demarcating classifications. This is a much more complicated situation. Rather than an animal's causal basis resting on high probability genes (the easy bit) which combines with other factors to produce an animal (the multi-level difficult bit), the causal basis of psychiatric classifications will be the difficult multiple level explanation. The more content placed in a model and the more levels of explanations involved the more complicated the model becomes (Zachar 2012, p.195) but without this content important factors may be lacking. Ceteris parabus, some genes do have near deterministic effects. By contrast, simplifying multi-level models risks ignoring important causes that cannot be simply idealised away ceteris parabus without producing something vastly more misleading than ceteris parabus explanations of biological species. Some biologists downplay the importance of genes, so called evo-devo emphasizing the importance of development (Garvey 2007, p.73) but nothing analogous currently exists in psychiatry. Also, even were multi-level explanations possible, philosophy of psychiatry has yet to attempt to describe what a multi-level explanation in psychiatry would look like (Campbell 2008, p.199). These problems are much more complicated than in biology.

Murphy hopes allying psychiatry with biology will make psychiatry more successful but the reasons why biology has been successful appear absent in psychiatry. Animals have far greater causal stability than most mental illnesses, making finding causes much easier. Also, most biological species were broadly correct prior to finding causes. The physical and behavioural characteristics associated with most species did not change radically when evolution was discovered (Dupré 1993, p153; Kitcher 1993a, p.32), also, Linnaean classifications match modern classifications quite well (Garvey 2007, p.131). This seems likely for medicine more generally, Solomon writing that “the rest of medicine has much more agreement about disease classification and rarely introduces diagnostic categories that are as complex and difficult to apply as those in the DSM” (Solomon 2015, p.70). For psychiatry we face the alternative, modern classifications as fundamentally mistaken. Plausibly,

causal investigation was successful in biology because categorization of animals was broadly already correct and high probability causes exist. Unlike biology, psychiatry lacks stable and broadly correct classifications as starting point for biological investigation. Also, biological causes typically have much lower effect in psychiatry than in biology. Differences between both sciences weaken the inference that it will be successful in psychiatry, undermining Murphy's optimism.

Murphy's metaphysical picture improves on Cooper's since Murphy allows realism over causally disunified things. Cooper's realism over mental illnesses as biological species rests on determining properties but causal properties in psychiatry are far less determining. This leaves mental disorders as insufficiently causally unified to be adequately modeled as biological species. However, Murphy's epistemological argument for realism seemingly presupposes many parallels with biological species. His epistemological optimism seems based on a metaphysical realism he rightly rejects, his epistemological optimism requiring stable causes which his metaphysics does not require. I will supply new epistemological arguments in chapter six but doing so will be based upon supplying a new metaphysical picture, which I now do.

4.3 Psychiatry, causal disunity and laws

4.3.1 Physical Systems and phenomena

Cooper links inductive power with determining properties whilst Murphy links success with causal mechanisms. I will show how psychiatric classifications can be inductive (and therefore scientifically valuable) without resting on determining properties or causal mechanisms. Some accounts of induction believe induction requires a presupposition of uniformity (Howson 2000 p.182; Macnamara 1991, p.30). Autism would not count as uniform given its underlying causal disunity. However, accounts of induction which require presupposition of uniformity bare little relationship to science (or indeed inductions outside of science) (Lipton 2004, p.12).

Science generally seeks to describe physical systems. A physical system is where many different causes interact to produce a typically non-uniform process. Examples of physical systems are severe storms, gas jets and turbulent flow of water

in a basin (Winsberg 2001, p.443).[28] Similar to the causal instability I have described, the

> "behaviour of most real physical systems is the result of the interaction of a very large number of distinct causal influences, which may not recur together in a regular or uniform fashion at all… the behaviour of some actual object in an electromagnetic field will reflect not just the electromagnetic force… but will also reflect the gravitational force incident on the particle, the effect of air resistance, and so forth… There will be no single law describing the net effect of all these factors on the objects" (Woodward 1992, p.194; also Giere 1999, p.24).

Physical systems usually have high levels of instability, changing from one moment to the next. Despite this, inductive and causal claims are still possible. We should consider an autistic person in an environment as a physical system, disunified causes internal to the person plus unstable causes from the environment interacting to create a non-uniform process called autism.

Scientists cope with unstable causes by isolating different causes and studying them independently. This typically occurs in laboratory settings, scientists creating situations where causes are controlled. For example, scientists might create a vacuum to analyse the effect of gravity in the absence of air resistance. This would ideally allow scientists to understand how each cause operates individually, allowing scientists to combine all those causes together to understand the causes affecting the phenomena. Unfortunately, this process has severe limitations for three reasons.

Firstly, studying different causes in complete isolation is generally impossible, there are almost always multiple causes influencing the experiment. Cartwright writes that

> "however small we choose the masses in tests of Coulomb's law, we never totally eliminate the gravitational interaction between them; in Galilean experiments on inertia, the plane is never perfectly smooth nor the air resistance equal to zero; we may send our experiments

28 Note that a physical system does not simply mean physics. A society could be seen as a physical system, just one harder to measure than turbulent water.

> deep into space, but the effect of the large massive bodies in the universe can never be entirely eliminated; and we can perform them at cryogenic temperatures, but the conditions will never, in fact, reach the ideal" (Cartwright 1999, p.84).

Secondly, there are too many causal factors present at any moment for scientists to account for them all. Trying to factor into account all causes is impossible, idealisations are required "to avoid computational intractabilities" (Bogen & Woodward 1988, p.324). Neither scientists nor their computers can make such calculations. Scientists need to limit the number of causal factors they attempt to accommodate. Thirdly, scientists need a level of generality to apply theories to multiple instances. Scientists often "rely on idealizations, approximations, and simplifications of various kinds... in order to secure generality" (Bogen & Woodward 1988, p.324). Scientists might establish the best possible causal description of a physical system on a specific day in a specific situation yet find that specific causal description near useless when applied to other seemingly similar physical systems – say a given leaf on a given day compared to another leaf on another day. Rather, scientists produce general theories accounting for the most common and most important causes. These are the means for understanding physical systems so, I claim, they are the means for understanding an individual with autism in a particular environment. Showing autism contexualised to specific instances helps understand what autism decontextualised from specific instances is, so lets consider Bogen & Woodward's account in greater detail.

4.3.2 Symptoms as phenomena

Bogen & Woodward famously demarcated between data and phenomena. Historically, philosophers of science believed data proves theories. Bogen & Woodward showed an important intermediate step. Data is localised to specific conditions, say a particular experiment, whereas phenomena is more generalisable, yielded by many different procedures such as different types of experiment (Bogen & Woodward 1988, p.317). "Data typically result from complex, loosely connected, short-lived assemblies of

causal factors... The causes that produce data sets are never exactly the same from one trial to another" (Bogen 2010, p.789). Specific experimental set-ups produce specific results due to different set-ups involving some different causes. Many causes are not relevant to the phenomena, since "in typical cases data are the result of many causal factors and at most [only] some of these will have to do with the phenomena of interest" (Woodward 2010, p.167). For example,

> "the outcome of any given application of a thermometer to a lead sample depends not only on the melting point of lead, but also on its purity, on the workings of the thermometer, on the way it applied and read, on interactions between the initial temperature of the thermometer and that of the sample, and a variety of other background conditions" (Bogen & Woodward 1988, p.309).

Thus data is yielded by experiments produced by specific causal factors but for scientific purposes we require a more generalizable notion. One measurement of melting lead might produce 326.7 degrees, another 327.2 degrees (the data) but the figure we arrive at (the phenomena) is 327 degrees. We might use statistics to reach 327 degrees or we might derive it from theories about molecules (I discuss phenomena formulation in chapter five). I now show how phenomena is used in science, how symptoms are phenomena and how this alleviates many concerns of Cooper and Murphy over symptoms.

Phenomena are idealised models, abstracting away many causes (Bogen 2010, p.781; Massimi 2008, p.13; Psillos 2009b, p.87; Teller 2010, p.820; Woodward 2011, p.170). Not fully accurately representing reality means they are models. Psillos writes that

> "phenomena (e.g., the melting point of a substance or the path of a planet) are abstracted from the data by means of a number of sophisticated modeling techniques based on a rather substantive set of assumptions and theories" (Psillos 2009, p.87)

and "nothing worldly satisfies the description associated with a model" (Psillos 2011a,

p.5), providing the example of the linear harmonic oscillator. Idealised models involve

> "abstraction [where] some features of the system under study are neglected/omitted... [and/or] idealisation, [in which] there is misrepresentation – the model attributes properties to the system it does not have and/or denies that the system has properties that it in fact possesses" (Psillos 2011a, p.7).

Note that phenomena as idealisation is fully compatible with various types of realist approaches (Bogen & Woodward 1988, p.337; Giere 2004, p.750; Massimi 2012, p.49; Psillos 2011a, p.4).

The distinction between data and phenomena fits psychiatry. Symptoms (phenomena) are idealized from tendencies to exhibit certain behaviour whereas behaviour (data) manifests in particular contexts, responding to additional specific causal factors alongside causes specific to the symptom. Take, for example, an individual who has the symptom of having low social skills. The symptom has biological and/or psychological causes but manifestations of low social skills – specific words spoken, degree and nature of social misjudgment – are also causally influenced by many additional factors. For example, who an individual is speaking to, for what purpose, the mood of the individual, even gender, age, time of day or time of year. In contrast, the symptom low social skills have generality. An individual has the symptom low social skills whether at work, at school and even when speaking to those rare others whom they exhibit normal levels of social skills around. Symptoms are conceptualised in a broad, generalisable manner, low social skills covering an immense variety of behaviour. There are many means of measuring low social skills, numerous questionnaires and experimental methods. Qualifying as having low social skills requires scoring below a particular figure rather than needing one specific magnitude. Psychological tests and statistical analysis reduce incidental factors, most generalised symptoms are measurable by literally hundreds of context specific experiments and psychiatrists have some competency at abstracting away incidental factors when assessing patients.

Phenomena, rather than data, are the basis of inductions.

> "What we call phenomena include stable regularities produced and sustained by a relatively small number of causal factors that are present and that interact in pretty much the same way within a range of different background conditions at different places and times" (Bogen 2010, p.779).

Similarly, phenomena have "stable, repeatable characteristics" (Woodward 2010, p.794). Also, phenomena are used in predictions (Bogen & Woodward 1988, p.326; see also Glymour 2000, p.30; Massimi 2014, p.421; Schindler 2007, p.162). Stable, repeatable and useful for predictions means phenomena can be used for inductions. Inductions require neither a certain property nor a certain cause to always be present; at most inductions need degrees of uniformity.

Scientists have choices over how to formulate phenomena (Feest 2011, p.68; Harris 2003, p.1516; Massimi 2008, p.34; McAllister 2007, p.886; Woodward 2011, p.174).[29] For the phenomena of how often a coin lands on each side, "a researcher who employs a significant test with a significance level of 0.05 has a different attitude... than a researcher who employs a significance level of 0.001) (Woodward 2011, p.176). One level of precision results in one phenomenon, another level in another phenomena (Woodward 2011, p.174). Here goals influence what data we obtain, influencing the phenomena we establish. Also, the same data can yield different but consistent phenomena (Brading 2010, p.838; Woodward 2011, p.175). We might interpret the same data differently depending on goals and theories. Finally there can be multiple levels of phenomena, one phenomena which has many subphenomena (Falkenburg 2011, p.161; Teller 2010, p.818). These may or may not cause the phenomena. Let's consider all this. Different goals may result in different data being produced. Different goals may result in the same data being interpreted differently. Different goals influence employing a particular phenomena or sub-phenomena. This means that the inductive regularities we find depend on our goals. Woodward writes, "inductive inference... relies on more or less explicit assumptions about epistemic *goals* or ends, including attitudes towards risk" (Woodward 2011, p.172). Similarly,

29 In contrast, Bogen considers phenomena as interest independent, though he considers data as interest dependent and from data phenomena are built (2010, p.781). He might mean that given a particular set of data then certain phenomena will entail, i.e. data is interest relative but the subsequent phenomena is not. This would leave him compatible with my account. Otherwise, I cannot make sense of his claim.

we can conceptualise regularities as coarse or fine-grained (Teller 2010, p.821), Teller writing that "[n]ature constrains what options we have, but from among nature's options the regularities that emerge are guided by our choices" (Teller 2004, p.739). Relatedly, projectability (inductiveness) may depend on level of detail (Batterman 2002, p.37) or level of noise (McAllister 2011, p.80). Employing phenomena inductively allows us multiple ways to formulate inductions.

We understand physical systems and phenomena through building models. The nature of the model will partially depend on our goals, on tractability and on theories. Often simpler, less accurate theories are preferred (Chakravartty, 2007 p.233). Models are constrained by "what could be modelled manageably and reliably" (MacLeod & Nersessian 2013, p.545). Various mathematical techniques are used to suppress complexity (Macleod & Nersessian 2013, p.539; Wilson 2010, p.495). More detail often means less tractability (Batterman 2002, p.22), reduces generality (Rohwer & Rice 2013, p.336) and applicability to future situations (Myrvold & Harper 2002, p.137). Such ideas seem additional developments of Bogen & Woodward's claim that idealisations are required "to avoid computational intractabilities" (1988, p.324). Modeling all causes of physical systems or phenomena in a physical system is generally impossible.

Phenomena show we can have inductions despite causal instability. The stability of inductions is not based upon the presence of one single cause or determining property but from the idealisation, conceptualising phenomena in a manner which is tractable rather than conceptualising phenomena to track every possible cause. Symptoms, then, do not need determining properties or Murphy style causal mechanisms to be inductive.

Since classification is composed of symptoms we have some indication of how classification contributes inductively. This is important because inductions then allow laws. This will be fully outlined after the next section.

4.4 What are classifications?

We have seen Cooper and Murphy argue psychiatric classifications need have determining properties or underlying causal mechanisms to merit belief. In the

absence of determining properties or causal mechanisms which explain the psychiatric classification surely all psychiatric classifications do is probabilistically correlate co-occurrences of symptoms? This situation would seemingly leave the sceptic correct. Remember how Cushing, described in the introduction, considers all possible things autism could be identified with – cognitive psychology, neuroscience, genetics – and since autism is not equatable with any of these he concludes autism is not a "thing" (Cushing 2013, p.38) and therefore needs replacing with something better (Cushing 2013, p.41). We will see, however, that probabilistic relationships are heavily involved in causes, explanations and laws. Consequently, psychiatric classifications can be closely related to causes, explanations and laws. From here, we can see that Cooper and Murphy have been unfairly downplaying the role of psychiatric classifications; they can provide causes, explanations and laws without determining properties or deeper causal mechanisms.

4.4.1 Causes

Let's start with causes. "When an apparently unconnected event occurs in conjunction more frequently than would be expected if they were independent then assume there was a common cause" (Salmon, 1998 p.110). If low social skills and intolerance of criticism co-occur more frequently than chance then a common cause seems possible. Sufficient statistical co-occurrences lead us to believe that common causes are present. Similarly, Salmon argues explanations are statements of statistical relevancy (Salmon 1984, p.37). One cause can lead to different effects so causal explanation is not saying cause A leads to effect B but instead a statement of the relevant causal factors making something less or more likely to happen (Salmon 1998, p.354). Claiming A causes B just means B has a statistical probability of occurring after A has occurred. For example, mixing sodium and oxygen has a very high statistical probability of causing sodium oxide though not 100% given impurities and various atmospheric conditions. Recessions have a statistical probability of causing inflation, one quite low because inflation also depends on imports and quantitative easing.

Salmon argues theoretical explanations are employed to join together causal connections. We observe the product of causes and fill in gaps between those effects

by positing theoretical explanations based on a belief in the common cause (Salmon 1998, p.113). Theoretical entities are posited to fill in gaps in our knowledge rather than simply charting how the causes of an independently identified entity lead to effects (Salmon 1998, p.109). Thus we might posit genes or abnormal psychology as leading to certain effects, such as symptoms. In their absence we can posit autism, a statement about statistical probability of effects plus belief common causes are typically responsible for statistically co-occurring effects. This in no way entails a common cause is simply one thing, it does not entail autism is one uniform thing. To assign a cause is to assign statistically relevant factors, autism itself is a statistical relevant factor which itself will be produced by many other unknown statistically relevant factors (biological, psychological, environmental). Arguments over the quality of autism as an explanation will be considered in chapter six. For now, we can note that scientists often create theories that join together effects and this is also true for psychiatry. Autism is a theoretical explanation contributing to causal explanations of individual symptoms. Autism groups together many unknown causes into one big cause which we call autism. Here we invoke a different notion of causation to those typically employed. Rather than causation as discrete causes which either are present or are not, instead we think of causation as what is required to explain effects. Autism is the cause which explains the probabilistic co-occurrence of symptoms, it does not entail that autism is, or is composed of, a single discrete cause or many regularly occurring discrete causes.

4.4.2 Mechanism

A related concept to causation is mechanism. "A mechanism, nowadays, is virtually any relatively stable arrangement of entities such that, by engaging in certain interactions, a function is performed or an effect is brought about" (Psillos 2011b, p.772). "What fixes the explanatory relevant description is surely the function it [the mechanism] performs" (Psillos 2011b, p.785). "When it comes to the search for [causal] mechanisms, *anything* can count as a quasi-mechanism provided it performs a function that is meant to explain" (Psillos 2011b, p.786; see also Lipton 2004, p.33).[30]

30 Psillos does not use the phrase 'quasi-mechanism' derogatively, rather, he considers 'quasi-mechanisms' as the only type of mechanism possible, rejecting purely mechanical accounts which

Mechanisms are not A pushes B but collections of entities we group together based upon particular effects that interests us. We posit mechanisms relevant to the effect which interests us, joining together diverse entities because they produce a particular outcome. Relatedly, Bogen argues belief in phenomena only makes sense if we believe something causes it (Bogen 2011, p.16). Like Salmon, Bogen is not stringent over the causally responsible thing. Bogen notes that laws are often identified long before how they operate is established. This can provide a "reasonable belief that there is a mechanism of some kind to do the job [although this] wouldn't warrant as much confidence as well confirmed ideas about how the mechanism operates" (Bogen 2011, p.20). Autism is short hand for the unknown biological and psychological entities that mechanistically produce symptoms.

These accounts of causation and mechanism often fit psychiatry well. Psychiatry is often highly causally unstable, "specific combinations of the same antecedent causes can lead to different clinical outcomes ("multi-finality") and different antecedent can lead to the same outcome through common developmental pathways ("equi-finality")" (Cloninger 2014, p.205; also Jablensky 2012, p.87; Kendler 2005b, p.1247; Meehl 2001, p.509). Tracing causes to effects involves choices, deciding how we combine many low probability causes in a manner that produces effects. The requirement of multi-level explanations also requires choices over how we trace interactions of levels to symptoms. We cannot take a 1.2% effect size gene and see what it causally results in; rather, we start with the classification and then link it to other regularities like genes. There are instances, like Down's syndrome, where high probability causes are linked to stable sets of symptoms but this is not how things generally exist in psychiatry.

4.4.3 Explanations

Kitcher provides an account of explanations, seeing explanations as unifying diverse phenomena. For example, evolution and Newton's gravitation link together seemingly diverse phenomena (Kitcher 1981, p.519).

require causation purely "in terms of *pushing* and *pulling*" (Psillos 2011b, p.774, emphasis original).

> "connections, common patterns, in what initially appeared to be different situations... *Science advances our understanding of nature by showing us how to derive descriptions of many phenomena, using the same patterns of derivation again and again, and, in demonstrating this, it teaches us how to reduce the number of types of facts we have to accept as ultimate (or brute* [unexplained]*)*" (Kitcher 1989, p.432 emphasis original).

Theories reduce the number of unconnected facts, increasing "systemitization of our beliefs" (Kitcher 1989, p.476). Derivations need not come from a few grand equations and often only supply probabilistic explanations. For example, the "derivations that are provided by genetics show why certain distributions of genes and traits are expected" (Kitcher 1989, p.456). This is statistical derivation, certain genes increase probability of certain traits occurring. Similarly, people exhibit much seemingly unrelated behaviour but employing autism reduces the number of unexplained phenomena, "seeing connections, common patterns, in what initially appeared to be different situations" (Kitcher 1989, p.432). The presence of autism means certain symptoms are statistically probable and autism is an explanation of those symptoms. Note that explanations do not need further explanations (Lipton 2004, p.22); we do not then need an explanation of autism for autism to be an explanation.

Kitcher constrains legitimate explanations to "those derivations which collectively provide the best systemitization of our beliefs" (Kitcher 1989, p.430) by making "the best tradeoff between minimizing the number of patterns of derivation employed and maximizing the number of conclusions generated" (Kitcher 1989, p.432), emphasising building a system of economy of thought (Kitcher 1993a, p.171). Chapter six will argue autism is a plausible candidate for this unificatory goal to best systematise our knowledge, vastly reducing unsystematised phenomena. Psillos associates Kitcher with the notion of laws which Psillos adopts (2002, p.264). For Psillos, the best laws, just like the best explanations, are those which are part of the simplest and strongest system, roughly Kitcher's trade-off.

4.4.4 Laws

Traditionally, laws are often considered exceptionless regularities but many or even all laws are not exceptionless (Cartwright 1999, p.34; Giere 2004, p.745; Psillos 2002, p.145; Teller 2004, p.731; Woodward 2000, p.228). There are many different accounts of laws but since I am approaching scientific realism from the perspective of Psillos I shall employ Psillos'. This is especially important because, as discussed in chapter six, his account of laws is central to his account of inference to the best explanation which is required for scientific realism. Additionally, my discussion in chapter five of systematisation is important partly because systematisation can strengthen psychiatry inductively and thus strengthen scientific laws. Psillos takes a Humean approach whereby laws are a special type of regularity. "According to this tradition, there are only regularities, that is, sequences of event types, which happen in constant conjunction: whenever one occurs, it is invariably followed by the other... when a metal gets heated, it expands" (Psillos 2002, p.139). This would suggest all regularities are laws, however, laws are

> "sufficiently different from accidents to demand a different treatment. After all, there is a clear intuitive difference between the regularity that all apples in the fruit bowl on the table are ripe and the regularity that all metals expand when heated. Even if all laws are regularities, not all regularities are laws" (Psillos 2002, p.139).

Psillos demarcates between laws and accidents not upon some strong metaphysical grounds but upon what we desire science to do. For Psillos, the correct laws are those which are part of the best balance between strength, simplicity and stringency of our systematised knowledge. "Laws are those regularities that are members of a coherent system of regularities, in particular, a system that can be represented as a deductive axiomatic system striking a good balance between *simplicity* and *strength*" (Psillos 2002, p.149 emphasis original). Simplicity is where we employ as few laws as possible. Strength is where we explain as much as possible. The strength of an individual law has two components, strength is where law covers as much phenomena as possible and stringency is where the statistical probabilities it describes should be

stringent, as high as possible.[31] We might have a law which covered everything in the universe but was near totally incapable of describing how those things related. Equally, we might have a law which showed two phenomena were one hundred percent correlated but was only applicable to two specific instances of phenomena such as 'when I enter my house I always open a window'. Neither of these would be part of the best balance between simplicity, strength and stringency. In contrast, Newton's laws have near universal applicability whilst also being extremely stringent in how they portray probabilistic relationships (plausibly so high as to often be called deterministic). For Psillos, “accidents are those regularities that do not find a place in the simplest and strongest true deductive system that systematizes our knowledge of the world” (Psillos 2002, p.150-151). Being laws rather than just lawlike is a route towards scientific realism.

