universität wien

MASTERARBEIT / MASTER'S THESIS

Titel der Masterarbeit / Title of the Master's Thesis

„Electrophysiological correlates of human long-term face adaptation“

verfasst von / submitted by

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angestrebter akademischer Grad / in partial fulfilment of the requirements for the degree of

Master of Science (MSc)

Wien, 2016

Studienkennzahl lt. Studienblatt / degree programme code as it appears on the student record sheet:	A 066 840
Studienrichtung lt. Studienblatt / degree programme as it appears on the student record sheet:	Masterstudium Psychologie
Betreut von / Supervisor:	Mag. Thomas Ditye, MRes PhD

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Abstract

When looking at an extended face for a few seconds a following normal face will appear compressed. This is called face adaptation and is one example of the flexible adjustment of the visual system to changes in the environment. The present study investigated the underlying mechanisms of adaptation in terms of the questions (a) whether there is more than one mechanism that subserving adaptation, (b) if adaptation can be regarded a form of learning and (c) whether there is a sensitive electrophysiological marker of adaptation (i.e., the N170 and P250) in the electroencephalography (EEG). To answer these questions extended and compressed faces were used as adapters in a computer experiment. One group of participants continuously adapted for 12 minutes, whereas the second group of participants adapted in 12 one-minute blocks. After adaptation both groups adapted to a second adapter for a shorter time to overwrite the original adaptation effect. Perceived face distortion and EEG were measured and revealed that the original adaptation effect returned once adaptation had stopped but there were no differences between the two groups. Furthermore, neither N170 nor P250 could be identified as an electrophysiological marker of adaptation. The results indicate that adaptation relies on more than one mechanisms. Adaptation does not benefit from a distributed condition as other forms of learning do and thus seems to be different from learning – at least concerning a spacing effect.

Key words: Adaptation, Visual aftereffects, Learning, Event Related Potential, N170, P250

ELECTROPHYSIOLOGICAL CORRELATES OF HUMAN LONG-TERM FACE ADAPTATION

Zusammenfassung

Wenn man für einige Sekunden ein auseinandergezogenes Gesicht sieht, wird ein normales Gesicht danach als zusammengedrückt wahrgenommen. Dies wird auch als Gesichtsadaptation bezeichnet und stellt ein Beispiel für die flexible Anpassung des visuellen Systems an Änderungen in der Umwelt dar. Die vorliegende Studie untersucht die der Adaptation zugrundeliegenden Mechanismen und wirft die Fragen auf (a) ob es mehr als einen Mechanismus gibt, der die Adaptation bestimmt, (b) ob Adaptation als eine Form des Lernens betrachtet werden kann und (c) ob es ein sensibles elektrophysiologisches Korrelat (N170 und P250) im Elektroenzephalogramm (EEG) gibt. Um diese Fragen beantworten zu können wurden auseinandergezogene und zusammengedrückte Gesichter als Adaptoren genutzt. Eine Versuchspersonengruppe hat 12 Minuten am Stück adaptiert, während die zweite Versuchspersonengruppe in 12 einminütigen Blöcken adaptierte. Nach der Adaptation adaptierten beide Gruppen auf einen weiteren Adaptor für eine kürzere Zeit, um den ursprünglichen Adaptationseffekt zu überschreiben. Die wahrgenommene Gesichtsverzerrung und das EEG wurden erhoben. Es konnte gezeigt werden, dass der ursprüngliche Adaptationseffekt nach der zweiten Adaptation zurückkam. Allerdings gab es keinen Unterschied zwischen beiden Gruppen. Außerdem konnte weder die N170 noch die P250 als elektrophysiologisches Korrelat von Adaptation bestimmt werden. Die Ergebnisse zeigen, dass Adaptation auf mehr als einen Mechanismus basiert. Adaptation profitiert, zumindest in Bezug auf den ‚spacing effect', nicht von einer verteilten Bedingung wie andere Formen des Lernens.

Schlüsselwörter: Adaptation, Visuelle Nacheffekte, Lernen, Ereigniskorrelierte Potentiale, N170, P250

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1. Introduction

„The reality, so characterized, is not true reality, but is appearance. “
(Bradley, 1897, Chapter III, para. 1)

This quote deals with one main assumption, namely that a true reality is not perceivable. True in this context means that everyone perceives the reality as it really is which in fact would imply that everyone perceives the reality or the environment in exactly the same way. There are a lot of examples ruling out the acceptance of one objective perception of reality. Thus, Bradley (1897) states a clear distinction between the appearance that is affected by an individual perception and a true reality.

If you look at a waterfall for a few minutes and then shift your gaze to the adjacent rocks, they will appear to be moving upward in the opposite direction of the water flow even though they are, of course, stationary. The question arises, whether the movement of the rocks is reality. Obviously, based on your physical knowledge, the movement of the rocks does not reflect reality but what if it is not that obvious?

The characterized phenomenon is one example that contradicts the assumption of one objective perception of reality. It points toward a perception of reality which is individually shaped by our visual systems and previous experiences. The described phenomenon is the so called waterfall illusion which was first mentioned by Robert Addams (1834) and which is now referred to as motion aftereffects (Thompson & Burr, 2009). Aftereffects in general are one convincing argument that not everyone perceives the environment in exactly the same way and further, that a true reality if it really exists is not directly visible. There is an insurmountable gap between reality and appearance which is determined by the brain and how it processes visual information. How the brain codes visual information defines and distorts the perception of reality. That is why visual aftereffects are of high interest.

Aftereffects are the result of an adaptation period. *Adaptation* is a prolonged exposure to a stimulus and reflects a mechanism of the visual system that refers to the flexible adjustment to changes in the environment. This mechanism occurs across the whole range of different perceptions like the perception of motion, brightness, colour,

orientation but also on higher visual levels like the identity or gender of faces. It is mostly unconscious and therefore, not controllable (Thompson & Burr, 2009).

Returning to the waterfall illusion, the exposure to the waterfall is an adaptation to motion that results in the distorted perception of stationary objects, the adjacent rocks, moving in the opposite direction of the adapted stimulus. Adaptation is a powerful tool to concretise the processes of the visual system. Thus, in a narrow sense, adaptation and aftereffects are one possibility to investigate the processing of visual information and the encoding of visual dimensions like colour, orientation, and movement. This advantage resulted in a steadily growing number of studies during the last 50 years. However, a lot of these studies neglect adaptation as a process (Webster, 2011). This master thesis focuses on the understanding of adaptation itself. Adaptation itself is worth looking at because, in the broadest sense, it gives an insight into the perception of our surrounding and is indispensable for living in a permanent-changing environment. (Strobach & Carbon, 2013).

Adaptation and visual aftereffects exist on different processing levels ranging from low levels like retinal cells adapt to contrast to lower cortical levels like cells in the primary visual area adapt to tilt lines up to high levels like the adaptation to faces and objects. In this thesis distorted faces were used during adaptation but the aim is to make statements about adaptation and higher level aftereffects rather than about the sole perception of faces. Faces were used because facial adaptation offers a broad range of literature to build up on, is a simple implementation and a suitable opportunity to link adaptation with learning.

All in all, this thesis first, aims to replicate previous findings that adaptation relies on temporally different mechanisms rather than on one single short-term mechanism (Mesik, Bao, & Engel, 2013), second, wants to reduce the gap between adaptation and learning and third, tries to identify an electrophysiological correlate of perceived visual aftereffects during adaptation.

1.1 Adaptation on single cell level and its contradiction to long-term adaptation

The waterfall illusion was firstly described by Robert Addams in 1834. One and a half centuries later, psychophysics offered the possibility to investigate the neuronal basis of adaptation and visual aftereffects in animals (Thompson & Burr, 2009).

One of the first models addressing the explanation of adaptation on a neuronal level was the fatigue model (Blakemore & Campbell, 1969). Barlow and Hill (1963) recorded the discharge of single units in retinal ganglion cells of a rabbit. The discharge of these single units were selectively activated in response to motion in different directions. They used a rotating disk with an irregular pattern to stimulate the ganglion cells. Two important results could be shown and were used to clarify visual aftereffects. First, a prolonged stimulation of these ganglion cells with the disk rotating in the preferred direction led to a reduced activity, whereas prolonged stimulation with the disk rotating in the non-preferred direction did not trigger a reduction in activity. Second, the ganglion cells which were stimulated with the preferred motion direction showed a decrease in discharge below the baseline activity directly after stimulation. Ganglion cells which were stimulated with the non-preferred motion direction and which have a preference for the opposite motion direction showed the same strength of signal after stimulation in comparison to the baseline activity. This imbalance after stimulation between these two types of ganglion cells could explain the distorted perception of motion in the reverse direction.

Thus, the fatigue model states adaptation to some extent by diminished responses of stimulus specific neurons. A prolonged presentation of a stimulus ‘fatigues’ relevant neurons. After adaptation, perception is distorted in the opposite direction because these stimulus relevant neurons respond less strongly than in normal conditions (Blakemore & Campbell, 1969).

However, this model is oversimplified. Adaptation is not limited to retinal cells as cortical cells in lower (Dragoi, Sharma, & Sur, 2000) as well as higher visual areas (Kohn & Movshon, 2003) also adapt in response to prolonged stimulation. An additional limitation of the fatigue model is the fact that the imbalance of discharge between for example motion sensitive cells is temporally restricted to a few seconds. This implies that visual aftereffects disappear after a few seconds (Barlow & Hill, 1963). In contrast to this implication, a growing body of evidence suggests that aftereffects could remain longer

than just a few seconds, especially higher visual aftereffects (Carbon & Ditye, 2011; Carbon et al., 2007; Ditye, Javadi, Carbon, & Walsh, 2013; Mesik et al., 2013). The fatigue model was based on low visual aftereffects like motion or contrast aftereffects. This contradiction between the fatigue model and long lasting visual aftereffects does not mean that adaptation is totally independent of a reduced discharge of stimulus sensitive neurons. It is rather unlikely that the fatigue model by itself is sufficient to explain the whole range of visual aftereffects for example the perception of facial visual aftereffects one week after adaptation.

