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Action language processing in Parkinson's disease: Characterization of neuro-oscillatory dynamics and linguistic performance

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List of Abbreviations

AoA	age of acquisition
BOLD	blood oxygen level dependent
CO	morphological complexity
DBS	deep brain stimulation
dB	decibel
DICS	dynamic imaging of coherent sources
ECG	electrocardiogram
EEG	electroencephalography
EPSP	excitatory postsynaptic potential
ERD	event-related desynchronization
ERP	event related potential
ERSP	event-related spectral perturbation
ERS	event-related synchronization
fMRI	functional magnetic resonance imaging
FR	word frequency
HC	healthy controls
Hz	Hertz
H	entropy
ICF	International Classification of Functioning, Disability and Health
IM	imageability
IPSP	inhibitory postsynaptic potential
L-Dopa	levodopa
LEDD	levodopa equivalent daily doses
LE	word length
LFP	local field potential
MCI	mild cognitive impairment
$\mathbf{MC_{high}}$	high motor content
$\mathbf{MC_{low}}$	low motor content
$\mathbf{MC_{pic}}$	motor content of pictures
$\mathbf{MC_{word}}$	motor content of verbs
MC	motor content
MEG	magnetoencephalography
MEP	motor evoked potential
μ**V**	microvolt
MoCA	Montreal Cognitive Assessment

MRI	magnetic resonance imaging
ms	milliseconds
NA	name agreement
$\mathbf{n_{response}}$	number of responses
OLD20	ortographic Levenshtein distance of the 20 nearest neighbors
$\mathbf{PD_{off}}$	Parkinson's disease patients off medication
$\mathbf{PD_{on}}$	Parkinson's disease patients on medication
PD	Parkinson's disease
rad	radians
REM	rapid eye movement
RE	reflexivity
RT	reaction time
s	seconds
SD	standard deviation
SEM	standard error of the mean
SNc	substantia nigra pars compacta
STN	subthalamic nucleus
TDCS	transcranial direct current stimulation
TMS	transcranial magnetic stimulation
TR	transitivity
UPDRS	unified Parkinson's disease rating scale
VC	visual complexity
WHO	World Health Organization

1 Overview

From a neuroscientific perspective, language processing depends on the temporally coordinated interplay of several brain areas, giving rise to dynamically evolving neural networks (Friederici & Singer, 2015). While the core language regions are located within the left peri-sylvian cortex (Friederici, 2011), it is increasingly acknowledged that further cerebral sites are also involved, especially in semantic processing (Binder et al., 2009; Huth et al., 2016). Critically, processing of action concepts seems to rely on the recruitment of frontal motor areas (Aziz-Zadeh & Damasio, 2008; Hauk et al., 2004; Tettamanti et al., 2005). Complementary to these findings, recent studies indicate that patients with conditions affecting motor control, such as Parkinson's disease, feature specific action language impairments (Bak, 2013; Birba et al., 2017; Cotelli et al., 2018). However, the mechanisms underlying this putative action language deficit are so far unresolved. It is unclear whether neural correlates of semantic access to action concepts are altered in Parkinson's disease. Therefore, the aim of this study was to characterize spatiotemporal neurophysiological patterns that accompany action language processing in patients with Parkinson's disease. To achieve this goal, two experiments were performed: Experiment 1 was a normative study in healthy participants to establish a data set of action pictures for the German language. In Experiment 2, this data set was used in an action naming task and combined with high-density electroencephalography to compare behavioral and oscillatory correlates of action language processing between patients with Parkinson's disease and healthy controls.

This study is structured as follows:

Chapter 2 provides an overview on the methodological foundations of electroencephalography and on oscillatory neural activity. Furthermore, an introduction to Parkinson's disease will be given and previous studies on action language processing will be reviewed. Finally, the research questions and hypotheses will be formulated.

In Chapter 3, the methodology and results of a picture naming normative study will be presented (Experiment 1). Psycholinguistic properties of 283 action pictures were characterized and their impact on naming latency was evaluated.

Chapter 4 presents an action naming study in patients with Parkinson's disease and healthy participants (Experiment 2). Time-frequency and source reconstruction analyses of high-density electroencephalographic recordings as well as behavioral modeling allowed for the characterization of neural correlates of action language processing in Parkinson's disease.

Finally, Chapter 5 discusses the results from Experiments 1 and 2 and places them in the context of relevant research literature.

2 Introduction

The time-resolved description of neuro-linguistic processes requires methods that are able to register neural activity on a time scale of milliseconds. By recording changes in the electric field produced by neuronal ensembles, electroencephalography (EEG) is especially suited to measure highly dynamic brain networks (Cohen, 2017). In the following Introduction, the foundations of EEG and neuronal oscillations will be reviewed first, as the latter constitute the phenomena under investigation in this study. Subsequently, the clinical and pathophysiological features of Parkinson's disease (PD) will be introduced. Clinically, the focus will be on non-motor symptoms, comprising linguistic abnormalities. Pathophysiologically, changes in oscillatory networks will be emphasized. Afterwards, a more detailed overview will be given on the status quo of action language research and on how PD has been associated with deficits in action language processing. A last introductory note will be directed towards the behavioral paradigm that is used in this study. Finally, the research questions and hypotheses will be formulated.

2.1 The EEG and neural oscillations

Neurophysiological methods have been pivotal in elucidating both the pathophysiology of PD as well as the neural mechanisms of language processing. One of these methods is EEG, a non-invasive technique to measure the brain's electric field (Cohen, 2017). Fluctuations in this field create distinct temporal, spatial and spectral patterns, which can be correlated to clinical information in order to subserve medical diagnosis (Schomer & Lopes da Silva, 2011). However, they also provide a means to decipher neural mechanisms that underlie human behavior and cognition (Lopes da Silva, 2013). In particular, the study of neural oscillations has become increasingly popular in both basic (Buzsáki, 2006; Buzsáki & Draguhn, 2004; Fries, 2015; Lisman & Jensen, 2013) and clinical neuroscience (Little & Brown, 2014; Simon & Wallace, 2016; Uhlhaas & Singer, 2010). In the following, the mechanisms by which the EEG is brought about will be briefly reviewed and an overview on the role of neural oscillations in physiological and pathophysiological conditions will be provided.

2.1.1 Generators of the brain's electric field

Changes in the electric field at scalp level reflect a superposition of fluctuations in microscopic local field potentials (LFPs) within a given brain volume (Nunez & Srinivasan, 2006). As a rule of thumb, a cortical area of about 6 cm^2 (enclosing around 60,000,000 neurons) must be synchronously active to produce a measurable scalp potential (Nunez & Srinivasan, 2006). In this volume, every event that gives rise to transmembrane currents modulates local electric fields (Buzsáki et al., 2012). Manifold of such mechanisms exist, which overlap in a complex spatial and temporal way. However, the main component contributing to local electric field changes is being attributed to synaptic activity (Buzsáki et al., 2012).

Synaptic activity

Whenever excitatory neurotransmitters activate postsynaptic ligand-gated ion channels, cations (mainly sodium) flow from the extracellular to the intracellular compartment producing a positive transmembrane current. This leads to a transient increase of the transmembrane potential called excitatory postsynaptic potential (EPSP) (Kandel et al., 2013). As a result, an excess of positive charge carriers emerges at the subsynaptic site, which in turn induces a so-called return current, i.e., an outward bound transmembrane flux of positive ions at a remote location from the EPSP (Buzsáki et al., 2012). Thereby, electroneutrality is being restored. Taken together, this configuration represents a dipole, i.e., spatially separated charges of opposing polarity: The extracellular site of synaptic activity reflects the sink (relative surplus of negative charge carriers) and the region of the outgoing return current reflects the source (excess of positive charges) (Jackson & Bolger, 2014) (Figure 2.1). Roughly the inverse happens with the release of inhibitory neurotransmitters, generating an inhibitory postsynaptic potential (IPSP) at the postsynaptic membrane and a dipole configuration that is mirror-inverted to the one produced by an EPSP (Jackson & Bolger, 2014). Depending on where EPSPs or IPSPs are being generated - near the soma or at a dendrite - both events can produce similar electric fields. Thus, at scalp level positive and negative EEG deflections can both emerge due to the collective emergence of EPSPs or IPSPs respectively (Jackson & Bolger, 2014).

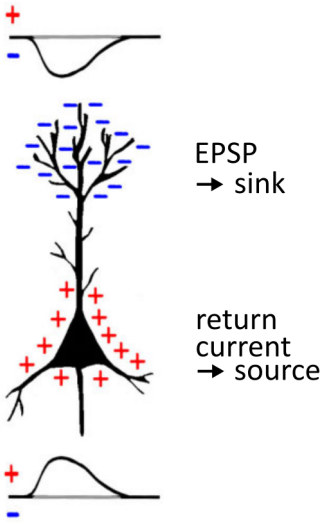

Figure 2.1: **The dipole model of a neuron.** At the dendrites, an EPSP induces an inward going positive current (i.e., sink), leading to an extracellular surplus of negative charge carriers (blue minuses). This results in an outward going positive return current at the soma (i.e., source), which manifests in an extracellular excess of positive charge carriers (red pluses). This way, charges of opposing polarity get spatially separated and form a dipole. The potential difference over the neuron can be recorded at either site by an electrode. Reused and adapted with permission from Jackson and Bolger (2014). © 2014 Society for Psychophysiological Research.

Non-synaptic activity

While synaptic activity plays the major role in generating extracellular electric fields, further mechanisms do also contribute. Calcium spikes triggered by presynaptic depolarization (Llinás et al., 1981) are both large and long enough to contribute significantly to local field fluctuations (Buzsáki et al., 2012). On the contrary, action potentials - albeit producing large transmembrane currents - are short-lasting events. Thus, they rarely temporally overlap in a given volume and as such their electric fields do not add up under most circumstances (Schomer & Lopes da Silva, 2011). Furthermore, very-low frequency local field changes may be caused by slow fluctuations in the membrane potential of glial cells (Buzsáki et al., 2012), extending the broad ensemble of possible generators to non-neuronal events.

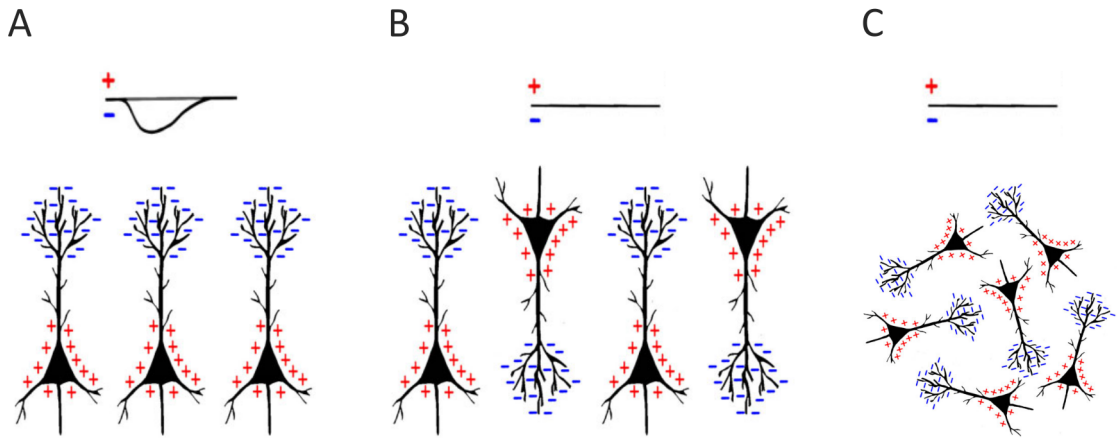

Figure 2.2: **Neuronal architecture prerequisites for EEG.** A) Parallel organization of dipoles gives rise to measurable potential differences. B) Parallel neural alignment but diverging dipole orientation leads to net cancellation of potential differences. C) Random orientation of neurons prevents individual potential differences from accumulating. Reused and adapted with permission from Jackson and Bolger (2014). © 2014 Society for Psychophysiological Research.

From microscopic to macroscopic potentials

The aforementioned mechanisms (alongside a variety of others) lead to changes in the electric field on a microscopic scale. By means of volume conduction, these potential changes ultimately reach the scalp, where they can be picked up by EEG electrodes. However, as mentioned above, a multitude of microscopic dipoles emerges simultaneously in a given volume. Thus, scalp EEG only represents compound activity of microscopic field changes, i.e., the sum of a large number of dipoles. Furthermore, two main conditions have to be met to detect macroscopic field changes at the scalp (Buzsáki et al., 2012). First, in a given area neurons need to be aligned in a parallel way and source-sink pairs, i.e., the effective dipoles, must orientate in the same direction (Jackson & Bolger, 2014) (Figure 2.2 A). Violations of these spatial patterns will lead to a net neutralization of source and sink currents (Figure 2.2 B & C). Second, for a given time window, fluctuations in field potentials have to be temporarily synchronized because accumulation is necessary to withstand the attenuating effects of volume conduction. As cortical pyramidal neurons are layered in the form of palisades, they form extended dipole sheets when receiving quasi-simultaneous input (Schomer & Lopes da Silva, 2011). Thus, the cerebral cortex is believed to be the main source of electric potentials recorded at scalp (Nunez & Srinivasan, 2006).

2.1.2 Oscillations in health and disease

Electric fields can fluctuate in a rhythmical manner, in other terms: They can oscillate (Cohen, 2014). Oscillatory components of EEG signals in humans have been described from the early days on, starting with the detection of the alpha rhythm by Hans Berger (1929). An oscillation is typically described by three properties (Cohen, 2014): First, amplitude is the distance between the maximum and minimum peak within a cycle and for EEG is measured in microvolt (μV). Power is amplitude squared and therefore expressed as μV^2. Second, the cycle width is defined as the duration of one cycle (i.e., until it repeats itself) expressed in seconds (s). It is inversely related to frequency, with the unit $\frac{1}{s}$ or Hertz (Hz). Finally, phase refers to the position along the cycle, denoted by radians (rad).

By subsuming oscillations of neighboring frequencies, a series of common rhythms has been classified: Among others, theta ranges from 4-7 Hz, alpha from 8-12 Hz, beta extends from 13-30 Hz and gamma summarizes frequencies above 30 Hz (Buzsáki, 2006). An exception to this frequency-centric classification is the mu rhythm, which shares the same frequency range with alpha but exhibits a different topographical distribution (Schomer & Lopes da Silva, 2011). Note that these frequency borders have evolved historically, are drawn somewhat arbitrarily and thus do not necessarily map to distinct functional entities. For example, the same underlying neurobiological mechanism can generate rhythms of different canonical frequency bands in different species (Buzsáki, 2006). Despite these limitations, it is still common practice to investigate neural oscillations under the framework of the aforementioned classical rhythms.

But how do these rhythms emerge as neurons communicate with each other? Three main mechanisms of rhythmogenesis follow from basic neural circuit theory (Wang, 2010). Mutual excitation of pyramidal neurons plays a role for slower rhythms below 10 Hz (Hansel et al., 1995), while networks of interneurons (Whittington et al., 1995) and reciprocal interactions between excitatory and inhibitory neurons (Sohal et al., 2009) are of importance for higher frequencies. However, these microscopic circuits are always embedded in large scale neuronal networks involving cortical and subcortical sites. Thus, compound neural activity, as picked up by EEG, is sculpted by both micro- and macroscopic neural circuit architecture (Wang, 2010).

Importantly, oscillations are not stationary. Their properties may be modulated by external or internal cues and they often do so in a characteristic way. A classical

example is the suppression of occipital alpha power after eye opening, which was first described by Berger (1929). One determinant of oscillatory power changes is the dynamically evolving temporal alignment of microscopic field fluctuations, i.e., the amount of synchrony of neuronal population activity (Cohen, 2014). Thus, a reduction in power relative to a reference period is regularly termed event-related desynchronization (ERD), whereas the opposite is called event-related synchronization (ERS) (Pfurtscheller & Lopes da Silva, 1999). In general, changes in oscillatory power associated with a specific event can be subsumed under the term event-related spectral perturbation (ERSP). Numerous behavioral phenomena, cognitive concepts and clinical conditions have been associated with specific ERSP patterns (Buzsáki, 2006). Ultimately, this study adds to this literature by characterizing ERSPs during action language processing in Parkinson's disease.

In the following, some of the canonical frequency bands that are relevant to this study will be introduced and the (patho)physiological phenomena they have been associated with will be outlined.

Alpha

Alpha oscillations (frequency range 8 to 12 Hz) are most abundant over occipital regions (Schomer & Lopes da Silva, 2011). Alpha power is higher in the resting wakeful state and decreases with external stimulation or increased cognitive demand (Schomer & Lopes da Silva, 2011). The physiological sources of alpha activity are still not well-defined. Whereas former data suggested a dominant role for thalamocortical networks (Lopes da Silva et al., 1973), more recent research emphasizes cortico-cortical circuits as the driving forces behind the alpha rhythm (Bollimunta et al., 2008). Functionally, alpha ERS has been associated with top-down inhibition of task-irrelevant cortical sites (Händel et al., 2010; Klimesch, 2012) by suppressing neuronal firing rate (Haegens et al., 2011). However, this inhibitory effect may be dependent on oscillatory phase, which has been shown to align with spiking activity and thereby possibly gating neuronal information transmission (Haegens et al., 2011; Lőrincz et al., 2009). Analogously, alpha band suppression was shown to reflect cortical disinhibition (Hanslmayr et al., 2005). In general, Klimesch (2012) proposed that the interplay between inhibition and temporal coordination promoted by alpha ERS and focal disinhibition (alpha ERD) subserves attention and automatic knowledge access.

Mu

There is terminological variety in the literature when it comes to the definition of mu rhythms (Hobson & Bishop, 2017). The consistent view is that mu oscillations comprise a frequency range from at least 8 to 12 Hz and concentrate over central electrodes (Cheyne, 2013). However, several authors consider mu rhythms as a combination of two distinct oscillatory components: A slower 8 to 12 Hz rhythm and a faster oscillation around 20 Hz (Hari, 2006; Schomer & Lopes da Silva, 2011). In the following and to avoid any confusion, the mu rhythm will be referred to as the slower component and ~20 Hz oscillations will be discussed in the context of beta as previously done (Cheyne, 2013).[1] Albeit sharing the same frequency band, the mu rhythm is distinguished from alpha by its topographic distribution and differences in reactivity. While resting state alpha is pronounced over the occipital cortex, mu oscillations are observed over the sensorimotor cortex, possibly originating from the post-central gyrus (Cheyne, 2013). The mu rhythm is most prominently modulated by motor control: It gets suppressed bilaterally right before movement onset and remains desynchronized throughout action execution (Hari, 2006; Schomer & Lopes da Silva, 2011; Stančák et al., 1997). Furthermore, it has been shown that mu is not only modulated by motor tasks but also by observing another person's actions (Avanzini et al., 2012; Muthukumaraswamy & Johnson, 2004), by imagining movements (Pfurtscheller et al., 2006; Yuan et al., 2010) and, critically, by action language processing (Moreno et al., 2013; van Elk et al., 2010). As these features are akin to the reactivity profile of the so-called mirror neuron system, mu suppression has been suggested to reflect mirror neuron activity on a macroscopic scale (N. A. Fox et al., 2016; Hari, 2006). However, this notion has also been criticized (Hobson & Bishop, 2016, 2017).

Beta

Oscillatory activity in the beta frequency range (13 to 30 Hz) can be recorded over frontal and central areas (where, as laid out above, it may be seen as part of the mu rhythm) (Schomer & Lopes da Silva, 2011). Likewise to the slower mu component, it is primarily associated with motor control and shows largely similar reactivity (Engel & Fries, 2010; Hari, 2006). However, slight discrepancies can be distinguished: In contrast to mu oscillations, beta activity shows a pronounced rebound after move-

[1] This terminology will be followed throughout the study.

ment execution (Pfurtscheller, 1981) and is somatotopically organized (Salmelin & Hari, 1994). Furthermore, beta likely originates from precentral motor areas instead of the post-central gyrus (Jurkiewicz et al., 2006; Salmelin & Hari, 1994). Importantly, analogous to mu oscillations, beta desynchronization has also been demonstrated during action observation (Hari et al., 1998) and action imagination (Yuan et al., 2010), with the beta ERD possibly scaling with the amount of movement executed or imagined (Avanzini et al., 2012; Yuan et al., 2010). Functionally, beta oscillations may act to actively sustain an ongoing motor state through cortical inhibition, hampering the execution of new motor commands (Pogosyan et al., 2009). Apart from motor control, increased beta activity has also been associated with top-down attentional control, reducing sensitivity to external stimuli (Buschman & Miller, 2007). Engel and Fries (2010) interpret both of these findings under the hypothesis of beta rhythms conserving the current neural state. This concept has received additional support by studies in patients with PD, who, among other symptoms, suffer from increased slowing of movements (bradykinesia). Depth recordings from PD patients undergoing deep brain stimulation (DBS) surgery indeed revealed elevated levels of beta activity (Brown, 2007), while levodopa (L-Dopa) intake suppressed beta oscillations as a function of clinical improvement (Kühn et al., 2006). Whether the association of elevated beta activity in PD with impaired motor performance (Neumann et al., 2016) may generalize to action language processing is being addressed in this study.

2.2 Parkinson's disease

As Parkinson's disease affects multiple brain regions and disrupts physiological information flow within various networks (Caligiore et al., 2016), neurophysiological methods like EEG have proven highly valuable in understanding the pathophysiology of PD. Having established the methodological foundations of EEG in the first section, PD will be introduced throughout the following section. PD is a chronic, incurable neurological condition (Berg, 2016). Being the second most common neurodegenerative disease after Alzheimer's disease, it is estimated to affect 2% of men and 1,4% of women over the course of their lifetime with increasing prevalence among the elderly (Ascherio & Schwarzschild, 2016). In the following, the clinical phenotype of PD will be introduced, emphasizing non-motor symptoms including linguistic abnormalities. After that, the current understanding on underlying etiology, pathogenesis and pathophysiology will be outlined. Finally, treatment options

for PD will be shortly discussed, focusing on the network effects of dopaminergic medication in the parkinsonian brain.

2.2.1 Clinical phenotype

Traditionally, PD has been labeled as a movement disorder. As such, core clinical symptoms are subsumed under the term parkinsonism, which is defined as the combination of bradykinesia (slowed and decreased movements) with rigidity (velocity-independent stiffness) and/or rest tremor (Postuma et al., 2015). While clinical diagnosis requires the presence of this syndrome (Postuma et al., 2015), it is increasingly acknowledged that PD additionally involves a variety of non-motor symptoms (Schapira et al., 2017). Such features can appear before onset of classical motor symptoms, giving rise to the term prodromal PD (Berg et al., 2015). Most notably, patients may suffer from neuropsychiatric symptoms such as depression or anxiety (Aarsland & Kramberger, 2015), rapid eye movement (REM) sleep behavior disorder (Postuma et al., 2012) or autonomic dysfunction (Adams-Carr et al., 2016) several years prior to formal diagnosis (Schrag et al., 2015). However, other non-motor symptoms primarily develop during late-stage disease, such as cognitive impairment (Aarsland et al., 2017). Importantly, non-motor symptoms worsen health-related quality of life to a greater extent than does movement deterioration (van Uem et al., 2016).

Building upon the World Health Organization (WHO) International Classification of Functioning, Disability and Health (ICF), problems in psychosocial functioning are most strongly associated with reduced quality of life of PD patients (van Uem et al., 2016). As preserved communication is a prerequisite for psychosocial interaction, it is therefore of utmost importance to address communication disorders in these patients (Miller, 2017). Such linguistic dysfunction is highly prevalent among patients with PD throughout the course of the disease (Miller et al., 2007) and is of multi-factorial origin (Miller, 2017). While reduced motor control may be responsible for hypophonic and dysarthric speech (Sapir, 2014), it can not account for the complete patholinguistic phenotype (Saldert & Bauer, 2017). Critically, disrupted semantic processing needs to be considered as well when addressing communication disorders in patients with PD (Miller, 2017; Saldert & Bauer, 2017). Previous research indicated impaired semantic access to action concepts in particular (Bak, 2013), a notion that will be laid out in more detail below.

2.2.2 Etiology, pathogenesis and pathophysiology

The majority of PD cases occur sporadically, while only 10% are of genetic cause (Ascherio & Schwarzschild, 2016). Risk factors for developing sporadic PD are diverse, with the most important determinant being age (Ascherio & Schwarzschild, 2016). However, exogenous factors like exposition to specific pesticides or increased dairy consumption may interplay with individual disposition as well, facilitating disease development (Ascherio & Schwarzschild, 2016). Both sporadic and genetic PD share core neuropathological features: Intraneuronal alpha-synuclein accumulation (within so-called Lewy bodies) and loss of dopaminergic cells in the substantia nigra pars compacta (SNc) (Rocha et al., 2018). It is assumed that misfolded and aggregating alpha-synuclein drives progressive neurodegeneration by maintaining a vicious circle of impaired proteostasis, mitochondrial dysfunction, increased oxidative stress and (mal)adaptive immune responses (Abeliovich & Gitler, 2016; Rocha et al., 2018). Over the course of the disease, Lewy body pathology can be found throughout successively more brain areas, giving rise to specific patterns of progression (Braak et al., 2003). Initially, aggregates are mainly restricted to the enteric nervous system and/or the olfactory bulb, but - putatively via prion-like transmission (Olanow & Brundin, 2013) - spread in a retrograde fashion to upstream areas, ultimately reaching the cerebral cortex (Braak et al., 2003). However, it has to be emphasized that both the pathogenic relevance of aggregated alpha-synuclein and its supposed cell-to-cell transmission are still heavily debated questions (Jellinger, 2009; Surmeier et al., 2017).

Motor symptoms begin to manifest after the loss of approximately 30% or more SNc neurons (H.-C. Cheng et al., 2010). Thereafter, reduced dopaminergic modulation of striatal activity leads to disruption of physiologic basal ganglia computational capacity (Yttri & Dudman, 2018). This is also reflected by distinct neurophysiological patterns in the beta band within the basal ganglia (Little & Brown, 2014): Elevated beta power in LFPs recorded from the subthalamic nucleus (STN) has been shown to correlate with parkinsonian symptom severity (Neumann et al., 2016; Steiner et al., 2017) and to accompany amplitude decrement during repetitive movements (Lofredi et al., 2019). However, as the basal ganglia are part of a network that also encompasses cortical sites and the thalamus (so-called cortico-basal ganglia-thalamic loops (Alexander et al., 1986)), widespread system-level abnormalities emerge as a consequence of subcortical degeneration (Caligiore et al., 2016; McGregor & Nelson,

2019; Ruppert et al., 2020).[2] Neurophysiologically, this has been demonstrated to manifest as increased (beta band) synchrony throughout several nodes of the cortico-basal ganglia-thalamic network (Litvak et al., 2011; Tinkhauser et al., 2018). For example, cortical beta band desynchronization is reduced for highly impaired patients during isometric hand contractions (Pollok et al., 2012). Increased oscillatory synchronization within this network has therefore been suggested to interfere with physiologic information conduction, ultimately leading to compromised behavioral performance (Brittain et al., 2014).

