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Amisulpride does not influence facial mimicry of happy and angry expressions

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Disclaimer

The following master's thesis is based on a series of experiments conducted within the projects "CS15-003 - "Wanting" and "Liking": The Neurochemical and Neurocognitive Basis of Primary and Social Reward in Humans" (Silani et al., n.d.). This also resulted in the study and accompanying paper "Facial mimicry is not modulated by dopamine D2/3 and opioid receptor antagonism" (Korb et al., 2023). However, the data processed here only refer to a subset of the sample described in that study. The data collection for this subset sample of the study was completed in July 2018, while data collection for the other study continued afterward. I hereby declare that the theoretical examination, as well as the processing and analysis of the data, were carried out independently and autonomously. Some overlaps in theoretical aspects were unavoidable due to the research question, experimental design and the lack of previous studies on dopamine and facial mimicry. Furthermore, artificial intelligence (ChatGPT) was used in the context of this thesis for linguistic corrections and translations.

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1 Theoretical background

Facial expressions are an especially important part of daily life (Kang et al., 2019). Communicating and connecting with others through imitation can be observed even in neonatal nonhuman primates as early as three days old, through rudimentary facial gestures (Ferrari et al., 2006). Human facial expressions and behavior display complex patterns. Mimicking these involves motor-related brain structures as well as emotional processing circuits (Rymarczyk et al., 2018). The neurological disorder Parkinson's disease is characterised by a dysfunctional dopaminergic system and show less mimicking in response to observed facial expressions (Kang et al., 2019). Yet little is known about the underlying mechanisms of neurotransmitters like dopamine. As social interactions are crucial for human beings, it is of great importance to understand those mechanisms.

1.1 Characteristics of Facial Mimicry

Mimicry is the tendency to copy expressions, behaviors, and postures while interacting with or observing others (Chartrand et al., 2005). Facial mimicry (FM) describes the activation of facial muscles congruent to an observed facial expression of emotion (Seibt et al., 2015). These muscular changes, which begin within the first 300–400 ms after stimulus presentation, appear to occur outside conscious awareness (Dimberg & Thunberg, 1998). As they are of very low intensity, it is typically measured with Electromyography (EMG) by using electrodes on facial muscles (Fridlund & Cacioppo, 1986). Facial EMG can be applied to different muscle groups of the face, but most research focusses on two muscles. The *Zygomaticus major* (ZM), which shows increased activation in response to happy expressions and the *Corrugator supercilii* (CS) showing increased activation toward angry expressions (Dimberg & Thunberg, 1998; Fridlund & Cacioppo, 1986; Guntinas-Lichius et al., 2023). As an automatic reaction, which can hardly be suppressed (Dimberg et al., 2002; Korb et al., 2010), it can be measured even with just subliminal presentation of happy and angry facial expressions (Bailey & Henry, 2009). Furthermore, facial mimicry is likely to facilitate the recognition of emotion categories through minor changes like an eyebrow raise (Jack et al., 2014). There are different approaches to explain the contributing processes to emotion experiences and recognition. The functions of FM involve the question of automatic processing as motor copies or motivational driven aspects, like seeking affiliation with others (Coles et al., 2019; Hess & Fischer, 2013, 2022; Wood et al., 2016) in sharing and understanding emotions (Hess & Fischer, 2013; Holland et al., 2021; Lakin et al., 2003).

1.2 Facial Mimicry, behavior and emotion

According to data of many studies, reviews and meta-analysis (Holland et al., 2021; Korb et al., 2014, 2015, 2019, 2020; Kraaijenvanger et al., 2017; Seibt et al., 2015; Wood et al., 2016), a variety of variables impacts facial mimicry. The function and the role in observing, experiencing and understanding expressions of emotion is still of great interest in scientific literature. Regarding underlying mechanisms of FM, neuronal networks and neurochemical basis are of great interest.

1.2.1 Facial Feedback

According to the *facial feedback hypothesis*, automatically mimicking or producing facial expressions generates feedback to different brain areas as motor copies, facilitating or influencing emotional experience and understanding (Davis et al., 2009; Niedenthal, 2007; Niedenthal et al., 2010). This is a concept also referring to other theories like the *embodiment of emotion*, which includes different aspects of expression of emotion (Niedenthal, 2007).

One of the most prominent studies in the facial feedback literature is the pen-in-mouth experiment, in which participants had to hold a pen between the lips or the teeth while watching funny cartoons (Strack et al., 1988). The position of the pen either inhibited (lips) or facilitated (teeth) smiling associated muscle contractions. Subjects reported to find the cartoons to be more humorous in the facilitating than in the inhibiting condition. This is an example, how facial feedback can modulate emotional experiences, when there are external stimuli present (Davis et al., 2009). Those findings could not be replicated in a series of 17 indirect replication studies (Wagenmakers et al., 2016), questioning the basic mechanisms of facial feedback. Noah, Schul and Mayo (2018) examined the monitoring of the subjects via camera as causing factor of the failed replication. Conducting the experiment without a camera led to the facial feedback effect and failed that effect in another condition with subjects being filmed.

Levenson, Ekman and Friesen (1990) conducted experiments, in which participants learned to activate certain muscles under supervision. Instructions based on the Facial Action Coding System (FACS; Ekman & Friesen, 1978) and were performed in front of a mirror. Those configurations resembled facial expressions of emotions (anger, disgust, fear, sadness, happiness and surprise), but without participants knowing the relation between the activated muscles and associated emotions. After 10s they were asked if they had sensations, memories or feelings coming up while holding these expressions. Participants reported associated

emotions above level of chance either with or without having a mirror during trials. With higher configuration accuracy, which was rated independently during the trials, the reports of emotions resembling increased. In this case facial feedback is the sufficient (initiating) factor for an emotional experience without external stimuli present (Davis et al., 2009).

Another question is, if facial feedback is a necessary factor for emotional experiences or the processing of facial expressions and data appears to be inconsistent.

Evidence from a study on bilateral facial paralysis showed no difference in detection, discrimination or the imaging of expression of emotion (Keillor et al., 2002). Subjects with unilateral facial paralysis on the left side displayed inconsistencies in responses to facial expressions of emotion (Korb, Wood, et al., 2016). Based on data that the left side of the face tends to be the more expressive side (Ross & Pulusu, 2013), the authors assumed possible impairments in emotion recognition. Data on participants with Moebius Syndrome, which causes facial paralysis, demonstrated lower autonomic activation in response to video cartoons and recognition impairments with pictures of facial expressions of emotion (Nicolini et al., 2019).

Coles et al. (2019) performed a meta-analysis on a great variety and factors of the facial feedback literature, because of inconsistent data and also controversies like Strack et al. (1988) and Wagenmakers et al. (2016). They found no differences in effect sizes between discrete/specific emotions and dimensional emotion scales (e.g. positivity – negativity) and no specific emotion to have a moderating effect on the facial feedback effect size. No evidence of differing effects because of awareness of manipulation was found. On the Question of subjects being aware of a video recording during trials: little evidence shows a methodological difference associated with different effect sizes and overall effect sizes are small. Despite large effect sizes being found with emotional audio and imagined scenario stimuli, other stimuli types are associated with small effect sizes. Interestingly Coles and colleagues examined the influence of facial movement on emotional experience and affective judgment. Affective judgement is about the emotional characteristics of a stimulus (Coles et al., 2019). Facial movements indicated a significant influence on both, but a larger effect on affective judgment, but was found to be created by publication bias. Furthermore, they could demonstrate facial movements having a larger initiating than moderating effect for emotional experiences.

Another term for the automatic view of mimicry of facial expressions and resulting facial feedback is called the *matched motor hypothesis* (Hess & Fischer, 2013). It refers to emotional mimicry, based on a *perception-behavior-link* and following named the

Chameleon-effect, describing imitating different observed behaviors like postures or facial expressions (Chartrand & Bargh, 1999; Lakin et al., 2003). It also suggests a corresponding motor representation in the observer, while mimicking facial expressions or other aspects of behavior, without involvement of intentions in interactions (Hess & Fischer, 2022). The *Matched motor hypothesis* focusses on aspects of perception and mimicry, where the *facial feedback hypothesis* is the broader approach on facial expressions and their influence on emotion experience and recognition (Coles et al., 2022).

1.2.2 Emotional Mimicry - Mimicry as a social regulator

FM is also described as resulting from the tendency to seek affiliation with others and as a regulator for social encounters (Bourgeois & Hess, 2008; Hess & Fischer, 2013, 2022; Lakin et al., 2003; Seibt et al., 2015). More precisely, mimicry as a social regulator is imitating the affiliative behavior of an observed person to foster affiliation (Hess & Fischer, 2022). While classic facial feedback views suggest that mimicry involves the configuration of certain muscles to initiate or modulate associated emotions and emotional experiences, emotional mimicry describes how people appraise these emotional signals as conveying intentions and information in particular contexts (Hess & Fischer, 2013, 2022). The main goal is to initiate and maintain positive interactions that lead to strong social bonds and interpersonal closeness. Therefore, it is also referred to as *social glue* (Lakin et al., 2003; Meier et al., 2016).