Carbon and Ditye (2011) found these durable visual aftereffects using distorted familiar faces as adapters for 25 minutes. In a test phase participants had to choose the veridical face in a two alternative forced choice task. The veridical face was the original face and the alternative face was a slightly distorted face in the same direction as the adapter. For example, if the adapter face is compressed, a normal face would be perceived as extended and a slightly compressed face would be perceived as normal (face figural aftereffects). Therefore, the participants would choose the slightly compressed face as the veridical face if the face figural aftereffect is present. This is exactly what they found. Closely after a 25 minutes adaptation to a distorted familiar face a slightly distorted face in the same direction as the adapter was perceived as normal. But more interestingly, they extended the time interval between the adaptation phase and the test phase to one week and the face figural aftereffects still existed even though they were weaker in comparison to a 5 minutes interval. The authors interpreted this result in favour of memory representations that are matched with the environment due to adaptation. Thus, adaptation reflects an integration of new information into already existing memory traces. The relationship between adaptation and learning is discussed in more detail in the next sub chapter (1.2).

Moreover, the persistence of visual aftereffects for one week indicates that adaptation operates on more than just one timescale. The question arose whether different mechanisms underlie adaptation that represent different time scales or whether just one mechanism regulates adaptation on all timescales. To answer this question, Mesik et al. (2013) conducted a series of experiments with different kinds of adaptation (adaptation to motion, to coherence and to faces) but with the same experimental design. First, an adapter was presented for 10 minutes (=adaptation) and was then followed by a shorter presentation of an adapter to delete the original adaptation effect (=deadaptation). For

instance, to induce facial visual aftereffects Barack Obama with eyes too far apart was used. After 10 minutes they presented Barack Obama with an eye distance that was too short (=deadaptation). A continued measurement of the original facial visual aftereffect (=adaptation) demonstrated that it returned even deadaptation had already cancelled out the effect. These results were observed for adaptation to motion, to coherence as well as to faces and thus, seem to be generalizable across lower and higher visual aftereffects.

The authors argued in favour of a multiple mechanism model, meaning that there are at least two basic mechanisms underlying adaptation. Longer adaptation involves both fast and slow mechanisms, whereas deadaptation, because of its short duration, might only affect the fast mechanism.

1.2 Adaptation and (perceptual) learning

Carbon and Ditye (2011) assumed that adaptation leads to changes in memory trances which could imply that adaptation reflects learning. Additionally, the effects found by Mesik et al. (2013) are comparable with interference. Deadapation can be seen as a task or process that interferes retroactively with the adaptation. As visual aftereffects of adaptation resisted against deadaptation on the one hand and consolidation can be defined as resilience to retroactive interference on the other hand (Krakauer, Ghez, & Ghilardi, 2005) the hypothesis that adaptation reflects consolidation or learning seems to be plausible.

This hypothesis is supported by other long term adaptation studies. For example, a study that investigated the changes of visual aftereffects depending on sleep. Here, higher visual aftereffects to faces were discovered after 90 minutes of sleep compared to wakefulness. These beneficial effect of sleep on adaptation could not be explained due to a pure reduction of visual interference. The wake group was blindfolded and thus, visual interference is not an alternative explanation for the higher visual aftereffects after a period of sleep compared to wakefulness. Additionally, there was a positive correlation between visual aftereffects and rapid eye movement (REM) sleep. Higher visual aftereffects were associated with a longer REM sleep period. The positive correlation indicates a more active role of sleep for adaptation than just a passive prevention of visual interference. In general, REM sleep have been related to recognition memory for faces

(Wagner, Kashyap, Diekelmann, & Born, 2007) and, interestingly, to visual perceptual learning (Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994; Yotsumoto et al., 2009).

So, the beneficial effects of REM sleep on adaptation might be explained by an overlap between adaptation and perceptual learning. In fact, the relationship between adaptation and perceptual learning is still unclear. In an experimental approach, perceptual learning involves the improvement in discrimination of a perceptual judgement due to training (Lu, Yu, Sagi, Watanabe, & Levi, 2011). In contrast, adaptation is not an improvement in discrimination but rather a loss of sensitivity because of exposure to a stimulus. Thus, adaptation leads to changes in appearance, whereas perceptual learning leads to changes in performance of perceptual judgements. Furthermore, perceptual learning is the result of training and reflects alterations in the interpretation of available neuronal signals of the visual system. Adaptation is normally not equated with training and seems to just change the strength of neuronal activity. On a neuronal level adaptation is distinguishable from perceptual learning because it is more a suppression rather than facilitation (Webster, 2011). One further difference that could be indicated is the course of time. Teich and Qian (2003) concluded relying upon a physiologically based model that adaptation can be seen as a short-term type of learning. However, adaptation does not have to be restricted to a short time period. As it was discussed above, visual aftereffects can exist for even a week or longer (Carbon & Ditye, 2011). This is one example showing that the delimitation between these two processes is not that unambiguous.

In a broad sense, perceptual learning describes long-lasting changes of the perceptual system based on changes in the environment, which leads to an enhanced ability to interact with the surrounding (Goldstone, 1998). Adaptation can be seen as a continuous recalibration of sensory processes initiated by the visual input (Kovács et al., 2006). These two definitions show some similarities, namely that adaptation as well as perceptual learning rest upon changes of visual information in the environment and make the visual system, as a consequence, more efficient.

Taken together, even if there are differences between adaptation and learning, the idea that adaptation could be a form of learning is not unreasonable. To examine the hypothesis that adaptation reflects learning we used a similar experimental design to Mesik et al. (2013). Additionally, we manipulated the adaptation phase in correspondence to the spacing effect. The spacing effect is well documented in context of learning. It describes that items learned once and studied again after a delay are recalled better than

items learned and restudied in fast succession (Cepeda, Pashler, Vul, Wixted, & Rohrer, 2006). Thus, we compared a blocked and a distributed adaptation phase to examine whether the spacing effect is also detectable in adaptation.

1.3 Adaptation on cell population level investigated with electroencephalography

The EEG is mostly applied in humans to investigate and identify the neurophysiological mechanisms, among other things, of adaptation. Several studies have identified an event related potential (ERP) that is quite specific for face processing, the N170 (Bentin, Allison, Puce, Perez, & McCarthy, 2010; Desjardins & Segalowitz, 2013; Eimer, 2000). This negative component is peaking at around 140-200ms after stimulus onset and is located over occipito-temporo-parietal regions (Burkhardt et al., 2010). The component shows a significant higher amplitude in response to faces compared to other objects like cars or houses (Eimer, 2000).

The N170 component is also affected by face adaptation. This indicates that face adaptation takes place on the same level as face processing- that is on a higher rather than on a lower neuronal level. Additionally, there is evidence that facial visual aftereffects are constant even when changing lower visual properties like size (Anderson & Wilson, 2005), colour (Yamashita, Hardy, De Valois, & Webster, 2005) or orientation (Rhodes, Jeffery, Watson, Clifford, & Nakayama, 2003) Exemplary, Rhodes et al. (2003) used distorted faces (extended or compressed) as adapters that were canted 45 degrees either clockwise or counter clockwise. The test faces were also canted 45 degrees but in the opposite direction of the adapter faces. When the adapter face was canted clockwise the test face was canted counter clockwise and vice versa. They found the typical face figural aftereffect. Totally independent of the opposite orientations of the faces, the target face was perceived as distorted in the same direction as the adapter face. This means that the face figural aftereffect is not affected by lower visual aftereffects (in this case orientation) and seems to occur on a higher neuronal level.

However, the N170 component is affected by face adaptation regarding to a decrease in amplitude and a rise in latency. After an adaptation period for instance to a female face, the afterwards presented test face elicited a N170 component with a reduced amplitude and an increased latency compared to a control condition (Kovács et al., 2006).

This changing pattern of the N170 component is not restricted to face adaptation in the domain of gender. Zimmer and Kovács (2011) presented extended and compressed faces as adapters and also found a reduction in amplitude and a longer latency of the N170.

Despite these results, the direct link between face adaptation and the modulation of the N170 component is not that clear because of two reasons. The first one is that the influence of the sole repetition of faces on the latency and amplitude of the N170 is ambiguous. The repetition of faces can lead to a reduced amplitude of N170 (Campanella, Quinet, Bruyer, Crommelinck, & Guerit, 1996), whereby some studies could not found such a reduction in amplitude (Schweinberger, Pickering, Burton, & Kaufmann, 2002). The second reason is that the N170 is not absolutely limited to face processing and face adaptation. Kovács et al. (2006) reported that also hand stimuli of females elicited a reduction in amplitude and the rise in latency of the N170 component.

Burkhardt et al. (2010) might identified a more sensitive electrophysiological correlate. The P250 is a positive component that peaks at 185-300ms over occipito-parietal regions. They also used compressed and extended faces to induce facial figural aftereffects. The P250 changed its pattern depending on the perception. The perception of a face as undistorted elicited a higher amplitude whereas the perception of a face as distorted led to a reduced amplitude. This effect was slightly pronounced over the right hemisphere (electrode position P08) as over the left hemisphere (electrode position P07). The authors interpreted the results in favour of population coding more than just a decline in neuronal activity. A similar pattern was found by Walther, Schweinberger, Kaiser and Kovács (2013) for identity-specific aftereffects. This indicates that the changes of the P250 component are not limited to face figural aftereffects.

1.4 Aim and research questions

Taken together, the aims of the present study are to first, reproduce the findings that adaptation is based on more than one mechanism. These different mechanisms might explain the different timescales of adaptation. The second aim is to concretise the relationship between adaptation and learning. The question is whether effects that benefits learning also have a beneficial effect on adaptation. The third aim is to identify an

electrophysiological marker of face adaptation that correlates with the subjective perception.