2.2.3 Therapy

Motor symptoms of PD are primarily treated pharmacologically, while non-pharmacological interventions complement therapy during various disease stages (S. H. Fox et al., 2018). No disease-modifying intervention is available yet, however, contemporary PD therapy provides various means aiming at symptom relief (S. H. Fox et al., 2018). Most pharmacotherapeutic options target the striatal dopaminergic deficit, e.g., through dopamine replacement via the dopamine precursor L-Dopa (LeWitt, 2015) or by means of dopamine receptor agonists (Torti et al., 2019). Among these and other agents, L-Dopa is regarded as a gold standard, due to it's clinical efficiacy and cost-effectiveness (de Bie et al., 2020; LeWitt, 2015). Functional neuroimaging studies have shown that L-Dopa intake restores system-level network changes towards physiologic states (Tahmasian et al., 2015). For example, Ballarini et al. (2018) revealed that increased connectivity throughout motor brain regions in PD patients off dopaminergic medication in comparison to healthy controls was reduced after L-Dopa intake. L-Dopa also affects neurophysiological markers of PD pathology. For instance, elevated beta power in the STN is suppressed after L-Dopa intake (Priori et al., 2004), which in turn correlates with symptom improvement (Kühn et al., 2006). The picture becomes less clear, though, when considering cortical oscillatory changes due to dopaminergic medication (Geraedts et al., 2018). Pre-movement mu and beta desynchronization over motor cortical regions is delayed in PD patients off medication in comparison to healthy controls and is reestablished after dopaminergic medication (Magnani et al., 2002). The amount of increase in desynchronization is thereby correlated with clinical improvement (Brown & Marsden, 1999). In contrast, resting state oscillatory beta power has also been shown to increase with L-Dopa intake, demonstrating a likewise positive association with

[2] During advanced disease stages, more widespread neurodegeneration including other cerebral regions may contribute to extensive network aberrations (Gratwicke et al., 2015)

clinical improvement (Cao et al., 2020). The latter study proposes that previous conflicting results could have emerged due to an interaction between disease duration and cortical beta power: While early-stage patients might exhibit increased cortical beta synchronization, the contrary might be the case for late-stage patients, which might show elevated beta power only after L-Dopa intake (Cao et al., 2020).

2.3 Grounded cognition

As laid out in the previous section, Parkinson's disease is not exclusively associated with impaired motor control. Its multi-faceted clinical phenotype includes cognitive symptoms as well. Intriguingly, PD has been associated with impaired semantic access to action concepts (Bak, 2013). This raises the question whether or not these cognitive symptoms are due to motor system degradation observed in PD and it directly leads to one of the most widely debated questions in cognitive neuroscience: Is semantic knowledge based on an amodal symbolic system, which operates irrespective of its contextual embedding (Fodor & Pylyshyn, 1988)?[3] Or, alternatively, does it depend on "perceptual, motor, and introspective states" which get reenacted spontaneously to form semantic representations (Barsalou, 2007, p. 618)? The latter view summarizes theories of grounded cognition and has gained increasing popularity over the last years in comparison to standard cognitive theories favoring strictly amodal processing (Barsalou, 2010). One of the main cognitive domains that have been investigated within the grounded cognition framework is language processing (Buccino et al., 2016). Empirical evidence indicates that understanding a words meaning is dependent on simulating the sensorimotor experiences to which this word refers (Galetzka, 2017). Much of this data has come from experiments investigating action language processing (Pulvermüller, 2005). In the following, studies which suggest action language grounding in the motor system will be reviewed first. After that, the implications this might have for patients with PD will be discussed.

[3]Throughout this study, the terms "semantic processing" and "conceptual preparation" are used as synonyms for the process of retrieving semantic concepts from memory. "Preparation" hereby indicates that this may subserve downstream steps of an overarching cognitive process, e.g., language production.

2.3.1 Action language grounding in motor networks

Behavioral Studies

Behavioral evidence in favor of embodied action language processing comes from a variety of research paradigms. One such approach has been to investigate the impact of concurrent motor execution on the access to action-related semantic concepts. Buccino et al. (2005) let participants listen to action sentences related to either the hand or the foot and instructed them to give semantic judgments about these sentences either with the hand or the foot. The authors found reaction time to be longer when sentential content matched the effector body part (e.g., hand-related sentences were to be met by hand responses). Complementary to this result, when stimulating the cortical motor area that somatotopically maps to the semantic content of a presented action sentence by means of transcranial magnetic stimulation (TMS), reduced motor evoked potentials (MEPs) were registered (Buccino et al., 2005). These findings were interpreted as to reflect interference between action language processing and motor control due to shared neural resources. In a similar vein, Gijssels et al. (2018) found that excitation of the premotor cortex via transcranial direct current stimulation (TDCS) led to impaired performance in a lexical decision task involving hand-related verbs. Furthermore, it has been shown that processing of hand action words negatively affected reaching kinematics when verbs were presented simultaneously with movement onset (Boulenger et al., 2006). However, while these (and other) findings suggest shared neuronal circuitry for action language and execution it has to be mentioned that most studies were lacking sufficient sample sizes and that adequately powered replications have come up with more mixed results (Gianelli & Dalla Volta, 2015).

Neuroimaging Studies

Besides behavioral evidence, many experiments utilizing functional neuroimaging methods have pointed to overlapping networks for motor control and action language processing as well (Aziz-Zadeh & Damasio, 2008; Courson & Tremblay, 2020; Pulvermüller, 2005; Pulvermüller & Fadiga, 2010). In a seminal study, Hauk et al. (2004) found that reading action verbs referring to specific body parts (e.g., 'lick', 'kick' or 'pick', which relate to the face, leg and arm respectively) elicited blood oxygen level dependent (BOLD) responses in the motor and premotor cortex com-

parable to those during execution of the corresponding physical movement. Similar findings have also been reproduced with participants listening to whole sentences containing action verbs (Tettamanti et al., 2005). Moreover, concurring activation patterns with action language processing have not only been revealed for movement execution but also for action observation (Aziz-Zadeh et al., 2006). More recently, a common neural network for action observation and action language has also been confirmed using advanced data analysis strategies exploiting machine learning methods (Horoufchin et al., 2018).

Neurophysiological Studies

Complementary to findings in neuroimaging studies, several neurophysiological investigations indicated a link between motor control and action language processing. Equivalently to Hauk et al. (2004), two studies combining high-density EEG with source reconstruction techniques localized event related potentials (ERPs) during processing of action verbs referring to different body parts in a somatotopic fashion around the motor cortex (Hauk & Pulvermüller, 2004; Pulvermüller et al., 2001). Similar results have been reported in respect to oscillatory responses, with desynchronization of beta and mu rhythms showing comparable somatotopic patterns (Niccolai et al., 2014). The involvement of mu and beta desynchronization in action language processing has been further investigated in a range of studies (Klepp et al., 2015; Klepp et al., 2019; Moreno et al., 2013; Moreno et al., 2015; van Elk et al., 2010; Vukovic & Shtyrov, 2014). In summary, these data suggest that mu and beta desynchronization during action language processing reflects patterns that have already been described for action execution and observation (see above). Importantly, in an action naming task, Cuellar and Del Toro (2017) could show that sensorimotor involvement, as indicated by mu desynchronization, occurs as early as 200 milliseconds (ms) after stimulus onset. This is in line with ERP data described above and strengthens the notion that motor cortex activation supports early lexico-semantic retrieval of action verbs instead of representing an epiphenomenonal process (Hauk et al., 2008).

Breakdown of Embodiment

As behavioral, neuroimaging and neurophysiological evidence suggests grounding of action language in motor networks, it can be hypothesized that pathological disruption of these circuits should lead to deficits in action language processing (García & Ibáñez, 2018). Indeed, data from clinical populations have shown that patients with aberrant motor networks display action language deficits: Examining patients with motor neuron disease, Bak et al. (2001) found a selective deterioration of verb over noun comprehension and production. While this case series however lacked substantial sample size, in a lesion mapping approach including 75 patients, Tranel et al. (2001) could link action naming deficits to lesions encompassing the left inferior frontal and premotor cortex. Subsequently, action language deficits could be demonstrated for a variety of movement disorders (Cotelli et al., 2018), most prominently PD. In the following, data characterizing action language deficits in PD will be presented in more detail.

2.3.2 Action language deficits in Parkinson's disease

Behavioral studies

First evidence linking PD to specific deficits in action language processing came from verb generation studies (Péran et al., 2003; Piatt et al., 1999). An early investigation by Piatt et al. (1999) demonstrated that PD patients with signs of dementia provided less verbs in an action fluency task than healthy controls. Additionally, in comparison to other verbal fluency tasks, action fluency deterioration was more pronounced. In non-demented PD patients, using a word generation task, Péran et al. (2003) showed that patients were performing less accurate than healthy controls when requested to provide verbs, while noun generation was preserved.

Several action naming studies have subsequently reproduced this disproportionate deficit in verb processing (Cotelli et al., 2018). Bertella et al. (2002) found that PD patients were providing less accurate responses than healthy controls during both action and object naming. Furthermore, they observed a dissociation between action and object naming to the disadvantage of the former. Later on, similar performance patterns have been reproduced by other authors (Cotelli et al., 2007; Rodríguez-Ferreiro et al., 2009; Salmazo-Silva et al., 2017).

However, as these studies have contrasted verbs to nouns, the question has been raised whether the observed effect is of grammatical or semantic origin. Fernandino et al. (2013) addressed this issue by comparing action and abstract verbs in a lexical decision task. Overall, PD patients' reaction time was slower than those of healthy controls, but while the latter showed faster processing times for action verbs than for abstract verbs, no such distinction could be found for patients. For Bak (2013, p. 677), these findings indicate that "it is the action rather than the verb component which determines the impairment." In keeping with this, extensive data on the neural representation of nouns and verbs suggest that semantic rather than grammatical differences are driving the neural discernability of these two word classes (Vigliocco et al., 2011).

Following this line of research, several action naming studies have investigated the effect of a verb's motor content (MC) on behavioral performance, thereby particularly focusing on semantic processing. MC has been defined as the amount of "movement [that is] needed in order to perform the action" a respective verb refers to (Herrera et al., 2012, p. 901). One study utilized stimuli with target verbs of varying MC and compared the naming accuracy of PD patients and healthy controls (Herrera et al., 2012). The authors could demonstrate that patients generally responded less accurate to stimuli than healthy controls. Importantly, a dissociation between high and low MC stimuli emerged with a disproportionate deficit for high MC target verbs. Similar results have been obtained by Bocanegra et al. (2015). In another study, Herrera and Cuetos (2012) investigated the effect of L-Dopa medication on action naming performance in PD patients while simultaneously manipulating the MC of stimuli. In the on medication state, no difference in reaction time and accuracy between high and low MC pictures could be found. In the off state, though, patients performed selectively worse when confronted to high MC stimuli.[4] These results have been interpreted as supporting the hypothesis that effective processing of action semantics relies on the integrity of the motor system (Birba et al., 2017).

[4]Note that Herrera et al. (2012) and Bocanegra et al. (2015) both assessed PD patients in their on medication state. The fact that they found an effect of MC while patients took dopaminergic medication seems to conflict with the results of Herrera and Cuetos (2012) who fail to do so. Reported clinical data (mean levodopa equivalent daily doses (LEDD) and unified Parkinson's disease rating scale (UPDRS)-III) however indicates that patients in Bocanegra et al. (2015) were more heavily affected than the participants evaluated by Herrera and Cuetos (2012), possibly leading to stronger motor system degradation and less potential functional restoration after L-Dopa intake. Herrera et al. (2012) do not provide clinical data.

Neuroimaging and neurophysiological studies

While a considerable amount of behavioral evidence has accumulated in support of an action language deficit in PD, far less data is available on underlying neural correlates from neuroimaging and neurophysiological studies. In a first functional magnetic resonance imaging (fMRI) study, Péran et al. (2009) found no difference in neural activation indicated by BOLD signal changes during object naming and action word generation in PD patients. Both tasks elicited an elevated BOLD signal in fronto-temporal and temporo-occipital areas, as well as in the supplementary motor area. However, action naming recruited a more widespread cortical network as a function of symptom severity (assessed by UPDRS-III), which the authors interpreted in terms of a compensatory mechanism. This is in line with another study which utilized functional connectivity measures to infer networks engaged during action naming in PD (Abrevaya et al., 2017). These authors found stronger recruitment of non-motor regions for patients in comparison to healthy controls. In a smaller follow-up study, Péran et al. (2013) showed that dopaminergic medication is accompanied with increased neural activity in the premotor cortex when contrasting action word generation and object naming. Considering the scarcity of data, neuroimaging studies were not able not clarify the neural underpinnings of action language deficits in PD (da Silva et al., 2014).

Neurophysiological correlates of action language processing in PD have been investigated even less. De Letter et al. (2012) examined source reconstructed ERPs in a verb reading task comparing PD patients on and off dopaminergic medication. They found generally higher current densities in the on medication state when considering the difference between action and non-action verbs in five pre-selected regions of interest, comprising motor and non-motor areas. However, due to small sample size (seven participants) and low-density EEG coverage (24 electrodes), these results have to be interpreted cautiously. While no study has so far assessed neuro-oscillatory patterns during action language processing in PD, Heida et al. (2014) investigated mu and beta ERSPs in PD patients during action observation. The authors could show absent mu and beta ERD in PD, raising the possibility that a similar pattern may emerge for action language processing.

Thus, the neural mechanisms associated with a putative action language deficit in patients with PD remain unclear. As such, it is unknown whether aberrant semantic access to action concepts in PD can be attributed to pathologically affected motor networks.

2.4 Probing action language production

The aforementioned studies used various psycholinguistic paradigms to behaviorally and neuroscientifically characterize action language processing. One frequently used approach is to let participants name visually depicted actions. As this task has also been used in this study, methodological considerations regarding the correct implementation of this paradigm will be briefly introduced.

This section has been published in a partly modified version in Busch et al. (2021).

2.4.1 Action naming

The action naming task can be subsumed under the more general framework of picture naming paradigms which already date back to the 19th century (Cattell, 1886). In these tasks, subjects are asked to name graphical stimuli with a single word. Oftentimes, they are additionally instructed to respond as fast as possible. Performance can be assessed by measuring naming latency (i.e., reaction time (RT)) or response accuracy if correct responses have been predetermined. Using the picture naming task, it is possible to examine which stimulus or response attributes affect language production (Perret & Bonin, 2019) and how these variables may interact with population characteristics under investigation (such as healthy individuals and patients). While there is substantial evidence on the behavioral patterns for naming objects (Glaser, 1992; Indefrey, 2011; Perret & Bonin, 2019), this is less the case for naming actions. However, as noun and verb processing may be subserved by distinct cognitive mechanisms (Vigliocco et al., 2011), there is considerable demand to characterize the factors specifically driving action naming performance. Furthermore, for factorial experiments, researchers need to know which parameters influence behavioral outcomes to rule out confounding variables by adequately matching sets of stimuli (van Casteren & Davis, 2007). So far, only few normative studies have undertaken such attempts, however, none of these were conducted for the German language (Cuetos & Alija, 2003; Khwaileh et al., 2018; Schwitter et al., 2004; Shao et al., 2014; Szekely et al., 2005). In the following, findings from these studies will be reviewed and additional variables that may affect naming performance will be introduced. These picture and verb characteristics were evaluated in Experiment 1 of this study and relate to the picture data set that was used in Experiment 2.

2.4.2 Variables affecting action naming performance

Mainly two picture characteristics have been evaluated in previous action naming normative studies. The variables that have been shown to influence naming performance most consistently have been summarized as name agreement indices by Szekely et al. (2005). This term comprises name agreement (NA), entropy (H) and the number of responses ($n_{response}$) to a picture and captures how uniformly responses are distributed among participants. Across all action naming normative studies, a strong negative correlation of naming latency with NA and likewise positive correlations with H and $n_{response}$ could be shown (Cuetos & Alija, 2003; Khwaileh et al., 2018; Schwitter et al., 2004; Shao et al., 2014; Szekely et al., 2005). On the contrary, the visual complexity (VC) of a picture - indicating difficulty to decode what is portrayed graphically - has been found to predict reaction time in only one study (Shao et al., 2014).

Besides picture characteristics, several response attributes have been assessed. A frequent finding is that the age of acquisition (AoA) of a word strongly correlates with naming latency, with words learned earlier in life being paralleled by faster reaction time (Cuetos & Alija, 2003; Schwitter et al., 2004; Shao et al., 2014; Szekely et al., 2005). Counter-intuitive results have been reported for word frequency (FR), i.e., a word's rate of occurrence within a language-specific vocabulary: One study showed that more frequent verbs were accompanied by slower reaction time (Szekely et al., 2005). Furthermore, high imageability (IM) of responses, meaning how easy verbs are able to evoke mental images, was found to predict lower naming latency in some studies (Cuetos & Alija, 2003; Khwaileh et al., 2018; Shao et al., 2014). Finally, word length (LE), while being uncomplicated to measure, has failed to predict reaction time in any previous normative study.

Besides these previously examined parameters, additional variables may affect naming performance based on evidence coming from other paradigms. As laid out above, the MC of verbs has been shown to influence reaction time or naming accuracy in clinical populations (Herrera & Cuetos, 2012; Herrera et al., 2012). However, systematic analyses within normative studies including healthy participants are lacking so far. Thus, the motor content of pictures (MC_{pic}) as well as the motor content of verbs (MC_{word}) was evaluated in this study. Additionally, data from lexical decision studies suggest that the neighborhood size of responses may be an important determinant of action naming performance as well (Yarkoni et al., 2008). Neighbors of words share most segments with each other (e.g., "house" and "mouse") (Marian et

al., 2012). Words with manifold orthographic neighbors were found to be associated with faster reaction time (Yarkoni et al., 2008). However, no data is available for action naming. Finally, grammatical properties of responses could also shape action naming outcomes: Transitivity (TR) relates to the number of arguments a verb can accommodate, reflexivity (RE) describes whether an action's agent and patient are identical and morphological complexity (CO) relates to the composite nature of verbs (e.g., verbs with prefix and stem). As behavioral data on these features is scarce (Kauschke & Stenneken, 2008; Kauschke & von Frankenberg, 2008), they were further assessed in this study.

2.5 Objective, research question and hypotheses

In conclusion, behavioral data indicate disrupted semantic access to action concepts in Parkinson's disease. Furthermore, data from healthy individuals suggest that action language processing and action execution may be associated with partly congruent neural correlates, possibly due to shared neural circuits in the sensorimotor cortex. This is specifically reflected in similar event-related modulations of central mu and beta oscillations. It is therefore hypothesized that the putative action language deficit in Parkinson's disease is reflected by aberrant modulation of mu and beta oscillations in the sensorimotor cortex.

The following formal research questions and hypotheses are therefore defined:

Experiment 1

Q1: Which psycholinguistic variables predict action naming reaction time?

H1: Name agreement indices and age of acquisition are expected to affect reaction time, while other picture and response characteristics may also explain variance in naming latency.

Experiment 2

Q2.1.1: Do PD patients off medication and healthy individuals show differing response accuracy and reaction time in an action naming task depending on the motor content of the stimuli?

H2.1.1: Patients with PD are expected to exhibit reduced response accuracy and longer reaction time than healthy controls for pictures with high motor content.

Q2.1.2: Do PD patients on and off medication show differing response accuracy and reaction time in an action naming task depending on the motor content of the stimuli?

H2.1.2: PD patients off medication are expected to exhibit reduced response accuracy and longer reaction time than patients on medication for pictures with high motor content.

Q2.2.1: Which spatiotemporal oscillatory patterns accompany action language processing in patients with PD off medication in comparison to healthy individuals?

H2.2.1: Action language processing is expected to elicit early desynchronization in the mu and beta frequency band for both patients and healthy controls. This desynchronization is expected to be weaker in patients with PD over the sensorimotor cortex for stimuli with high motor content.

Q2.2.2: Which spatiotemporal oscillatory patterns accompany action language processing in patients with PD in the on medication state in comparison to the off medication state?

H2.2.2: Action language processing is expected to elicit early desynchronization in the mu and beta frequency band for both medication states. This desynchronization is expected to be weaker in patients with PD off medication over the sensorimotor cortex for stimuli with high motor content.

To address these questions, two experiments were conducted: Experiment 1 was an action naming validation study in healthy participants. It aimed at establishing a normative data set of action pictures for the German language. Experiment 2 combined an action naming task (using the stimuli validated in Experiment 1) with EEG recordings to compare behavioral and oscillatory correlates of action language processing between patients with Parkinson's disease and healthy controls.

3 Experiment 1

This chapter has been published in a partly modified version in Busch et al. (2021).

Experiment 1 consisted of two parts, subsequently called Experiment 1.1 and Experiment 1.2. In Experiment 1.1, a timed action naming task was carried out using stimuli from two freely available picture databases (Bayram et al., 2017; Szekely et al., 2005). Within this part, responses, reaction times, name ageement indices and MC_{pic} values were collected. Experiment 1.2 was an online survey used to assemble MC_{word}, IM and AoA values, which included a subset of responses that were given in Experiment 1.1. Additional picture and verb characteristics that do not depend on individual ratings (e.g., VC) were assessed in both parts. Approval by the institutional ethics committee at the Medical Faculty of the University of Marburg was given for both experiments (study number 198/17).

3.1 Methods

3.1.1 Experiment 1.1

Participants

59 right-handed native speakers of the German language without any history of neurological or psychiatric conditions were recruited in this part of Experiment 1 (37 women, mean ± standard deviation (SD) age: 24.6 ± 3 years, formal education: 17.7 ± 2.2 years). All participants provided written informed consent.

Materials

A selection of 286 black-and-white drawings, derived from two sources (Bayram et al., 2017; Szekely et al., 2005) was compiled. The illustrations depicted action scenes, most of them involving human beings. Pictures were rescaled to a width of

500 pixels, keeping the aspect ratio of each picture fixed, and superpositioned on a white rectangle surrounded by a black background.

Procedure

Experiment 1.1 was performed in a dimly lit and soundproof laboratory. All stimuli were displayed at fixed distance on a VG28QE computer screen (Asus) using *Psychophysics Toolbox* (Brainard, 1997) for Matlab 2016b (The MathWorks). Responses were recorded using a SF-920 microphone (Elegiant) while MC_{pic} ratings were registered with a N001 numeric keypad (Jelly Comb). After several practice trials, stimuli were presented in randomized order (Figure 3.1). A total of six self-paced breaks were intercalated after blocks of 50 trials each. Participants were instructed to name the illustrated scene with a single verb. They were additionally asked to respond as fast as possible and to avoid hesitations, corrections or non-lexical utterances. A time-limit of 5 s was imposed after which responses were excluded. After this period, a motor content rating scale was displayed underneath the stimulus alongside a request to quantify the amount of movement required for the depicted action. No deadline was enforced upon this task, however, participants were asked to respond quickly. After providing a MC_{pic} value via button press, the next trial was triggered with a jittered inter-stimulus interval of three to four seconds. Participants required about 55 minutes to complete the task.

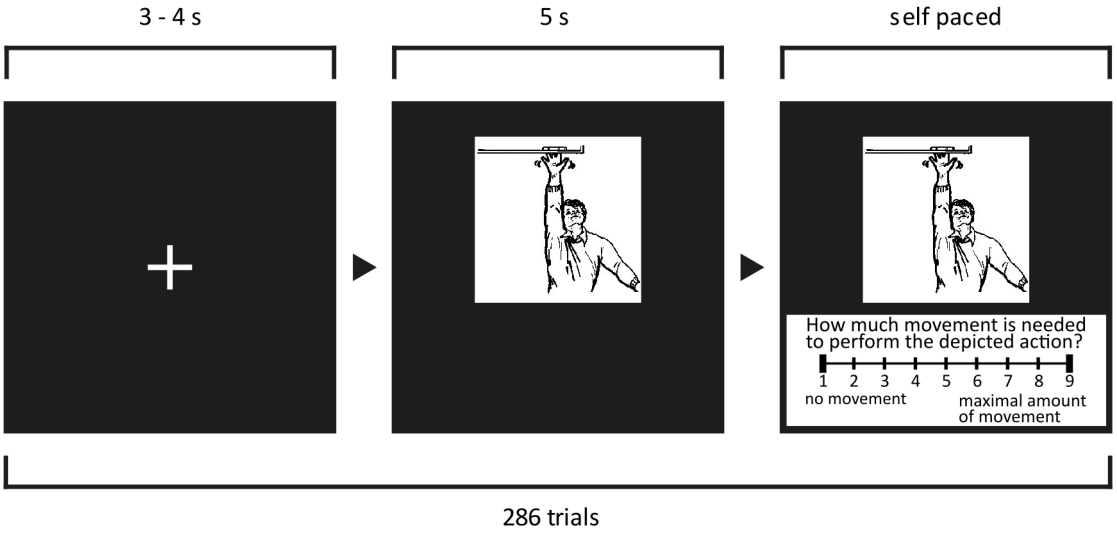

Figure 3.1: **Paradigm description.** Sequence of a single trial repeated over the course of the experiment. Reused and adapted from Busch et al. (2021). Licensed under CC-BY 4.0.

Trial preprocessing

Responses were transcribed manually while considering pictorial context. Thus, in the case of homophones, the verb was chosen that most likely matched the depicted scene. Trials which fulfilled one of the following conditions were error-flagged:

- Missing, non-comprehensible, multi-word or late answer (given after the time limit of 5 s).
- Response onset not determinable.
- Response not being listed in the online dictionary DUDEN (www.duden.de) as of August 2018.

Outcome variables

Naming latency RT was determined for all valid trials semi-manually using *CheckVocal* (Protopapas, 2007).

Name agreement indices The distribution of responses to each picture was characterized by three (related) variables: $n_{response}$, NA and H. $n_{response}$ was calculated on a purely lexical basis: If verbs differed in their word form, they were counted as distinct even if they were semantically comparable. NA was calculated by dividing the number of times the most frequent response per picture was given by the total number of participants providing a valid answer. Finally, H of responses was computed following Shannon and Weaver (1998):

$$H = \sum_{i=1}^{n_{response}} p_i \log_2 p_i \tag{3.1}$$

With p_i being the response frequency of the i^{th} answer. Thus, high H indicates more uniformly distributed responses.

Motor content of the picture A nine-point rating scale was used to quantify MC_{pic} (1: No movement, 9: Maximal amount of movement). The rating prompt

in German translated to: “How much movement is needed to perform the depicted action?”

Additional variables

Visual complexity Objective VC was computed on the basis of jpeg file size as suggested by Székely and Bates (2000). To account for differing stimulus dimensions, each picture was first superimposed onto a fixed-size black rectangle the dimensions of which equaled the size of the largest picture.

3.1.2 Experiment 1.2

Participants

150 native German speakers without any history of neurological or psychiatric conditions took part in Experiment 1.2 (103 women, mean ± SD age: 24 ± 4.1 years, formal education: 16.6 ± 2.4 years). All participants provided written informed consent.

Materials

A set of 600 verbs was assembled. The verbs comprised a selection of responses derived from Experiment 1.1 as well as additional frequent German verbs. A total of 1044 unique responses were provided in Experiment 1.1, rendering a full evaluation of every answer hardly feasible. Thus, a thresholding approach was implemented to extract those verbs that accounted for the majority of trials per picture: For each stimulus, the minimum number of verbs that jointly represented the responses of more than half of the participants was chosen. For example, in the case of stimulus X with response 1 being given by 40%, response 2 by 35% and response 3 by 25% of participants, responses 1 and 2 were selected for Experiment 1.2. Due to transcription errors in Experiment 1.1, three pictures had to be excluded, because valid response data were missing for these stimuli. Taken together, 299 verbs were

chosen from Experiment 1 by this approach. The remaining 301 verbs were compiled from the SUBTLEX-DE database (Brysbaert et al., 2011).