Various factors contribute to FM concerning social regulation. These include the context or setting of an interaction, the relationship between individuals, and characteristics of both the expresser and observer (e.g., attitudes, cooperativeness, fairness, similarity, familiarity, gaze, mood, state, personal traits, and empathy). The type of facial expression further shapes this complex system of influences that impact FM (Hess & Bourgeois, 2010; Hess & Fischer, 2013, 2022; Holland et al., 2021; Seibt et al., 2015).

Likowski et al. (2008) presented different avatars that were introduced with certain traits, such as kind, reserved or deceitful, to manipulate attitudes toward them. The emotional expressions used were happiness, a neutral expression and sadness. They showed that attitudes impacted the degree of FM and also how participants reacted. Happy and sad expressions were mimicked more strongly (especially sad faces) and congruently with the expressions of positively perceived avatars. Negatively received characters were mimicked with less ZM activation toward smiles and even a relaxation (incongruent) of the CS toward sad expressions.

People also tend to show increased FM toward politicians or individuals if they share the same political opinions and interests (Bourgeois & Hess, 2008). Furthermore, subjects show stronger FM toward interaction partners they like, which leads to them being more liked and increases overall closeness in a social situation (Chartrand & Bargh, 1999; Lakin et al., 2003; McIntosh, 2006; Salazar Kämpf et al., 2018; Stel & Vonk, 2010). Individuals are more likely to mimic people who are physically similar to them (Olszanowski et al., 2022), they want to cooperate with (Seibt et al., 2013) and those they consider part of the same social group (Bourgeois & Hess, 2008; Van Der Schalk et al., 2011).

Eye Gaze is particularly associated with stronger ZM response to happy expressions and a stronger CS response to angry and neutral facial expressions (Schrammel et al., 2009).

The observation and mimicry of facial expressions are also influenced by mood state (Likowski, Weyers, et al., 2011). Being in a sad mood decreases facial reactions of the observer, while a happy mood leads to congruent facial mimicry. Subjects in the sad mood condition also showed a tendency to deactivate the CS but not to activate the ZM.

Mauersberger und Hess (2019) examined subjects in a conflict interaction. They demonstrated that interaction quality was higher rated when individuals where mimicked in an affiliative manner despite disagreement, compared to when they were mimicked in an antagonistic manner.

The link between compound empathy (which includes state vs. trait empathy and cognitive vs. emotional empathy) and FM is significant but rather weak. It appears to be moderated by different factors and suggested to be non-direct (see for review: Holland et al., 2021).

In summary, substantial evidence supports the affiliative and regulatory role of FM in social encounters, highlighting its function (Hess & Fischer, 2014; see for review: Hess & Fischer, 2022; Seibt et al., 2015).

1.2.3 Sensorimotor simulation

Evidence highlights the relevance for *facial feedback* and *social regulation* approaches as an interplay function (Orlowska et al., 2023) and is integrated and broadened in *sensorimotor simulation* (Wood et al., 2016). It describes “as-if” loops for simulation and/or recreation of facial expressions that feedback and overlap across somatosensory and motor areas. These can be modulated by social context or motivation to facilitate emotion recognition. Like the *facial feedback hypothesis* it also is theoretically connected to *embodiment of emotion theory* (Niedenthal, 2007). In contrast to *facial feedback* and *social regulation theories*, the absence

of FM measures does not constitute the absence of *sensorimotor simulation* (Wood et al., 2016). According to Wood et al. (2016), automatic FM causes activation of associated emotion systems or at least parts of it and reflects or augments *sensorimotor simulation.* Moreover, as mimicry can hardly be suppressed, its probability and intensity depend on motivational aspects of engaging with and understanding different emotions (Carr & Winkielman, 2014). Therefore, to some extent, it establishes a connection to *social regulation theories* of mimicry. Moreover, the connection between the two theories particularly highlights processing mechanisms in the brain that are relevant to the research question of this thesis.

1.3 Types of facial expressions

One important aspect of FM is to distinguish specific kinds of expressions in terms of the extent to which they are mimicked and why. It is crucial to take certain settings or states into consideration. As they are used in many studies and are especially relevant for this thesis, the focus will be on angry expressions (associated with CS activation) and happy expressions (smiles, associated with ZM activation). Another commonly studied emotional expression in research is sadness, along with, at times, surprise, disgust, and fear (Hess & Fischer, 2013; Topolinski & Strack, 2015).

1.3.1 Anger Expressions

Anger is mimicked more when it comes from an ingroup than an outgroup member (Van Der Schalk et al., 2011), is more mimicked by man (Soussignan et al., 2013) and anger mimicry was found despite subjects trying to voluntarily control their reaction (Dimberg et al., 2002). Some studies found no anger mimicry in general (Rymarczyk et al., 2016) or covert facial mimicry toward angry expressions (Wingenbach et al., 2020). Incongruent reactions to angry expressions appear in romantic relationships, in which anger is mimicked with smiling (Häfner & IJzerman, 2011). CS activation appears to be complex in interpretation. It has been found to correlate with moral judgements about harm (Cannon et al., 2011) and to be activated due to experiencing surprise (Topolinski & Strack, 2015). In summary, mimicry of anger exists, but shows rather mixed and incongruent results.

1.3.2 Happy Expressions

Strong smiles are judged as more authentic (Korb et al., 2014), indicate the need for social connection (Martin et al., 2017), capture attention (Campos et al., 2015) and can be described as rewarding stimuli, which increase ZM response (Sims et al., 2012). FM of smiles can be a congruent reaction to happy expressions, even in a competitive setting (Seibt et al., 2013). ZM reaction also occurs as an incongruent response to sad expressions in a cooperative setting (Likowski, Mühlberger, et al., 2011).

In summary, smiles can have affiliative, rewarding and even dominant qualities (Niedenthal et al., 2010), which can be characterized by different patterns of facial muscle configurations (Rychlowska et al., 2017). The rewarding nature of smiles is also related to higher dopamine levels being associated with positive affect (Ashby et al., 1999; Wiswede et al., 2009).

1.4 Neuronal and hormonal framework

1.4.1 The mirror neuron system and related brain areas

Some studies suggest that the neural basis of FM lies in the mirror neuron system (MNS) (Likowski et al., 2012; Niedenthal, 2007). These findings are closely related to the assumptions of *embodiment theories* and *sensorimotor simulation*, which include FM processes as feedback mechanisms in various brain areas, influenced by different factors and contexts (Wood et al., 2016).

Likowski et al. (2012) identified different areas of the MNS involved in FM. For facial expressions in general, these areas include the inferior frontal gyrus, the inferior parietal lobule, the middle temporal gyrus, the superior temporal sulcus, the precentral gyrus, the cerebellum, the hippocampus, the amygdala and also caudate, putamen, insula, and the posterior cingulate cortex. Additionally, for happy expressions, the middle cingulate cortex, the parahippocampal gyrus, the precuneus and the supplementary motor area are also involved, while for sad expressions the precuneus is activated.

Rymarczyk, Żurawski, Jankowiak-Siuda and Szatkowska (2018) further concluded motor and emotion processing areas – namely, MNS associated regions and their extensions – play a pivotal role in processing observed expressions of emotions. Specifically, during the simultaneous activation of the ZM and orbicularis oculi (OO) muscles in response to happy expression, the putamen, the nucleus accumbens and the globus pallidus were implicated.

This provides evidence that regions of the basal ganglia, including the caudate (as mentioned above) are linked to FM.

Korb et al. (2019) additionally demonstrated activation of the medial prefrontal cortex during incongruent trials, such as observing happy expressions associated with loss or angry expressions associated with winning.

In an rTMS study, inhibition of the primary motor cortex (M1) led to a reduction in smile mimicry among female participants, whereas no effect was observed in male participants (Korb et al., 2015). This suggests differences in the underlying neural circuitry of FM for women and men.

Prefrontal areas and the basal ganglia are also regions affected in Parkinson's disease (see section 1.5.6 Parkinson's disease) (Costello et al., 2022; Surmeier et al., 2014).

1.4.2 Hormones

Hormones such as oxytocin, cortisol and testosterone can impact these neural structures, modulate processing of emotion and influence facial emotional processing (Kraaijenvanger et al., 2017; Romero-Martínez et al., 2021).

Korb, Malsert, Strathearn, Vuilleumier and Niedenthal (2016) administered oxytocin intranasal and found increased FM compared to the placebo group – especially toward angry facial expressions of infants, while other data using the same drug administration could not show a direct effect (Trilla et al., 2020). Oxytocin appears to improve the ability of decoding emotional facial expressions accurately (Romero-Martínez et al., 2021).

Cortisol appears to reduce activity of the ZM and facial mimicry toward smiles (Nitschke et al., 2020) and testosterone leads to a general decrease of facial mimicry (Hermans et al., 2006).

