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The activation of visual face memory and explicit face recognition are delayed in developmental prosopagnosia

Joanna Parketny, John Towler, Martin Eimer*

Department of Psychological Sciences, Birkbeck College, University of London, UK

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ABSTRACT

Individuals with developmental prosopagnosia (DP) are strongly impaired in recognizing faces, but the causes of this deficit are not well understood. We employed event-related brain potentials (ERPs) to study the time-course of neural processes involved in the recognition of previously unfamiliar faces in DPs and in age-matched control participants with normal face recognition abilities. Faces of different individuals were presented sequentially in one of three possible views, and participants had to detect a specific Target Face ("Joe"). EEG was recorded during task performance to Target Faces, Nontarget Faces, or the participants' Own Face (which had to be ignored).

The N250 component was measured as a marker of the match between a seen face and a stored representation in visual face memory. The subsequent P600f was measured as an index of attentional processes associated with the conscious awareness and recognition of a particular face. Target Faces elicited reliable N250 and P600f in the DP group, but both of these components emerged later in DPs than in control participants. This shows that the activation of visual face memory for previously unknown learned faces and the subsequent attentional processing and conscious recognition of these faces are delayed in DP. N250 and P600f components to Own Faces did not differ between the two groups, indicating that the processing of long-term familiar faces is less affected in DP. However, P600f components to Own Faces were absent in two participants with DP who failed to recognize their Own Face during the experiment. These results provide new evidence that face recognition deficits in DP may be linked to a delayed activation of visual face memory and explicit identity recognition mechanisms.

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 et al., 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 and Avidan, 2005; Duchaine and Nakayama, 2006a; see Towler and Eimer, 2012; Susilo and 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 et al., 2007; Duchaine, 2011).

The functional and neural causes of the face recognition impairments in DP are still largely unknown. Functional neuroimaging studies have often observed relatively normal brain activation patterns to faces versus non-face objects within the core posterior face processing network (Hasson et al., 2003; Avidan et al., 2005; Avidan and Behrmann, 2009; Furl et al., 2011; Avidan et al., 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 et al., 2007; Garrido et al., 2009).

Due to the limited temporal resolution of fMRI-based measures, these studies cannot reveal possible differences in the time-course of face perception and recognition processes between DPs and

* Corresponding author. Fax: +44 20 7631 6312.
E-mail address: m.eimer@bbk.ac.uk (M. Eimer).

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participants with unimpaired face recognition. Such differences can be revealed by ERP measures. Most ERP studies of DP have focused on the face-sensitive N170 component that emerges as 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 et al., 1996; Eimer et al., 2010; Eimer, 2011; Rossion and Jacques, 2011; see also Thierry et al., 2007, and Rossion and Jacques, 2008, for debates about the functional interpretation of the N170). A recent study from our lab (Towler et al., 2012) demonstrated that the generic face-sensitivity of the N170 does not differ between DPs and control participants (see also Towler et al., 2014), but found atypical effects of face inversion on N170 amplitudes for individuals with DP. The N170 component is usually not affected by the familiarity of a face (Bentin and Deouell, 2000; Eimer, 2000; but see Caharel et al., 2011), and is believed to reflect processes involved in the perceptual structural encoding of faces that occur prior to the recognition and identification of individual faces. For this reason, studies focused on the N170 component alone cannot provide direct electrophysiological markers of impaired face recognition that is at the core of the face processing deficits in DP.

ERP markers of identity-related face processing emerge at post-stimulus latencies beyond 200 ms. A repeated encounter with the face of a particular individual elicits an enhanced negativity at inferior occipito-temporal electrodes at around 250 ms after stimulus onset (e.g., Schweinberger et al., 1995; Begleiter et al., 1995; Schweinberger et al., 2002; Zimmermann and 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 and Burton, 2003). The N250r is larger for repetitions of famous faces as compared to unfamiliar faces (Herzmann et al., 2004), suggesting that pre-existing long-term representations of individual faces are activated particularly strongly when a matching face is perceived. A similar N250 component is also triggered by famous faces versus novel faces (e.g., Gosling and Eimer, 2011), and is assumed to reflect the match between a perceptual representation of a particular familiar face and a representation of the same face that is stored in long-term visual face memory. If the N250 component is generated during the activation of visual memory traces for a particular individual face, studying whether and when this component is elicited in participants with DP may yield new insights into possible impairments of early visual face recognition processes in DP.

In a recent ERP study (Eimer et al., 2012), we employed the N250 component to investigate the recognition of pre-experimentally known famous faces in DP. Participants with DP and control participants had to discriminate faces of famous versus unfamiliar individuals. As would be expected, DPs 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 and 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 (P600f; Gosling and 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 explicit categorization or identification of task-relevant stimuli (e.g., Folstein and Van Petten, 2011). In our earlier 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 the conscious recognition of an individual face.