Procedure

An online survey was conducted to gather ratings for MC_{word}, IM and AoA using *SoSci Survey* (Leiner, 2014). Participants were randomly split into three groups of 50 subjects, each evaluating one variable of interest. After instructing participants to provide ratings as fast and intuitively as possible, verbs were presented in randomized order on a total of 10 pages with each page containing 60 words. Rating scales were displayed besides each word for participants to respond in a self-paced manner.

Outcome variables

Motor content of the word A nine-point rating scale was used to assess MC_{word}, analogous to MC_{pic} ratings (1: No movement, 9: Maximal amount of movement) and following Bayram et al. (2017).

Imageability IM, that is how easily a word is able to evoke a mental image, was evaluated according to Paivio et al. (1968) utilizing a seven-point rating scale (1: Low imageability, 7: High imageability).

Age of Acquisition Participants were asked to estimate the age (in years) at which they had understood the respective verb's meaning for the first time. In line with Birchenough et al. (2017), participants could provide a single year estimate instead of time periods.

Additional variables

Word frequency, orthographic neighborhood and word length The frequency of each verb was derived from the SUBTLEX-DE database (Brysbaert et al., 2011).

In order to counter FR overestimation of verbs that could also be interpreted as nouns when capitalized, the lower-case word form was used exclusively. To quantify the orthographic neighborhood of each verb, the ortographic Levenshtein distance of the 20 nearest neighbors (OLD20) was calculated according to Yarkoni et al. (2008), using the SUBTLEX-DE database (Brysbaert et al., 2011) as corpus. Lastly, LE was computed as number of letters. In contrast to all other variables, that were gathered in Experiment 1.2, FR, OLD20 and LE were also computed for every other response from Experiment 1.1.

Transitivity, reflexivity and morphological complexity Two experienced linguists classified each verb into binary categories for each variable respectively: Intransitive vs. transitive and ditransitive, partly reflexive and non-reflexive vs. reflexive, non-complex vs. complex.

Quality check

As participants provided ratings in an unsupervised environment, special care had to be taken to ensure that the survey was completed with due diligence. Thus, in a first step, participants who filled out less than 70 % of the survey or took more than 24 hours for completion were excluded. In a second step, fraudulent cases were identified by detecting unusual response patterns (high difference from median entropy and high proportion of answers exceeding one median absolute deviation from the median value) or completion times (high difference from median time to completion) by a multivariate outlier detection approach using the Minimum Covariance Determinant (Hubert & Debruyne, 2010). This was performed for each group separately.

3.1.3 Data analysis

A two-step procedure was employed for analyzing the data gathered in Experiments 1.1 and 1.2. First, a stimulus database with according normative data was established to be used in Experiment 2. Second, trial-based statistical analyses were carried out to investigate associations among all assessed outcome variables and to elucidate which picture and verb characteristics uniquely explained variance in nam-

ing latency. All data were analyzed using custom-made code for R (R Core Team, 2020).

Data preprocessing

For all further analyses, trials marked as errors (4.9% of all trials) and those containing responses given only by a single participant per picture (7.4%) were exlcuded. In sum, 87.7% of all trials were therefore considered for statistical analyses and establishment of normative data. 91% of these trials were fully characterized in Experiment 1.2 (79.8% of all trials). For statistical analyses, some variables were transformed in order to obtain normally distributed values. RT, $n_{response}$, VC, AoA, FR and OLD20 were logarithmized and IM was exponentiated based on visual assessment. Multivatiate outlier trials were then detected and exlcuded using the Minimum Covariance Determinant (2% of fully characterized trials) (Hubert & Debruyne, 2010). Finally, variables were z-transformed and both transitivity (TR) and CO were coded with dummy variables (0 = intransitive/non-complex, 1 = transitive and ditransitive/complex). RE was not incorporated in statistical analyses as 99.4% of trials fell into the pre-defined category of non-reflexive and partly reflexive verbs.

Normative data

To generate a normative dataset to be used in Experiment 2, variables assessed in Experiments 1.1 and 1.2 were averaged over participants for each picture. After that, descriptive statistics of each variable were computed over pictures.

Repeated measures correlation

As a first step of statistical analysis, associations among naming latency and picture as well as verb characteristics were assessed. To this end, repeated measures correlation coefficients were computed using the *rmcorr* package (Bakdash & Marusich, 2017). This method takes the non-independence of repeated measures within each participant into account and estimates the common association between two variables among participants (Bakdash & Marusich, 2017). Basic assumptions of

linearity, homoscedasticity, and normal distribution of errors were tested using the Rainbow test, the Breusch-Pagan test, and the Kolmogorov-Smirnov test, respectively. Overall, no severe violations of these assumptions were detected. P-values were corrected for multiple comparisons by means of the Benjamini-Hochberg procedure (Benjamini & Hochberg, 1995). Weak ($R \geq 0.1$), moderate ($R \geq 0.3$) and strong ($R \geq 0.5$) correlations were reported following Bakdash and Marusich (2017).

Linear mixed effects model

To evaluate which picture and verb characteristics uniquely explained variance in RT (research question Q1), linear mixed effects modeling was employed as a second step. For this purpose, the *lmer* function from the *lme4* package was used (Bates et al., 2015). As fixed effects H, MC_{pic}, VC, AoA, IM, MC_{word}, FR, LE, OLD20, CO and TR were included. A maximal random effects structure as dictated by study design was established (Barr et al., 2013): By-participant random intercepts were incorporated as well as random slopes for H, MC_{pic}, VC, AoA, IM, MC_{word}, FR, LE, OLD20, CO and TR. Likewise, by-picture random intercepts were included as well as random slopes for MC_{pic}, AoA, IM, MC_{word}, FR, LE, OLD20, CO and TR. By-picture random slopes for H and VC were not modelled as these variables did not show within-picture variation. As name agreement indices were highly intercorrelated, only H was included. The full model was specified as follows:

$$\begin{aligned} \text{RT} \sim & 1 + \text{H} + \text{MC}_{\text{pic}} + \text{VC} + \text{AoA} + \text{IM} + \text{MC}_{\text{word}} + \text{FR} + \text{LE} + \text{OLD20} + \text{CO} + \text{TR} + \\ & (1 + \text{H} + \text{MC}_{\text{pic}} + \text{VC} + \text{AoA} + \text{IM} + \text{MC}_{\text{word}} + \text{FR} + \text{LE} + \text{OLD20} + \text{CO} + \text{TR} \mid \textit{Participant}) + \\ & (1 + \text{MC}_{\text{pic}} + \text{AoA} + \text{IM} + \text{MC}_{\text{word}} + \text{FR} + \text{LE} + \text{OLD20} + \text{CO} + \text{TR} \mid \textit{Picture}) \end{aligned} \tag{3.2}$$

Multicollinearity of predictors was tested by computing the variance inflation factor, which was < 2 for all variables and thus indicated that multicollinearity was not a concern. The model was iteratively estimated by the restricted maximum likelihood procedure and p-values were computed by t-tests using Satterthwaite's approximation for degrees of freedom. Assumptions of normal distribution of residuals and homoscedasticity were checked visually using the *performance* package (Lüdecke et al., 2020). No severe violations of these assumptions were detected.

3.2 Results

3.2.1 Included datasets

In Experiment 1.1, three participants were excluded due to technical problems during data acquisition and one participant due to naming latency exceeding two standard deviations above the mean. Thus, data of 55 participants was analyzed. In Experiment 1.2, twenty cases were excluded due to missing data, exceedingly long time to completion or unusual response patterns (see above). Therefore, 130 data sets were confirmed eligible for statistical analysis, i.e., 44 data sets for imageability, 41 for motor content and 45 for age of acquisition.

3.2.2 Normative data

The distribution of normative data for each variable across pictures is illustrated in Figure 3.2. Descriptive statistics of the normative data are reported in Supplementary Table S1.

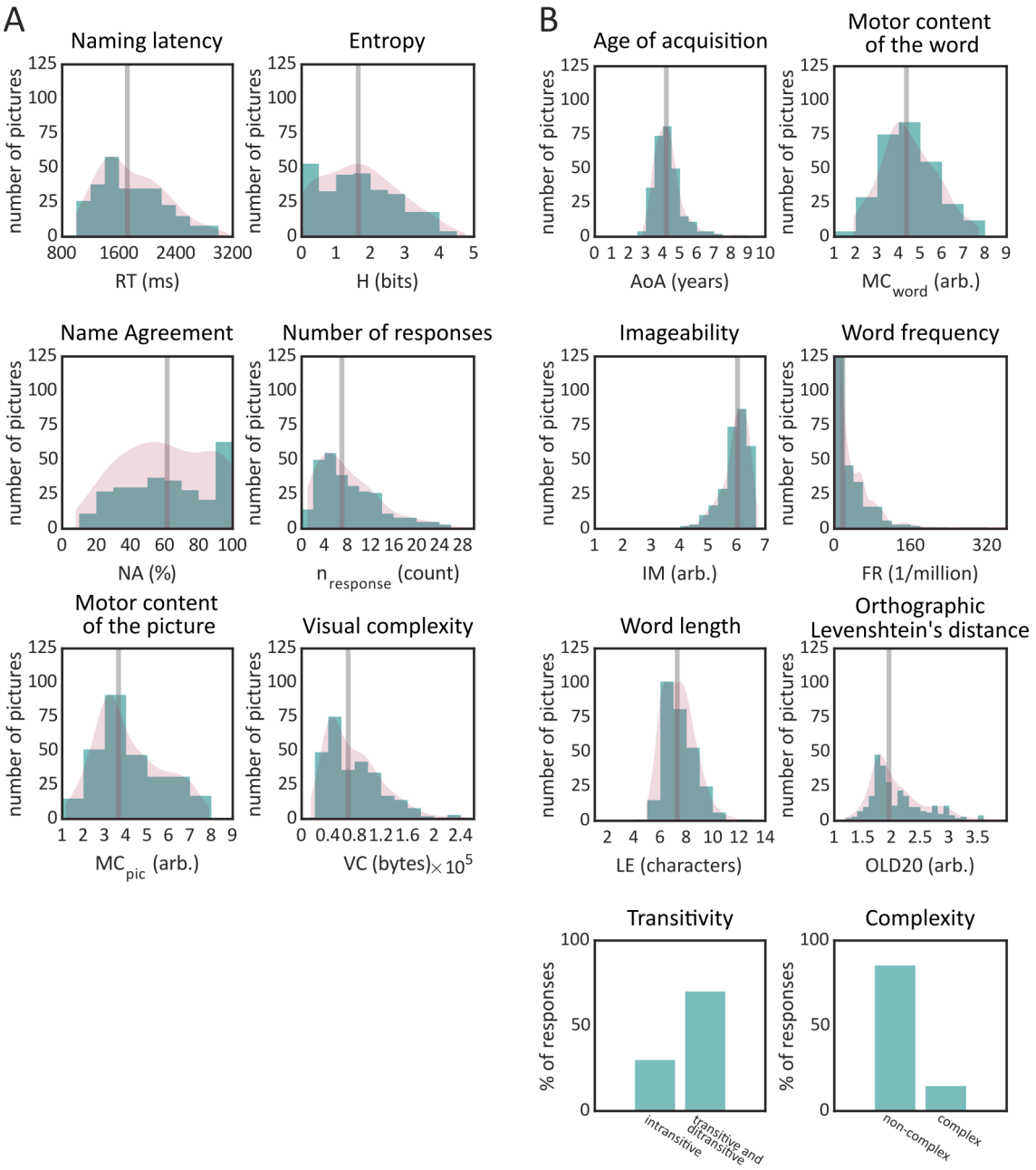

Figure 3.2: **Distribution of variables.** Distribution of A) naming latency and picture characteristics and B) verb characteristics. Grey vertical lines indicate median values while red areas represent estimated probability densities. Reused and adapted from Busch et al. (2021). Licensed under CC-BY 4.0.

3.2.3 Repeated measures correlation

All results from the repeated measures correlation analysis are summarized in Figure 3.3 and Supplementary Table S2.

Correlations of naming latency with picture and verb characteristics naming latency strongly increased as a function of H and $n_{response}$ and decreased with NA (Figure 3.3 A). Additionally, naming latency weakly increased with higher VC and weakly decreased with increasing IM. No other correlations fulfilled the criteria of at least weak correlation ($R \geq 0.1$).

Correlations between verb characteristics A strong positive correlation between LE and CO and a likewise negative association between FR and AoA was found (Figure 3.3 B). OLD20 correlated positively and moderately with AoA, LE, and CO. Additionally, a moderate positive association between MC_{word} and IM and a moderate decrease of OLD20 with higher FR was detected. Furthermore, higher AoA was accompanied by a weak increase in CO and LE, while word frequency weakly decreased with CO and LE. A small negative correlation could be observed between IM and all remaining word characteristics except FR. No other correlations fulfilled the criteria of at least weak correlation ($R \geq 0.1$).

Correlations between verb and picture characteristics MC_{pic} and MC_{word} showed a strong positive association(Figure 3.3 C). Name agreement indices mainly correlated with IM and CO: Moderate increases in IM for higher NA, lower H and lower $n_{response}$ were observed. Similarly, CO was found to weakly decrease with NA and increase with H and $n_{response}$. Furthermore, positive, but only weak correlations between $n_{response}$ and TR and between MC_{pic} and IM were detected. Finally, VC decreased weakly with higher MC_{word}, IM and AoA. No other correlations fulfilled the criteria of at least weak correlation ($R \geq 0.1$).

Correlations between picture characteristics Name agreement indices all strongly correlated with each other: A negative association between NA on one side and $n_{response}$ and H on the other side was observed, while conversely $n_{response}$ and H correlated positively (Figure 3.3 D). Furthermore, VC correlated with all name agreement indices: It weakly decreased with NA, and moderately increased with H and $n_{response}$. No other correlations fulfilled the criteria of at least weak correlation ($R \geq 0.1$).

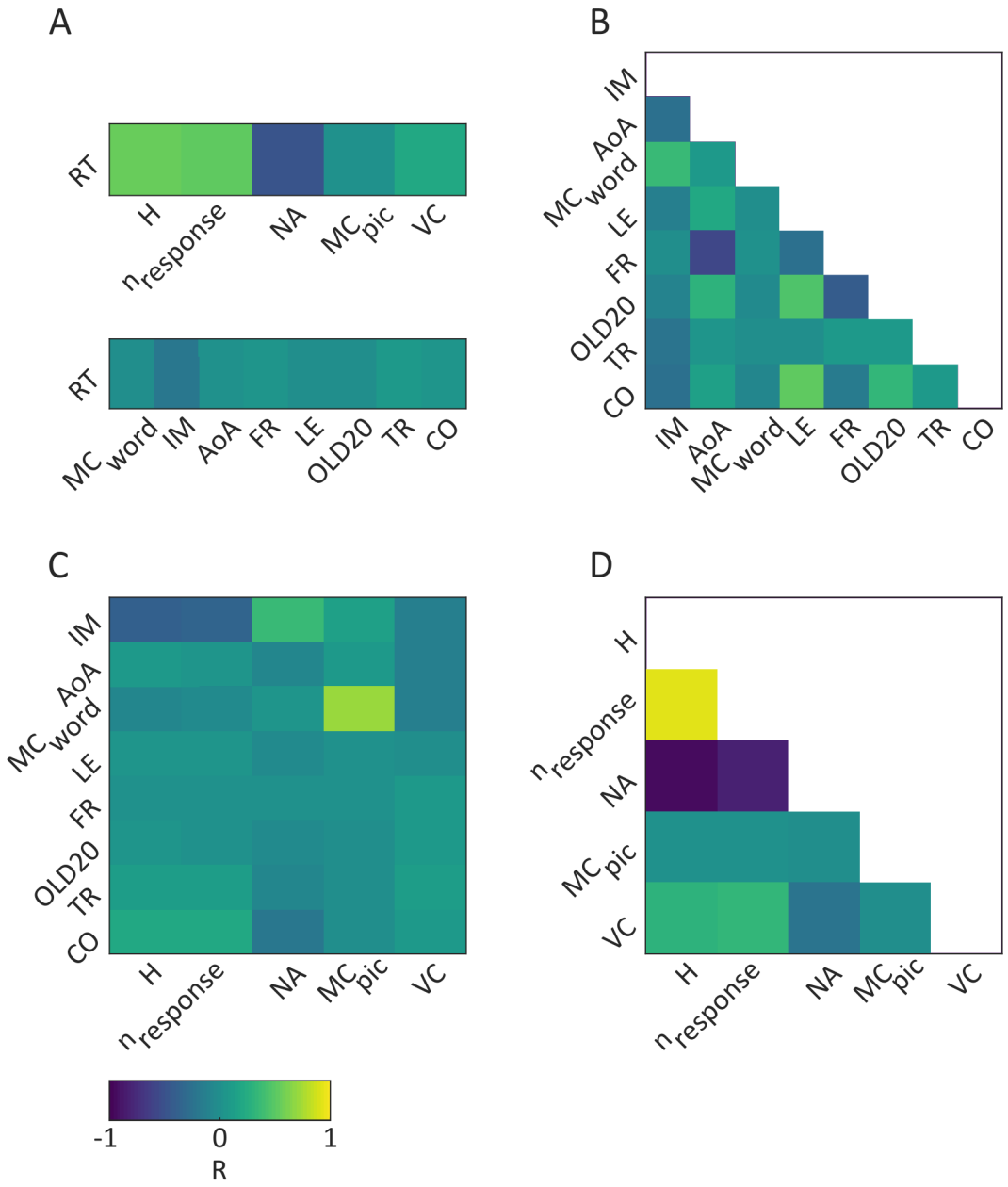

Figure 3.3: **Correlation among variables.** Correlational analyses between A) naming latency and picture and verb characteristics, B) verb characteristics, C) picture and verb characteristics and D) picture characteristics. R = repeated measures correlation coefficient. Reused and adapted from Busch et al. (2021). Licensed under CC-BY 4.0.

3.2.4 Linear mixed effects model

The linear mixed effects model predicting naming latency revealed independent contributions of H, FR, MC_{word} and MC_{pic} (Table 3.1). Specifically, higher H, FR and MC_{word} predicted slower RT, whereas higher MC_{pic} predicted faster RT. All other picture or verb characteristics did not independently predict naming latency. The combination of fixed and random effects explained 58 % of variance in naming latency (R^2[conditional] = 0.58), while fixed effects alone accounted for 25 % (R^2[marginal] = 0.25). Note that zero-estimates for MCpic and CO (0) random effects indicate a singular fit of the model. In an additional model, these terms were therefore excluded to compute a non-singular fitted model. However, the results of this model were practically identical to the reported model (data not shown). This is in line with Brauer and Curtin (2018), who state that singular model fits do not necessarily affect fixed effects estimates.

Fixed effects					
Variable	Estimate	SE	df	t	p
(Intercept)	0.01	0.07	77.1	0.1	0.92
H	0.49	0.02	240.3	20.1	< 0.001
MC_{pic}	-0.04	0.01	202.5	-3.3	0.001
VC	0.02	0.02	232.6	0.9	0.35
AoA	-0.02	0.02	190.2	-0.7	0.48
IM	-0.03	0.02	237.0	-1.5	0.14
MC_{word}	0.05	0.02	170.0	2.6	0.01
FR	0.07	0.02	196.4	3.1	0.002
OLD20	-0.001	0.02	154.8	-0.1	0.94
LE	-0.02	0.02	193.5	-1.2	0.22
CO (1)	-0.002	0.05	156.2	0	0.96
TR (1)	0.04	0.04	163.1	1.0	0.30
Random effects					
Group	Variable	Variance	SD		
Picture	(Intercept)	0.016	0.128		
	MC_{pic}	0	0		
	AoA	0.014	0.118		
	IM	0.012	0.111		
	MC_{word}	0.006	0.079		
	FR	0.011	0.105		
	OLD20	0.009	0.095		
	LE	0.008	0.089		
	CO (0)	0.050	0.224		
	CO (1)	0.021	0.144		
	TR (0)	0.008	0.090		
	TR (1)	0.018	0.136		
Participant	(Intercept)	0.17	0.41		
	H	0.008	0.092		
	MC_{pic}	0.001	0.031		
	VC	0.0003	0.017		
	AoA	0.003	0.057		
	IM	0.001	0.024		
	MC_{word}	0.0003	0.016		
	FR	0.002	0.047		
	OLD20	0.001	0.033		
	LE	0.0003	0.016		
	CO (0)	0	0		
	CO (1)	0.012	0.108		
	TR (0)	0.211	0.459		
	TR (1)	0.193	0.440		
Residual		0.439	0.663		
Model fit					
R^2(marginal)		0.25			
R^2(conditional)		0.58			

Table 3.1: Results of the linear mixed effects model.

4 Experiment 2

4.1 Methods

4.1.1 Participants

19 right-handed patients with PD were recruited from the movement disorders outpatient clinic at the Department of Neurology at the University Hospital Marburg (3 women, mean ± SD age: 61.1 ± 10.5 years, formal education: 16.3 ± 4.4 years). Clinical diagnosis was established by a movement disorders specialist. Included patients did not report any history of other neurological or psychiatric conditions, concomitant intake of neurotropic medication or impaired eyesight or hearing disability. Screening for mild cognitive impairment (MCI) was performed using the German version of the Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005) with patients scoring less than 24 points being excluded (Thomann et al., 2020). PD patients were assessed in both the on (PD_{on}) and off medication state (PD_{off}) over two separate experimental sessions, mostly taking part on different days. For off medication sessions, L-Dopa was withdrawn for a minimum of 12 hours, while long-acting dopamine receptor agonists were last taken 72 hours before the experiment started. Session order was counterbalanced among patients. Prior to each session, motor symptoms were assessed by means of the UPDRS-III (Goetz et al., 2008). Full demographic and clinical details are reported in Table 4.1. A cohort of 20 right-handed healthy participants served as controls (3 women, mean ± SD age: 61 ± 7 years, formal education: 15.9 ± 4 years). The two groups were matched for gender, age and formal education. Subjects were recruited via public notice and received financial compensation. They were free of any neurological or psychiatric condition, took no neurotropic medication and did report normal eyesight and sense of hearing. In contrast to patients, healthy subjects only performed one experimental session. Full demographic details of the control group are outlined in Table 4.2. All patients and healthy controls provided written informed consent. The experiment was approved by the institutional ethics committee at the Medical Faculty of the University of Marburg (study number 198/17).

ID	Gender	Age (years)	Education (years)	Disease duration (years)	LEDD (mg)	Symptom dominant side	MoCA	UPDRS On	UPDRS Off
1	male	77	10	11	1725	left	27	29	49
2	male	62	15	1	675	left	27	8	10
3	male	49	21	4	300	left	29	21	28
4	male	70	24	3	308	right	29	22	33
5	male	57	17	4	375	left	29	29	34
6	male	78	18	4	600	right	30	26	34
7	male	50	18	2	450	right	29	8	14
8	male	55	15	6	1170	right	27	52	61
9	male	50	12	8	180	right	28	6	7
10	male	63	13	8	1024	left	30	10	23
11	male	52	17	1	250	left	29	5	6
12	male	57	15	7	450	right	30	39	59
13	male	55	19	5	600	right	30	14	19
14	male	65	18	5	800	right	29	12	17
15	male	67	11	4	562	right	28	17	24
16	male	44	21	4	540	left	28	24	38
17	female	69	24	8	400	left	25	58	68
18	female	80	9.5	5	450	left	25	12	20
19	female	61	12	13	800	left	30	17	41
mean (SD)		61.1 (10.5)	16.3 (4.4)	5.4 (3.2)	614 (372)		28.4 (1.6)	21.5 (14.9)	30.8 (18.4)
ratio	m:f 5.3:1					l:r 1.1:1			

Table 4.1: Demographic and clinical details of patients. LEDDs have been determined following Tomlinson et al. (2010).

ID	Gender	Age (years)	Education (years)
1	male	56	12
2	female	60	13
3	male	64	13
4	male	59	12
5	male	50	22
6	female	69	16
7	female	67	20
8	male	51	12
9	male	66	18
10	male	66	23
11	male	61	12
12	male	49	22
13	male	56	19
14	male	71	13
15	male	58	21
16	male	68	13
17	male	72	14
18	male	65	17
19	male	57	13
20	male	54	13
mean (SD)		61 (7)	15.9 (4)
ratio	m:f 5.7:1		

Table 4.2: Demographic details of healthy controls.

4.1.2 Materials

A collection of 228 stimuli was assembled from the picture database validated in Experiment 1. To assess the putative semantic effect of motor content, the stimuli were classified into two groups of low (MC_{low}) and high (MC_{high}) motor content based on MC_{pic} normative data. The stimuli were then subdivided into a pair of sets to avoid learning effects in patients as they participated in two experimental sessions. Thus, each session consisted of 114 trials. H, $n_{response}$, NA, VC, IM, AoA, FR, OLD20, LE, TR, RE and CO were matched within and between sets using the program *Match* (van Casteren & Davis, 2007). Wilcoxon signed-rank tests confirmed

	Set 1		Set 2	
	MC_{low}	MC_{high}	MC_{low}	MC_{high}
MC_{pic}	2.9	5.3	2.8	5.3
H	1.7	1.7	1.8	1.7
$n_{response}$	8.6	7.8	8.9	7.9
NA	62.4	61.5	61.7	60
VC	82398	79980	74222	84780
IM	5.9	6	5.8	6
AoA	4.3	4.3	4.2	4.4
FR	40.8	33.3	42.5	52.7
OLD20	2.1	2.1	2.1	2.2
LE	7.2	7.5	7.5	7.4
TR	0.76	0.67	0.68	0.76
RE	0.02	0.01	0.01	0
CO	0.14	0.13	0.18	0.15

Table 4.3: Average picture and verb characteristics of the two stimulus sets used in Experiment 2.

that no significant differences were observed (all $p \geq 0.05$). Conversely, MC_{pic} was significantly different within sets (both $p < 0.05$) but not between sets (both $p \geq 0.05$). Full characteristics of the experimental stimuli are reported in Table 4.3. Set order was randomized for patients. For control subjects, one of the two sets was randomly assigned beforehand. All pictures were visually displayed as has been described in Experiment 1, however no motor content rating scale was presented. Post-hoc, for each set a subset of pictures was selected that only comprised pictures with NA greater than 80 %, while matching for all other variables was conducted as laid out above (52 stimuli in total). Characteristics of these stimuli are reported in Supplementary Table S3.

4.1.3 Procedure

Experiment 2 was conducted in the same dimly lit and soundproof laboratory that had been used in Experiment 1. All stimuli were presented on a VG28QE computer screen (Asus) at fixed distance using *Psychophysics Toolbox* (Brainard, 1997) for Matlab 2016b (The MathWorks). A SF-920 microphone (Elegiant) was used to acquire responses. The experiment started with three practice trials that did not

enter statistical analysis. Over each session, a total of 120 pictures were presented in randomized order, six of which were unrelated to this study. Before each trial, a fixation cross was displayed at the center of the screen. After 30 stimuli each, a self-paced pause block was introduced. Participants were asked to name the action that was depicted on each stimulus, providing responses as fast as possible. Only single-word answers given in the infinitive form were allowed. They were also instructed to avoid any utterances unrelated to the response. After five seconds, the stimulus was blanked out and incoming responses were discarded. Subsequently, a jittered inter-stimulus-interval of four to five seconds commenced, after which the next trial started automatically. Throughout each session, a 128-channel EEG was recorded as laid out below. Total duration of the task summed up to about 20 minutes.

