**Electrophysiology reveals two stages of face recognition impairment**

**in developmental prosopagnosia**

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**Abstract**

Event-related brain potentials (ERPs) were employed to study the time course of face recognition in developmental prosopagnosia (DP). Participants with DP and age-matched control participants with normal face recognition abilities had to detect a pre-experimentally unknown Target Face (“Joe”). Single faces were presented sequentially at fixation, and appeared in one of three possible views. On different trials, the face was the Target Face, one of seven different Nontarget Faces, or the participants’ Own Face (which had to be ignored). EEG was recorded during task performance, and two ERP components were measured as markers of matching a particular face to a stored representation in visual face memory (N250) and of attentional processes associated with the conscious awareness and recognition of a particular face (P600f). N250 components to Target Faces were present in the DP group, but they emerged reliably later than in the control group. N250 components to Own Faces did not differ between the two groups. P600f components were strongly attenuated in the DP group for both Target and Own Faces. These results show that the activation of visual face memory for previously unknown learned faces is delayed in DP, and that the transfer of activated visual face memory representations to central cognitive control structures involved in the attentional processing and conscious recognition of individual faces is also impaired. They imply that the face recognition deficits in DP are likely to be generated at multiple successive stages of face processing.

Keywords: Face processing; face recognition; prosopagnosia; event-related brain potentials

1. **Introduction**

Individuals with prosopagnosia are unable to recognize and identify the faces of familiar individuals, despite normal low-level vision and intellect (Bodamer, 1947). This problem can be caused by impairments at early perceptual stages of face processing (apperceptive prosopagnosia) or by selective deficits of long-term face memory (associative prosopagnosia; De Renzi, Faglioni, Grossi, & Nichelli, 1991). Acquired prosopagnosia (AP) usually results from lesions to face-sensitive regions in occipito-temporal visual cortex, including the fusiform gyri (e.g., Barton, 2008). In contrast, individuals with developmental prosopagnosia (DP) have no history of neurological damage (Behrmann & Avidan, 2005; Duchaine & Nakayama, 2006a; see Towler & Eimer, 2012; Susilo & Duchaine, 2013; for recent reviews). In DP, face recognition deficits are typically present from an early age, and are believed to be linked to a failure to develop normally functioning face recognition mechanisms. All individuals with DP have a core deficit in recognising familiar individuals, whereas other aspects of face processing may or may not be affected. For example, some DPs perform poorly on perceptual face matching tasks while others perform within the normal range (Duchaine, Yovel & Nakayama, 2007; Duchaine, 2011).

Functional neuroimaging studies of DP have identified face processing abnormalities in both posterior and anterior cortical regions. Experiments investigating fMRI responses to faces versus classes of non-face objects have generally observed relatively normal fMRI activation patterns within the core posterior face processing network (Hasson, 2003; Avidan, Hasson, Malach, & Behrmann, 2005; Avidan & Behrman, 2009; Furl, Garrido, Dolan, Driver, & Duchaine, 2011; Avidan, Tanzer, Hadj-Bouziane, Liu, Ungerleider, & Behrmann, 2014). However, temporal face areas were found to be reduced in size and showed less face-selectivity in DPs (Furl et al., 2011), and face-selective activation in the inferior anterior temporal lobe was absent in a group of DPs (Avidan et al., 2014). Other subtle structural differences between DP and control participants have been observed in multiple occipito-temporal regions (Behrmann, Avidan, Gao, & Black, 2007; Garrido, Furl, Draganski, Weiskopf, Stevens, Tan, Driver, Dolan, & Duchaine, 2009). Most event-related brain potential (ERP) studies of DP have focused on the face-sensitive N170 component that reflects an enhanced negativity to faces versus non-face objects between 150 and 200 ms after stimulus onset over lateral occipito-temporal areas (e.g., Bentin, Allison, Puce, Perez, & McCarthy, 1996; Eimer, Kiss, & Nicholas, 2010; Eimer, 2011; Rossion & Jacques, 2011). A recent study from our lab (Towler, Gosling, Duchaine, & Eimer, 2012) demonstrated that the generic face-sensitivity of the N170 does not differ between DPs and control participants (see also Towler, Gosling, Duchaine, & Eimer, 2014), but found atypical effects of face inversion on N170 amplitudes for individuals with DP.

Because the N170 is usually not affected by the familiarity of a face (Bentin & Deouell, 2000; Eimer, 2000), this component is believed to reflect processes involved in the perceptual structural encoding of faces that precedes the recognition and identification of individual faces. Studies focused on the N170 component alone will therefore not be able to obtain direct electrophysiological markers of impaired face recognition that is at the core of the face processing deficits in DP. Such markers emerge in ERP waveforms only at post-stimulus latencies beyond 200 ms. A repeated encounter with the face of a particular individual triggers an enhanced negativity at inferior occipito-temporal electrodes at around 250 ms after stimulus onset (e.g., Schweinberger, Pfütze, & Sommer, 1995; Begleiter, Porjesz, & Wang, 1995; Schweinberger, Pickering, Jentzsch, Burton, & Kaufmann, 2002; Zimmermann & Eimer, 2013). This repetition-induced N250r component has been linked to the activation of a representation of a specific face in visual memory that is triggered by its match with a currently presented face (Schweinberger & Burton, 2003). The N250r is larger for repetitions of famous faces as compared to unfamiliar faces (Herzmann, Schweinberger, Sommer, & Jentzsch, 2004), suggesting that pre-existing long-term representations of individual faces are activated particularly strongly by an identity match. A similar N250 component is also triggered by famous faces versus novel faces (e.g., Gosling & Eimer, 2011), and is assumed to reflect the match between a perceptual representations of a particular familiar face and a representations of the same face that is stored in long-term visual face memory.