Under Psillos' Humean approach, laws determine causes (2002, p.293). He believes causes are just probabilistic relationships of regularities derived from laws (2002, p.293) and the probabilistic relationship causes describe depend upon demarcating laws (2007, p.105). Some laws are not causal, Psillos giving the example of coexistence (2002, p.171), examples of which are relationships between length and period of pendulums, pressure, volume and temperature in gases, electrical conductivity in metals relates to thermal conductivity. Grouping together characteristics into a classification are instances of coexistence (Weber 1999, p.485) and these are explanatory (Weber 1999, p.486). Whilst causes are a potential route for belief the law itself can potentially merit belief in the absence of causes. Also, lawfully describing probabilistic relationships between one set of regularities (symptoms) with another (biological, psychological and environmental regularities typically called causes) is easier when employing more laws. The more laws employed the more specific they can be. By employing more laws each law could describe a narrower range of probabilistic relationships or some laws might have very wide coverage whilst others describe a narrower range of probabilistic relationships. Describing a narrower range can strengthen the probabilistic relationships, so much so that they might be considered causal. A single law might state that after a regularity occurs then many other regularities can occur, multiple laws would allow narrowing down of probabilities. Imagine a law describes how the co-occurrence of regularity A,

31 Psillos typically just talk of strength vs simplicity, only talking of stringency when discussing what strength entails. For convenience I talk about strength, simplicity and stringency.

B, C, D and E has a 70% chance of no subsequent regularity occurring and a 10% chance each of regularity X, Y and Z occurring. Now imagine an alternative law which states the co-occurrence of A, B and C has a 80% chance of no subsequent regularity occurring, a 20% chance of regularity X occurring and a 0% chance of Y or Z occurring (i.e. Y and Z do not occur without regularity D or E). Assume we could also employ another two laws which equally allowed greater probabilities of Y occuring, and Z occurring, following a certain combination of A, B, C, D and E. By employing these three laws we can increase the specificity of the probabilistic relationships. This makes the probabilistic relationship more causal by increasing the probabilistic relationships of certain regularities occurring following other regularities occurring. More law make deriving causes easier. In psychiatric terms, linking a set of symptoms with biological, psychological and environmental regularities becomes easier, so much so that we might take those probabilistic relationships as causal. However, "simplicity and strength pull in opposite directions" (Psillos 2002, p.149) since simplicity demands fewer laws whereas more laws makes fully accounting for all phenomena easier. A system of many laws, each of which has relatively narrow coverage, is often quite easy to demarcate strong causes from but runs contrary to the simplicity constraint of laws. The best balance between strength and simplicity might be one with fewer laws and fewer (or weaker) causes, rather than more laws and more (or stronger) causes (I cover this in greater detail in chapter 6).

Though Salmon's and Kitcher's approach could fit, I endorse Psillos' account of laws. Under this, a philosophically reconstructed science takes a set of regularities, deriving laws from the statistical co-occurrences of those regularities, the law being explained by an entity with causal properties. The most sophisticated account of law in science is a model based regularity guide as advocated by Giere and Teller, one endorsed by Psillos. Laws of nature "cannot by themselves be used to make any direct claims about the world" (Giere 2010, p.270), rather,

> "[t]hey function more like recipes for constructing models than like general [universal] statements... the behaviour of many types of real world systems can in fact successfully be represented by models constructed using this recipe" (Giere 1994, p.293; also Winsberg 2001,

These highly idealised laws to which we can add more detail, can be used to create more specific models, eventually constructing models representing physical systems. Teller describes such models as “regularity guides” (Teller 2004, p.735). These heavily relate to previously discussed notions. Unifications and explanations act as regularity guides (Teller, 2004 p.740), and they relate to causes (Giere 1999, p.185), idealised models from which regularities of physical systems can be represented. The idea that regularity guides can be used to produce a hierarchy of models which can be used to guide building nested sets of regularity formulations also perfectly fits Bogen and Woodward's notion of phenomena (Teller 2010, p.825). Highly abstract theories connect to the less abstract phenomena which are connected to non-abstract instances of data. This position is fully compatible with scientific realism and Psillos cites Giere in support of his account of how models and theories relate (Psillos 2011a, p.7). For Psillos, “the truth of theories does not give them straightforward representational content vis-a-vis the physical world. Their representational content is mediated (at least partly) by abstract objects – the models” (Psillos 2011a, p.9) and he describes models as “abstract objects that can stand in representation relations to worldly systems” (Psillos 2011a, p.8). Psillos argues relationships between these layers of models can be explanationary without being causal, writing that “not all explanation is causal (e.g., the explanation of low-level law by reference to high level laws)” (Psillos 2010, p.957). Theories represent the world through the models built from those theories and there is no need for relationships between different layers to be causal.

- 4.4.4.1 Autism as a law

Regularity guide models are how we should understand autism. Recall that Cushing thinks autism need be associated with a concrete thing rather than behavioural correlates to be real (Cushing 2013, p.38) but laws are abstract models for guiding the building of more detailed models rather than concrete things. People exhibit instances of behaviour, call these data. We abstract from those something more abstract but more generalizable, call these phenomena or symptoms. The co-occurrence of the symptoms we explain or believe caused by autism, a model acting as a regularity guide, a statement about statistical probabilities. Autism systematises phenomena,

showing occurrence of certain phenomena has a probabilistic relationship with other phenomena, guiding the building of models of symptoms. We might apply those symptoms to an individual, combining autism with factors related to their individual psychology, economic status, geographic locations, etc., and also considering which specific symptoms of autism the individual exhibits, to produce more concrete models. We might apply these to a physical system, an autistic individual in a specific situation. All stages add details, sharpen the probabilities. These probabilities can be implicit judgements rather than mathematical calculations, the important point is that autism is playing a significant role in formulating such judgements. Imagine a physical system, an autistic individual in a library wishing to borrow a book already on loan. The abstract model autism predicts substantially higher probabilities of a meltdown than models of typical humans. We then establish what particular symptoms of autism that individual has, further sharpening the probabilities. We can add non-pathological traits, non-autistic symptoms, socio-economic status, geographic location, etc., further sharpening the probabilities. Autism, by itself, suggests high probabilities of meltdown and each more detailed model either increases or decreases the probabilities, assisting an informed judgement over expectations that particular individual on that particular day will have a meltdown. Autism is an abstract model that guides the building of less abstract models, each model providing rough statistical probabilities of regularities occurring. This abstract model allows us to make general claims about specific individuals. As occurs in physics,

> "[g]enerality requires abstractness: otherwise the general cannot cover the particular. There is not a theory of concrete springs, and another of concrete pendula... there is a theory of the LHO [Linear Harmonic Oscillator] which covers many concrete structures that are inexact tokens of the LHO" (Psillos 2010, p.951)

Thus autism can be explanatory and inductive without being a concrete thing or having a strong causal basis.

- 4.4.4.2 Autism as an entity

Entities for Psillos are conceptualised based upon explanatory requirements, to account for regularities via laws. “The process of positing theoretical entities... is associated with specific problems-situations, in which an entity is posited in order to stand for the cause of some phenomena” (Psillos 1999, p.294; also Kitcher 1993a, p.172; Salmon 1998, p.109). For Psillos, entities are clusters of properties demarcated upon causal relationships they enter into; which causal relationships obtain depend upon laws – which are an idealised description of regularities. Similarly, a natural kinds are “functionally relevant clusters of properties” (Massimi, 2014 p.428). Systematised phenomena are further systematized through providing theoretical explanation of phenomena by conceptualizing entities with properties. For Kitcher, the “causal structure of the world, the division of things into kinds, the objective dependencies among phenomena are all generated from our efforts at organisation” (Kitcher 1993a, p.172). Sometimes, already conceptualised entities can be appealed to when explaining regularities by laws (see Massimi 2007, p.250). A new regularity in, for example, fundamental physics could likely be accounted for by an already employed fundamental particle. If none can account for the regularity then conceptualising a new one is legitimate. Conceptualising entities with causal powers which give rise to lawful regularities is legitimate. Autism is an entity which causally explains symptoms and about which we know of through the lawful relations it gives rise to, the idealised statistical probabilities of regularities described by the classification.

4.5 Mind-independence, causes and regularities

Cooper and Murphy seemingly value causes because they are taken as mind-independent parts of reality. Cooper sees clustering properties which are grouped into natural kinds as “reflect[ing] real structures in nature” (Cooper 2005, p.49) and Murphy says the “causes of mental illness [that] are genuinely out there in the structure of the world, waiting to be discovered” (Murphy 2014a, p.62). Unfortunately, exactly what is meant by the world having a real structure or an objective structure is left unstated. Let’s compare this with Psillos' account.

Psillos sees regularities, rather than causes, as mind-independent. “Regularities are real and mind-independent: they would exist as (perhaps very complicated) patterns among events even if there were no minds around” (2015, p.18). From this, Psillos' approach to laws is objective, since “we can claim that there exists a true deductive system of (our knowledge of) the world [balancing strength, simplicity and stringency], irrespective of whether or not we may ever come to know it” (Psillos 2002, p.150). There exists a best balance of our knowledge but we only make subjective judgements over what that best balance is (2002, p.210). Though we may model regularities as phenomena based upon our purposes, the laws we employ to describe those phenomena will still differ in degree to the objective structure of the mind independent regularities. Consequently the best balance is absolute truth whereas our subjective judgements only give degrees of closeness to that best balance, meaning science deals with approximate rather than absolute truth.

On this account regularities are mind-independent. We model these regularities by generating data and abstracting phenomena based upon our interests. Salmon's causes, Kitcher's explanations and Psillos' laws are all derived from attempts to account for phenomena. Scientists see phenomena co-occur probabilistically, either occurring together or one following the other. They account for this co-occurrence by employing Salmon style causes, Kitcher style explanations and Psillos style laws. In each case, the cause, explanation or law is not mind-independent waiting to be found in any strong sense. They are constructed to attempt to best explain the phenomena. Causes are not mind-independent in the manner Cooper and Murphy defend. Murphy cites Kitcher in his defence (2014a, p.66) but Kitcher explicitly states

> “there is no sense to the notion of causal relevance independent of that of explanatory relevance and that there is no sense to the notion of explanatory relevancy except that of figuring in the systematization of belief in the limit of scientific inquiry, as guided by the search for unification” (Kitcher 1989, p.499).

Also, “I recommend rejecting the idea that there are causal truths that are independent of our search for order in the phenomena” (Kitcher 1989, p.497; see also Kitcher 2001, p.187).

We now have a fundamental starting point. Cooper and Murphy take a cause first approach. They see causes as real and from these causes they want our theories about the world to be derived. In contrast, Salmon, Kitcher and Psillos take a regularity first approach. Regularities are real and from theories describing regularities modelled as phenomena we derive causes (mediated by explanations for Kitcher and laws for Psillos). I endorse a regularity first approach rather than a cause first approach.

4.6 Conclusion

Metaphysical realism can constrain epistemological realism. The evidence required for true theories which accurately describe reality depends upon the nature of reality. By outlining a new metaphysical picture for psychiatry, a new way in which things exist, we allow different types of evidence to entail belief over theories describing reality. In this chapter I outline a new metaphysical picture; the new epistemological arguments will come in chapters five and six.

The metaphysical realism Cooper explicitly adopts and Murphy implicitly adopts restricts their epistemic realism. They require very specific epistemic conditions to consider a psychiatric classification as meriting belief. By outlining a philosophical account of causation, including showing its connection to explanations, laws and entities, we better understand the role causation plays. We also understand how other things can potentially contribute epistemically as much or more than Cooper's determining properties and Murphy's causal mechanisms. If Cooper's determining properties or Murphy's causal mechanisms are not required for belief then we should not automatically adopt epistemic anti-realism over psychiatric classification lacking these.

Popular accounts of causes, explanations and laws all heavily relate to statistical relationships between otherwise seemingly independent things. In this sense autism could be a Salmon style common cause theoretical entity, a Kitcher style unifying explanation or a Psillos style law. The approach to reality I adopt here is Psillos's account of scientific laws. This is regularity first, rather than causes first. For Psillos, a philosophically reconstructed science takes regularities, establishes law-like

relationships then derives entities which causally explain those regularities. I have shown that symptoms should be understood as phenomena, which means they are regularities. I have also shown regularities are often not causally unified, the product of many unstable causes, yet they still work inductively. This means symptoms can work inductively without Cooper's determining properties or Murphy's causal mechanisms. Rather, the stable cause that is responsible for the symptoms (plus its interaction with the environment) is an entity constructed to explain those regularities. We can assign autism as an entity which we primarily know of via the lawful probabilistic relationships of symptoms which autism causally explains. For this, we do not need Cooper's determining properties or Murphy's causal mechanisms. These might assist epistemological arguments for belief in psychiatric classification but they are not necessary.

5.0 Chapter 5 – The interaction between symptoms and classifications

5.1 Introduction

In this chapter I argue psychiatric classifications can play a more important role than often assumed. Psychiatric classifications group together symptoms. This seems to suggest symptoms have a level of independence from classifications; we detect symptoms then make independent decisions about how to classify them. Even if historically symptoms are detected after the classification was formulated, it is assumed that in principle those symptoms could have been detected without the classification. I argue this picture can be deeply mistaken. Symptoms can have far greater connection to classification than typically assumed since psychiatric classifications can influence symptom formulation. The behaviour considered indicative of a symptom may depend upon classifications, literally identical behaviour being considered instances of different symptoms due to a psychiatric classification. This process can potentially increase the epistemic strength of psychiatric symptoms which in turn strengthens our belief in psychiatric classifications.

My argument challenges the belief that classifications add nothing or that classifications without identified or inferred causes are highly inadequate. Murphy believes psychiatry is unsuccessful and classifications need basing on theories (Murphy 2006, p.11). Remember how Cushing, editor of a recent book entitled *The Philosophy of Autism,* gives a particularly striking example of devaluing classifications. Attempting to locate autism in the world, Cushing fails to find autism within psychology, genetics or neuroscience (Cushing 2013, p.41) and believes autism only exists behaviourally which makes it "arbitrary or solely politically/economically motivated" (Cushing 2013, p.38). From this he claims "progress can only come from abandoning it [the classification of autism] and starting from scratch" (Cushing 2013, p.41). Although not wishing to abandon the entire classification, leading autism researchers believe autism needs basing upon theory, such as theory of mind (Baron-

Cohen 1997, p.137; Happé 1994, p.98). Social constructivists often advocate even more extreme solutions, the complete abandonment of psychiatric classifications in preference for focusing on symptoms for research and diagnosis (Bentall 1992a, p.50; Boyle 1990, p.83). Highlighting the important role classifications can play should dampen dissatisfaction with classifications and dampen the desire to base classifications on external theory or abandon classification.

Classifications play an important role in systematizing experience. Behaviour is systematised into symptoms, scientifically useful behavioural characteristics. Symptoms are not ready-made, waiting to be detected but are created through combining behaviour with concepts. My claims here build on those of chapter four which outlined symptoms as phenomena, idealised models designed based upon our interests. Combining behaviour with concepts places behaviour within a systematitised science, turning the world from unordered appearances to structured scientific concepts. From this novel position I can show a crucial role for classifications, acting as concepts to conceptualise behaviour and produce symptoms. This process can epistemically strengthen symptoms by making them more statistically relevant to the world, increasing their inductive adequacy by more accurately portraying reality. Since psychiatric classifications are laws describing statistical probabilities of symptoms, strengthening the symptoms also strengthens the applicability of the laws to reality, epistemically improving psychiatric classifications.

This means symptoms have greater dependence on classifications than often assumed. This places limits on moving symptoms between classifications or joining and merging classification since doing so may require changing the formulation of the symptom. It also limits investigating the causal basis of symptoms independent of existing classifications such as advocated by RDoC since existing classifications may have influenced symptom formulation. Finally, this limits the possibility of completely abandoning classifications since symptoms may have been formulated upon classifications.

Note that by saying symptoms are not ready-made and are created by classifications I do not mean Hacking's looping effects (1999, p.117), it has no relation to his claim people act differently once classified. His argument and my arguments add and detract nothing from each other, they are totally unrelated.

I start by outlining Massimi's concept of systematisation, the bringing of

unordered appearances into systematised phenomena, and then show how symptoms should be considered systematized concepts. I then show how the classification can be employed to systematise appearances into symptoms, meaning symptom formulation can depend upon classifications. I then explore various epistemic advantages this brings to symptoms and classifications before using this position to show how it places limits upon attempts to modify classifications.

5.2 Classifications and Symptoms

5.2.1 Classifications

Classifications group together symptoms. We generate facts about people deemed mentally ill and build classifications covering those facts. What exactly are classifications built from? The common but slightly misleading answer is symptoms, building classifications from statistical analysis of symptoms, determining how often symptoms co-occur through factor or cluster analysis.

> "When a psychiatrist identifies a syndrome on the basis of observations of a select sample of patients he notes that certain behaviours and signs go together and form a functional unity... Factor analysis is simply a more systematic rigorous procedure" (Lorr 1966, p.5).

The "factors identified would represent the behaviour syndromes which are now established entirely through clinical observation" (Lorr, 1966, p.5). The word 'entirely' here is instructive. On this picture we observe behaviours without needing to employ classifications. Classifications are built from symptoms but supposedly not the reverse, we supposedly do not build symptoms from classifications. There are certainly additional possible steps to building classifications, such as corroboration with other factors (Kincaird 2014, p.151; Meehl 1995, p273) or establishing causal mechanisms (Murphy 2006, p.5). Also, building classification requires judgements over how to group symptoms, ones based upon our interests (Cooper, 2005, p.50; Murphy 2006, p.316; Zachar 2014, p.154). All these notions are still based upon

seeing symptoms as being the foundation of classifications. Classifications are certainly built from symptoms but I shall argue that the reverse can also be true, symptom formulations can often depend upon classifications. Let's first consider what symptoms are.

5.2.2 Symptoms

Typically, symptoms are roughly described as behavioural characteristics. Referring to the DSM, Jablensky says "[t]he primary material out of which the diagnostic entities in psychiatry are constructed consists of patterns of human behaviour" (Jablensky 1999, p.140). Murphy describes symptoms as "observable characteristics" (Murphy 2006, p.209). Certain humans exhibit certain behaviour with sufficient regularity, their behaviour forming a pattern, a behavioural tendency or characteristic.

Symptoms are behavioural characteristics, a tendency to exhibit certain behaviour. This means symptoms and actual instances of behaviour are two different things. We observe instances of behaviour and from this we infer a behavioural characteristic. As discussed in chapter four, behaviour manifests in particular contexts, responding to various causal factors, more than the causes we might specifically attribute to a symptom. Low social skills have particular biological and/or psychological causes. In common speaking, these cause the symptom. However, exactly how those low social skills manifest – the words spoken, the degree and nature of the social misjudgement – will also be influenced by many other causal factors such as where the individual is, who and how many people they are talking to, why they are talking, their gender, their age, even day or time of day. We observe behaviour manifested in a context, produced by both those psychological and biological causes attributable to the symptom and those other causal factors specific to that situation. In contrast, the symptom low social skills have a level of generality not present in behavioural manifestations of the behaviour. An individual has the symptom low social skills whether at work, at school and even speaking to the few individuals whom they are capable of exhibiting normal levels of social skills around. This abstracted, generalised notion of low social skills is distinct from instances of behaviour.

Excessive causal factors affect all sciences and motivate Bogen & Woodward's famous distinction between data and phenomena. As described in chapter four, data is localised to specific conditions, say a particular experiment, whereas phenomena is generalizable and yielded by many different procedures such as many different types of experiment (Bogen & Woodward 1988, p.317). Similarly with behaviour and symptoms, instances of behaviour involve many specific causal factors absent in more generalized behavioural characteristics (symptom). Philosophers disagree upon exactly how phenomena is formulated, some emphasising phenomena as built from theories (Brading 2010, p.830; Massimi 2007, p.249; Psillos 2009b, p.87; Schindler 2007, p.162; Teller 2010, p.824; Woodward 2010, p.797) or at least determined by some non-empirical considerations (Bogen 2010, p.779; Glymour 2000, p.32; Woodward 2010, p.797). Without denying the possibility of purely employing statistical analysis of data which McAllister (2007, p.225) favours and Woodwood sometimes considers sufficient for phenomena formulation (2011, p.171), below I show how classifications can be employed to decide which causal factors are relevant and which incidental. Classifications thereby contribute to formulating the symptom. In this sense symptoms can depend upon classifications, in contrast to the typical portrayal of symptoms which has formulation being independent of classifications. I outline this through Massimi's account of systematicity.

5.3 Systematicity

5.3.1 Constructing Symptoms

Bogen & Woodward have not fully outlined exactly what phenomena are. Bogen nicely describes them as “ontological furniture” (Bogen 2011, p.8) employed in science. Both Bogen & Woodward take a realist approach to phenomena (1988, p.337). Massimi suggests they believe phenomena to be out there for scientists to discover (even if phenomena only occur under experimental conditions) (2011a, p.109). This seems a plausible reading of their pioneering 1988 paper but, as discussed in chapter four, Woodward more recently argues phenomena is relative to purpose (Woodward 2011, p.174) (Bogen is more ambiguous (2010, p.781)). This leaves

phenomena sitting between two clear positions of being real and being constructed. Bogen & Woodward need further philosophical work to argue phenomena fit both categories; Massimi has produced a detailed account which accommodates phenomena as both real and being constructed.

Massimi sees "conceptually determined appearance or *phenomena* as the proper object of scientific knowledge" (Massimi 2008, p.14, emphasis original). Science is the act of subsuming appearances under concepts, producing conceptually determined experiences (Massimi 2008, p.11). She shows how in Galileo's experiments

> "the goal of the inclined plane experiment was to extract from the appearance (motion of a bronze ball along an inclined plane) the property of uniform acceleration.... we should not think that what we observe, say, a free-falling object, is the rough-and-ready observable phenomena... If we stick to the level of observable[s]... Galileo may seem no more right than Aristotle" (Massimi 2008, p.25).

In this sense, "phenomena are something that... we *make*, rather than something that comes to us as ready-made in nature" (Massimi 2008, p.8, emphasis original). There are important differences between the appearance of the ball and the property of uniform acceleration, they are not the same thing. Similarly, in psychiatry we subsume appearances under concepts and this produces symptoms which are quite distinct from observations of instances of behaviour. We do not simply use theory to understand observation, rather, we modify what we observe using theories to create symptoms.

Let's compare symptoms and appearances. Consider an individual with the symptom low social skills. Throughout their pre-diagnosis life many observations have been made and many behavioural characteristics have been formulated. They are rude, insensitive, slow, unintelligent, self-centred, etc. However, we eventually diagnose this individual as having low social skills. Thus behavioural characteristics change, the individual now having the behavioural characteristic low social skills rather than behavioural characteristics rude, insensitive, slow, unintelligent, self-centred. It could be responded the individual has low social skills and those other

behavioural characteristics, or low social skills covers those other behavioural characteristics. However, total behaviour associated with low social skills is different to total behaviour associated with the combination of being rude, insensitive, slow, unintelligent, self-centred, etc. Some behaviour associated with behavioural characteristic being rude (and the others) will not occur in low social skills, some behaviour will be missing. Also, there will be additional behaviour associated with low social skills not associated with behavioural characteristics of being rude. The individual may exhibit similar behaviour prior to and after diagnosis but the behavioural characteristics we associated with the individual – and therefore what specific behaviour we believe they will manifest – changes, pre-scientific appearances being different to the symptoms assigned.

Chapter four outlined symptoms as phenomena, providing probabilistic statements about likely behaviour. This is how behavioural characteristics should be understood; having behavioural characteristic of being rude means we believe, given suitable opportunity, someone is significantly more likely to be rude than your average person. The combined regularity guides of being rude, slow, unintelligent and self-centred are different to the regularity guide of low social skills. Symptoms change the probability statements, both of which behaviour is expected and their probabilities. We take the same behaviour (someone prior to diagnosis and afterwards) and generalised it differently to give different statistical probabilities of likely behaviour. Scientists need to decide which data is relevant and which incidental for phenomena (Bogen & Woodwood 1988, p.317), needing to pick data sufficiently caused by phenomena under investigation. Massimi writes that the "phenomena we infer depends on the way we have carved and 'massaged' those data" (Massimi 2011, p.104). Similarly, "[d]ecisions to ignore or discard data play a central role in virtually all data-to-phenomena reasoning" (Woodward 2000, p.177). Our individual diagnosed with low social skills may exhibit an entire range of behaviour, including behavioural manifestations of rudeness not associated with low social skills but at much lower statistical frequencies than those we consider as having the behavioural characteristic rudeness. For formulating symptoms, aspects of rudeness not covered by low social skills are abstracted away, the symptoms they have provide no higher than average probability for such instances.