4.1.4 Behavioral data preprocessing

Reaction time was calculated semi-manually using *CheckVocal* (Protopapas, 2007). Before entering linear mixed effects modeling they were log-transformed and z-transformed. Response transcription and error-labeling was conducted as described in Experiment 1. Trials marked as errors (mean ± SD 11.9 % ± 8.6 % per session) were excluded. Accuracy of responses was determined picture-wise by checking each answer against those responses that were given by at least two participants in Experiment 1. For reaction time analyses, trials labeled as inaccurate (14.2 % ± 4.7 % per session) were discarded as well. Thus, 88.1 % ± 8.6 % of trials per session qualified for statistical analysis of response accuracy and 74 % ± 10 % of trials per session were included for reaction time analyses.

4.1.5 Behavioral data analysis

In the following, *subject type* refers to either Parkinson's disease patients off medication (PD_{off}), Parkinson's disease patients on medication (PD_{on}) or healthy controls (HC) while *MC* encompasses MC_{high} and MC_{low}. Based on this, *condition* indicates a combination of subject type and MC (e.g., PD_{on} patients confronted to MC_{high} stimuli).

Behavioral data were analyzed using custom-made code for R (R Core Team, 2020). First, descriptive statistics were computed for each condition separately. Second,

linear mixed effects modeling was used to analyze variance in accuracy and naming latencies across subjects. Eight models were established, addressing research questions Q2.1.1 and Q2.1.2, respectively. The first model assessed differences in accuracy between PD patients off medication and healthy controls (disease state) as well as between stimuli with $\mathrm{MC}_{\mathrm{high}}$ and $\mathrm{MC}_{\mathrm{low}}$. Additionally, the interaction between disease state and MC was estimated. These parameters were therefore included as fixed effects. Furthermore by-participant and by-picture random intercepts were incorporated. The first model was specified as follows:

$$\begin{aligned}\text{Accuracy} \sim & 1 + \text{MC} + \text{disease state} + \text{MC} : \text{disease state} + \\ & (1 \mid \mathit{Participant}) + (1 \mid \mathit{Picture})\end{aligned} \tag{4.1}$$

The second model aimed at evaluating variance in accuracy due to medication state and MC. A possible interaction between medication state and MC was considered as well. The full specification of the second model was as follows:

$$\begin{aligned}\text{Accuracy} \sim & 1 + \text{MC} + \text{medication state} + \text{MC} : \text{medication state} + \\ & (1 \mid \mathit{Participant}) + (1 \mid \mathit{Picture})\end{aligned} \tag{4.2}$$

Models three and four evaluated the variance in naming latencies. Both models were specified analogous to the latter two models:

$$\begin{aligned}\text{RT} \sim & 1 + \text{MC} + \text{disease state} + \text{MC} : \text{disease state} + \\ & (1 \mid \mathit{Participant}) + (1 \mid \mathit{Picture})\end{aligned} \tag{4.3}$$

$$\begin{aligned}\text{RT} \sim & 1 + \text{MC} + \text{medication state} + \text{MC} : \text{medication state} + \\ & (1 + \mid \mathit{Participant}) + (1 \mid \mathit{Picture})\end{aligned} \tag{4.4}$$

Models five to eight mirrored models one to four but only included the subset of stimuli with high name agreement (NA $>$ 80 %). All models were estimated with the restricted maximum likelihood procedure using the *glmer* (for accuracy) and *lmer* function (for naming latencies) from the *lme4* package (Bates et al., 2015), respectively. With *glmer*, p-values were computed via Wald z-tests, while with *lmer*, p-values were established by t-tests using Satterthwaite's approximation for degrees

of freedom. A visual check for assumptions of normal distribution of residuals and homoscedasticity using the *performance* package (Lüdecke et al., 2020) revealed no severe violations for all models.

4.1.6 EEG recordings

Scalp EEG was recorded from 128 active Ag/AgCl electrodes at a sampling rate of 2500 Hz with a resolution of 0.1 μV using four BrainAmp amplifiers (BrainProducts). Signals were passed through an analogue high-pass filter of 0.1 Hz and a low-pass filter of 1000 Hz. Electrode montage followed a standardized layout for high-density recordings (Oostenveld & Praamstra, 2001) (Figure 4.1). The reference electrode was located at FCz, while ground was at FPz. Electrode impedances were ensured to be less than 5 kiloohm prior to starting the experiment. Additionally, three electrodes attached to both ankles and to the left chest were used to record an electrocardiogram (ECG). All data were processed and stored using BrainVision recorder software (BrainProducts).

4.1.7 EEG data preprocessing

EEG data were preprocessed using custom-made code based on the *Fieldtrip* toolbox (Oostenveld et al., 2010) for Matlab 2020b (The Mathworks). Initially, the continuous EEG data of each session was segmented trial-wise into epochs of eight seconds. The point of stimulus onset was set to 0 seconds with a 3 s pre-stimulus and a 5 s post-stimulus interval. Each trial was then demeaned and detrended. To cancel out line-noise corruption, a notch-filter was applied at 50 Hz, 100 Hz and 150 Hz. After that, the data was high-pass filtered with a 4th order non-causal butterworth filter at a cut-off frequency of 0.5 Hz. To reduce computation time for subsequent analyses, all EEG datasets were downsampled to 1000 Hz. This was followed by a first manual trial-rejection step based on excessive signal variance. After that, an independent component analysis was run using the infomax algorithm (Bell & Sejnowski, 1995), preceded by a decomposition into 128 principal components. With this method, components representing blink artifacts, movement artifacts and other characteristic technical artifacts were identified. For ECG artifact removal, the unmixing matrix that was generated by the independent component analysis was applied on EEG data segmented around automatically identified QRS complexes

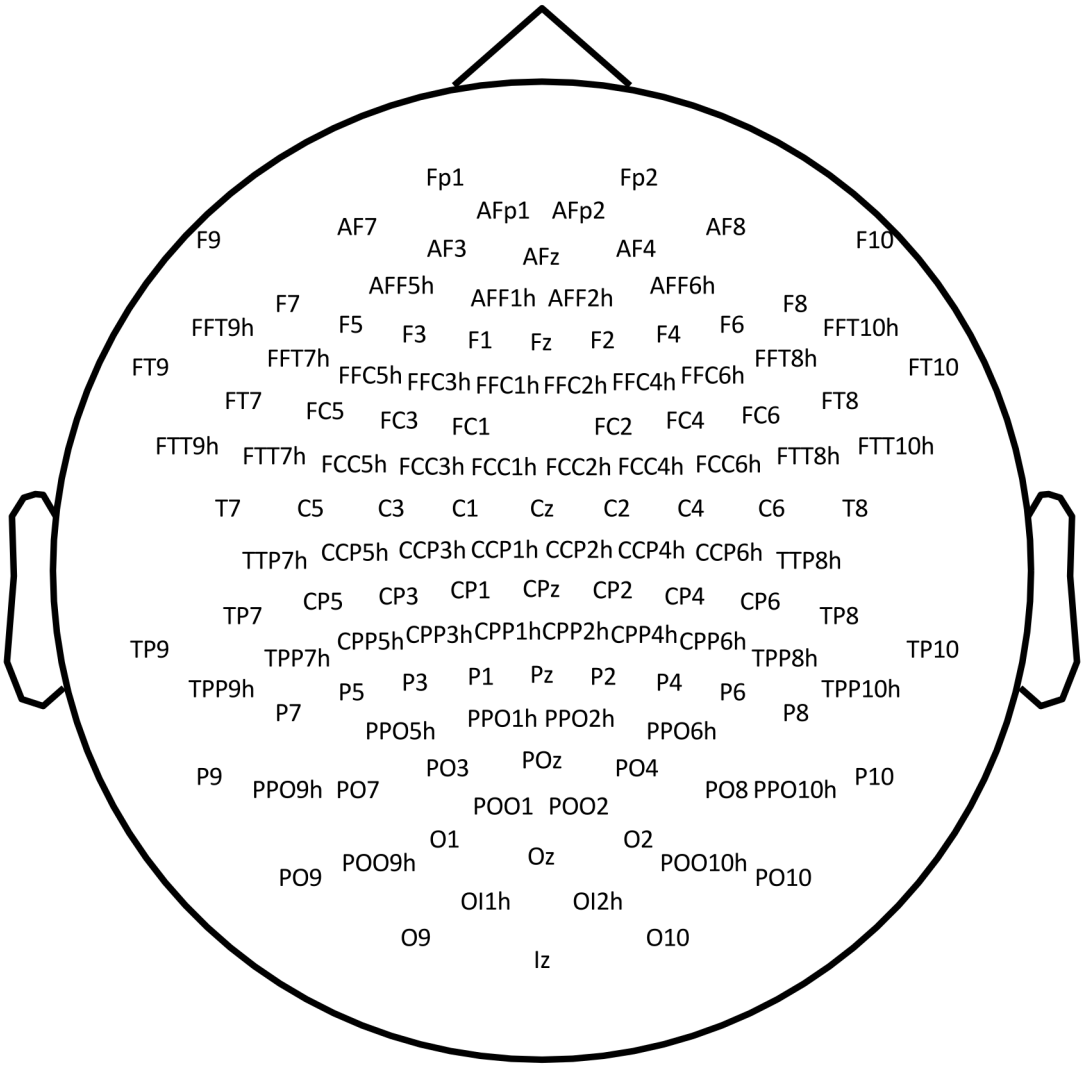

Figure 4.1: **EEG layout**. Representational scalp distribution of the 128 EEG channel montage.

in the ECG channel. By averaging over these segments, components representing ECG artifacts were identified. The remaining components were backprojected onto the channel level. This way, mean ± SD 6.8 ± 2.6 components were rejected per session in total. Subsequently, channels with low signal quality were identified visually and interpolated with data from neighboring channels.[1] This resulted in 2 ± 2.4 channels being discarded for each session. A second manual trial-rejection step was followed by visually checking each trial and excluding trials which featured clear artifacts. Both trial rejection steps taken together, 15.2 ± 11.1 trials were excluded per session. After that, trials labeled as errors or as inaccurate were rejected (23.2 ± 10.5). In sum, 38.4 ± 12.4 trials per session were discarded based on signal quality

[1]Neighbors were defined by triangulation of subject-specific electrode coordinates generated with a Fastrak digitizer (Polhemus), or (in case of missing subject-specific data) template coordinates.

and behavioral outcome. Finally, all EEG channels were rereferenced to a common average reference. For subsequent analyses, EEG data of each session were split into MC_{high} and MC_{low} trials. Thus, for each condition 37.8 ± 6.4 trials were included for statistical analysis on average. As was done with the analysis of behavioral data, all computations were furthermore carried out on a subset of stimuli with high NA. Here, on average 9 ± 2.1 trials were available for each condition. The EEG dataset from the on medication session of patient 19 had to be discarded as a whole due to technical failure.

4.1.8 EEG data analysis

The analysis pipeline was implemented in Matlab 2020b (The Mathworks) using the *Fieldtrip* toolbox (Oostenveld et al., 2010).

Time-frequency transformation

To investigate induced oscillatory dynamics in the form of ERD and ERS, time-domain EEG data had to be transformed to the time-frequency domain. To this end, a discrete Morlet-wavelet transformation was applied trial-wise over frequencies from 8 Hz to 30 Hz in steps of 1 Hz. The number of cycles per wavelet was 4 and each wavelet was centered around successive time points separated by 10 ms intervals. With this procedure, time-resolved and frequency-specific power and cross-spectral density estimates were generated for each channel per trial (time-frequency-channel-trial quadruplets). The latter were only used for source reconstruction (see below). The time-frequency transformed data were then partitioned into a baseline period (-0.7 s to -0.2 s relative to stimulus onset) and an activation period (0 s to 1.8 s relative to stimulus onset). Baseline data were averaged over time and subsequently expanded to the same duration as the activation period (i.e., 1.8 s). For the subset of stimuli with high NA, the activation period was defined from 0 s to 1.4 s. In each case, these periods were chosen as to reflect average naming latencies. For both baseline and activation periods, time-frequency transformed data were then averaged over trials within each data set (leading to time-frequency-channel triplets).

Baseline correction and frequency selection

Absolute power can be influenced by a variety of individual factors such as electrode impedance or head anatomy (Cohen, 2014). Furthermore, absolute EEG power is frequency-dependent showing a characteristic $\frac{1}{f}$ decline. To account for these task-unrelated aspects, only power changes relative to a neutral baseline (i.e., ERSPs) were being compared between subjects. For this purpose, the power values of the activation period were normalized to the respective baseline period by computing the sample-wise change in decibel (dB) for each dataset. Note that for the statistical comparison between activation and baseline period (see below), no baseline normalization was performed. To increase statistical power, the time-frequency transformed data were furthermore averaged over the the mu band (8 to 12 Hz) and over the beta frequency range (13 to 30 Hz), resulting in separate time-channel doublets (Pernet et al., 2020).

Statistics

Statistical analyses of the time-frequency transformed EEG data were based on non-parametric cluster-based permutation tests (Maris & Oostenveld, 2007). Evaluating statistical differences in multidimensional EEG data gives rise to a multiple comparisons problem due to the high number of data points. Cluster-based permutation tests address this problem by 1) computing a single summary statistic per contrast which is sensitive to the clustered structure of EEG data and 2) comparing the observed statistic against a surrogate distribution which is obtained by repeatedly drawing random partitions and calculating the summary statistic for each one of these. Thereby, the method tests the null hypothesis of exchangeability of conditions (Maris & Oostenveld, 2007).[2] Here, cluster-based permutation tests were implemented as follows: For each contrast, the difference between conditions was quantified by means of a t-test at each time-channel doublet. Data points that yielded a p-value < 0.05 and which met temporal and spatial adjacency criteria were formed to a cluster. Within each cluster, all t-values were added up. The summary statistic was defined as the maximum of these sums. For the surrogate distribution, this procedure was iterated 1000 times, each time permuting data-to-condition affiliation. The observed summary statistic was then compared to the surrogate

[2]Here and throughout this subsection, "conditions" refer to abstract categories that are being compared statistically instead of indicating specific combinations of subject type and MC.

distribution of statistics. The null hypothesis of exchangeability was rejected if the observed statistic fell under the 2.5th or over the 97.5th percentile of the surrogate distribution. Two groups of contrasts were investigated. First, the activation period was compared to the baseline period for each subject type and MC separately. This way, the statistical significance of ERSPs after stimulus presentation could be established. Second, stimulus-induced ERSPs were compared between MC and subject types. This was implemented by comparing baseline-corrected activation periods between 1) PD_{off} and HC and 2) PD_{off} and PD_{on}, respectively. Separate permutation tests were carried out for the main effects (subject type and MC) and for each interaction. For the main effect of subject type, MC_{high} and MC_{low} subsets were averaged for each subject, while for the main effect of motor content, PD_{off} and HC as well as PD_{off} and PD_{on} were treated as the same subject type. To assess interaction effects, the difference between MC_{high} and MC_{low} was computed and compared between PD_{off} and HC and between PD_{off} and PD_{on}, respectively. With these tests, research questions Q2.2.1 and Q2.2.2 were addressed. To explore possible associations between behavioral outcomes and EEG power changes across participants, average RT per subject were correlated to the mean baseline corrected power over those channels and time-points that constituted a significant cluster via Pearson correlation. Cluster-averaged power was furthermore correlated between different clusters by means of Pearson correlation to test whether distinct ERSP patterns might be related. All statistical contrasts were carried out on both the whole set of trials and on the subset of trials with high NA stimuli.

Source reconstruction

To localize the sources of neural activity associated with statistically different oscillatory patterns on sensor level, a frequency-domain beamforming approach was employed: Dynamic imaging of coherent sources (DICS) estimates source level activity by applying a spatially adaptive filter on the sensor level EEG data (Gross et al., 2001). For this, a forward model had to be generated first, which describes how source level activity would project onto sensor level. Here, a standard forward model was used for each subject. It was composed of a volume conduction model based on a high-resolution T1 magnetic resonance imaging (MRI) derived head-model (Holmes et al., 1998) processed with the boundary element method (Oostendorp & van Oosterom, 1991), scalp-projected template electrode locations of the 128-channel EEG montage and a volumetric grid of 3470 source locations. Second, to estimate the inverse model, i.e., deriving source level activity from sensor level

activity, the cross-spectral density between all channel combinations (see above) and the forward model were incorporated into an optimization procedure (Gross et al., 2001). This in turn yielded the adaptive filter which was applied on the sensor-level data to obtain estimates of source-level power. Here, a common filter was computed for both activation and baseline periods and subsequently applied to each period separately. Baseline correction of source power was then conducted in analogy to sensor level power. Finally, the difference in baseline-corrected source power between conditions was computed as percent change and projected onto a standard cortical surface mesh. Only time-frequency periods and contrasts of interest (selected based on sensor-level statistical results) were projected onto source level.

4.2 Results

4.2.1 Behavioral results

Descriptive statistics for each condition are shown in Tables 4.4 and 4.5. Average response accuracy and RT per participant and condition are depicted in Figure 4.2.

No behavioral action naming deficit in PD patients

Eight linear mixed models were established to assess the impact of subject type, MC and their interaction on response accuracy and RT. In the first model, accuracy was compared between PD_{off} and HC (Table 4.6). The fixed effects in this model explained 0.6 % of variance in accuracy (R^2[marginal] = 0.006), while fixed and random effects jointly accounted for 30 % of variance (R^2[conditional] = 0.3). No predictor made a significant independent contribution. The second model contrasted PD_{off} to PD_{on} in regards to accuracy (Table 4.7). In this model, 0.6 % of variance was explained by fixed effects (R^2[marginal] = 0.006). Fixed and random effects together accounted for 32 % of variance in response accuracy (R^2[conditional] = 0.32) with no independent significant predictor. This indicates that PD_{off} patients did not show a deficit in action naming accuracy in comparison to PD_{on} and HC.

In the third model, RT was investigated for PD_{off} and HC (Table 4.8). Fixed effects explained 0.5 % of variance (R^2[marginal] = 0.005), while both fixed and random effects represented 52 % of variance (R^2[conditional] = 0.52). As with the preceding models, no independent contribution was detected for any predictor. RT was furthermore analyzed in model 4, comparing PD_{off} and PD_{on} (Table 4.9). Here, 0.3 % of variance was explained by fixed effects (R^2[marginal] = 0.003) and both fixed and random effects together accounted for 54 % of variance (R^2[conditional] = 0.54). In this model, PD_{on} predicted longer naming latencies independently ($p < 0.001$). Thus, action naming latency was not inferior in PD_{off} patients in comparison to PD_{on} and HC.

When only including high NA stimuli (models 5 to 8), no independent contribution of any predictor under investigation was observed (Supplementary Tables S6 - S9).

	PD_{off}	PD_{on}	HC
MC_{low}	81.2 (1.2)	81.4 (1.7)	82.5 (1.8)
MC_{high}	85.9 (1.3)	85.3 (1.4)	86.2 (1.5)

Table 4.4: Mean (standard error of the mean (SEM)) response accuracy in % over all subjects.

	PD_{off}	PD_{on}	HC
MC_{low}	1816 (74)	1858 (82)	1701 (53)
MC_{high}	1831 (86)	1917 (82)	1697 (66)

Table 4.5: Mean (SEM) RT in ms over all subjects.

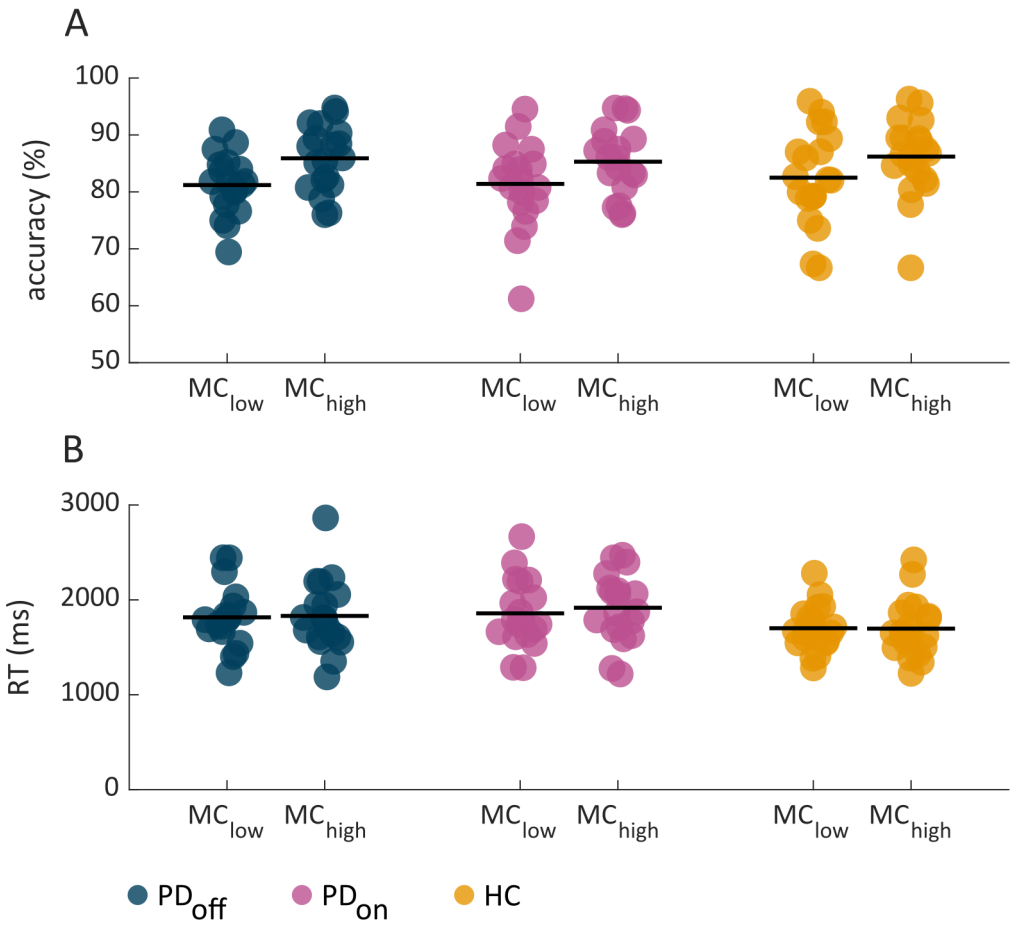

Figure 4.2: **Descriptive statistics.** Mean accuracy (A) and RT (B) per participant for each subject type and motor content. Black horizontal lines indicate grand means.

Fixed effects				
Variable	Estimate	SE	z	p
(Intercept)	2.2	0.17	13	<0.001
MC_{low}	-0.33	0.20	-1.64	0.1
PD_{off}	-0.09	0.19	-0.46	0.65
MC_{low}:PD_{off}	-0.01	0.19	-0.03	0.97
Random effects				
Group	Variable	Variance	SD	
Picture	(Intercept)	1.22	1.1	
Participant	(Intercept)	0.15	0.39	
Model fit				
R^2(marginal)		0.006		
R^2(conditional)		0.3		

Table 4.6: Results of the linear mixed effects model 1, evaluating accuracy as a function of MC and disease state.

Fixed effects				
Variable	Estimate	SE	z	p
(Intercept)	2.1	0.17	12.05	< 0.001
MC_{low}	-0.32	0.21	-1.53	0.13
PD_{on}	0.06	0.14	0.44	0.66
MC_{low}:PD_{on}	0.04	0.2	-0.18	0.86
Random effects				
Group	Variable	Variance	SD	
Picture	(Intercept)	1.37	1.17	
Participant	(Intercept)	0.13	0.37	
Model fit				
R^2(marginal)		0.006		
R^2(conditional)		0.32		

Table 4.7: Results of the linear mixed effects model 2, evaluating accuracy as a function of MC and medication state.

Fixed effects					
Variable	Estimate	SE	df	t	p
(Intercept)	-0.02	0.11	69.9	-0.15	0.88
MC_{low}	0.04	0.09	251.6	0.46	0.64
PD_{off}	0.16	0.14	39.9	1.18	0.24
MC_{low}:PD_{off}	-0.02	0.05	3067.9	-0.37	0.72
Random effects					
Group	Variable	Variance	SD		
Picture	(Intercept)	0.37	0.61		
Participant	(Intercept)	0.17	0.41		
Residual		0.51	0.71		
Model fit					
R^2(marginal)		0.005			
R^2(conditional)		0.52			

Table 4.8: Results of the linear mixed effects model 3, evaluating naming latency as a function of MC and disease state.

Fixed effects					
Variable	Estimate	SE	df	t	p
(Intercept)	0.15	0.12	32.33	1.19	0.24
MC_{low}	0.04	0.09	261.43	0.42	0.67
PD_{on}	0.15	0.04	2948.69	4.13	< 0.001
MC_{low}:PD_{on}	-0.1	0.05	2952.63	-1.77	0.08
Random effects					
Group	Variable	Variance	SD		
Picture	(Intercept)	0.38	0.62		
Participant	(Intercept)	0.21	0.46		
Residual		0.51	0.7		
Model fit					
R^2(marginal)		0.003			
R^2(conditional)		0.54			

Table 4.9: Results of the linear mixed effects model 4, evaluating naming latency as a function of MC and medication state.

4.2.2 EEG results

In the following, results of the statistical analysis of EEG data are presented. Note that whenever cluster patterns are described, this is deliberately done in a coarse way, as the exact spatio-temporal extent is highly contingent on the choice of cluster-forming parameters (Sassenhagen & Draschkow, 2019). This issue will be further explored in the Discussion but is already mentioned here for clarification reasons.

A sustained and widespread post-stimulus mu ERD emerged across conditions

To assess changes in EEG power after stimulus presentation, the activation period was compared to the baseline period by means of cluster-based permutation tests for each condition separately.

For the mu frequency band, the activation period significantly differed from the baseline period in every condition (all $p < 0.004$). These changes were driven by ERD clusters of different spatial and temporal extent (Figure 4.3): Generally, for PD patients mu power suppression was observed over occipital, parietal and central electrodes starting around 200 - 300 ms after stimulus presentation. With increasing latency, ERD was present at a higher number of central electrodes and ultimately extended towards frontal and prefrontal recording sites after about 700 ms. Less mu ERD was evident at temporal electrodes throughout the whole activation period. For HC, the observed clusters were partly comparable. ERD started around 500 ms (MC_{low}) to 600 ms (MC_{high}) but was permanently confined to frontal and central recording sites, sparing occipital and parietal regions. No significant ERS cluster was observable in all conditions (all $p > 0.14$). When only considering stimuli with high name agreement (NA > 80 %), ERSP patterns were largely comparable to the results of the full set of stimuli (all $p < 0.01$, Supplementary Figure S1).

