

Susceptibility to Geometrical Visual Illusions in Parkinson's Disorder

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Scope Statement

This study concerns susceptibility to visual illusions in Parkinson's disease (PD), which makes it a very good fit for the Research Topic selected below: Geometrical Illusions: What They Tell Us about Human Vision in Health and Disease. Furthermore, it is the first of its kind empirical investigation into susceptibility to visual illusions in PD, which provides valuable insight into the role of dopamine and pathophysiology of the basal ganglia on susceptibility to high-level visual illusions. Furthermore, it helps to understand the visual deficits of PD patients. This paper is therefore a novel contribution to current PD and visual illusions research.

Conflict of interest statement

The authors declare a potential conflict of interest and state it below

The author(s) declared that they were an editorial board member of Frontiers, at the time of submission. This had no impact on the peer review process and the final decision

CRediT Author Statement

Calum Hartley: Supervision, Writing - review & editing. Megan Readman: Conceptualization, Investigation, Project administration, Resources, Writing - review & editing. Radoslaw Wincza: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Project administration, Validation, Writing - original draft, Writing - review & editing. Sally Linkenauger: Methodology, Software, Supervision, Validation, Writing - review & editing. Trevor Jeremy Crawford: Conceptualization, Supervision, Writing - review & editing.

Keywords

Parkinson's disease, Visual Illusions, Ebbinghaus illusion, Ponzo illusion, Muller-Lyer illusion, Depth Perception

Abstract

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Parkinson's disorder (PD) is a common neurodegenerative disorder affecting approximately 1-3% of the population aged 60 years and older. In addition to motor difficulties, PD is also marked by visual disturbances, including depth perception, abnormalities in basal ganglia functioning, and dopamine deficiency. Reduced ability to perceive depth has been linked to an increased risk of falling in this population. The purpose of this paper was to determine whether disturbances in PD patients' visual processing manifest through atypical performance on visual illusion (VI) tasks. This insight will advance understanding of high-level perception in PD, as well as indicate the role of dopamine deficiency and basal ganglia pathophysiology in VIs susceptibility. Groups of 28 PD patients (Mage = 63.46, SD = 7.55) and 28 neurotypical controls (Mage = 63.18, SD = 9.39) matched on age, general cognitive abilities (memory, numeracy, attention, language), and mood responded to Ebbinghaus, Ponzo, and Muller-Lyer illusions in a computer-based task. Our results revealed no reliable differences in VI susceptibility between PD and neurotypical groups. In the early- to mid-stage of PD, abnormalities of the basal ganglia and dopamine deficiency are unlikely to be involved in top-down processing or depth perception, which are both thought to be related to VI susceptibility. Furthermore, depth-related issues experienced by PD patients (e.g., increased risk for falling) may not be subserved by the same cognitive mechanisms as VIs. Further research is needed to investigate if more explicit presentations of illusory depth are affected in PD, which might help to understand the depth processing deficits in PD.

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In review

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In review

Susceptibility to Geometrical Visual Illusions in Parkinson's Disorder

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Authors' Contributions:

R.W. designed the study, as well as conducted the majority of the research. R.W. has also written the manuscript and conducted the analyses. M.R. helped by providing access to PD patients, as well as by providing theoretical feedback. S.L., C.H., and T.C. supervised the project, provided theoretical feedback, and assisted with editing the manuscript and revisions.

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In review

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Abstract

49 Parkinson's disorder (PD) is a common neurodegenerative disorder affecting approximately 1-
50 3% of the population aged 60 years and older. In addition to motor difficulties, PD is also
51 marked by visual disturbances, including depth perception, abnormalities in basal ganglia
52 functioning, and dopamine deficiency. Reduced ability to perceive depth has been linked to an
53 increased risk of falling in this population. The purpose of this paper was to determine whether
54 disturbances in PD patients' visual processing manifest through atypical performance on visual
55 illusion (VI) tasks. This insight will advance understanding of high-level perception in PD, as
56 well as indicate the role of dopamine deficiency and basal ganglia pathophysiology in VIs
57 susceptibility. Groups of 28 PD patients ($M_{age} = 63.46$, $SD = 7.55$) and 28 neurotypical controls
58 ($M_{age} = 63.18$, $SD = 9.39$) matched on age, general cognitive abilities (memory, numeracy,
59 attention, language), and mood responded to Ebbinghaus, Ponzo, and Muller-Lyer illusions in
60 a computer-based task. Our results revealed no reliable differences in VI susceptibility between
61 PD and neurotypical groups. In the early- to mid-stage of PD, abnormalities of the basal ganglia
62 and dopamine deficiency are unlikely to be involved in top-down processing or depth
63 perception, which are both thought to be related to VI susceptibility. Furthermore, depth-related
64 issues experienced by PD patients (e.g., increased risk for falling) may not be subserved by the
65 same cognitive mechanisms as VIs. Further research is needed to investigate if more explicit
66 presentations of illusory depth are affected in PD, which might help to understand the depth
67 processing deficits in PD.

68 *Keywords:* Parkinson's disease, visual illusions, Ebbinghaus illusion, Ponzo illusion,
69 Muller-Lyer illusion, depth perception

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Susceptibility to Geometrical Visual Illusions in Parkinson's Disorder

Visual illusions (VIs) occur when the configuration of a stimulus causes the viewer to incorrectly perceive relationships between its parts (Notredame et al., 2014). VIs have been widely used as a tool to investigate how visual perception develops (e.g., Doherty et al., 2010) and the impact of neuropsychological disorders such as schizophrenia (for a review see King et al., 2017; Costa et al., 2023) and autism (for a review see Gori et al., 2016). Although impairment of visual perception (e.g., hallucinations) is now well established in Parkinson's disorder (PD) (Nieto-Escamez et al., 2023; Sauerbier and Chaudhuri, 2013; Weil et al., 2016), research has yet to investigate how PD affects susceptibility to VIs. Furthermore, depth perception – which is linked to VI susceptibility (e.g., Doherty et al., 2010; Gregory, 1963, 1966) and increased risk of falling (Cummings et al., 1995) – is shown to be affected in PD (Maschke et al., 2006). Therefore, studying VI susceptibility in this population may indicate how neuropsychological characteristics of PD (e.g., dopamine deficits and the pathophysiology of the basal ganglia) impact depth perception and top-down visual processing.

PD is a common neurodegenerative disorder affecting approximately 1-3% of the population aged 60 years and older (Ball et al., 2019; Pringsheim et al., 2014). It is characterised by motor deficits including tremors, rigidity, bradykinesia (slowed movement execution and initiation), and postural instability (Berardelli et al., 1983; Guttman et al., 2003). Although PD was traditionally considered to be a paradigmatic motor disorder, non-motor disruptions (including visual distortions) are experienced by the majority of PD patients (Chaudhuri et al., 2011). Visual distortions in PD include decreased contrast sensitivity (Sauerbier and Chaudhuri, 2013; Uc et al., 2005; van der Lijn et al., 2022), decreased colour discrimination (Pieri et al., 2000), deficits in motion and spatial perception (Uc et al., 2005), visual acuity deficits (Uc et al., 2005), and visual hallucinations (Barnes and David, 2001; Weil et al., 2016).