Participants adapted to distorted faces (=adaptation). While one half of participants adapted twelve minutes straight, the other half adapted twelve minutes distributed over time. Afterwards, an adaptation to the opposite direction of distortion (=deadaptation) took place and was directly followed by a test phase.

We assumed that the continuation of the original adaptation effect is influenced by (a) the manipulated adaptation condition (blocked vs. distributed) and (b) the size of the initial adaptation effect. Further, we hypothesized that the continuation of the original adaptation effect directly influenced (a) the strength of the return of the original adaptation effect within the test phase and (b) the time it takes to deadapt from the initial effect. On a neuronal level, the adapter should elicit a higher amplitude of the P250 component at the end of adaptation compared to the beginning because a higher amplitude is associated with a more undistorted perception. The increase in amplitude should be directly correlated with the continuation of the original adaptation effect. The hypothesis concerning the N170 component is not that simple. Nearly all studies analysed the changes of the N170 in response to the test face and not to the adapter face. If the N170 acts as a marker of a distorted perception, the amplitude should be reduced and the latency should be longer at the early stages compared to the late stages of adaptation.

2. Method

2.1 Participants

To ensure a sufficiently large sample size to detect potential effects which means to guarantee an appropriate power a prospective power analysis was conducted. It was based on the behavioural results of a preliminary, not published study with the same experimental design. In this preliminary study a MANOVA was calculated with two between subject factors (DISTORTION extended vs. compressed and ADAPTATION blocked vs. distributed) and four dependent variables (baseline, adaptation, deadaptation, post-test). The effect size of the almost significant difference between the distributed and blocked condition, $p = .062$, $\eta^2 = .275$, was used as the effect size estimate. The power analysis was conducted using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). Under the assumption of the effect size estimate a power of 80% is obtained with a sample size of 24 participants (see Figure A1).

Because of the expectation to subsequently exclude subjects, 30 participants (17 females, aged between 18-40) were recruited via the Sona LABS System of the University of Vienna and from the investigators circle of friends and acquaintances. All participants had a normal or corrected-to-normal vision, were right handed and blind to the purpose of the study. They gave their written informed consent to participate in the study. The final sample for the behavioural analysis consisted of 24 participants (14 females, $M_{age} = 26.08$, $SD_{age} = 5.36$) which corresponds to a data loss of 20%. The data of one participant was lost because of technical problems. Two participants were excluded from further analysis since they did not adapt at all during adaptation. The variance of the subjectively rated face distortion of three participants was too high during the last 50 trials of adaptation. One participant mixed up the keys during baseline. The baseline was interpolated based on the other subjects in the same condition, so the participant was kept in the sample.

The final sample for the electrophysiological analysis persisted of 18 participants (9 females, $M_{age} = 25.94$, $SD_{age} = 4.81$). Two participants had to be excluded because of technical issues during EEG recordings. 4 Participants had less than 20 trials without artefacts in the early or late adaptation phase.

For the behavioural as well as for the electrophysiological analysis the presence of outliers was checked. There were no extreme outliers, $x < Q_1 - 3 * (Q_3 - Q_1)$ or $x > Q_3 + 3 * (Q_3 - Q_1)$, Q_1 = first quartile, Q_3 = third quartile. So, all remaining participants were kept in the sample.

2.2 Experimental design

The present study followed a 2x2x4 design with the two between-subject factors DISTORTION (extended vs. compressed) and ADAPTATION (blocked vs. distributed) and the within-subjects factor PHASES (baseline, adaptation, deadaptation and post-test). Participants were randomly assigned to one of four experimental conditions: (a) blocked and extended, (b) blocked and compressed, (c) distributed and extended and (d) distributed and compressed. The factor distortion describes the direction of distortion of the adapter face that was used during adaptation. The factor adaptation designates the two different kinds of adaptation.

2.3 Stimuli

Distorted versions of a coloured picture of the actor George Clooney on a grey background were presented. George Clooney was used because representations of familiar faces already exist. It is easier to influence the perception in the long term if the adapted faces are familiar (Carbon & Ditye, 2011). All participants reported that they know George Clooney.

The distortion of George Clooney was induced through an extension or compression of the face along the vertical axis. A nonlinear transformation was applied to the original face (distortion degree, d = 0) to create gradually extended faces (d = [+10, +20, …, +180]) and gradually compressed faces (d = [-10, -20, …, -180]). In total, 18 gradations of distortion in each direction, compressed and extended, and the undistorted face (d = 0) were used as stimuli (see Figure 1). To avoid retinal effects induced by position, the images were slightly shifted along the vertical axis between trials. The images were shown on a 19-inch tube monitor with a screen resolution of 1024x768

pixels. The size of the images was 350x257 pixel which refers to a visual angle of 11° horizontal and 15° vertical.

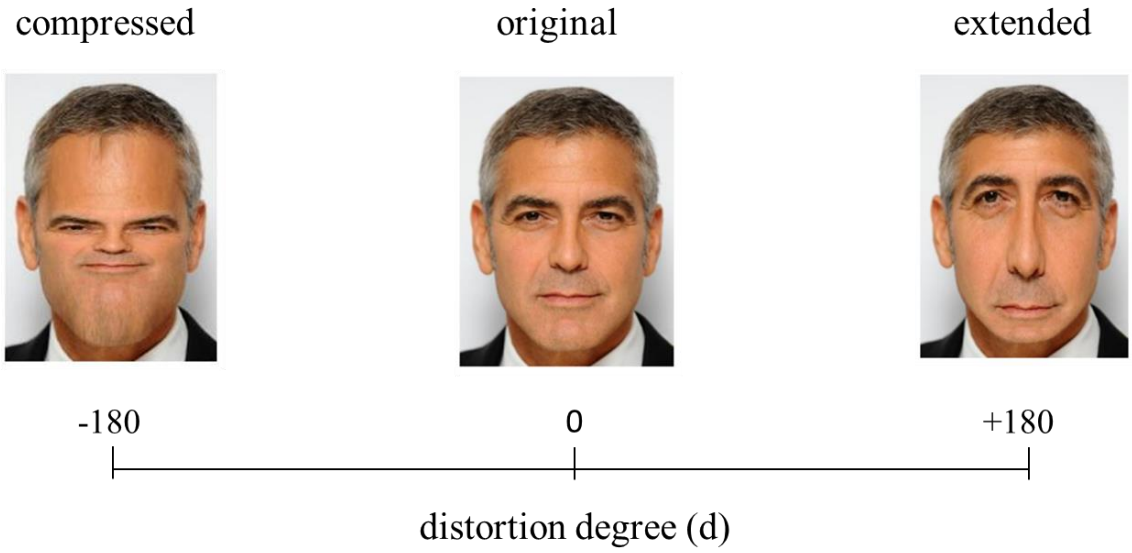

Figure 1. The stimuli that was used with 36 gradations ranging from extremely compressed (d = -180) to extremely extended (d = +180) in steps of d = 10. D = 0 is the undistorted normal face.

2.4 Procedure & Task

At the beginning of testing all participants gave their consent to voluntarily take part in the study. After an oral instruction, they were tested in a dimly lit room with a chin rest to fixate the head position and with a distance to the screen of roughly 50 cm. During the entire test session, the EEG was recorded. The experiment was controlled by Matlab (2013b, The MathWorks, Natick, MA) using Psychophysics Toolbox extensions (Brainard, 1997).

The task consisted of 4 phases: baseline, adaptation, deadaptation, and post-test. On each baseline trial, a target face was presented for 200 ms. Target faces refer to all faces that should be assessed by participants according to their extension or compression. The target face was preceded by a fixation cross for 1400 ms and was followed by a white screen for a maximum of 1500 ms. Participants now had to indicate whether they

perceived the target face as extended or compressed using two different keys (f and j) on a keyboard.

The baseline lasted for 3 minutes and consisted of 30 trials. Within the first trial the original face (d = 0) was used as the target face. A staircase procedure was applied to track the perception in every trial. Every time a participant perceived the target face as too compressed, the target face was extended (d = +10) in the following trial and vice versa. Therefore, an approximation to the perception of the target face was possible.

At adaptation, a distorted face (d = +180 or d = -180) was presented as the adapter for 1400 ms. Following the adapter face, again the target face was shown for 200 ms. Participants should again assess whether they perceived the target face as compressed or extended. There were two conditions concerning the kind of adaptation: blocked and distributed. During the blocked adaptation, participants adapted for twelve minutes straight (180 trials), whereas during the distributed adaptation participants adapted for twelve minutes distributed over twelve blocks (one block = one minute). Each of the twelve blocks was followed by a simple reaction time task for three minutes. Thus, the adaptation and the reaction time task alternated in the distributed condition. In the blocked condition the reaction time task was carried out before the adaptation and took 36 minutes (see Figure 2).