Our justification for this distinction is systematization. We desire a workable science, probabilistic statements better allowing prediction, control and interaction with the world. Any individual could exhibit almost any behaviour and any behavioural characteristic can manifest in many different ways. Producing science requires substantial selectivity; it's not possible to mention every possible behavioural occurrence. This limitation requires taking some behaviour as relevant and some not. Our major motive is statistical significance; only behaviour sufficiently likely to occur is systematised into instances of a symptom. Hence whether an individual has low social skills or rudeness depends upon how often they exhibit behavioural manifestations of rudeness not associated with low social skills. Rudeness present at average levels means just low social skills, present above a statistical threshold and they have rudeness (potentially with low social skills as well). The importance of behaviour may also influence the thresholds at which it is considered noteworthy. For example, suicide is such a significant event that even those who have a relatively low chance of killing themselves may be noted as being at "risk of suicide". Pragmatic reasons fully justify being selective over which behaviour is systematised into a symptom.

Symptoms are thus created rather than being ready-made waiting to be discovered. The symptoms associated with autism are systematized, creating something quite different to appearances, both prior to the historical conceptualisation of autism and typically prior to knowing someone is autistic.

5.3.2 Employing classifications to systematise symptoms

Formulating symptoms requires deciding which causal factors are localised and incidental causal factors and which are part of the symptom. I now show how classifications can be employed to make this decision. Imagine two different individuals exhibit literally identical behaviour in a particular situation. Imagine both individuals exhibit anxiety and the environmental cause was an unexpected change, a belief a particular course of events will occur and anxiety resulting when unexpected environmental changes results in an alternative course of events occurring. If sufficient in intensity and if occurring sufficiently often then the behaviour likely

counts as a manifestation of a symptom. This symptom likely would be formulated as anxiety, a symptom with many diverse causes. The unexpected change is a localised causal factor, not considered part of the symptom, much like how the individual being male, it being Tuesday and feeling insecure may causally influence behaviour expressed but are localized to that manifestation rather than being part of the abstracted symptom. This, however, can change when a wider range of behaviour is considered, taking into account behaviour other than this anxiety resulting from the unexpected change. Imagine one individual generally acts within boundaries of normality whereas the other individual exhibits many symptoms of autism, sufficient that they have a diagnosis of autism. Autistic individuals often struggle with unexpected changes. An autistic person exhibiting anxiety following an unexpected change will likely not be assigned the symptom anxiety; they are assigned the symptom disliking unexpected changes. The classification changes a causal factor from being a localised causal factor present in specific manifestations to being part of the symptom. The symptom anxiety considers the unexpected change as one of many possible causes of anxiety, assigning the cause to localised manifestations of anxiety rather than considering the cause part of the symptom anxiety. In contrast, the symptom disliking unexpected changes considers the unexpected change as part of the symptom rather than just another localised causal factor like the individual being male and it being Tuesday. Which symptom an individual is considered to manifest can thus depend upon a classification.

Disliking unexpected changes is considered typical in autism, but is rarely discussed outside of the literature on autism. The forty four pages on anxiety in DSM-5 do not mention disliking unexpected changes whereas they are mentioned in the five pages on autism. The closest notion found in the anxiety literature applicable to non-autistic individuals is intolerance of uncertainty. This is when "faced with ambiguous situations, the uncertainty schema will be activated and could lead to the perception of difficulties where problems do not really exist, leading to non-reality based worries" (Freeston 1994, p.800). Intolerance of uncertainty is distinct from other aspects of anxiety worry over uncertain future events (Rosen et al 2014, p.68) and perception of threat (Bredemeier & Berenbaum 2008, p.36). Factor analysis of publications on intolerance of uncertainty showed researchers focus on two elements, desire for predictability and feeling stuck over decision making (Birrell et al 2011, p.1205). This

is very different to notions of autistic people reacting strongly to environmental changes, typically ones neither feared nor predicted in advance. Notions similar to disliking unexpected changes are present to a limited degree in intolerance of uncertainty. Of the twenty questions on the intolerance of uncertainty scale, two are “[u]nforeseen events upset me greatly” (USC, p.1) and “[o]ne should always look ahead so as to avoid surprises” (USC, p.1). These are among the most highest scoring items (Freeston 1994, p.798). Though relatively rare, disliking unexpected changes can be present among non-autistic people who exhibit anxiety. However, this is conceptualised as part of intolerance of uncertainty, itself conceptualised as related to worry which is conceptualised as related to anxiety. A non-autistic individual who reports anxiety following unexpected changes would likely be noted as exhibiting 'anxiety' and/or 'worry'. In contrast, the autistic individual would be very likely to be given the symptom 'disliking unexpected changes', whether or not also given the symptom 'anxiety'.

Additionally, classifications often enable judgments as to whether behaviour is relevant or irrelevant. Systematisation emphasises economy of thought, reducing needless conceptual baggage. Statistical relevancy is often a good measurement of relevancy and formulating symptoms on classifications can improve statistical relevancy. Of all the myriad factors potentially accompanying symptoms, some will occur very frequently and some very infrequently. For the general population anxiety is infrequently caused by unexpected changes whereas for autistic people anxiety is often caused by unexpected changes. Given pragmatic goals, we need to decide what factors are sufficiently common to merit mention within the DSM, on training courses, on undergraduate psychology degrees, etc. We could list days of the week, describing statistical correlations between days of the week and anxiety (perhaps anxiety increases on Mondays after restarting work) but such factors are likely vastly much less significant than many others. Only considering certain factors as relevant rather than incidental is justified; leaving days of the week as likely just conceptual baggage. Statistical occurrences above pragmatically determined thresholds are good reason to demarcate relevant from incidental when formulating symptoms.

Since statistical relevancy is relative to populations these statistical occurrences should be relative to psychiatric classifications and psychiatry would be worse of if they were not. For practical purposes, unexpected changes is best

formulated as relevant only for the population where it is quite common and has substantial impact, namely autistic people rather than all humanity. Classifications are good reason to conceptualise the identical behaviour as displaying either anxiety or disliking unexpected changes.

Alternatively, we could create a classification 'unexpected changes anxiety' disorder with the symptom anxiety which includes unexpected changes within its formulation just so we can apply this symptom to the few non-autistic people for whom anxiety is regularly caused by unexpected changes. So we have general anxiety disorder with the symptom anxiety which does not mention unexpected changes and we have the new classification, 'unexpected changes anxiety' disorder, with the symptom anxiety that does mention unexpected changes. Here though we risk over-inflating the number of classifications we employ. Let's recall Kitcher's approach from chapter four. He desires "the best tradeoff between minimizing the number of patterns of derivation employed and maximizing the number of conclusions generated" (Kitcher 1989, p.432). Similarly, I have argued the psychiatric classification of autism is a scientific law and, as discussed in chapter six, Psillos argues scientific laws are legitimate if part of the best tradeoff between strength (covering as many things as possible), simplicity (employing a few laws as possible) and stringency (whilst being stringent in how those things co-occur) (2002, p.149). If we add a new classification whilst only slightly increasing the coverage of science, we only very slightly make the phenomena we conceptualise more applicable to the world. This does not enhance the goals of systematisation, the aim of employing as few classifications as possible whilst covering as much phenomena as possible. Creating a whole new classification whilst only adding single rarely occurring phenomena is not generally good systematisation.

5.4 Systematicity helping with other background theories and ethics

Determining between relevant and incidental behaviour when formulating symptoms often involves theories and ethics. These can be controversial but systematicity can reduce some of the difficulties. Let's consider theories first.

5.4.1 Theories

Consider delusions as an example. In deciding that someone has the symptom of being deluded we must decide which factors are relevant and which incidental and sometimes we employ theories for this. Typically, the subject matter of delusions is considered incidental, being a delusion whether about dogs or chairs. However, DSM controversially considers certain content as relevant. “Ideas that appear to be delusional in one culture (e.g., witchcraft) may be commonly held in another” (APA 2013, p.103). This is even stronger in an earlier edition; the DSM-IV states religious content are not delusions if “ordinarily accepted by other members of the person's culture or subculture” (APA 1994, p.xxiv). In the case of religious content we interpret the behaviour differently depending on the content of the belief, and no longer see the behavioural manifestations as the symptom delusions. Dogs or chairs are incidental but common religious experiences are not incidental and this changes imagining non-existent things from being a symptom into a non-symptom.

Many philosophers dislike this approach, either objecting or desiring the exclusion clause be justified. Let’s consider the problems. Many approaches define or explain delusions through belief formation, seeing delusions produced by errors in belief formation. However, such approaches risk counting many “normal” people as deluded - as most people hold some beliefs without much evidence or in-spite of evidence (Gillett & Mullen 2014a, p.32). The mechanism responsible for delusions is contested, there is disagreement between rationalist approach where mistaken beliefs causes abnormal feelings or empiricist approach where abnormal feeling causes mistaken beliefs (Campbell 2001, p.91). Mechanistic approaches also disagree over whether delusions are sharply separated from normality or lie on a continuum, schizophrenia at the bottom and normality near the top (Radden 2014, p.45). Some prefer demarcating delusions on values rather than mechanisms but this problematically requires identifying the correct values (Bentall 2014, p.40). Even endorsing the DSM distinction between common vs uncommon beliefs creates problems since different parts of a culture vary over what is considered common, such as different religions having different standard practices (Rashed 2010, p. 201). Some question if adequately demarcating delusions from non-delusions using philosophy is

possible, Radden writing that “attempts to define delusions in contrast to them by appeal to their truth value, or how they have been acquired or maintained, will face daunting double counterexamples at every turn” (Radden 2014, p.43). Similarly, helpful interventions by psychiatrists may be “distorted if our efforts have to be shoe-horned into an over intellectualised view of human psychology” (Gillett & Mullen 2014b, p.48).

Systematicity can help relieve such problems. Systematicity emphasises symptom formulation upon the most relevant factors. Imagine if half of all behavioural manifestations of the existing notion of delusions involved computers possessing the individual. Here we might formulate such behavioural manifestations as a different symptom, since perhaps this particular content can be associated with additional behavioural manifestations, has theoretical connotations not present from typical content or that specific content might lead to specific treatments. Similarly so with common religious experience type delusions. In this regard, conceptualising religious based delusions differently to typical delusions maybe justified on grounds of systematicity.

Systematicity also suggests a radically different approach to symptom formulation than employed by philosophers of psychiatry. Remember systematicity is population dependent. What counts as relevant or incidental depends on the population. Systematicity suggests there is no correct definition or explanation of delusions as such, only of delusions given a particular population. Philosophers implicitly set the population as all humanity. Consequently, the definition of delusions offered need ideally be sufficiently general to potentially cover all humans whilst sufficiently specific to avoid counter-examples. The generality is required so philosophers cannot respond 'but what about this person who is deluded and your theory cannot account for them'. The specificity is required so philosophers cannot respond 'but what about this person who is not deluded but your theory suggests they are'. This task seems impossibly difficult, no philosopher yet succeeding. Systematicity suggests an alternative approach, defining delusions based upon populations smaller than all humanity.

We might define (or explain) delusions relative to two populations, one composing all schizophrenic individuals and one composing everyone else. Factors can change from incidental to relevant depending upon the population. Delusions

associated with schizophrenia statistically take a wide variety of forms whereas in non-schizophrenic individuals unjustified beliefs with the content of common religious experience (alongside seeing ghosts and UFOs) are more common among all content of delusions. This should be a factor in symptom formulation. If common religious experiences becomes statistically insignificant as content of delusions by schizophrenic individuals then delusions with such content by schizophrenic individuals are simply delusions. If common religious experience are much more statistically common than most content associated with delusions then arguably we should formulate delusions with such content differently. We could make common religious experience not delusions for non-schizophrenic individuals but delusions for schizophrenic individuals. Note that a related approach is commonplace, since we do not simply just have delusions but also have specific delusions like paranoia and Capgras delusion. These, however, are taken as different types of symptoms formulated of different behaviour. The Capgras delusion is recognised as different to a standard schizophrenic delusion. Also, each different type of delusion can occur in many different psychiatric classifications. The process I describe is where the same behaviour counts as different symptoms based upon classifications. Let's consider the advantages.

Allowing the definition of delusion to be specific to population allows more relevant formulations of delusions. Statistical significance of various behavioural manifestations taken as instances of delusions may be differently measured by schizophrenic individuals compared to a wider population. Certain behavioural manifestations being more common in schizophrenia and having additional connotations (statistical correlation to additional distress, statistically correlated with further behaviour or amenability to specific treatment) plausibly makes such behavioural manifestations worth mentioning in DSM, training courses and undergraduate degrees. Meanwhile, they might be too common to merit mentioning for the wider population. Secondly, population specific definitions of delusions reduce possible counter-examples. Assume we demarcate delusions as involving irrational thinking. Measuring all humanity supplies endless counter-examples of non-deluded individuals being irrational. These counter-examples are irrelevant to more specific populations, the irrationality of non-schizophrenic individuals becomes irrelevant to a notion of delusion defined for the population of schizophrenia.

Philosophy of psychiatry is deeply mistaken to search for *the* correct definition or explanation of delusions. Assuming a near universally accepted definition of delusions were established, perhaps concrete necessary and sufficient conditions, this in no way entails inclusion in a systematized science. Rather, necessary and sufficient conditions must be statistically significant. Correct definitions and correct explanations should be population specific. On this basis, classification can have an immensely useful role in setting populations.

5.4.2 Ethics

Classifications can also help with ethical problems. The DSM demands mental illness cannot just be normal behaviour or reactions to society, they must "reflects a dysfunction in the psychological, biological or developmental processes underlying mental functioning" (APA 2013, p.20). "Socially deviant behaviour (e.g., political, religious, or sexual) and conflicts that are primary between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual" (APA 2013, p.20). Also, "[m]ental disorders are usually associated with significant distress or disability in social, occupational, or other important activities" (APA 2013, p.20). An individual can exhibit a symptom pattern without actually suffering, only a propensity to suffer is required. Following the DSM, we look at individual behaviour and decide if it meets both these criteria, being caused by a dysfunction and causing suffering. Having decided which behaviour meets these criteria we then see how that behaviour clusters, thus forming a classification. This approach can be problematic and these problems can be partially alleviated using a classification to help decide when any particular behaviour meets either criteria.

Classification can easily help sidestep the first criteria of requiring the behaviour be caused by a dysfunction. Very few symptoms have identified biological or psychological mechanisms. Therefore, we can instead consider if the classification which groups those symptoms has a mechanism which is causally responsible for multiple symptoms. Whilst most classifications also do not have identified causes, some classifications have very diverse co-occurring symptoms and repeat across so many people that it seems highly implausible that a biological and/or psychological

dysfunction is not present (Cooper 2007, p.46; Murphy 2006, p.319). This is not to suggest it is a strong cause or mechanism (I argued in chapter four that Cooper and Murphy were mistaken to rely upon strong causes or mechanisms), merely that stability of the classification suggest some abnormal mechanisms are present. As Psillos writes, "[w]hen it comes to the search for [causal] mechanisms, *anything* can count as a quasi-mechanism provided it performs a function that is meant to explain" (Psillos 2011b, p.786; see also Lipton 2004, p.33). Classifications help ethical problems by helping decide if behavioural characteristics are likely generated by a mechanism and therefore a symptom.

More difficult is the association between psychiatric classifications and suffering. This leads to controversy, for example, grief is a normal response to death of someone significant to the individual and it is unclear when grief should be so excessive as to be medicalised (Wakefield 2012, p.510). Bolton endorses the DSM-IV definition of disorder but argues this means demarcation between illness and normality depends on individual psychology, individual life circumstances and cultural expectations (Bolton 2013, p.445). This is concerning because these might be far more important than the classification itself for causing suffering. Therefore, we risk the association between the symptom and suffering just being artificial noise, actually caused by some other factors. Imagine a symptom were statistically associated with suffering, 5% of the individuals diagnosed suffered greater than average. However, imagine individual personality (say, being positive) or life circumstance (say, poor housing) had a greater than 5% association with suffering. This makes it difficult to say if any given behavioural manifestation of that classification actually is an instance of suffering, thereby resulting in difficulty establishing the correlation between that behavioural manifestation and suffering. Also, cultures may disagree significantly over what, and how much, is normal suffering, limiting our ability to apply the classification widely. When formulating behavioural manifestations into symptoms we need judge if those behavioural manifestations have statistical associations with abnormal suffering and this can be problematic. Other factors may overshadow that link by being more associated with suffering and some associations with suffering are so dependent upon cultural expectations. Consequently, uncertainty over behavioural manifestations means uncertainty over what factors are relevant for symptom formulation.

We can avoid such problems by changing the population from which statistical probabilities are measured. In the above paragraph I implicitly used all humanity as the population. We take an average human as exhibiting average levels of suffering then try to establish how much the presence of a symptom influences the likelihood of suffering among average humans. Measured by all humanity, being shy might have some statistical correlation to suffering but vast numbers of other factors could have higher correlations with suffering. It is possible the statistical correlation is driven by other factors than shyness. Here classifications can help. If we set the population as a particular classification rather than all humanity and if that classification had a 5% association with suffering then those other factors are less likely to overshadow the association between a particular symptom and suffering but overshadowing could still easily happen. Autism is heavily associated with suffering, with studies showing 40% of autistic children and adolescents meeting a clinical diagnosis for anxiety (Francisca et al 2011, p.309) and 43% of autistic adults meeting a clinical diagnosis for depression (Sterling et al 2008, p.1013). The association with suffering is less sensitive to individual personality, individual circumstance and cultural expectations than a psychiatric classification with only a 5% association with suffering. This approach simply shows there is a range of behaviour which is associated with suffering and we can be sure of that association because the association is so high. This is not to claim any particular symptoms specifically causes suffering in all autistic individuals, or that all autistic individual suffer, only that high numbers of autistic people undergo periods of suffering. Rather it suggests our belief that autism has a strong statistical association with suffering among a population of individuals is on much safer grounds than similar beliefs for diagnostic categories less associated with suffering. It shows that the symptoms do have a strong association with suffering, substantially reducing the possibility that the association with suffering is just statistical noise and that the suffering is not related to being autistic but is actually caused by some other factor. This is difficult to establish when implicitly employing all humanity as a population for measurement, consequently, when classifications are sufficiently associated with suffering this assists symptom formulation.

5.5 Historical evolution and interaction of symptoms and classifications

There is an obvious problem with my argument. I accept classifications are built from symptoms but I also argue that symptoms are built from classifications. This appears a chicken and egg argument, both relying on the other. Additionally, I have suggested formulating symptoms on classifications increases the epistemic strength of symptoms but surely this epistemic strength is constrained by the epistemic strength of the classifications – which surely must be constrained by the epistemic strength of the symptom. So hypothetically from pre-psychiatric appearances we formulate bad symptoms but hope the classification could be used to improve those symptoms yet that classification itself is already built from those bad symptoms – a negative epistemic feedback relation. This argument might look convincing from certain philosophical armchairs, those looking for secure foundations or non-circular justifications. A closer analysis of scientific practice shows this not always to be the case. Woodward outlines how this can occur between phenomena and theory.

> "[T]wo sets of considerations [phenomena and theory] are [can be] in a positive feedback relation with each other. That *P* [phenomena] apparently obtains might in turn to be important evidence in favor of *T* [theory]. This is a case in which *T* figures in the overall process leading to acceptance of *P* in an important way and *P* in turn supports *T*... it is far from obvious that the process described is automatically viciously circular or that it fails to provide a legitimate basis for increased confidence in *P* (or *T*, for that matter) (Woodward 2011, p.178).

Massimi and Kitcher emphasise that science is fundamentally a historical process, scientists taking one concept of the world, investigating and testing it, making modifications some of which work and some of which do not. Given time, they hopefully adopt the better supported parts, making improvements and eventually, providing the process works, we end up with something worthy of belief. Kitcher writes that

> "we begin with a haphazard collection of beliefs, and, in our epistemic maturity, we regenerate them in a more adequate fashion... it regards

> the epistemic status of the beliefs of current subjects as dependent on the reliability of a social-historical process that extends into the distant past" (Kitcher 1993b, p.160).

Massimi writes that

> "[i]t is not a God-given metaphysics of natural kinds that ultimately supports our inductive inferences but rather how well-entrenched our conceptual resources are in our cultural history. The resilience and historical evolution of our natural kinds testifies to how entrenched our scientific taxonomy must be to grant us comprehensible experience of nature" (Massimi 2014, p.438).

She outlines how phenomena and theory are often modified in light of one another, mutually improving the systematisation. We do not simply systematise but systematise in light of what we already know, then modify what we already know in light of how well (or not) our systematization worked, and throughout this process we hopefully keep the parts that work, the inductively reliable parts worthy of belief (Massimi 2007, p.260; Massimi 2010, p.21).

I suggest autism has undergone an evolution in a manner that helps justify belief. Prior to the conceptualisation of autism symptoms were organised in a particular way. Then autism was introduced in 1943, systematising symptoms in a new way but also symptoms were modified. Behaviour which was previously considered as low intellect was now seen, sometimes correctly, as aloneness or insistence on sameness. The symptoms were conceptually improved when applied to a particular population. In the 1950s Kanner's symptoms were applied to another two populations, older autistic people and schizophrenic children, resulting in the symptoms being formulated in a more general manner. In the 1980s Wing found the most important statistical factor within her classification was socialising. She found "severity of social impairment gave more statistically significant associations with behavioural, psychological, and medical variables" (Wing & Gould 1979, p.25) than earlier psychiatric classification which were employed. Having removed the older

classifications by formulating an autistic spectrum she then recast the symptoms as forming a triad of impairment of social communication, social interaction and social imagination. Having modified the classification the symptoms were then given new emphasis as relating to social defects. Symptoms and classification have been modified in light of one another in a manner which has resulted in symptoms being formulated with higher inductive reliability, being more relevant to the new population.

5.6 Consequences

Many are dissatisfied with existing classification and think they should be reformulated. Let's assess proposals to reformulate classification in light of my arguments over symptoms depending upon classifications.

Statistical significance of symptoms depends upon populations, consequently modifying classifications may modify statistical significance of symptoms. We could reformulate existing classifications by adding or removing symptoms from a classification, by merging two classifications and by splitting a classification. Generally, it is assumed modifying classifications needs no modification to symptoms. We just modify how we believe those symptoms cluster. Kendler & Zachar seemingly appeal to this picture when suggesting symptoms remain unmodified even when classifications undergo change. They write that

> "another way for researchers to protect themselves from the effects of diagnostic instability at the level of syndromes [classifications regularly being modified] is to also study these underlying traits [symptoms]... formalised measures of traits will not change according to a programmed schedule like the DSM and the ICD" (2008, p.374).

This picture can be mistaken. When symptom formulation depends upon populations then modifying the population may ideally require also modifying the symptoms. Factors relevant to one population may not be relevant to another population. Exactly what we consider statistically significant involves a level of choice but we should not

assume behaviour that passes that threshold in one classification will do so in another classification, potentially risking reducing epistemic strength of symptoms.