97 It is widely regarded that visual disturbances in PD are caused by a reduction of
98 dopamine (Bodis-Wollner, 1990). Dopamine, a key neurotransmitter in the mammalian brain
99 (Bibb, 2005), is believed to play a crucial role in visual perception (Harris et al., 2003). For
100 example, Andreou and colleagues (2015) showed that dopamine influences neurotypical
101 adults' sensitivity to detecting an object in snowy (noisy) black-and-white pictures. Dopamine
102 has also been shown to influence visual perception in PD. Multiple studies have found that
103 retinal dopamine levels and dopaminergic innervation surrounding the fovea are reduced in PD
104 (Harnois and Di Paolo, 1990; Nieto-Escamez et al., 2023; Sauerbier and Chaudhuri, 2013),
105 resulting in visual perception deficits such as poorer light adaptation and decreased contrast
106 sensitivity (e.g., Armstrong, 2015; Pieri et al., 2000). [Other visual deficits that are linked to
107 dopamine deficiency include greater thresholds for motion detection \(e.g., Trick et al., 1994\),
108 colour discrimination \(e.g., Buttner et al., 1994\), as well as visuospatial deficits \(e.g., Gibson
109 et al., 1987; for an overview of dopamine-related deficits in PD, see Brandies & Yehuda, 2008\).](#)

110 Another hallmark of PD is the pathophysiology of the basal ganglia (Obeso et al., 2000).
111 The basal ganglia are believed to control motor and cognitive functioning (Macpherson &
112 Hikida, 2019); however, recent research has implicated their role in visual perception (Maschke
113 et al., 2006; Nieto-Escamez et al., 2023). Maschke and colleagues (2006) showed that PD
114 patients and patients with spinocerebellar ataxia (a movement disorder) made greater errors
115 when estimating the slant of an illusory display (Ames Trapezoidal Window). The difficulties
116 evidenced by PD patients were attributed to differences in the basal ganglia's functioning.
117 Furthermore, dopamine losses across key components of the basal ganglia (e.g., subthalamic
118 nucleus, substantia nigra, and globus pallidus) are observed in PD (Benazzouz et al., 2014).
119 [Dopamine deficiency in the basal ganglia is of particular interest, as the link between these two
120 is thought to be related to the processing of visual information. Sil'kis \(2007\) proposed a
121 mechanism in which the basal ganglia modulates the efficiency of synaptic transmission in an](#)

122 [interconnected parallel circuit that involves the limbic cortex, basal ganglia, thalamus, and](#)
123 [cortex. This process is contingent on dopamine-dependent processes. It is, therefore, plausible](#)
124 [to suspect that changes to this circuit in PD, could result in abnormal VIs susceptibility.](#)

125 Given the well-documented abnormalities in depth perception in PD (Maschke et al.,
126 2006; Ou et al., 2018), which could be linked to dopamine deficiency and the role of the basal
127 ganglia (e.g., Maschke et al., 2006), it may be that susceptibility to depth-related VIs (e.g., the
128 Ponzo illusion) is atypical in this population. Studying VIs in PD will enable us to comprehend
129 the potential relationship between dopamine losses and basal ganglia pathophysiology with
130 susceptibility to VIs. Consequently, VIs could offer a promising approach to address perceptual
131 depth deficits in PD.

132 Although abnormalities in the basal ganglia and deficiency in dopamine levels could
133 potentially influence sensitivity to depth-related VIs in PD, there are reasons to believe that
134 sensitivity to *high-level* VIs may be preserved. The term 'high-level VIs' is used to classify
135 illusions that are thought to emerge at a later stage of visual processing (from approximately
136 the V1 and beyond) compared to low-level illusions that are mediated at the retinal level and
137 up to V1 (King et al., 2017). The Ebbinghaus, Ponzo, and Muller-Lyer are examples of high-
138 level illusions, while the Brightness and Herman Grid illusions are examples of low-level
139 illusions (King et al., 2017).

140 Milner and Goodale's (1992) classic theory proposes that there are two visual streams
141 in the brain. The ventral stream is responsible for perception for vision, while the dorsal stream
142 is responsible for perception for action. VIs represent a unique method for investigating
143 differences between these two streams. Research shows that even if the Ebbinghaus illusion is
144 perceived, grip aperture is not affected by the illusion in neurotypical adults (e.g., Haffenden
145 et al., 2001). Also, for the Ponzo illusion, it has been shown that grasping in neurotypical adults

146 is not 'fooled' by illusory displays (Ozana & Ganel, 2020). Studies on differences in perception
147 and action relating to VIs have been used to demonstrate the dichotomy between dorsal and
148 ventral streams. Research examining the functioning of ventral and dorsal visual streams in PD
149 patients has revealed abnormalities in vision for action in a blind walking task coupled with
150 intact performance on a line matching task (Giovannini et al., 2006). These findings suggest
151 that impairments in visual perception in PD may be explained by abnormalities in dorsal stream
152 processing, while the ventral stream remains unaffected, potentially preserving sensitivity to
153 high-level VIs. In line with these findings, PD patients also experience deficits associated with
154 higher level visual processing of motor actions including slower motor imagery (Poliakoff,
155 2013) and difficulties observing other people perform actions (Tremblay et al., 2008). These
156 differences in processing visual action signal possible impairments in dorsal stream
157 functioning.

158 This study is the first to test PD patients on their susceptibility to the Ebbinghaus,
159 Ponzo, and Muller-Lyer illusions using the method of adjustment. PD patients and neurotypical
160 age-matched controls completed a series of online illusion tasks in their own homes. On one
161 hand, based on evidence of depth perception abnormalities in PD (e.g., Ou et al., 2018), we
162 anticipated that PD patients may be less susceptible to these VIs than controls. However, we
163 also believe the differences are likely to be stronger for VIs with most explicit depth, like the
164 Ponzo illusion. However, on the other hand, we recognized that PD patients' susceptibility to
165 these VIs could be unaffected due to a lack of severe disruption to the ventral stream. Our
166 findings will advance theoretical understanding of how PD impacts susceptibility to high-level
167 VIs and ventral stream visual processing.

168 **Method**

169 **Participants**

170 *Power Analysis*

171 G*Power software (Faul et al., 2007) was used to perform an a priori power analysis
172 to ascertain the necessary sample size required. Power ($1 - \beta$) was specified as .80 and the
173 significance level (α) was set to .05. The anticipated effect size was modelled on the results
174 obtained by Grzeczowski et al., (2018). Due to this, we anticipated a medium effect size of d
175 = 0.46. For the frequentist parameters defined, a sample size of $N = 56$ is required to achieve
176 a power of .80 at an alpha of .05. Hence, we aimed to recruit 56 participants.

177 *Demographics*

178 Participants included 27 PD patients (15 females, 12 males) and 28 neurotypical
179 participants (17 females, 11 males). PD participants were recruited from the Department of
180 Psychology database of PD patients at Lancaster University, while controls were recruited via
181 convenience sampling ($n = 18$) and sign-ups to the Centre for Aging Research at Lancaster
182 University ($n = 10$). All PD patients were medicated. Participants were predominantly white
183 British ($n = 47$). Participants were largely well-educated, with the majority holding at least an
184 undergraduate degree ($n = 35$). None of the participants reported having a cognitive impairment
185 or any neurological illness. Nine participants reported having a psychiatric illness (anxiety: n
186 = 5; [3 in the control group](#), and depression: $n = 4$; [3 in the control group](#)). Eleven participants
187 reported visual impairments for which they were receiving treatment, including glaucoma ($n =$
188 [3; 1 in the control group](#)), age-related macular degeneration ($n = 2$), double vision ($n = 3$),
189 astigmatism ([control group](#)), keratoconus, and short-sightedness ([control group](#); all $n = 1$). All
190 participants confirmed that they had corrected-to-normal vision despite having these
191 conditions, [and the aforementioned difficulties did not affect their ability to perceive the VIs.](#)
192 [Participants' visual acuity was not assessed as previous research indicates that VIs](#)

193 [susceptibility is not related to it \(Cretenoud et al., 2021\) as well as in PD visual acuity remains](#)
 194 [largely perseverated \(Hunt et al., 1995\).](#)

195 No significant differences between PD patients and neurotypical controls were
 196 observed for age ($t = 0.05, p = .96$), years of formal education ($t = 0.21, p = .835$), scores for
 197 mild cognitive dysfunction ($t = -0.706, p = .484$), anxiety ($t = 0.599, p = .07$), and depression
 198 ($t = 0.15, p = .882$). These non-significant group differences indicate that the groups were
 199 closely matched (see Table 1 for more details). Full details of the PD patients' cohort are
 200 presented in Table 2 below.

