

THESIS

In order to obtain the title of

**DOCTOR OF COGNITIVE PSYCHOLOGY FROM
UNIVERSITÉ GRENOBLE ALPES**

**DOCTOR OF PSYCHOLOGY FROM
GHENT UNIVERSITY**

Specialty: **Cognitive Sciences, Psychology, Neurocognition &
Educational Sciences**

Arrêté ministériel : May 25, 2016

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Prepared as a joint PhD in

**Laboratoire de Psychologie et NeuroCognition (UGA) &
Psychopathology and Affective Neuroscience Laboratory (UGent)**

In the **Doctoral School EDISCE (UGA)** and the **Doctoral School of
Social and Behavioural Sciences (UGent)**

Psychophysiological characteristics of verbal rumination

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RÉSUMÉ

To be detailed...

OVERZICHT

To be detailed...

ABSTRACT

To be detailed...

ACKNOWLEDGEMENTS

Acknowledgements will be included in the final version of this thesis.

PREFACE

This PhD has been realised as a joint PhD between Univ. Grenoble Alpes (Grenoble, France) and Ghent University (Ghent, Belgium). It has been funded by Univ. Grenoble Alpes (UPMF)...

The underlying code as well as an online version of this thesis is available at: https://github.com/lnalborczyk/phd_thesis.

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Part I

Theoretical chapters

THEORETICAL FRAMEWORK

All human things are subject to
decay, and when fate summons,
Monarchs must obey

Mac Flecknoe
John Dryden

As you read these words, you might notice the presence of a familiar companion. A voice-like phenomenon that remains unnoticed until we pay attention to it. However, if I ask you to focus on that little voice while you are reading these lines, you would probably be able to provide a relatively fine-grained description of this thing that we call inner speech. Whose voice is it? Is it yours? Is it gendered? It is usually possible to examine these aspects as well as more low-level features like the tone of this soundy companion, its pitch, its tempo, or virtually any sensory aspect of it. This first set of very basic observations already provide us some very important insights. First, if we can think about inner speech, then it should be something different from thinking itself. Rather, inner speech (or *covert speech*) can be construed as a vehicle for conscious thought (instead of *verbal thinking*, for instance)¹. Second, the set of observations we can make about our inner voice also tautologically reveals that inner speech is accompanied by sensory percepts (sounds, kinaesthetic feelings, etc.). It thus raises another set of fascinating questions about the origin and nature of inner speech percepts: where do these percepts come from? Why do they look like the one we experience when we *actually* (overtly) speak?

This first set of questions refer to the *nature* of inner speech, that is, *what* is it? In the current work, we are mostly concerned with this first question. Another related set of fascinating questions revolve around the *function* question, that is, *what is it for* (e.g., Alderson-Day & Fernyhough, 2015)? The influential Vygotskian theory of inner speech development suggests that inner speech evolves from *private speech* (i.e., self-addressed overt speech) during childhood. As such, we (as others have argued elsewhere) postulate that the functions of inner speech are inherited from the functions of private speech, via a mechanism of internalisation. The specific features of this internalisation process are

¹We will not dwell on the touchy question whether inner speech is a necessary condition for consciousness. For the current purpose, it is sufficient to say that thinking and inner speech are ontologically separable.

worthy of investigation on their own (and we briefly discuss them later on) but we are mostly interested in the *what is* (the nature) question here. Thus, we will only sparsely address the *functions* question in the following text.

That being said, it is interesting to look at situations in which these functions do not work as intended. These *dysfunctions* (that can also be considered as *mis-exadaptation*, Agnati et al., 2012) are as spread as... They can generally be understood as transdiagnostic processes (i.e., a process that is not specific to a single pathology), and cover various...

1.1 Rumination as a form of repetitive negative thinking

1.1.1 Theoretical perspectives on rumination

The cognitive approach (Koster, De Lissnyder, & De Raedt, 2013; Koster, De Lissnyder, Derakshan, & De Raedt, 2011)... from the RST to contrl theories and the mental habit theory...