Our previous ERP results (Eimer et al., 2012) suggest that when DPs successfully identify a pre-experimentally known famous face, the processes involved in the matching of perceptual and long-term visual 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 and Eimer, 2012, for more detailed discussion). The goal of the present study was to investigate the recognition of pre-experimentally unfamiliar target faces in participants with DP. When the face of a particular unfamiliar individual is designated as task-relevant, a visual representation of this face is stored in short-term face memory. The activation of this representation by a match with a currently seen face should therefore elicit an N250 component, and the subsequent attentional processing and explicit recognition of this face should give rise to a P600f component. Comparing these two components and their time-course between DPs and control participants could therefore reveal differences in the processes involved in the recognition of learned unfamiliar faces that may be linked to the face recognition impairments in DP.

A second issue addressed in the present study was whether participants' own faces would show a normal pattern of visual face memory activation and explicit recognition in DPs. Because one's own face is highly familiar and salient, and is strongly represented in long-term face memory, it should be rapidly recognized even when it is not explicitly task-relevant, and this should be reflected by N250 and P600f components to own versus unfamiliar faces. The question whether and to what degree the recognition of one's own face is impaired in prosopagnosia has not yet been studied systematically. Some patients with severe AP fail to recognize themselves in the mirror (Sergent and Poncet, 1990) and some individuals with DP also report difficulties in recognizing their own face (e.g., Duchaine et al., 2007). Our earlier study (Eimer et al., 2012) has shown that long-term visual memory representations of famous faces are activated when DPs successfully recognize one of these faces. In the present experiment, we investigated whether this is also the case for participants' own faces under conditions where these faces are formally task-irrelevant.

To address these questions, we adopted an experimental paradigm that was developed by Tanaka et al. (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 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 as well as the activation of a recently learned representation of a previously unfamiliar face in short-term memory. 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 et al., 2009). Target Faces and Own faces also elicited a sustained positivity that peaked around 500 ms post-stimulus in the study by Tanaka et al. (2006), analogous to the P600f component observed in our previous studies of famous face recognition (Gosling and Eimer, 2011; Eimer et al., 2012).

Ten participants with DP and a group of ten age-matched control participants had to memorize a particular Target Face

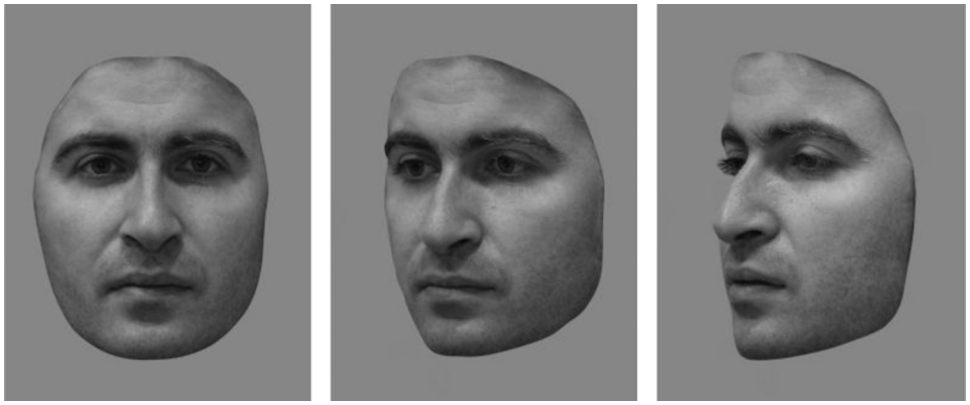

Fig. 1. The target face ("Joe"), shown from the three viewpoints (front view, 30° side view, 60° side view) in which it was presented in this study.

("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, which had to be ignored. In contrast to Tanaka et al. (2006), all faces appeared randomly in one of three possible views (see Fig. 1). To find out whether neural processes that contribute to the recognition of learned previously unfamiliar task-relevant faces are impaired in DP, we compared N250 and P600f components triggered by Target versus Nontarget Faces between the DP and control groups. If the normal ability to acquire and activate short-term representations of novel task-relevant faces and to explicitly recognize these faces was fully retained in DP, these two components should not differ between DPs and controls. Any delay and/or attenuation of N250 or P600f components to Target faces in the DP group would point towards a specific impairment in the time-course or efficiency of these processes in participants with DP. We also measured N250 and P600f components to Own Faces versus Nontarget Faces in both groups. An atypical pattern of Own Face N250 or P600f components for participants in the DP group would show that the recognition of one's own face can also be impaired in DP.

2. Methods

2.1. 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. The DP participants were recruited after contacting our research website (http://www.faceblind.org). 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 and Nakayama, 2006b). The Old-New Face Recognition test (ONT, Duchaine and 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.