In a recent ERP study (Eimer, Gosling, & Duchaine, 2012), we employed this N250 component to investigate famous face recognition in DP in a task where faces of famous and unfamiliar individuals had to be discriminated. Participants with DP correctly detected less than 30% of all famous faces (even though subsequent tests revealed that they knew 95% of these individuals). However, those relatively few famous faces that were successfully recognized triggered N250 components that were similar to those observed for participants with unimpaired face recognition (Gosling & Eimer, 2011). For six of the twelve DPs tested, N250 components were triggered by famous faces on trials when these faces were judged to be unfamiliar, suggesting that stored visual face representations can be activated even when faces are not explicitly recognized (covert recognition). The explicit recognition of a particular individual face is associated with a sustained broadly distributed positivity that emerges around 400 ms after stimulus onset. This late positive component (termed P600f by Gosling & Eimer, 2011) is similar in its time course and scalp distribution to the P3b component that is observed in many target-nontarget discrimination tasks, and is assumed to be linked to the allocation of attentional resources during the categorization or identification of task-relevant stimuli (e.g., Folstein & Van Petten, 2011). In our study (Eimer et al., 2012), P600f components were only elicited on trials where DPs correctly reported a famous face, in line with the view that the P600f reflects explicit face recognition.

Our previous ERP results (Eimer et al., 2012) show that when DPs successfully identify a pre-experimentally known famous face, the processes involved in the matching of perceptual and long-term memory representations (as reflected by the N250 component) and explicit face recognition (marked by the P600f component) are not qualitatively different from participants with unimpaired face processing abilities (see Towler & Eimer, 2012, for more detailed discussion). One question addressed in the present study was whether this is also the case for the recognition of pre-experimentally unfamiliar target faces in a task where these faces have to be discriminated from other unfamiliar nontarget faces. Another question was whether familiar faces will activate matching face memory representations in DPs when they are not task-relevant. To address these questions, we adopted an experimental paradigm that was developed by Tanaka, Curran, Porterfield, and Collins (2006). Single face images were presented sequentially, and participants had to respond to a previously studied but otherwise unknown target face (“Joe”), while ignoring other task-irrelevant distractor faces. One of these distractors was the participants’ own face. Tanaka et al. (2006) found that both target faces and participants’ own faces triggered occipito-temporal N250 components, even though the latter were task-irrelevant. This shows that the N250 reflects the activation of long-term face memory (one’s own face) as well as the activation of a recently learned face representation (the target face). The N250 to participants’ own face was already present in the first half of the experiment, while the N250 to target faces only emerged during the second half, suggesting that an episodic representation of a previously unfamiliar target face builds up gradually (see also Kaufmann, Schweinberger, & Burton, 2009). Target faces and participants’ own faces also elicited a sustained positivity that peaked around 500 ms post-stimulus, suggesting that attention was selectively allocated to these faces (Tanaka et al., 2006).

In the current experiment, ten participants with DP and a group of ten age-matched control participants had to memorize a particular target Face (“Joe”), in order to recognize photographs of this face among sequentially presented distractor face photographs. The stimulus set included seven unfamiliar nontarget faces, as well as photographs of each participant’s own face. In contrast to Tanaka et al. (2006), all faces appeared randomly in one of three possible views (see Figure 1). For the control group, both target faces and participant’s own faces should trigger occipito-temporal N250 components, reflecting a match between seen faces and stored short-term or long-term face memory representations. The N250 should be followed by a late positivity (P600f), which indicates the in-depth attentional processing and explicit recognition of a target face or one’s own face.

The critical question was whether the same pattern of ERP results would also be observed for participants with DP. Our earlier study (Eimer et al., 2012) has shown that long-term visual memory representations of famous faces are activated (as reflected by the N250 component) when DPs successfully recognize one of these faces. Since one’s own face is very familiar and thus presumably strongly represented in long-term face memory, own faces may also trigger N250 components in the DP group. However, some patients with severe AP fail to recognize themselves in the mirror (Sergent & Poncet, 1990) and some individuals with DP may also have difficulties in recognizing their own face (e.g., Duchaine, Germaine, & Nakayama, 2007). In addition, and in contrast to our previous study where famous and unfamiliar faces had to be discriminated (Eimer et al., 2012), participants’ own faces were now task-irrelevant. The presence of N250 and P600f components to own faces in the DP group would demonstrate that highly familiar faces trigger face recognition processes in DPs even when they have to be ignored. We also tested whether these ERP correlates of face recognition would also be present for target faces in the DP group. Unlike famous faces and one’s own face, these faces were pre-experimentally unfamiliar and thus not represented in long-term face memory. If DPs are able to acquire and activate short-term representations of novel target faces and explicitly recognize these faces in a face identity matching task, N250 and P600f components should be elicited to target faces in the DP group. Any delay in the emergence of the N250 to target faces for DPs as compared to controls would indicate the visual memory representations for learned unfamiliar target faces are activated less rapidly in individuals with DP.