1.4.3 Neurotransmitter

When it comes to neurotransmitters, little is known about their direct influence on FM. Blocking the mu-opioid system with naltrexone leads to a strengthened response of the CS to happy facial expressions (Meier et al., 2016), but administration of morphine, as an agonist of the mu-opioid system, showed no significant effect on facial mimicry (Massaccesi et al., 2022).

Serotonin has been found to influence the modulation of emotional facial expression processing in participants with autism spectrum disorder (Daly et al., 2012).

Additionally, in a study with ±3,4-Methylenedioxymethamphetamine (MDMA), which is known to have serotonergic effects, Hoshi, Bisla and Curran (2004) concluded a possible effect on the recognition of fearful expressions.

1.5 Dopamine

Dopamine (DA) plays a crucial role in various brain functions, including executive function, emotion, motor control, learning, and reward. It operates through three major pathways: nigrostriatal, mesocortical and mesolimbic (Ko & Strafella, 2012).

For this thesis, the mesolimbic pathway is of particular interest. Originating from the ventral tegmental area (VTA), it projects through the nucleus accumbens to the limbic system and frontal cortex and is associated with reward and emotion (Haber & Knutson, 2010).

In clinical pharmacology, DA receptors are well studied targets (see section 1.5.5 Amisulpride). A variety of disorders and conditions – such as Parkinson's disease, depression, bipolar disorder, schizophrenia, restless leg syndrome, hyperprolactinaemia, pituitary tumours, hypertension, gastroparesis, nausea and erectile dysfunction – are associated with the dopaminergic system (Beaulieu et al., 2015).

1.5.1 Dopaminergic reward processing

As a transmitter of the reward system, DA plays a role in motivational aspects in eliciting "wanting" – namely, generating effort to gain specific rewards (Berridge, 1996; Berridge & Kringelbach, 2015).

Evidence for dopaminergic reward processing in humans is scarce and primarily derived from research on Parkinson's disease (PD), as well as pharmacological studies.

PD patients exert more effort to receive a reward depending on whether they are ON or OFF dopaminergic medication (Chong et al., 2015). Treatment with DA replacement therapy for DA dysregulation syndrome, particularly DA stimulation through D2/D3 receptor agonists, has been shown to increase "wanting," contributing to addictive behaviors (Callesen et al., 2014; Kim et al., 2013; O'Sullivan et al., 2009). These behaviors fall under impulsive control and repetitive behavior disorders, including hypersexuality, compulsive shopping, pathological gambling, and compulsive eating.

In contrast, blocking D2/D3 receptors with Amisulpride dampens cue-induced responding with a Pavlovian-instrumental transfer task and reduces reward impulsivity with a delay discounting task (Weber et al., 2016).

A study on testosterone administration and reward anticipation using a monetary incentive delay task found a significant influence of testosterone on the ventral striatum's response to potential reward or no-reward signals. The authors suggested that testosterone enhances preparation for the effort – ergo, "wanting" - in reward anticipation via mesolimbic DA pathways (Hermans et al., 2010).

1.5.2 Dopamine and facial mimicry

No direct link between DA and FM has been established yet, but existing data suggest possible connections.

For example, spontaneous mimicry of happy expressions is associated with positive valuation (i.e., reward value) in the orbitofrontal cortex (Hsu et al., 2018), which has extensive connections to the dopaminergic system (Kringelbach, 2005).

Some connections have been identified through pharmacological approaches that found how possible dopaminergic mechanisms influence responses to emotional, social, and behavioral stimuli (Schuster et al., 2022; Wardle & De Wit, 2014) or have been suggested by research on PD (Argaud et al., 2016; Arioli et al., 2022; Gray & Tickle-Degnen, 2010; Livingstone et al., 2016; Prenger & MacDonald, 2018). The relevant findings will be presented in the following section.

1.5.3 MDMA

Research on MDMA is not only relevant for serotonergic studies, as MDMA also increases the release of dopamine (Schenk & Highgate, 2021; Wei et al., 2018). In a fMRI Study, Bedi, Phan, Angstadt and De Wit (2009) found an increased response to positive social stimuli, highlighting the connection between dopaminergic reward circuitry and social affiliative behavior, as well as enhanced social reward processes. Research on the link between MDMA and facial mimicry indicates that higher intensity is required to perceive angry expressions, while responses to happy expressions are increased in female participants (Wardle & De Wit, 2014).

1.5.4 Haloperidol

Schuster et al. (2022) conducted an experiment to examine the influence of haloperidol, a D2 receptor antagonist, on emotion recognition from dynamic motion cues of

whole bodies. They used a working memory (WM) task as a proxy for DA synthesis capacity. Low WM span is associated with lower DA synthesis capacity and different behavioral responses to dopaminergic modulation compared to higher WM span. Their findings showed that participants with low WM span improved in emotion recognition, but no main effect of haloperidol on emotion recognition was found.

1.5.5 Amisulpride

Amisulpride is a benzamide derivative and an atypical antipsychotic primarily used for treating schizophrenia (Curran & Perry, 2001). At low doses (50-300mg/day), it facilitates DA transmissions by blocking presynaptic D2/D3 autoreceptors, whereas at high doses (400-800mg/day) it blocks postsynaptic DA D2/D3 receptors, reducing DA transmission (Rosenzweig et al., 2002). In general, Amisulpride is a highly selective antagonist of DA D2/D3 receptors, with a preference for the limbic system (Schoemaker et al., 1997). Amisulpride is rapidly absorbed, reaching peak plasma concentrations after approximately one hour, followed by a second peak after 3-4 hours (Bergemann, 2004). In healthy individuals, a single high dose (400mg) does not affect selective and divided attention, motor activity, sensory-motor coordination, or vigilance (Rosenzweig et al., 2002). As mentioned earlier, blocking D2/D3 receptors with Amisulpride has been shown to decrease motivation to gain immediate rewards in two different tasks (Weber et al., 2016).

1.5.6 Parkinson's disease

Parkinson's disease (PD) is primarily characterized by a dysfunctional dopaminergic system, caused by the degeneration of dopamine-producing cells in the striatum (Surmeier et al., 2014). As a result, PD it is associated with impairments in reward processing and motivation, as well as deficits in prefrontal areas, the basal ganglia, and the midbrain, leading to impairments in initiation and control of motor, cognitive and emotional functions (Costello et al., 2022; Surmeier et al., 2014). Some of these brain regions have been implicated in FM and emotional activation (Likowski et al., 2012; Prenger & MacDonald, 2018), and individuals with PD tend to show reduced activity in parts of the MNS (Arioli et al., 2022).

Additionally, PD is associated with diminished or absent facial expressions due to basal ganglia dysfunction, which causes bradykinesia—slowness of movement—as well as difficulties in initiating, planning, and executing both voluntary and spontaneous movements (Jankovic, 2008).

A meta-analysis by Gray and Tickle-Degnen (2010) found a link between PD and impaired recognition of emotions from facial and prosodic expressions.

Although research on PD and FM is scarce, existing studies report similar findings. Argaud et al. (2016) found that participants with mild PD showed no activation of the ZM and OO muscles in response to joyful and neutral expressions, but did exhibit FM toward expressions of anger. In an emotion recognition task using avatars displaying joy, anger, and neutral expressions, participants with PD had reduced accuracy in recognizing joyful and neutral expressions.

Similar results were shown in subjects with mild-moderate PD (Livingstone et al., 2016). PD participants activity of the ZM in response to happy expressions and medial frontalis (MF) activity in response to sad expressions was reduced and delayed. Additionaly, a negative correlation was observed between the amplitude of these activations and response times for emotional identification ratings.

These findings are further supported by studies on voluntary and spontaneous FM (Kang et al., 2019). In both conditions, ZM activity in PD subjects was significantly lower than in the control group.

Overall, subjects with PD show reduced or absent facial muscle activation, particularly in the ZM in response to happy expressions. Furthermore, they exhibit difficulties with accurate emotion recognition, with greater difficulty recognizing anger (44%) compared to happiness (27%) (Argaud et al., 2018).

2. Research Question and Hypothesis

Facial mimicry (FM) refers to the tendency to copy observed facial expressions, primarily in a congruent manner. It likely facilitates emotion recognition and understanding. While FM can modulate emotional experience or even be sufficient for it, it is not a necessary factor, as studies on individuals with bilateral facial paralysis suggest. These descriptions are based on data supporting the idea that FM originates as an automatic process of facial feedback.

Research also suggests that FM results from the motivational drive to seek affiliation in social interactions and regulate emotions. From this perspective, various factors—such as attitudes, mood, gaze, personal characteristics, and more—influence the extent of FM. Additionally, the type of facial expression plays a crucial role. While research on FM in response to angry expressions shows mixed results, studies on happy expressions and smiles present more consistent findings. Smiles are particularly significant in terms of motivation and affiliation-seeking due to their attention-grabbing, rewarding, and socially connective nature.