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	45	M	−8.88	−2.77	−2.56	−0.63	−8.16
CC	30	F	−5.02	−2.52	−1.74	−0.49	−5.69
CM	31	M	−7.72	−4.29	−3.1	−2.89	−14.34
CT	40	M	−5.97	−2.64	−1.19	1.64	−2.78
MW	58	M	−3.67	−2.14	−1.6	−0.2	−6.49
KS	31	F	−8.49	−2.9	−0.92	−1.05	−9.03
DD	45	M	−5.21	−2.77	0.17	−0.77	−3.36
GW	21	M	−8.49	−2.52	−1.33	−1.05	−6.41
JA	48	F	−5.41	−2.64	−0.92	−0.49	−3.35

2.2. Materials and procedure

Stimuli were photographs of the faces of nine 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 Fig. 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/m^2) 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/m^2. 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 Fig. 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 first training block, participants were asked whether they felt comfortable with the task and were ready to start the main experiment. All ten control participants stated that they were ready to proceed. All participants with DP requested another training block of 40 trials, and five of the ten DPs asked for a third training block before they felt ready to proceed to 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 100 ms before to 700 ms after stimulus onset. Epochs with EEG activity exceeding 30 μV in the HEOG (horizontal eye movements) or 60 μV at electrode Fpz (eye blinks or vertical eye movements) were excluded from subsequent analysis, as were epochs with voltages exceeding 80 μV 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 electrodes Cz and Pz in the 400–700 ms time window (P600f component). The N250 was quantified at P7/8 because previous studies of face recognition in unimpaired participants (Gosling and Eimer, 2011) and DPs (Eimer et al., 2012) have shown that this component is maximal at these electrodes.

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, while faces of a single individual were used to compute Target and Own Face ERPs for each participant. For these reasons, we conducted additional analyses where Nontarget Face ERPs were computed for a single Nontarget Face (analogous to the procedure used by Tanaka et al., 2006). 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 once for a control participant. 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 assess differences in the onset latencies of N250 components between the two groups, difference waveforms were computed by subtracting ERPs to Nontarget Faces from ERPs to Target Faces and Own Faces, respectively. A jack-knife based procedure (Miller et al. (1998) was employed to determine and compare the onset of the N250 and P600f components to Target Faces between DPs and control participants. With this method, onset latencies were measured on the basis of grand-averaged difference waveforms (Target Faces – Nontarget Faces) computed for subsamples of participants, where one participant is subsequently excluded from the original sample. N250 onset was defined as the point in time where Target-Nontarget Face difference waveforms exceeded an absolute threshold value of -1 μV. The onset of the P600f component was determined with an absolute amplitude criterion of $+2.5$ μV. N250 and P600f onset latency differences between the DP and control groups were evaluated in analyses with the factor group, with F values (F_c) corrected according to the formula described by Miller et al. (1998). To confirm that the absolute onset criterion values used in these jack-knife based analyses were appropriate, N250 and P600f onset latency estimates were also obtained with an alternative procedure. ERP waveforms to Target versus Nontarget Faces were compared for each post-stimulus sampling point with paired t-tests, separately for DPs and control participants. The onset of N250 and P600f components in each group was defined as the point in time where these waveforms started to differ significantly, and this difference remained reliable for at least five successive sampling points. The onset latency estimates obtained with this procedure were then compared to the onset latencies obtained with the jack-knife based procedure.

3. Results

3.1. 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;

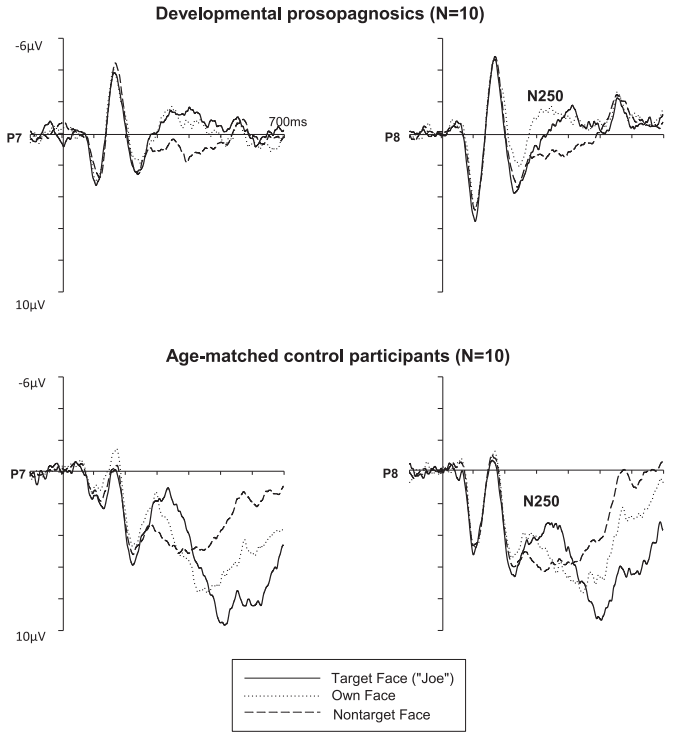

Fig. 2. Grand-averaged ERPs elicited at lateral temporo-occipital electrodes P7 (left hemisphere) and P8 (right hemisphere) in the 700 ms interval after stimulus onset in response to Target Faces, Own Faces, and Nontarget Faces. ERPs are shown separately for the DP group (top panel) and the age-matched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

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%; $t(18)=2.87$, $p<.02$). During the training phase that preceded the main experiment, target detection performance for control participants was already close to ceiling (97% correct), while DPs successfully detected 82.5% of all target faces. A comparison of target detection rates between the training phase and the main experiment for participants with DP showed there was no significant performance improvement (82.5% versus 87%; $t<1$).