As suggested by Christoff, Irving, Fox, Spreng, & Andrews-Hanna (2016), rumination and other forms of spontaneous thoughts can be considered in a common conceptual space (see Figure 1). This space is built upon two dimensions: *deliberate constraints* and *automatic constraints*. These dimensions represent two general mechanisms that allow to constrain the contents of these related mental states and the transitions between them. The first constrain correspond to a deliberate processus and is implemented through **cognitive control** (Miller, 2000). The second constrain is referring to more automatic constrains like sensory afferences. In this framework, rumination is characterised by the highest level of automatic constraints and spread all along the *deliberate constraints* dimension.

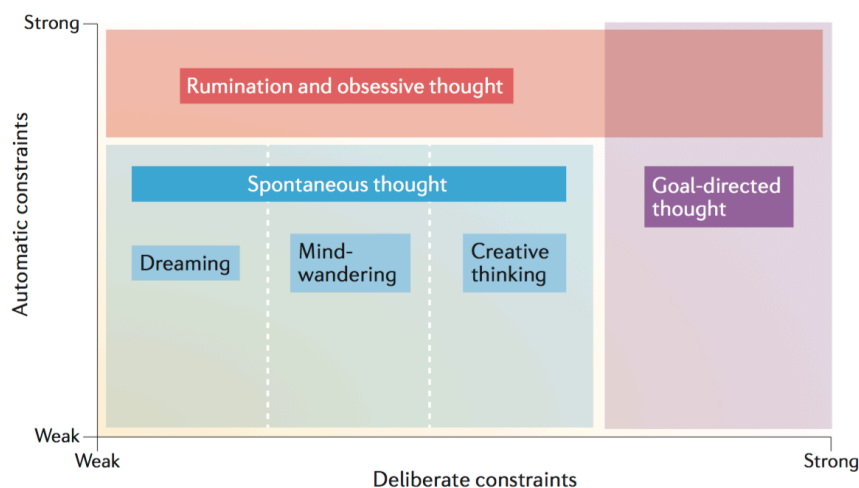


Figure 1.1: Conceptual space of different types of thought (Christoff et al., 2016)

...

1.1.2 On the verbal and sensory properties of rumination

...

1.2 What is that little voice inside my head ?

...description, functions, nature...

1.2.1 Brief historical overview of inner speech investigations

...add a brief historical timeline here (from egger to kapur)...

1.2.2 Inner speech as a form of motor imagery

...

1.2.2.1 Action representation and internal models

Voir Marc Jeannerod (1994) and Wolpert, Ghahramani, & Jordan (1995), Wolpert & Ghahramani (2000)...

Box 1.1: What is an internal model ?

Blah blah...

...

1.2.3 Theoretical perspectives about inner speech production

The inner voice as the sensory consequence (prediction, see Loevenbruck et al., 2018) of imagined speech. Analogy with raising the arm: what we perceive when we imagine raising our arm are the sensory consequences (e.g., visual) of what would happen if we actually raised our arm, these are then kind of predictions. The same thing happens during inner speech production: the inner voice is the predicted auditory consequence of actual speech, except that it's predicted. The two actions might seem very different, partly because of differences in the degree of automaticity. Imagining raising our arm might need a voluntary/deliberate/conscious (choose a word) intention (i.e., I want to raise my arm > I raise my arm) while speech imagery (i.e., inner speech) seems more automatic: we do not express consciously the intention to speak, we just speak...

Learning how to internalise speech might be similar to learning how to internalise playing an instrument... Let's consider the analogy between speaking and playing an instrument (e.g., the piano). Playing piano results from the learning of an infinitely complex coordination of fine motor sequences, that in turn produce sensory (kinesthetic, auditory, visual, etc) feedback to the producer of the action (the agent). It seems that (from a certain level of analysis), the act of speech can be paralleled with the act of playing an instrument in that it consists in the coordination of infinitely complex movements that result in some modifications in the environment that in turn generate sensory feedbacks for the agent. Thus, pursuing the analogy, we argue that imagining playing piano and imagining speaking (i.e., producing inner speech) might rest on similar mechanisms... see O'Shea & Moran (2018) on expert pianists...

1.2.3.1 MVTV Cohen (1986)

...