According to the theory of *sensorimotor simulation*, particularly neural mechanisms are important for FM and consequently emotion recognition. The mirror neuron system (MNS) appears to serve as the neural basis of FM, involving motor areas. Some regions are innervated by DA and affected in patients with PD.

Consequently, neurotransmitter systems are essential for understanding FM. Studies on Parkinson's disease (PD) and pharmacological interventions suggest that dopaminergic pathways—particularly the mesolimbic pathway, which is involved in reward processing—play a role in FM.

Taken together, facial expressions, especially smiles, can be described as rewarding stimuli. Positive affect is related to higher dopamine levels and spontaneous mimicry observing happy expressions is associated with positive valuation (i.e., reward value) in the orbitofrontal cortex which is connected with the dopaminergic system.

Additionally, blocking DA receptors has been shown to dampen motivation for obtaining rewards.

Research on PD and MDMA suggests a possible dopaminergic influence on FM, particularly in the form of reduced and/or delayed zygomaticus major (ZM) activation in response to happy and angry expressions in individuals with PD. MDMA studies indicate

increased ZM responses to happy expressions in women, though higher intensity is required to perceive angry expressions.

Despite these findings, no direct connection between DA and FM has been firmly established. The aim of this thesis is to examine the relationship between the dopaminergic system and FM. Specifically, this thesis investigates how blocking DA D2/D3 receptors with Amisulpride—a highly selective D2/D3 receptor antagonist—affects facial responses when observing emotional facial expressions.

Based on the literature on FM, facial expressions, dopamine and dopaminergic reward processing presented above, the following hypothesis are proposed:

1) Changes in muscle activity over time (FM) are expected in response to facial expressions of emotion in both experimental conditions.
   a) Observing angry facial expressions in the condition *HappyToAnger* is expected to increase activity in CS and decrease activity in the ZM.
   b) Happy facial expressions in the condition *AngerToHappy* are expected to increase activity in the ZM and decrease activity in the CS.
2) There is a difference in FM of CS between experimental groups when responding to happy (*AngerToHappy*) and angry expressions (*HappyToAnger*).
3) Blocking D2/D3 receptors with Amisulpride is expected to affect FM in response to happy (smiles) (*AngerToHappy)* and angry expressions (*HappyToAnger*) by diminishing ZM activation compared to placebo group.

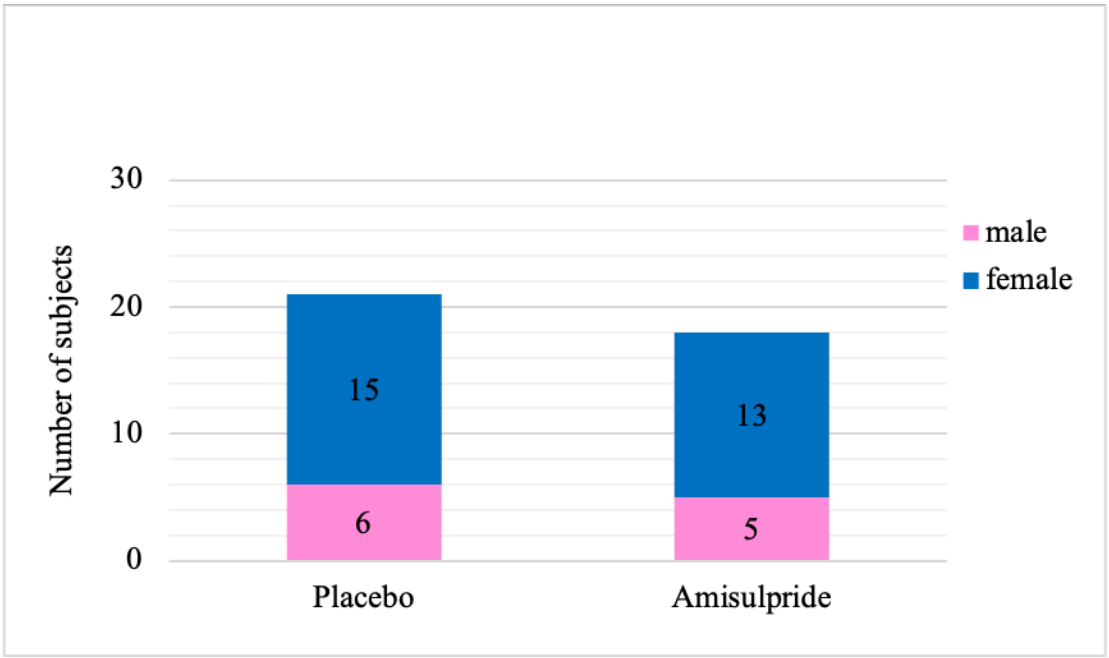
3. Methods

3.1 Participants

A total of 39 healthy participants between 19 and 35 years old ($M = 23.38$, $SD = 4.40$) were recruited between February and July 2018 and randomly assigned to experimental groups. One participant was excluded because of technical problems with the EMG. All 39 remaining subjects reported to be right-handed, smoking less than five cigarettes per day, stating no current or former abuse of drugs and no history of psychiatric or neurological disorders. The study was conducted in a randomized, double-blind placebo-controlled manner as a pharmacological intervention. In a between-subjects design, 18 (5 male) participants received 400 mg of Amisulpride (a dopamine D2/3 receptor antagonist) or 21 (6 male) participants mannitol, a placebo (see Figure 1). The procedure was approved by the ethics committee of the Medical University of Vienna. It followed the regulations of the Declaration of Helsinki (World Medical Association, 2013). Participants received an informed consent form to sign, along with explanations by a medical doctor regarding possible side effects and the anonymous nature of their participation in the study. In the end subjects received a monetary compensation of at least 90 € for the whole study of "Facial responses of adult humans during the anticipation and consumption of touch and food rewards" (Korb et al., 2020).

Figure 1

Sample sizes of experimental groups, divided by gender.



3.2 Study "Facial responses of adult humans during the anticipation and consumption of touch and food rewards"

The study on the influence of DA on FM was a part of a greater set of experiments at the Department of Psychiatry and Psychotherapy, Medical University of Vienna (General Hospital "Allgemeines Krankenhaus AKH", Währinger Gürtel 18-20, 1090 Vienna). The first session of the study included a comprehensive pre-screening, while the second session being the experiment regarding the role of the dopamine and opioid systems in the "wanting" and "liking" of social (touch) and primary (food) rewards.

During the pre-screening session, participants completed a computer task measuring stimulus-reward associations. This was followed by a detailed medical and psychological evaluation conducted by a doctor, which included electrocardiography, blood tests, and psychiatric assessments to determine suitability for the study. Participants with no pathological findings were invited to the second session, scheduled to occur within 1 to 60 days, lasting approximately 6 hours.

The second session began with drug administration, followed by a psychophysiological study assessing the "wanting" and "liking" of primary and social rewards. This session also included the facial mimicry task and three shorter computer-based experiments: a probability weighting task, a reward-learning task (repeated), and a working memory task. The participants' health status and any side effects were monitored by the doctor around 180 and 300 minutes after drug administration.

3.3 Procedure FM task

FM of dynamic happy and angry facial expressions was measured with facial EMG of the CS and ZM, starting around 4h after drug/placebo intake (the second plasma peak of Amisulpride).

Participants sat between 50-60cm in front of a computer screen, watching 4 sets of 24 videos each set (5.12s duration time). Each video showed a happy expression morphing gradually into an angry one and vice versa. Participants were instructed to press a button as accurately as possible to indicate the moment the initial emotional expression was no longer visible. Responses were recorded using a key on a keyboard. Movie clips continued to play in full, regardless of participants' responses. Each movie clip was preceded by a white fixation cross displayed on a black background for an average duration of 2.5 seconds (ranging from 2 to 3 seconds). After the clip ended, perceived offset time (in milliseconds from the start of the

clip) was displayed for one second on a black screen. If no response was recorded, the text "No response" appeared for the same duration.
Sets were then repeated four times, for a total of 96 trials shown in semi-random order (three successive stimuli with the same emotion max), in two blocks of 48 trials. In the beginning, subjects completed 4 practice trials.

3.4 Stimuli

The stimuli were described and successfully used before in other studies (Halberstadt & Niedenthal, 2001; Korb et al., 2015; Korb, Malsert, et al., 2016; Niedenthal et al., 2001). They based on photos of 10 faces (5 male) of happy and angry expressions, applying a morphing software (Morpheus Photo Morpher, version 3.17). Happiness and anger show distinguishable patterns of FM, as well as ZM and CS muscles are distanced enough from another (Korb, Malsert, et al., 2016). The Videos of faces morphing from angry to happy expressions constituted the condition AngerToHappy (AH) and videos of happy expressions morphing into angry expressions the condition HappyToAnger (HA). See figure 2 for an example with 5 frames, the first and the last frame are the initial and ending expressions for condition AH or vice versa. Dynamic expressions were used because of advantages compared to static images. Those advantages include higher ecological validity, presenting dynamic stimuli as more natural and also more powerful (Sato et al., 2015), especially eliciting stronger responses in the ZM (Rymarczyk et al., 2016).