3.2. ERP results

Fig. 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 700 ms epoch after stimulus onset, for participants with DP (top panel) and control participants (bottom panel). Visually evoked P1 and N1 components were followed by N250 components to both Target and Own Faces relative to Nontarget Faces. These N250 components were present not only in the control group, but also for participants with DP. Visual N1/N170 components were generally larger in the DP group than in the control group, similar to our previous study that focused on the N170 component in DP (Towler et al., 2012). This was reflected by a main effect of group (DPs versus controls) for N1 mean amplitudes (measured for the 150–200 ms post-stimulus interval), $F(1,18)=6.1$, $p=.02$, $\eta_p^2=.25$. The amplitudes of visual-evoked P1 and N1 components typically differ considerably across participants, due to individual variability in the spatial orientation of neural generator processes in the visual cortex, which determines the size of visual ERP components recorded from the scalp surface.

Target versus Nontarget Faces – N250 component. As can be seen in Fig. 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 (Target versus Nontarget Face), hemisphere, and the between-participant factor group (DPs versus controls). There was a highly significant main effect of face identity, $F(1,18)=32.7$, $p<.001$, $\eta_p^2=.65$, that did not interact with group, $F(1,18)<1$, demonstrating that N250 components to Target Faces of similar size were elicited in both groups. There was no identity x hemisphere interaction, $F=2.5$. 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, $F(1,18)=15.9$, $p=.001$, $\eta_p^2=.47$, 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 right-hemisphere N250 component to Target Faces was delayed in the DP group. This is illustrated in Fig. 3 (top panel), which shows ERP difference waveforms for right-hemisphere electrode P8 obtained by subtracting ERPs to Nontarget Faces from ERPs to Target Faces. Jack-knife-based N250 onset comparisons showed that the N250 component emerged 35 ms later in the DP group than in the control group (318 ms versus 283 ms; $F_c(1,18)=7.3$, $p=.02$). There was a corresponding 10 ms onset latency difference at left-hemisphere electrode P7 (295 ms versus 285 ms), which was however not reliable $F_c(1,18)<1$. The alternative method of obtaining N250 onset estimates based on successive *t*-tests for ERP waveforms to Target versus Nontarget Faces at P8 (see Methods section) yielded very similar results, with N250 onset latencies of 318 ms and 283 ms for the DP group and the control group, respectively.

Analyses with Nontarget ERPs that were computed for a single randomly selected Nontarget Face yielded essentially identical results. A main effect of face identity, $F(1,18)=30.2$, $p<.001$, $\eta_p^2=.63$, that did not interact with group, $F<1$, showed the presence of N250 components to Target Faces in both groups. The N250 emerged reliably later in the DP group relative to the control group at right-hemisphere electrode P8 (341 ms versus 287 ms; $F_c(1,18)=19.3$, $p<.001$).

Target versus Nontarget Faces – P600f component. At lateral posterior electrodes, the N250 components to Target Faces was followed at around 400 ms post-stimulus by a sustained positivity in the control group, but not in the DP group (Fig. 2). As shown in Fig. 4, this late positive component (P600f) to Target faces was maximal at posterior midline electrode Pz, where it was present not only for control participants but also for DPs, although it was reduced in amplitude and delayed in the DP group. Scalp topographies of the P600f to Target Faces that were obtained by subtracting ERP mean amplitudes measured in the 400–700 ms post-stimulus time window to Nontarget Faces from ERPs to Target Faces are shown in Fig. 4 (bottom panel) for both groups. The P600f component showed a clear focus over Pz in the control group, and was attenuated and more broadly distributed in the DP group. The attenuation and delay of the P600f to Target Faces for participants with DP is illustrated in Fig. 3 (bottom panel), which shows Target Face – Nontarget Face difference waveforms measured at Pz for both groups.

To assess these Target P600f differences between DPs and controls statistically, ERP mean amplitudes to Target and Nontarget Faces measured at Pz in the 400–700 ms post-stimulus time window were analysed with the factors face identity and group. A