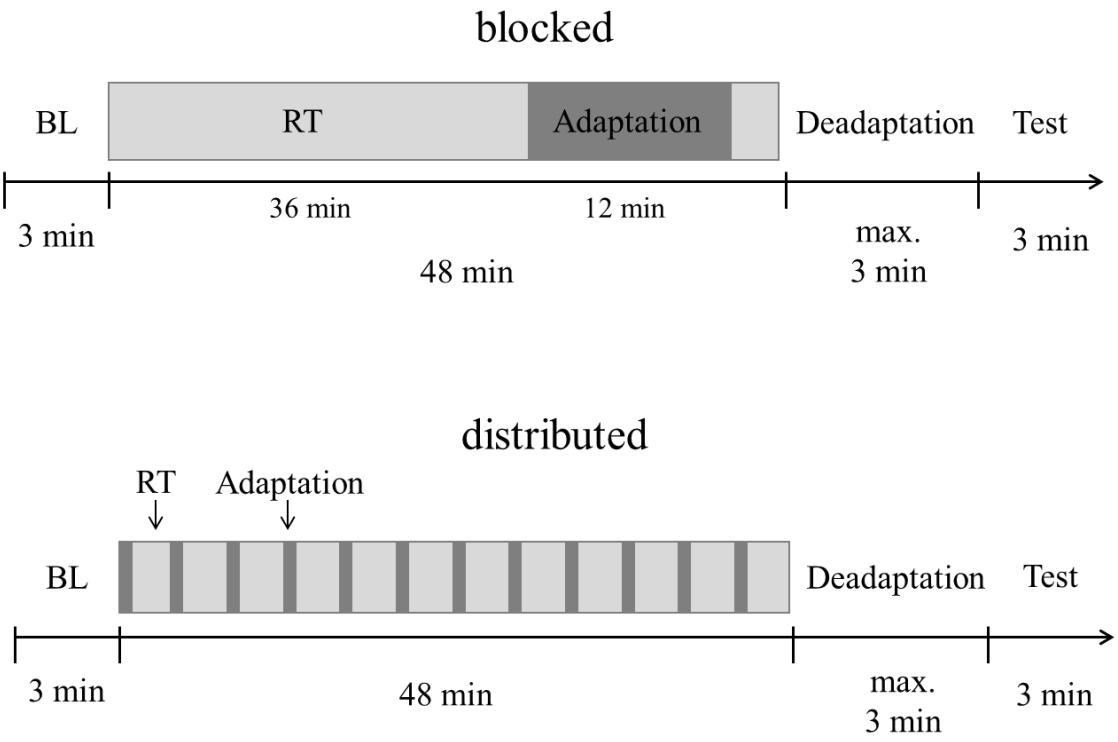

Figure 2. Experimental procedure. The experiment was the same for all participants except the adaptation period. One group (blocked condition) started with a 36-minute reaction time task (RT) which was followed by a blocked 12-minute adaptation. The other group (distributed condition) alternately conducted the RT (3 min) and the adaptation period (1 min). In total, the second group performed 12 adaptation periods and 12 RTs so that both groups needed 48 minutes for the adaptation. Before adaptation a 3-minute baseline was carried out. The adaptation was followed by a deadaptation period that lasted for a maximum of 3 minutes and a 3-minute post-test.

The procedure of the presentation of faces during deadaptation was the same as during adaptation. An adapter was presented and then followed by a target face. The target face was assessed as to its distortion, extended or compressed. The target face in the first trial of deadaptation was distorted to the same degree as the target face in the last trial of adaptation. The adapter was distorted (d = +180 or d = -180) in the opposite direction to the adapter that was used during adaptation in order to compensate for the original adaptation effect. To ensure this compensation, an abort criterion was applied. For participants who adapted to the extended face (d = +180), the abort criterion in the deadaptation phase was defined as: $d(t_x) < 0$ and $d(t_x+1) < 0$ or $x > 40$. Otherwise, the

abort criterion in the deadaptation phase was analogously defined as: $d(t_x) > 0$ and $d(t_x+1) > 0$ or $x > 40$. 't_x' refers to trial x during deadaptation. Therefore, deadaptation stopped after a maximum of 40 trials.

The post-test was conducted identically to the baseline except for the length. In total, 100 trials were presented during post-test. Please see Figure 3 for an example of one trial of each of the four phases: baseline, adaptation, deadaptation and post-test.

The overall experimental time was about 60 minutes.

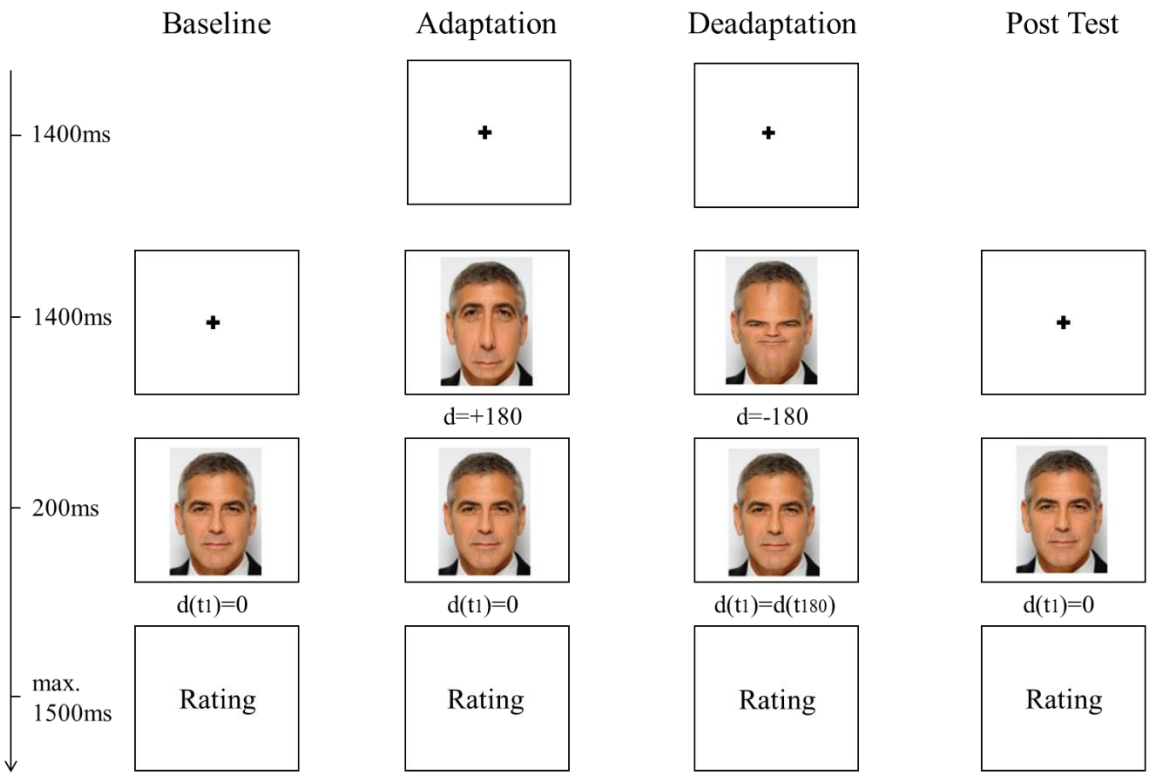

Figure 3. The four phases of the experiment. Baseline and post-test started with a 1400 ms fixation cross which was followed by the test phase. In trial one of both phases the distortion degree (d) was 0, so the face was not distorted. Adaptation and deadaptation also began with a 1400 ms fixation cross but right afterwards an adapter was presented. The distortion degree of the adapter was either +180 (extended) in the adaptation period and -180 (compressed) in the deadaptation period or vice versa. The test face of the deadaptation phase had the distortion degree of the last trial of the adaptation phase ($d(t_1) = d(t_{180})$). After the 200-ms test face the participants had maximal 1500 ms to rate whether they perceive the test phase as compressed or extended.

2.5 EEG recordings and analysis

A full-band DC-EEG system (neuroConn GmbH, Ilmenau, Germany) with 64 active electrodes was used. The electrodes were placed according to the 10-20 system (Jasper, 1958) and were referenced to FCZ. Horizontal and vertical electrooculograms (EOG) were recorded to allow offline eye movement artifact correction. The recordings were done with a sampling rate of 1,000 Hz, a low pass filter of 100 Hz and a high pass filter of 0.1 Hz. Impedances were kept below 5 kΩ. For offline processing of the EEG signal EEGLab (Delorme & Makeig, 2004) implemented in Matlab (2013b, The MathWorks, Natick, MA) was used.

The raw EEG signal was filtered utilizing a 40 Hz low pass filter and was re-referenced against the averaged mastoid electrodes (TP9 and TP10).

For further analysis, 1200 ms long epochs were segmented, starting 200 ms before stimulus onset (Burkhardt et al., 2010). For automatic artifact correction different criteria were defined. To clean the data from eye movements a threshold for the vertical eye movements was set at -60 µV or 60 µV and for the horizontal eye movements at 30 µV or -30 µV. All trials with identified eye artifacts were eliminated. To reject movement artifacts channels with a gradient higher than 50 µV/ms, an amplitude higher than 80 µV or lower than -80 µV or a lower difference than 0.5 µV between the minimum and maximum within a 500 ms interval were excluded.

Artefact-free epochs were averaged over a maximum of 60 trials of early and late adaptation. Because not all 60 trials were artefact-free the used number of trials was lower and varied between the two periods of adaptation. For the early adaptation period the average number of trials was 42 (SD = 7.92, minimum = 26, maximum = 54) and for the late adaptation period the average number of trials was 36 trials (SD = 10.13, minimum = 23, maximum = 53). A problem is that the number of trials of the early adaptation period was significantly higher than the number of trials of the late adaptation period, $t(1, 20) = 3.20$, $p = .005$. This issue is debated in the discussion section.

Based on the grand average the peak amplitude and latency of N170 and P250 within a time window of 125-180 ms or 185-260 ms post stimulus were selected. As previous studies have found clear waveforms for the processing of faces at PO7 and PO8 (Burkhardt et al., 2010; Kovács et al., 2006) the same electrode positions were used in this study.

2.6 Statistical analysis

For the statistical analysis SPSS 22 (IBM Corporation, Armonk, USA) was used. The significance level was set at $\alpha = 5\%$ ($p < .05$) and a statistical trend was defined as $\alpha = 10\%$ ($p < .10$).

On the behavioural level two measurements were used to compare the two different conditions of adaptation and the directions of the distortion of the adapter face.