Figure 2

Sequence of frames in the condition AngerToHappy (AH).

3.5 EMG recording and processing

Facial areas were degreased, cleaned and peeled with alcohol, water, and an abrasive paste. Ag/AgCl electrodes - 4 mm inner diameter and filled with highly conductive water electrolyte gel (SignaGel, Parker Laboratories) - were attached bipolarly on the left CS and ZM muscles (Fridlund & Cacioppo, 1986). A ground electrode was placed on the upper forehead and a reference electrode on the mastoid bone behind the left ear. EMG signals were amplified with a samplerate of 1200 Hz and impedances below 20kOHM using a g.USBamp amplifier (g.tec Medical Engineering GmbH). Then data was processed in Matlab R2014b (www.themathworks.com), partly using EEGLAB toolbox (Delorme & Makeig, 2004), running with a 20 Hz to 400 Hz bandpass filter, a 50 Hz notch filter, rectified and smoothed with a 40 Hz low-pass filter.

Data was prepared in Windows Excel 16.78.3 2019 and Statistical Analysis was performed with SPSS 29.0.0.0.

The EMG scores were obtained from 0.5 s before to 5 s after stimulus onset, calculated as a percentage of the baseline values (an average of the 500 ms preceding stimulus onset), averaged over 1-second intervals (5 in sum per trial). FM is measured as changing muscle activity over 5 1-second intervals.

Trials were excluded from analyses if their average values exceeded 2 SD's above or below the mean for that subject and muscle, or if their peak values deviated by more than 2 SD's from the average peak. Using the same criteria, trials with outlier values in the baseline period were also removed. EMG scores with artefacts were also excluded from analysis. In total, 18.12% of trials were removed and both groups (Amisulpride: M = 17,72, SD = 4,56; Placebo: M = 18,46, SD = 3,92) did not differ significantly in percentage of excluded trials (t(33,84) = -0,54, p = 0,596).

3.6 Data analysis

Windows Excel 16.78.3 2019 was used for preparation of the data and creating diagrams. Statistical Analysis was performed with SPSS 29.0.0.0. Because of using dynamic imagery as stimuli, FM measures are regarded and processed as changing muscle activity over time.

For testing the first hypothesis, a two-factor repeated measures ANOVA was conducted with Time (5 measurement points: CS1–CS5, ZM1–ZM5) and Muscle (CS vs.

ZM) as within-subject factors, and Condition (AngerToHappy vs. HappyToAnger) as a between-subjects factor.

To examine hypothesis 2 and 3, whether Amisulpride (Amisulpride vs. Placebo) influenced muscle activity over time (FM), separate repeated measures ANOVAs were conducted for CS and ZM in each condition (AngerToHappy, HappyToAnger). The within-subjects factor was Time (5 measurement points: CS1–CS5 or ZM1-ZM5), and the between-subjects factor Drug Group (Amisulpride vs. Placebo). In all cases corrected test values of Greenhouse-Geisser were used, because of significant Mauchly's test for sphericity.

The threshold of significance α was determined to $p = 0.05$.

4. Results

1.) Changes in muscle activity over time (FM) are expected in response to facial expressions of emotion in both experimental conditions.

The two-factor repeated measures ANOVA with Time (5 measurement points: CS1–CS5, ZM1–ZM5) and Muscle (CS vs. ZM) as within-subject factors, and Condition (AngerToHappy vs. HappyToAnger) as a between-subjects factor revealed a significant main effect of Time ($F(2.30, 174.60) = 5.93$, $p = .002$, $\eta_p^2 = .072$), ergo muscle activity changed significantly over time.

The main effect of Muscle was not significant ($F(1, 76) = 0.033$, $p = .856$, $\eta_p^2 = .000$), so overall activation levels of CS and ZM did not differ across all time points.

Furthermore, there was no significant main effect of Condition ($F(1, 76) = 1.028$, $p = .314$, $\eta_p^2 = .013$). Therefore, overall muscle activity did not differ between the two experimental conditions.

The analysis could show a significant Time × Muscle interaction ($F(2.23, 169.18) = 17.74$, $p < .001$, $\eta_p^2 = .189$), indicating that CS and ZM followed different activation patterns over time.

Additionally, there were differences in activation between CS and ZM depending on the experimental condition with a significant Muscle × Condition interaction ($F(1, 76) = 20.23$, $p < .001$, $\eta_p^2 = .210$).

Most notably, the three-way interaction of Time × Muscle × Condition was significant ($F(2.23, 169.18) = 9.58$, $p < .001$, $\eta_p^2 = .344$). This indicates that the activation patterns of the two muscles not only changed over time and differed between muscles but were also influenced by the experimental condition (see Table 1 and 2 for means and standard deviations of the CS and ZM). Mean value plots were created (see Figure 3 and 4) to illustrate the specific temporal progression of muscle activity in the two conditions. Where CS activation is decreasing in AH and increasing in HA, ZM activation is increasing in AH and decreasing in HA.

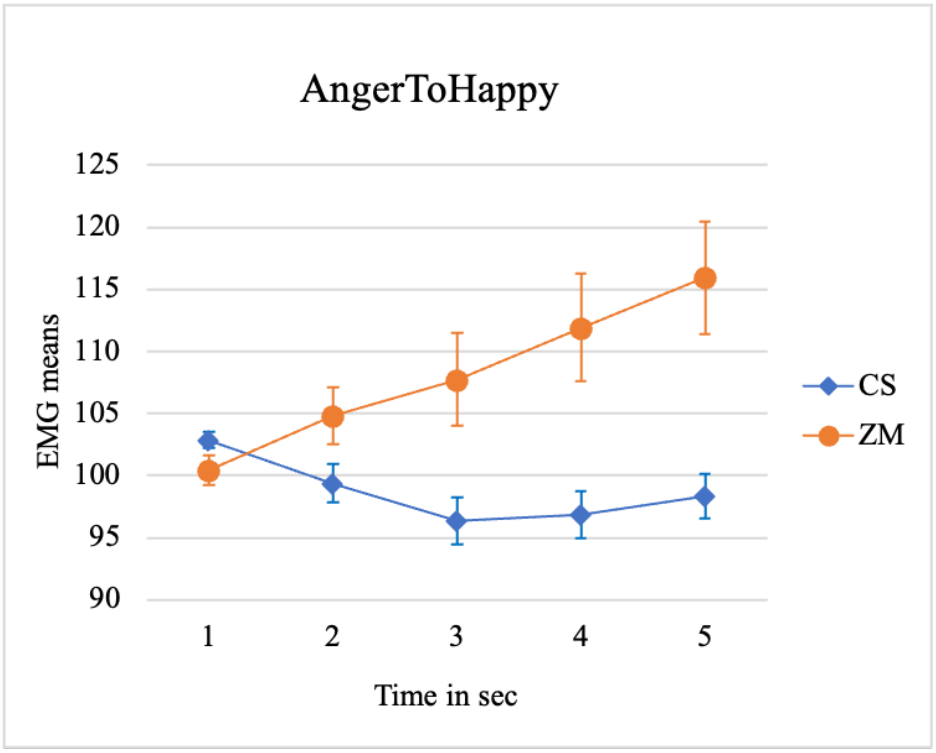
Table 1

Means and standard deviations for both muscles and five measurement points in AH.

	Muscle			
	CS		ZM	
Time	***M***	***SD***	***M***	***SD***
1	102,87	3,96	100,47	7,55
2	99,38	9,76	104,85	14,32
3	96,35	11,97	107,75	23,48
4	96,85	12,02	111,91	27,00
5	98,34	11,09	115,95	28,36

Figure 3

Mean Data of CS and ZM in condition AH.



Note. Error bars show standard error of the means.

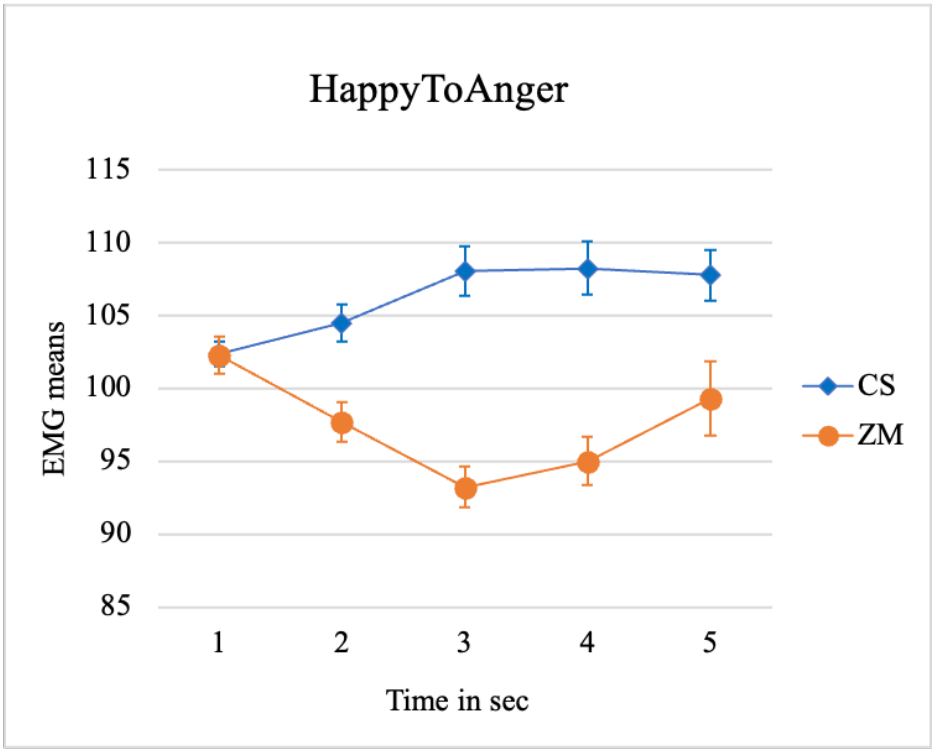
Table 2

Means and standard deviations for both muscles and five measurement points in HA.