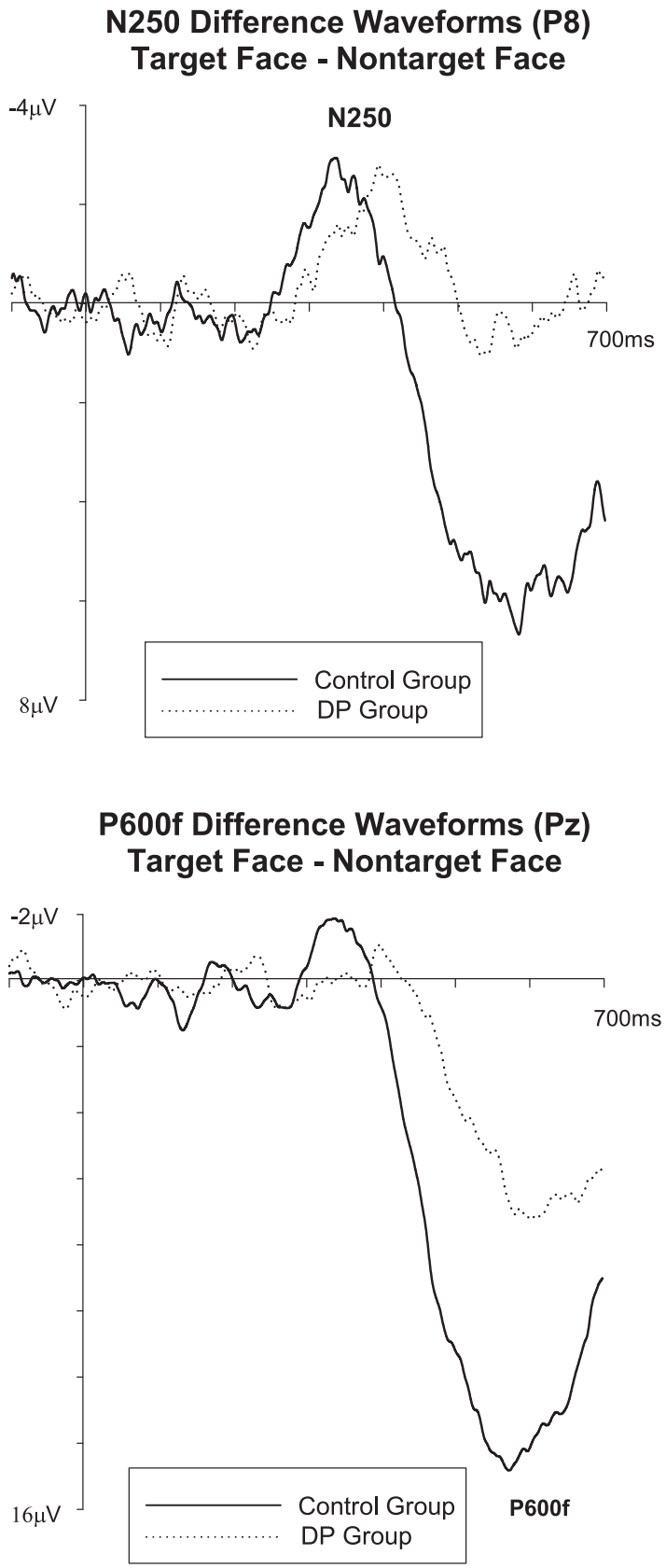

Fig. 3. Top panel: N250 difference waveforms obtained for right posterior electrode P8 by subtracting ERPs to Nontarget Faces from ERPs to Target Faces, separately for the control group and the DP group. Bottom panel: P600f difference waveforms obtained at electrode Pz by subtracting ERPs to Nontarget Faces from ERPs to Target Faces, separately for the control group and the DP group. The onset of both components was delayed in the DP group, and P600f amplitude was also attenuated in this group.

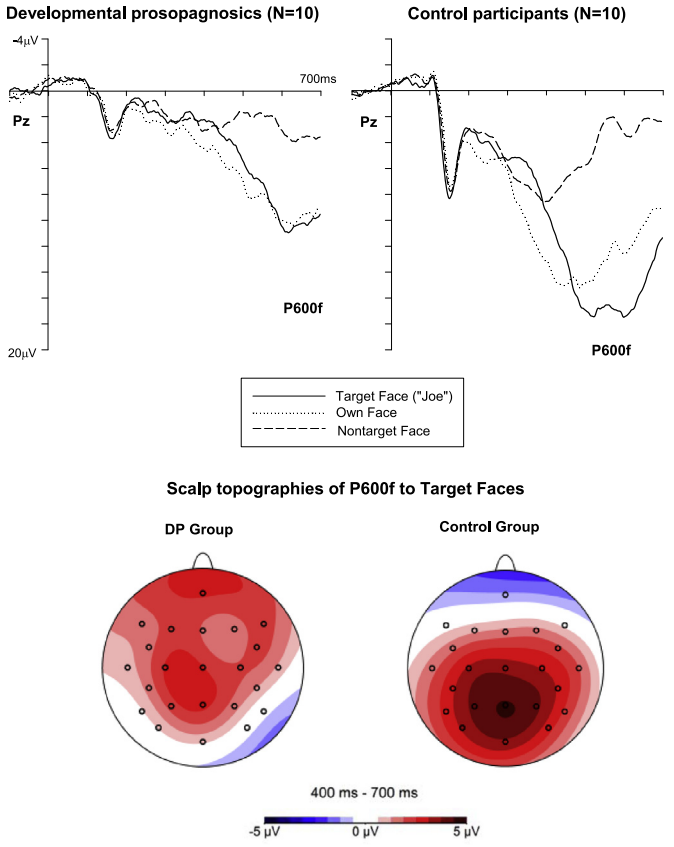

Fig. 4. Top panel: Grand-averaged ERPs elicited at posterior midline electrode Pz in the 700 ms interval after stimulus onset in response to Target Faces, Own Faces, and Nontarget Faces, shown separately for the DP group (left panel) and the control group (right panel). P600f components to Target Faces and Own Faces were attenuated in the DP group. Bottom panel: Topographical maps showing the scalp distribution of P600f components to Target Faces for the DP group and the control group. 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.