1.2.3.2 Predictive models

Learning how to internalise speech might be similar to learning how to internalise playing an instrument... Let's consider the analogy between speaking and playing an instrument (e.g., the piano). Playing piano results from the learning of an infinitely complex coordination of fine motor sequences, that in turn produce sensory (kinesthetic, auditory, visual, etc) feedback to the producer of the action (the agent). It seems that (from a certain level of analysis), the act of speech can be paralleled with the act of playing an instrument in that it consists in the coordination of infinitely complex movements that result in some modifications in the environment that in turn generate sensory feedbacks for the agent. Thus, pursuing the analogy, we argue that imagining playing piano and imagining speaking (i.e., producing inner speech) might rest on similar mechanisms... see O'Shea & Moran (2018) on expert pianists...

1.2.3.3 Loevenbruck et al., HMOSAIC

...

Copy-pasted from zygote old intro...

Speech production might be one of the most complex motor action ever studied. Involving the fine-grained coordination of more than 100 muscles in the upper part of the body (Simonyan & Horwitz, 2011), its expression is shaped by a subtle combination of physiological, cultural and evolutionary determinants. In adult humans, its covert counterpart (i.e., *inner speech* or *verbal imagery*) has developed to allow the full reconstruction of usual overt speech situations. In the same way as visual imagery allows to mentally examine visual scenes, *verbal imagery* can be used as an internal tool, allowing –amongst other things– to rehearse or to prepare past and future speech situations (for a review, see Perrone-Bertolotti, Rapin, Lachaux, Baciú, & Loevenbruck, 2014). In consideration of its self-evident motoric nature, a parallel can be drawn between verbal imagery and other forms of motor imagery (e.g., imagined walking or imagined writing). As such, inner speech studies might benefit from insights gained from the study of motor imagery and the field of motor cognition (e.g., Haggard, 2005; Marc Jeannerod, 2006). Whereas previous research have demonstrated that it is possible to record muscle-specific electromyographic correlates of inner speech production using invasive intramuscular needle electrodes, more recent research using surface electromyography lead to mixed results. Building upon previous work, we describe an experimental set-up using surface electromyography with the aim of refining the description of the involvement of the speech motor system during inner speech production.

1.3 Theoretical perspective on motor imagery

Considerable experimental evidence has accumulated to suggest that movement execution and MI share substantial overlap of active brain regions (for review, see Guillot et al., 2012). Such apparent functional equivalence supports the hypothesis that MI draws on the similar neural networks that are used in actual perception and motor control (Jeannerod, 1994; Grezes and Decety, 2001; Holmes and Collins, 2001)...

See introduction of O'Shea (2017) phd thesis introduction... See Stinear's chapter in Guillot's book for intracortical and spinal mechanisms involved during motor imagery (p.55-57).

1.3.1 Overt and imagined actions

Wittgenstein's (1953) famous query: "When I raise my arm, what is left after subtracting the fact that my arm raised?". We posit that what is left is an internal model (a representation) of what should happen if and when my arm goes up (Jeannerod, 1999)...

1.3.2 The motor simulation theory

In his seminal paper, Marc Jeannerod (1994) introduces the idea of a close functional equivalence between motor imagery and motor preparation, as suggested by the similar neural and physiological correlates observed in both imaging and preparing (and by the effects of motor imagery on motor learning and training). According to Jeannerod, actions are driven by an "internally represented world rather than directly by the external world" (page 187). In the next paragraph(s), Jeannerod seems to equate motor representations with motor images or what he calls *pragmatic representations* (page 187). For Jeannerod, motor imagery is a part of a broader phenomenon (the motor representation) related to intending and preparing movements (page 189). He also echoes the distinction (coming from sport psychology) between *internal* (imagining an action from the first perspective, which would be more kinesthetic) and *external imagery* (imagining an action from the third perspective, which would be more visual). For him, motor imagery necessarily belongs to the former type (page 189), whereas the term of *motor imagery* in sport psychology seems to more broadly encompass both first-person and third-person motor imagery [e.g.,].