5.6.1 DSM

This has important consequences for future DSM revisions. The current approach is conservativism, DSM not undergoing major changes across editions. Though not fully endorsing this, Zachar (2012, p.31) and Kendler (2012b, p.320) seem quite sympathetic to this general approach, accepting that progress can often come through small incremental changes rather than radical changes. Poland argues cautious updating will not improve classifications if they are already sufficiently bad (Poland 2014, p.50). My argument supports both sides. Modifying good classifications risks taking symptoms relevant to that classification and finding them less relevant in the new classification. Changes should be small and the relevancy of symptoms needs carefully checking within the new classification. However, if existing classifications are already bad, lacking strong reason to group symptoms, then formulating symptoms relevant to that classification is no strong epistemic accomplishment. Imagine we abandoned the classifications of whales and octopuses, merging them into one species. Relevant behaviour characteristics could be produced (they swim, they eat fish) but this relevancy here would not justify the new classification. Plausibly, a bad classification with relevant symptoms is better than reformulating that classification to produce another bad classification but with less relevant symptoms. However, if bad classification constrains quality of symptoms then small incremental changes to classification risk being constrained by those conceptually weak symptoms. Consequently, for good existing classifications an incremental approach might be best whereas for bad classifications something more radical than cautious progressivism might be justified.

5.6.2 RDoC

My argument also raises problem for RDoC which intends to investigate biological

causes of mental illness without reference to existing classifications, fearing weaknesses of existing classifications negatively effect biological investigation. They desire circumnavigating classifications by directly linking symptoms to biological findings. The RDoC aims to be "trans-nosographically organized" (Aragona 2014, p.39). No longer employing existing classification, alternative populations need be employed. Consider how RDoC founder Insel suggests choosing populations for biological investigation. Since the

> "group of individuals to be entered into the study... will not be identical to a DSM or ICD diagnosis, other criteria will have to be applied. In some cases, this might simply comprise all patients presenting at a certain type of clinic, such as for anxiety disorders or serious mental illness" (Cuthbert & Insel 2013, p.5).

A study might try correlating biology with symptoms of everyone at a particular clinic rather than everyone with a particular classification. Other options include anyone attending a particular type of clinic (such as anxiety clinic), anyone displaying a particular symptom (rather than classification), anyone with particular risk factors and anyone with particular environmental causes (Stanislow et al 2010, p.635). Epistemic optimism surrounding RDoC is based upon existing classifications playing little role, seemingly based upon an image of symptoms out there waiting to be found. However, some symptoms have been formulated based upon existing classifications, as highlighted by my anxiety vs disliking unexpected changes example earlier. This means existing classifications still have influence upon RDoC process. Biological investigation will not simply link biology with symptoms but link biology with symptoms potentially formulated by classifications RDoC desire to circumnavigate. This suggests RDoC is more closely tied to existing classifications than realised, limiting the project.

5.6.3 Symptom-based approaches to psychiatry

Some theorists advise completely abandoning all use of psychiatric classifications. For

example, Boyle accepts people we call schizophrenic hear voices and have confused thinking, writing that we

> "acknowledge the person's behaviour and experiences and devote enormous energy and resources to trying to understand why these phenomena occur and what variables influence them, but without inferring unsupported concepts like schizophrenia" (1990 p.166).

However, when symptoms are formulated on psychiatric classifications, abandoning all psychiatric classifications still likely entails employing symptoms possibly formulated partially based upon existing classifications. Symptom-based approaches to psychiatry underestimate the difficulty of removing all influence of existing psychiatric classifications. Where this situation is applicable then just diagnosing symptoms without diagnosing classifications will not escape the influence of current classifications, as highlighted by the anxiety vs disliking unexpected changes example. Additionally, since formulating symptom on classifications can increase epistemic strength, those desiring to abandon psychiatric classifications deny themselves an epistemic tool, disallowing themselves the possibility of improving existing classification which might also improve symptoms. This substantially limits symptom-based projects of abandoning psychiatric classifications.

5.7 Classification, systematisation and belief

My ultimate aim is arguing autism merits belief, so does the systemitisation process described increase belief? To many observers this process likely increases their concern. Some believe that "posited attributes [symptoms assigned to an individual], rather than being possessed by subjects, exist, so to speak, in the heads of researchers" (Boyle 1990, p.223), just a bunch of psychiatrists making arbitrary decisions (Bentall 1992b, p.293; Horwitz 2002, p.5). Throughout this chapter I have shown how psychiatrists have 'constructed' a new type of anxiety just for autistic people, even though that type of anxiety can also affect non-autistic people. This might raise fears psychiatrists are medicalising based upon their own personal preferences. A social

constructivist would quite likely take the process I describe as evidence psychiatrists are 'just making things up'. Such an approach to thinking about belief is extremely unhelpful. Psychiatric symptoms are constructed but, following Massimi, all phenomena in science is constructed. Also, they are constructed for good reasons, since literally accounting for every causal factor is likely impossible. Also, it would not be pragmatically wise to try and do since science can work without accounting for all causal factors.

5.7.1 Enhancing Inductiveness

As chapter four showed, phenomena are employed inductively and we need decide which factors are relevant to phenomena and which incidental. Assume we knew all the factors causally influencing a specific instance of a phenomena. Formulating the phenomena to include all those factors would only increase inductiveness to the degree to which those factors are generally present in future instances of that phenomena whilst factors included which are rarely present actually decreases inductiveness. Remember that the factors which occur typically change from one instance to the next, so including some factors will reduce inductiveness. Consequently, formulating phenomena should be (as discussed in chapter four) constrained by "what could be modelled managably and reliably" (MacLeod & Nersessian 2013, p.545). More detail in phenomena often means less tractability (Batterman 2002, p.22), reducing generality (Rohwer & Rice 2013, p.336) and applicability to future situations (Myrvold & Harper 2002, p.137). We thus have a tension. Adding factors associated with a phenomena makes the phenomena more inductive if those factors occur regularly and less inductive if those factors occur infrequently. If unexpected changes are frequently a causal factor of anxiety then including unexpected changes within notions of anxiety increases its inductiveness. However, if unexpected changes are very rarely causal factors of anxiety then including them reduces the inductiveness. Adding unexpected changes to anxiety leaves anxiety less specific, covering a wider variety of factors, but whether this loss in specificity is made up for by applicability depends upon how often that additional factor is present in instances of anxiety. Regularly and anxiety becomes more

inductive, rarely and anxiety becomes less inductive. As I have argued, within the population 'all humanity' unexpected changes rarely cause anxiety, meaning notions of anxiety would become less inductive if included with the general symptom anxiety. However, within the population 'all autistic people' unexpected changes often cause anxiety, meaning including unexpected changes within our concept of anxiety for that population increases the inductiveness of phenomena. The inductiveness of phenomena is improved by formulating phenomena of anxiety differently for different populations demarcated by classifications.

Since some classifications are best thought of as scientific laws, strengthening the inductiveness of phenomena in turn strengthens the scientific law. Scientific laws describe probabilistic co-occurrences of regularities and regularities are inductively reliable occurrences conceptualised into phenomena depending upon our interests (as shown in chapter four). Making phenomena more inductive by making it more applicable means we strengthen the law describing probabilistic relationships of those phenomena. Conceptualising anxiety for autistic people differently than for non-autistic people strengthens autism as a scientific law, contributing towards belief. This also increases the presence of the theoretical virtue unification. As discussed in chapter six, unification is where as few theories are employed to cover as much phenomena as stringently as possible, increasing the truth of theories.

5.7.2 Systematisation and theory-ladenness

Symptoms being derived from populations set by classifications is related to the theory laden-nature of evidence. Specifically, employing one population (autistic people) or employing another (people suffering from anxiety) can result in the same person being seen as manifesting different symptoms. By employing a particular population scientists are more likely to focus upon particular aspects of patients and may conceptualize different symptoms for individuals within that population. Of the three types of theory ladeness discussed in chapter three, this is not perceptual loading. It does not change perceptions, at least in the examples I have provided. It is semantic theory loading and salience, giving emphasis to different behavioural manifestations when formulating symptoms. This is not particularly deep theory-

ladenness. I suggest psychiatrists will likely often switch from employing one population to another. Also, if aware of this process, psychiatrists would not face massive challenges to actively formulating symptoms as statistically relevant to psychiatric classifications. Theory ladenness is only harmful when based upon bad theories and chapter six will show autism is a good theory. Therefore, when employing autism, theory-ladenness is not an epistemic threat, unlike potentially theories like psychoanalysis and cognitive psychology.

5.8 Conclusion

Many believe classifications are a necessary or unnecessary evil. Those who consider them unnecessary wish them dispensed with, those who consider them necessary desire new and superior classifications. Both approaches fail to recognise a role classifications can play, acting as a population to decide statistical relevancy. This previously unrealised epistemological role strengthens symptoms, making them more applicable to the world and thus more inductive. Additionally, this means symptoms are more closely tied to existing classifications, limiting the possibility of radical reformations. All this means classifications are far more important than typically believed.

Symptoms are not ready-made, waiting to be found. Symptoms are abstracted behavioural manifestations and this abstraction involves choices. Symptoms are idealisations, missing many factors, and systematisation encourages this. Listing all factors is generally impossible and totally impractical for a predictive science. Systematisation aims to conceptualise symptoms based upon relevancy, typically substantially a statistical matter. We generally should formulate symptoms based upon frequently occurring factors.

Statistical relevancy depends upon populations. Measuring on more specific populations can greatly enhance systematicity. Measured by all humanity, unexpected changes should plausibly not be included within symptom formulation because they are rarer than many other factors. Measured by autistic people, they are more common, increasing their relevance so plausibly should be included in symptom formulation. In this regard symptoms can depend upon classifications. Additionally,

formulating symptoms on theories and ethics involves decisions over relevancy. Specific populations can increase relevancy of theoretical and ethical claims, also making applying them easier. Symptom attribution is improved by considering smaller populations than all humanity.

The importance of classifications has substantial implications for reformulating psychiatry. Moving symptoms between classifications, splitting and lumping may involve symptoms formulated based upon existing classifications, meaning existing classification can play an influence, limiting any radical potential of such reformations. Also, RDoC aims to circumnavigate psychiatric classifications by directly linking causes to symptoms whilst some social constructivists advocate for abandoning psychiatric classifications to just focus on symptoms. However, some symptoms are formulated on existing classifications, reducing the possibility of fully avoiding existing classifications.

I have shown classifications sometimes play a more important role than previously considered. Some commentators see autism as arbitrary and not making a scientific contribution but the systematicity process allows psychiatric classifications to make non-arbitrary contributions. Additionally, this systematicity process strengthens autism as a law. This is significant because my argument for belief is based upon autism being a scientific law.

6.0 Chapter six – Scientific Realism and Autism

6.1 Introduction

In this chapter I describe criteria under which a psychiatric classification merits belief and show autism meets those criteria. Questions over scientific realism have huge ramifications for decisions over keeping or abandoning psychiatric classifications. Many scientific theories have been abandoned because they did not merit belief and "it is possible that [for example] schizophrenia is not a meaningful scientific concept and that it should be abandoned along with all the other meaningless concepts (for example, the four humours, phlogyton, the luminiferous ether)" (Bentall 1992a, p.24). Cushing and Timimi have called for autism to be abandoned. Scientific realism being justified over autism would be a good reason not to abandon autism.

Belief matters because we need psychiatric classification to have certain levels of adequacy to be useful. If psychiatric classifications merit belief, we could expect them to provide a good basis for helping people. Many do not believe in existing classifications and desire that we abandon them, completely modifying how we cluster symptoms. Many see biological or psychological causes of mental illnesses as required for belief in a classification and desire to abandon classifications without identified or inferred causes. Successfully providing alternative means to belief than identified causes would be good reason to decide a psychiatric classification should turn up in DSM-6.

For belief about scientific claims to be justified involves three steps. First, an inference to the best explanation, establish how theoretically virtuous theories are. Secondly, the inference to the best explanation must be justified. We must assess if the conditions under which the inference to the best explanation is made reliably generate true claims. Thirdly, the level of risk involved in the inferences must not exceed a personal limit on acceptable epistemic risk.

Philosophers of psychiatry commonly focus upon causation as a justification

for belief. I portray causation as a theoretical virtue. Causation can be a route to belief but other theoretical virtues can (under reliable circumstances) grant belief in the absence of causation. I suggest many philosophers making such arguments have adopted an implicit neo-Aristoleanism, whereas I provide neo-Humeanism, neo-Kantianism and pragmatism as alternative underlying philosophies.

I apply these arguments to autism. I argue autism strongly exhibits the theoretical virtue unification. It unifies together a whole set of otherwise seemingly unrelated symptoms in a stringent manner. I argue this assessment of theoretical virtues has taken place under reliable conditions. I appeal to epistemic conditions such as high accessibility, having a short causal chain between autism and data, not requiring highly specialized experiments, not requiring highly precise results, not requiring high selectivity of data, not requiring precise demarcations, not requiring questionable theories and not requiring high idealization. These will be inapplicable to some parts of psychiatry but is applicable to most, though not all, claims about symptoms and classification of autism. My discussion of what is required for belief is applicable to all psychiatric classifications; I employ autism as a case study showing an example where belief is justified.

I start by discussing scientific realism in greater detail, then I establish how autism passes an Inference to the Best Explanation (IBE), then I show how IBEs over autism are reliable and then I discuss issues relating to epistemic risk.

6.2 Scientific Realism

My measure of scientific realism is taken from Stathis Psillos, a central figure in modern philosophy of science on scientific realism. Let's first establish some basic parameters.

Psillos' central claim is "if scientific theories are true, then the entities posited by them are real" (Psillos 2009c, p.44). One might interpret this under typical notions of realism, translating Psillos' quote as 'when science discovers mind independent entities then our theories are true'. Notice, however, Psillos takes the opposite approach – true theories decide which entities are real, rather than real entities determine which theories are true. Psillos puts theories first and this has massive

ramifications for thinking about true psychiatric classifications. As I will show, this allows a neo-Humean regularity first approach to scientific realism rather than a neo-Aristolean cause first approach to scientific realism.

Scientific realism is quite metaphysically minimal, only requiring "whatever commitments are necessary for securing the Possibility of Divergence" (Psillos 2012, p.211). This is where the world might diverge from our scientific theories. Psillos takes a strong realism as, quoting Putnam, "some fixed totality of mind-independent objects. There is exactly one true and complete description of 'the way the world is' " (Putnam in Psillos 2012, p.197). In contrast, Psillos writes that

> "if it were rejected that there is such a fixed totality of objects and a fixed set to their intrinsic properties, it would still seem possible that we might be unable to represent the world and that the world might be independent of any particular representation we have of it" (2012,

We do not need a metaphysics of a fixed set of mind-independent objects. We need sufficient metaphysics that our theories might make false claims, specifically, our inductive and causal claims might turn out false. As I will show, Psillos endorses a Humean scientific realism, rather than a richer metaphysics of causal powers for instance offered by neo-Aristolelians.

Psillos demarcates between fundamentalism and scientific realism. A metaphysical fundamentalist believes certain facts are more fundamental than others, the less fundamental facts produced by more fundamental facts. Scientific realism aims to establish the facts, not which facts are fundamental. For example, logicism argues maths purely reduces to logic, making logic the fundamental facts. "But from the claim that '7 + 5 = 12' does not represent a *sui generis* mathematical fact it does not follow that it does not represent a fact. Reductionism does not show that something is unreal. It shows that is not *sui generis*" (Psillos 2009c, p.37, emphasis original). Failure to offer a reductionist account of autism does not entail anti-realism.

Finally, scientific realists aim for approximate truth rather than full truth. Scientific realism broadly argues for the approximate truth of laws, causes and entities (Psillos 1999, p.277). The inexactness of science means only approximate truth is possible (Psillos 1999, p.276). Realism over scientific laws governing gases is realism

over ideal gases only occurring in particular situations. Scientific realism means real gases behave just like ideal gases providing those real gases were in certain conditions where other factors not accounted for by the law are non-existent. In other words, if real gases were placed in the same conditions as the ideal gases (which may never occur) then both would act the same (Psillos 1999, p.277). Scientific realism is realism about scientific claims and therefore allows approximations and idealisations.

6.3 Existing approaches in philosophy of psychiatry

Belief in psychiatric classifications, both for philosophers and non-philosophers, usually relates to causation. We saw how Murphy values causal mechanisms and Cooper values determining properties. This seems similar to notions of validation employed in psychiatry. “The validity question is how 'good' are these symptoms at representing the hypothesised underlying illness” (Goodyer 2012, p. 335). Validity relates to ontological reality, questions about real entities or diseases (Pies 2008, p.49). Most attempts at validation relate to causation, either directly found or inferred from family studies and treatment responses. Finding biological factors will help “identify actual neural or genetic mechanisms. . . actual causes” (Andreasen, 1995, p.162). In contrast, psychiatric classifications which lack identified or inferred causes are not typically considered as meriting belief.

Unfortunately, which notions of causation or belief is being employed is rarely described. Murphy talks about “causes of mental illness [that] are genuinely out there in the structure of the world, waiting to be discovered” (Murphy 2014a, p.62). In contrast, other standards of assessing psychiatric classification such as reliability or questions over co-morbidity, do not relate to reality (Murphy 2014b, p.75). This is because psychiatric classification involves values, be them ethical or pragmatic values, and such values are not part of reality. Relatedly, Cooper sees clustering properties which are grouped into natural kinds as “reflect[ing] real structures in nature” (Cooper 2005, p.49). The clusters which allow groupings of natural kinds are themselves produced by determining properties. Cooper describes these determining properties as “[g]enuine properties, such as possessing negative charge, endow entities with particular causal powers, and ground objective similarities” (Cooper 2005, p.52).

In contrast, such genuine determining properties are absent in non-natural kinds. Murphy and Cooper respectively emphasise identifying or inferring causation as routes to belief over psychiatric classifications. Unfortunately, none of this explains why causes relate to reality or explains what is accomplished when something is real. We need to more deeply explore notions of causation and reality.

6.3.1 Neo-Humeanism vs Neo-Aristoleanism

Psillos contrasts two major philosophical traditions, neo-Aristoleanism and neo-Humeanism. Neo-Aristoleans see regularities as produced by causes whilst neo-Humeans see causes as produced by regularities. Each approach gives a different status to causes and different status of how causes relate to reality. Let's consider each approach.

Psillos characterises neo-Aristoleanism as having a

> "view of the deep structure of reality... [a] commitment to this rich metaphysics... a metaphysical accounts of causation and laws and dispositional essentialism about properties and natural kinds" (Psillos 2014, p.91).

Neo-Aristoleans sees causal powers as fundamental and irreducible, being synonymous with properties of entities. Causal powers of entities interacting gives rise to scientific laws. Psillos groups a wide variety of philosophers under the name neo-Aristoleanism. They have some different commitments. For instance, Ellis believes natural kinds have dispositional essences, essential properties which give rise to laws through their causal interactions (Ellis 1998, p.22). Slightly different is Cartwright who believes the world consists of a patchwork of laws produced by capacities which can activate causally when in suitable environments (Cartwright 1999, p.50). They differ because Ellis sees dispositions as necessitated universals, the causal power always activating in certain situations (Ellis 1998, p.22), whereas Cartwright does not think capacities are universal or necessitated, the causal power often not activating in a suitable environment (Cartwright 1999, p.72). We can assign neo-Aristotelianism

two key claims – causal powers are real and they are not derived from anything more fundamental.

In contrast to neo-Aristoleanism, neo-Humeanism takes any talk of causation as parasitic on regularities. The neo-Humean world consists of regularities. Laws are a special type of regularity, ones which form part of the best balance between strength and simplicity. To say one regularity caused another regularity means the second regularity occurred after the first. However, regularities often follow one another but causes are only those regularities which follow one another and are part of the best system balancing strength and simplicity. Causes which cluster in the world are conceptualised as properties of an entity. Entities having properties means causal relationships have clustered together. Both properties and entities are derived from causes and causes are derived from laws, therefore properties and entities are ultimately derived from laws. Psillos describes some divergent views as neo-Humean, considering Kitcher and Salmon (described in chapter 4) as neo-Humeans (note that Kitcher is a neo-Kantian but Psillos considers neo-Kantianism a variation on neo-Humeanism). Kitcher sees science as building a systematised explanatory structure which subsumes regularities into a unified system of inter-connected phenomena (Kitcher 1989, p.476). Causes are derived from probabilistic relations between phenomena, showing how one phenomena can follow another. Causes are derived from efforts at unification (Kitcher 1993a, p.172). Salmon employs the common cause principle to derive causes (Salmon, 1998 p.110). Events occurring with greater than chance frequency suggests a cause is present. Assigning causes depends upon regularities of events. Similarly, unobservable entities are derived to fill in gaps between observations, the entity giving a causal explanation of how one observation relates to another (Salmon, 1998 p.113). All these views are much more metaphysically minimal than neo-Aristoleanism, taking regularities as fundamental whilst causes, entities, natural kinds and dispositions are not fundamental but are dependent on regularities. We can assign neo-Humeanism two key claims – regularities are real and causes are derived from regularities.

6.3.2 Murphy, Cooper and neo-Aristoleanism

Murphy and Cooper have substantial similarities to neo-Aristoleanism. Let's consider how well neo-Aristoleanism fits other parts of their general argument.

Whether neo-Aristoleanism is suitable in the life science is debateable. Psillos thinks it is unclear that neo-Aristoleanism can be extended to biological species (2014, p.93). Neo-Aristolean Ellis would agree, associating Aristoleanism with an essential unsuitable for biological species since "within a species, there is often a lot of genetic variation, and sometimes there are no sharp genetic distinctions between different species" (2001, p.169). In contrast, though he does not identify as a neo-Aristolean, Dupré's position resembles neo-Aristoleanism through his emphasis on causal powers and he considered biological species as natural kinds (1993, p.57). Even if it could be extended to biological species it would not easily deliver Cooper's or Murphy's position. They both suggest psychiatry could be improved to become more real. However, if causes make something real then Cooper's and Murphy's position is problematic. Remember that stronger causes are easier to find so we have probably found most of them already. This means Cooper's claim that modern classifications do not reflect the causal structure of the world does not then entail a superior classificatory system would likely reflect the causal structure of the world. Psychiatry has likely found most determining properties, leaving us close to the limits of natural kinds modelled as biological species that we will discover. Most biological species have determining properties but we should not expect this picture replicated in psychiatry. Similarly, Murphy could no longer be an optimist about the future because most psychiatric classifications with strong causes have likely already been found. All this contrasts with biological species which have a strong genetic basis. Neo-Aristoleanianism does not easily entail there being lots of real psychiatric classifications waiting to still be discovered.

6.4 Alternative approaches to scientific realism

In this chapter I outline various alternatives to neo-Aristolean approach to reality and apply them to psychiatry. Though they do not all identify as neo-Humean, all fall on neo-Humean side of the divide Psillos places between neo-Aristolean and neo-Humean – each position thinks belief can be legitimated in the absence of finding (or

inferring) fundamental causes. The four positions are neo-Humeanism, neo-Kantianism, anti-realism and pragmatism.

There are three stages to scientific realism. Firstly, an inference to the best explanation. Secondly, that inference to the best explanation must be reliable. Thirdly, the epistemic risk involved in the inference to the best explanation must not exceed ones position on epistemic risk. All three stages need passing, passing one or two is insufficient to attain scientific realism. I will outline each stage in turn.

6.4.1 Inference to the best explanation

The fundamental challenge to belief in science is the underdetermination argument. As described in chapter two, more than one theory can fit the evidence, therefore theories are underdetermined by the evidence. The evidence alone does not show which theory is true. Therefore, something more is needed to decide a theory is true.

Realists employ an inference to the best explanation (IBE) by taking competing explanations and deciding one is best on the grounds that is most theoretically virtuous. Alongside empirical adequacy, theory assessment need

> "take into account several theoretical *virtues* such as coherence with other established theories, consilience, completeness, unifying power, lack of ad hoc features and capacity to generate novel predictions. These virtues capture the *explanatory power* of a theory, and explanatory power is potentially confirmatory" (Psillos 1999, p.171, emphasis original)

These theoretical virtues give reason to believe a scientific theory offers an explanation which merits belief, and allows scientists to decide which theory among competing theories merits belief.