The first measurement that should be susceptible to the face figural aftereffect during adaptation is the strength of return of this face figural aftereffect within the test phase. To test the hypothesis that the distributed adaptation leads to a stronger return of the face figural aftereffect compared to the blocked adaptation (between-subjects factor ADAPTATION) and that the face figural aftereffect is generalizable across the extended and compressed distortion (between-subjects factor DISTORTION) throughout baseline, adaptation and post-test (within-subjects factor PHASES) a 2 (distributed vs. blocked) x 2 (extended vs. compressed) x 3 (baseline vs. adaptation vs. post-test) repeated measures analysis of variance (ANOVA) was conducted. The distortion degree, indicating the perceived face figural aftereffect, was defined as the dependent variable and was averaged over the last 20 trials of the baseline, last 50 trials of the adaptation as well as the last 50 trials of the post-test. According to the Shapiro-Wilk test, the residuals of the dependent variable for all three phases (baseline, adaptation and post-test) did not differ significantly from a normal distribution, baseline: $W = .928$, $p > .05$; adaptation: $W = .959$, $p > .05$; post-test: $W = .928$, $p > .05$. Additionally, the Q-Q-Plot did not demonstrate a striking deviation from a linear relation between expected normally distributed and observed values. As the Mauchly's test showed that the assumption of sphericity was not met, $\chi^2(2) = 9.45$, $p = .009$, degrees of freedom were corrected using the Greenhouse-Geisser procedure, $\varepsilon = .72$.

The second measurement that should be directly influenced by the face figural aftereffect during adaptation is the amount of trials needed to deadapt from this aftereffect. The assumption is that participants in the distributed condition need more trials to deadapt compared to the blocked condition. The number of trials of the deadaptation phase in the extended condition should not significantly deviate from the number of trials in the compressed condition. The distortion degree in the last trial of the adaptation phase was the first one in the deadaptation phase. Therefore, participants with

a higher distortion degree at the end of adaptation automatically needed more trials to reach the abort criterion of the deadaptation phase and vice versa. To take this into account the relative length of deadaptation as the dependent variable was calculated as follows: the absolute number of trials of the deadaptation phase – the minimal number of trials needed to reach the abort criterion. As a result, the relative length of deadaptation could range between 0 and 40.

The residuals of the relative length of deadaptation were not normally distributed, Shapiro-Wilk test: $W = .87$, $p < .05$. Thus, a first Mann-Whitney-U-test was used to investigate the different length of deadaptation between the extended vs. compressed condition (DISTORTION) and a second one was used to test if the distributed adaptation led to a longer deadaptation phase compared to the blocked adaptation (ADAPTATION).

Due to the fact that the electrophysiological analysis was restricted to an early and late adaptation period, a similar ANOVA was calculated for the behavioural effects.

To examine if the distributed adaptation led to stronger perceived distortions at the end of adaptation in comparison to the blocked adaptation (between-subjects factor ADAPTATION) and in comparison to the beginning of adaptation (within-subjects factor EARLYLATEADAP) a 2 (distributed vs. blocked) x 2 (extended vs. compressed) x 2 (early vs. late adaptation) repeated measures ANOVA was executed. Again, the second between-subjects factor DISTORTION was taken into account to control for the generalisability of the face figural aftereffect across the extended and compressed condition. The dependent variable was still the distortion degree but for this analysis it was averaged over the first 50 and the last 50 trials of adaptation.

The Shapiro-Wilk test did not reveal a significant difference between the distribution of the residuals and the normal distribution, neither for the first 50 trials, $W = .935$, $p > .05$, nor for the last 50 trials of adaptation, $W = .959$, $p > .05$. Moreover, the Q-Q-Plot did not show a noteworthy deviation. So, the ANOVA was suitable.

On a neuronal level the N170 and P250 were investigated. Indeed, the previously described 2 (distributed vs. blocked) x 2 (extended vs. compressed) x 2 (early vs. late adaptation) repeated measures ANOVA did not show any differences, neither between the distributed and blocked condition nor between the extended and compressed condition (see the results section). Thus, all groups were analysed together.

To identify an electrophysiological marker for the perceived distortion four repeated measures ANOVAs were applied. To examine the assumption that the P250

reflects the perceived degree of distortion (higher amplitude of P250 means a lower subjective perception of distortion) and further, that the perceived distortion of the adapter face decreases during adaptation a 2 (early vs. late adaptation) x 2 (PO7 vs. PO8 electrode) repeated measures ANOVA was performed. As dependent variables the amplitude and latency of P250 were used. They were defined for the average of the first 42 trials and the last 36 trials of adaptation (within-subjects factor EARLYLATEADAP) at the electrode positions PO7 and PO8 (within-subjects factor HEMISPHERE).

The residuals of the amplitude and latency of P250 were nearly all normally distributed, amplitude | early adaptation | PO8: $W = .949$, $p > .05$, amplitude | late adaptation | PO8: $W = .935$, $p > .05$, amplitude | late adaptation | PO7: $W = .945$, $p > .05$, latency | early adaptation | PO8: $W = .918$, $p > .05$, latency | early adaptation | PO7: $W = .953$, $p > .05$, latency | late adaptation | PO7: $W = .942$, $p > .05$. But the residuals of the amplitude at early adaptation at electrode position PO7, $W = .885$, $p < .05$, and the latency at late adaptation at electrode position PO8, $W = .837$, $p < .05$, did significantly deviate from a normal distribution (see Figure A2 and A3 for the Q-Q-Plot). In the fact that just one variable per ANOVA was not normally distributed and an equal cell size was given the ANOVA was nevertheless calculated.

The same 2 (early vs. late adaptation) x 2 (PO7 vs. PO8 electrode) repeated measures ANOVA was used to test whether a changing pattern of the amplitude and latency can also be found for the N170 component. The amplitude and latency of the N170 component were defined based on the same averaged trials as the amplitude and latency of the P250 component.

The residuals of the amplitude and latency for the early and late period of adaptation at the electrode positions PO7 and PO8 did not significantly differ from a normal distribution, amplitude | early adaptation | PO8: $W = .963$, $p > .05$, amplitude | early adaptation | PO7: $W = .930$, $p > .05$, amplitude | late adaptation | PO8: $W = .914$, $p > .05$, amplitude | late adaptation | PO7: $W = .945$, $p > .05$, latency | early adaptation | PO8: $W = .910$, $p > .05$, latency | late adaptation | PO8: $W = .916$, $p > .05$. However, the latency of early, $W = .824$, $p < .05$, and late, $W = .874$, $p < .05$, adaptation at PO7 did significantly deviate from a normal distribution (see Figure A4 and A5 for the Q-Q-Plot). The same argument mentioned before justifies the application of an ANOVA. Just two out of four variables are not normally distributed, the cell size was equivalent and the ANOVA is relatively robust against violations of assumptions.

For all analyses partial eta squared (η^2) was indicated to clarify the effects of the repeated measures ANOVAs. $\eta^2 > .01$ refers to a small, $\eta^2 > .06$ to a medium and $\eta^2 > .16$ to a large effect (Rasch, Friese, Hofmann, & Naumann, 2010). All multiple comparisons (post hoc tests and the two Mann-Whitney-U-tests) were corrected according to Bonferroni (p_{corr} refers to the corrected p-value and p_{crit} refers to the corrected significance level).

3. Results

3.1 Behavioural Results

The 2 (distributed vs. blocked) x 2 (extended vs. compressed) x 3 (baseline vs. adaptation vs. post-test) repeated measures ANOVA revealed that the face figural aftereffect significantly changed throughout baseline, adaptation and post-test, $F(1.44, 28.74) = 68.06$, $p < .001$, $\eta^2 = .773$. The face figural aftereffect (distortion degree) during the last 50 trials of adaptation was significantly stronger compared to the baseline, $p_{corr} < .001$. Additionally, the face figural aftereffect was stronger in the post test than in the baseline, $p_{corr} = .005$, but weaker than during adaptation, $p_{corr} < .001$.

There were no significant differences in the face figural aftereffect neither between the compressed and extended condition, $F(1, 20) = 1.03$, $p = .322$, $\eta^2 = .049$, nor between the blocked and distributed condition, $F(1,20) = 0.16$, $p = .659$, $\eta^2 = .010$. Moreover, the increase of the face figural aftereffect during adaptation and the slightly decrease in the post-test did not differently vary between the compressed and extended condition, $F(1.44, 28.74) = 1.40$, $p = .258$, $\eta^2 = .065$, and the blocked and distributed condition, $F(1.44, 28.74) = 0.35$, $p = .636$, $\eta^2 = .017$ (see Figure 4).

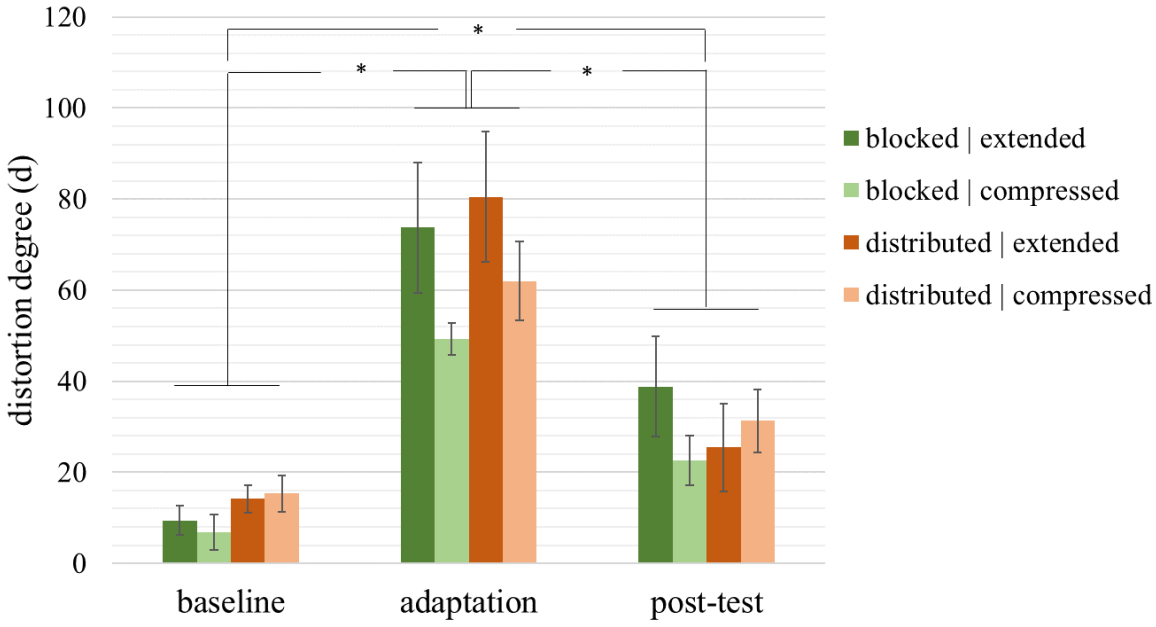

Figure 4. Behavioural results. The distortion degree (d) as indicator for the face figural aftereffect for the blocked vs. distributed and extended vs. compressed condition across the three phases baseline, adaptation and post-test. There was a significant increase in face figural aftereffects from baseline to adaptation and a significant decrease from adaptation to post-test for all groups. The original face figural aftereffect recovered during post-test (significant increase in comparison to baseline) but was weaker compared to adaptation. Depicted are the means of distortion degree and the standard error.