Motor preparation is unconscious while motor imagery is conscious. However, Jeannerod suggests that the difference between the two might be a difference of degree, rather than one of kind, as the difference between the two would only be determined by the time spent on each (i.e., if one makes motor preparation lasting for longer than it's usually needed, it can "become" motor imagery, page 190). In plain words, the idea is that motor representations/intentions become known to the subject when they are not followed by execution (page 190), so there would be a continuum between motor preparation and motor imagery.

EMG lines of evidence page 191: Eye movements have been recorded during the recall of visual scenes (e.g., Jeannerod & Mouret 1962). Jacobson (1930) first suggested that muscles were selectively activated during imagined action. Shaw (1940) reported that during imagined weight lifting, electromyographic (EMG) activity of forearm muscles increased linearly with the magnitude of the weight, etc.

Neural structures and physiological correlates page 192-193.

Evidence from mental chronometry and temporal equivalence (e.g., Landauer, 1962), showing that it takes approximately the same amount of time to say the alphabet or series of numbers aloud and to think them to himself.

The famous walking + load experiment of Decety et al. (1989) reveals that force, rather than duration, is the encoded variable, and that estimated duration is merely derived from the level of centrally represented force. When the subjects carried the load, they centrally programmed a greater force to overcome any resistance, because subjects did not actually walk (page 194). For Jeannerod, the programming of force as a subjective correlate: the sensation of effort, which can become perceptible to the actor independently of overt movement execution and can be monitored experimentally (page 194). Page 196: representation of kinematic regularities (e.g., Fitts' law, Viviani's experiments, etc.).

In the following (section 5, starting page 197), Jeannerod develops the idea that the goal of an action includes an internal representation of both the external object toward which it

is directed and the final state of the organism when that object has been reached. Then, he develops on the idea of a distinction between a pragmatic mode and a semantic mode (page 198). He also makes a distinction between two functions of representations: a sensory one (to extract from the external world the attributes of objects or situations that are relevant to a given action) and a motor one (to encode some aspects of that action) (page 199).

NB: in this paper (Jeannerod, 1994), there is no explicit mention of a “simulation” process... Jeannerod only discusses the conscious access to the content of a representation, and defines it as motor imagery (cf. notes above as well as the distinction drawn by Grush, 2004). Interestingly, in the commentaries, Morton suggests that many of the cases Jeannerod reports are actually instances of simulations, rather than mere imagination, because these cases contain part of what would be involved in actual performance (page 215). In his response, Jeannerod seems to agree with and to endorse this view (e.g., page 231).

In the commentary of this paper Grush suggests that... (Motor models as steps to higher cognition (Rick Grush)) there should exist an inverse model, and points that nothing mentioned in the target article is actually capable of generating mental imagery. For Grush, this inverse model would act centrally, taking as input the efferent copy (for instance) and would output a mock version of the afferent signals the peripheral system would have produced given the same input (i.e., an emulator, see page 209).

For M. Jeannerod (1995), motor imagery is necessarily first-perspective. Third perspective imagery is imagery, but not MOTOR imagery... Motor representations are conceived here as ‘internal models’ of the goal of an action.

In Marc Jeannerod (2001), “Covert and overt stages thus represent a continuum, such that overtly executed action implies the existence of a covert stage, whereas a covert action does not necessarily turns out into an overt action” (page S103). Jeannerod introduces the term S-states to designate mental states which involve an action content and where brain activity can be shown to simulate that observed during the same, executed, action. These S-states correspond to intended action, imagined action, and many others (see Table 1, page S104).

In Marc Jeannerod (2006), *The Simulation Hypothesis of Motor Cognition* (pages 129-164). See Schematic summary of motor simulation according to Jeannerod (page 133).

“Simulation is used here as the off-line rehearsal of neural networks involved in specific operations such as perceiving or acting (page 129). According to our view [...], the simulation relates not to complete actions but to unspecific elements that comprise actions. Motor representations are automatically assembled in response to immediate task requirements and do not rely on memorized actions (page 134).”