1. **Methods**

* 1. **Participants**

Ten participants with developmental prosopagnosia (5 females; aged 21-58 years; mean age 40.0 years) and ten gender and age-matched control participants (5 females; aged 25-54 years; mean age 39.1 years) were tested. All participants gave written informed consent prior to the experiment, and all had normal or corrected-to-normal vision. None of the control participants reported any face recognition difficulties in real life. In contrast, all developmental prosopagnosics reported problems with face recognition since childhood. A battery of behavioural tests was administered on the two separate testing sessions to assess their reported face recognition deficit. Table 1 shows the performance of each of the ten participants with DP expressed as z-scores in four behavioural tests.

In the Famous Faces Test (FFT), participants have to identify the faces of 60 famous individuals from the popular culture such as actors, musicians, politicians or athletes. In the Cambridge Face Memory Test (CFMT), participants’ task is to memorize six target faces photographed from three different angles. In the subsequent test phase, one of the target faces has to be discriminated from the two simultaneously presented distractor faces (for details see Duchaine & Nakayama, 2006b). The Old-New Face Recognition test (ONT, Duchaine & Nakayama, 2005) requires participants to memorize ten target faces. In the test phase, these target faces are presented amongst 30 novel faces, and an old/new discrimination is required on each trial. In the Cambridge Face Perception Test (CFPT, Duchaine et al., 2007) one target face in a three-quarter view is shown above the six frontal-view morphed test faces that contain a different proportion of the target face. These test faces have to be rearranged according to their similarity to the target face. Faces are presented either upright or inverted. As can be seen in Table 1, all DPs had severe impairments in the face recognition tests (with z-values below -2 in the FFT, CFMT, and ONT for each of the ten participants with DP), and most of them also showed some deﬁcits in face perception.

***2.2* Materials and procedure**

Stimuli were photographs of the faces of eight different individuals and of each participant’s own face that were taken under identical lighting conditions in three different views (front view, 30° side view, 60° side view, see Figure 1). Each participant was photographed prior to the experimental session and was told that the images would be added to our face database. They were not informed explicitly that their own face would be the part of the current experiment. All face images were converted into grayscale, cropped to remove external facial features, including the hairline, and resized to create identical-size images for each for the three views, using Creative Suite 6 software (Adobe Photoshop).

All face stimuli were presented at the centre of a CRT monitor against a light grey background (15 cd/m2) at a viewing distance of 100 cm. The visual angle covered by a face was 4.3° x 3.1° (front and 30° side view) or 4.3° x 2.9° (60° side view). The average luminance of the face images was 8 cd/m2. Ten successive experimental blocks were run, with 81 trials per block. On each trial, one face was presented at fixation for 400 ms. The interval between the offset of a face and the onset of a face on the next trial was 1100 ms. Participants’ task was to detect a pre-specified Target Face (“Joe”, see Figure 1), to press a response key with their right index finger whenever the Target Face was presented in any of its three possible views, and to refrain from responding to all other faces. The same individual face (shown in three different views) served as target for all participants in this study. In each block, the Target Face was presented on 9 trials, and participants’ Own Face on another 9 trials. In the remaining trials, Nontarget Faces were presented. Each of the seven Nontarget Faces appeared on 9 randomly interspersed trials. The view in which a particular face was presented (front view, 30° side view, 60° side view) was randomly determined for each trial.

Prior to the first experimental block, participants were shown each of the three views of the Target Face “Joe” on the computer screen for 5 s, and were asked to memorize this individual face. Next, they were given a training block of 40 trials. On 10 of these training trials, “Joe” was presented. On the other 30 trials, the face of one of three other individuals was shown. These three nontarget faces were not used in the main experiment. At the end of the experiment, participants were briefed about the purpose of the experiment and asked whether they could identify any of the faces shown. Two participants with DP reported to have been unaware of the presence of their own face during the experiment.

***2.3.* Electroencephalography recording and data processing**

EEG was DC-recorded with a BrainAmps DC ampliﬁer (Brain Products, Munich, Germany; high cut-off filter 40 Hz, 500 Hz sampling rate) and Ag–AgCl electrodes mounted on an elastic cap from 23 scalp sites (Fpz, F7, F3, Fz, F4, F8, FC5, FC6, T7, C3, Cz, C4, T8, CP5, CP6, P7, P3, Pz, P4, P8, PO7, PO8 and Oz, according to the extended international 10–20 system). Horizontal electrooculogram (HEOG) was recorded from the outer canthi of both eyes. An electrode placed on the left earlobe served as the reference for online recording, and EEG was re-referenced off-line to the average of both earlobes. Electrode impedances were kept below 5 kΩ. No off-line ﬁlters were applied.

The EEG was epoched ofﬂine from 100ms before to 700ms after stimulus onset. Epochs with EEG activity exceeding 30µV in the HEOG (horizontal eye movements) or 60µV at electrode Fpz (eye blinks or vertical eye movements) were excluded from subsequent analysis, as were epochs with voltages exceeding 80µV at any other electrode. Because these rejection criteria led to a loss of more than 50% of all trials for three participants with DP, artefact rejection thresholds were increased by 10 µV for one participant and by 20 µV for the other two participants. Analyses were focused on the N250 and P600f components, which were quantified on the basis of ERP mean amplitudes measured at lateral posterior electrodes P7 and P8 in the 250-400 ms post-stimulus time window (N250 component) and at midline electrode Pz in the 400-700 ms time window (P600f component).