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Table 1*Means and Standard Deviations for PD Patients and Neurotypical Adults*

	Total	Age	Education	Depression	MOCA	Anxiety	Screen Size
PD Patients	27	63.3(7.64)	15.11(4.17)	4.67(2.73)	24.89(2.04)	5.78(3.94)	35.48(9.93)
Neurotypical Adults	28	63.18(9.39)	14.89(3.52)	3.93(2.61)	24.54(2.04)	5.64(2.64)	36.93(4.3)

Note. Higher values for depression and anxiety indicate more severe symptoms. Higher MOCA scores indicate better cognitive functioning. Screen size is reported in centimetres.

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Table 2*Characteristics of PD Patients*

Participant	Age	Gender	Years since the PD diagnosis	Years since PD onset	LEDD	Last dose	MOCA	HADS-A	HADS-D	Hoehn and Yahr Stage
1	51	Female	3	5	555	204	22	4	1	1
2	62	Female	8	11	760	30	26	11	6	1
3	65	Male	5	5	660	148	25	1	2	0
4	63	Female	5	6	350	180	26	6	7	2
5	57	Female	2	6	375	85	25	15	6	2
6	56	Male	6	8	1000	136	18	3	5	2
7	58	Male	2	3	973	120	26	6	7	1
8	74	Female	4	5	195	2	26	1	1	2
9	59	Male	5	15	220	0	23	2	3	1
10	70	Male	5	7	595	210	25	4	3	1
11	67	Male	5	10	960	720	25	1	5	1
12	67	Male	9	21	N.A.	204	26	3	0	2
13	70	Male	13	30	N.A.	90	26	2	2	2
14	71	Female	3	6	400	230	26	2	1	0
15	59	Male	4	7	590	25	26	4	3	2
16	67	Male	6	10	840	60	27	12	5	1
17	63	Male	4	5	475	240	25	7	6	1
18	59	Female	6	7	362	420	25	14	5	0
19	75	Female	7	7	1680	127	25	5	7	2
20	51	Female	3	5	555	150	24	3	1	1
21	70	Female	11	2	578	0	27	6	3	2
22	57	Female	2	5	300	150	24	8	5	2
23	67	Female	5	6	500	120	26	6	8	1
24	51	Female	1	4	800	210	20	11	8	2
25	59	Female	5	16	355	1440	26	5	9	1
26	81	Female	7	7	640	255	26	8	9	2
27	60	Male	4	6	715	45	26	6	8	3

Note. The time since the last dose is in minutes. HADS-A and HADS-D correspond to anxiety and depression, respectively (described in further detail below). LEDD corresponds to L-dopa equivalent daily dose, which is amongst the most common medication for PD (Julien, 2021). Hoehn and Yahr's scale refers to the severity of symptoms in PD, ranging from 0 (least severe) to 5 (most severe) (MDS, 2008).

208 **Materials**

209 All study stimuli were developed using Unity 3D© Gaming Engine and were visually
210 displayed to participants using the 'screen share' function in Microsoft Teams. [The stimuli](#)
211 [were modeled on existing work in the field \(e.g., Chouinard et al., 2013; Sperandio et al., 2023\).](#)
212 These studies were conducted virtually as a precaution to protect both participants and
213 experimenters from COVID-19. [Though it may be seen as a potential confound, previous](#)
214 [research indicates that online testing yields reliable measurements, however, the effect sizes](#)
215 [tend to be smaller \(e.g., Chuey et al., 2021; Pallen et al., 2022\).](#) As participants viewed the
216 stimuli through screen share on their personal devices, screen size ranged between 23 and 61
217 inches. An independent samples *t*-test indicated that screen sizes of PD patients ($M = 35.48$,
218 $SD = 10.56$) and neurotypical controls ($M = 36.93$, $SD = 4.30$) did not significantly differ, $t(53)$
219 $= -0.706$, $p = .484$. Also, no significant correlations were observed between illusion strength
220 and screen size.

221 Three visual illusions were used: the Ebbinghaus illusion, the Ponzo illusion, and the
222 Muller-Lyer illusion. Participants were required to adjust the size of a line or circle (depending
223 on the illusion) until they perceived it as equivalent in size to the reference stimuli. The size
224 was adjusted using the right and left arrow keys, and trials were progressed using the *ENTER*
225 key. The experimental software obtained a measure of reaction time (ms). RT data was only
226 used to detect skipped trials (responses faster than approximately 5 seconds, which were
227 accompanied by large Z-score values, at least 2 standard deviations (SDs) away from the
228 mean). Average RTs significantly differed between illusions [$F(1.63, 88.05) = 5.37$, $p = .006$]
229 but not between participant groups [$F(1, 54) = 1.12$, $p = .294$]. Post hoc comparisons with Holm
230 correction showed differences between RTs for the Ebbinghaus ($M = 21.24$, $SD = 6.11$) and
231 the Muller-Lyer ($M = 23.87$, $SD = 9.10$) illusions, $t = -2.75$, $p = .014$, as well as between the
232 Muller-Lyer ($M = 23.87$, $SD = 9.10$) and Ponzo illusions ($M = 21.08$, $SD = 6.01$), $t = 1.92$, $p =$

233 .013. No difference was detected between RTs for the Ebbinghaus and Ponzo illusions; $p =$
234 [.867. Furthermore, we conducted correlations between the illusion's strength and RTs for both](#)
235 [groups individually, and the whole sample, to assess if prolonged exposure affected VIs](#)
236 [susceptibility \(Bressan & Kramer, 2021\). None of the correlations approached significance.](#)

237 *The Ebbinghaus Illusion*

238 The two orange centre circles were surrounded either by eight pink large inducers (125
239 pixels in diameter, positioned 35 and 90 pixels away from the central circle) or eight pink small
240 inducers (50 pixels in diameter, positioned 32 and 80 pixels away from the central circle)
241 presented on a black background (see Figure 1). The orange centre circle was 100 pixels in
242 diameter (an example display is illustrated in Figure 1). There were 16 trials in total. The
243 starting size of the adjustable centre circle was 50 pixels in 8 trials and 150 pixels in 8 trials.
244 The side of appearance (left or right) and inducer size (large or small) for the adjustable circle
245 varied between trials, with four trials for each size and side combination. The order of
246 presentation was randomised.

247 **Figure 1**

248 *Example Ebbinghaus Illusion Trial*

249

250 *Note. Participants were required to manipulate the size of the right orange circle to match the*
251 *size of the left orange circle (or vice versa).*

252 *The Ponzo Illusion*

253 Four pink converging lines were used as inducers (two at 420 pixels in length at a 64-
254 degree angle, and two at 380 pixels in length at a 10-degree angle). The adjustable and reference
255 horizontal lines were orange and 135 pixels apart. The reference line for both methods of

256 measurement was held constant at 100 pixels. An example display can be found in Figure 2.
257 There were 8 trials in total; in 4 trials the adjustable line started at 50 pixels, and in 4 trials the
258 adjustable line started at 150 pixels. In half of the trials, the adjustable line appeared above the
259 horizontal midline and half below. The order of presentation was randomised.