main effect of face identity, $F(1,18)=53.9$, $p<.001$, $\eta_p^2=.75$, was accompanied by an interaction between face identity and group, $F(1,18)=9.3$, $p=.007$, $\eta_p^2=.34$, confirming that the amplitude of the P600f to Target Faces was reduced in the DP group. Follow-up analyses showed that the P600f elicited by Target Faces was reliably present not only in the control group, $F(1,18)=39.4$, $p<.001$, $\eta_p^2=.81$, but also for DPs, $F(1,18)=14.6$, $p=.004$, $\eta_p^2=.62$. Very similar results were obtained for an analysis of P600f amplitudes at vertex electrode Cz. Again, a main effect of face identity, $F(1,18)=9.8$, $p=.006$, $\eta_p^2=.40$, was accompanied by an interaction between face identity and group, $F(1,18)=7.3$, $p=.02$, $\eta_p^2=.30$, indicating that the Target Face P600f was attenuated in the DP group. To evaluate Target P600f onset differences between both groups, a jack-knife-based P600f onset latency analysis was conducted for Target-Nontarget Face difference waveforms measured at Pz. This analysis confirmed that the P600f to Target Faces was significantly delayed by 68 ms in the DP group relative to the control group (486 ms versus 418 ms; $F_c(1,18)=11.9$, $p=.003$). The P600f onset estimates obtained with the alternative procedure based on paired *t*-tests yielded very similar results (498 ms versus 428 ms, for DPs and controls, respectively).

The parallel set of analyses that was based on Nontarget ERPs for a single randomly selected Nontarget Face yielded identical results. For P600f mean amplitudes, there was main effect of face identity, $F(1,18)=36.6$, $p<.001$, $\eta_p^2=.67$, and an interaction between face identity and group, $F(1,18)=8.1$, $p=.01$, $\eta_p^2=.31$, confirming the attenuation of the P600f component to Target Faces in the DP group. In addition, there was a significant P600f onset delay in the DP group (492 ms versus 423 ms; $F_c(1,18)=6.5$, $p=.02$).

ERPs to Own versus Nontarget Faces. As can be seen in Fig. 2, Own Faces triggered N250 components in both groups. Subsequent analyses of ERP mean amplitudes to Own and Nontarget Faces measured in the N250 time window (250–400 ms post-stimulus) at electrodes P7/P8 conducted with the factors face identity (Own versus Nontarget Face), hemisphere, and group revealed no differences of N250 components to Own Faces between DPs and controls, A main effect of face identity, $F(1,18)=12.3$, $p=.003$, $\eta_p^2=.41$, that did not interact with hemisphere, $F(1,18)<1$, or group, $F(1,18)=2.1$, confirmed the presence of reliable Own Face N250 components in both groups. The N250 component to Own Faces was already reliably present in the first half of the experiment, $F(1,18)=5.0$, $p=.04$, $\eta_p^2=.22$, and there was no interaction between face identity and group, $F<1$, confirming that N250 components to Own Faces emerged early for both DPs and control participants. There was also no difference in the onset latency of this component between DPs and controls over either the left or right hemisphere, both $F_c(1,18)<2$. Analyses based on Nontarget ERPs computed for a single randomly selected face yielded identical results. There was a main effect of face identity, $F(1,18)=8.2$, $p=.01$, $\eta_p^2=.31$, that did not interact with group, $F<2$, and N250 onset latency differences between the two groups, both $F_c(1,18)<2$.

Fig. 4 shows that relative to Nontarget Faces, Own Faces elicited P600f components in both groups, and that the amplitude of this Own Face P600f was attenuated in the DP group. An analysis of P600f mean amplitudes measured at Pz in the 400–700 ms post-stimulus time window with the factors face identity and group, revealed a main effect of face identity, $F(1,18)=67.1$, $p<.001$, $\eta_p^2=.79$, that was accompanied by a marginally significant interaction between face identity and group, $F(1,18)=3.8$, $p=.07$, $\eta_p^2=.18$. Follow-up analyses showed that Own Face P600f components were reliably present in the control group, $F(1,18)=60.5$, $p<.001$, $\eta_p^2=.87$, and also for participants with DP, $F(1,18)=16.9$, $p=.003$, $\eta_p^2=.65$. Analogous results were found at vertex electrode Cz, where a main effect of face identity, $F(1,18)=74.2$, $p<.001$, $\eta_p^2=.81$, and an interaction between face identity and group, $F(1,18)=7.8$, $p=.01$, $\eta_p^2=.31$, were present, again reflecting the attenuation of the P600f to Own Faces in the DP group. A jack-knife-based P600f onset latency analysis conducted for Own Face-Nontarget Face difference waveforms measured at Pz demonstrated that the P600f component was significantly delayed by 95 ms in the DP group relative to the control group (438 ms versus 343 ms; $F_c(1,18)=8.0$, $p=.01$). Similar P600f onset latency values were obtained with the alternative onset estimation procedure based on successive paired *t*-tests (426 ms versus 354 ms, for DPs and controls, respectively). The same results were obtained in the additional analysis based on a single randomly selected Nontarget Face. A main effect of face identity, $F(1,18)=56.9$, $p<.001$, $\eta_p^2=.76$, was accompanied by a borderline significant face identity x group interaction, $F(1,18)=4.4$, $p=.05$, $\eta_p^2=.2$. The Own Face P600f component was significantly delayed by 93 ms in the DP group relative to the control group (443 ms versus 350 ms, $F_c(1,18)=7.7$, $p=.01$).