Separate analyses on ERP mean amplitudes were conducted for ERPs to Target Faces versus Nontarget Faces, and ERPs to Own Faces versus Nontarget Faces, with the within-participant factor face identity (Target/Nontarget or Own/Nontarget) and between-participant factor group (DP/control). For the main analyses reported below, ERPs to Nontarget Faces were based on all trials where these faces were presented. Because Nontarget Faces appeared more frequently than Target or Own Faces, comparisons of ERPs between these three different face types might be affected by differences in signal-to-noise ratios, and/or the fact that faces of different individuals contributed to Nontarget ERPs, but not to Target and Own Face ERPs. For these reasons, we conducted additional analyses with Nontarget Face ERPs that were computed for a single Nontarget Face. This Nontarget Face was randomly selected for each participant, with the restriction that each Nontarget Face was selected at least once for a DP participant and a control participant, respectively. ERPs to Target Faces only included trials where this face was correctly reported, and ERPs to Own and Nontarget Faces were based on trials where no response was recorded. Because participants with DP missed only 13% of all Target Faces (see below), ERPs to undetected Target Faces could not be computed due to an insufficient number of trials. Analyses of N250 amplitudes also included the factor hemisphere (P7/P8). Additional follow-up analyses were conducted separately for the DP and control groups, and for N250 components measured in the first half of the experiment (blocks 1 – 5). To determine whether the activation of visual face memory by Target Faces or Own Face started later in the DP group relative to the control group, we employed a jack-knife based procedure (Miller, Patterson, & Ulrich, 1998) to estimate N250 onset latencies and compare them between the two groups. Difference waveforms were computed by subtracting ERPs to Nontarget Faces from ERPs to Target Faces and Own Faces, respectively. N250 onset latencies were determined from grand averages that were computed for ten subsamples of participants in each group (where one of the ten participants in each group was successively excluded from the original sample). N250 onset was defined according to an absolute criterion where the voltage on the ascending flank of the N250 difference wave exceeded a threshold value of -1 µV within a 200-400 ms post-stimulus time window. N250 onset latency differences between the two groups were compared with paired *t*-tests, with *t* values) corrected according to the formula described by Miller et al. (1998). P600f components were quantified on the basis of mean amplitudes measured at Pz in the 400 – 700 ms post-stimulus interval.

1. **Results**
   1. **Behavioural results**

As expected, target detection performance was impaired for participants with DP relative to control participants. Reaction times (RTs) on trials where the target face was successfully detected were more than 150 ms slower in the DP group (674 ms; SD: 126.4 ms) than in the control group (520 ms; SD: 80.6 ms), and this difference was reliable, *t*(18) = 3.25, *p* < .005). DPs detected 87% of all target faces, while control participants correctly responded on 97% of all target trials. This difference between groups was significant, *t*(18) = 2.872, *p* < .01. False Alarms to nontarget faces also occurred more frequently in the DP group relative to the control group (3.35% versus 0.25%; *t*(18) = 2.87, *p* < .02).

* 1. **ERP results**

Figure 2 shows grand averaged ERP waveforms elicited at lateral occipito-temporal electrodes P7 and P8 in response to the Target Face (“Joe), the participants’ Own Face, and Nontarget Faces in the 700ms epoch after stimulus onset, for participants with DP (top panel) and control participants (bottom panel). Visual N1/N170 components were generally larger in the DP group than in the control group. A main effect of group (DPs versus controls) for N1 mean amplitudes (measured for the 150-200 ms post-stimulus interval), *F*(1,18) = 14.2, *p* < .001, ηp2=.26, that did not interact with hemisphere (left versus right), *F* < 1, confirmed this observation.

*N250 components to* *Target versus Nontarget Faces.* As can be seen in Figure 2, Target Faces triggered N250 components in both groups. This was confirmed in an ANOVA of N250 mean amplitudes at electrodes P7/P8 with the factors face identity (Own versus Nontarget Face), hemisphere, and the between-participant factor group (DPs versus controls). There was a main effect of face identity, *F*(1,18) = 23.9, *p* < .001, ηp2=.57,that did not interact with group, *F*(1,18) = 1.5, confirming that N250 components in response to Target Faces were similar in size in both groups. There was no identity x hemisphere interaction, *F* = 1.6. Analyses conducted separately for both groups confirmed the presence of reliable N250 components to Target Faces in the control group, *F*(1,9) = 16.7, *p* = .003, ηp2=.65, as well as in the DP group, *F*(1,9) = 7.7, *p* = .02, ηp2=.46. Analyses conducted for ERPs to Target and Nontarget Faces recorded in the first five blocks of the experiment revealed a reliable effect of face identity, *F*(1,18) = 17.8, *p* = .001, ηp2=.49, that did not interact with group, *F* < 2, demonstrating that Target N250 components were already present in the first half of the experiment in both control participants and DPs.