260 **Figure 2**

261 *Example Ponzo Illusion Trial*

262

263 *Note. Participants would be required to manipulate the length of the bottom orange line to*
264 *match the length of the top orange line (or vice versa).*

265 **The Muller-Lyer Illusion**

266 Two orange lines with inwards or outwards facing arrows (40 pixels in length) at a 45-
267 degree angle were presented. The reference line for both methods of measurement was held
268 constant at 150 pixels. An example display can be found in Figure 3. There were 16 trials in
269 total with four trials for each side of the presentation (left or right) and arrow type (inwards or
270 outwards facing) combination. The starting size of the adjustable line was 75 pixels in 8 trials
271 and 225 pixels in 8 trials. The order of presentation was randomised.

272 **Figure 3**

273 *Example Muller-Lyer Illusion Trial*

274

275 *Note. Participants would be required to manipulate the left orange line (between the*
276 *arrowheads) to match the length of the right orange line (between the arrowheads), or vice*
277 *versa.*

278 *Questionnaires and Screening Tools*

279 Questionnaires and screening tools were administered to participants via an online
280 interview. These included the Hospital Anxiety and Depression Scale (HADS; Snaith, 2003),
281 the Montreal Cognitive Assessment (MOCA; Nasreddine et al., 2005), and the Movement
282 Disorder Society – Unified Parkinson Disease Rating Scale (MDS-UPDRS; Goetz, 2007).
283 These measures were included to test whether potential differences in susceptibility to VIs were
284 influenced by participants' cognitive abilities and/or mood.

285 HADS consists of 14 statements that measure traits of depression (7 items) and anxiety
286 (7 items). Each statement has four corresponding answers which the interviewee can choose
287 between. For example, for the statement 'I feel tense or wound up' (an anxiety item), the
288 response options are: 'most of the time' (3 points), 'a lot of the time' (2 points), 'from time to
289 time, occasionally' (1 point), and 'not at all' (0 points). Higher scores indicate more severe
290 symptomology. During the interview, the participant was instructed to think about their
291 feelings over the past week. The statements were read out loud, followed by the answers, and
292 then the participant chose one of them. If they were unsure, the interviewee was asked to make
293 their best guess. For half of the questions the response options were read in order from negative
294 to positive, and for the other half the response options were read in order from positive to
295 negative.

296 The MOCA includes 13 tasks measuring a variety of cognitive functions, including
297 visuospatial/executive functions, naming, memory, attention, language, abstraction, delayed
298 recall, and orientation. As the study was conducted online, small changes were implemented.
299 The first part of the visuospatial/executive task (connecting numbered dots) was omitted as the
300 participant was unable to respond due to online administration. Also, in the orientation task,
301 participants were not asked about their present location as the researchers were unable to

302 validate their responses. The participant could therefore score up to 27 points (30 points
303 originally).

304 The MDS-UPDRS consists of four subscales measuring: I – non-motor aspects of
305 experiences of daily living (1.1 – 1.6), (questions 1.7 to 1.13 were excluded as they were
306 unrelated to our study's objective); II – motor aspects of experiences of daily living (2.1 –
307 2.13); III – motor examinations (3.1 – 3.8; 3.15 – 3.18) (questions 3.9 to 3.14 were dropped as
308 the study's online nature prevented the researchers from correctly assessing the participant's
309 performance); IV – motor complications (4.1 – 4.6). Parts I, II, and IV included questions
310 asking participants to rate their difficulty engaging with a variety of daily tasks (e.g., getting
311 dressed and getting out of a deep chair) from normal to severe on a five-point scale. Part III
312 involved a motor examination of the participants, who performed tasks as they were described
313 by the researcher (e.g., holding their hands still in front of them). The researcher then scored
314 the performed action according to the MDS-UPDRS guidelines.

315 **Procedure**

316 All participants were tested online via Microsoft Teams. Before taking part in the online
317 session, participants were required to complete a survey requesting basic demographic
318 information (e.g., age and gender), history of PD and diagnosis, and current medication intake.
319 Then, all participants were screened for mild cognitive impairment (MOCA) and mood
320 disorders (HADS). Individuals with PD symptoms were also assessed using the MDS-UPDRS.
321 Participants were then given control over the researcher's laptop using the Teams share
322 function [~~if that was~~which was not possible in some cases (< 5), participants were asked to
323 provide oral instructions to the researcher, however, our RT correlations with VIs susceptibility
324 failed to reach significance, hence the different modes of entering data were not deemed
325 problematic]. Once control was given, participants were presented with the experimental

326 stimuli and asked to manipulate the size of a line (Müller-Lyer or Ponzo display) or centre
327 circle (Ebbinghaus display; either to increase or decrease) using the right and left (left to
328 decrease, right to increase) arrow keys [\(see Figure 4\)](#). Once the participant believed that their
329 stimulus matched the size of the reference non-adjusted line or circle, they were prompted to
330 press *Enter*. If the participant was unable to take control, they were asked to orally instruct the
331 researcher to either increase or decrease the sizes until they were happy with it. Participants
332 were prompted to be as accurate as possible in their judgements and [instructed to make their](#)
333 [judgements as quickly as possible](#). In both scenarios, the researcher looked away from the
334 screen to prevent the participant from feeling pressured to respond quickly or to prevent any
335 gaze cues. The order of illusion blocks and trials within blocks were randomised. Once the
336 experiment finished, participants were fully debriefed and encouraged to ask questions. The
337 study took between 45 to 60 minutes to complete.

338 [Figure 4](#)

339 [Example Trial](#)

340 [Note. During adjustment, the participant used the arrows on their keyboard to match the larger](#)
341 [of the two orange, inner circles with the other](#), target [circle](#). [Once they perceived the circles as](#)
342 [equal](#) in size, they pressed enter to proceed to the next trial.

343 **Analysis Plan**

344 The data were screened to assess for normality of distribution. The magnitude of the
345 illusion was calculated as the difference between the actual size of the target and the
346 participant's response. A 2 (Group: PD patients, neurotypical controls) x 3 (Illusion:
347 Ebbinghaus, Ponzo, and Muller-Lyer) repeated measures ANOVA was conducted.
348 Correlations between VIs, demographic data, and Parkinsonian symptoms were computed
349 using both frequentist and Bayesian analyses. [Multiple comparisons were analysed with Holm](#)

350 [correction \(e.g., Grzeczowski et al., 2018\)](#). Screening analyses were performed using IBM
351 SPSS Statistics (Version 27) and all the remaining analyses were performed in JASP Team
352 (2022).

353 **Results**

354 **Normality of The Data Set**

355 Each participant's data were screened for outliers (40 responses per participant) located
356 at least two SDs away from the response mean (unusually low or high values reported), and
357 compared against the population's mean for each particular illusion. Outliers were screened for
358 PD patients and neurotypical adults separately. To ensure consistency across responses, all
359 individual outliers were replaced with a second value for the same trial type.

360 Several outliers were identified across the data. For the Ebbinghaus illusion, there were
361 27 outliers (3.01%) out of 896 trials, including 18 in the PD group (16 belonged to one
362 participant, meaning every single trial of that participant was outside ± 2 SDs away from the
363 mean, resulting in the exclusion of this participant) and 9 in the neurotypical group. For the
364 Ponzo illusion, there were 20 outliers (4.46%) out of 448 trials, including 14 in the PD group
365 and 6 in the neurotypical group. For the Muller-Lyer illusion, there were 31 outliers (3.45%)
366 out of 896 trials, including 18 in the PD group and 13 in the neurotypical group. The majority
367 of outliers were due to the participant pressing the *enter* key too forcefully, which resulted in
368 skipping a trial (this was identified by unusually quick reaction times of less than 3 seconds).
369 These scores were replaced with the participant's second score in the same condition.