	Muscle			
	CS		ZM	
Time	*M*	*SD*	*M*	*SD*
1	102,39	5,25	102,29	8,03
2	104,49	7,87	97,68	8,60
3	108,05	10,62	93,22	8,76
4	108,27	11,27	95,01	10,46
5	107,79	10,88	99,30	16,01

Figure 4

Mean Data of CS and ZM in condition HA.



Note. Error bars show standard error of the means.

2.) There is a difference in FM of CS between experimental groups when responding to happy (AngerToHappy) and angry expressions (HappyToAnger).

AngerToHappy

A significant main effect of Time was found ($F(1.92, 70.87) = 8.78$, $p < .001$, $\eta_p^2 = .203$), thus CS activity changed significantly over time in both groups.

The main effect of Drug Group was not significant ($F(1, 37) = 0.015$, $p = .903$, $\eta_p^2 = .000$). No overall difference in CS activity between the Amisulpride and Placebo group could be shown.

Temporal changes in CS activity did not differ between drug groups ($F(1.92, 70.87) = 0.32$, $p = .716$, $\eta_p^2 = .009$; no significant Time × Drug Group interaction for AH).

In Table 3 and Figure 5 CS activity for experimental Groups in condition AH is displayed.

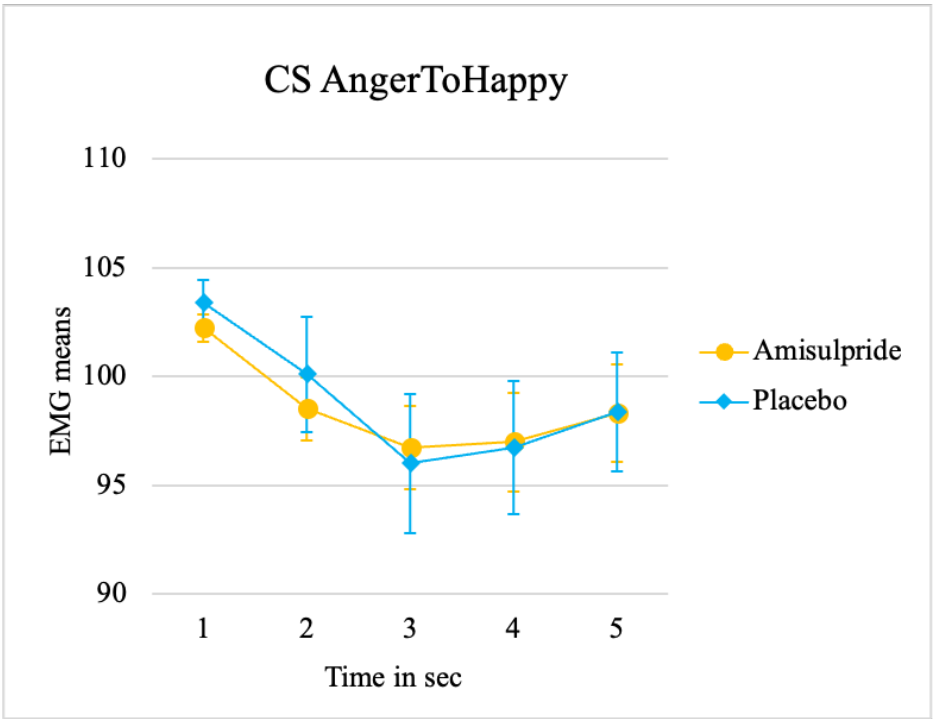
Table 3

CS means and standard deviations for DrugGroups and measurement points in AH.

	DrugGroup			
	Amisulpride		**Placebo**	
Time	***M***	***SD***	***M***	***SD***
1	102,24	2,63	103,40	4,82
2	98,52	6,10	100,12	12,17
3	96,74	8,14	96,02	14,69
4	97,00	9,54	96,72	14,04
5	98,32	9,52	98,36	12,51

Figure 5

CS activity over 5 measurement points for experimental groups in AH.



Note. Error bars show standard error of the means.

HappyToAnger

CS activity changed significantly over time in condition HA ($F(1.63, 60.24) = 15.76$, $p < .001$, $\eta_p^2 = .294$; main effect of Time).

CS activity did not differ significantly between experimental groups ($F(1, 37) = 0.01$, $p = .946$, $\eta_p^2 = .000$; no main effect of Drug Group)

The Time × Drug Group interaction was also not significant ($F(1.63, 60.24) = 1.25$, $p = .289$, $\eta_p^2 = .032$) consequently FM of CS did not differ between the Amisulpride and Placebo groups in the HappyToAnger condition. See Table 4 and Figure 6 for mean values in both groups.

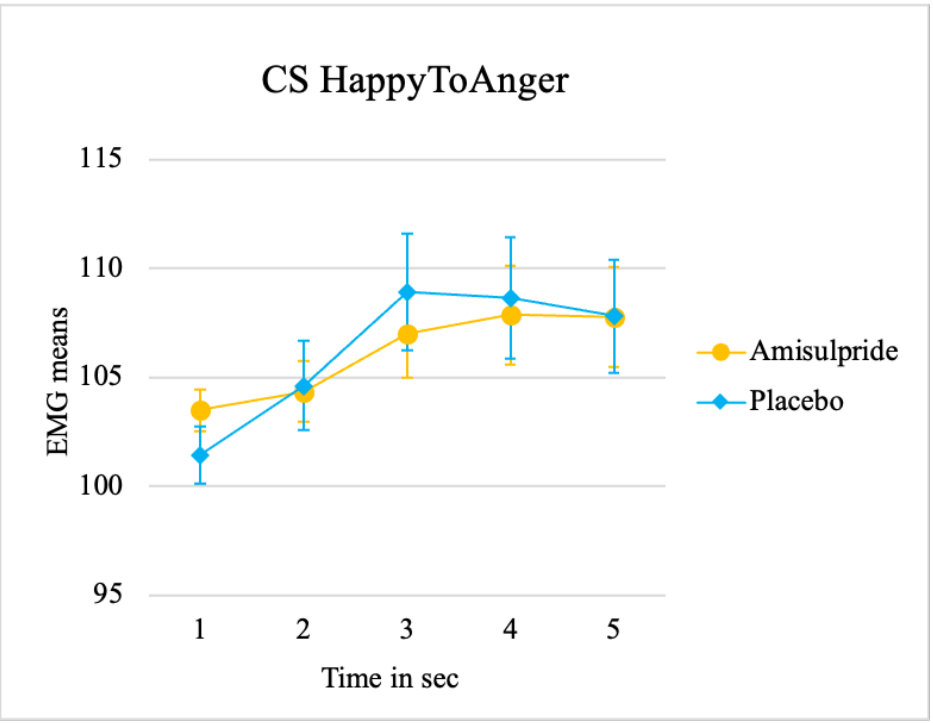
Table 4

CS means and standard deviations for DrugGroups and measurement points in HA.

	DrugGroup			
	Amisulpride		Placebo	
Time	***M***	***SD***	***M***	***SD***
1	103,49	3,97	101,44	6,07
2	104,35	5,96	104,61	9,36
3	107,00	8,54	108,94	12,27
4	107,85	9,57	108,63	12,78
5	107,77	9,79	107,80	11,97

Figure 6

CS activity over 5 measurement points for experimental groups in HA.



Note. Error bars show standard error of the means.

3) Blocking D2/D3 receptors with Amisulpride is expected to affect FM in response to happy (smiles) (AngerToHappy) and angry expressions (HappyToAnger) by diminishing ZM activation compared to placebo group.

AngerToHappy

For ZM activity, a significant main effect of Time was observed ($F(2.20, 81.69) = 7.17$, $p = .001$, $\eta_p^2 = .177$). Thus, ZM activity varied significantly across the five time points.