Importantly, the N250 to Target Faces emerged later in the DP group than in the control group. This is illustrated in Figure 3 (left panel), which shows ERP difference waveforms for right-hemisphere electrode P8 obtained by subtracting ERPs to Nontarget Faces from ERPs to Target Faces. Jackknife-based N250 onset comparisons showed that the N250 component was delayed by 40 ms (322 ms versus 282 ms; *tc*(1,9) = 2.76, *p* = .02).

Analyses with Nontarget ERPs that were computed for a single randomly selected Nontarget Face yielded essentially identical results. A main effect of face identity, *F*(1,18) = 22.4, *p* < .001, ηp2=.55, that did not interact with group, *F* < 1, showed the presence of N250 components to Target Faces in both groups. This component already emerged in the first half of the experiment, *F*(1,18) = 9.6, *p* =.006, ηp2=.35, in both groups, as shown by the absence of an interaction between face identity and group, *F* < 1. The N250 emerged later in the DP group relative to the control group, as reflected by a reliable onset latency difference at right-hemisphere electrode P8 (339 ms versus 292 ms; *tc*(1,9) = 2.45, *p* = .04).

*N250 components to* *Own versus Nontarget Faces.* As can be seen in Figure 2, Own Faces elicited N250 components in both groups, which emerged earlier than the N250 to Target Faces, analogous to earlier observations by Tanaka et al. (2006). The similar time course of the N250 to Own Faces in both groups is illustrated in the Own Face – Nontarget Face difference waveforms in Figure 3 (right panel). N250 mean amplitudes to Own and Nontarget Faces at electrodes P7/P8 were analysed in an ANOVA with the factors face identity (Own versus Nontarget Face), hemisphere, and group. There was a main effect of face identity, *F*(1,18) = 14.2, *p* = .001, ηp2=.44, that did not interact with hemisphere, *F*(1,18) = 2.8, or with group, *F*(1,18) < 1, indicating that Own Faces elicited reliable N250 components for participants with DP and control participants. The presence of reliable N250 components to Own Faces for both groups was confirmed by follow-up analyses conducted separately for each group, which revealed significant effects of face identity, *F*(1,9) =11.7, *p* = .008, ηp2=.56, and *F*(1,9) = 5.8, *p* = .04, ηp2=.39, for the control and DP groups, respectively. The N250 component to Own Faces was already reliably present in the first half, *F*(1,18) = 6.95, *p* = .02, and did not interact with group, *F* < 1, confirming that N250 components to Own Faces emerged early for both DPs and control participants. As can be seen in Figure 3 (right panel), there was no difference in the onset of the N250 component to Own Faces between the two groups, *tc* < 1.

Again, analyses based on Nontarget ERPs that were computed for a single randomly selected Nontarget Face yielded identical results. A main effect of face identity, *F*(1,18) = 11.9, *p* = .003, ηp2=.39, that did not interact with group, *F* < 1, showed the presence of N250 components to Own Faces in both groups. This component was already present in the first half of the experiment, *F*(1,18) = 8.8, *p* = .008, ηp2=.33, and did not interact with group, *F* < 1. There were no N250 onset latency differences between the DP and Control groups, *tc* < 1.

*P600f components.* At electrodes P7 and P8 (shown in Figures 2 and 3), N250 components to Own and Target Faces were followed at around 400 ms post-stimulus by a sustained positivity in the control group, which was appeared to be absent in the DP group. As can be seen in Figure 4 (top panel), this late positive component (P600f) elicited by Own Faces and Target faces (relative to Nontarget Faces) was much larger at posterior midline electrode Pz. The scalp topographies of P600f components to Target Faces and Own Faces are shown Figure 4 (bottom panel) for the DP and control groups. In control participants, the P600f to Target and Own Faces was broadly distributed across posterior and more anterior electrodes, with a maximum at posterior midline electrode Pz. In the DP group, P600f amplitudes appear to be considerably smaller.

To assess these differences statistically, ERP mean amplitudes measured at Pz in the 400–700 ms post-stimulus time window were compared between Target and Nontarget Faces, and between Own and Nontarget Faces, in two analyses with the factors face identity and group. For Target versus Nontarget Faces, a main effect of face identity, *F*(1,18) = 30.31, *p* < .001, ηp2=.63, was accompanied by an interaction between face identity and group, *F*(1,18) = 6.92, *p* = .02, ηp2=.28, confirming that P600f components to Target Faces were smaller in the DP group. Follow-up analyses showed that the P600f elicited by Target Faces was reliably present in the control group, *F*(1,18) = 22.9, *p* < .001, ηp2=.77, but was only marginally significant in the DP group, *F*(1,18) = 4.6, *p* = .06, ηp2=.34 For Own versus Nontarget Faces, a main effect of face identity, *F*(1,18) = 75.5, *p* < .001, ηp2=.81, was again accompanied by an interaction with group, *F*(1,18) = 6.3, *p* = .02, ηp2=.26, showing that the reduction of the P600f to Own Faces in the DP group was reliable. Follow-up analyses revealed the that the Own Face P600f was present not only in the control group, *F*(1,18) = 71.9, *p* < .001, ηp2=.89, but also for participants with DP, *F*(1,18) = 17.0, *p* = .003, ηp2=.65.