The peripheralist versus centralist interpretation of physiological inner speech correlates (page 153): Subvocal speech was first interpreted as a source of peripheral kinesthetic information which, when projected to central nervous structures, generated auditory images of the corresponding words. The same interpretation was given to the low intensity EMG recorded during mental motor imagery of limb actions, which was thought to be the origin of the feelings experienced by the subject during mental rehearsal (Jacobson 1930), or to the eye movements recorded during mental visual imagery (e.g. Brandt and Stark, 1997). However, this interpretation of mental processes as consequences of peripheral feedback is now disproved by recent experiments showing complete absence of muscular activity in many subjects during motor imagery. When present, this activity is rather assumed to be a consequence of incomplete inhibition of motor output during mental states involving motor simulation. This same interpretation might also hold for

inner speech.

Motor imagery can be defined as the mental process by which one rehearses a given action, without engaging in the physical movements involved in this particular action. One of the most influential theoretical explanation of this broad phenomenon, the *motor simulation theory* (MST, Marc Jeannerod, 1994, 2001, 2006), contains the three following postulates at its core: i) there exists a continuum between the covert (the mental representation) and the overt execution of an action, ii) action representations can operate off-line, via a *simulation* mechanism, and iii) covert actions rely on the same set of mechanisms as the overt actions they simulate, except that execution is inhibited (O'Shea & Moran, 2017).

In this framework, the concept of simulation refers to the “offline rehearsal of neural networks” (Marc Jeannerod, 2006), and motor imagery is conceptualised as a simulation of the covert stage of the same executed action (O'Shea & Moran, 2017). The MST shares some similarities with the theories of embodied and grounded cognition (Barsalou, 2008) in that both allow to account for the phenomenon of motor imagery by appealing to a simulation mechanism. However, the concept of simulation in grounded theories is assumed to be multi-modal (not just motoric) and to operate in order to achieve a particular abstract knowledge (O'Shea & Moran, 2017), which is not the concern of the MST².

The MST is supported by a wealth a findings, going from mental chronometry studies showing that the time taken to perform an action is often found to be similar to the time needed to imagine the corresponding action (though not always, see Glover & Baran, 2017, for a review of controversial findings and for an alternative conceptualisation of motor imagery), to neuroimaging and neurostimulation studies showing that both motor imagery and overt actions tend to recruit similar frontal, parietal and sub-cortical regions (e.g., Héту et al., 2013; Marc Jeannerod, 2001). The involvement of the motor system during motor imagery is also supported by repeated observations of autonomic responses and peripheral muscular activity during motor imagery (we discuss these observations in section 3.4.1.2).

²We should also make a distinction between *embodiment of content*, which concerns the conceptual content of language, and *embodiment of form*, which concerns “the vehicle of thought”, that is, proper speech production (Pickering & Garrod, 2013).

Box 1.2: Centralism versus peripheralism

The motor theory of perception: mental states arise from movement (more precisely, from innervation associated with movements (the perceived image of an object arises from the muscular discharges produced during the movements involved in exploring it). The peripheral variant of the motor theory is the idea according to which the mental image of that object is also produced by covert muscular discharges in the related muscles (cf. work from James, 1890; Jacobson, 1930).

But the idea developed in this paper is quite the reverse of the peripheral motor theory and would be more akin to a centralist version of the motor theory (page 190). Jeannerod says that existence of muscle discharge during a mental state does not imply a bottom-up influence of these discharges on the mental state. The key phenomenon in Jeannerod's conception is the motor intention, which is thought to be largely endogenous (page 190). Motor imagery would represent the results of conscious access to the content of the intention, and the content of the intention would constrain the expression of the image (page 190). Jeannerod also states that this view is in agreement with the corollary discharge model. The key idea is that the activation of peripheral mechanisms is not needed to generate the image (but they can be activated, as a consequence of the activation of the image/representation)...

1.3.3 Emulation and internal models

NB: in this paper emulator is used as a synonym for “forward models” (pages 378-379)...

Grush (2004) proposes a unified framework for explaining a wide variety of representational functions of the brain: the emulation theory of representation. This theory has its origins in control theory (forward models) and signal processing (Kalman filters).