370 **Group Differences Between PD Patients and Neurotypical Controls**

371 To examine differences between PD patients and neurotypical participants on their
372 susceptibility to the Ebbinghaus, Ponzo, and Muller-Lyer illusions, a 2 x 3 repeated measures
373 ANOVA was conducted. Both Levene's test for equality of variance for all three illusions and

374 Mauchly's W test of sphericity indicated that the assumptions for a two-way ANOVA were
375 met; $p = .349$, $p = .777$, $p = .663$, and $p = .057$ respectively. The results revealed a significant
376 effect of the illusion, $F(2, 108) = 628.63$, $p < .001$, $\eta^2 = .87$. The difference between PD patients
377 and neurotypical approached significance, $F(1, 54) = 3.79$, $p = .057$, $\eta^2 = .003$, as did the
378 Population x Illusion interaction $F(2, 54) = 3.07$, $p = .050$, $\eta^2 = .004$. Given our a priori
379 predictions, we proceeded to conduct post-hoc comparisons though note that these should be
380 treated with caution as the interaction was only marginally significant. Post-hoc comparisons
381 using Holm correction (after Grzeczowski et al., 2017) showed that PD patients were
382 significantly less susceptible ($M = -0.18$, $SD = 0.08$) than controls ($M = -0.23$, $SD = 0.09$) to
383 the Ponzo illusion; $t(54) = 2.19$, $p = .033$, $d = 0.59$. No significant differences were observed
384 for the Ebbinghaus (PD; $M = -0.14$, $SD = 0.04$ and controls; $M = -0.13$, $SD = 0.05$) and Muller-
385 Lyer illusions (PD; $M = -0.54$, $SD = 0.07$ and controls; $M = -0.57$, $SD = 0.08$).

386 Similar results were observed by conducting a Bayesian 2 x 3 repeated measures
387 ANOVA. Based on Jeffreys' (1939) rule of thumb for interpreting Bayesian results (1-3, 3-10,
388 and 10+, are considered weak, moderate, and strong effects, respectively), we observed weak
389 evidence for an effect of VIs (BF = 0.89), very weak evidence for an effect of group (BF <
390 0.001), and weak evidence for an interaction (BF = 0.681). Bayesian t -tests yielded similar
391 results for differences between the groups on each VIs. Weak evidence was observed for group
392 differences on the Ebbinghaus, Ponzo, and Muller Lyer illusions; $B = 0.399$, $B = 1.911$, and B
393 $= 0.67$, respectively. Evidence from these Bayesian analyses indicates a lack of differences
394 between PD patients and neurotypical controls on the three tested illusions.

395 **Correlations**

396 Several correlations were performed to assess whether severity of PD symptoms was
397 associated with differences in susceptibility to VIs. The variables of interest included

398 susceptibility scores for each illusion, time since the last medication dose, years since PD
399 diagnosis, years since starting medication, years since symptom onset, LEDD score, and the
400 total MDS-UPDRS score. As some variables were not normally distributed, Spearman's
401 correlations and their Bayes equivalent were conducted. No frequentist or Bayesian
402 correlations approached significance, indicating that susceptibility to VIs was not correlated
403 with patients' PD characteristics.

404 **Figure 5**

405 *Individual Data Points for the Ebbinghaus Illusion for PD Patients (PDP) and Healthy Control*
406 *Participants (HCP)*

407
408 *Note.* Both groups show overlapping similarities in their susceptibility to the Ebbinghaus
409 illusion.

410 **Figure 6**

411 *Individual Data Points for the Ponzo Illusion for PD Patients (PDP) and Healthy Control*
412 *Participants (HCP)*

413
414 *Note.* Both groups show overlapping similarities in their susceptibility to the Ponzo illusion.

415 **Figure 7**

416 *Individual Data Points for the Muller-Lyer Illusion for PD Patients (PDP) and Healthy Control*
417 *Participants (HCP)*

418
419 *Note.* Both groups show overlapping similarities in their susceptibility to the Muller-Lyer
420 illusion.

421

Discussion

422 This study investigated whether PD patients – a population characterised by basic and
423 complex visual disturbances (e.g., Maschke et al., 2006) – and neurotypical adults differ in
424 their susceptibility to the Ebbinghaus, Ponzo, and Muller-Lyer visual illusions. We formulated
425 two competing hypotheses: (a) PD patients may be less susceptible to VIs than neurotypical
426 adults due to abnormalities in the basal ganglia and dopamine deficits affecting their visual
427 processing, or (b) sensitivity to VIs may not be impacted by PD due to their visual deficits
428 specifically affecting dorsal stream processing of actions. Our analyses did not identify robust
429 differences between the two populations' responses for any illusion. These results suggest that
430 dopamine deficiency and basal ganglia pathophysiology may not be directly related to VI
431 susceptibility and that these may affect different aspects of visual perception (Maschke et al.,
432 2006). Furthermore, our data imply that the ventral stream's processing of vision for perception
433 in PD is largely free from pathology when viewing VIs.

434 Previous research has shown that depth perception deteriorates in older adults (Salonen
435 and Kivela, 2012) and that the inability to perceive depth correctly increases their risk of falls
436 (Cummings et al., 1995; Ivers et al., 2000; Lord and Dayhew, 2001). There is also an extensive
437 body of evidence documenting abnormal depth perception in PD (e.g., Ou et al., 2018),
438 including in illusory contexts (Maschke et al., 2006). Our analysis, however, showed only
439 marginal evidence for abnormal depth perception. PD patients appeared to have reduced
440 susceptibility to the Ponzo illusion. The Ponzo illusion is considered a classic example of a
441 depth illusion (Gregory, 1963), and creates the most apparent experience of depth among the
442 tested illusions. These findings suggest that dopamine deficiency and/or pathophysiology of
443 the basal ganglia may, marginally, affect depth perception as shown by the illusory depth in
444 the Ponzo illusion, adding to already existing evidence concerning such deficits (e.g., Maschke
445 et al., 2006). It is, however, important to note that the depth here is only illusory (induced), and

446 arguably less apparent compared to the Ames Window illusion (such as in Maschke et al.,
447 2006), and it is not real, 3D depth. PD patients might still have difficulties in perceiving depth
448 in everyday situations (e.g., Cummings et al., 1995). Potentially, only a slight indication of
449 reduced susceptibility was observed because PD participants in this study were mostly in the
450 early- and mid-stages of PD. Therefore, it might still be possible that susceptibility to VIs starts
451 deteriorating as PD develops, as other aspects of vision like colour and contrast discrimination
452 abilities get progressively worse (Diederich et al., 2002).

453 Reduced ability to interpret and process depth cues may result in abnormal
454 susceptibility to the Ponzo illusion. Thus, an incorrect perception of an object's position in the
455 world (whether it appears as closer/further away than it is), could contribute to the increased
456 risks of falls in the elderly. In line with this assumption, many PD patients are shown to exhibit
457 difficulties in perceiving depth, experiencing both teleopsia (objects appear to be further away
458 than they are) and pelopsia (objects appear to be closer than they are; Sasaki et al., 2022).
459 Furthermore, it is unlikely that these differences observed between PD patients and controls
460 arise due to the abnormal role of top-down influences in susceptibility to the Ponzo illusion, as
461 such a deficit should also be observed for the Ebbinghaus illusion, which is considered a
462 context sensitivity illusion (Kaldy and Kovacs, 2003).