What is the status of these theoretical virtues? Why should we care about them and how do they relate to reality? The major issue relates to just how theoretical, rather than empirical, these theoretical virtues are.

> "[R]ealists regard theoretical values as *testable presuppositions* about the world, and antirealists regard theoretical values as *pragmatic constraints* on theorizing. Realist acknowledge that the world might be simple or complex, unified or disunified (and thus explainable by theories of wide or narrow scope), hierarchical or self-organizing, and even best modeled by consistent or inconsistent theories (since humans may not be capable of exactly representing the world). Theoretical values are testable and, historically, evidence has disposed of theoretical values when they do not survive empirical tests. Thus, for the realist, theoretical successes are not only secondary to empirical success, but valued only when they bring empirical success" (Solomon,

Of the four position described below, two (neo-Humeanism and neo-Kantianism) see theoretical virtues as testable whereas the other two (anti-realism and pragmatism) see theoretical virtues as pragmatic constraints.

Let's start with Psillos' neo-Humeanism. He believes the world has an objective structure and scientific theories have higher approximate truth the more accurately they describe it. For Psillos,

> "the world has an objective structure, in which (fully mind-independent) regularities stand in certain relations to each other... explanatory relations are subjected to some external – and mind-independent – standard of correctness: *the nomological structure of the* world" (Psillos 2002, p.293 emphasis original).

The regularities hold relationships and this can be captured by a system of laws. The system of laws "should be as informative as possible *vis-a-vis* the regularities that hold in the world" (Psillos 2002, p.149). The system of laws which best balances simplicity, strength and stringency (as discussed in chapter four and described again in detail later) will maximise information. Theoretical virtues of simplicity, strength and stringency create the most informative system. Reality is nothing more than regularities (including the relationship between regularities) and greater belief means being more informative of those regularities. We connect laws to the mind

independent regularities by nested layers of models, the most basic model being phenomena which are then accounted for by higher models (Psillos 2011a, p.7). Theoretical virtues make a theory more informative of regularities and thus more worthy of belief.

Neo-Kantianism also sees truth as the most theoretically virtuous scientific description of regularities. However, neo-Kantianism differs from neo-Humeanism by denying the world has an objective structure of regularities. Kitcher's neo-Kantian aims for the most theoretically virtuous explanatory system but does not take the most theoretically virtuous explanatory system as being closest to an objective structure of reality (Kitcher 1993a, p.171; see also Psillos 2002, p.292). Rather, correct explanations are those which would be found at "the limit of the rational development of scientific practise" (Kitcher 1989, p.498). Similarly, neo-Kantian Massimi sees science as building mathematical structures accounting for phenomena but more than one structure can fit the world which undermines notions of an objective structure (2010, p.20). Despite this, theories merit greater belief the more they fit phenomena into unified mathematical models, effectively endorsing theoretical virtues (Massimi 2010, p.21; Massimi 2011, p113). Neo-Kantianism sees science as a reliable means of turning appearances into phenomena through subsuming them under concepts, believing the world heavily constrains this process. The world constrains the data from which phenomena is constructed. It also constrains the background theories used to formulate the phenomena since background theories were constrained by the world when themselves formulated from data and phenomena. It is this constraining process, rather than closeness to an objective structure, which makes scientific theories worthy of belief to neo-Kantians (Kitcher 1993b, p.160; Massimi 2014, p.438).

Let's now consider two positions which take theoretical virtues as pragmatic tools. Anti-realists typically accept that truth over unobservables is potentially possible but usually restrict belief only to empirical adequacy. They are not semantic anti-realists, they believe truth is potentially obtainable and scientists should seek to make their theories true (Laudan 1984, p.105; Stanford 2006, p.193; Van Fraassen 1980, p.10)**.** However, anti-realists typically doubt that theoretical virtues deliver true theories. Anti-realism is compatible with both a neo-Humean objective structure and a neo-Kantian lack of objective structure; the difference is not over notions of truth but over whether truth has been obtained. Van Fraassen argues there is no rationally

compelling reason to believe in scientific theories except over their empirical adequacy (1980, p.99). Laudan believes scientists should aim for truth but doubts they usually attain it (1984, p.136). Stanford does not believe in most unobservables but does believe in some, such as unobservable processes responsible for fossils of dinosaurs (Stanford 2011, p.893). Anti-realists can be characterised as believing truth is attainable but generally believe we lack evidence to believe science has obtained truth. They typically value theoretical virtues for their pragmatic benefits rather than for truth, whereas neo-Humeans and neo-Kantians value theoretical virtues because they deliver truth and pragmatic benefits.

The final position is pragmatism. Pragmatism sees truth as usefulness. Therefore, to the degree those theoretical virtues produce useful theories (and they generally do), theoretical virtues provide truth. Fine argues both realism and anti-realism are mistaken, seeing assertions of truth as adding nothing above claiming something is scientific (Fine 1996, p.133). Unlike anti-realists, pragmatists cannot claim truth is possible but currently unattained, except for potentially claiming more useful theories might be obtained. Consequently, they cannot endorse an objectively true structure. Also, anti-realists consider unobservables as useful but not true, a position unreconcilable with pragmatism. Neither neo-Kantians nor pragmatists accept an objective structure but they disagree over notions of truth. The constraining process neo-Kantians value may produce useful beliefs and therefore contribute to truth for pragmatists. However, usefulness is not synonymous with this constraining process. Potentially, some constrained beliefs might not be useful. Inversely, not all useful beliefs are constrained. Usefulness is different to constraint by subsuming appearances under theories.

- 6.4.1.1 Ethics

Theoretical virtues help solve a problem relating to ethics. Some philosophers are unwilling to associate psychiatric classifications with truth, reality and belief because psychiatric classifications involve values whereas reality is considered value free. For these reasons Murphy (in his 2014a) denies that psychiatric classifications are amenable to belief. Discussing validity in terms of scientific realism, Murphy writes

“we can't validate a diagnosis. We can just correlate it with part of the world's structure” (Murphy 2014a, p.75). In contrast, “causes of mental illness are genuinely out there in the structure of the world, waiting to be discovered” (Murphy 2014a, p.62). For Murphy interest dependent classifications cannot be real whereas interest independent causes are. This view seemingly has echoes in notions of validation and in RDoC, both seeing belief in terms of finding causes. It seemingly also influences social constructivists and commentators like Cushing and Timimi et al who believe autism should be abandoned because it has no identified causes. Other philosophers do not consider this a problem, seeing values and reality as compatible through adopting moral realism (Loughlin & Miles 2014, p.151 ; Thornton 2007, p.179; Wrigley 2007, p.395). If values are part of the world then they are not incompatible with scientific realism. By applying the correct values to correct knowledge of symptoms and their clustering then psychiatric classifications would merit belief. I do not wish to specifically criticise either approach, only note that the causal approach has the disadvantage of not actually delivering realism over psychiatric classification while the moral realism approach requires the problematic stance of moral realism. Instead, I shall provide an alternative approach.

Either approach can be avoided if we consider ethics as having the same status as theoretical virtues. Note that some philosophers demarcate between different types of values in science, such as hot cognitive values, cold cognitive values, internal factors, external factors and decision vectors (see Solomon 2001, p.54-55) but this need not trouble us. For convenience I still describe ethical issues as theoretical virtues, all that matters is showing that we can treat ethical values similarly to other theoretical virtues. This works slightly differently for different positions.

Thinking of ethical virtues as theoretical virtues easily fits pragmatism. Psychiatric classifications which accomplish the aims of pragmatists are useful and thus true. If providing a diagnosis to people who are suffering is a goal of psychiatry then psychiatric classification are theoretically virtuous when they pick out people who are suffering. Thus psychiatric classification exhibiting the theoretical virtue ethics is fully compatible with belief to a pragmatist.

Ethics attains the status of a theoretical virtue to neo-Humeans and neo-Kantians if they are testable, that being, we can gather information about whether they are present in any specific instance. Theoretical virtues do not make a theory worthy

of belief because they provide some special property of belief. Rather, theoretical virtues are simply things scientists value which are sometimes present or are not. Thus hypothetically scientists might decide they valued above all else 'things measuring exactly five metres' and build their theories around this. They design all their theories to be about things measuring exactly five metres. They then test each theory, finding very few theories actually do exhibit this theoretical virtue. Those few theories would attain the status of scientific realism not because things measuring exactly five metres has some special status which makes it more real than anything else. Rather, those theories attain scientific realism because those theories describe something which scientist's value. On this picture we should not claim simplicity is a theoretical virtue because it is mind-independent whereas ethics cannot be a theoretical virtue because it depends on our interests. Rather, both simplicity and ethical concerns are based upon our interests and theories incorporating these can sometimes describe the world. Sometimes the world is in a state which we describe as simplicity; similarly sometimes it is in a state we describe as suffering. Psychiatrists should test their classifications on ethical grounds. They would likely find that homosexuality is not associated with suffering thus does not display these ethical theoretical virtues, whereas some mental illnesses will. Ethics as theoretical virtues pose no special problem to neo-Humeans and neo-Kantians; ethical considerations are theoretical virtues, psychiatric classification need exhibit them to pass an IBE and psychiatric classifications lacking them will not pass an IBE.

6.4.2 Justifying IBE

We have considered four stances on theoretical virtues. Let's now consider different stances on justifying IBEs. All the above four positions consider theoretical virtues as important and as related to truth. Theoretical virtues are required for scientific truths (except perhaps for pragmatism, but they value theoretical virtues). However, theoretical virtues do not guarantee truth. We might believe something exhibits theoretical virtues when actually it does not, our beliefs that a theoretical virtue is present may be mistaken. IBEs are only successful in certain circumstances and we need independent reasons to justify why we believe in the IBE (Lipton 2004, p.139).

Similarly, employing IBE for scientific realism requires “a clear account of when scientific success argues for realism” (Day & Kincaid 1994, p.292). Antirealists also demand IBE's are reliable, but doubt we have such reliable means for truth inferences over IBEs (Lauden 1984, p.136; Stanford 2006, p.205). Let’s consider why IBE need justifying and how this is typically done.

IBEs need justifying because scientists can be mistaken about when theoretical virtues are exhibited. This can occur in two places, phenomena generation and theory generation. Claiming a theoretical virtue is present, as opposed to merely appearing present, is a substantial epistemic claim for neo-Humeans, neo-Kantians, anti-realists and pragmatists. I employ unifications in the below examples, since it is the theoretical virtue which I argue autism exhibits. Before going into detail, it is helpful to consider successful (i.e. generally taken as epistemologically reliable) unification, unsuccessful ones and some uncertain ones.

Galileo unified terrestrial and astronomical phenomena whilst Newton unified terrestrial and astronomical motions (Maxwell 2014, p.136). Lavoisier's chemical revolution resulted in millions of elementary substances being replaced with around one hundred chemical elements (Maxwell 2014, p.136). These unifications largely survived centuries of further development and data generation. Additionally, these unifications seem made under good epistemic conditions; it seems unlikely that, for example, there are large areas of the earth where actually gravity does not apply but we have not noticed this, or that there are millions of chemical elements on earth which we simply have not noticed yet.

Examples of unifications which once seemed epistemologically reliable but now no longer considered so would be the four humors, rational choice theory and behaviourism. In each case, the number of principles, entities or laws required to provide explanations is relatively low, providing substantial unification. However, each one is radically insufficient, only able to account for the evidence with very low stringency. Despite this, each theory was once very popular. These theories were taken as epistemologically reliable but a positive epistemic stance towards these was unjustified; attribution of belief was not made under good epistemic conditions. Even theories which make extremely precise predictions, and whose predictions are carried forward into later theories, may turn out as bad unifications. The Ptolemaic model of the universe could make all the same predictions as the Copernican and accommodate

all the new astronomical evidence supplied by the telescope yet Ptolemaic model was inferior to the Copernican model because the process of adding more celestial spheres was so unconstrained (Forster & Sober 1994, p.14). Notions of an all pervading material ether appear unifying but its removal actually increased unification (Maxwell 2014, p.147).

Unifications can often involve much uncertainty. Physics has moved from corpulses to particles with fields, then just to fields, then just to quantum fields and now moving towards strings, with each step unifying more phenomena under less entities (Maxwell 2014, p.144). However, so far there are no empirical predictions from string theory (Maxwell 2014, p.146). Similarly, superstrings and quantum gravity increase unification but their empirical content is not specific (Falkenburg 2012, p.333) and it is unclear if unifying entities like messenger particles are or are not adhoc in the manner of Ptolmaic astronomy (Falkenburg 2012, p.342). Also, quantum theory may turn out to be much less unifying than currently believed if a hidden variable were discovered, though this would depend upon the nature of the hidden variable. Trying to decide, in advance of future possible evidence, just how much these theories unify requires considerations of the epistemic conditions under which the evidence is gathered and how these evidence is then turned into theories.

Let's first consider phenomena generation. As described in chapter four, phenomena generation consists of reliably gathering data and reliably constructing phenomena from that data. Consider this hypothetical example. A data gathering process resulted in a phenomena being formulated. The theory which accounts for these phenomena is a simple theory, thus contributing to unification. However, assume the data gathering process was unreliable and crucial data was missed. Had that crucial data been detected, the phenomena formulated would have been quite different, being formulated in such a way which did not allow a simple theoretical explanation. Alternatively, suppose instead the superior data gathering process resulted in a second, additional phenomena being formulated. Imagine the theoretical explanation could not cover both phenomena whilst still being a simple theoretical explanation, it needed all sorts of auxiliary extensions. In these hypothetical examples simplicity appeared to be present, strengthening the theoretical virtue unification, but simplicity is only attributed because an unreliable data gathering process missed crucial data which would produce phenomena incompatible with the simple theory.

Additionally, and independent of the reliability of data, flaws may occur in formulating phenomena from the data. Hypothetically, the gathered data might be interpreted badly, perhaps weighing some particular data too heavily without good reason. A superior process of formulating data from phenomena might produce phenomena which is incompatible with the simple theory. Alternatively, perhaps the phenomena interpretation is made via flawed background theories. The resulting phenomena might once again be compatible with a simple theoretical explanation but superior phenomena, interpreted via superior background theories, might not be.

Having generated phenomena, the next step is generating a theory which explains the phenomena. This process can be mistaken in two ways. Firstly, the relationship between the phenomena formulated and the theory which explains it could be flawed. Scientists might believe a theory accommodates particular phenomena when actually it does not. Alternatively, the way in which the theory accommodates the phenomena could be misunderstood. A theory may cover two different phenomena, thus the theory appears to contribute to simplicity. However, those two phenomena might contradict one another, being incompatible and best covered by two separate theories rather than one single simple theory. Similarly, a theory might be taken as simple because it covers certain phenomena, however, there might be other phenomena within the domain the theory covers which could only be accommodated by making the theory more complicated. Scientists should try to cover that other phenomena but perhaps theoretical or social bias mean they miss it. Secondly, theoretical explanations are typically formulated in light of background theories. Those background theories may themselves be false or unreliable. Alternatively, even if those background theories are reliable, errors could occur when employing those background theories to formulate a theoretical explanation of particular phenomena. Here we face theory-laden nature of evidence, though theory-laden evidence is only epistemically problematic when weak background theories are employed or when good background theories are employed badly. A third reason for doubt is the pessimistic meta induction (PMI). Rather than looking at the specifics of reliability of inferences or upon the quality of a specific background theory, PMI takes a broader view. As discussed in chapter one, a theory undergoing sufficient change across its history gives reason to doubt it. Rather than needing to specifically identify unreliable inferences or identify flawed background theories we instead look to

history to legitimately doubt modern theories. An unification may appear present but this was only due to flaws in producing a theory which accounted for the phenomena.

Philosophers counter such concerns by considering the conditions under which the IBE attribute is made. Some situations are amenable to reliable IBE attributions whereas others are not. Philosophers need assess specific theories and establish if the situation under which the IBE attribution was made is reliable. The most well-known means of justifying IBE is the no miracles argument (NMA). NMA argues that there are only two explanations of the success of science, truth or a miracle. Miracles are not allowed in philosophy, therefore truth is only legitimate explanation. Psillos employs NMA to show that IBE is reliable, that "it tends to generate true conclusions when fed with true premises" (Psillos 2011c, p.24). NMA is a "grand IBE... [NMA] aims to defend the reliability of scientific methodology in producing approximately true theories" (Psillos 2011c, p.23). NMA argues the best explanation of miracle like predictively successful scientific theories is that those theories are true. Given this, and given that scientific theories dealing with miracle like evidence are typically established by an IBE, it follows that IBEs over theories producing miracle like evidence are reliable. We need reason to believe IBEs are reliable and Psillos offers the no miracles argument; those IBEs involving miracle like evidence are reliable, those without are not reliable. NMA ensures the reliability of inferences and background theories – they would not produce miracle like predictions unless reliable. We thus only need know a theory makes miracle like predictions, rather than specifically show inferences and background theories are reliable.[32] This position is popular. Chakravartty considers NMA an IBE (2007, p.5), Kitcher also employs the NMA as reliable means for belief (Kitcher 2001, p.166 and p.180 and, related, see Kitcher 1993b, p.160), Massimi sees miracle like predictions as providing "good epistemic conditions" (Massimi 2010, p.15) for further inferences, Giere focuses upon the reliability of our methods (though not just the NMA) (Giere 1999, p.76). Most defenses of IBE focus upon NMA, an argument not generally available in psychiatry (difficulties with NMA in psychiatry are discussed in sections *6.5.2.1* and *6.5.2.4*). We

32 I suggest here that NMA is a way to respond to PMI despite PMI historically being conceptualised to respond to NMA. A general PMI potentially threatens (when supported by historical evidence) all theories. NMA can then be restricted to theories making miracle like predictions. PMI can respond by finding theories based upon miracle like evidence which did undergo historical change. NMA can then be restricted to theories making miracle like predictions and novel predictions (Psillos 1999, p.105). The nature of PMI determines what sort of NMA is required to escape PMI.

discuss alternatives to NMA later.

6.4.3 Epistemic Risk

The final step for belief over scientific theories is a stance on epistemic risk. Both everyday and scientific inference involve the possibility of error, they can turn out false. Consequently, those inferences are only legitimate if they do not violate a personal stance on acceptable epistemic risk. Even claiming a theory is empirically adequate involves risk, meaning anti-realists accept some level of epistemic risk. If belief in empirical adequacy is epistemically acceptable then, argue realists, belief in unobservables can also be epistemically acceptable. Anti-realists respond that unobservables involve more epistemic risk. A realist and anti-realist might fully agree about the level of epistemic risk a theory holds but disagree about whether the theory merits belief, each holding different stances on acceptable levels of epistemic risk. Both realists and anti-realists have discussed the importance of judgments over epistemic risk, such as Chakravartty (2014), Giere (1999, p.185), Kitcher (1993a, p.152) and Psillos, as described below. Epistemic risk has two components.

Firstly, not exceeding epistemic risk depends on the level of epistemic risk involved. Some inferences require much more risk than others. For example, Van Fraassen consider claims over observables to not to exceed his stance on epistemic risk, unlike claims over unobservables. There are other factors than observable vs unobservable which influence levels of epistemic risk. This partly relates to good epistemic conditions since these reduce epistemic risk. Consequently, inferences made under good epistemic conditions are more likely to pass acceptable levels of epistemic risk.

Secondly, a judgement over how much epistemic risk an individual considers as warranted is required. Psillos argues belief in some unobservables only requires slightly more risk than belief in some observables. Since belief in observables is rational then potentially belief in unobservables can also be. In contrast, Van Fraassan argues restricting belief to unobservables is rationally acceptable (2001, p.162). This issue is fairly intractable, potentially down to a judgement which is not particularly amiable to argumentation. Relatedly, in relation to NMA, Psillos believes inferring

that truth rather than empirical adequacy is the best explanation of miracle like success involves a judgment (Psillos 2011b, p.31). He writes that “is truth or empirical adequacy the best explanation [of the success of science]? Not much progress can be made on this front” (Psillos 2011b, p.31). Psillos' believes NMA has some “epistemic force” (Psillos 2011b, p.32) for favouring truth but nothing more. Similarly, Fine (1991, p.82) and Massimi (2012, p.37) claims IBE only provide truth if you assume they work. Realists are typically willing to make that assumption, anti-realists are not.

We have concluded outlining my approach to the possible grounds of belief. Let’s apply this to psychiatry then autism.

6.4.4 Validity

Let’s now reconsider existing approaches to belief within philosophy of psychiatry in light of the above claims about theoretical virtues, justifying inference to the best explanation and attitudes towards epistemic risk.

Most psychiatrists approach questions over reality of psychiatric classification through notions of validity. What validity means is rarely adequately described but Kendell & Jablensky famously tried make explicit an account of what validation accomplishes. Kendell & Jablensky believe validity has two important qualities, both absent in non-valid psychiatric syndromes. Something is either valid or not valid, partial validity not being possible, also, validity is independent of context (2003, p.10). In contrast, utility comes in varying levels of degradation and can vary with context (2003, p.10). These notions seem intuitive, something either is true or is not true and truth is a product of the world rather than subjective human purposes. These ideas seem implicit in most accounts of validity employed by psychiatrists. Our above discussion and the discussion in chapter four undermines these claims, as I now show.

Kendell & Jablensky argue a valid syndrome is fully valid, meaning it merits full rather than partial belief. “Validity, as we define it, is an invariant characteristic of a diagnostic category. There may be considerable uncertainty about the category's validity because the relevant empirical information is lacking, but in principle a category cannot be partly valid” (2003, p. 10). However, Kendell & Jablensky are mistaken to see validity as all or nothing. This notion of validity is untenable for two

reasons. As described above, theoretical virtues are required for belief in scientific concepts. More theoretically virtuous means greater belief is entailed. Unless a theory was as theoretically virtuous as conceivably possible (for neo-Humeans this is where it fully reflected the objective structure of regularities, for neo-Kantians it reached the absolute limit of rational inquiry, for pragmatists it reach absolute usefulness), then truth claims vary in degrees. Additionally, as chapter four shows, science deals with idealised models so only provides approximate descriptions formulated to our interests. Science cannot provide context independent absolute truth, only interest dependent approximate truths, as I now show. Psillos writes that "[d]emanding the exact truth in science would amount to demanding the exclusion of all approximations, simplifications, idealisations, approximate derivations, sources of error in measurement and calculation" (Psillos 1999, p.276). A strict dichotomy between valid and invalid is unsustainable. Truth in science is not invariant, the strongest scientists can achieve is degrees of approximate truth, even in principle. Similarly, we must consider classifications as having varying degrees of approximate validation, from very high to very low. Also, Psillos writes that "there is no reason to think that empirical evidence cannot lend a different credence to the several theoretical constituents of the theory. Nor is there any reason to think that all parts of a theory are equally well supported by the evidence" (1999, p. 125). 1700's chemistry falsely postulated that an element named phlogiston was emitted during combustion. Most descriptions and predictions phlogiston theory entailed had strong evidential support even though the element phlogiston was weakly supported by evidence (1999, p.291). The same is true of, for example, autism. Arguably, the idea that autistic people have weak social skills has immense evidential support and should be considered approximately true whereas the idea that there are theory of mind deficits in autism is conceptually questionable and weakly supported by the evidence (Maise 2013, p.182), making it only weakly approximately true or potentially completely false. Establishing that a classification merits some belief does not then confer full belief to all symptoms, whereas Kendell & Jablensky take syndromes as either fully meriting belief or not meriting belief at all.

Kendell & Jablensky believe valid syndromes are context free. "Validity does not depend on the context" (2003, p. 10). However, validity must depend on context. Idealised models are simplifications, scientists reduce the number of causes by being

selective and this selectivity typically is based upon purpose. Lipton writes that

> "the causes that explain depend on our interests... take a particular eclipse. The number of causal factors is enormous... We do not explain the eclipse *tout court*, but only why it lasted as long as it did, or why it was so partial, or why it was not visible from a certain place. Which aspect we ask about depends on our interests, and reduces the number of causal factors we need consider for any particular phenomenon, since there will be many causes of the eclipse that are not, for example, causes of its duration" (2004, p. 33).