The parallel set of analyses that was based on Nontarget ERPs for a single randomly selected Nontarget Face yielded identical results, confirming the attenuation of P600f components to Target Faces and Own Faces in the DP group. For Target versus Nontarget Faces, there was a main effect of face identity, *F*(1,18) = 29.2, *p* < .001, ηp2=.62, and an interaction between face identity and group, *F*(1,18) = 6.8, *p* = .02, ηp2=.27. Similarly, for Own versus Nontarget Faces, a main effect of face identity, *F*(1,18) = 61.4, *p* < .001, ηp2=.77, was accompanied by an interaction with group, *F*(1,18) = 4.8, *p* = .04, ηp2=.21.

1. **Discussion**

Individuals with DP have severe difficulty in recognising the faces of familiar individuals, but the causes for this impairment are still largely unknown. We investigated whether the processes involved in the recognition of a task-relevant face that was previously unfamiliar and thus involves the activation of learned short-term face memory representations differ between a group of participants with DP and a group of age-matched unimpaired control participants. We also tested the recognition of participants’ own face in DP (i.e., the activation of a long-term representation) under conditions where the own face was task-irrelevant.

As expected, the DP group performed considerably worse than the control group in detecting the Target Face (“Joe”) among other task-irrelevant faces. Response times on trials where the Target Face was successfully detected were delayed by 150 ms in the DP group, and participants with DP failed to report the presence of the Target Face more often (on 13% of all trials) than unimpaired control participants (on 3% of all trials). Given the severe face recognition impairments revealed for all ten DPs in the CFMT (see Table 1), the fact that they were able to detect the memorized Target Face on most trials is remarkable, especially because these faces could appear in three different views. This dissociation between the DPs’ poor performance in the CFMT and their relatively high accuracy in to the task employed in the ERP experiment is most likely due to the differences in the memory demands of these two tasks. While only one task-relevant individual (“Joe”) had to be memorized in the ERP experiment, the CFMT required the simultaneous maintenance of the faces of six different individuals shown from three different angles. The fact that DPs were reasonably accurate in the “Joe” task shows that they are able to activate and maintain visual representations of one particular task-relevant face, and to successfully use these representations in a face identity matching task. Their poor performance in the CFMT reflects the much higher memory demands of this task, and demonstrates that the face recognition impairments in DP are particularly pronounced when multiple representations of individual faces have to be simultaneously retained and matched to a particular test face.

As can be seen in Figure 2, N1/N170 components were generally larger in the DP group relative to the control group, similar to our previous study that focused on the N170 component in DP (Towler et al., 2012). The amplitudes of visual-evoked components such as the P1 and N1 typically vary considerably across participants. This is linked to variability in the specific spatial orientation of neural generator processes in the visual cortex of individual participants, which determines the size of visual ERP components recorded from the scalp surface. The observation that N1 amplitudes were larger in the DP group is likely to reflect such subtle anatomical differences between individual participants in the two groups.

More importantly, differences in the processing of Target versus Nontarget faces between the DP and control groups were revealed by N250 and P600f components. A reliable N250 component was triggered by Target Faces in both groups, and this component did not differ in size between DPs and controls, indicating that the activation of a short-term memory trace of a previously unfamiliar learned task-relevant face operated in a qualitatively similar fashion in DPs and controls. Importantly, the onset of the N250 to Target Faces was reliably delayed by approximately 40 ms in the DP group (Figure 3). This observation provides the first direct evidence that the time course of face identity processing is altered in DP. The activation of short-term memory representations of a recently learned individual face by a matching seen face is delayed in individuals with DP relative to age-matched unimpaired control participants. Since the N250 is assumed to reflect an early visual stage of face recognition (Schweinberger & Burton, 2003; Gosling & Eimer, 2011), the delayed onset of this component in DPs is likely to reflect a deficit in perceptual face processing (e.g., an impairment in representing identity-specific properties of a currently seen face) and/or in visual aspects of face memory (e.g., lower precision of stored representations of a learned Target Face). In either case, the temporal delay in the onset of face identity matching processes is likely to contribute to the face recognition impairments that are characteristic for individuals with DP. It should be noted that the onset delay observed for the N250 component in the DP group relative to the control group was considerably smaller than the difference in RTs to correctly detected target faces between the two groups (150 ms). This suggests that in addition to a delay in face identity matching, other factors contributed to the impaired task performance observed for individuals with DP.

The fact that the P600f component to Target Faces was present but reliably reduced in the DP group relative to the control group suggests that identity-related processes that follow the initial matching of perceptual and memorized face representations also differed between the two groups. The P600f has been linked to the attentional processing and the explicit recognition of individual task-relevant faces, as well as to the retrieval of semantic or episodic information about these faces (e.g., Tanaka et al., 2006; Gosling & Eimer, 2011; Eimer et al., 2012). Because the Target Face “Joe” was pre-experimentally unfamiliar, the P600f component triggered by this face is unlikely to be linked to an activation of semantic or episodic memory, and should thus primarily reflect focal-attentional processing and explicit face recognition. The attenuation of the P600f to Target Faces for participants with DP therefore suggests that these processes were less target-selective or less consistently elicited across trials in the DP group. A reduced selectivity in the attentional processing of Target versus Nontarget Faces in this group is likely to affect the explicit individuation of a particular face as target, and will thus delay responses to Target Faces relative to the control group. In addition, the P600f amplitude reduction for Target Faces in the DP group may also reflect increased temporal variability of face recognition processes for DP participants, which could also be due to the less efficient allocation of focal attention to Target Faces.