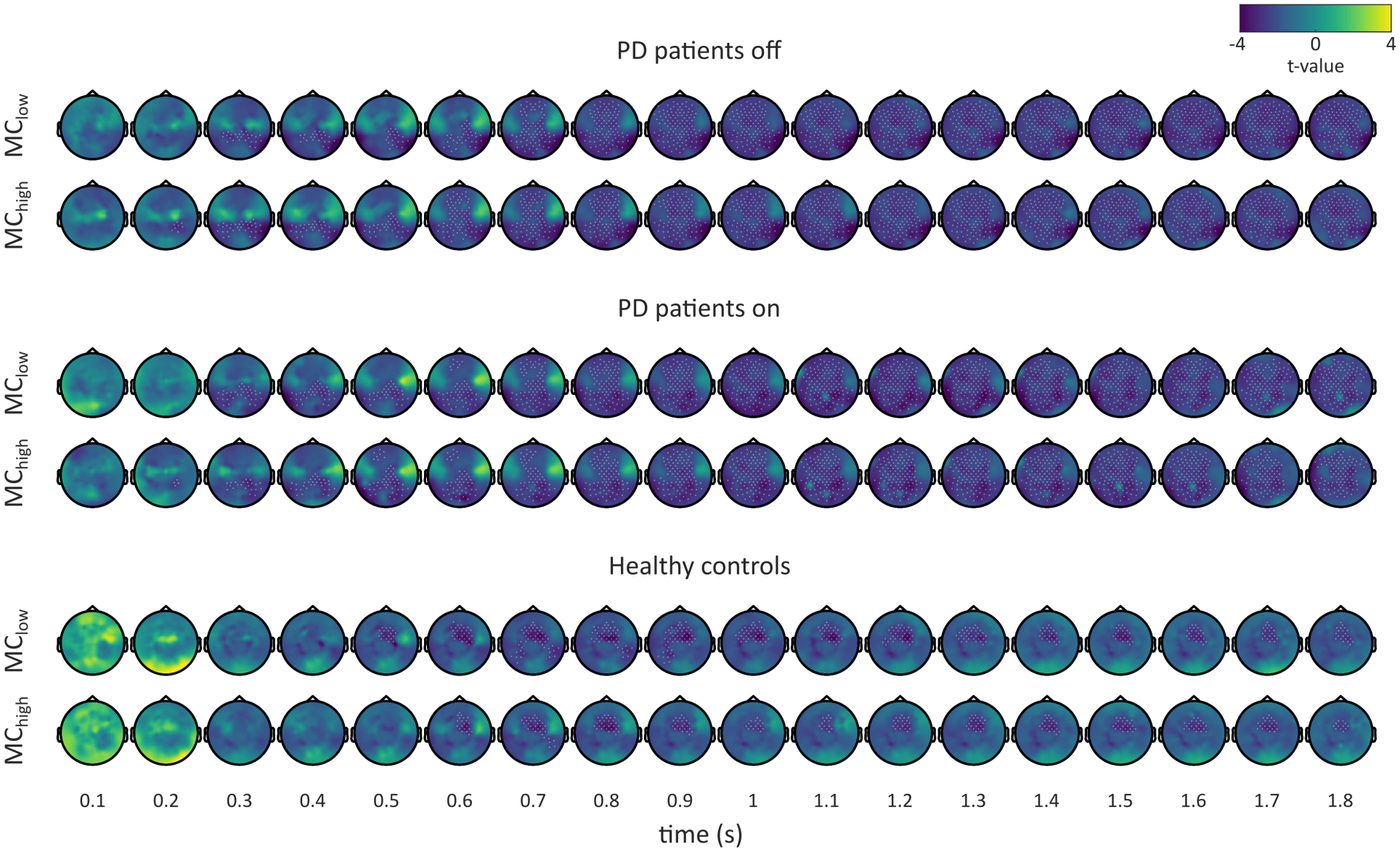

Figure 4.3: **Mu power activation versus baseline.** T-values at each time-channel doublet are displayed for the contrast between activation and baseline periods in the mu frequency range for all conditions. Higher t-values indicate increased power, whereas lower t-values mark suppressed power in the activation period as compared to the baseline period across patients. Channels included in clusters associated with significant differences between periods are highlighted by gray dots.

Stronger mu suppression in PD_{off} versus HC

Oscillatory patterns after stimulus presentation were hypothesized to differ systematically depending on MC or subject type. Therefore, cluster-based permutation tests were carried out on baseline-corrected activation period time-frequency data to investigate differences between PD patients off medication and HC, between MC_{high} and MC_{low} and their potential interaction.

In the mu frequency range, a significant difference between PD_{off} and HC during the activation period could be observed for the main effect of subject type ($p = 0.02$, Figure 4.4). The associated cluster commenced around 700 ms after stimulus onset with a spatial emphasis on occipital electrodes. Later, it also included frontocentral electrodes and lasted until the end of the analysis window, further encompassing parietal electrodes by that time. A similar pattern was observed when only considering high NA stimuli ($p = 0.015$, Supplementary Figure S5). The negative polarity of the cluster indicates that power was lower in PD_{off} than in HC. To further assess the nature of this effect, all baseline-normalized power values contained in the cluster were averaged for each subject separately and subsequently compared between PD_{off} and HC. This revealed that throughout the cluster both PD_{off} patients and HC showed mu suppression relative to baseline, however, this effect was stronger in PD_{off} (Figure 4.5 A). Furthermore, a significant difference was found for the main effect of MC ($p = 0.03$, Supplementary Figure S3). The positive cluster associated with this difference was spatially constrained to right hemispheric prefrontal to fronto-temporal electrodes. In contrast to the main effect of subject type, this cluster was shorter (800 - 1100 ms) and did not arise with high NA stimuli ($p = 0.12$). Finally, when only including high NA stimuli, a significant interaction effect associated with a short-lived (1200 - 1300 ms) and focal parieto-occipital cluster was present ($p = 0.035$, Supplementary Figure S5), which did not emerge with the complete stimulus set ($p = 0.44$).

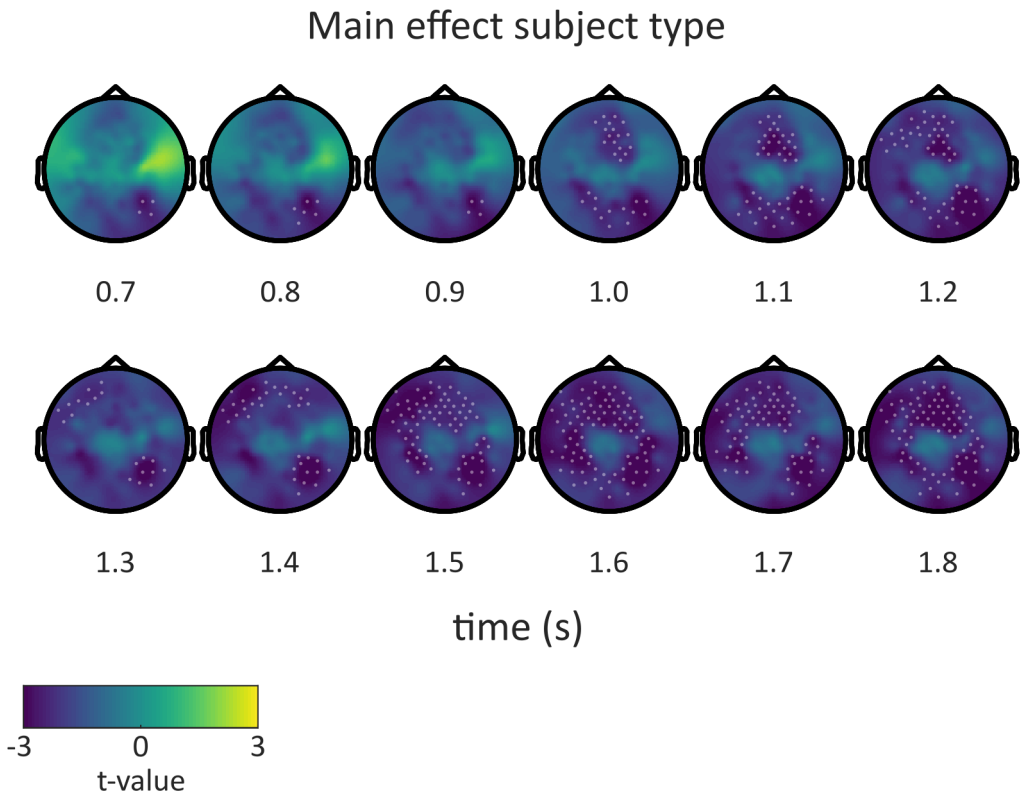

Figure 4.4: **Mu power PD_{off} versus HC - main effect subject type.** Higher t-values indicate higher power in PD_{off} than in HC and vice versa. Channels included in the cluster are highlighted by gray dots.

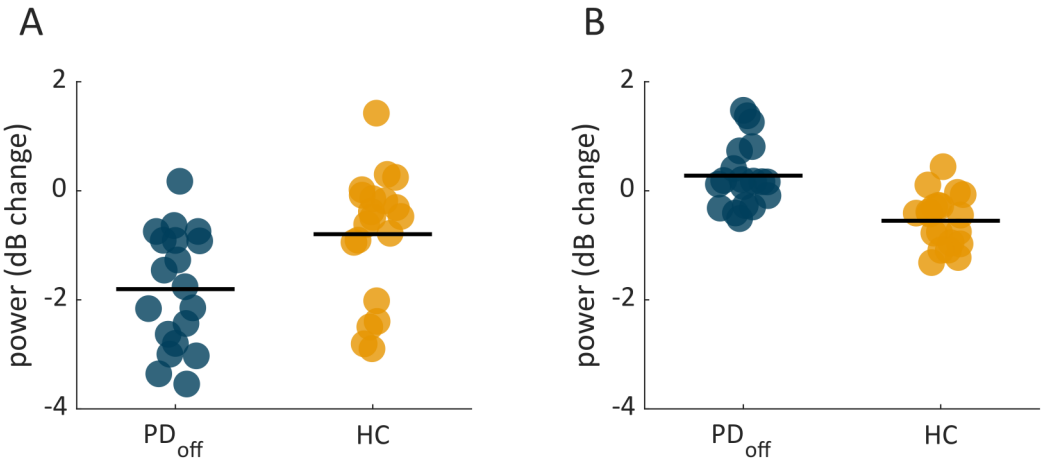

Figure 4.5: **Cluster-averaged power main effect subject type.** Mean baseline-corrected power in the mu (A) and beta (B) cluster for each PD_{off} and HC subject. Black horizontal lines indicate grand means.

Differences in mu power between PD_{off} and HC were anatomically unspecific

The strongest observed difference between PD_{off} and HC in the mu frequency range was the main effect of subject type associated with an extensive ERD starting at 700 ms after stimulus presentation. Therefore, mu power was averaged over the cluster period (Figure 4.4) and projected onto source level. This revealed widespread mu suppression, only featuring little spatial emphasis on the right occipital to parietal cortex (Figure 4.6).

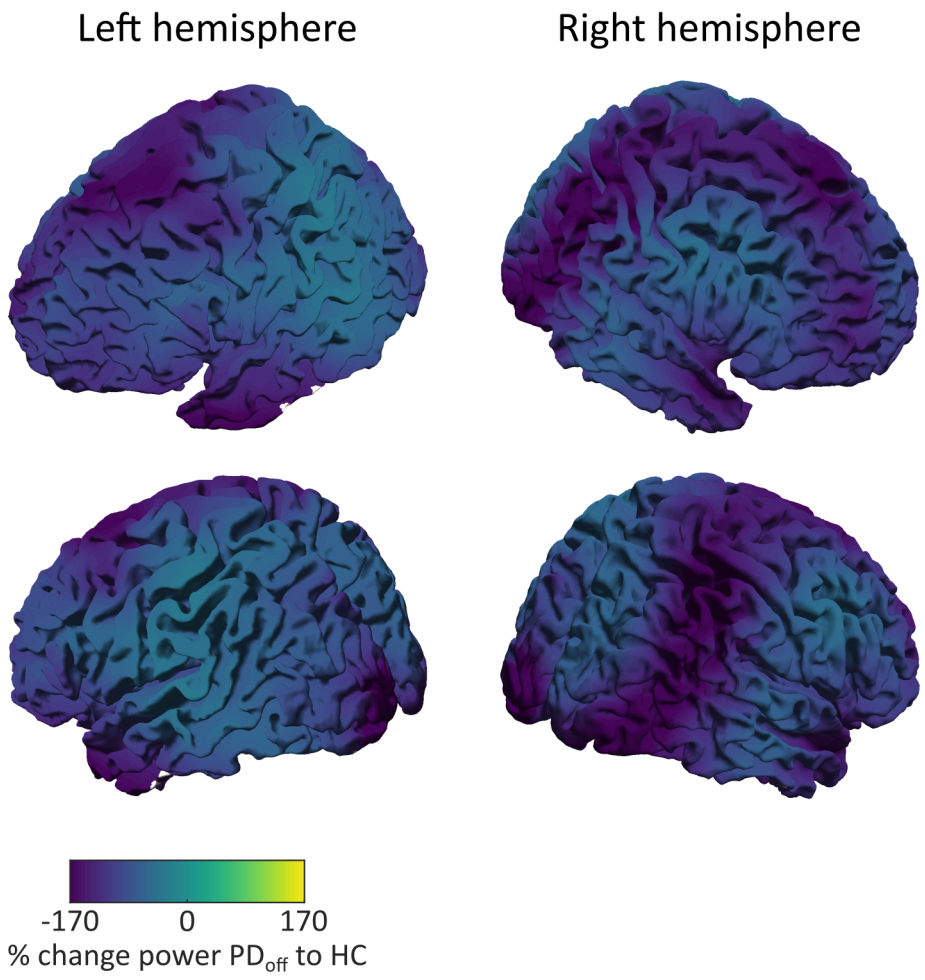

Figure 4.6: **Source reconstruction mu power PD_{off} vs. HC - main effect subject type.** Source-projected percentage difference in mu power averaged over the time interval of the cluster depicted in Figure 4.4.

An early post-stimulus ERS was accompanied by a sustained ERD for the beta frequency range in PD

Significant differences between the activation and baseline period were also observed for every condition in the beta frequency band (all $p < 0.001$, Figure 4.7). In PD patients, the differences were accompanied by both positive and negative clusters, i.e., ERS and ERD. First, a temporary ERS could be discovered approximately between 300 ms and 500 ms: Power increased at central and frontotemporal electrodes, partly extending to frontocentral electrodes but sparing the central midline. Second, similar to the mu patterns, an ERD commenced around 300 ms to 600 ms at parieto-occipital electrodes and progressively involved central and frontal recording sites during later stages. Note that depending on medication state and MC, the exact cluster shapes varied to some extent for both suppression and elevation of beta power. For HC, the ERD patterns largely recapitulate those observed in PD patients. However, beta suppression occurred slightly earlier and involved midcentral electrodes between 300 ms and 500 ms. Importantly, in contrast to PD patients, no ERS could be detected in HC. For the high NA stimulus set, comparable ERD patterns were detected (all $p < 0.001$, Supplementary Figure S2). However, the beta ERS component in PD patients did not reach statistical significance anymore (all $p > 0.07$).

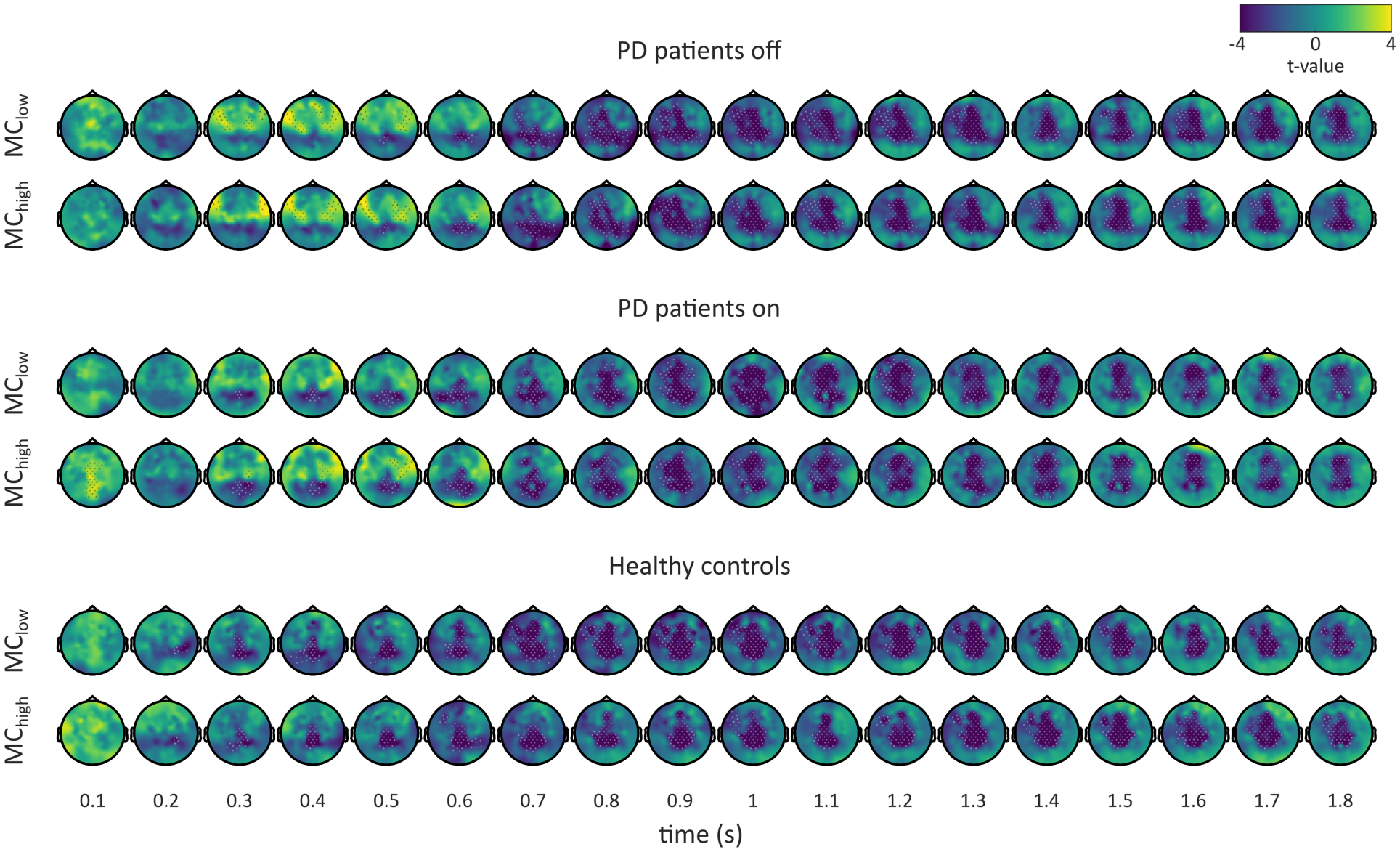

Figure 4.7: **Beta power activation versus baseline.** T-values at each time-channel doublet are displayed for the contrast between activation and baseline periods in the beta frequency range for all conditions. Higher t-values indicate increased power, whereas lower t-values mark suppressed power in the activation period as compared to the baseline period across patients. Channels included in clusters associated with significant differences between periods are highlighted by gray dots.

Elevated beta power was found in PD_{off} in comparison to HC

For the beta band, a main effect of subject type was the only significant difference during the activation period when comparing PD_{off} to HC ($p = 0.006$) (Figure 4.8). However, in contrast to mu, this difference was associated with a positive cluster instead, indicating higher power in PD_{off} than in HC. The cluster commenced around 300 ms, lasted until 700 ms and was focused on central and frontal electrodes in a symmetrical, slightly left-lateralized way. Cluster-wise averaged power indicated that this difference was due to suppression of beta power in HC while a minor increase was detected in PD_{off} instead (Figure 4.5 B). The cluster remained present when only considering high NA stimuli ($p = 0.006$, Supplementary Figure S6). For the beta frequency range, neither a main effect of MC, nor an interaction between subject type and MC could be found (all $p > 0.16$, Supplementary Figures S4 and S6).

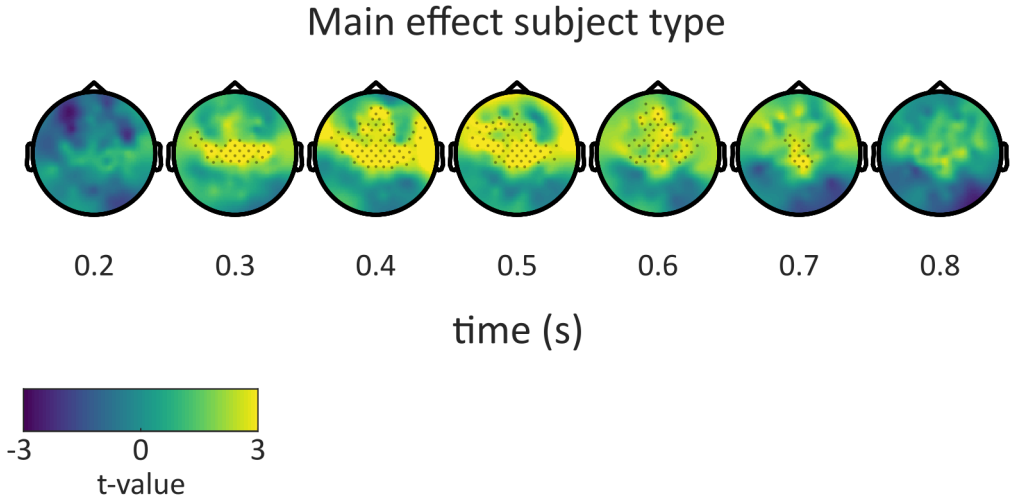

Figure 4.8: **Beta power PD_{off} versus HC - main effect subject type.** Higher t-values indicate higher power in PD_{off} than in HC and vice versa. Channels included in the cluster are highlighted by gray dots.

Differences in beta power between PD_{off} and HC localized to motor areas

As for the mu frequency range, a significant difference in beta power between PD_{off} and HC was detected for the main effect of subject type. Differences in beta activity contained within the associated cluster (Figure 4.8) were averaged over time and source-localized. Two foci emerged with this procedure (Figure 4.9). First, increased beta activity was observed in the anterior temporal lobe and a fraction of the orbitofrontal cortex of the right hemisphere in a spatially confined way. Second, on the left hemisphere, elevated beta power in PD_{off} over HC was evident in the pre- and postcentral gyrus, partly extending to the inferior frontal gyrus and the parieto-temporal junction.

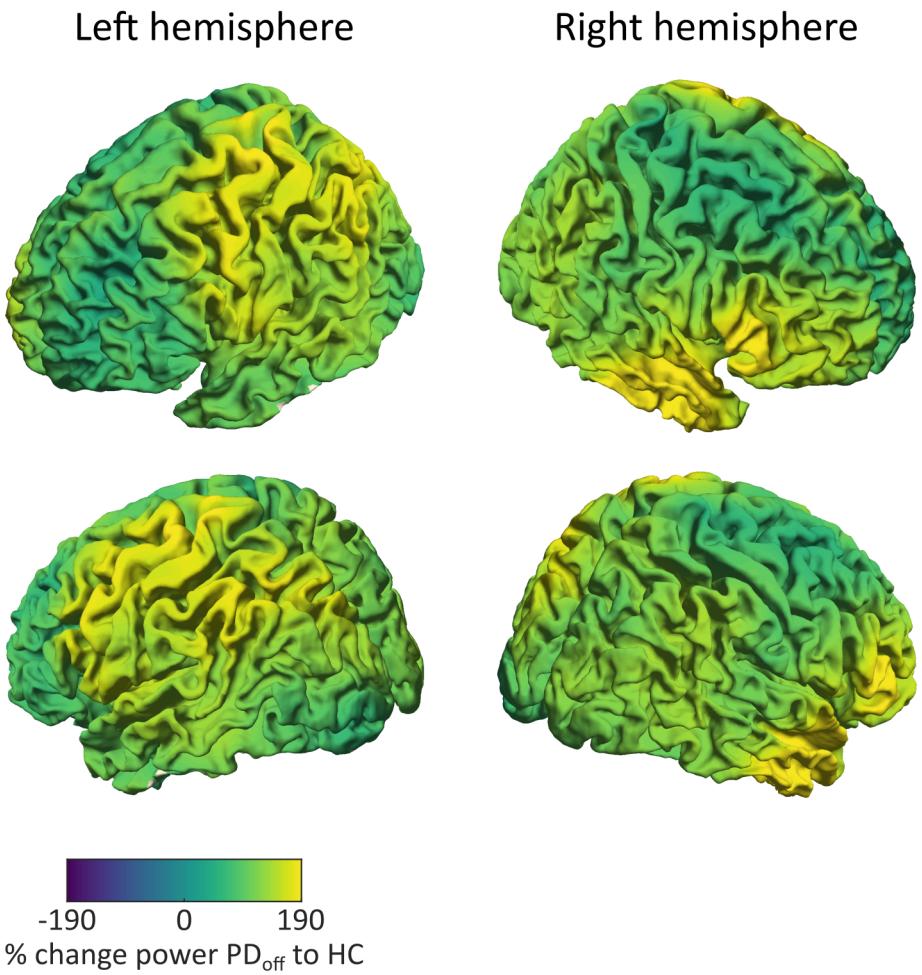

Figure 4.9: **Source reconstruction beta power PD_{off} vs. HC - main effect subject type.** Source-projected percentage difference in beta power averaged over the time interval of the cluster depicted in Figure 4.8.

No association between naming latency and ERSP magnitude

Furthermore, a possible relationship between neurophysiological patterns and naming latency was investigated in an exploratory fashion. However, correlating cluster-averaged power with mean RT per subject revealed no significant association for both frequencies and subject types (all $|R| < 0.14$, all $p > 0.58$, Supplementary Figure S7). Moreover, a potential association between mu and beta ERSPs was examined. This analysis revealed no correlation between cluster-averaged power in the mu and beta frequency band for PD_{off} patients ($R = 0.18$, $p = 0.46$, Supplementary Figure S8).

Dopaminergic medication did not change ERSP patterns

Analogous to the comparison between PD_{off} and HC laid out above, cluster-based permutation tests were carried out to investigate differences in ERSPs due to medication state within PD patients. With this analysis, no significant difference in ERSP patterns in both the mu and beta frequency ranges during the activation period could be observed when comparing PD patients on and off medication (all $p > 0.25$).

5 Discussion

5.1 Experiment 1

This section has been published in a partly modified version in Busch et al. (2021).

In Experiment 1.1, 283 action pictures were compiled from two sources (Bayram et al., 2017; Szekely et al., 2005) and related psycholinguistic variables were validated for the German language. In Experiment 1.1, data from 55 healthy participants were obtained and normative values for motor content, the distribution of verbal responses to each picture and visual complexity were established. Further, precise naming latencies were measured within the controlled experimental setup. In Experiment 1.2, 600 German verbs (including 299 responses from Experiment 1.1) were assessed. Three groups of 41 - 45 healthy participants each rated imageability, motor content or age of acquisition of every verb. In addition, word frequency, word length, ortographic Levenshtein distance of the 20 nearest neighbors, transitivity, reflexivity and morphological complexity were determined for each verb.