Which specific causes a model describes depends on what we explain. Imagine we identified the biological causes of low social skills in autistic people. This would not provide full causal information. Exactly how any autistic person manifests low social skills will be influenced by many other causal factors such as individual personality, previous experiences and, in specific manifestations, who they are talking to and why. The model needs describe only some rather than all causes, meaning the model provides only approximate truths about reality, and which causes the model describes depend upon our interests, as described in chapters four and five. The constraint is how greatly we maximise theoretical virtues, choosing theories based both upon how well they have the theoretical virtue of describing causes and how well they do on many other theoretical virtues. A theory is not automatically superior if describing causes; this depends upon the wider context of how much the theory adds to a simple system of laws. Dependence on a context is not a barrier to approximate truth. Kendell & Jablensky seemingly see knowledge of reality as being of absolute reality whereby something is either true or is not and this is independent of our interests. However, science can only partially describe reality and which parts a theory describes depends upon our interests, leaving Kendell & Jablensky's claims over what qualities we should ascribe to valid psychiatric syndromes as untenable.

6.5 Arguments for belief

6.5.1 Autism and inference to the best explanation

I now provide my own arguments for belief over autism. Let's take the first step, establishing autism as the best explanation, before later establishing reliability of IBE attributions. IBEs are established through appeal to theoretical virtues. Of various competing explanations we deem one as the best explanation because it exemplifies qualities that good explanations should have. Most IBE emphasise unification or causation (Day & Kincaird 1994, p.275), let's start with unifications.[33]

- 6.5.1.1 Unification and laws

The most powerful IBE for autism is unification. Chapter four argued autism could be understood as unifying diverse phenomena. Symptoms co-occur and we explain this co-occurrence by appealing to autism. I discussed Kitcher's notion of unification, let's consider a more general account. Lipton describes unification as covering scope, simplicity and consilience (Lipton 2004, p.138-139) whereas Psillos considers unification and parsimony as separate, though very similar, virtues (Psillos 2009d, p.184-185). Simplicity is simplifying our explanatory picture of the world. Rather than appealing to multiple explanations we might instead be able to appeal to fewer or a single explanation for particular phenomena. However, we might simplify too much, grouping together much diversity under one theory. So we need more than just simplicity but also strength. This partially comes from scope of the claims. Newton's gravitation is universal so gains great strength from scope. This has obvious relationship with simplicity. However, this may lead to a single theory covering all phenomena. So the third criteria is stringency, the statistical strength of probabilistic relationships of phenomena. Some phenomena co-occur frequently, some less frequently. Additionally, the number of phenomena increases strength. The best balance between simplicity, strength and stringency maximises unity.

Let's consider how this works in psychiatry. We ideally take all symptoms described by psychiatry and put them into a unified system, all symptoms being

33 Lipton also lists mechanism and background belief (Lipton 2004, p.138-139) and Psillos also lists completeness, importance, parsimony and precision (Psillos 2009d, p.184-185). Many, though not all of these, are substantially related to unification and causation.

accounted for by a classification. We maximise simplicity by employing as few classifications as possible. We also wish those classifications to cover as many symptoms as possible since this increases strength. By employing fewer classifications each classification can cover more symptoms. Here reducing the number of classifications (simplicity) increases scope of the resulting classification (strength). However, this leads to classifications covering symptoms which co-occur weakly, lacking in stringency. Hence we need employ as few classifications as possible, those classifications need cover as many symptoms as possible and those symptoms should co-occur as strongly as possible. A psychiatric classification unifies well when it covers a very large number of diverse phenomena which co-occur with high stringency. Belief in a psychiatric classification, under unification, is where it contributes to a system of simple, strong and stringent classifications.

An individual classification has the theoretical virtue unification to the degree which it contributes to a unified system. Autism covers a very large number of symptoms, most of which are highly unrelated. Social-emotional reciprocity, low eye contact, deficient body language, abnormal, facial expressions, lack of shared imaginative play, stereotyped movements, idiosyncratic phrases, adherence to routines, excessively circumscribed interests, disliking unexpected changes, rigid thinking, extremely variable intelligence levels, sensory issues, clumsiness, difficulty with planning are all mentioned by DSM-5 (APA 2013, p50-55) whilst other symptoms associated with autism but are not mentioned by DSM-5 include obsessive interests, peculiar visual perceptions, arrogance and good memory.[34] There are many classifications much worse at unifications. Social anxiety disorder has only two main symptoms, far less than autism. Also, the symptoms seem closely related. Being anxious in social situations and fear of social situations are highly related; fear and anxiety are very similar and one refers to an instance of socialising, the other to future socialising. Whilst not identical, they look like different aspects of the same symptom. On both these grounds social anxiety disorder unifies weakly. Many other psychiatric classifications also do poorly on unifications, such as personality disorder and milder learning difficulties. Covering so few symptoms means they unify weakly. Some classifications do better than this. For example, depression has nine symptoms in

34 Note that symptoms can causally influencing one another (Zachar 2014, p.131). Repetitive behaviour in autistic people likely causally contributes to low social skills but it seems implausible that one entirely causes the other without contribution from common causes.

DSM, meaning depression has some strength. However, very diverse symptom patterns can be diagnosed as depression, as many complain. Depression is weak statistically since, Horwitz claims, any five symptoms grants diagnosis (2014, p.219, also p.222) though one of those symptoms must be depressed mood or loss of interest or pleasure (APA 2013, p.160). By contrast, diagnosis of autism require multiple symptoms from three different categories (now only two different categories since DSM-5), making diagnostic criteria for autism much more stringent than depression. Having ten random symptoms of autism does not mean someone is autistic unless some of those symptoms are very specific ones from multiple categories. By contrast, having five random symptoms of depression (providing one is depression or loss of interest) means someone has depression. Autism does well on unifications because it has strength, covering many symptoms, and it has stringency, being specific in how those symptoms must co-occur.

Usually described in terms of validity or internal validity, statistical studies show interrelations between the multiplicity of symptoms of autism. Disagreement exists over exactly how symptom clusters correlate. DSM-IV autism had three domains, social, communicative and RRBI [repetitive, restrictive behaviours and interests] whereas DSM-5 has two domains, social communication (merging social and communication) and RRBI. Most studies favour a two domain approach:

> "studies give weight to the suggestion that social and communication symptoms should be combined conceptually into one core domain of impairment, distinct from the restricted/repetitive behaviour domain" (Kuenssberg, McKenzie & Jones 2011, p.2190; also Mandy, Charman & Skuse 2012, p.49)

Some disagree, accepting that "social and communication impairments relate more closely together than either does with RRBIs" (Dworzynski et al 2009, p.1208) but "overlap between social and communicative impairments was not complete" (Dworzynski et al 2009, p.1208). Though debated exactly how symptoms relate, all studies show wide ranging symptoms do co-occur together. The theoretical virtue of unification has been tested and has passed, meaning neo-Humeans and neo-Kantians can see this part of the world as has having a degree of unity.

For a stronger correlation we would ideally desire autism maps onto a single factor, rather than two or three factors as described above. However, if reality takes a form where a set of behaviour (from which symptoms are derived) co-occur regularly when measuring many individuals, but which specific behaviour any given individual exhibits from that set has some variance, then reality might not best be modeled as a single factor. Also, autism is an idealised law which describes probabilistic co-occurrence of symptoms which abstracts away many specific details of individuals with autism; for reasons of age, of environment, of how the specific symptoms an individual has interact together, of how the specific symptoms an individual has interacts with non-pathological traits, we should not expect manifestations of autism to correspond with the high level law. Consequently, we should not assume greater social and communicative impairment means greater impairment of RRBI. Thus, "it maybe unwise to think that severity of symptoms in autism is always highly correlated with level of functioning" (Szatmari et al, 2002, p.472; also Dworzynski et al 2009, p.120;8 Kamp-Becker et al 2009, p.568). Despite all this, individuals are to varying degrees impaired in very different domains and autism can describe how occurrences of symptoms probabilistically relate. We only need autism as a high level model to have stringency, describing probabilistic relationships between symptoms which provide guidance on which symptoms an individual might exhibit.

Thinking autism need lie only on a single factor may stem from mistaken views on causation. If autism had a single cause then we might expect that when the cause is stronger then individuals will exhibit more autism, i.e. more symptoms or symptoms manifest more severely. However, modern causal investigation shows an inherently multi-factorial element. Notions of stronger vs weaker causes need replacing with notions that a wide variety of causes can result in a wide variety of symptoms. Maximising information is only best served by employing a single factor when the world takes particular forms, ones which rarely occur in psychiatry.

The history of autism, rationally reconstructed, shows various ways of trying to balance strength, simplicity and stringency. We can see psychiatry of 1925 to 1943 as having a set of laws, ones which inadequately described all phenomena by leaving a gap within the described probabilistic relationships. Consequently, Kanner created a new scientific law in the form of autism, covering part of that previously not described phenomena. This increased strength and stringency, at an acceptable cost of simplicity

by adding a new law. Between 1943 to 1978 there were two main approaches, those who employed childhood schizophrenia and mental retardation and those who just employed childhood schizophrenia, its subtypes (autism, children with circumscribed interests and symbiotic psychosis) and mental retardation. We can imagine this as a debate over strength, simplicity and stringency, psychiatrists each trying to get closer to the ideal balance. Both approaches had similar strength, since both approaches could diagnose the same people. Each approach differed on simplicity and stringency. Those employing the subtypes of childhood schizophrenia did so at the cost of simplicity, employing more laws than is needed. However, plausibly they gained in stringency, the additional classifications allowing more specific probabilistic relationships to be applied. Those just employing childhood schizophrenia and mental retardation gained in simplicity but lost in stringency. I make no judgements over who was correct, merely that we can rationally reconstruct them as two separate subjective judgements attempting to reach that best balance between strength, simplicity and stringency. Kolvin's and Rutter's approach may have gained in simplicity but vastly lost strength, being unable to cover many people who were previously covered. It did gain in some stringency because the symptom pattern of early onset schizophrenia, quite different to that of the majority of childhood schizophrenia and its subtypes, is now associated with adult schizophrenia. DSM-III-R is quite similar to Kanner's approach, an overarching pervasive developmental disorders whereby PDD-NOS covers higher functioning individuals whilst autism covers more impaired individuals. It thus combines simplicity and stringency. DSM-IV is even closer to Kanner's position, the overarching category pervasive developmental disorders with specific subtypes of PDD-NOS, Asperger's syndrome, autism, childhood disintegrative disorder and Retts disorder. Simplicity is lost but stringency is gained. DSM-5 removed those subtypes, becoming similar to Bender's approach, an overarching category of autism without demarcating specific subtypes. Simplicity has been gained at the cost of stringency. Both DSM-IV and DSM-5 significantly balances strength, simplicity and stringency.

- 6.5.1.2 Causes

The other major theoretical virtue is causation. If one regularity follows another to a sufficient probabilistic degree then we consider the first regularity to cause the second one. For Psillos (and Salmon and Kitcher) causes are probabilistic relationships of regularities demarcated by laws. We establish the laws and then see which causes are derived. Finding causes is different to co-occurrence of regularities which I described above. Symptoms of autism co-occur together, but they do not seem to follow one another (i.e. the regularity low social skills are not followed by the regularity low eye contact). In contrast, causation of psychiatric classifications would be where one regularity (gene, brain structure, psychological state) precedes the co-occurrence of symptoms. Establishing the theoretical virtue causation, whereby certain types of relationships between regularities are established, increases belief in a scientific theory.

Some psychiatric classifications gain substantial belief from the theoretical virtue causation. Down's syndrome and Huntingdon's have strong probabilistic relationships between one set of very regularly co-occurring regularities (symptoms) and biological regularities (the genes). These are considered so strong to make the gene a cause. These are good reasons to believe in Down's syndrome and Huntingdon's.

Unfortunately, as discussed in chapter four, such causes are extremely rare in psychiatry. Very few psychiatric classifications gain much belief from the theoretical virtue causes. Imagine a psychiatric classification was associated with, say, a hundred genes all between 1% to 2% effect size. Depending on one's stance towards belief, either these would provide no belief via the theoretical virtue of causes or they would provide very little belief. Given that modern evidence suggests most causes have extremely small effect size in psychiatry, establishing belief on causes through a theoretical virtues approach looks very unpromising except for a limited number of psychiatric classifications.

- 6.5.1.3 Ethics

To pass an IBE a psychiatric classification must exhibit ethical theoretical virtues. I have previously outlined in chapter five how autism accomplished various ethical

goals of psychiatry. The diverse symptoms of autism make it a strong likelihood autism is not just a reaction to society, hence autism passes this IBE whereas personality disorders would struggle to do so. Psychiatric classifications need also cause suffering. Autism has a very high statistical probability of suffering meaning autism passes this criteria whereas some classifications would struggle to. Psychiatric classification must also not be variations on normal behaviour. The diversity of the symptoms means autism passes the IBE in a manner which mild learning disability would not. Note that this would not necessarily be so with boarderline cases of autism, here I only refer to individuals who meet DSM criteria. Autism exhibits theoretical virtues of ethics more than many other psychiatric classifications.

- 6.5.1.4 Other inference to the best explanation arguments

There are other types of IBEs, usually considered an indirect way of establishing causes. Firstly, a psychiatric classification having very particular course, such as the progressive degeneration exhibited in Huntingdon's. In this case we value strength, showing how Huntingdon's changes. This links one state with another state, covering more phenomena. We also value stringency since Huntingdon's is extremely predictive. The course of autism can change as individual’s age, though nothing as striking as Huntingdon's. Secondly, response to drugs, specific response to specific drug allowing inference to shared biology. Here we value strength by linking drugs with end results, plus we value stringency though this depends upon the specificity of predictions. This is currently inapplicable to autism. Thirdly, family studies, whereby co-occurrence of psychiatric illness within families suggests underlying shared causes. Here we value strength by furthering the web of probabilistic relationships. We also value stringency if those probabilities are strong. Such family studies plausibly should contribute to belief since autism has a heritability of 80% (Lichtenstein et al 2010). All these are potential means for IBEs though how strong each need be for IBE inferences needs arguing for. We also might employ multiple IBE's for each psychiatric classification. However, each IBE is rare in psychiatry and, as discussed in chapter four, we should not assume reality is composed of currently undiscovered things existing in these forms. Additionally, the next section shows how valuing these can

sometimes reduce the IBE's strength. Finally, the IBE attribution needs justifying, as discussed in section 6.5.2. It would need be shown how these claims would be justified.

- 6.5.1.5 Balancing IBEs

Unification and causation are often in tension in psychiatry. A psychiatric classification has the theoretical virtue of unification if it covers many symptoms which co-occur stringently and it has the theoretical virtue of causation if it has an identified (or inferred) cause. Sometimes these are fully compatible, such as with Down's syndrome which has a very strong cause and relatively little variation in symptomatology. However, this is rarely the case in psychiatry. Most currently identified causes in psychiatry only have an extremely small effect size for existing psychiatric classifications.

Psychiatry could strengthen causes by modifying classifications. We could split up and shrink psychiatric classifications, say split autism into numerous sub-classifications and see if any genes have higher probabilistic relationships to each sub-classification. Assume this worked, the causal basis of each subclassification being stronger than currently exists for autism, many genes going from 1.2% to, say, 5% or 10% effect size. This strengthens the theoretical virtue causation, contributing to belief. However, doing this means employing more classifications, thereby decreasing simplicity. Remember that there is a tension since “simplicity and strength [including stringency] pull in opposite directions” (Psillos 2002, p.149). Simplicity demands fewer laws but this means those laws become more general, making them less stringent. By employing more laws, we can describe probabilistic relationships of phenomena (including causal connection) with higher stringency. So in our hypothetical example we need compare the strength gained (linked to more causes) and the stringency gained (strong probabilistic associations between those causes and the classifications) with how much simplicity has been sacrificed. All this depends on how many new classifications are created and how greatly (both number and degree of probabilistic associations) they link to causes. Ideally, we would need formulate relatively few classifications and each linked to causes of relatively high effect size. I

shall not discuss precise figures, this all depends upon a judgment. Instead, I point out that focusing heavily on causes could result in reducing overall balance of theoretical virtues.

The same could occur when inferring causes from the form of the psychiatric classification. For example, Kendell & Jablensky famously argued that, in the absence of identified causes, psychiatric classifications can be valid when "demonstrated to be an entity, separated from neighbouring syndromes and normality by a zone of rarity" (2003, p.8). A zone of rarity is where symptoms of a syndrome have little overlap with other syndromes; a syndrome is valid if few symptoms in that syndrome are found in any other syndrome. This seems similar to notions of sensitivity and specificity. This then allows an inference that the psychiatric classification has a unique biology, different to other psychiatric classification (2003, p.8). However, trying to increase zones of rarity, trying to ensure all those diagnosed have similar symptoms and those symptoms do not occur in other psychiatric classification, can reduce unifications. Zachar writes that

> "good diagnostic criteria are [typically seen as] both sensitive indicators of a disorder and specific indicators of a disorder. For this reason a symptom such as irritability is not an ideal criterion for depression because it is sensitive to depression but not specific to depression. Highly anxious people are also irritable. . . overlapping symptoms contribute to our understanding of how complicated cases might develop. When. . . [overlapping] symptoms are ignored the gaps between clusters look larger (or more real) than they are" (2014, p.

Ignoring symptoms when modelling to produce zones of rarity reduces the number of symptoms employed and thus weakens the law. Placing irritability as a symptom of depression will increase the number of symptoms and thus increase the strength of the law (providing this does not negatively affect stringency). Scientists often prefer to capture more causes by not seeking strictly demarcated entities. Many scientific entities have zones of rarity only in an idealised sense, only having a unique set of causes if scientists ignore many causes. For example, systems biologists produce "models [that] try to ignore the complexities at the lower level, in order to capture the

general features of a system's dynamic" (MacLeod & Nersessian 2013, p. 553). The complexity means the model's power is increased by not seeking strictly demarcated entities. Attempting to maximise both laws and zones of rarity when formulating classifications will, in many situations, produce a conflict. The strength of laws comes from the number, diversity and unrelatedness of statistically associated phenomena. Greater diversity often means greater overlapping causes, reducing the zone of rarity. Where establishing a zone of rarity requires ignoring many symptoms then enforcing zones of rarity will likely not be the best explanation.

6.5.2 Justifying inference to the best explanation attributions

- 6.5.2.1 Reliability

We now need establish the second step of scientific realism, justifying IBE attributions. If we attribute autism as the best explanation then why believe such attributions are reliable? Specifically, we might decide a particular explanation is the best explanation when actually it is not. Therefore, we need reason to believe such attributions actually are the best explanation.

Many scientific realists employ NMA to justify IBE, a condition under which IBE attributions are reliable, but we need seek an alternative for autism. In some instances NMA is highly applicable in psychiatry. For instance, knowing someone has HTT gene means they either die early or develop Huntingdon's seems miracle like. Equally, the near perfect sensitivity and specificity of causes to Down's syndrome seems miracle like. Finally, perhaps sufficient responsiveness to a medication might be miracle like (Lithium might be a suitable example). Such situations are extremely rare in psychiatry. Beyond such instances, perhaps NMA could still grant some very weak form of approximate truth for since, referring to NMA, Kendler ponders, "would these advances [measurement, psychopharmacology, neurobiology and genetics] have been possible if all our attempts as psychiatry were, at a fundamental level, deeply flawed" (Kendler 2012b, p.100). This argument has potential, especially if we also consider the success of classifications at systematizing phenomena. However, the level of success here is, with rare exception, far below miracle like predictions. Weak

success delivers weak realism, far below what most realists aim for (Lauden 1984, p.115). Perhaps many philosophers of psychiatry would welcome such weak realism, seeing it as better than existing high levels of scepticism. However, I will argue for an alternative to NMA which can deliver a moderate, rather than weak, scientific realism for autism.

Perhaps the most secure basis for IBE attributions is observability. A traditional battleground for scientific realism was observablility vs non-observablility, however, any divide between observable vs non-observable cannot be made on a-theoretical vs theoretical grounds (Feyerabend 1975, p.212; Hempel 1973, p.70; Kuhn 1996, p.4; Popper 1959, p.106; Quine, 1951). A more promising approach to observables is minimising epistemic risk. Van Fraassen seeks to minimise epistemic risk by extending belief only to empirical adequacy, scientific entities only accounting for observables rather than merit belief. However,

> "[i]n either case we stick our neck out: empirical adequacy goes far beyond what we can know at any given time. (All the results of measurement are not in; they will never all be in; and in any case, we won't measure everything that can be measured). Nonetheless there is a difference: the assertion of empirical adequacy is a great deal weaker than the assertion of truth" (Van Fraassen 1980, p.69).

Claims about observables involve epistemic risk but less than realism, a claim realists agree on (Azzouni 2004, p.374; Magnus 2003, p.466; Psillos 1999, p.200). They disagree with Van Fraassen over exactly how much epistemic risk unobservables involve and how much epistemic risk is acceptable. Let's consider why observables involve less epistemic risk.

Psillos argues belief in scientific claims is justified by getting background theories correct (Psillos 1999, p.189). Observables need interpreting by background theories just like unobservables do (Psillos 1999, p.31). Inferences for observables are typically epistemically safe because observables have limited ontological options of what they can be, of the form they can take. Discussing why Bridge (a card game) players are successful in their inferences, Azzouni writes

> "[w]hen we *already have* an ontological description of a collection of objects (cards, people, etc.) and a *restricted range of properties we can attribute to them*, inference to properties can be closely keyed to the success of the inferences... restriction to *observable entities* restrict *ontological options* – both of what things can exist, and what sort of properties they can have: we can trust inferences about distributions *of cards* – if successful – because we are severely constrained in the ontological options that it's reasonable to even consider" (2004, p.381 emphasis original).

Pointing to Azzouni's use of the work 'reasonable', Psillos in response writes "what options it is reasonable to consider when it comes to observables depends on several background theories and assumptions" (Psillos 2009b, p.97). Observables are epistemically justified because they are generally interpreted by good background theories and assumptions (Psillos 2009b, p.88) and sometimes this is also true of unobservables (Psillos 2009b, p.98). We must establish how good the background theories and assumptions employed for autism are.

There are two stages of epistemic risk, firstly inferring phenomena from data and secondly inferring theories from phenomena. Below I highlight epistemic risk found at both stages, it is a representative rather than exhaustive account of epistemic risk in science – different sciences face different challenges. I show how problematic epistemic risk is in physics (thus why Psillos needs NMA) and show how autism faces vastly reduced epistemic risk. I start with the epistemic risk of inferring phenomena.

- *6.5.2.2 Phenomena*

From phenomena we infer theoretical claims but these inferences often require phenomena take a very particular form, one requiring extremely high precision which is highly epistemically risky. For example, "getting neutrinos to produce records which are accessible to the human sensory system will require a great deal of subtle contrivance and that the causal chain running from the neutrinos to such records will be long and complex" (Bogen & Woodward 1988, p.320). Scientists do not simply

detect neutrinos but take very specific phenomena as, via a long causal chain, being produced by neutrinos.

Establishing phenomena can involve very complicated experiments. For example,

> "in the neutral current experiments conducted at CERN, researchers followed the strategy of first getting the neutrinos to interact with matter to produce charged particles and then getting the charged particles to interact with a standard detector (in this case, a bubble chamber) in such a way as to produce records which were visually detectable" (Bogen & Woodward 1988, p.320).

There is clear epistemic risk when building complicated experiments to study phenomena, ones not associated with many psychiatric symptoms.