463 The Ebbinghaus illusion arises due to the perceptual system's top-down integration of
464 display elements (Kaldy and Kovacs, 2003). Our data show that susceptibility to the
465 Ebbinghaus illusion is not significantly different in PD, indicating typical abilities to integrate
466 context in this population. This finding aligns with previous research reporting intact top-down
467 influences on PD patients' responses in visual priming tasks (Straughan et al., 2016) and visual
468 search tasks (Horowitz et al., 2006). By contrast, Mannan and colleagues (2008) found that PD
469 patients were impaired in visual search tasks involving highly salient targets, indicating
470 difficulties with bottom-up processing. The illusions tested in this study belong to a category

471 of high-level VIs that rely on complex cognitive processing and top-down mechanisms,
472 whereas low-level VIs (e.g., the Brightness illusion) are mediated at the level of the retina and
473 bottom-up perception (King et al., 2017). While PD may not impact top-down processing
474 involved in experiencing complex VIs, deficiency of retinal dopamine may result in abnormal
475 susceptibility to low-level VIs. As deficiency in retinal dopamine results in a diminished ability
476 to differentiate contrast (as in colour, e.g., Pieri et al., 2000; Price et al., 1992), PD patients
477 could have higher thresholds in matching colour in Brightness or Adelson's Checkerboard
478 illusions. Therefore, we recommend that future research investigates whether susceptibility to
479 low-level VIs is affected by PD.

480 Our findings suggest that the pathophysiology of the basal ganglia and dopamine
481 deficits may not affect PD patients' sensitivity to the Muller-Lyer illusion. Therefore, illusions
482 such as the Ebbinghaus and Muller-Lyer may be subserved by neural mechanisms that are
483 largely free from pathophysiology in PD, such as those located in the visual cortex (Cheng et
484 al., 2011; King et al., 2017). The Muller-Lyer illusion is considered to rely on depth cues
485 (Gregory, 1966), just like the Ponzo illusion, which is considered a classic example of a depth
486 illusion (Gregory, 1963). Therefore, the inability to perceive depth cannot be a major factor
487 driving the illusion, at least in the version used here. In line, with Doherty and colleagues'
488 (2010) claims that subtle depth cues are likely to play a part in susceptibility to the Ebbinghaus
489 illusion, the depth cues in the Muller-Lyer illusion are also subtle, hence no differences in
490 susceptibility to those two illusions might have been observed. Thus, the pathophysiology of
491 the basal ganglia and/or dopamine deficits might only be related to more explicit perceptions
492 of depth, and are not directly linked with susceptibility to the Muller-Lyer illusion.

493 Overall, our observed results support the alternative hypothesis that susceptibility to
494 VIs is largely unaffected in PD patients due to their visual perception difficulties originating
495 from abnormalities in dorsal stream functioning, rather than ventral stream functioning. PD

496 patients showed similar susceptibility to the Ebbinghaus and Muller-Lyer illusions and only
497 marginal evidence for reduced susceptibility to the Ponzo illusion was observed. From this, we
498 conclude that perception of depth is more crucial for executing motor actions than the
499 integration of context. This is, in line with findings by Giovannini and colleagues (2006) who
500 observed that PD patients display abnormalities in their vision for action in a blind walking
501 task, but not a line-matching task. Arguably, the line-matching task does not rely on depth
502 integration, therefore PD patients performed similarly to controls.

503 Extending this line of research to grasping behaviour, which is guided by the dorsal
504 stream, would potentially provide valuable insight into differences between the dorsal and
505 ventral streams in PD. Previous findings on the dichotomy between the two streams have
506 largely focused on whether individual illusory effects are larger on the ventral stream than the
507 dorsal stream. Here, testing PD patients would allow for a different perspective; one would still
508 assume that the perceptual stream is affected by the illusion in both PD patients and healthy
509 controls, but the action stream is affected by the illusion only in PD patients.

510 This study has several limitations. Firstly, we did not directly assess our participants'
511 dopamine levels or pathophysiology of the basal ganglia. In line with other studies in the field
512 (e.g., Maschke et al., 2006), our target population was selected based on robust pre-existing
513 knowledge that PD is characterised by dopamine loss and basal ganglia pathophysiology which
514 are known to adversely affect visual perception. Therefore, our conclusions that dopamine loss
515 and the pathophysiology of the basal ganglia do not influence susceptibility to high-level VIs
516 should be interpreted with caution. Furthermore, the online administration of the study resulted
517 in several potential shortcomings. First, varying Internet speed could cause a lag in the delivery
518 of the experiment, impacting the smoothness of the increase/decrease of the targets which the
519 experimenter could not control for. Secondly, although participants were frequently reminded
520 to rely on their visual perception alone, the experimenter could not verify whether the

521 participants truly did so. Finally, our study did not check for the presence of everyday VIs (that
522 are similar to geometrical VIs, but they occur during everyday activities of the patients), that
523 recently gained interest in medical research on PD (Nishio et al., 2018; Sasaki et al., 2022).

524 In conclusion, our findings suggest that PD patients and neurotypical controls do not
525 differ in their susceptibility to the Ebbinghaus, Ponzo, and Muller-Lyer illusions. The lack of
526 differences was especially evident in the Ebbinghaus and Muller-Lyer illusions that more
527 strongly rely on context sensitivity rather than depth perception. Only a marginal indication of
528 abnormalities in depth perception was indicated by reduced susceptibility to the Ponzo illusion,
529 which compared to the other VIs is a classical illusion of depth. Collectively, our data suggest
530 that context integration, a key component of VIs susceptibility, remains unaffected in the early
531 to mid-stage of PD. Furthermore, our findings suggest that visual deficits in PD are more likely
532 to be related to the dorsal visual stream. This study makes a novel contribution to a growing
533 literature exploring visual deficits in PD and advances the understanding of how visual
534 perception may be affected by dopamine deficiency and abnormalities in the basal ganglia.