5.1.1 Associations among classical psycholinguistic variables

The relationship between picture and verb characteristics revealed in Experiment 1 largely confirmed results from previous studies. A strong correlation between AoA and FR was found, with more frequent words being learned earlier in life. This is a well-established effect that has already been demonstrated for the German language (Kauschke & von Frankenberg, 2008; Schröder et al., 2012) and in a range of other languages (Cuetos & Alija, 2003; Schwitter et al., 2004; Shao et al., 2014; Szekely et al., 2005). In addition, H and $n_{response}$ decreased while NA increased moderately as a function of IM, which is a robust finding across studies investigating these variables (Cuetos & Alija, 2003; Kauschke & von Frankenberg, 2008; Khwaileh et al., 2018; Shao et al., 2014). Furthermore, words learned at an earlier age were easier to imagine, which has also been found consistently in previous timed and untimed action naming studies (Akinina et al., 2015; Bayram et al., 2017; Cuetos & Alija, 2003; Kauschke & von Frankenberg, 2008; Khwaileh et al., 2018; Masterson &

Druks, 1998; Shao et al., 2014).Together, the replication of results from German and non-German studies regarding associations among picture and verb characteristics indicates that these are robust across groups, settings and languages.

5.1.2 Predictors of naming latency

Picture and verb characteristics were differentially correlating with naming latency. RT of visually depicted actions strongly increased with $n_{response}$ and with H but decreased with NA. These results are in accordance with previous studies investigating action naming. Several studies report similar correlations between naming latency and name agreement indices (Cuetos & Alija, 2003; Kauschke & von Frankenberg, 2008; Khwaileh et al., 2018; Schwitter et al., 2004; Shao et al., 2014; Szekely et al., 2005) and also between naming latency and imageability (Kauschke & von Frankenberg, 2008; Khwaileh et al., 2018; Shao et al., 2014).

The linear mixed model analysis confirmed the effect of H on naming latency. This goes in line with a range of picture naming studies for actions and objects in other languages included in a meta-analysis by Perret and Bonin (2019). In addition, linear mixed modeling revealed that RT was longer for more frequent words. Most previous normative action naming studies did not find an independent contribution of word frequency to naming latency (Cuetos & Alija, 2003; Kauschke & von Frankenberg, 2008; Khwaileh et al., 2018; Schwitter et al., 2004) In these experiments, the authors used restrictive measures of word frequency based on news articles or books. However, recent studies showed that FR derived from television and film subtitles, as used in Experiment 1, is superior to estimates based on written sources (Brysbaert et al., 2011; Brysbaert & New, 2009). The higher quality of word frequency estimates employed here may have revealed the predictive value of word frequency on naming latency. The somewhat unexpected relationship between high FR and slower RT is in line with one previous action naming study (Szekely et al., 2005). These authors speculated that participants fall back on high-frequency multipurpose verbs for difficult items, leading to inflated naming latency due to the more effortful (and ultimately unsuccessful) search for a specific verb (Szekely et al., 2005). However, this hypothesis remains to be tested in future studies, e.g., by obtaining parameters capturing semantic specificity. In addition, the relatively weak effect of word frequency as revealed in Experiment 1 warrants further replication in other languages with contemporary word frequency measures and should be interpreted with caution.

In contrast to previous action naming normative studies (Cuetos & Alija, 2003; Schwitter et al., 2004; Shao et al., 2014; Szekely et al., 2005) an effect of age of acquisition on naming latency was not found in Experiment 1. A large body of evidence shows that AoA correlates with FR (Johnston & Barry, 2006). If one follows the assumption that frequent words occasionally served as fallback verbs for more difficult pictures, one could speculate that these items, which are associated with slow reaction time, obscured the otherwise expected positive correlation between AoA and RT. Furthermore, IM did not exert an independent effect on naming latency, as previously shown (Kauschke & von Frankenberg, 2008). This suggests that the effect of IM on naming latency is mediated by name agreement indices.

5.1.3 Motor content and further psycholinguistic variables

Beyond the standard variables reported in action naming normative studies in other languages, MC_{pic} and MC_{word} have been investigated in Experiment 1. Both variables were highly inter-correlated and were positively associated with imageability. An unexpected finding was the opposing effect of MC_{pic} and MC_{word} on RT in the linear mixed model. While high MC_{pic} was associated with faster naming latency, the contrary was the case for MC_{word}. However, both parameter estimates were small in relation to the effect of H. Both MC_{pic} and MC_{word} have not yet been formally studied in action naming normative studies. A possible dissociation between MC_{pic} and MC_{word} may be investigated in future studies. Here, generating MC normative data was primarily necessary to establish clearly defined sets of stimuli in Experiment 2.

A second rarely reported variable, OLD20, was positively correlated with word length and age of acquisition and was negatively associated with word frequency, corroborating previous findings (Yarkoni et al., 2008). However, OLD20 did not predict naming latency, in contrast to the findings of Yarkoni et al. (2008). Morphological complexity and transitivity both showed no considerable association with naming latency. As to be expected, complex verbs were longer and exhibited a sparser orthographic neighborhood than non-complex words, but they were also less imaginable.

5.1.4 Conclusion

In summary, a first data set of picture and verb characteristics for a compilation of 283 freely available action pictures (Bayram et al., 2017; Szekely et al., 2005) was characterized for the German language. This normative data set including standard and new parameters will be useful for future behavioral and neuroscientific studies on the cognitive processes underlying action naming. Similar relationships between picture and verb characteristics in comparison to action naming studies in other languages were found, indicating high construct validity. Entropy of responses, motor content of pictures and words as well as word frequency constituted independent predictors of naming latency. These findings are partly in keeping with hypothesis H1, with the exception that AoA did not predict naming latency in this study.

5.2 Experiment 2

In Experiment 2, an action naming task using stimuli validated in Experiment 1 was carried out in 19 patients with PD and in 20 healthy controls. Accuracy of responses and naming latency were investigated as behavioral outcome parameters. Simultaneous high-density EEG recordings were used to assess neurophysiological correlates of action language processing. Changes in oscillatory power in the mu and beta frequency range were compared between baseline and activation periods as well as between subject types and stimuli with high and low MC.

5.2.1 No evidence for action language deficits in Parkinson's disease

In this study, both differences in accuracy of responses and in naming latency were investigated as a function of subject type and MC. This was motivated by previous research indicating that patients with PD produce more response errors when naming actions in comparison to objects, while no such difference is observable in healthy controls (Rodríguez-Ferreiro et al., 2009). Moreover, naming performance was shown to specifically deteriorate for actions with high MC (Herrera et al., 2012) and in PD patients off medication (Herrera & Cuetos, 2012), suggesting impaired semantic access to action concepts in PD_{off}.

In contrast to these data, no difference in response accuracy due to subject type, MC or their interaction could be observed in this study. What may account for the discrepancy to previous findings? When looking at similar studies (Cotelli et al., 2007; Herrera & Cuetos, 2012; Herrera et al., 2012), demographics may play a role. In this study, mostly highly educated participants were included (average years of education: 15.9 years (HC) and 16.3 years (PD)), whereas the average level of education was considerably lower in Herrera et al. (2012) (approx. 6 years), Herrera and Cuetos (2012) (approx. 10 years) and in Cotelli et al. (2007) (approx. 7 years). There is converging evidence for a positive correlation between years of education and object naming performance in healthy subjects (Neils et al., 1995; Stasenko et al., 2019; Zec et al., 2007) and a recent study indicated a similar effect for action naming (Papagno et al., 2020). Thus, deteriorated response accuracy in action naming in PD may be only uncovered in less educated patients, consistent with the hypothesis of a larger "cognitive reserve" with higher levels of education (Meng & D'Arcy, 2012).[1] Additionally, the observation that action naming accuracy in PD is positively correlated with executive functions (Cotelli et al., 2007) and the latter being associated with higher education in PD (Loftus et al., 2021) may support this explanation. Thus, action naming impairments may be - to some extent - a consequence of executive dysfunction, which is more apparent in PD patients with less years of education.

While the latter reasoning may account for a missing main effect of subject type, it is unlikely that this interpretation can also explain the absent interaction between MC and subject type. This specific effect was hypothesized to arise from differences in semantic processing and may be more difficult to explain through executive dysfunction (Herrera et al., 2012). While indeed an association between higher cognitive reserve and improved semantic processing has been shown in patients with MCI (Darby et al., 2017), no such relationship could be established for PD, though (Guzzetti et al., 2019). Following this, higher education levels in the study sample may not account for the absent interaction effect.

However, further methodological differences between this study and the two studies by Herrera et al. concern stimulus characteristics and may have contributed to conflicting results. In the latter two studies, n = 25 high name agreement stimuli (average NA approx. 90 %) were presented to participants within each MC group. In

[1] It could be argued that this hypothesis could have been tested in this study as well, by correlating years of education with behavioral outcomes. However, this would only provide evidence regarding a potential association within highly educated participants, which can not be generalized to a population with a more diverse educational background in a straightforward way.

this study, though, with the aim of collecting a high number of trials for subsequent EEG analyses, n = 57 pictures were compiled for each MC bin, which required the inclusion of stimuli with low name agreement. This may have obscured a semantic effect of MC, due to less homogeneous semantic concepts being elicited in participants. Furthermore, the concept of naming accuracy assumes a certain degree of uniformity of responses: Considering a response as right or wrong is only meaningful when there is consensus about the ground truth. Thus, to address this issue, a sub-selection of stimuli with NA $\geq$ 80 % was drawn, amounting to similar NA levels as in Herrera and Cuetos (2012) and Herrera et al. (2012) (compare Supplementary Table S3). However, while naming accuracy was generally higher in this subset for all subject types and for both stimuli with high and low MC, no difference between conditions could be observed as well. Note that due to the strict name agreement constraint, each subset only comprised n = 13 stimuli. Therefore, statistical inferences were based on a markedly lower number of trials, reducing the statistical power of this analysis. In sum, previous findings of reduced action naming accuracy in PD could not be replicated in this study, potentially owing to higher levels of education among participants. Furthermore, a missing interaction between subject type and MC challenges prior accounts of a specific action naming deficit for MC_{high} stimuli in PD_{off} after accounting for methodological variations across studies.

Alongside response accuracy, naming latency was measured as a further outcome parameter. For the computation of naming latency, only accurate trials were considered. This way it could be assured that the intended semantic concept was in fact accessed by each participant and subtle deficits in semantic processing would be reflected in longer naming latency. However, similar to the results for accuracy, no effect of MC, subject type or their interaction was observed that would support the hypotheses of this study. In contrast, when considering all stimuli, the model comparing PD patients off and on medication indicated longer naming latency for PD_{on} irrespective of MC. This finding could not be replicated in the sub-selection of high NA stimuli, which more stringently controlled for the semantic concept that was invoked in each participant. In contrast, Herrera and Cuetos (2012) observed an interaction between subject type and MC that would be in keeping with a specific deficit for MC_{high} stimuli in PD patients off medication. However, the authors report the computation of a compound score encompassing both RT and accuracy. Unfortunately, it is not clear whether their results refer to this score or to uncorrected RT, rendering a direct comparison difficult. The authors report average RT for every combination of subject type and MC, though, which clearly indicate longer naming latency in PD_{off} than in PD_{on}. In the light of these results, the significant main effect

for medication state in this study remains unclear: It is not present when focusing on more clearly defined stimuli and it is not associated with complementary changes in accuracy. Thus, there is only weak evidence for a genuine behavioral deficit in PD_{on} patients in comparison to PD_{off}.

To conclude, this study does not support the hypothesis of a specific behavioral action naming deficit for high MC stimuli in PD patients off medication as has been formulated in H2.1.1 and H2.1.2. Future studies may consider other psycholinguistic paradigms (e.g., assessing language comprehension instead of language production) to examine a potential task-dependency of action language processing performance in Parkinson's disease.

5.2.2 Mapping EEG patterns to language production components

While behavioral correlates in favor of a proposed deficit in semantic processing of action language in PD could not be detected in this study, differences in EEG patterns may still be informative in regards to the neural processes underlying action concept retrieval and whether they are altered in PD. However, interpreting oscillatory motifs as reflecting a specific cognitive process is not straightforward and builds upon several assumptions. Thus, before interpreting the electrophysiological results, some of these prerequisites and their relation to this study will be discussed.

Experiment 2 built upon the hypothesis that action naming in PD is accompanied by altered semantic access to action concepts in sensorimotor areas. First and foremost however, this hypothesis comes with the implicit assumption that, in language production, a cognitive process as semantic access with its neural implementation does indeed exist. This argument has been consistently formulated in theories of language production, which themselves build upon the synthesis of a large body of psycholinguistic data: For example, one influential model of language production has been proposed by Levelt et al. (1999), which assumes distinct processing stages connected in a largely sequential and feed-forward way. In this model, conceptual preparation is an early step in the process of picture naming, which aims at binding the perceived picture to a semantic concept that can be expressed as a word (i.e., a lexical concept). After conceptual preparation has completed, further processing stages are following, ultimately leading to articulation. While other models differ

e.g., regarding the relationship between processing steps (compare Dell (1986) for an interactionist account), it is nevertheless largely agreed upon "that there are processing levels of meaning, form, and articulation" (Indefrey, 2011, p. 2). Thus, without going into further detail on how these theories establish conceptual preparation as a pivotal step in language production, this assumption is supported by a wide base of psycholinguistic data (Indefrey & Levelt, 2004; Indefrey, 2011).

Second, after justifying that conceptual preparation as a cognitive process exists, it is reasonable to narrow down its time course. This way, spatiotemporal oscillatory patterns that are in keeping with previously reported time frames can more likely be interpreted as reflecting conceptual preparation and in the case of conflicting patterns, false-positives can be identified likewise. In an attempt to specify the time courses of processing steps involved in language production, Indefrey (2011) conveyed a meta-analysis of behavioral and ERP data from semantic decision tasks and estimated an interval of 0 - 200 ms after stimulus presentation for conceptual preparation. This time window was largely confirmed by Strijkers and Costa (2011), reviewing overt word production studies. More recently, in a picture naming task similar to the one employed in this study, it was found that the manipulation of semantic stimulus properties was associated with differential ERPs around 150 ms (Miozzo et al., 2015). Notably, by varying action features of the presented stimuli, the authors found differential cortical activation at this time in the ventral premotor cortex and the inferior frontal gyrus, which is in keeping with the notion that the motor system supports semantic processing of action concepts.

In principle, these results can be used as a framework to facilitate correct identification and interpretation of oscillatory patterns found in this study. However, direct transferability is limited due to several reasons: First, virtually all studies that have been included in meta-analyses of language production (Indefrey & Levelt, 2004; Strijkers & Costa, 2011) employed stimuli depicting objects instead of actions. In language comprehension, though, differential effects of objects and actions on ERP waveforms arise around 200 ms (Pulvermüller et al., 1999). However, by analyzing spatiotemporal ERP evolution using magnetoencephalography (MEG) recordings during action and object naming tasks, Sörös et al. (2003) and Liljeström et al. (2009) showed similar activation patterns for both actions and objects. Thus, these data indicate that component time course estimates of object naming can also be applied to action naming. Second, the relative time frames of language production components vary as a function of task demands and stimulus characteristics. In their meta-analyses, Indefrey and Levelt (2004) and Indefrey (2011) stress the fact

that their average word production component estimates were based on studies using repeated naming of the same stimuli. This is in contrast to this study in which participants were confronted to each stimulus only once. Repeating stimuli induces a priming effect that shortens the late stages of object identification, conceptual preparation and lexical as well as phonological selection (Francis, 2014).[2] However, early object identification and articulation are left unaffected by stimulus repetition. Additionally, most studies that were considered in the two meta-analyses employed stimuli with very high name agreement, which are associated with shorter naming latency. To increase the number of trials and thus improve the signal-to-noise ratio for EEG analyses, low NA stimuli were also used in this study instead. Low name agreement prolongs the processing stages of visual recognition, conceptual preparation and lexical selection (X. Cheng et al., 2010; Vitkovitch & Tyrrell, 1995). In the light of these aspects - absent priming and stimuli with low name agreement - the time course estimates of Indefrey (2011) have to be scaled accordingly: It is plausible to assume that visual recognition took considerably longer in this study due to absent repetition priming and low name agreement stimuli. Additionally, conceptual preparation itself was probably prolonged as well, due to the same effects. Thus, in this study conceptual preparation likely started after a period of prolonged visual recognition and finished far later than the 200 ms estimate from Indefrey (2011) when considering the whole set of presented stimuli. In an attempt to at least control for the effect of low name agreement, all EEG analyses were carried out on a subset of stimuli with high NA as well. However, this comes with decreased statistical power due to the limited number of pictures meeting high NA criteria.

5.2.3 Action naming induces mu and beta event-related desynchronization

After having discussed some of the underlying assumptions for attributing oscillatory patterns to psycholinguistic processes, the dynamics of observed ERSPs will now be put in the context of previous literature. After that, it will be evaluated whether differences in these oscillatory dynamics may reflect behaviorally insignificant but altered semantic access to action concepts in the sensorimotor system.

[2] Studies investigating effects of repetition priming on picture naming usually employ stimuli depicting objects instead of actions. In a rather pragmatic way, similar effects for action naming are assumed here.

In this study, the time courses of both mu and beta power changes depicted in Figures 4.3 and 4.7 were predominated by post-stimulus power suppression. While changes in oscillatory power compared to a low-level baseline (e.g., fixation cross) have not yet been described in detail in picture naming tasks, one study reported desynchronization of 9 to 13 Hz activity over frontal and sensorimotor sites, which followed an earlier occipital ERD in the same frequency range (Salmelin et al., 1994). This is partly in keeping with the present study, where suppression of 8 to 12 Hz activity was observable at frontal sites for both PD patients and healthy controls, starting around 700 ms post-stimulus. However, while the ERD pattern in healthy controls was spatially confined to frontal sites, widespread suppression over almost all electrodes was evident in patients with PD. Surprisingly, in contrast to Salmelin et al. (1994) no preceding occipital 8 to 12 Hz ERD was present in healthy controls.

Further studies on the role of mu and beta dynamics in action semantics employed other paradigms than overt picture naming but are largely in accordance with the early oscillatory patterns up to approximately 600 ms found here. In a single word reading experiment, van Elk et al. (2010) observed an ERD in the 20 to 30 Hz range 500 ms to 600 ms after action verb presentation, which they associated with conceptual processing. The authors reported beta desynchronization mainly over fronto-central electrodes, which resembles the pattern found in HC in this study. Similar topographic results for the beta frequency range have been obtained by comparing listening to action verbs and abstract verbs (Moreno et al., 2013). Here, in contrast to HC, no beta ERD was present in PD at these recording sites. Van Elk et al. (2010) also observed an ERD in the mu frequency range, showing a similar topographic distribution to beta but starting already after 150 ms. This is in contrast to the results described here, where mu desynchronization over central and frontal electrodes only emerged after 600 ms for all subject types. However, it has to be emphasized that in picture naming upstream visual processing is probably more complex than in word reading. This may lead to a relative delay in semantic processing in picture naming. In a covert action naming task, Cuellar and Del Toro (2017) found mu and beta desynchronization over pre-defined independent components which were selected to reflect sensorimotor mu and beta oscillations. Generally, this is supporting the findings of this study as well, however, a formal comparison is complicated by the specific analysis approach employed.

The subsequent long-lasting desynchronization over both mu and beta frequency bands after approximately 700 ms is in keeping with prior descriptions of oscillatory patterns accompanying preparation for speech movements. Salmelin et al. (2000)

observed a 20 Hz ERD near the central sulcus of both hemispheres, starting around 700 ms before speech onset in an overt word reading task. Similarly, by employing a verbal repetition task, Herman et al. (2013) found ongoing suppression of 4 Hz to 13 Hz activity starting around 850 ms prior to speech onset. Furthermore, decreased beta power was detected from 600 ms pre-speech onset onwards over left motor and premotor cortices. This is compatible with the general observation of mu and beta ERD accompanying motor preparation and is thought to reflect activation of the motor cortex (Pfurtscheller & Lopes da Silva, 1999). Thus, the consistent and ongoing desynchronization of mu and beta activity over central to frontal electrodes observed over the last 600 ms to 900 ms prior to average speech onset may be in large part explained by motor preparation and execution.

In sum, while fully characterized spatio-temporal reference patterns of mu and beta dynamics in action naming are missing, the differential changes in oscillatory power during the activation period described here are largely in keeping with prior data and may support motor cortex engagement during different stages of word production.

5.2.4 Altered sensorimotor beta oscillations in Parkinson's disease may be linked to semantic processing

The comparison between baseline and activation period revealed relatively consistent spatiotemporal oscillatory patterns across subject types and for both MC_{high} and MC_{low}. However, to detect differences between conditions that could ultimately be linked to differential semantic processing, cluster-based permutation tests were carried out on the baseline-corrected time-frequency representations.

The most intriguing result of these analyses was the main effect of subject type in the beta frequency range when comparing PD_{off} versus HC. Beta power was suppressed in HC over central electrodes in the period between approximately 300 to 700 ms, whereas in PD_{off} elevated beta power was observed (compare Figures 4.8 and 4.5). As stated above, it is reasonable to assume that conceptual preparation started after a longer period of visual recognition in this study. Thus, it may be possible that processing of action semantics at least partly overlapped with the interval of 300 to 700 ms and as such can be linked to an absent beta ERD in PD_{off}. Furthermore, the anatomical pattern of differential beta activity included the left sensorimotor cortex (Figure 4.9), which is largely in keeping with previous data on the source of cortical

beta oscillations (Bai et al., 2005; Salmelin & Hari, 1994). Given the anatomical localization and the strong association of beta oscillations with sensorimotor processing (Kilavik et al., 2013), the focal and temporally confined differences in beta power between PD_{off} patients and HC may reflect a differential engagement of the motor system in the semantic processing of action concepts. This pattern strongly reflects findings from related experimental paradigms. Heida et al. (2014) studied mu and beta ERD in patients with PD in contrast to HC during an action observation task. While HC showed reduced mu and beta power over central electrodes during movement observation, this effect was absent in PD patients. Interestingly, increased beta power in PD_{off} also localized to the right hemispheric anterior temporal lobe. The temporal poles are critical in the access to semantic knowledge as evidenced by their degeneration in semantic dementia, a condition leading to pronounced anomia due to the loss of semantic representations (Hodges et al., 1992). Thus, it has been proposed that the anterior temporal lobes may act as a neural "hub", coordinating modality specific access to semantic concepts grounded in distributed brain regions (Patterson et al., 2007). Altered retrieval of action concepts from the sensorimotor cortex may therefore be mediated by the right anterior temporal lobe and could be jointly reflected by increased beta activity in PD_{off}. However, it has to be emphasized that the findings were not accompanied by a behavioral correlate and that higher cluster averaged beta power was not associated with increased naming latency in PD. Furthermore, the experimental paradigm did not allow to exactly narrow down the interval of conceptual processing. Thus, the interpretation is solely based on adopting previously established time course estimates (Indefrey, 2011), an indirect method which has been criticized elsewhere (Strijkers & Costa, 2011). Future studies may therefore complement analyses of oscillatory patterns with investigations on even-related potentials to provide additional evidence on the temporal sequence of processing steps (including conceptual preparation) in action naming.

Moreover, the absent interaction effect between subject type and MC questions whether differential beta modulation truly reflects differences in conceptual preparation instead of other subject type specific alterations in neural processing. What may account for this absent interaction? The hypothesis of attenuated mu and/or beta desynchronization for MC_{high} stimuli in PD_{off} patients was partly based on studies demonstrating a relationship between the extent of mu/beta modulation and the magnitude of executed or imagined simple (hand) movements (Avanzini et al., 2012; Stančák et al., 1997; Yuan et al., 2010). In this study however, the stimuli illustrated mainly complex movements, which were depicted as a still image.

Thus, the kinematics of the presented concepts may have been more difficult to decode by participants. In fact, it has been shown that action naming in response to videos induces stronger mu and beta desynchronization as compared to pictorial stimuli (Cuellar & Del Toro, 2017). Furthermore, there are also reports on absent scaling of beta ERD with movement velocity (Tatti et al., 2019). Finally, it has to be noted that incongruent time windows for semantic processing across participants constitute an unlikely source for false negative results: All EEG analyses were carried out on a high NA subset of stimuli as well, facilitating a temporal overlap of conceptual preparation. These analyses yielded highly comparable results to those of the full stimulus set, though. Thus, any absent effect reported here was unlikely to arise from temporal heterogeneity in regards to semantic processing. In sum, the absence of an interaction between MC and subject type may speak against MC dependent grounding of action semantics in the sensorimotor system. However, employing pictorial instead of video stimuli may have impeded the detection of such an effect. Furthermore, this negative result does not preclude the possibility of MC independent grounding of action concepts.

Besides a cluster of elevated beta power in PD_{off} as compared to HC early after stimulus presentation, a late prolonged and spatially extensive cluster of stronger 8 Hz to 12 Hz desynchronization in PD_{off} was observed as well. This pattern encompassed almost all recording sites and was anatomically unspecific, rendering mu ERD as a sole contributor to this contrast unlikely. It was discussed above that the missing behavioral differences between subject types may have been due to higher education levels in the study sample. Following this rationale and presuming that the neurophysiological correlates in the beta band reflect altered semantic processing, one could have speculated that the observed stronger mu reactivity may represent a correlate of a compensatory mechanism in PD patients with higher cognitive reserve. However, this interpretation can be challenged from several perspectives. First, no clear oscillatory patterns have been associated with the concept of cognitive reserve so far, including absent evidence of increased mu reactivity (Balart-Sánchez et al., 2021). Second, higher beta power in the early cluster was not associated with lower mu power in the late cluster. Furthermore, the oscillatory pattern in the mu frequency range associated with the main effect of subject type was spatially and temporally widespread. This complicates attempts of linking it to a specific neurocognitive process that could be a candidate compensatory mechanism. While timing information alone would suggest more downstream processes in language production (e.g., phonetic encoding and articulation, (Indefrey, 2011)) the widespread spatial pattern contradicts a singular process. Finally, higher mu desynchronization was

not associated with reduced RT, speaking against a behaviorally facilitative effect attributable to this pattern. Thus, the significance of this finding remains rather unclear.

It is furthermore interesting that a difference between Parkinson's disease patients off medication and HC reasonably attributable to semantic processing could only be observed in the beta band in this study. Prior reports have also identified mu ERD as a correlate of action semantic processing (Cuellar & Del Toro, 2017; Moreno et al., 2013; Moreno et al., 2015; van Elk et al., 2010). This may suggest that motor cortical engagement is specifically altered during action language processing in PD, given the different source areas of mu and beta oscillations. However, as both mu and beta ERD are attenuated in PD during action observation (Heida et al., 2014), this finding is rather surprising. For now, the functional relevance of this dissociation has to be left unexplained.