Relatedly is the need for controlling confounding factors. Phenomena are the product of multiple causes, many of which need controlling when seeking precision. Sciences like physics involve attempts to control these via complicated experimental set-ups. For example, when studying electrical activity in nerve membranes,

> "[to] control the nerve's temperature and shield the recording equipment from ambient heat, Hill sealed them in the core of a double walled container. To see how the nerve behaved at 0C, he filled the space between the walls with crushed ice. For experiments at higher temperatures he used paraffin oil. To bring the inner temperature to within a desired range above 0C he pumped in air from the laboratory. For lower temperature experiments he used oxygen. When the core was cooled slowly or when oxygen was introduced, the equipment sometimes recorded 'curious fluctuations' in nerve temperature. Hill supposed the fluctuations were artifacts of warming caused by little rainstorms in the chamber. The rainstorms occurred when the charge on the wire he used to stimulate the nerve initiated condensation in the supersaturated air surrounding the thermopile. Assuming that the gases he used to manipulate the temperature inside the core contributed to

> supersaturation, he took pains to remove moisture from his oxygen and adjusted the temperature of the room to keep the air he pumped into the container from getting colder than the air in the core" (Bogen 2011, p.15).

Once again, all this involves many epistemic challenges and we simply need not do this for most psychiatric symptoms.

We need be selective over data when constructing phenomena. Physics often needs very high selectivity of data when producing phenomena. For example,

> "physicists successfully detected the phenomena of weak neutral currents in 1973... The data obtained at CERN consisted of approximately 290,000 bubble chambers photographs of which roughly 100 were thought to provide evidence for the presence of neutral currents... at NAL... 8 of approximately 330 records were interpreted as evidence for neutral currents" (Bogen & Woodward 1988, p.315).

The level of data which is discarded is immense and the level of data considered legitimate is tiny. This contains huge epistemic risk and there seems nothing analogous in psychiatry.

All this creates great difficulty demarcating phenomena. For example,

> "identifying WNC [Weak Neutral Current], however, is highly problematic. In spark chamber experiments, where the production and detection of muons is spatially separated, wide-angle muons could escape the detector. In bubble chamber experiments muons could get stuck in the shielding of the chamber. If one didn't take extra care in estimating these undetected but nevertheless present muons, one could end up counting as WNC events what in fact were merely charged current events. Moreover, in bubble chamber experiments, incoming neutrinos could knock off neutrons within the shielding, which in turn would propagate into the chamber where they would scatter of hadrons, thus emulating WNC events" (Schindler 2011, p.49).

This inference from data to phenomena here carries immense epistemic risk. There is very high possibility errors could be made when applying this process. There seems little analogous in psychiatry.

The phenomena scientists employ are often highly idealised, conceptually constructed from potentially questionable theories. The

> "phenomena scientists investigate are often the end product of these series of intermediate steps, at quite a distance from the original data. Not only can they be unobservable, as Bogen and Woodward have rightly pointed out; they may also require a significant amount of *conceptual construction*" (Massimi 2008, p.13 emphasis original).

Heavily discussed in chapters four and five, phenomena are idealised regularities that only indirectly describe data, constrained by "what could be modelled managably and reliably" (MacLeod & Nersessian 2013, p.545), formulated on considerations like tractability, generality and applicability. Also, theories often heavily influence phenomena interpretation. The greater the idealisation and greater reliance upon false or heavily idealised theories means greater epistemic risk.

Neutrinos are very inaccessible, a long causal chain lying between them and their effects. The causal path from neutrinos to their effects involves interaction of vast numbers of other causal factors. Consequently, we need account for these phenomena in two steps. Firstly, highly specialized experiments which minimize these other effects. Secondly, since these effects can never be fully dispensed with, only some experimental results (data) are taken as relevant to the effects of neutrinos (phenomena). We typically need extremely precise measurements when deciding relevancy of data for precisely demarcated phenomena. We need produce extremely precise measurements and judge which precisely measured data is relevant for producing precise phenomena. "Quite plainly we are more likely to be wrong... when we attempt precise specification of magnitude" (Kitcher 1993a, p.140). Additionally, we may interpret phenomena using highly abstract, idealised and epistemologically insecure background theories when deriving phenomena. Phenomena can potentially have immense epistemic risk, as highlighted by examples from physics. Let's now

consider psychiatry in detail.

Entities and their laws are typically very imprecisely defined in psychiatry, removing the need for precise phenomena. The level of precise information indirectly gathered is radically lower for formulating low social skills compared to weak neutral currents. We might need considerable complexity for very specific types of memory or very technical notions of thought disorders but not for most symptoms of autism. In psychiatry only rough figures are needed, low social skills covering a very wide range of behaviour. Rather than requiring the exact figure 4.82 on a particular scale to have low social skills the score merely need fall within a particular range. Consequently, we can employ imprecise observations, interviews and statistical studies, rather than highly specialised experiments with associated high epistemic risk. Establishing phenomena for demarcating autism from schizophrenia need not be precisely formulated, does not need specialised experimental set-ups, need not discard ninety nine percent of data gathered, need not be so stringent over countering causal influence of unwanted factors. Minute causal factors need controlling for, including highly selectivity of data, when dealing with some of the phenomena studied in physics. In psychiatry we need not control for the individual being male, it being winter, he has four friends rather than five, is working class, he lives in Hull rather than Bristol. These factors can causally influence data but need not be controlled for when formulating symptoms.

Relatedly, we often have much greater access to phenomena in psychiatry than in physics. Consider social interaction. Most humans engage in many conversations throughout their day and speak with perhaps thousands of individuals across their life. These conversations can occur in many different environments (work, school, home), differ from lasting seconds to hours, occur among many different people and about many different subjects. Most conversations successfully transmit some portion of the information required. Doing so requires a whole range of assumptions about comprehensibility of language, shared semantic understanding, psychological states, intentionality and much else besides. However, none of these seem particularly problematic for most instances of conversations. Humans have an ability to “reliably and robustly sustain interaction with other's intensional states” (Michael & MacLeod 2013, p.226). Sometimes, however, interactions are not sustained or are abnormal. Most humans will, given sufficient evidence, be aware this occurred. Most humans

can intuitively form conversations and intuitively detect abnormal conversations. Zachar argues this more generally.

> "Consider temperature, weight and length. We have some native abilities to detect these phenomena, however crude. Something similar, albeit more conceptually complex may be true for detecting aberrant behavioural patterns (supplemented by some widely shared assumptions about normal functioning)" (Zacher 2012, p.31).

The role of empathy and intuitive understanding has been emphasised by Avramides (2013, p.279) and Potter (2013, p.302). Woodward argues causal reasoning is robust and reliable in everyday contexts (Woodward 2012, p.962), similarly so for social interactions. Much less conceptual construction or highly abstract idealisation is required than can occur in physics. Also, good access is still present when applying specialized interviews and questionnaires to increase the accuracy of our judgments. Additionally, evidence gathered under a wide range of circumstances is safer than single experiments (Stanford 2011, p.893). This level of access conveys substantial epistemic reliability. Also, we need only use implicit rather than detailed theoretical commitments to demarcate social skills. For low social skills, complicated theories are rarely employed (Ochs et al 2004, p.160). The above problems might be inapplicable for some psychiatric phenomena. For example, most psychiatrists either never or rarely have hallucinations, meaning levels of access are far lower than social skills. Similarly, theories used for hallucinations are much more epistemically problematic. Additionally, demarcating hallucinations from delusions or thought disorders is difficult. However, the epistemic risk when constructing the phenomena low social skills is much lower than for many phenomena in physics.

- *6.5.2.3 Entities*

We have considered symptoms (phenomena) in psychiatry. I have argued that some phenomena in psychiatry involve far less epistemic risk than can occur in physics. Let's consider the second inference, from phenomena to theoretical entities.

Indirect access of scientific entities increases epistemic risk. For example, "the causal chain running from the neutrinos to such records will be long and complex" (Bogen & Woodward 1988, p.320). Kitcher observes, "[q]uite plainly we are more likely to be wrong when we… make claims about things that are causally remote from us" (Kitcher 1993a, p.140). In contrast, inference from symptoms (phenomena) to autism (theory) seems much smaller, the causal chain between theory and phenomena being much smaller than often present in physics.

The inference from phenomena to theory can involve high epistemic risk. We need theories to differentiate electrons from neutrinos (Psillos 1999, p.256). Here instances of phenomena have only very minor differences to which we apply very highly idealised theories, claiming each almost identical phenomena is (via a long and abstract causal chain) the product of two different particles. Also, there may be plausible alternative ways of demarcating entities. Eliminating alternatives will often depend upon assumptions, often highly questionable ones (Stanford 2006, p.41). For example, modern physics often relies upon very specific notions of causation but there may be alternatives not yet conceived of (Stanford 2006, p.171). In contrast, psychiatric theories are loose probabilistic relationships, much less precise than probabilistic claims typically found in physics. Psychiatric classifications as probabilistic theories tells us certain behaviour regularly co-occurs and from this common causes (typically in an INUS sense) can be assigned. Providing the co-occurrences are strong then this is a relatively uncontroversial way to do science. It merely infers an entity consisting of a loose collection of unknown co-occurring properties (biological, psychological) which cause the symptoms. Also, imagine if the rough statistical probabilities assigned to autism were slightly, though not massively, false. This means the classification is less ideal at systematising the world than possible, reducing the level of approximate truth over the entity. Contrast this with physics where we demarcate one very precise electron from another very precise neutron, requiring very precise theories and assumptions. Mistaken laws in physics could be utterly fatal for demarcating electrons from neutrons given the specificity of the claims involved. The epistemic barrier in psychiatry for entity inference is much lower compared with physics.

We need also conceptually construct entities. In psychiatry we consider entities responsible for a range of behaviour. By specifying a very loose range of behaviour

we equally can specify a very loose entity. In physics we believe very specific entities are responsible for very specific phenomena. This typically involves very high levels of idealization and assumptions. For example, extremely high levels of idealization and selectivity of data creates difficulty "confidently ascertain[ing] what its [physics'] basic quantities comprise" (Wilson 2010, p.994). Additionally, closeness to common sense is epistemically advantageous (Stanford 2006, p.200) but physics is often radically different to common sense. Huge conceptual construction increase epistemic risk that we have idealized or abstracted away something crucially important that, if accommodated, would produce a radically different alternative. The looseness of psychiatric entities carries much less epistemic risk compared to the specificity of entities in physics.

Whilst epistemic risk is lower in parts of psychiatry this does not entail psychiatry has more truth than physics. Firstly, plausibly physics accomplishes more, i.e. psychiatry might rarely meet the lower bar whereas physics might sometimes meet the higher bar. Secondly, psychiatry typically deals with imprecision whereas physics deals with precision. This means that approximate truth is typically higher in physics when obtained, whereas the easier to obtain approximate truth in psychiatry is a lower approximate truth. All this is case by case and depends on judgments over epistemic risk; I merely show how parts of psychiatry can potentially deliver truths when parts of physics fail to without implausibly claiming psychiatry is scientifically superior to physics.

- 6.5.2.4 Pessimistic Meta Induction

Where applicable, PMI is good reason to doubt reliability of IBE attributions. If previous IBE attributions appeared reliable yet turned out false then we have reason to doubt IBE attributions. Chapter one argued autism had not undergone sufficient change to hold substantial epistemic risk. Let's consider my claims in light of the above arguments.

Most symptoms associated with autism involved good epistemic conditions. This would be undermined if PMI was applicable to symptoms, if psychiatrists had been mistaken about symptoms in earlier notions of autism. However, we saw that

many symptoms of modern autism are recognisable in autism historically, from around 1950, if measured by childhood schizophrenia and its subtypes. Kanner describes symptoms like social imperceptiveness, excessive literalness, obsessiveness, narrow interests and low intellect (Kanner & Eisenberg 1956, p.558-559) whilst Bender describes preferring structured environments, intelligent, enjoying academic study, disliking new situations, rigid in approach, little awareness of self, fearing social situations, restricted range of actions and being obsessive (Bender 1959b, p.506). Many symptoms of modern autism are not at risk from the PMI but there are some exceptions. Following PMI, we cannot trust modern claims about thought processes of autistic individuals; we might be missing something which earlier psychiatrists were getting right. This claim is enhanced when we consider the salience aspect of theory-laden nature of evidence. Modern psychiatrists may have their attention directed away from such abnormal thinking. Consequently, the possibility that modern science misses such abnormal thinking places a limit on the approximate truth of modern autism. The same may also be true of age of onset, though I suggested in section 2.7.1. that age of onset is less worrying because it describes what age symptoms occurred rather than which symptoms occurred, plus modern psychology has much more information about age which symptoms manifest than was available in the 1940s to 1970s.

The psychiatric classification autism has undergone substantial changes but not generally in a problematic way. PMI is not applicable to the first step to belief of assessing theoretical virtues. We desire the balance of theoretical virtues to be as informative as possible. Scientists can disagree about which is the most informative balance. This does not entail one scientist is wrong and the other correct, only that one might be more correct than the other, i.e. a theory might be more theoretically virtuous, be more informative and therefore have higher approximate truth. Rationally reconstructed, Kanner and Bender adopted two different approaches to unifications. This type of disagreement does not entail PMI. Rather, PMI applies to the second step to belief, justifying IBE attributions. PMI generates epistemic worries when earlier versions of a theory lacked theoretical virtues, inductively suggesting scientists are mistaken about the presence of theoretical virtues in the modern theory. PMI against unifications would require earlier versions of autism which lack unification. Bender taking one all-encompassing childhood schizophrenia and Kanner preferring narrow

subtypes does not deliver PMI because both approaches exhibit the theoretical virtue unification. We lack reason to believe PMI is applicable.

Another problem is claims regarding causes are highly amenable to PMI. History of psychiatry is replete with psychiatrists being mistaken about causal origins of psychiatric classifications. Kanner's 1935 textbook is an excellent source of mistaken causal claims, Kanner wisely withholding judgment on which were true (1935, p.9). Bovet remarks that "the hope of imminent understanding [of psychiatric causes] has always been with us" (2015, p.134). Even very broadly there has been considerable change, social factors were emphasised a hundred years ago and now biological factors are emphasised (Horwitz 2002, p162). Horwitz wonders if "future historians of early twenty first-century psychiatry might ask: "why didn't they realise that poor social relationships, not neurochemicals, create distress" " (Horwitz 2002, p.206). Here I only raise general concerns, likely applicable to many psychiatric classifications, applying it to specific psychiatric classifications requires specific evidence from that classification's history.

Strategies for escaping PMI involve restricting which theories are counted. Scientific realists only consider theories from mature sciences as candidates for NMA (Psillos 1999, p.105). Large scale, well-funded research into causes of psychiatric classifications under a modern biopsychosocial framework using modern techniques like neuroscience started around 1980, so arguably prior disproved theories can be ignored when assessing reliability of causal IBEs. Unfortunately, in 2006 Murphy picks theory of mind as an example of how we might define autism causally (Murphy 2006, p.319). However, theory of mind is highly problematic (see Volkmar et al for review of evidence showing limitations with theory of mind (2004, p.142-144), Peterson for contradictory experimental evidence (2002, p.1456) and Maise for a philosophical criticism (2013, p.182)). Murphy's recommendation does not just have risk, here is a clear instance where we have good reason to believe theory of mind is a false causal claims. Similarly, the dopamine hypothesis has been the major causal theory of schizophrenia since the 1960s. After some initial successes it was repeatedly modified in face of contrary evidence when it should have been abandoned, though Kendler thinks it will be abandoned soon (Kendler 2015, p.293). Also, some theories are arguably insufficiently developed. The mechanisms assigned to neuroscience are likely to change as neuroscience develops which likely means substantial modification

of any psychiatric classifications formulated on current neuroscience (Sullivan 2014, p.276). Consider how this quote highlights how the field of genetics is undergoing regular development.

> "inheritance patterns of ASD [Autism Spectrum Disorders] could be due to gene–gene interaction, but not simply to a few genes of major effect, even if they interacted to generate risk. Research in the past decade has begun to uncover numerous genes and loci and the mechanisms that govern their action, but there are hundreds of other ASD risk loci estimated to exist [20,38,80–82] that await further genetic and functional characterization. Moreover, there has been rudimentary progress in identifying multiple 'mutations' in single individuals [95–97], suggesting possible multigenic threshold models for ASD. These variants include multiple CNVs [95,96], smaller sequence-level changes [97], variants affecting apparent non-coding regions of the genome [20,72], and combinations of each [24,72], all of which are predicted to be etiologic due to both the rarity in populations and the presumed damaging effect on the genes. An approach with significant promise is to apply informatic tools and databases, or to perform laboratory based interaction or expression mapping, to link apparently discrete ASD genes into common functional pathways or convergent networks [98,99]. Such heuristic genetic patterns may correlate with ASD endophenotypes and/or overlap with other brain and developmental disorders" (Devlin & Scherer 2012, p.233-334)

When is the field sufficiently developed to demarcate new classifications? Taking this step in 2000, or 2005, or 2010 would have risked the initial evidence for subdivisions being soon disproved. This risk remains present today and judging when this risk will sufficiently diminish is difficult.

PMI seems much less successful against causal claims involving miracle like evidence. Undermining PMI over miracle like genetic prediction requires historical evidence of scientists being mistaken about miracle like genetic prediction. Consequently, I raise no concerns over the reliability of IBE attribution via causes

over Down's syndrome.

- 6.5.2.5 Theory Laden Evidence

Where applicable, theory laden evidence would also be good reason to doubt the reliability of IBE attributions. Reliability of IBE attribution requires good epistemic conditions. When data to phenomena inference (including production of data) or phenomena to theoretical explanation inferences are based upon bad theories then the reliability of those inferences is undermined.

Chapter three considered the two main candidates for epistemically uncertain theories applicable to autism, cognitive psychology and psychoanalysis. If many symptoms or the classifications rested upon either theory then, where applicable, we would have reason for epistemic doubt. I argued both pose relatively little risk to the symptoms and classification of autism. Neither the majority of symptoms nor the classification appeared particularly theory laden by psychoanalysis or cognitive psychology except when those theories were explicitly employed or except for causal claims. Above I have suggested the theories involved in psychiatry are often much less controversial, sitting on a much stronger epistemic basis.

Chapter five provided an in depth discussion of how theory laden evidence can have a positive epistemic effect. Justifying IBEs requires good epistemic condition and one aspect of this is ensuring the background theories involved are good. I showed how some symptoms of autism were theory laden by autism itself. I have also argued that autism is a good scientific theory. Therefore, autism can act as a background theory for formulating symptoms. Where applicable, this means those symptoms have been generated under good epistemic conditions. This then conveys reliability to the IBE attribution over autism.

Of course, this is clearly a circular argument. Autism is a good theory because the IBE attribution over autism has been made under good epistemic conditions, whilst those good epistemic conditions partly reply upon autism being a good theory. There is no good reason to automatically assume this type of circular argument is negative. Massimi has provided detailed case studies showing how such circular justifications can strengthen scientific theories. She outlines how data to phenomena

inferences and phenomena to theory inferences can merge together to produce statistically strong theories (Massimi 2007, p.260; Massimi 2010, p.21). The data to phenomena inference is made to get it closer to the phenomena to theory inference, whilst the theory to phenomena inference is also made to get it closer to the data to phenomena inference. Relatedly, Psillos thinks scientific realism rests upon an unharmful circular argument. The realist claims IBE is a reliable method and as proof offers the history of science to show that IBE generally produces truths, therefore IBE is a reliable means to truth. However, those scientific theories in the history of science which are taken as evidence that IBE is successful have themselves typically been proved via IBE. Therefore, IBE is being used to prove IBE, a seemingly circular argument (Psillos 1999, p.81). Psillos argues many common arguments are circular, such as modus ponens and induction (Psillos 1999, p.81). Circularity alone is insufficient to doubt previously successful uses of IBE (or modus ponens and induction).

We saw in chapter one how Kanner derived a new classification from the existing classification scheme, filling a gap. We then saw how Kanner effectively modified autism around the 1950s, keeping the diagnostic criteria the same but associating it with new symptoms. Similarly, childhood schizophrenia incorporated elements of Kanner's 1943 autism and two other subtypes were developed, children with circumscribed interests and symbiotic psychosis. Both Kanner's and Bender's approach exhibited the theoretical virtue unification. Wing's modifications produce a different unification, one based around level of social functioning. From this she gave symptoms a different emphasis, formulating them as part of a triad of impairment of social communication, social understanding and social imagination. All this suggests employing autism as a background theory is a good epistemic condition for inferences over symptoms. This looks like a

> “positive feedback relation... it is far from obvious that the process described is automatically viciously circular or that it fails to provide a legitimate basis for increased confidence in *P* [phenomena](or *T* theory], for that matter) (Woodward 2011, p.178).

There is certainly no reason to believe it has a negative impact.

6.6 Epistemic Risk

All inferences need not exceed a personal limit on epistemic risk to merit belief. This depends upon both the level of epistemic risk involved and the level at which the limit has been set. I have identified three areas which might carry worrying levels of epistemic risk, let's consider each in turn in relation to autism.

Inferences over phenomena carry epistemic risk. I have shown above how many problems which challenge scientific realists in physics are absent over the symptoms of autism. The scale of the inferences, the reliance upon such idealised and speculative theories is not present. On this basis the epistemic risk is generally much lower than in physics. All this is case specific but we should not be excessively worried here. This position does not seem particularly controversial, most people would accept that accurately diagnosed autistic people do have low social skills and low eye contact. For autism the psychiatric skeptic would need look elsewhere to justify their concerns.

Inferences over entities carry epistemic risk. I have shown that epistemic conditions in psychiatry are sometimes superior to epistemic conditions under which such inferences over entities are made in physics. Also, claims about entities are weaker in psychiatry. The weakness of the claims has seemingly lead some to doubt psychiatry. Remember how Cushing (2013, p.41) thinks autism needs be associated with a thing, whilst Timimi (2011, p.139) considered autism arbitrary because it was not biological. This may stem from a flawed understanding of what 'things' are. To say something is an entity is to assign it a role in a theory (Psillos 1999, p.294). Psillos writes that "theories traffic in abstract entities. They assume their existence. They describe them and not concrete objects" (Psillos 2011a, p.8). Even neo-Aristolean Chakravartty argues that there is no way to know how many properties are needed for something to be an object (2007, p.65). In this regard, we see entities in physics and psychiatry as involving the same type of commitment, but in differing degrees. The major difference between psychiatry and physics is that statistical probabilities in physics are extremely high, consequently the properties cluster together very closely, whereas in psychiatry the statistical probabilities cluster together in a much weaker

manner, therefore properties cluster together in a much weaker manner. Since the probabilities are so precise in physics there is greater room for error, therefore greater epistemic risk. Of course, sufficiently low probabilities in psychiatry means unification will not be applicable. Where probabilities are sufficiently strong that unification applies, but not sufficiently strong to carry high epistemic risk, then psychiatric entities will not have high epistemic risk. I have argued autism meets these criteria.

The pessimistic meta induction does carry some risk but not excessive amounts. The history of autism shows three areas of substantial change. Firstly, the period between 1943 to around some time between the late 1940s to mid-1950s. Here autism was a very specific diagnosis which was not associated with social impairment or low intellect. We can appeal to the maturity clause to accept that a theory may undergo substantial changes in its early phases of development. By accepting this provision we reduce epistemic risk. Secondly, we have seen how various child psychiatrists took varying stances on how they formulated their classifications (i.e. just childhood schizophrenia or childhood schizophrenia and its subtypes or a spectrum covering childhood schizophrenia and much else, or an autistic spectrum). I have portrayed these as varying attempts at unifications. If we take there as being an absolute best unification then each attempt varies in degree of approximate truth to that best balance. Multiple credible ways to formulate unifications does not entail each option is not unifying. Lack of knowledge of which is closest to the best unification only entails that we do not know which of the various, quite similar approaches to autism has highest approximate truth. This does not result in high epistemic risk. PMI is largely problematic because of Kolvin and Rutter's late 1970s and early 1980s modifications. Their approach was very different from what went before and came after. This strengthens the PMI but child psychiatrists quickly reverted their approach. The level of historical discontinuity is not large. DSM-III autism strengthens PMI and thus epistemic risk but not in my judgment to fatal levels.