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In review

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In review

Figure 1.JPEG

In review

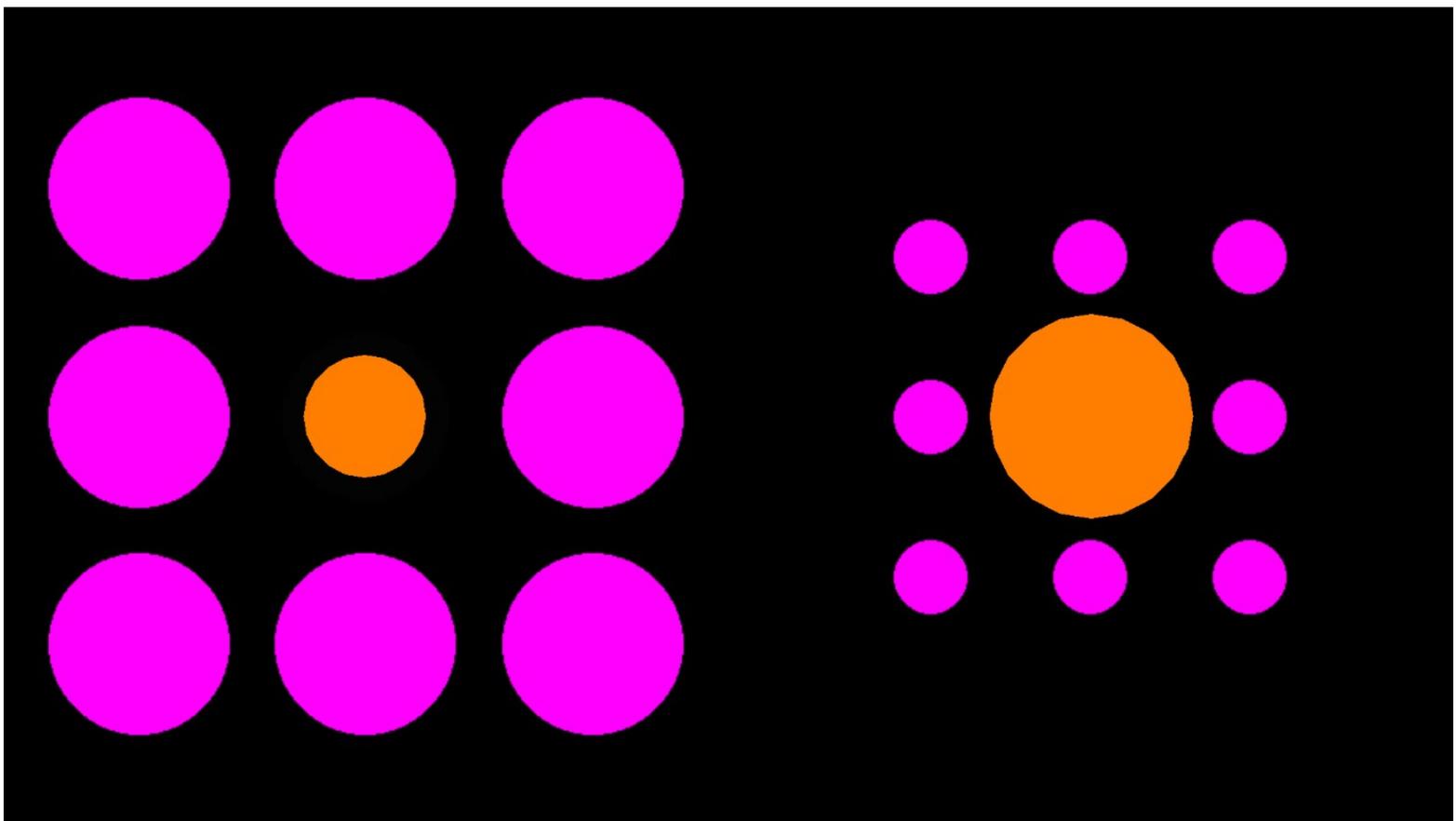


Figure 2.JPEG

In review

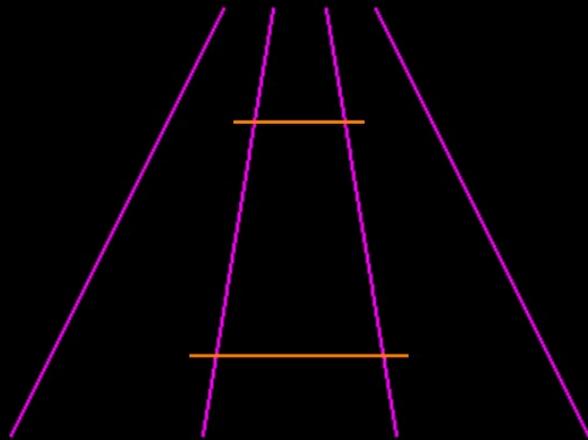
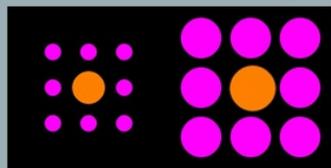


Figure 3.JPEG

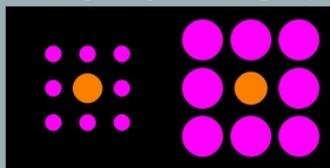


Figure 4.JPEG

In review



Adjustment



Participant's perceived equality

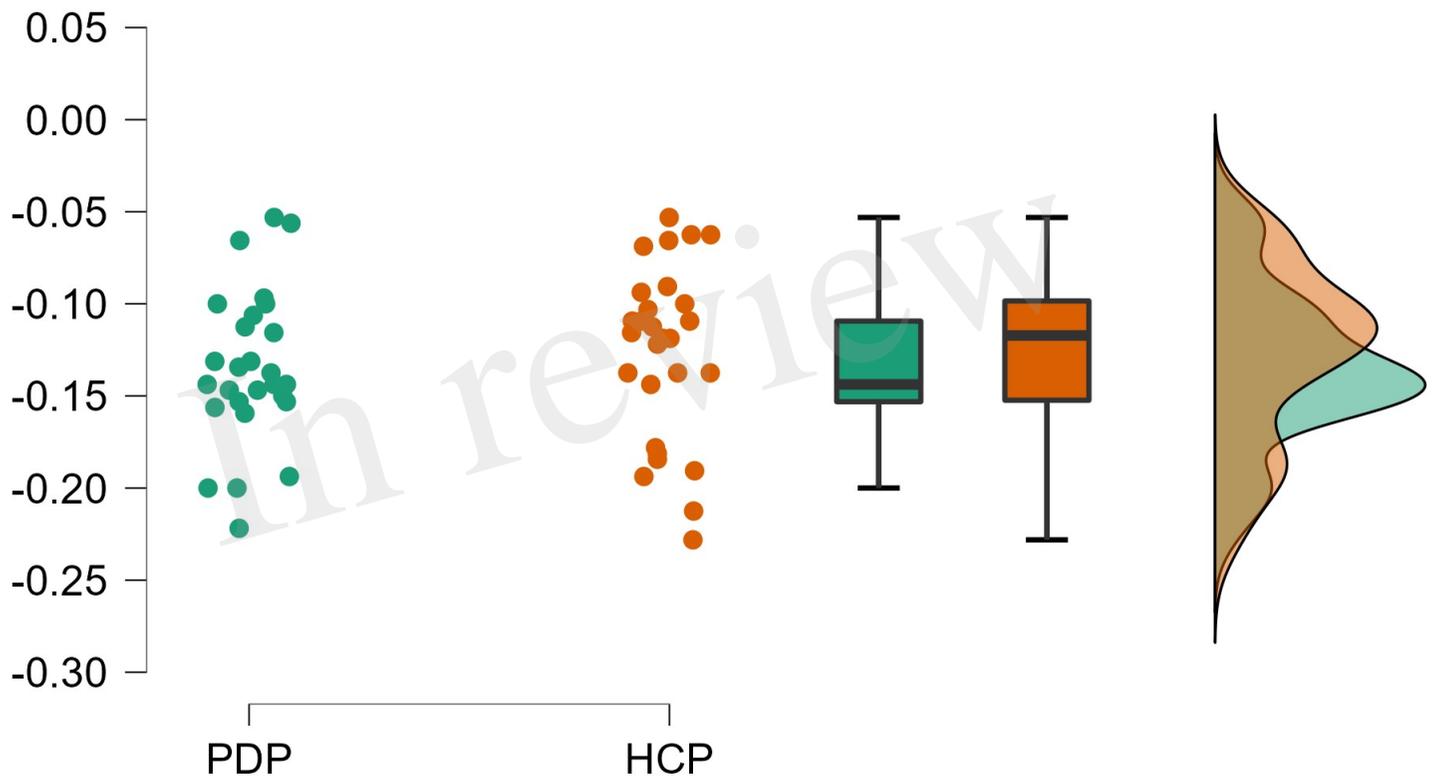
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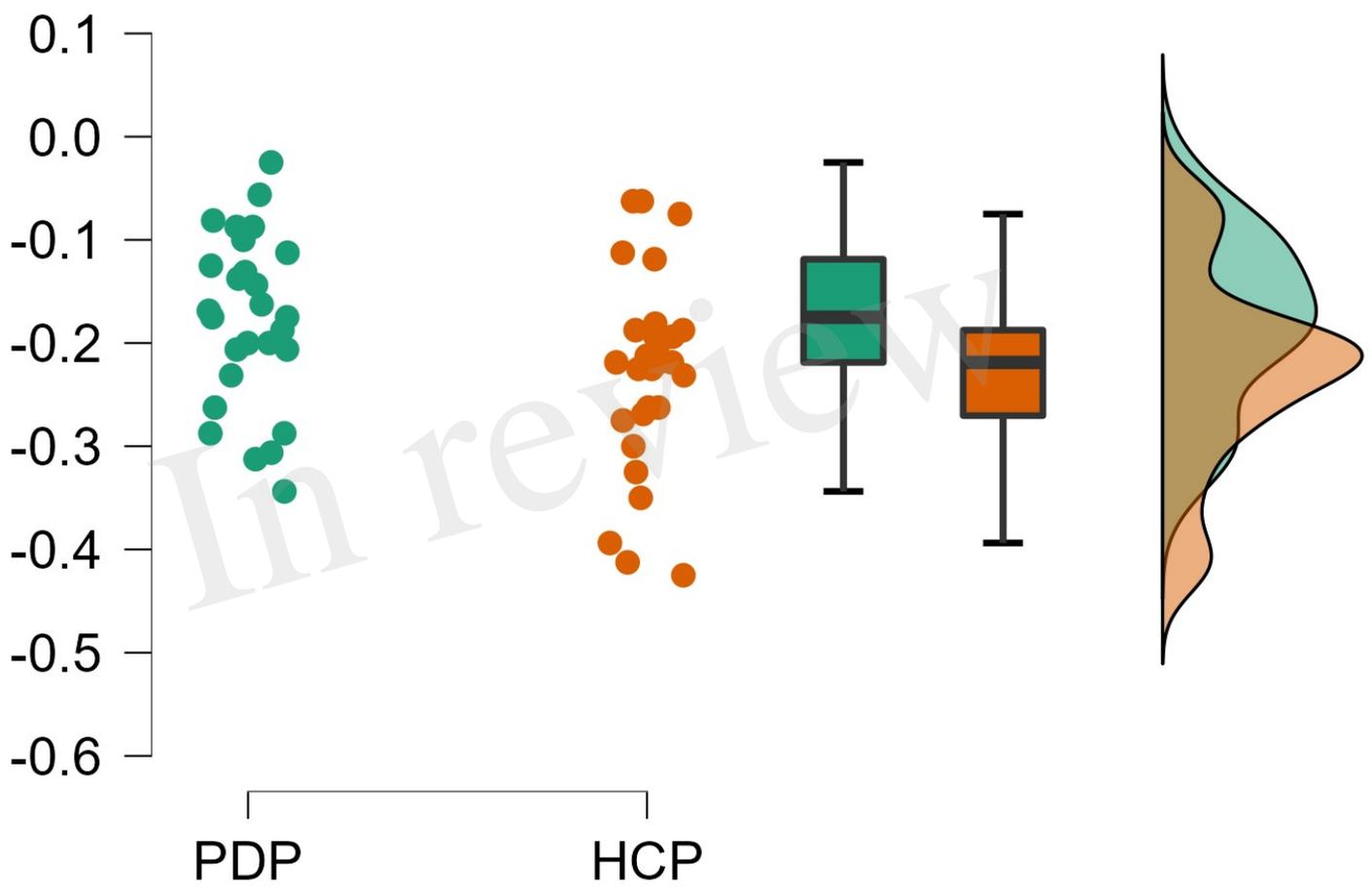
1-second mask