Further comments have to be provided on the absent medication effect when comparing PD_{on} to PD_{off} patients. L-Dopa intake did not change oscillatory dynamics in both the mu and beta frequency ranges. Specifically, it did not reestablish the spatiotemporal patterns seen in HC. This is surprising, given the fact that an improvement in motor function was evident by an UPDRS decrease of about 30 %, indicative of effective treatment. Previous data on the effects of L-Dopa on mu and beta oscillations have shown a decrease in resting state subthalamic beta power associated with clinical improvement (Kühn et al., 2006) as well as increased movement-related cortical mu reactivity in PD patients (Magnani et al., 2002). Thus, it was hypothesized that L-Dopa would have led to stronger mu and/or beta reactivity in PD patients during action language processing. This claim is therefore not supported by the results of this study. Indeed, while the desynchronizing effect of L-Dopa on subcortical oscillations has been repeatedly shown (Giannicola et al., 2010; Kühn et al., 2006; Sure et al., 2021; Tinkhauser et al., 2017), cortical beta hypersynchronization could be observed after L-Dopa intake instead (Cao et al., 2020; Melgari et al., 2014). This suggests that the functional properties of cortical versus subcortical beta oscillations are not fully congruent and this may explain why cortical oscillatory correlates of action language processing were not modulated by L-Dopa in this study. Furthermore, behavioral data indicated a naming latency disadvantage for PD_{on} patients in comparison to PD_{off}. However, as no neurophysiological correlate of this effect could be observed, the specificity of this result is questioned from this perspective as well.

To sum up, the hypothesis of an interaction effect between MC and subject type was not supported by the results of this study. However, elevated beta power in PD_{off} over HC irrespective of MC showed a characteristic spatiotemporal pattern suggestive for altered recruitment of the motor cortex during action semantic processing. Thus, hypothesis H2.2.1 receives partial support by the results of this study. However, dopaminergic medication did not show any behavioral or neurophysiological effect, speaking against hypothesis H2.2.2.

5.2.5 Limitations

Several methodological limitations have to be emphasized in order to interpret the here presented results appropriately.

First, the exact patterns of observed ERSPs are contingent on the methods and parameter choices used for time-frequency transformation and statistical analysis. For example, the number of wavelet cycles determines the time/frequency trade-off, with lower number of cycles leading to improved temporal but worse spectral precision and vice versa (Cohen, 2014). This means that both the temporal or spectral extent of ERSPs can be both over- or underestimated depending on wavelet width. As temporal precision was prioritized over spectral precision for revealing the hypothesized EEG patterns, a rather short cycle width of 4 was chosen.[3] Additionally, cluster-based permutation tests allow for a large range of parameter combinations. As already indicated in the Results, this can substantially influence the spatiotemporal cluster patterns. For example, choosing a lower p-value for the sample-level t-test leads to a more restrictive cluster extent and vice versa. Thus, the exact temporal or spatial extent of a neurophysiological process of interest can therefore not be inferred from cluster shape (Sassenhagen & Draschkow, 2019). Furthermore, the frequency boundaries employed here followed a consensual definition (Pernet et al., 2020), raising the possibility that individually specified peak frequencies may yield divergent results (Donoghue et al., 2021). However, there is no agreement on optimal choice regarding any of the aforementioned parameters and as such, data interpretation is heavily reliant on transparent reporting (Pernet et al., 2018; Pernet et al., 2020). In fact, past studies on mu and beta oscillations during action language processing have implemented varying analysis pipelines (Cuellar & Del Toro, 2017; Moreno et al., 2013; Moreno et al., 2015), limiting comparability across studies.

[3] For the frequency band centered analysis carried out in this study, spectral averaging was conducted, which limited the advantage of higher spectral precision.

Second, changes in EEG power after stimulus presentation may not represent proper differences in oscillatory power in the sense of ERD or ERS (Donoghue et al., 2020). Instead, several phenomena are able to mimic such an effect: The peak-frequency of an oscillation may shift outside the analyzed window of interest, broadband instead of narrowband power may change, or the slope of the $\frac{1}{f}$ power decline may be modulated (Donoghue et al., 2020). In this study however, it was implicitly assumed that any event-related changes in EEG power could be ascribed to real changes in oscillatory power, neglecting the aforementioned alternative explanations. In fact, changes in broadband power or in the $\frac{1}{f}$ slope can yield the impression of oscillatory power changes even in the absence of true oscillations in either the baseline or activation period (Donoghue et al., 2020). Thus, accounting for and explicitly modeling these additional parameters in follow-up studies may yield a more accurate and complete description of the observed EEG dynamics.

Third, EEG source reconstruction was implemented using a template headmodel and standard electrode locations. Thus, the estimated forward model did not fully represent subject-specific biophysical properties. This can reduce the spatial specificity of source-localized neural activity (Akalin Acar & Makeig, 2013). Future studies may incorporate personalized headmodels based on individual MRIs and electrode positions to improve anatomical precision.

Fourth, as already mentioned above, the time period during which semantic processing probably happened could only be derived indirectly from meta-analyses of prior studies (Indefrey & Levelt, 2004; Indefrey, 2011). In addition, due to averaging trials with varying corresponding naming latency, the different processing stages involved in word production may not perfectly align across trials and subjects, even when only considering high NA stimuli. This holds also true when carrying out statistical inferences across participants. In sum, this leads to constrained temporal specificity of observed oscillatory differences.

Fifth, male PD patients were over-represented in this study. This limits the generalizability of the here reported findings to a wider population of patients. Of note, it was not intended to restrict the inclusion of female patients. However, as subjects were recruited from a highly specialized tertiary care center, the total number of possible study participants was limited and albeit attempts to recruit more female patients, a balanced study sample could not be achieved.

5.2.6 Conclusion

This study characterized behavioral and neurophysiological correlates of action language processing in PD. Behavioral results did not indicate an action naming deficit in Parkinson's disease patients off medication and thus are not in keeping with hypotheses H2.1.1 and H2.2.2. This may be partly explained by different education levels between participants included in this study and those assessed in prior reports. On the neurophysiological level, differential oscillatory patterns could be observed: Whereas Parkinson's disease patients off medication showed a mild and transient beta ERS over central to frontal electrodes between 300 ms and 700 ms, a beta ERD was present in HC. This difference in oscillatory power localized to left hemispheric sensorimotor areas and to the right anterior temporal lobe. These findings are therefore partly supporting hypothesis H2.2.1, providing weak evidence that semantic processing of action concepts in the motor system may be altered in PD_{off}. However, as no interaction of subject type and MC could be observed, these findings warrant further investigation. Finally, no changes in oscillatory patterns could be observed when comparing PD_{off} to PD_{on}, which stands in contrast to hypothesis H2.2.2.

6 Summary

Human language capacity is based on temporally coordinated neural activity across distributed brain regions. Although the left hemispheric perisylvian cortex constitutes the core region of language processing, a network of additional sites is further involved. For example, in the healthy brain, semantic access to action concepts has been associated with increased neural activity within frontal motor areas. These findings are complemented by studies demonstrating impaired action language processing in patients with Parkinson's disease, a condition leading to impaired motor control. Therefore, both lines of inquiry suggest an involvement of sensorimotor brain regions in the semantic access to action concepts. However, as the neural underpinnings of the putative action language deficit in Parkinson's disease are unknown, the contribution of motor areas to this phenomenon remains unresolved.

This study therefore aimed at resolving this question by characterizing neurophysiological and behavioral correlates of action language processing in patients with Parkinson's disease. For this purpose, two experiments were carried out. The goal of Experiment 1 was to compile and validate a data set of action pictures for the German language. This part of the study aimed at identifying psycholinguistic variables affecting naming latency in a picture naming task, allowing the selection of matched sets of stimuli in prospective studies. Experiment 2 built upon these data and employed an action naming task and high-density electroencephalography to characterize oscillatory patterns during action language production in both healthy participants and patients with Parkinson's disease. Specifically, this part of the study examined whether action language processing is accompanied with aberrant oscillatory patterns in the mu and beta frequency range over motor cortical areas in the parkinsonian state. Furthermore, the influence of dopaminergic medication on these patterns was assessed.

In Experiment 1, a total of 283 freely available action pictures could be assembled and characterized. The principal variables affecting naming latency describe the agreement in responses across subjects: Less homogeneous response distributions were associated with longer reaction times. Furthermore, word frequency as well as the motor content of the pictures and responses were significant predictors of naming latency. Experiment 2 could not replicate the behavioral action naming deficit in patients with Parkinson's disease when compared to healthy participants. How-

ever, differential neurophysiological correlates of action naming were observed. In contrast to healthy subjects, a transient episode of beta hypersynchronization was present over central to frontal electrodes in Parkinson's disease patients off medication within 300 to 700 ms after stimulus presentation. Cluster-based permutation tests confirmed this difference in oscillatory power and by reconstructing the sources of neural activity it could be localized to the left pre- and postcentral cortex and to the right anterior temporal lobe. Furthermore, subsequent mu power suppression (from 800 ms onwards) was stronger in patients with Parkinson's disease than in healthy controls.

The associations between psycholinguistic variables and naming latency found in Experiment 1 were largely consistent with action naming normative studies carried out in other languages. The data set of 283 action pictures may therefore constitute a valuable resource for future psycholinguistic investigations of action language processing. In Experiment 2, behavioral results were not in keeping with a specific action language deficit in patients with Parkinson's disease, which stands in contrast to prior studies. However, patients included in this study attained a higher level of education as those examined in earlier reports, potentially compensating the hypothesized deficit. On the neurophysiological level though, exaggerated beta power in Parkinson's disease patients showed a spatiotemporal pattern which may reflect aberrant semantic access to action concepts grounded in the motor system: Differential neural activity was partly observed during a previously established time frame for semantic processing and located to brain regions that have been associated with access to action concepts, including the sensorimotor cortex.

In conclusion, this study established a methodological basis for further psycholinguistic studies on action language processing by validating a normative action picture data set for the German language. By applying this data set in an action naming task and recording high density electroencephalography in Parkinson's disease patients and healthy controls, neurophysiological correlates of action language processing were examined. While behavioral results were not in keeping with a hypothesized action naming deficit, differential oscillatory activity in the beta frequency range suggests a contribution of the motor system to altered semantic processing of action concepts in patients with Parkinson's disease.

7 Zusammenfassung

Die menschliche Sprachfähigkeit beruht auf zeitlich koordinierter neuronaler Aktivität in multiplen Hirnregionen. Auch wenn der linkshemisphärische perisylvische Kortex die Kernregion der Sprachverarbeitung darstellt, ist darüber hinaus ein Netzwerk zusätzlicher Areale beteiligt. Im gesunden Gehirn wurde beispielsweise der Abruf von semantischen Konzepten, die für Handlungen kodieren, mit einer erhöhten neuronalen Aktivität in frontalen motorischen Arealen in Verbindung gebracht. Diese Ergebnisse werden durch Studien ergänzt, die eine gestörte sprachliche Verarbeitung von Handlungskonzepten bei Patienten mit Morbus Parkinson zeigen - einer Erkrankung, die zu motorischen Beeinträchtigungen führt. Beide Forschungsrichtungen deuten daher auf eine Beteiligung sensomotorischer Hirnareale am semantischen Abruf von Handlungskonzepten hin. Da jedoch die neuronalen Grundlagen des mutmaßlichen sprachlichen Defizits in Bezug auf Handlungskonzepte bei Morbus Parkinson unbekannt sind, ist der Beitrag des motorischen Systems zu diesem Phänomen bislang unklar.

Die vorliegende Studie zielte daher darauf ab, diese offene Frage durch die Charakterisierung neurophysiologischer und behavioraler Korrelate der sprachlichen Verarbeitung von Handlungskonzepten bei Parkinson-Patienten zu adressieren. Zu diesem Zweck wurden zwei Experimente durchgeführt. Das Ziel von Experiment 1 war es, einen Datensatz von Bildern, auf denen Handlungen dargestellt sind, für die deutsche Sprache zu kompilieren und zu validieren. In diesem Studienteil sollten psycholinguistische Variablen identifiziert werden, die sich auf die Antwortlatenz in einer Bildbenennungsaufgabe auswirken. Dies ist notwendig, um in zukünftigen Studien aufeinander abgestimmte Stimuli auszuwählen. In Experiment 2 wurde dieser Datensatz in einer Bildbenennungsaufgabe eingesetzt und mit hochauflösenden elektroenzephalografischen Ableitungen verbunden. Hiermit konnten oszillatorische Muster, die mit der Verbalisierung von Handlungskonzepten einhergehen, bei gesunden Teilnehmern und bei Patienten mit Morbus Parkinson charakterisiert werden. Insbesondere wurde in diesem Teil der Studie untersucht, ob die sprachliche Verarbeitung von Handlungskonzepten bei Parkinson-Patienten mit abweichenden oszillatorischen Mustern im Mu- und Beta-Frequenzbereich in motorischen kortikalen Arealen einhergeht. Außerdem wurde der Einfluss dopaminerger Medikation auf diese Muster untersucht.

In Experiment 1 konnten insgesamt 283 frei verfügbare Bilder kompiliert und charakterisiert werden. Die Variablen mit den stärksten Effekten auf die Antwortlatenz beschreiben den Grad an Übereinstimmung der abgegebenen Antworten: Inhomogenere Antwortverteilungen waren mit längeren Reaktionszeiten verbunden. Darüber hinaus waren die Worthäufigkeit sowie das Bewegungsausmaß der auf den Bildern dargestellten Handlungen und der assoziierten Antworten signifikante Prädiktoren der Antwortlatenz. Experiment 2 konnte ein Defizit in der Benennung bildlich dargestellter Handlungen bei Patienten mit Morbus Parkinson im Vergleich zu gesunden Teilnehmern nicht replizieren. Es wurden jedoch unterschiedliche neurophysiologische Korrelate während der Bildbenennungsaufgabe festgestellt. Im Gegensatz zu gesunden Probanden war bei Parkinson-Patienten, die nicht unter dem Einfluss dopaminerger Medikation standen, eine vorübergehende Episode von Beta-Hypersynchronisation über zentralen bis frontalen Elektroden in einem Zeitfenster zwischen 300 bis 700 ms nach Stimuluspräsentation zu beobachten. Clusterbasierte Permutationstests bestätigten diesen Unterschied in oszillatorischer Power und durch eine Quellenrekonstruktion neuronaler Aktivität konnte dieser auf den linken prä- und postzentralen Kortex sowie den rechten vorderen Temporallappen lokalisiert werden. Außerdem war eine spätere Suppression oszillatorischer Power im Mu-Frequenzbereich (ab 800 ms) bei Parkinson-Patienten stärker ausgeprägt als bei gesunden Kontrollpersonen.

Die in Experiment 1 gefundenen Zusammenhänge zwischen psycholinguistischen Variablen und der Antwortlatenz deckten sich weitgehend mit Resultaten aus Validierungsstudien zu Bildbenennungsaufgaben in anderen Sprachen. Der Datensatz von 283 Bildern könnte daher eine wertvolle Ressource für zukünftige psycholinguistische Untersuchungen der sprachlichen Verarbeitung von Handlungskonzepten darstellen. Im Gegensatz zu früheren Studien gingen die behavioralen Resultate aus Experiment 2 nicht mit einem Defizit im semantischen Abruf von Handlungskonzepten bei Parkinson-Patienten einher. Allerdings wiesen die Patienten in der vorliegenden Studie ein höheres Bildungsniveau als die Teilnehmer früherer Studien auf, was das angenommene Defizit möglicherweise kompensiert haben könnte. Auf neurophysiologischer Ebene zeigte die Beta-Hypersynchronisation bei Patienten mit Morbus Parkinson hingegen ein Muster, das möglicherweise eine veränderte sprachliche Verarbeitung von Handlungskonzepten im motorischen System widerspiegelt: Abweichende neuronale Aktivität wurde teilweise während eines für semantische Abrufprozesse etablierten Zeitfensters beobachtet und war in Hirnregionen lokalisiert, die mit dem Abruf von Handlungskonzepten in Verbindung gebracht werden, darunter auch der sensomotorische Kortex.

Zusammenfassend hat die vorliegende Studie durch die Validierung eines Datensatzes an Bildern von Handlungen eine methodische Grundlage für weitere psycholinguistische Studien zur sprachlichen Verarbeitung von Handlungskonzepten für die deutsche Sprache geschaffen. Durch die Anwendung dieses Datensatzes in einer Bildbenennungsaufgabe in Verbindung mit hochauflösenden elektroenzephalografischen Ableitungen bei Parkinson-Patienten und gesunden Probanden wurden neurophysiologische Korrelate der sprachlichen Verarbeitung von Handlungskonzepten untersucht. Während behaviorale Ergebnisse nicht mit der Hypothese eines Defizits in der Benennung bildlich dargestellter Handlungen einhergingen, deutet die abweichende oszillatorische Aktivität im Beta-Frequenzbereich jedoch auf einen veränderten semantischen Abruf von Handlungskonzepten in motorischen Hirnarealen bei Patienten mit Morbus Parkinson hin.

Supplementary

	Mean	SD	Min	Max
RT	1780.9	467.2	995.6	3129.4
H	1.7	1.1	0	4.7
$n_{response}$	8.1	5.5	1	29
NA	61.6	25.7	8.1	100
MC_{pic}	4.1	1.6	1.2	7.9
VC	79458.9	43071.8	13948	250804
MC_{word}	4.5	1.3	1.9	7.7
AoA	4.3	0.8	2.8	8.8
IM	6	0.5	4.1	6.7
LE	7.4	1.2	4.9	12
FR	41.9	75.8	0.2	705.7
OLD20	2.1	0.5	1.2	4
		Frequency		
TR				
- intransitive		29.9 %		
- transitive and ditransitive		70.1 %		
RE				
- Non-reflexive and partly reflexive		99.3 %		
- Reflexive		0.7 %		
CO				
- Non-complex		85.4 %		
- Complex		14.6 %		

Table S1: Descriptive statistics of naming latency as well as picture and verb characteristics for 283 action pictures.

	RT	H	$n_{response}$	NA	MC_{pic}	VC	IM	AoA	MC_{word}	LE	FR	OLD20	TR	CO
RT														
H	0.55*													
$n_{response}$	0.53*	0.92*												
NA	-0.5*	-0.95*	-0.82*											
MC_{pic}	0.01	0.02	0.03*	-0.03*										
VC	0.19*	0.31*	0.33*	-0.25*	-0.01									
IM	-0.21*	-0.38*	-0.36*	0.37*	0.15*	-0.15*								
AoA	0.01	0.08*	0.04*	-0.09*	0.07*	-0.13*	-0.27*							
MC_{word}	-0.02	-0.07*	-0.04*	0.06*	0.69*	-0.13*	0.35*	0.08*						
LE	-0.01	0.06*	0.04*	-0.06*	0.01	-0.01	-0.13*	0.19*	-0.03*					
FR	0.05*	0.01	0.03*	0	0	0.07*	-0.02*	-0.57*	0.03*	-0.26*				
OLD20	-0.01	0.04*	0.02	-0.04*	-0.03*	0.08*	-0.11*	0.31*	-0.04*	0.46*	-0.42*			
TR	0.07*	0.1*	0.11*	-0.09*	-0.03*	0.1*	-0.23*	0.06*	-0.01	-0.03*	0.08*	0.09*		
CO	0.06*	0.19*	0.2*	-0.19*	-0.03*	0.07*	-0.26*	0.14*	-0.09*	0.52*	-0.18*	0.33*	0.09*	

Table S2: R-values of repeated measures correlation among all variables under investigation. * = $p < 0.05$ after Benjamini-Hochberg correction.

	Set 1		Set 2	
	MC_{low}	MC_{high}	MC_{low}	MC_{high}
MC_{pic}	2.7	5.4	2.6	6
H	0.4	0.4	0.3	0.5
$n_{response}$	2.8	3.2	2.5	3.5
NA	93.7	93.5	95.6	90.8
VC	53672	61637	66971	68549
IM	6.3	6.3	5.9	6.3
AoA	4.3	4.6	4	4.3
FR	46.9	15.6	65.7	29.9
OLD20	2	2	2.1	2.1
LE	6.8	7.5	7.6	7.4
TR	0.63	0.69	0.69	0.7
RE	0	0	0	0
CO	0	0.07	0.08	0

Table S3: Average picture and verb characteristics of the two subsets of stimuli with high NA analyzed in Experiment 2.

	PD_{off}	PD_{on}	HC
MC_{low}	91.1 (2)	90.4 (2)	90.5 (1.8)
MC_{high}	94.1 (1.4)	95.4 (1.3)	92.7 (2.8)

Table S4: Mean (SEM) response accuracy in % over all subjects. Only stimuli with NA $\geq$ 80 % were considered.

	PD_{off}	PD_{on}	HC
MC_{low}	1448 (80)	1427 (67)	1343 (42)
MC_{high}	1436 (79)	1517 (83)	1337 (57)

Table S5: Mean (SEM) RT in ms over all subjects. Only stimuli with NA $\geq$ 80 % were considered.

Fixed effects				
Variable	Estimate	SE	z	p
(Intercept)	2.97	0.37	8	¡0.001
MC_{low}	-0.23	0.44	-0.53	0.59
PD_{off}	0.13	0.44	0.28	0.78
MC_{low}:PD_{off}	0.02	0.53	0.03	0.97
Random effects				
Group	Variable	Variance	SD	
Picture	(Intercept)	0.84	0.91	
Participant	(Intercept)	0.34	0.58	
Model fit				
R^2(marginal)		0.04		
R^2(conditional)		0.27		

Table S6: Results of the linear mixed effects model 5, evaluating accuracy as a function of MC and disease state. Only stimuli with NA $\geq$ 80 % were considered.

Fixed effects				
Variable	Estimate	SE	z	p
(Intercept)	3.1	0.39	7.9	¡0.001
MC_{low}	-0.23	0.49	-0.47	0.64
PD_{on}	0.48	0.46	1.06	0.29
MC_{low}:PD_{on}	-0.73	0.59	-1.24	0.21
Random effects				
Group	Variable	Variance	SD	
Picture	(Intercept)	0.93	0.97	
Participant	(Intercept)	0.18	0.43	
Model fit				
R^2(marginal)		0.03		
R^2(conditional)		0.28		

Table S7: Results of the linear mixed effects model 6, evaluating accuracy as a function of MC and medication state. Only stimuli with NA $\geq$ 80 % were considered.

Fixed effects					
Variable	Estimate	SE	df	t	p
(Intercept)	-0.09	0.15	81.9	-0.57	0.57
MC_{low}	0.02	0.16	60.1	0.11	0.92
PD_{off}	0.17	0.16	45.3	1.05	0.3
MC_{low}:PD_{off}	-0.04	0.1	800.2	-0.41	0.68
Random effects					
Group	Variable	Variance	SD		
Picture	(Intercept)	0.25	0.5		
Participant	(Intercept)	0.2	0.45		
Residual		0.58	0.76		
Model fit					
R^2(marginal)		0.006			
R^2(conditional)		0.44			

Table S8: Results of the linear mixed effects model 7, evaluating naming latency as a function of MC and disease state.. Only stimuli with NA $\geq$ 80 % were considered.

Fixed effects					
Variable	Estimate	SE	df	t	p
(Intercept)	0.09	0.16	47.94	0.57	0.57
MC_{low}	-0.05	0.157	64.58	-0.33	0.75
PD_{on}	0.12	0.08	811.18	1.62	0.11
MC_{low}:PD_{on}	-0.04	0.11	811.05	-0.35	0.72
Random effects					
Group	Variable	Variance	SD		
Picture	(Intercept)	0.25	0.5		
Participant	(Intercept)	0.27	0.52		
Residual		0.57	0.76		
Model fit					
R^2(marginal)		0.004			
R^2(conditional)		0.47			

Table S9: Results of the linear mixed effects model 8, evaluating naming latency as a function of MC and medication state. Only stimuli with NA $\geq$ 80 % were considered.

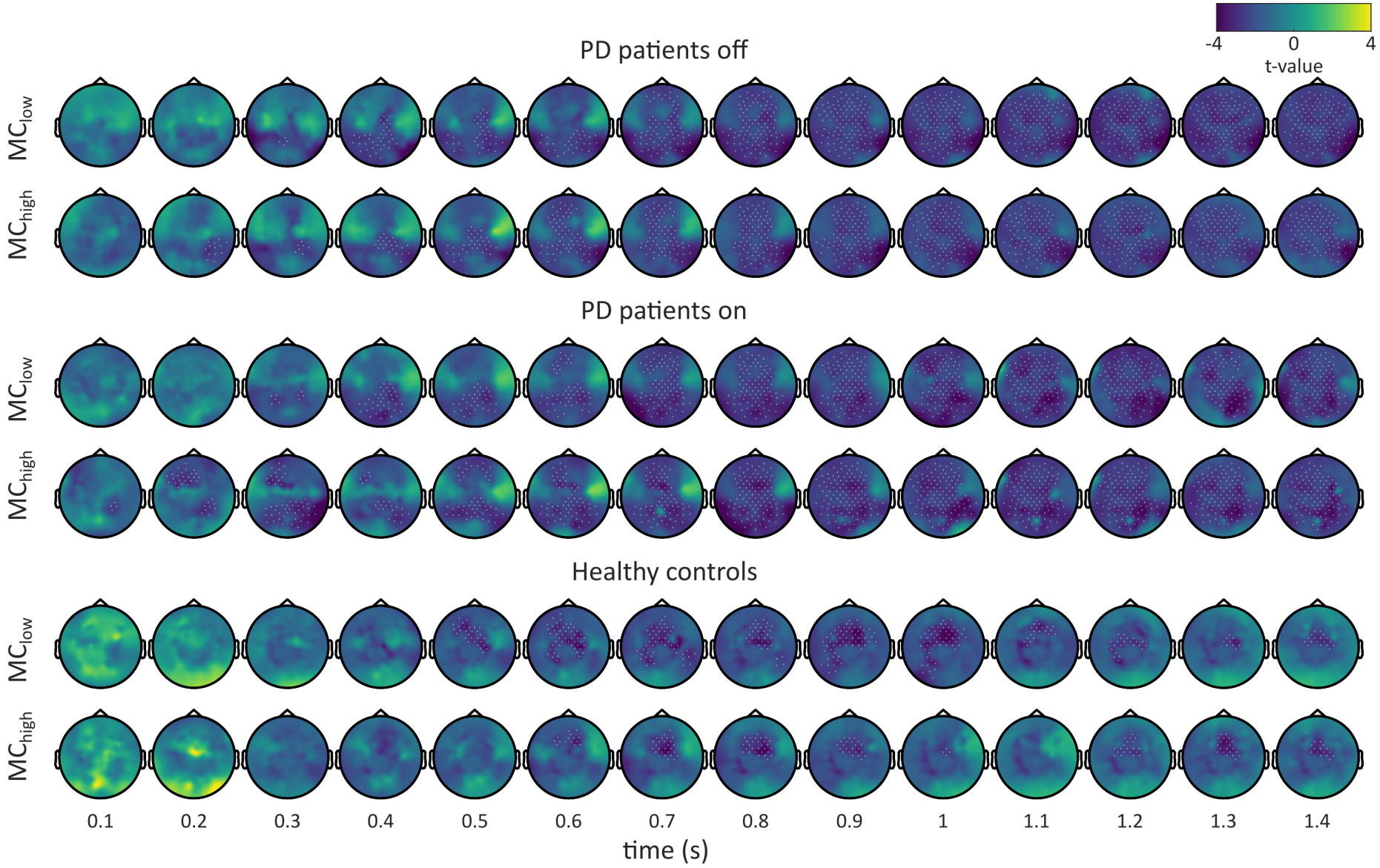

Figure S1: **Mu power activation versus baseline - high NA.** T-values at each time-channel doublet are displayed for the contrast between activation and baseline periods in the mu frequency range for all conditions. Higher t-values indicate increased power, whereas lower t-values mark suppressed power in the activation period as compared to the baseline period across patients. Channels included in clusters associated with significant differences between periods are highlighted by gray dots. Only stimuli with a NA >= 80 % were included.