The Mann-Whitney-U-tests underpinned the result that the distributed adaptation did not enhance the face figural aftereffect compared to the blocked adaptation. Precisely, the distributed adaptation did not lead to a longer deadaptation phase, $U(10, 14) = 70.5$, $p = .977$, $p_{crit} = .025$. The same pattern could be found for the distortion. The length of the deadaptation was statistically the same for both, the extended and compressed condition, $U(11, 13) = 77$, $p = .749$, $p_{crit} = .02$.

The 2 (distributed vs. blocked) x 2 (extended vs. compressed) x 2 (early vs. late adaptation) repeated measures ANOVA disclosed that the face figural aftereffect was clearly stronger in the late stage compared to the early period of adaptation, $F(1, 20) = 29.36$, $p < .001$, $\eta^2 = .595$. Additionally, the results supported the findings of the 2 (distributed vs. blocked) x 2 (extended vs. compressed) x 3 (baseline vs. adaptation vs. post-test) repeated measures ANOVA. Namely, that there were no significant differences

in the face figural aftereffect neither between the compressed and extended condition, $F(1, 20) = 1.79$, $p = .195$, $\eta^2 = .082$, nor between the blocked and distributed condition, $F(1,20) = 0.56$, $p = .462$, $\eta^2 = .027$. Because there seems to be no distinction in behaviour between the extended and compressed condition and the distributed and blocked condition, all participants were further analysed together.

3.2 Electrophysiological Results – ERPs

The 2 (early vs. late adaptation) x 2 (PO7 vs. PO8 electrode) repeated measures ANOVAs for the P250 showed that the amplitude, $F(1, 17) = 0.17$, $p = .682$, $\eta^2 = .010$, and the latency, $F(1, 17) = 0.13$, $p = .724$, $\eta^2 = .008$, did not significantly differ between the early and late phase of adaptation. There was no deviation between the amplitudes, $F(1, 17) = 0.24$, $p = .632$, $\eta^2 = .014$, but an almost significant difference between the latencies, $F(1, 17) = 3.42$, $p = .082$, $\eta^2 = .167$, at the electrode positions PO7 and PO8. Additionally, the interaction between the electrode positions and the early and late phases of adaptation was significant for the latency, $F(1, 17) = 7.39$, $p = .015$, $\eta^2 = .303$, but not for the amplitude, $F(1, 17) = 0.47$, $p = .505$, $\eta^2 = .027$. Post-hoc tests revealed that the latency was higher at PO8 compared to PO7 but just at the early, $p_{corr} = .016$, and not at the late stage of adaptation, $p_{corr} = .813$. Further, the latency tended to be higher at the late stage compared to the early stage of adaptation but just at the electrode position PO7, $p_{corr} = .085$, and not at electrode position PO8, $p_{corr} = .218$.

The 2 (early vs. late adaptation) x 2 (PO7 vs. PO8 electrode) repeated measures ANOVAs for the N170 revealed that neither the amplitude, $F(1, 17) = 0.02$, $p = .883$, $\eta^2 = .001$, nor the latency, $F(1, 17) = 0.08$, $p = .785$, $\eta^2 = .004$, changed during adaptation. There was no difference between the amplitudes, $F(1, 17) = 0.42$, $p = .524$, $\eta^2 = .024$, and latencies, $F(1, 17) = 0.15$, $p = .708$, $\eta^2 = .008$, at electrode positions PO7 and PO8. The interaction between the early and late phase of adaptation and the electrode positions did not reach significance for the amplitude, $F(1, 17) = 2.24$, $p = .153$, $\eta^2 = .116$, and latency, $F(1, 17) = 1.83$, $p = .194$, $\eta^2 = .097$. Please, see Figure 5 for the plotted grand average ERPs.

Since there were no consistent and clear differences between the N170 or P250 at the early and late stage of adaptation no further analyses were conducted to link the behavioural effects with electrophysiological changes.

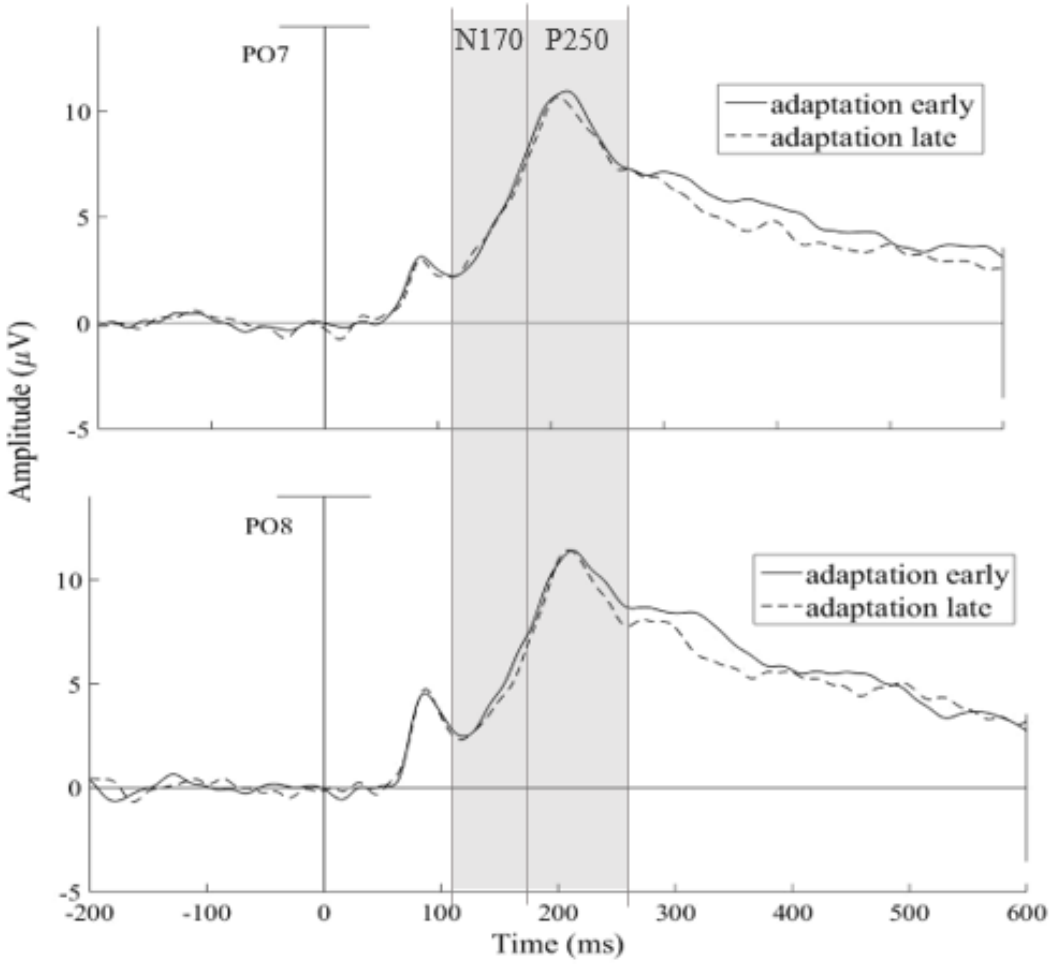

Figure 5. The plotted grand average ERPs for the early and late phase of adaptation at the electrode positions PO7 (top figure) and PO8 (bottom figure). The grey areas represent the time windows that were used for analyses.

4. Discussion

Adaptation reflects a rapid calibration of the visual system to a permanently changing environment. In this thesis, the underlying mechanisms of adaptation were examined. To replicate previous findings that there is more than one mechanism subserving adaptation participants adapted to a distorted face. After adaptation a shorter deadaptation took place and was followed by a post-test without any face adapter. To investigate the relationship between adaptation and learning one group of participants adapted in a distributed condition whereas another group adapted in one block. During the whole experiment EEG was recorded to identify an electrophysiological correlate of the distorted perception.

We could replicate the finding that more than one mechanism underlies adaptation. The benefit of distributed learning could not be found for adaptation. Thus, distributed adaptation did not lead to higher visual aftereffects compared to blocked adaptation. Additionally, neither the N170 nor the P250 component reflected the distorted perception of the faces during adaptation.

4.1 Adaptation- more than one mechanism

The first part of this thesis aimed at replicating previous findings indicating that adaptation is based on more than one mechanism (Mesik et al., 2013). These different mechanisms might imply that adaptation acts on different time scales. We could demonstrate that an adaptation of 12 minutes to a distorted face led to strong face figural aftereffects. These face figural aftereffects returned after a second, shorter adaptation (deadaptation) to a face that was distorted in the opposite direction. According to Mesik et al. (2013), the 12-minutes adaptation and the shorter deadaptation are based on different mechanisms. The longer adaptation seems to rely on a slower mechanism which acts on longer time scales. The shorter deadaptation seems to rely on a faster mechanism that involves a shorter time scale. It takes more time to change the slow mechanism but consequently the effect will last longer in comparison to the fast mechanism. Thus, deadaptation or the fast mechanism could not totally delete the longer adaptation or the

slow mechanism and just diminished it. The contribution of multiple mechanisms is not exclusive for higher level aftereffects like faces as they are also involved in lower level aftereffects like contrast (Bao & Engel, 2012).