The fact that the Target Face ("Joe") was always male could have affected the P600f component to Own Faces, with larger P600f amplitudes for the Own Faces of male participants, because these may have been more similar to the male Target Face than the Own Face of female participants. Furthermore, the similarity between Own Faces and Nontarget Faces might also have an impact on the P600f to Own Faces, with smaller P600f amplitude differences between Own and Nontarget Faces of the same gender than

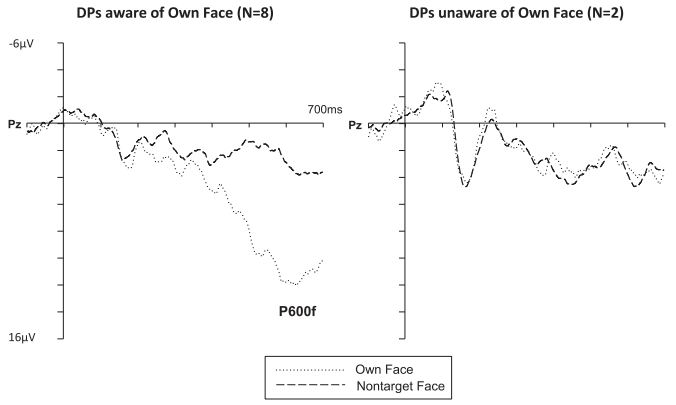

Fig. 5. ERPs elicited at posterior midline electrode Pz in the 700 ms interval after stimulus onset in response to Own Faces and Nontarget Faces, shown separately for those eight participants with DP who reported to have been aware of the presence of their Own Face during the experiment (left panel) 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.

between Own and Nontarget Faces that differed in their gender. To assess these possibilities, two analyses compared ERPs obtained at Pz in the P600f time window to Own versus Nontarget Faces, separately for male and female Nontarget Faces. These analyses included participant's gender as an additional between-subject factor. There were no interactions between participants' gender and face identity, both $F<1$, and no three-way interactions between participant's gender, face identity, and group, both $F<2$, indicating that the Own Face P600f component was not systematically modulated by any gender-related differences in the similarity of Own Faces to the Target Face or to Nontarget Faces.

Because two of the ten DPs tested reported that they did not recognize that their Own Face was present among the Nontarget Faces during the experiment, we computed separate Own Face and Nontarget Face ERP waveforms for these two DPs, as well as for the remaining eight DPs who claimed to have been aware of the presence of their Own Face (as shown in Fig. 5 for electrode Pz). P600f components to Own Faces were entirely absent for those two DPs who failed to recognize their Own Face. In contrast, Own Faces elicited a distinct P600f component in the other eight participants with DP. For all eight of them, Own Faces triggered an enhanced positivity relative to Nontarget Faces at Pz in the P600f time window. In fact, statistical comparisons of Own Face P600f amplitudes and onset latencies between these eight participants with DP and control participants obtained no significant between-group differences, both $F<2$, suggesting that for DP participants who recognized their Own Faces, P600f components to these faces were similar to the Own Face P600f measured for control participants.

ERPs to Target versus Nontarget Faces – Additional Analyses. If removing the two DPs who did not recognize their Own Face from the sample eliminates the differences of P600f components to Own Faces between the two groups, the question arises whether this might also be the case for N250 and P600f components to Target Faces. To test this, we repeated the analyses of N250 and P600f mean amplitudes to Target versus Nontarget Faces reported earlier, leaving out the two participants with DP who failed to recognize their Own Face. Results were virtually identical to the results obtained for the full set of DP participants. For N250 amplitudes to Target Faces, there was a main effect of face identity, $F(1,16)=26.1$, $p<.001$, $\eta_p^2=.62$, but no interaction between face identity and group, $F<2$. For P600f amplitudes to Target Faces, a main effect of face identity, $F(1,16)=48.4$, $p<.001$, $\eta_p^2=.65$, interacted with group, $F(1,18)=9.5$, $p=.007$, $\eta_p^2=.37$. This demonstrates that removing these two DPs from the sample does not change the pattern of N250 and P600f components to Target Faces.