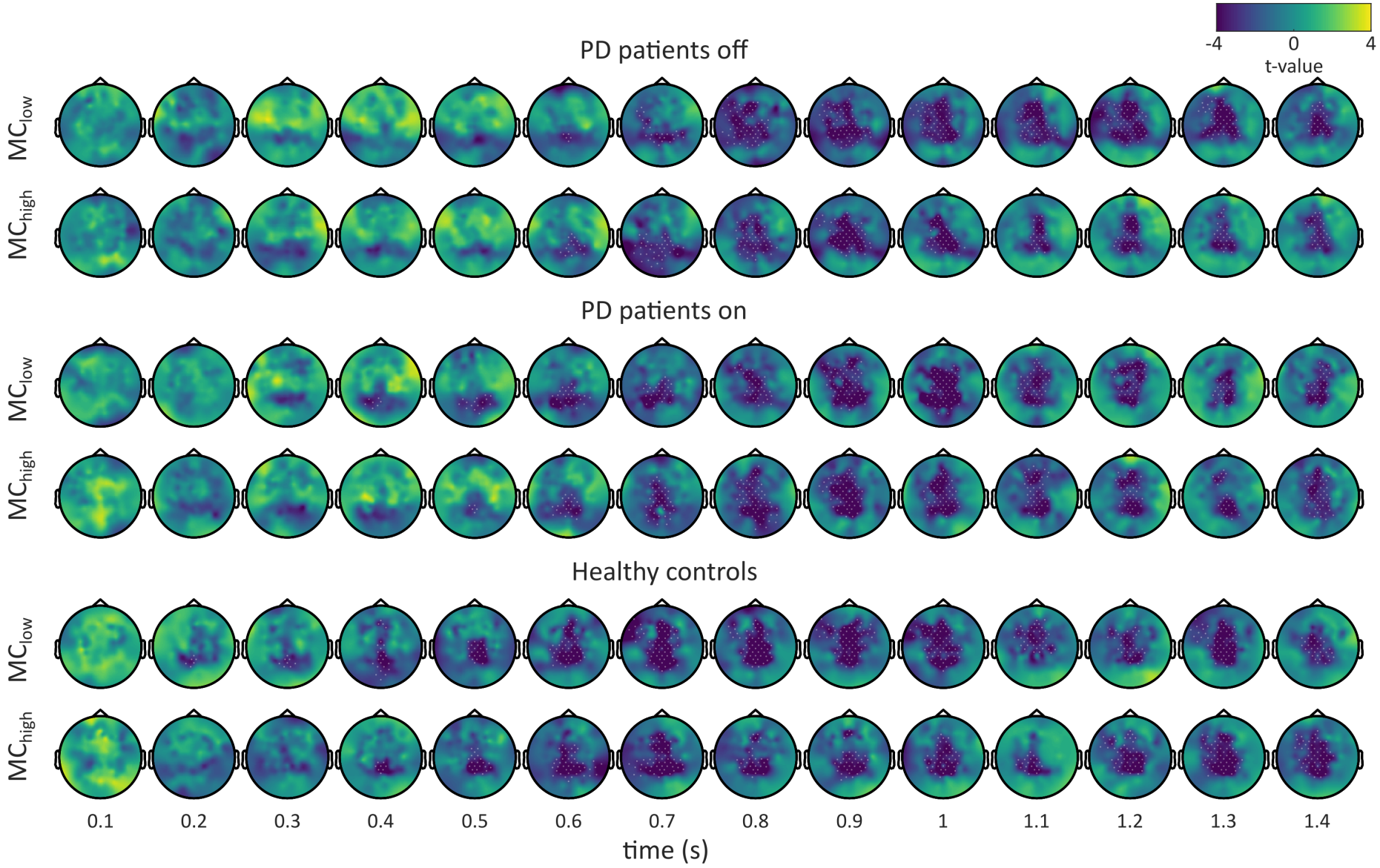

Figure S2: **Beta power activation versus baseline - high NA.** T-values at each time-channel doublet are displayed for the contrast between activation and baseline periods in the beta frequency range for all conditions. Higher t-values indicate increased power, whereas lower t-values mark suppressed power in the activation period as compared to the baseline period across patients. Channels included in clusters associated with significant differences between periods are highlighted by gray dots. Only stimuli with a NA >= 80 % were included.

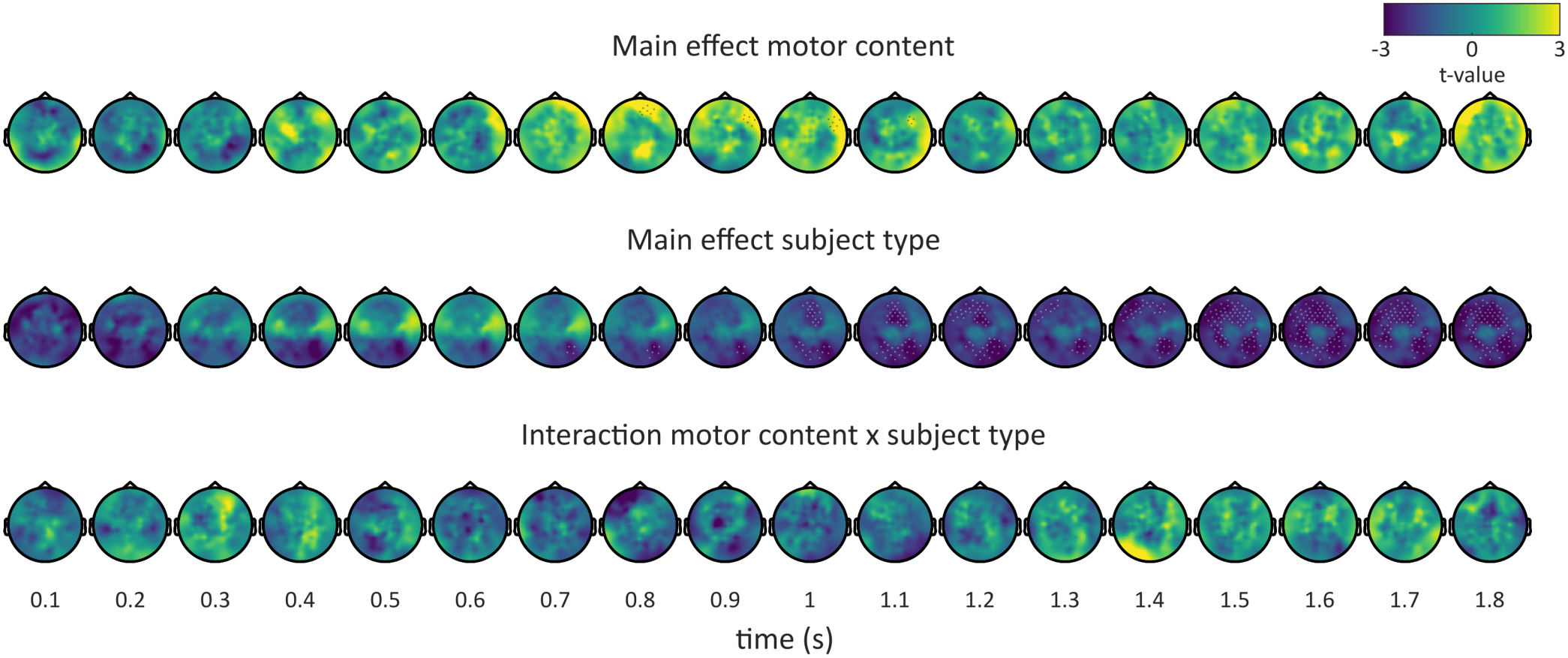

Figure S3: **Mu power PD_{off} versus HC.** T-values at each time-channel doublet are displayed for the contrast between PD patients off medication and healthy controls in the mu frequency range for the main effects of subject type and MC as well as their interaction. Higher t-values indicate 1) greater power for MC_{high} than for MC_{low} (main effect of MC), 2) higher power in PD_{off} than in HCs (main effect of subject type) and 3) higher difference in MC_{high} over MC_{low} power for PD_{off} as compared to HCs (interaction) and vice versa. Channels included in clusters associated with significant differences between conditions are highlighted by gray dots.

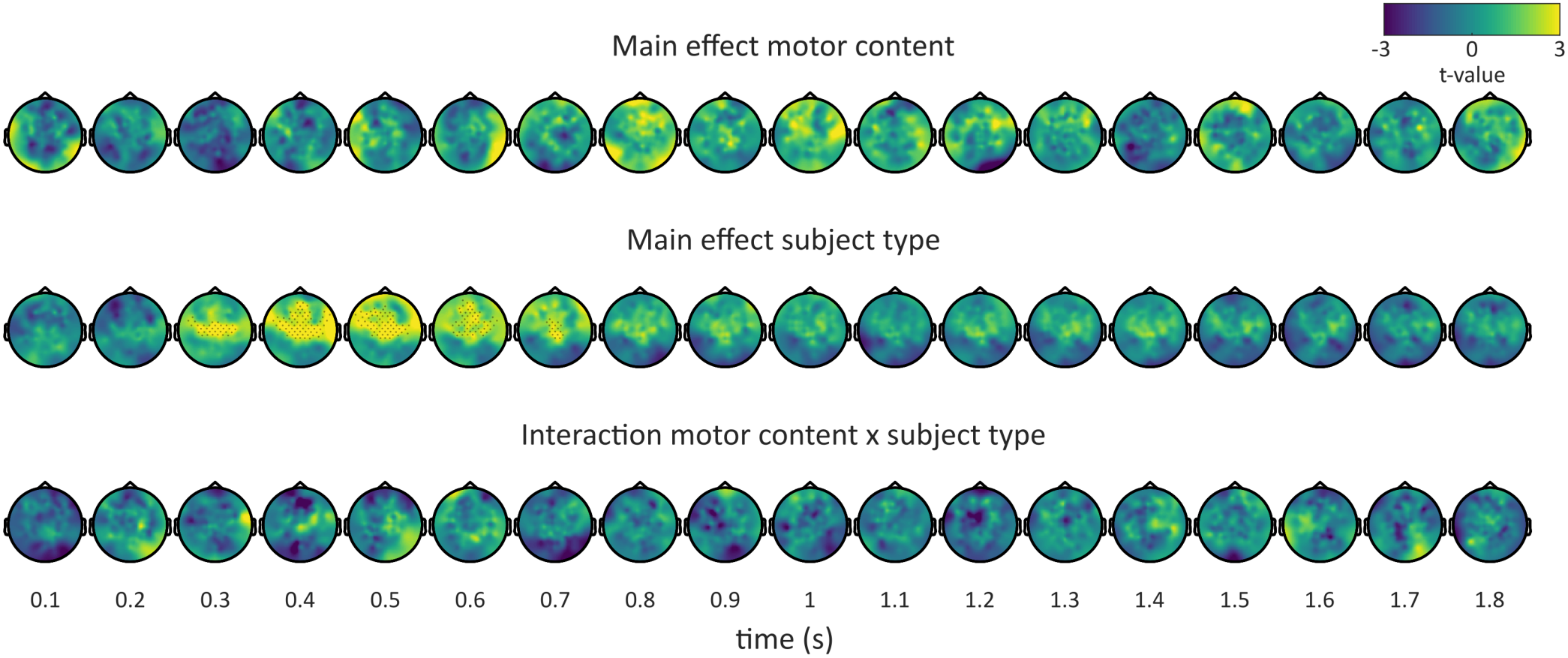

Figure S4: **Beta power PD_{off} versus HC.** T-values at each time-channel doublet are displayed for the contrast between PD patients off medication and healthy controls in the beta frequency range for the main effects of subject type and MC as well as their interaction. Higher t-values indicate 1) greater power for MC_{high} than for MC_{low} (main effect of MC), 2) higher power in PD_{off} than in HCs (main effect of subject type) and 3) higher difference in MC_{high} over MC_{low} power for PD_{off} as compared to HCs (interaction) and vice versa. Channels included in clusters associated with significant differences between conditions are highlighted by gray dots.

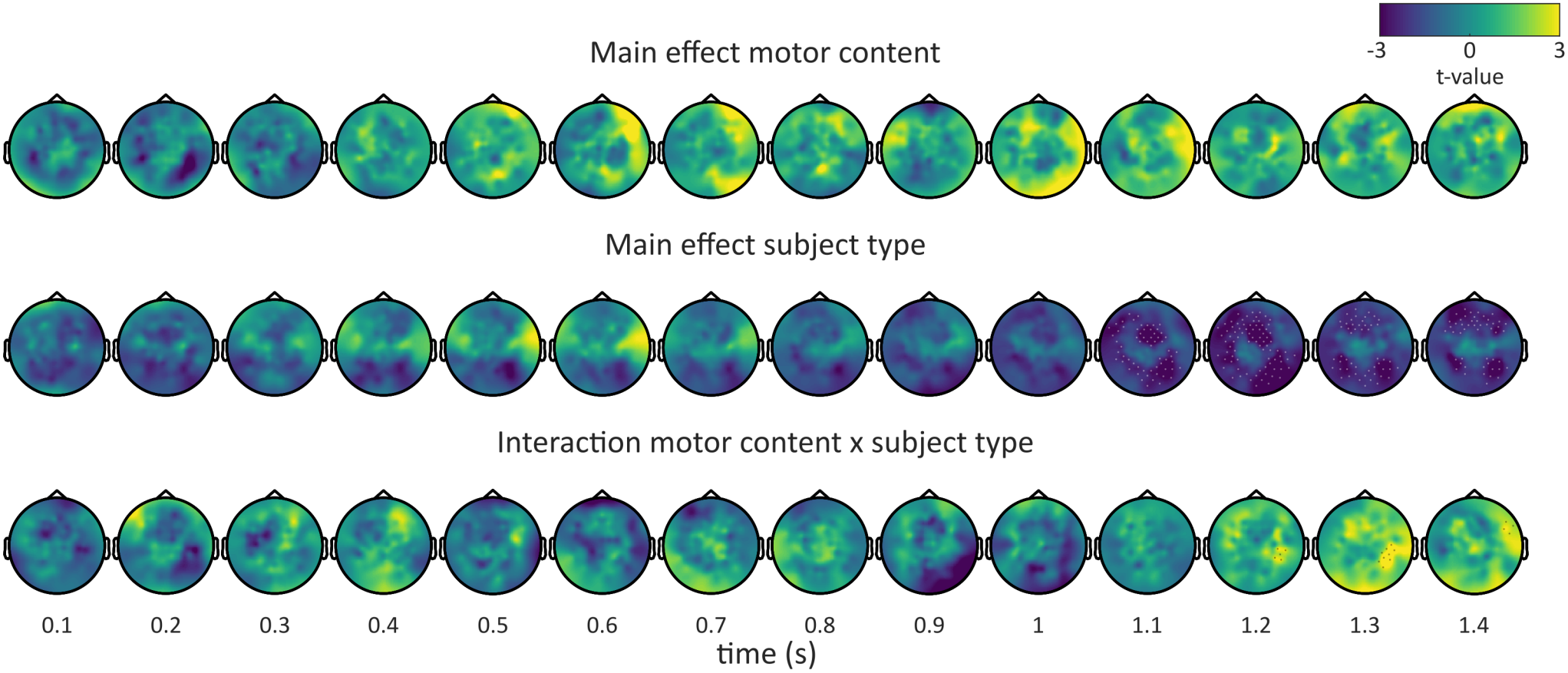

Figure S5: **Mu power PD_{off} versus HC - high NA.** T-values at each time-channel doublet are displayed for the contrast between PD patients off medication and healthy controls in the mu frequency range for the main effects of subject type and MC as well as their interaction. Higher t-values indicate 1) greater power for MC_{high} than for MC_{low} (main effect of MC), 2) higher power in PD_{off} than in HCs (main effect of subject type) and 3) higher difference in MC_{high} over MC_{low} power for PD_{off} as compared to HCs (interaction) and vice versa. Channels included in clusters associated with significant differences between conditions are highlighted by gray dots. Only stimuli with a NA >= 80 % were included.

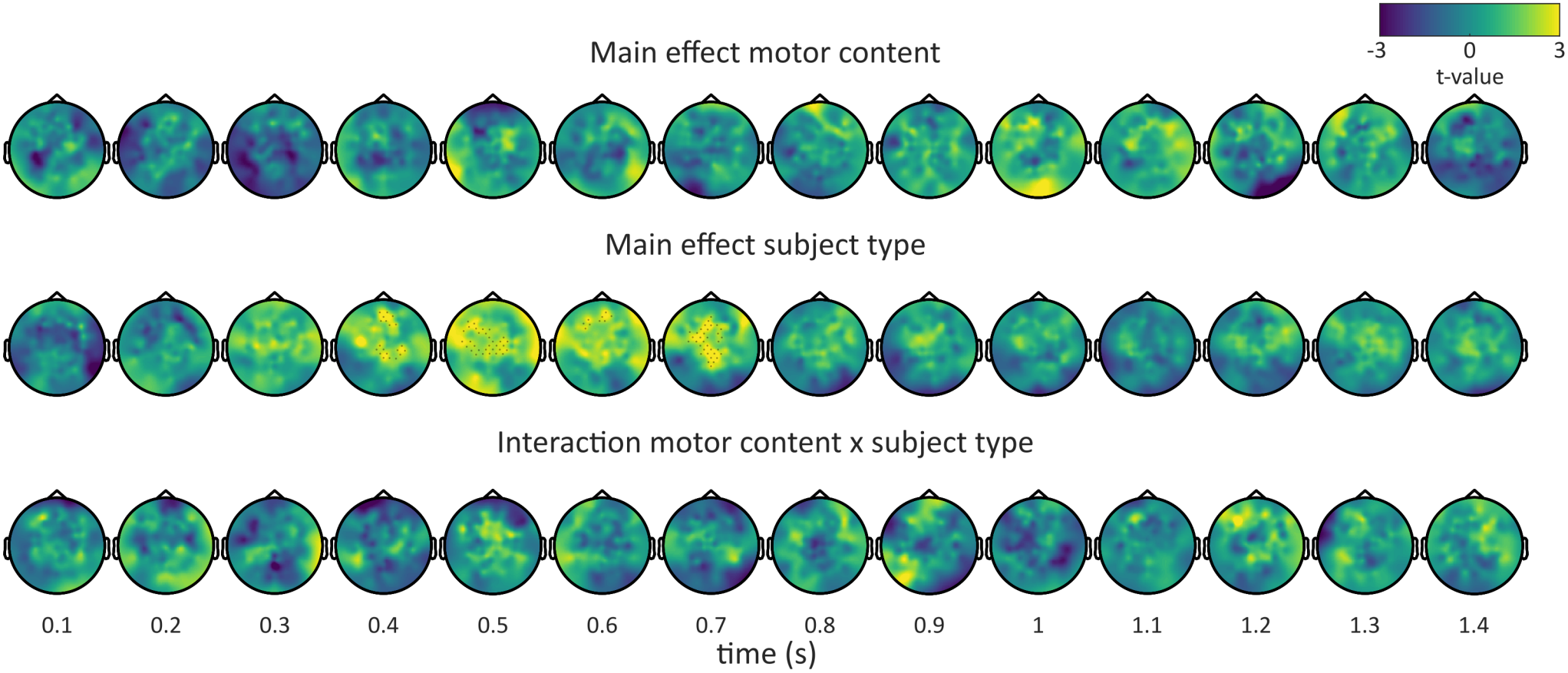

Figure S6: **Beta power PD_{off} versus HC - high NA.** T-values at each time-channel doublet are displayed for the contrast between PD patients off medication and healthy controls in the beta frequency range for the main effects of subject type and MC as well as their interaction. Higher t-values indicate 1) greater power for MC_{high} than for MC_{low} (main effect of MC), 2) higher power in PD_{off} than in HCs (main effect of subject type) and 3) higher difference in MC_{high} over MC_{low} power for PD_{off} as compared to HCs (interaction) and vice versa. Channels included in clusters associated with significant differences between conditions are highlighted by gray dots. Only stimuli with a NA >= 80 % were included.

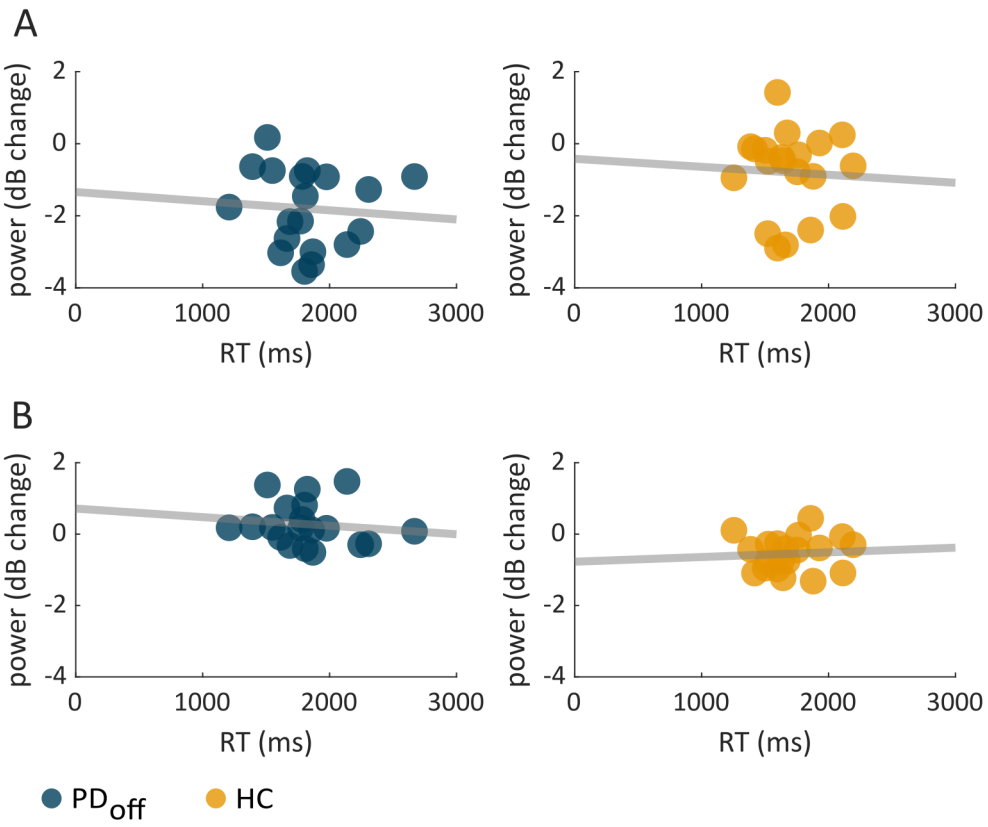

Figure S7: **Association between RT and cluster-averaged power.** Mean baseline-corrected power in the mu (A) and beta (B) cluster for each PD_{off} and HC subject as a function of mean RT per subject. Gray lines indicate linear fits. All $|R| < 0.14$, all $p > 0.58$.

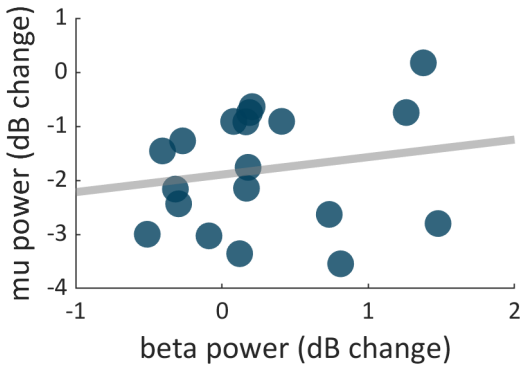

Figure S8: **Association between beta and mu cluster-averaged power.** Mean baseline-corrected power in the mu cluster as a function of mean baseline-corrected power in the beta cluster for each PD_{off} subject. Gray lines indicate linear fits. $R = 0.18$, $p = 0.46$.

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Anhang

1 Lebenslauf

Der Lebenslauf ist aus Datenschutzgründen in der Druckversion nicht enthalten.

2 Verzeichnis der akademischen Lehrenden

Meine akademischen Lehrenden waren in Marburg die Damen und Herren:

Bartsch, Becker, Becker, Bien, Cetin, Czubayko, Daut, Decher, Dodel, Donner-Banzhoff, Engenhart-Cabillic, Fuchs-Winkelmann, Gress, Hertl, Hey, Hofmann, Hoyer, Kill, Kinscherf, Kircher, Kruse, Lill, Lohoff, Mahnken, Maier, Moll, Moosdorf, Mueller, Neff, Neubauer, Nimsky, Pagenstecher, Renz, Ruchholtz, Sachs, Schäfer, Schieffer, Schratt, Sekundo, Stiewe, Teymoortash, Thieme, Timmermann, Vogelmeier, Wagner, Weber, Weihe, Wulf

Meine akademischen Lehrenden waren in London die Damen und Herren:

Chataway, Foltynie, Fox, Kullmann, Leff, Limousin, Manji, Rossor, Trip, Warner

3 Danksagung

Für die stete Hilfe, den langen Atem und die geweckte Begeisterung möchte ich mich an erster Stelle bei Carina Oehrn und Immo Weber mehr als bedanken. Von der ersten Zeile Code bis zum abgeschlossenen Manuskript war es eine lange Reise und ich bin sehr froh darüber, eine erstklassige Betreuung genossen zu haben.

Außerdem wäre diese Arbeit wohl kaum entstanden, wenn ich nicht mit Femke Häußler eine furchtlose Kommilitonin gehabt hätte, mit der ich nicht nur viel Zeit im EEG-Labor, sondern auch am Skattisch verbringen durfte. Die Hochs und Tiefs der Promotion gemeinsam durchlebt zu haben - dafür bin ich ungemein dankbar.

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4 Ehrenwörtliche Erklärung

Ich erkläre ehrenwörtlich, dass ich die dem Fachbereich Medizin Marburg zur Promotionsprüfung eingereichte Arbeit mit dem Titel *Action language processing in Parkinson's disease: Characterization of neuro-oscillatory dynamics and linguistic performance* in der Klinik für Neurologie unter Leitung von Prof. Dr. Lars Timmermann in Zusammenarbeit mit Prof. Dr. Frank Domahs sowie mit Unterstützung durch Dr. Carina Oehrn und Dr. Immo Weber ohne sonstige Hilfe selbst durchgeführt und bei der Abfassung der Arbeit keine anderen als die in der Dissertation aufgeführten Hilfsmittel benutzt habe. Ich habe bisher an keinem in- oder ausländischen Medizinischen Fachbereich ein Gesuch um Zulassung zur Promotion eingereicht, noch die vorliegende oder eine andere Arbeit als Dissertation vorgelegt.

Ich versichere, dass ich sämtliche wörtlichen oder sinngemäßen Übernahmen und Zitate kenntlich gemacht habe.

Mit dem Einsatz von Software zur Erkennung von Plagiaten bin ich einverstanden.

Vorliegende Arbeit wurde in Teilen in folgendem Publikationsorgan veröffentlicht:

Busch, J. L., Haeussler, F. S., Domahs, F., Timmermann, L., Weber, I., & Oehrn, C. R. (2021). German normative data with naming latencies for 283 action pictures and 600 action verbs. Behavior Research Methods.

Johannes Busch

Die Hinweise zur Erkennung von Plagiaten habe ich zur Kenntnis genommen.

Prof. Dr. Lars Timmermann

Prof. Dr. Frank Domahs