Additionally, the present results are in line with neurophysiological experiments that have shown diverse alterations in different cortical areas in response to lower level adaptation. The involvement of different cortical areas indicates the existence of multiple mechanisms in adaptation procedures (Rieke & Rudd, 2009). More precisely, in primary visual cortex (V1) neurons have different preferences for instance for different orientations. These preferences can be depicted in tuning curves that represent the activity (spiking rate) of neurons in dependence of various orientations. In relation to adaptation, an exposure to an oriented grating (e.g. -45 degrees) led to a shift of the cells' tuning curve in the opposite direction of this grating (e.g. +40 degrees as the highest peak instead of +20 degrees as the highest peak before adaptation). Thus, the neurons become less sensitive to stimuli having the same orientation as the adapter (Dragoi et al., 2000). Interestingly, the converse shift of the tuning curve was found for neurons in higher cortical areas in response to adaptation. A prolonged stimulation to drifting gratings resulted in a shift of the tuning curve towards the adapter. This implies that the sensitivity to stimuli having similar properties like the adapter is not strongly reduced (Kohn & Movshon, 2004). Therefore, different mechanisms of adaptation might be explained by diverse alterations of neurons in different cortical areas. This assumption is worded in very general terms. Of course a lot more evidence is required to close the gap between our and previous behavioural findings and the just described neurophysiological results.

However, the existence of different timescales relating to adaptation can also be framed in a broader sense. The norm based model (Valentine, 1991) states that faces are evaluated in terms of their deviation from a prototype face. Adaptation to a distorted face could lead to changes of this prototype face and thus, induces a shift of the face norm. Such shifts of the face norm appear when modifications are persistent (Webster 2011). A prolonged adaptation (slow mechanisms) could cause a change of the face norm whereas a shorter adaptation (fast mechanisms) could not. This might be one reason why the original adaptation effect withstood deadaptation.

4.2 Adaptation, learning and the spacing effect

Another aim of this thesis was to concretise the relationship between adaptation and learning. The question was whether the beneficial effect of distributed learning (spacing effect) can also be found for adaptation. However, this was not the case. The group of participants that adapted in a distributed way – compared to those who adapted in a blocked way – did not show higher visual aftereffects at the end of adaptation nor at the post-test. Additionally, the distribution of adaptation did not lead to a longer deadaptation.

The spacing effect has been shown for a large number of learning conditions and learning materials such as vocabulary learning (Callan & Schweighofer, 2010), face learning (Xue et al., 2011) and learning of unfamiliar stimuli like non-words (Russo, Mammarella, & Avons, 2002). However, all these learning conditions rely on explicit memory. Additionally, the instruction was mostly intentional, meaning that participants were instructed to learn the presented material. If adaptation reflects learning, it would rely on implicit memory rather than explicit memory and it would be incidental rather than intentional learning.

Regarding implicit memory and incidental learning, the spacing effect is not stable. For instance, a list of words was presented either distributed or blocked. There were no instructions about a later recall which reflects incidental learning. Following a distractor phase, participants were tested with an implicit memory task. In the test phase no beneficial effect was found for words that were presented in a distributed way compared to a blocked presentation (Perruchet, 1989). Therefore, an advantage of a distributed presentation of learning material – the spacing effect – is not present when implicit memory is involved and when learning is incidental. As a consequence, it is not surprising that we could not demonstrate a benefit of a distributed adaptation in comparison to a blocked adaptation period.

Contrary to our and the previously discussed results, Magnussen and Johnsen (1986) reported a spacing effect in an adaptation procedure. Participants had to adapt to a tilted line in clockwise or counterclockwise direction. To measure visual aftereffects, a test line had to be vertically aligned. The 10-minutes adaptation period was conducted distributed over 15 minutes or blocked. Interestingly, distributed adaptation led to stronger visual aftereffects than the blocked adaptation. The authors interpreted this result

in favour of a two-stage model instead of the comparability of adaptation and learning. The model implies that adaptation occurs on two stages which is in line with our results demonstrating that more than one mechanism underlies adaptation.

One limitation and a reason why we could not replicate the findings of Magnussen and Johnsen (1986) might be the sample size despite the fact that an a priori power analysis was conducted. As the power analysis was premised on a nearly significant result the effect size estimate could be overestimated. This would result in an underestimation of the needed sample size to detect the effect.

4.3 N170 and P250 might not reflect distorted perception

The last purpose of this thesis was to identify an electrophysiological marker of face adaptation that correlates with the subjective perception.

Many studies related the N170 to face adaptation (Kovács et al., 2006; Kovács, Zimmer, Webster, 2011; Volberg, Lavric, & Rossion, 2013). After an adapter face a test face elicited a reduction in N170 peak amplitude and an increase in latency when it was perceived as distorted but just over the right hemisphere (Zimmer & Kovács, 2011). We could not found these effects since there was no difference between the N170 amplitude and latency between early and late adaptation nor between the electrode positions. In fact, the changes of amplitude and latency were analysed in response to the adapter face rather than to the test phase. This can be stated as one limitation of the present study. The test face changed trial by trial depending on the rating of participants. The staircase procedure did not allow an analysis of the target faces because they were perceived as normal. To investigate an electrophysiological correlate of distorted perception in long-term adaptation we had to analyse the adapter faces at the beginning and end of adaptation.

Our results concerning the N170 component were in line with the findings of Burkhardt et al. (2010). They presented distorted faces ranging from extremely extended to extremely compressed. The amplitude of the N170 component was not affected by the varying forms of distortion. Moreover, they demonstrated a more sensitive electrophysiological marker, the P250. The P250 peak amplitude decreased with an increase in distorted perception of faces but just for extended faces. The effect for compressed faces was weak and not always present. Comparable results have been shown

for identity aftereffects (Walther et al., 2013). We expected a similar pattern, meaning a reduction in amplitude and an increase in latency at the beginning compared to the end of adaptation in response to the adapter face. In contrast to that expectation, the P250 peak amplitude did not differ between the early and late phase of adaptation and between the electrode positions. Ganis and Schendan (2008) analysed ERPs in response to adapter faces and found a reduction for a N250 and not for a P250 component at occipito-temporal sites. In addition to the before described studies, this result illustrates that the literature is quite divergent with respect to the kind of adaptation (e.g. adaptation to distorted faces or to identities), the stimulus that is used for analysis (adapter face or test face) and the connection between electrophysiological and behavioural effects.

For the P250 latency we found a significant trend but just at PO7 which contradicts the most previous studies. Usually, the effects are present at both electrode positions but are more dominant at PO8 compared to PO7. So, the effect is questionable.

In this context, there is one noteworthy limitation regarding our ERP analyses. Like mentioned in the method section, the number of trials was significantly higher in the early adaptation than in the late adaptation period. ERPs are methodologically based on the idea that the signal-to-noise ratio is better when a lot of trials were averaged. As the noise is randomly distributed across the trials and the signal is locked to a stimulus onset averaging improves the signal-to-noise ratio. It is valid that the more trials, the better the signal-to-noise ratio. Thus, a considerable difference between the number of trials in each factor level (in this case early and late adaptation) would lead to an imbalance in precision of the signal and further, to inaccurate results.

Additionally, no trial matching was carried out. Trial matching could be necessary if many trials were excluded because of artefacts. In our case 60 trials were used for the early adaptation period and 60 for the late adaptation period but artefacts were still included. The problem is now that maybe for most participants the first 30 trials of the early adaptation and the last 30 trials of the late adaptation were rejected because of artefacts. As a consequence, the effects of the early and late adaptation period would be diminished or even cancelled out. Thus, a matching between the trials of the two factor levels would be necessary to more accurately assess the effects.

5. Conclusion

Participants had to adapt to distorted faces to concretise the underlying mechanisms of adaptation, its relationship to learning and its electrophysiological correlates. The results demonstrated that adaptation and learning seems to be different at least concerning a spacing effect. To definitely distinguish adaptation from learning following investigations have to be carry out. Moreover and controversial to previous findings, no electrophysiological marker could be identified for face figural aftereffects. Especially these results highlight the difficulties in comparing findings of studies using diverse experimental designs. To clearly determine electrophysiological marker further studies with an equivalent design are needed.

It could be shown that adaptation is not limited to one mechanism and more generally, adaptation lead to an individual distortion in perception. Relating to the opening quotation adaptation and visual aftereffects are convincing examples for the fact that “the reality (…) is not true reality, but is appearance“ (Bradley, 1897, Chapter III, para. 1).