4. 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 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 face ("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 a visual representation of one particular task-relevant face, and to successfully use this representation 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.

The pattern of N250 and P600f components measured in response to Target Faces showed that there were systematic differences in the identity-related processing of these faces between the DP and control groups. 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 a short-term memory trace of a previously unfamiliar learned task-relevant face was activated by a match with the currently seen face in both groups. Importantly, the onset of the N250 to Target Faces was reliably delayed by approximately 40 ms in the DP group over the right hemisphere. 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 generated during an early visual stage of face recognition (Schweinberger and Burton, 2003; Gosling and Eimer, 2011), the later 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 may be an important factor contributing 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 later onset of face identity matching processes, other factors played a part in the impaired task performance observed for individuals with DP.

The additional observation that the P600f component to Target Faces emerged later and was reduced in amplitude 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 and 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 P600f to Target Faces emerged almost 70ms later in the DP group relative to the control group, indicating that DPs identified these faces considerably slower than control participants. Some of this P600f delay is likely to directly result from the delay of the preceding face identity matching process that was reflected by the Target Face N250 onset latency difference between the two groups. The fact that P600f onset differences between DPs and control participants were considerably larger than the corresponding differences observed for N250 components suggests that there may also have been an additional independent delay in the emergence of explicit face recognition in the DP group.

The attenuation of P600f amplitudes to Target Faces observed for participants with DP suggests that attentional and recognition-related 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 delay the explicit individuation of a particular face as target, and will thus result in slower RTs 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, Own Faces were highly familiar stimuli that are represented in long-term memory. In our previous ERP study of famous face recognition in DP (Eimer et al., 2012), famous faces that were successfully recognized by DPs triggered an N250 component, suggesting that the activation of visual face memory during the recognition of familiar faces is not selectively impaired in individuals with DP. In this earlier study, famous faces were task-relevant, whereas Own Faces served as irrelevant distractor stimuli in the present experiment. In spite of this fact, we found no evidence for systematic differences in the processing of Own Faces between the DP and control groups. Own Faces elicited very similar N250 components in both groups, suggesting that the activation of long-term representations of highly familiar faces in visual memory is not selectively impaired in DP. However, the P600f to Own Faces was delayed and reduced in amplitude in the DP group relative to the control group. These P600f differences between the two groups were primarily driven by those two participants with DP who reported to have been unaware of the presence of their Own Face during the ERP experiment. For these two DPs, P600f components to Own Faces were entirely absent. For the remaining eight DPs who recognized their Own Face, P600f component to Own Faces did not differ from the Own Face P600f observed in the control group (see Fig. 5).

The absence of an Own Face P600f for DPs who failed to recognize their Own Face is consistent with previous observations that this component is absent in response to non-recognized famous faces (Eimer et al., 2012), and provides further evidence that the P600f component is closely linked to explicit face recognition. One of the two DPs who did not recognize their Own Face also had no Own Face N250 component, while all other DPs (including the other one who reported to have been unaware of their Own Face) showed N250 components to Own Faces. The dissociation between the activation of visual face memory by their Own Face (reflected by the N250) and the absence of explicit Own Face recognition 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.

The delayed onset of N250 and P600f 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 et al., 2008). If view-independent representations of individual faces are stored in anterior temporal cortex (Anzellotti et al., 2013), a reduction in the density of white matter tracts connecting this region to posterior occipito-temporal face-selective regions could delay the onset of visual face identity matching processes, as was found in this study for DPs. The 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 the additional larger delay of the subsequent attentional processing and conscious recognition of Target Faces that was reflected by the later emergence of the P600f to Target Faces in the DP group. The fact that systematic N250 and P600f onset latency differences between DPs and controls were only observed for previously unfamiliar Target Faces but not for participant's Own Face suggests that these impairments are less pronounced when face recognition can be based on long-term representations of facial identity, and may primarily affect the recognition of newly learned facial identities.

In contrast to the earlier study by Tanaka et al. (2006), who found that the N250 component to newly learned Target Faces emerged reliably only in the second half of the experiment, we found that N250 components to Target Faces were already significant during the first five blocks of the current study, both for DPs and control participants. This difference may be due to the fact that the present study included a training block where the Target Face had to be identified among distractor faces, while participants in the Tanaka et al. (2006) experiment were merely asked to study the Target face prior to the first experimental block. It should also be noted 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). Memorizing different views of a previously unfamiliar task-relevant face is likely to involve view-independent working memory representations, whereas memorizing a single constant view, as in the Tanaka et al. (2006) study, can be based primarily on image-based view-dependent representations. It is possible that representations of facial identity are formed more rapidly in face learning tasks where task-relevant faces appear in multiple views and image-based cues are therefore less useful for face recognition.

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 an additional delay 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. However, even though Own Faces are highly familiar and personally relevant, the current study has shown that explicit self-recognition processes can be strongly impaired in some individuals with DP, up to the point where the presence of their Own Face goes entirely undetected.

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