The inclusion of participants’ Own Faces enabled us to investigate whether face-based self-recognition processes might also be impaired in DP. In contrast to Target faces, which were pre-experimentally unfamiliar, one’s own face is a highly familiar stimulus that is represented in long-term memory. In our previous ERP study of famous face recognition in DP (Eimer et al., 2012), successfully recognized famous faces triggered an N250 component, suggesting that the activation of visual face memory during the recognition of familiar task-relevant faces is not selectively impaired in individuals with DP. In contrast to this earlier study, Own Faces were task-irrelevant in the present experiment. In spite of this fact, Own Faces elicited very similar N250 components in both groups. In the control group, Own Faces triggered N250 components that were followed by a P600f, confirming previous observations (Tanaka et al, 2006). For DPs, a reliable N250 was also present in response to Own Faces. The size and onset latency of this N250 component did not differ between the DP and control groups, indicating that the activation of long-term representations of highly familiar faces in visual memory is not impaired in DP. In contrast, P600f components to Own Faces were reduced in size across all ten DPs tested relative to the control group. This suggests that later processes linked to the selective attentional processing and explicit recognition of one’s own face may operate less efficiently in participants with DP.

Because no responses were required to Own Faces, it was not possible to determine whether or not DPs had detected their Own Face on individual trials. The reduction in P600f amplitudes across all Own Face trials in the DP group may therefore be due to DPs failing to recognize their own faces on a subset of these trials. In fact, two of the ten DPs tested did not recognize that their Own Face was present among the Nontarget Faces in the experiment. For these two DPs, P600f components to Own Faces were entirely absent. This is illustrated in Figure 5, which shows ERPs by Own Faces and Nontarget Faces at electrode Pz, separately for the eight DPs we reported to have been aware of the presence of their Own Face and the two DPs who were not. Own Faces elicited a distinct P600f component in the former group, and additional analyses confirmed that an enhanced positivity for Own Faces as compared to Nontarget Faces was present for all eight DPs in this group. The size of the P600f for these eight participants was numerically but not reliably smaller than the P600f to Own Faces measured in the control group (*p* = .094). The absence of a P600f to Own Faces for two participants with DP is consistent with previous observations that this component is absent in response to famous faces that DPs fail to recognize (Eimer et al., 2012), and thus provides further evidence that the P600f component is closely linked to explicit face recognition. For one of the two DPs who failed to recognize their Own Face, the N250 component was also absent, while the other DPs (as well as all other individuals with DP) showed N250 components to Own Faces at lateral posterior electrodes. The dissociation between the activation of visual face memory (as reflected by the N250) and explicit face recognition (marked by the P600f) observed for one DP in the current study is in line with previous N250 evidence for the covert recognition of famous faces in DP (Eimer et al., 2012), and suggests that face recognition impairments can result from a disconnection between early visual stages of identity-related face processing and subsequent stages that mediate conscious access to a particular facial identity.

In contrast to the earlier study by Tanaka et al. (2006), who found that N250 components to Own Faces were present from the start of the experiment, while the N250 to Target Faces emerged reliably only in the second half, we found that N250 components to both Own and Target Faces were already significant during the first five blocks of the current study. There was also no difference in the early emergence of the N250 to Target Faces between the DP and control groups. One difference between these two studies was that the present experiment included a 40-trial training block where the Target Face had to be identified among distractor faces, while participants in the Tanaka et al. (2005) were merely asked to study the Target face prior to the start of the experiment. The inclusion of a training block may be responsible for the earlier emergence of N250 components to Target Faces in the present study. Another difference was that faces could appear in one of three possible views in the current experiment, while all faces were shown in a front view by Tanaka et al. (2006). Because different views of the previously unfamiliar Target Face had to be memorized and detected in our task, it is likely to have involved a view-independent working memory representation of its identity. In contrast, target detection in the Tanaka et al. (2006) study could have been based primarily on image-based view-dependent representations. Representations of facial identity may be formed more rapidly in face learning tasks where task-relevant faces appear in multiple views and image-based cues are therefore less useful than when all faces are encountered from the same view.

The delayed onset of N250 components to Target Faces observed in this study for participants with DP may be linked to impaired connectivity between posterior face-selective brain areas and anterior regions in the temporal and frontal cortex in DP (Thomas, Avidan, Humphreys, Jung, Gao, & Behrmann, 2008). If view-independent representations of individual faces are stored in anterior temporal cortex (Anzellotti, Fairhall, & Caramazza, 2013), a reduction in the density of white matter tracts connecting this region to posterior occipito-temporal face-selective regions could result in the delay in the onset of visual face identity matching processes found in this study for DPs. The fact that such delay was only observed for previously unfamiliar Target Faces but not for participant’s Own Face suggests that this deficit may be most pronounced during the learning of new facial identities. The additional reduction in the connectivity between posterior face-selective visual cortex and frontal cortex in DP that was also described by Thomas et al. (2008) could be linked to further impairments in the subsequent attentional processing and conscious recognition of relevant face identities that was reflected by the attenuation of P600f components observed for participants with DP in the present study.