Autism does a good job at unifying diverse symptoms and is thus, in my judgment, the best explanation for the symptoms of certain individuals seen by psychiatrists. This is not something I can prove, only show the evidence for and appeal to the reader to make their judgement based upon their own stance on epistemic risk. At most, I suggest autism is acceptable to a moderate position on

epistemic risk. A sceptic might put the standards so high no existing psychiatric classification passes an IBE. An optimist might put the standard so low all existing psychiatric classifications pass an IBE. Now imagine a moderate position between the sceptic and the optimist where most DSM psychiatric classification does not merit belief but a small number do. Let's grant that causally based IBE's allow Huntingdon's, Down's syndrome, Rett's and Williamson's to occupy many spaces on the small list of psychiatric classifications meriting belief. Unless this is the only type of IBE we epistemically commit to then unification IBEs are possible. Above I argued autism does substantially better on unifications than many other psychiatric classifications. Autism would be among the most plausible candidates for the remaining few psychiatric classifications meriting belief offered by the position between the sceptic and optimist. I judge the position between the sceptic and optimist a plausible stance on epistemic risk whilst offering evidence autism is a strong candidate for that position.

6.7 Conclusion

In this chapter I outlined criteria for considering a psychiatric classification as meriting belief, meaning it is not in need of replacement. I have employed a three step argument, IBEs via theoretical virtues, justifying IBE attributions and not exceeding a personal limit on epistemic risk. These steps fit three approaches to belief, neo-Humeanism, neo-Kantianism and pragmatism. I have presented these as alternatives to neo-Aristoleanism, which (until shown otherwise) appears to have limited applicability in psychiatry.

IBE is establishing the best explanation via theoretical virtues. The two main theoretical virtues are unifications and causation. Most psychiatric classifications do badly on causation. I give autism as an example of a strong unification, showing it unifies a vast range of phenomena and does so in a stringent manner. Modifying classifications to make causes stronger can reduce the strength of the unifications, potentially leaving a classification less theoretically virtuous.

The IBE attribution needs to be reliable, made under good epistemic conditions. The main tool employed for this by philosophers of science is the no

miracles argument but this is difficult to mount in psychiatry, including for causal claims. However, many conditions carry much less epistemic risk in psychiatry for inferring phenomena from data and inferring theories from phenomena. The major advantages are higher accessibility and no requirement for precise specification of entities or phenomena, conditions that are not applicable to all psychiatric classifications but are applicable to autism. I also show PMI and theory laden evidence are means to doubt IBE attributions. Neither is problematic for autism but PMI is problematic for some causal claims.

All inferences involve risk but inferences are still justified providing the level of risk does not exceed a personal limit on epistemic risk. I show the epistemic risk over autism is not great. The strongest challenge is from PMI but this is not large. Autism does involve some risk but this is insufficiently high to exceed plausible levels of epistemic risk.

Lets summarize how each anti-realist argument relates to each step of belief. Theoretical virtues could be undermined by underdetermination but autism has sufficient theoretical virtues to counter underdetermination. Good epistemic conditions could be undermined by PMI and theory-laden evidence. Autism largely avoids these, however, modern beliefs about abnormal thinking is affected by both these and, too a much lesser degree, modern belief about age of onset is affected by PMI. This does put some limit on approximate truth. Epistemic risk could be undermined by PMI, underdetermination and theory-laden evidence (i.e. the stronger these are the greater the epistemic risk). These largely generate little epistemic risk, with the exception of some, though not massive, epistemic risk generated by PMI for the classification due to DSM-III autism.

Passing an IBE and that IBE being made under reliable circumstances means, given plausible levels of epistemic risk, autism merits scientific realism. This acts as an example which highlights a new route to belief potentially available for psychiatric classifications: unifications under good epistemic conditions.

7.0 Conclusion

7.1 Aim and novel contributions

My principal aim is establishing a new route to belief for psychiatric classifications and to provide an example of a psychiatric classification which attains belief via that route. I have made three novel contributions. Firstly, I re-orientate discussions of reality away from implicit neo-Aristoleanism towards neo-Humeanism, neo-Kantianism and pragmatism. Secondly, I showed an example of how three arguments against belief in psychiatry can be either undermined or showed as inapplicable. Thirdly, I showed limitations of existing arguments for belief whilst providing a novel positive argument for belief. Let's consider all this in turn.

7.2 Reality

Philosophers of psychiatry typically associate reality with causes but without fully explaining why. They typically argue that directly finding causes of a psychiatric classification can make a psychiatric classification merit belief. Alternatively, the causes can be inferred through psychiatric classifications being corroborated with family studies or drug responses, through psychiatric classification resembling biological species or through psychiatric classifications having a zone of rarity. Why, however, do these philosophers value causes so much? Almost no details here are provided, beyond statements like "causes of mental illness are genuinely out there in the structure of the world, waiting to be discovered" (Murphy 2014a, p.62) or claims of "real structures in nature" (Cooper 2005, p.49). Which philosophical account of causation and reality do these philosophers employ? Unfortunately, such discussions are near absent. They appear to be adopting something like an implicit neo-Aristotelianism, with a metaphysics of mind-independent causal powers. From here a division between such causes (or things produced by such causes) and groupings based only in subjective opinion seems clear. Sometimes our subjective opinions

group psychiatric symptoms in ways reflecting those causes and sometimes they do not. From here, we have a clear principle for demarcating real psychiatric classifications from unreal ones. Additionally, applying this principle suggests most current psychiatric classifications are not real. Also, this principle suggests we should reformulate psychiatric classifications to reflect such causes. It is debatable what the prospects are for making neo-Aristoleianism, or some other philosophy similarly emphasising mind independent causal powers, explicit to adequately show it allows true psychiatric classifications. To retain Cooper's and Murphy's position, the adopted philosophy must entail classifications ultimately are made true if they reflect the causal structure of the world. Also, there must be many other undiscovered psychiatric classifications out there waiting to be found. If these cannot be accomplished then Cooper's and Murphy's picture will not survive fully intact. Regardless, all this is unconcerning to me because I provide an alternative account of what causes and reality are. Additionally, the alternative account I offer does not simply provide philosophical justifications for existing approaches to belief over psychiatric classifications, rather, it opens up new epistemic arguments.

Seeking to establish a scientific realist account of autism, I formulated an alternative account to neo-Aristoleanism, one applicable to neo-Humeanism, neo-Kantianism and pragmatism. For neo-Humeans and neo-Kantians causes are not mind-independent in any strong sense. Rather, regularities are mind-independent. From regularities phenomena, which are models of regularities, are derived. Causes are then derived from laws. The laws, and therefore the causes, are derived from attempts to build the most informative theories. This is accomplished by building a system of theories which best balance strength, simplicity and stringency. Deciding which theories are most informative is based upon theoretical virtues. The two main theoretical virtues are unifications and causation. Unifications are where theories are strong, because they cover lots of phenomena, and stringent, because they accurately describe probabilistic relationships between those phenomena. Stronger and more stringent theories means more phenomena have been covered. This contributes to simplicity because fewer theories are required. The other main theoretical virtue, causes, is where one regularity follows one another probabilistically (rather than simply co-occur probabilistically). From this new picture of reality we can see that causes are important but not all important; the best balance between strength,

stringency and simplicity may not involve causes. From this new picture of reality let's consider arguments over belief in psychiatric classifications.

7.3 What are psychiatric classifications?

Psychiatric classifications sometimes are taken as not making any contribution, as arbitrary or even harmful. Unfortunately, exactly what making a contribution would consist of or what a non-arbitrary psychiatric classification would be is rarely stated. Such arguments seemingly have metaphysical and epistemic routes. If assuming an implicit neo-Aristoleanism then sceptics could claim psychiatric classifications contribute nothing because they do not reflect the causal structure of the world, and hence are inductively unreliable. For truth, a neo-Aristolean could demand psychiatric classifications have identified causes or could infer psychiatric classifications have causes because they take a form resembling biological species, have a zone of rarity, or are correlated with factors like family studies or treatment responses. We may have psychiatric classifications which meet neither criteria. Additionally, we may have psychiatric classifications which appear to meet either criteria but we still doubt causes are present because of the pessimistic meta induction, underdetermination or theory laden nature of evidence (when bad theories are involved). If psychiatric classifications do not reflect the causal structure of the world then psychiatric classifications must have been formulated on other grounds, such as arbitrary factors like personal opinion, dubious ethics, speculative theories or socio-political concerns.

Unfortunately, this image does not fit psychiatric classification well. The neo-Aristolean image seemingly rests upon an image of stable unified causes producing sets of stable, unified symptoms. Whilst this can happen in psychiatry, such as with Down's syndrome, such situations are rare. Also, since strong causes are easier to find, we have likely already found most of them already. The notion that the domain of psychiatry consists of many strong causes waiting to be found is not supported by current evidence. Consequently, Cooper's metaphysical picture of mental disorders existing on the model of biological species seems largely inapplicable. Murphy's 2006 metaphysical picture is less problematic, willing to be realist over psychiatric classifications which lack strong causes. However, his epistemic optimism about a

future psychiatry seems based upon a metaphysical picture he rightly rejects. He implicitly treats psychiatric classifications epistemologically as though they have a similar status to biological species, something untenable unless endorsing something like Cooper's metaphysical picture. Since causal disunity is more prevalent in psychiatry we should not demand causal unity when assessing epistemological arguments.

I have outlined a new metaphysical picture for psychiatry. The world consists of regularities and probabilistic relationships between those regularities. Sometimes the nature of the world is where one regularity follows another with high probabilities, sometimes the nature of the world is where one regularity follows another with very low probabilities. Under my picture, both situations are equally real. Reality on my picture can involve causal disunity whereas Cooper assumes (and Murphy's epistemic arguments implicitly assume) that real things must be causally unified. Such causal disunity would be insufficient for realism for Cooper, perhaps also for Murphy. However, though weaker, these causes may still legitimate belief over psychiatric classifications; we should not demand stable causes to match up with stable symptom profiles when deciding what is real, rather, reality in psychiatry generally is disunity between symptoms and causes. From here we can judge psychiatric classifications as true regardless of causal disunity.

Moving away from neo-Aristoleanism means we need seek an alternative notion of what psychiatric classifications accomplish. I portray psychiatric classification as scientific laws, understood as idealised models guiding regularity attributions. Scientific theories seek to account for phenomena. Similarly, psychiatric classifications seek to account for symptoms. The world contains regularities, call these behaviour. Some behaviour which occurs sufficiently frequently is, for a variety of reasons, of interest to psychiatrists. They take this behaviour, these regularities, and formulate them into more general regularities that we call symptoms. We then notice these symptoms co-occur. Consequently, we group them together into a scientific law, the psychiatric classification. Too the degree which symptoms have been adequately formulated from behaviour and to the degree which the psychiatric classification accurately reflects co-occurrence of symptoms, a psychiatric classification is more informative of regularities. At one level of abstraction symptoms co-occur with certain probabilities and psychiatric classifications seek to describe these. This higher level

model can be turned into a lower level model, probabilistic relationships becoming more accurate as more detail is added. The psychiatric classifications, when combined with more information, guides assigning symptoms to specific individuals, creating less abstract models. These less abstract models can then be made even less abstract, guiding predictions about how an individual will act in specific situations. Thus, through varying levels of abstraction, psychiatric classifications can potentially be inductive by giving an idealised description of reality. There is nothing automatically arbitrary about a psychiatric classification which simply accounts for such regularities. Those regularities are real and thus a psychiatric classification reflects reality to the degree it accurately describes them.

We may then notice probabilistic connections between the occurrence of the psychiatric classification and other regularities, such as genes, neurobiology, psychology or a variety of environmental factors. If the probabilistic relationships are sufficiently high then these may be causes. Accounting for these makes the psychiatric classification more informative of reality. On this basis describing such causes increases how much information a psychiatric classification covers, thus increasing how much it merits belief. However, causes are not required. Firstly, a psychiatric classification can still be informative of the world without causes, thus giving it some partial truth. Secondly, since unifications and causes often pull in different directions, the best balance may be one where psychiatric classifications do not reflect unified causes. Causes could help psychiatric classifications form a good balance of strength, simplicity and stringency but good balances can be obtained without causes.

I also showed how psychiatric classifications can be employed to formulate symptoms. Science systematises by taking data, conceptually turning them into phenomena and explaining phenomena by theories. Autism is one of many theories for interpreting symptoms. When turning data into symptoms we need decide which factors are relevant and which are incidental. Relevancy depends upon populations. In one population a factor is incidental, in another the factor is statistically relevant. Some factors are so rare to a particular population they are not worth mentioning whilst being sufficiently common to be worth mentioning for another population. Autism can set the population, producing symptoms formulated relevantly to a population, assisting systematisation. Psychiatric classifications help increase the statistical relevancy of symptoms, thus strengthening psychiatry inductively. Being

more inductive then in turn strengthens psychiatric classifications. Additionally, classifications can assist with many problematic philosophical issues surrounding ethics and background theories. Classifications can play a far more crucial role than previously understood. Replacements to existing classifications need also play this role.

Having portrayed an alternative account of what psychiatric classifications are, let's now consider how to establish if a psychiatric classification merits belief.

7.4 Belief

The approach to belief which I adopt contains three steps. Firstly, an inference to the best explanation is required, assessing the strength of theoretical virtues. Secondly, that IBE attribution must be made under epistemically reliable situations. Thirdly, the inferences must not pass a personal limit on epistemic risk. This three step approach is an alternative to neo-Aristoleianism. It is compatible with neo-Humeanism, neo-Kantianism and pragmatism, and is also acceptable to anti-realism (though they doubt certain steps are usually attained).

7.4.1 Anti-realist arguments

Underdetermination argues that any particular set of evidence can be accounted for by more than one theory, therefore there is no way to establish which theory is the correct one. Under a neo-Aristolean picture, the claim is effectively that we cannot establish which theory reflects the mind-independent causes. On my three steps of belief, underdetermination casts doubt over the first step to belief, the establishing IBE step. Underdetermination claims we have no reason to believe either theory is the best explanation. Under neo-Humeanism and neo-Kantianism, we respond by assessing each theory for theoretical virtues. Here we might find no theory under consideration exhibits much theoretical virtues. However, if one theory is much more theoretically virtuous than another then that theory is the best explanation. I have argued this was the case when comparing historically continuous autism (autism and childhood

schizophrenia from the late 1940s to late 1970s, then autism from the mid-1980s onwards) with DSM-III autism. If two theories make quite similar claims and are roughly equally theoretically virtuous then underdetermination also fails. Whilst we cannot tell which the correct theory is, we do know both theories are both good candidates for the best explanation. I suggest this is the situation with DSM-IV and DSM-5.

The pessimistic meta induction argues historical examples of scientists mistakenly believing in theories based upon good evidence inductively suggests scientists today who believe in theories based upon good evidence will also be mistaken. Under neo-Aristoleanism, PMI gives reason to believe psychiatric classifications do not reflect mind-independent causes. Psychiatrists may believe they have directly identified a cause or believe a psychiatric classification reflects causes but PMI may challenge this. Under my three stages to belief, PMI seeks to undermine the second step of belief, the reliability of IBE attributions. Scientists might have a theory which they believe is a good one but could be mistaken. Previous scientists being mistaken about a theory exhibiting theoretical virtues would give reason to doubt scientists are correct about the presence of theoretical virtues in later instances of the theory. The PMI is largely not applicable to the psychiatric classification autism. From around the late 1940s, many symptom patterns of autism and childhood schizophrenia were described which bare strong resemblance to symptom patterns associated with modern autism. Kanner, Bender and Wing disagreed over how to account for the symptom patterns, effectively offering alternative methods of unification. Different approaches to unification across the history of a psychiatric classification are only problematic if one is much less theoretically virtuous but here each approach unifies well. Each approach effectively tries to form the best unification, each exhibiting various degrees of approximate truth. Believing in any of them means a level of approximate truth has been obtained. DSM-III autism creates some concern, showing the historical continuity of autism is not fully complete. However, many child psychiatrists did not properly employ DSM-III notion of autism, diagnosing children as autistic who did not meet the criteria. Also, the DSM-III changes were partially reversed in DSM-III R. PMI gains some limited strength from DSM-III autism but not vast amounts. This makes inferences to belief over autism more risky but not excessively risky given reasonable levels of epistemic risk.

However, some aspects of modern autism differ from pre-DSM-III autism and childhood schizophrenia, specifically earlier psychiatrists sometimes described children with abnormal thinking and late onset. The presence and then absence of these allows a PMI, leaving modern beliefs about abnormal thinking not meriting belief whilst some lesser doubts are raised about age of onset.

Theory laden nature of evidence argues that evidence needs interpreting via theories and this can, when bad theories are involved, provide a false understanding of the evidence. For neo-Aristoleanians, if the real causal powers were not, for example, poor mothering then it would be a considerable coincidence if a psychoanalytical approach formulated autism in a way reflecting the real causal powers. Under my three stages to belief, this is a problem for the second step. Inferring phenomena and inferring theories to account for phenomena will typically involve background theories. Weak background theories mean such inferences may be unreliable, leaving IBE attributions unreliable. I have considered the two background theories which best resemble a classic theory-laden picture, psychoanalysis and cognitive psychology. Some causal claims seemingly are theory laden in a negative way by these. I have shown that very similar symptoms were reported under both theories. Additionally, the psychiatric classification turns out similar under both theories unless specifically formulated on those theories. I have also shown how psychiatric classification can themselves be used as theories to interpret phenomena, improving the statistical relevancy of symptoms and therefore improving them inductively. Reliability of IBE attributions can be increased by employing good theories and autism is a good theory for interpreting symptoms.

7.4.2 Inference to the best explanation

Belief in scientific theories requires an inference to the best explanation whereby theories are assessed for theoretical virtues. The two main theoretical virtues are unification and causation. Either theoretical virtue, or both in combination, can legitimate belief over psychiatric classifications. Those taking an implicit neo-Aristoleanism are correct to suggest causes are a route to belief. However, such instances of strong causal unity are rare in psychiatry and we should not expect there

to be many undiscovered strong causes which may one day be discovered. Consequently, I have appealed to unifications as an alternative approach, a means of establishing belief in the absence of unified causes.

Unifications are where a set of phenomena is accurately accounted for using as few theories as possible. Seemingly unrelated phenomena are shown as probabilistically co-occurring and this is accounted for by a unifying theory. Using as few theories as possible, whilst retaining accuracy, maximises information about those regularities. In terms of psychiatric classifications, this is where as few psychiatric classifications are employed as possible to accurately account for as many symptoms as possible, including accounting for the probabilistic relationships between symptoms. I provide autism as an example of where unifications can be employed to attain belief. Autism shows very many diverse and seemingly otherwise unrelated symptoms co-occur. Additionally, these symptoms do not simply co-occur but cluster under each triads of impairment and those triads co-occur. This makes autism very stringent. Measured by unifications, autism is extremely theoretically virtuous. I make no claims that unifications will be widely applicable in psychiatry.

I have also shown how focusing on causes, at the cost of unifications, can decrease approximate truth. We could split up autism to make it more specific to causes but this would reduce the unification. The best balance between strength, simplicity and stringency may be one from which we cannot demarcate causes. The strength gained by demarcating causes may not compensate the strength lost by reducing the number of symptoms covered. Where the strength from unification is already low then this might be worth doing. However, autism has strength from unification and subdividing on causes would vastly reduce that strength from unification. In the absence of very specific types of causes, subdividing on causes would leave autism less theoretically virtuous and no longer the best explanation.

7.4.3 Justifying IBEs

Scientific realism needs another component beyond IBE. We need also justify our IBE attributions since such attributions may be unreliable. We may consider something as unificatory when actually it is not. Hence scientific realism requires two separate

steps, an IBE and justifying IBE attribution. Both steps are required, meeting just one step is insufficient. We justify IBE attributions by considering epistemic conditions under which IBE attribution is made. Some IBE attributions are made under reliable conditions. Let’s consider justifying an IBE attribution over autism.

Justifying IBE attributions requires considering the conditions under which the IBE is made. Some aspects of psychiatry do extremely well here. Psychiatric classifications rarely deal with entities vastly remote from us which we find out about by making very precise things happen under very specific situations. We have much greater access to autism than the Higgs Bosen, vastly reducing epistemic risk. There is a short causal chain between autism and data, meaning we do not require highly specialized experiments, highly precise results, high selectivity of data, precise demarcations, questionable theories or high idealization. Extremely specific conditions or extremely specific results are not needed to demarcate the symptom of autism or their clustering. Many challenges present in other science are absent in autism, meaning symptoms and classification carry much less epistemic risk. Additionally, as described above, PMI and theory-laden nature of evidence can be reasons to doubt IBE attributions but they do not affect most symptoms and the classification. IBE attributions involving those symptoms and the classification are much more likely to be reliable.

Existing accounts of realism of psychiatric classification seem unaware of this second step. Down's syndrome and Huntingdon's genes plausibly make miracle like prediction so pass this second step. This is not the case with most causes in psychiatry. Many causes involve extremely high levels of inference, sufficiently so that they carry high levels of epistemic risk, additionally, they are at great risk to PMI so have high epistemic risk. Philosopher's employing non-miracle like causes need show such causes pass this justifying IBE step but this faces major challenges.

7.5 Scientific Realism

The realism established is not full truth but only approximate truth. Notions of full truth differ between different philosophical approaches. Neo-Humeans see absolute truth as the best balance of simplicity, strength and stringency which is closest to the

objective structure of regularities. Neo-Kantians see absolute truth as the limits of rational inquiry, best balancing strength, stringency and simplicity. Pragmatists see absolute truth as absolute usefulness and best balancing strength, simplicity and stringency would help reach this. Autism makes a substantial contribution towards attaining the best balance of strength, simplicity and stringency, therefore it attains some approximate truth. I have not argued autism is the absolute best unification. Neither have I argued that modern autism is the only good unification. I suggested Kanner's approach (childhood schizophrenia with autism as a sub-type), Bender's approach (childhood schizophrenia which includes the symptoms of Kanner's autism) and Wing's approach (the 1987 DSM-III-R notion of an autistic spectrum) all do well as unification. Kanner's approach, Bender's approach and Wing's approach all have substantial approximate truth. Not all approaches will do so well, as we saw with DSM-III autism. Also, psychiatric classifications involve many idealisations and approximations, both over evidence for IBEs and over what is inferred from the IBE. Additionally, we often deal with imprecision. This makes justifying IBEs much easier, since we do not require extremely precise conditions. However, such IBEs convey less approximate truth than those involving more precision. IBE in physics may be harder to obtain but once obtained they involve greater approximate truth than present over psychiatric classifications.

Establishing which notion of autism is best depends on two substantial uncertainties. Let’s take Kanner's approach, Bender's approach, DSM-IV and DSM-5 as all good unifications made under reliable situations. We can claim DSM-IV and DSM-5 are superior to Kanner's and Bender's approach because they do not cover late onset hallucinating individuals. Given how different these are to typical autistic people this increases stringency. However, modern autism places much less value on abnormal thinking than occurred during Kanner's and Bender's era. If modern autism is missing out on these symptoms then potentially Kanner's and Bender's approach was superior. Further scientific studies are required to establish this, ideally through modern psychiatry and psychology placing much more emphasis on the subjective psychic life of the individual. On this basis, DSM-IV and DSM-5 have one clear advantage over Kanner's and Bender's approach, removing hallucinating individuals, but there is one substantial uncertainty which may leave Kanner's and Bender's approach superior, questions over whether more focus should be placed upon abnormal thinking. In

relation to DSM-IV and DSM-5, we need give DSM-5 much longer to be implemented, for the strengths and weaknesses to come out both in the clinic and through scientific study. Until then, I cannot commit to claiming one is superior to the other. What can be established is that Kanner's approach, Bender's approach, DSM-IV autism and DSM-5 autism all have substantial approximate truth and DSM-6 should include a classification which strongly relates to these four approaches to autism.

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