In contrast to the presumption, no general differences between the Amisulpride and Placebo groups were found ($F(1, 37) = 0.000$, $p = .985$, $\eta_p^2 = .000$; no significant main effect of Drug Group).

Thus, results did not depict ZM activity patterns differing between groups, showing no significant interaction of Time × Drug Group ($F(2.20, 81.69) = 0.69$, $p = .519$, $\eta_p^2 = .019$) (See Table 5 and Figure 7 for FM of ZM in condition AH).

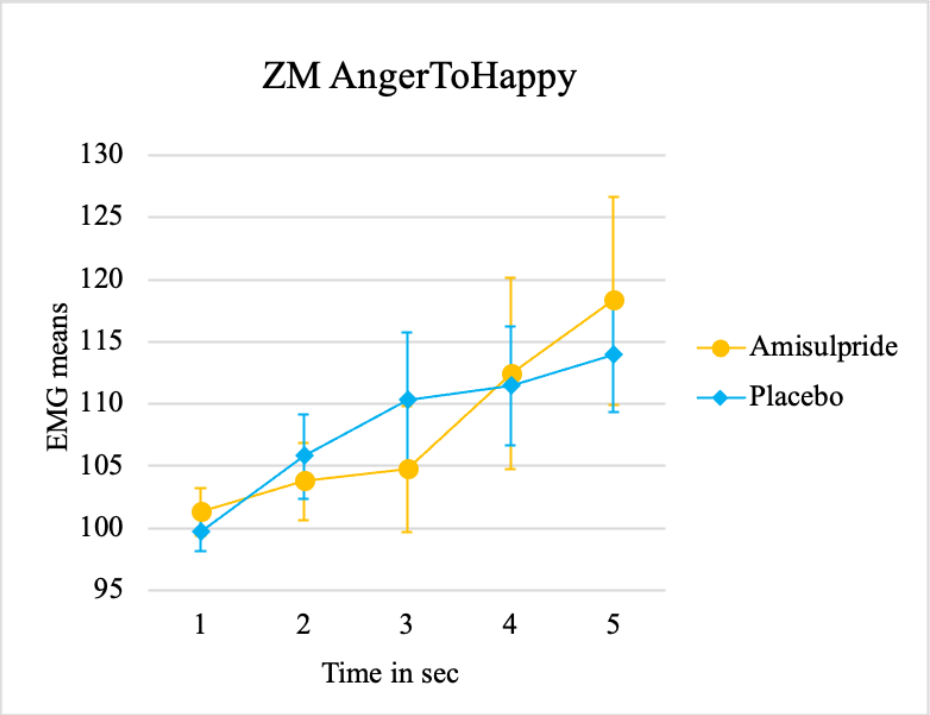
Table 5

ZM means and standard deviations for DrugGroups and measurement points in AH.

	DrugGroup			
	Amisulpride		**Placebo**	
Time	***M***	***SD***	***M***	***SD***
1	101,34	8,14	99,73	7,12
2	103,76	13,23	105,79	15,46
3	104,80	21,49	110,28	25,31
4	112,42	32,62	111,48	21,92
5	118,32	35,48	113,92	21,17

Figure 7

ZM activity over 5 measurement points for experimental groups in condition AH.



Note. Error bars show standard error of the means.

HappyToAnger

In condition HA, observed ZM activity changed significantly over the five measurement points ($F(2.20, 81.27) = 5.70$, $p = .004$, $\eta_p^2 = .189$; significant main effect of time).

The main effect of Drug Group was not significant ($F(1, 37) = 1.13$, $p = .294$, $\eta_p^2 = .030$). The experimental groups showed no significant difference in general ZM activation.

ZM activity patterns over time did not differ between drug groups ($F(2.20, 81.27) = 0.13$, $p = .972$, $\eta_p^2 = .004$; Time × Drug Group interaction not significant). Mean results of ZM activation of both groups can be seen in Table 6 and Figure 8.

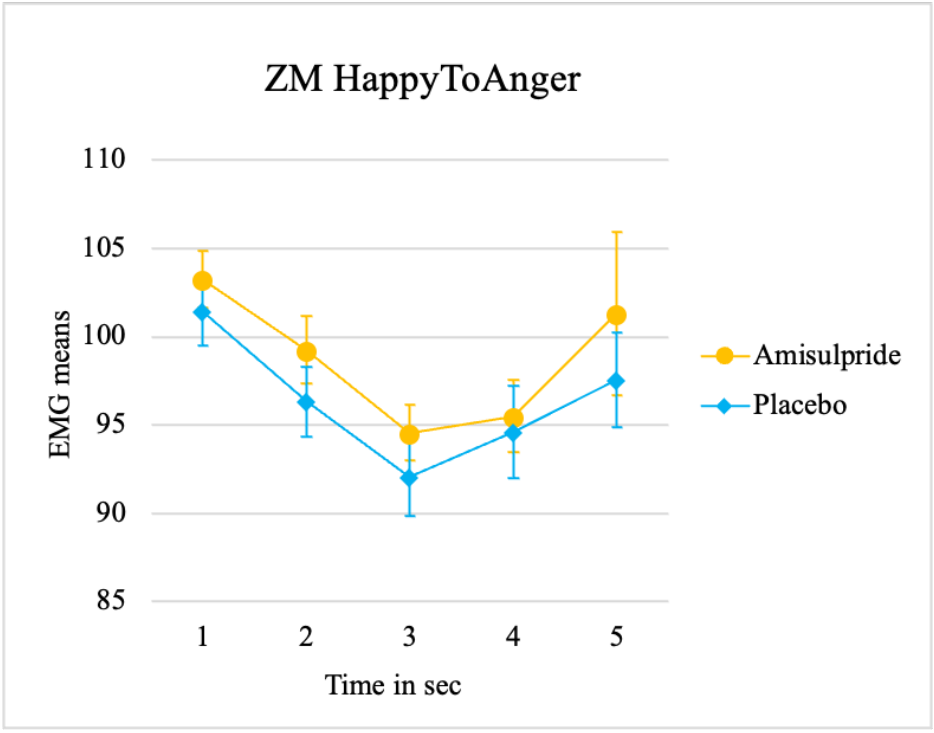
Table 6

ZM means and standard deviations for DrugGroups and measurement points in HA.

	DrugGroup			
	Amisulpride		Placebo	
Time	***M***	***SD***	***M***	***SD***
1	103,27	6,86	101,45	9,00
2	99,25	8,05	96,34	9,01
3	94,56	6,73	92,07	10,21
4	95,51	8,65	94,59	11,99
5	101,31	19,73	97,58	12,23

Figure 8

ZM activity over 5 measurement points for experimental groups in condition HA



Note. Error bars show standard error of the means.

5. Discussion

FM refers to the tendency to mimic facial expressions when interacting with or observing other human beings. The main goal of this thesis was to investigate whether blocking dopaminergic D2/D3 receptors with Amisulpride influences this response. One key function of the DA system, particularly the mesolimbic pathway, is its role in the reward system of the brain, specifically in motivational aspects of reward-seeking behavior – referred to as *wanting* – the extent of effort in gaining certain rewards. In social interactions, smiles can be perceived as rewarding stimuli. Based on this assumption, blocking DA receptors involved in reward processing should modulate FM responses to rewarding emotional facial expressions.

To test this, subjects were divided into two groups: one receiving Amisulpride (a highly selective DA D2/D3 receptor antagonist) or Mannitol (a Placebo). Facial muscle activity – specifically the CS and ZM – was measured using facial EMG while participants observed videos of faces morphing either from an angry to happy expression (AH) or from a happy to an angry expression (HA).

Results indicated that experimental condition significantly influenced how CS and ZM activity changed over time. Additionally, ZM and CS exhibited response patterns across time. A significant three-way interaction (Time × Muscle × Condition) suggested that the experimental conditions affected the temporal development of muscle activity. This finding confirmed the first hypothesis, which established FM in response to the stimuli, allowing for further investigation into potential differences between drug groups. Specifically, the study examined whether CS activation differed between groups in both AH and HA conditions, and whether ZM activation is diminished in AH and HA conditions in the Amisulpride group.

Across all conditions, the main effect of Time was significant, supporting presence of FM responses in both drug groups for both muscles. However, Amisulpride had no significant influence or modulating effect on FM. No significant differences were found in CS activation between groups, nor was evidence of reduced ZM activation in the Amisulpride group compared to Placebo group in the AH condition or any difference in the HA condition.

Overall, these results suggest that while muscle activity (CS and ZM) changes significantly over time, Amisulpride administration did not significantly impact facial muscle activity or its temporal development in either experimental condition (AngerToHappy or HappyToAnger).

5.1 Interpretation of the results

1.) Changes in muscle activity over time (FM) are expected in response to facial expressions of emotion in both experimental conditions.

Both the ZM and CS responded differently over time while subjects observed facial expressions of emotion, which is consistent with previous EMG research (Bailey & Henry, 2009; Dimberg et al., 2002). CS activity increased when observing happy expressions morphing into angry expressions and decreased while watching video sequences of angry expressions morphing into happy ones. In contrast, ZM activity followed an inverse pattern – increasing activity observing angry turning into happy expressions and decreasing watching facial expressions turning from happy to angry ones.