In summary, the present ERP study has provided several new insights into the modus operandi of face recognition processes in DP, and into how these processes differ from face recognition mechanisms in neuro-typical individuals. The detection of task-relevant faces of a particular previously unfamiliar individual is slowed in DP, due to a systematic delay in the activation of recently acquired short-term visual face representations as well as to additional impairments in the subsequent focal-attentional analysis of task-relevant faces and their explicit recognition. There appear to be no systematic differences in the activation of visual face memory by photographs of participants’ Own Faces between DPs and controls, but even for these personally highly relevant faces that are stored in long-term memory, explicit self-recognition processes can be impaired in DP, up to the point where the presence of one’s own face goes entirely undetected.

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**Figure Legends**

**Figure 1.** The target face (“Joe”), shown from the three viewpoints (front view, 30° side view, 60° side view) in which it was presented in this study.

**Figure 2.** Grand-averaged ERPs elicited at lateral temporo-occipital electrodes P7 (left hemisphere) and P8 (right hemisphere) in the 700 ms interval after stimulus onset in response to Target Faces, Own Faces, and Nontarget Faces. ERPs are shown separately for the DP group (top panel) and the age-matched control group (bottom panel). Target Faces and Own Faces triggered N250 components in both groups.

**Figure 3.** N250 difference waveforms obtained for right posterior electrode P8 by subtracting ERPs to Nontarget Faces from ERPs to Target Faces (left panel) or from ERPs to Own Faces (right panel), separately for the control group and the DP group. The onset of the N250 component to Target Faces was delayed in the DP group, while no such delay was present for the N250 to Own Faces.

**Figure 4.** Top panel: Grand-averaged ERPs elicited at posterior midline electrode Pz in the 700 ms interval after stimulus onset in response to Target Faces, Own Faces, and Nontarget Faces, shown separately for the DP group (left panel) and the control group (right panel). P600f components to Target Faces and Own Faces were strongly attenuated in the DP group. Bottom panel: Topographical maps showing the scalp distribution of P600f components to Target Faces and participant’s Own Face, for the DP group (left) and the control group (right). These maps were computed by subtracting ERP mean amplitudes measured in the 400 – 700 ms post-stimulus interval in response to Nontarget Faces from ERPs to Target Faces and Own Faces, respectively.

**Figure 5.** ERPs elicited at posterior midline electrode Pz in the 700 ms interval after stimulus onset in response to Own Faces and Nontarget Faces, shown separately for those eight participants with DP who reported to have been aware of the presence of their Own Face during the experiment (left panel) and for the other two DPs who failed to recognize their Own Face (right panel). P600f components to Own Faces were absent for the two DPs who were unaware of their Own Face.

**Table 1.** Z-values for the ten DP participants included in this study on the Famous Faces Test (FFT), the Cambridge Face Memory Test (CFMT), the Cambridge Face Perception Test (CFPT) with upright or inverted faces, and the Old-New Test (ONT).

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Participant** | **Age** | **Sex** | **FFT** | **CFMT** | **CFPT**  **upright** | **CFPT**  **inverted** | **ONT** |
| MZ | 51 | F | -4.25 | -2.52 | -1.33 | 0.22 | -6.47 |
| JG  CC  CM  CT  MW | 45  30  31  40  58 | M  F  M  M  M | -8.88  -5.02  -7.72  -5.97  -3.67 | -2.77  -2.52  -4.29  -2.64  -2.14 | -2.56  -1.74  -3.1  -1.19  -1.6 | -0.63  -0.49  -2.89  1.64  -0.2 | -8.16  -5.69  -14.34  -2.78  -6.49 |
| KS  DD  GW  JA | 31  45  21  48 | F  M  M  F | -8.49  -5.21  -8.49  -5.41 | -2.9  -2.77  -2.52  -2.64 | -0.92  0.17  -1.33  -0.92 | -1.05  -0.77  -1.05  -0.49 | -9.03  -3.36  -6.41  -3.35 |
|  |  |  |  |  |  |  |  |

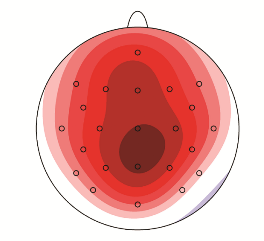
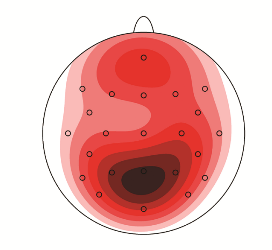
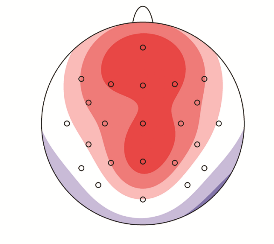
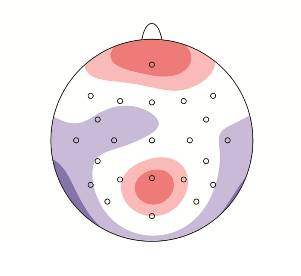
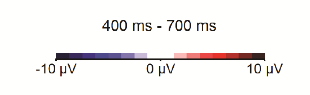
Figure 1









DP Group

Control Group

**P600f scalp topographies**

*Target Face*

*Own Face*

*Target Face*

*Own Face*