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List of figures

List of abbreviations

d	distortion degree
EEG	electroencephalography
EOG	electrooculogram
ERP	event related potential
REM	rapid eye movement
V1	primary visual cortex

Appendix

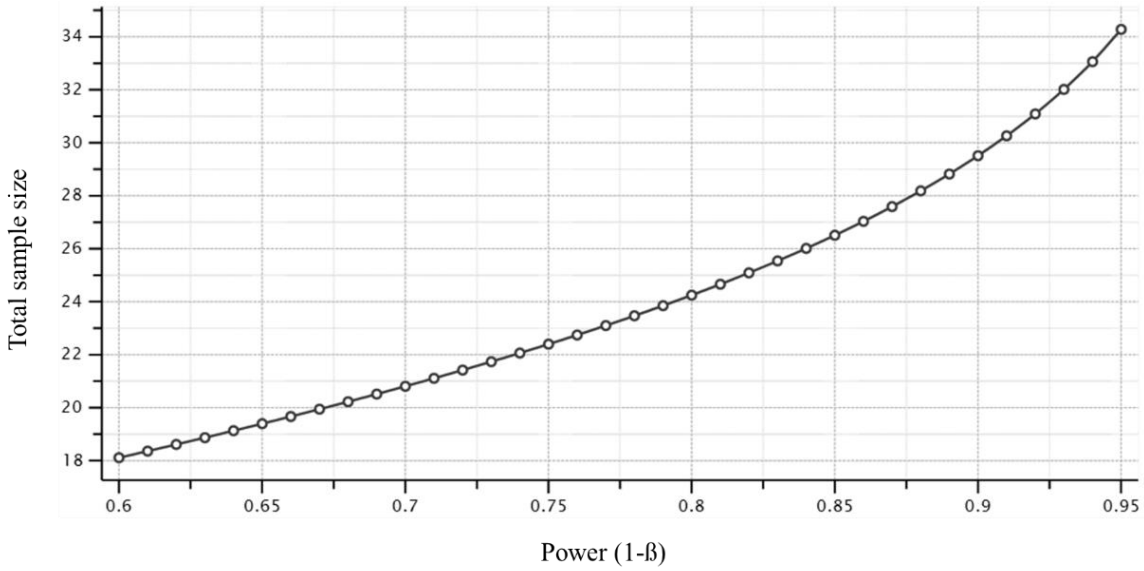

Figure A1. The relationship between power and the total sample size based on an a priori power analysis. 80% power is obtained with a total sample size of 24 participants

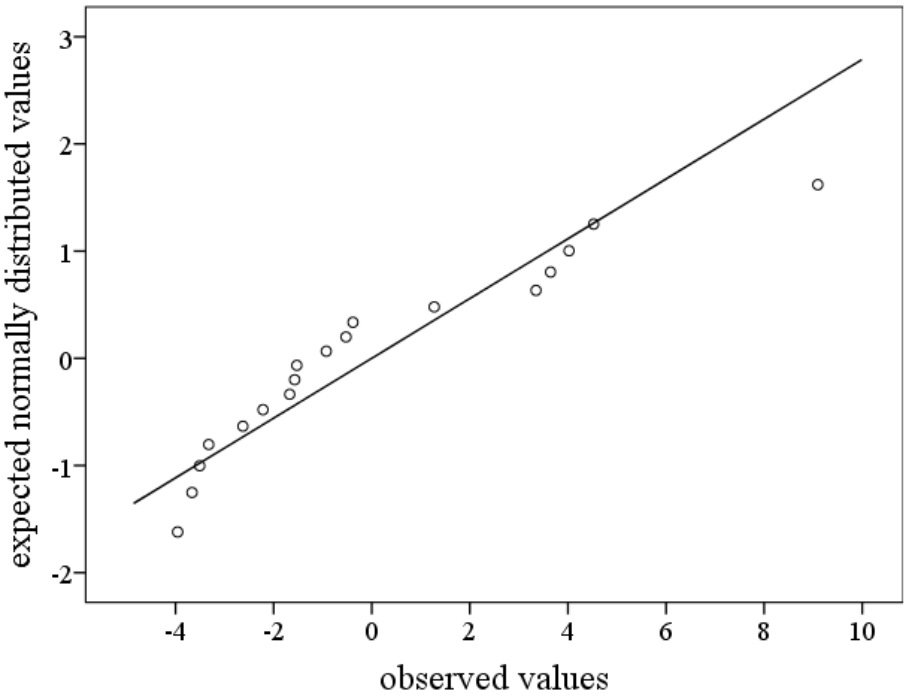

Figure A2. The Q-Q-Plot of the amplitude of P250 at the early adaptation phase at electrode position PO7

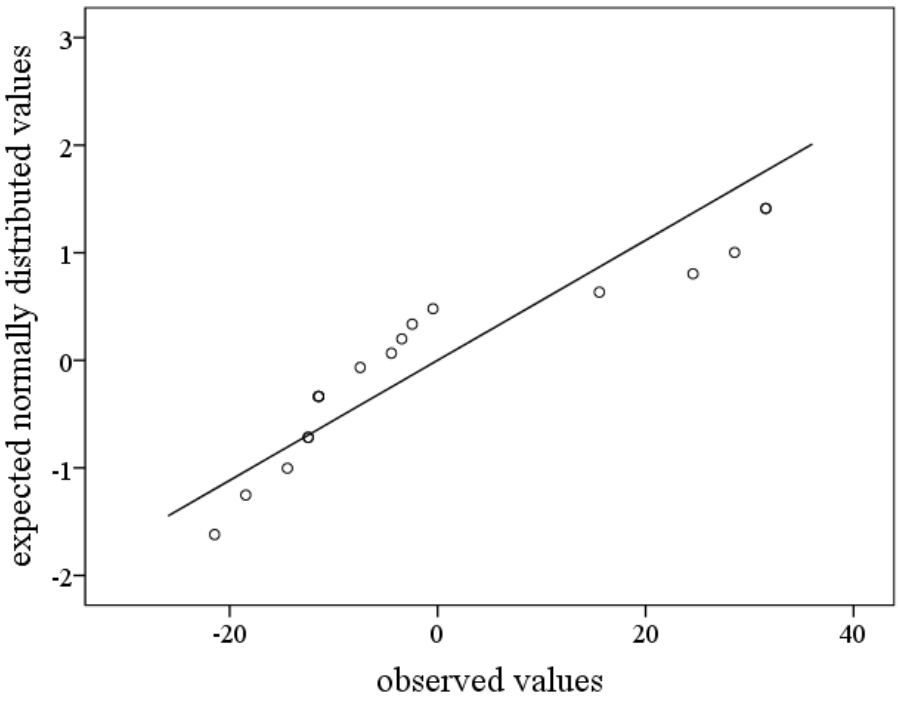

Figure A3. The Q-Q-Plot of the latency of P250 at the late adaptation phase at electrode position PO8

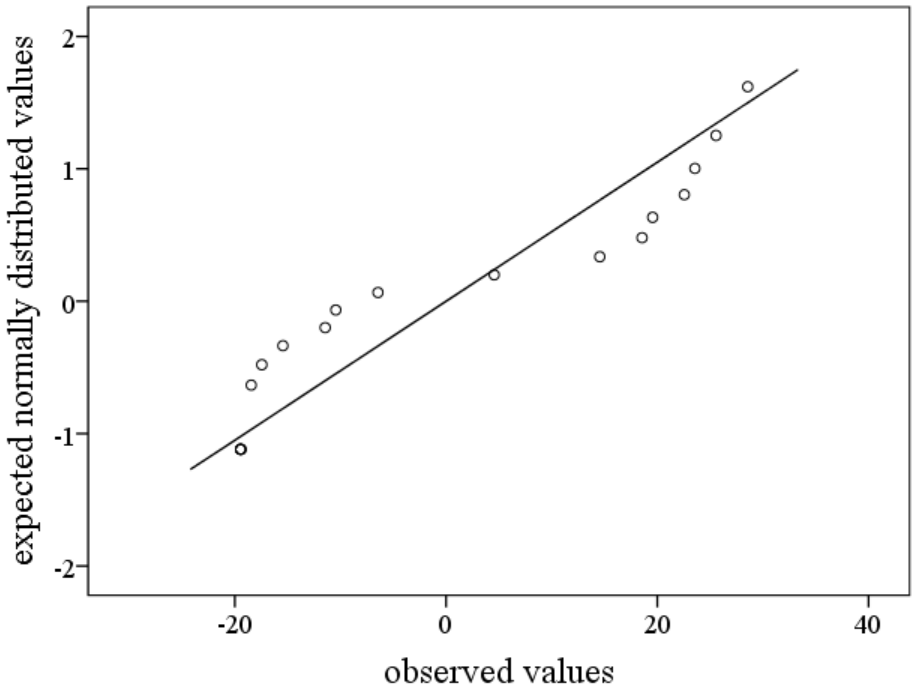

Figure A4. The Q-Q-Plot of the latency of N170 at the early adaptation phase at electrode position PO7

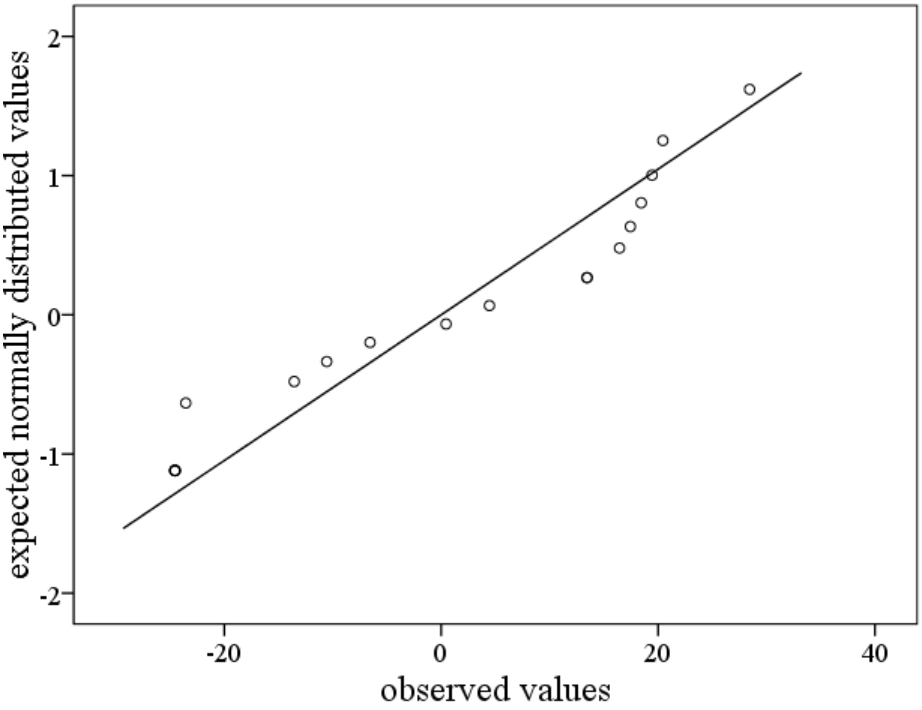

Figure A5. The Q-Q-Plot of the latency of N170 at the late adaptation phase at electrode position PO7