Next trial

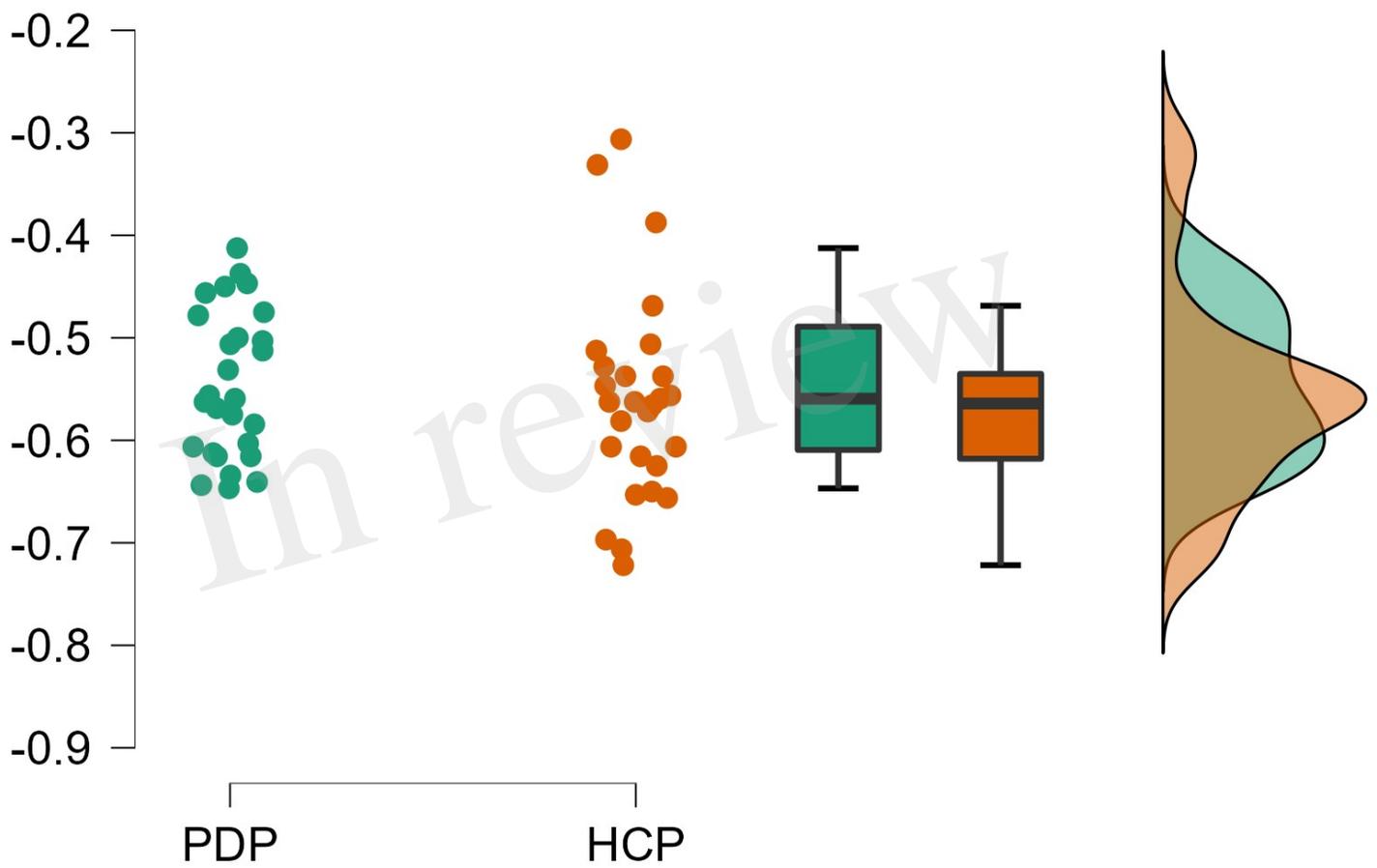
Figure 5.JPEG



Group Differences for the Ebbinghaus Illusion



Group Differences for the Ponzo Illusion



Group Differences for the Muller-Lyer Illusion