These findings confirm that the stimulus material reliably evoked facial muscle responses (activation and relaxation), consistent with studies using these materials before (Halberstadt & Niedenthal, 2001; Korb et al., 2015; Korb, Malsert, et al., 2016; Niedenthal et al., 2001). Establishing these response patterns, was a necessary requirement before investigating possible effects of Amisulpride on FM.

2.) There is a difference in FM of CS between experimental groups when responding to happy (AngerToHappy) and angry expressions (HappyToAnger).

Results did not display any significant difference in CS activation between drug groups in either condition. One explanation is that angry expressions, unlike happy ones, may not constitute rewarding cues of interactions and therefore remain unaffected by dopaminergic modulation.

In general, studies on FM of angry expressions show more inconsistent data (Häfner & IJzerman, 2011; Rymarczyk et al., 2016; Topolinski & Strack, 2015; Van Der Schalk et al., 2011).

Additionally, research on hormones, other drugs and neurotransmitter systems like oxytocin suggest that various factors modulate FM in response to angry expressions (Korb, Malsert, et al., 2016). For instance, blocking the mu-opioid system enhances CS responses to happy expressions (Meier et al., 2016), while morphine, a mu-opioid agonist, does not seem to influence FM (Massaccesi et al., 2022).

Regarding dopaminergic modulation, findings from MDMA and PD studies suggest, that higher intensity stimuli or wider timeframes may be necessary to detect differences in FM responses to anger (Livingstone et al., 2016; Wardle & De Wit, 2014). It is possible that

stimulus material did not reach the intensity or the timeframe needed to elicit measurable effects.

The CS results align with certain PD studies. While some findings report no difference in anger mimicry between groups (Livingstone et al., 2016), others do (Argaud et al., 2016; Kang et al., 2019), despite the fact that PD patients generally show difficulties in identification of emotions, especially anger and also sad expressions (Argaud et al., 2018; Livingstone et al., 2016).

3) Blocking D2/D3 receptors with Amisulpride is expected to affect FM in response to happy (smiles) (AngerToHappy) and angry expressions (HappyToAnger) by diminishing ZM activation compared to placebo group.

No evidence was found that Amisulpride influenced FM responses. This contrasts with previous research linking dopaminergic modulation to reward-related behavior, including motivation for rewards (Callesen et al., 2014; Chong et al., 2015; Hermans et al., 2010; Kim et al., 2013; O'Sullivan et al., 2009; Weber et al., 2016). Happy expressions (smiles) are often considered socially rewarding stimuli (Niedenthal et al., 2010; Sims et al., 2012) and prior studies suggest a connection between DA, reward, emotion recognition, and FM (Argaud et al., 2016, 2018; Bedi et al., 2009; Gray & Tickle-Degnen, 2010; Kang et al., 2019; Livingstone et al., 2016; Wardle & De Wit, 2014).

The results challenge the assumption that mesolimbic DA affects FM, particularly the idea that a dysfunctional dopaminergic system – such as in PD - diminishes activation of the ZM in response to happy expressions.

Similarly, the null finding in the condition HA contradicts the assumption based on prior PD research (Kang et al., 2019) suggesting reduced activation of ZM toward angry expressions.

5.2 Summary

One question arising from these results is whether happy expressions can be considered as rewards in an experimental setting, given that smiles can convey different social meanings (Korb et al., 2014). While some research supports the idea that happy expressions or smiles are rewarding (Sims et al., 2012), it may depend more on rewarding aspects of interactions such as regulating and smoothing social interactions (Hess & Bourgeois, 2010; Lakin et al., 2003; Seibt et al., 2015).

The more relevant question is whether DA modulates FM through another pathway. Amisulpride selectively blocks D2/D3 receptors, primarily affecting the limbic system (Schoemaker et al., 1997). However, research suggests that FM differences observed in PD patients may stem from disruptions in other dopaminergic pathways, such as the nigrostriatal pathway (Kang et al., 2019), which is involved in motor control and cognitive functions (Ko & Strafella, 2012).

5.3 Limitations and outlook

Several limitations should be considered.

Sample size and gender distribution

Overall sample size of the study was small, and future research should ensure balanced distributions of gender. Additionally, hormonal levels potentially differing by gender (e.g., testosterone) should be accounted for, as they may impact FM.

Timing and experimental conditions

The FM task started around 4h after drug administration. By this time, subjects may have experienced fatigue, particularly since participants came fasting and later consumed besides water and milk, sugary snacks and cocoa. This could have raised issues with concentration, let alone sitting in front of a screen, which then may have caused trials without participants actually watching the clips. Future research should control these factors.

Stimulus presentation and ecological validity

FM should be tested in a more naturalistic setting, potentially involving real-time social interactions rather than pre-recorded videos. Given that social rewards depend on context, incorporating participant ratings of reward value and effort-related measures could provide deeper insights into FM.

As there are some indications that with impaired DA systems, FM and emotion recognition could be delayed, it should be considered to use other timeframes for measuring FM.

Pharmacological considerations

Future research should examine interindividual differences in FM responses and incorporate measures of hormone levels, serum levels, and neurotransmitter synthesis

capacities.

Alternative dopaminergic pathways

Since the dopaminergic system extends beyond reward processing, future studies should investigate how other pathways, such as the nigrostriatal pathway, contribute to FM.

5.4 Conclusion

The study's findings suggest that while FM occurs reliably in response to facial expressions, blocking D2/D3 receptors with Amisulpride does not significantly impact this process. Future research should explore alternative dopaminergic mechanisms, individual differences, and more ecologically valid experimental designs to further the understanding of FM and its neural underpinnings.

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7. Appendix

Abstract

Facial mimicry is the tendency to imitate observed facial expressions, which plays a crucial role in social interactions. Previous research suggests that the dopaminergic system, particularly the mesolimbic pathway, is involved in reward processing and social behavior. This study investigates whether blocking dopamine D2/D3 receptors with Amisulpride affects FM in response to happy and angry facial expressions. A randomized, double-blind, placebo-controlled experiment was conducted with 39 healthy participants, who received either 400 mg of Amisulpride or a placebo. Electromyographic activity of the *Zygomaticus major* and *Corrugator supercilii* muscles was recorded while participants observed dynamic facial expressions morphing from happy to angry or vice versa. Results showed that muscle activity changed significantly over time in both conditions, but no significant differences were found between the Amisulpride and placebo groups. These findings suggest that mesolimbic dopamine D2/D3 receptor antagonism does not influence facial mimicry, indicating that other neurotransmitter systems may be more relevant for this process. Future research should explore alternative pathways and the potential role of dopamine in broader social and affective functions.

Keywords: Facial Mimicry, Amisulpride, Dopamine, Dopaminergic System, EMG

Abstract

Gesichtsmimikry beschreibt die Tendenz, beobachtete Gesichtsausdrücke zu imitieren, was eine zentrale Rolle in sozialen Interaktionen spielt. Frühere Forschung deutet darauf hin, dass das dopaminerge System, insbesondere der mesolimbische Pfad, an der Belohnungsverarbeitung und sozialen Verhaltensweisen beteiligt ist. Diese Studie untersucht, ob das Blockieren von Dopamin-D2/D3-Rezeptoren durch Amisulprid die Gesichtsmimikry fröhlicher und wütender Gesichtsausdrücke beeinflusst. In einer randomisierten, doppelblinden, Placebo kontrollierten Studie erhielten 39 gesunde Versuchsteilnehmende entweder 400 mg Amisulprid oder ein Placebo. Die Aktivität der Muskeln *Zygomaticus major* und *Corrugator supercilii* wurde mittels Elektromyografie gemessen, während die Versuchspersonen Videos dynamischer Gesichtsausdrücke anschauten, die von fröhlich zu wütend oder umgekehrt morphten. Die Ergebnisse zeigen, dass sich die Muskelaktivität über die Zeit hinweg signifikant veränderte. Jedoch konnten keine signifikanten Unterschiede zwischen der Amisulprid- und der Placebogruppe festgestellt werden. Diese Ergebnisse zeigen, dass das Blockieren der mesolimbischen Dopamin-D2/D3-Rezeptoren keine Auswirkungen auf die Gesichtsmimikry hat. Das könnte ein Hinweis sein, dass andere Neurotransmittersysteme für diesen Prozess relevanter sind. Zukünftige Studien sollten alternative Signalwege sowie die potenzielle Rolle von Dopamin in sozialen und affektiven Funktionen weiter untersuchen.

Keywords: Gesichtsmimikry, Amisulprid, Dopamin, dopaminerges System, EMG

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List of Abbreviations

AH	AngerToHappy
CS	Corrugator Supercilii
FM	Facial Mimicry
EMG	Electromyography
HA	HappyToAnger
PD	Parkinson's Disease
ZM	Zygomaticus Major