Difference between emulation theory of motor imagery and simulation theory à la Jeannerod (pages 384-385): “[...] Given these definitions, it should be clear that the simulation theory and the emulation theory are not at all the same thing. They agree that the efferent motor centers are active during imagery. The simulation theory takes this by itself to be sufficient for motor imagery; the emulation theory does not, and claims that in addition, an emulator of the musculoskeletal system is needed and imagery is produced when the efferent motor centers drive this emulator. This distinction should be entirely obvious. To make an analogy: The emulation theory claims that motor imagery is like a pilot sitting in a flight simulator, and the pilot's efferent commands (hand and foot movements, etc.) are translated into faux “sensory” information (instrument readings, mock visual display) by the flight simulator which is essentially an emulator of an aircraft. The simulation theory claims that just a pilot, moving her hands and feet around but driving neither a real aircraft nor a flight simulation, is sufficient for mock sensory information.”

Distinguishing between simulation and emulation theories (page 385): “[...] The only way to get from the former (signals in motor format) to the latter (signals in proprioceptive and kinesthetic format) is to run the motor signal through something that maps motor plans/signals to proprioception and kinesthesia. And the two possibilities are (a) the body (yielding real proprioception and kinesthesia), and (b) a body emulator (yielding faux proprioception and kinesthesia) [...] A motor plan is one thing, a sequence of

proprioception and kinesthesia is another. The simulation theorist conflates them.”

In Moulton & Kosslyn (2009), In brief: they tried to characterize mental imagery as an information processing system, considering it in the perspective of Marr’s three levels of explanation (i.e., the computational, algorithmic, and implementation levels).

Imagery is also characterized by its reliance on perceptual representations and activation of perceptual brain systems. We use this conception of imagery to argue that all imagery is simulation— more specifically, it is a specific type of simulation in which the mental processes that ‘run’ the simulation emulate those that would actually operate in the simulated scenario. This type of simulation, which we label emulation, has benefits over other types of simulations that merely mimic the content of the simulated scenario. They note (page 1273): “[...] ‘motor imagery’ (which actually appears to be proprioceptive or kinaesthetic imagery—one experiences the bodily sensations of movement, not the movement commands themselves: Jeannerod, 1994).”

A second class of explanatory models of motor imagery are concerned with the phenomenon of *emulation* and with *internal models* (see Gentsch, Weber, Synofzik, Vosgerau, & Schütz-Bosbach, 2016, for a review of the similarities and dissimilarities of simulation and emulation models).

Internal model theories share the postulate that the motor system is represented by *internal models*, whose function is to estimate and anticipate the outcome of a motor command. One of its variant, the *motor control theory* (e.g., Kawato, 1999; Wolpert et al., 1995), assumes two kind of models: a forward model that predicts the sensory consequences of motor commands from efference copies, and an inverse model that calculates the feed-forward motor commands from the desired movement (Gentsch et al., 2016).

Emulation theories (e.g., Grush, 2004; Moulton & Kosslyn, 2009) borrow from both previously discussed framework (i.e., simulation theories and internal model theories) to posit a specific kind of simulation. While the MST postulates that during simulation the motor system is guided exclusively by internal motor representations, the emulation theories suggest that both motor and sensory systems are emulated in parallel (Grush, 2004; O’Shea & Moran, 2017).

In the emulation model proposed by Grush (2004), the *emulator* is a device that implements the same input-output function as the body (i.e., the musculoskeletal system and relevant proprioceptive/kinaesthetic systems). When the emulator receives a copy of the control signal (which is also sent to the body), it produces an output signal (the emulator feedback), identical or similar to the feedback signal produced by the body³. This feedback would be responsible for the presence of sensory percepts (e.g., visual, auditory, kinaesthetic) during motor imagery.

One important difference between the emulation theory of motor imagery and the MST though, is that the latter takes the mere activation of efferent motor centres as being sufficient for explaining motor imagery, while the emulation theory postulates that an emulator of the musculoskeletal system is needed (Grush, 2004, pp. 384–385). Grush (2004) suggested an analogy to illustrate this difference: “The emulation theory claims that motor imagery is like a pilot sitting in a flight simulator, and the pilot’s efferent commands (hand and foot movements, etc.) are translated into faux “sensory” information (instrument readings, mock visual display) by the flight simulator which is essentially an emulator of an aircraft. The simulation theory claims that just a pilot, moving her hands and feet around but driving neither a real aircraft nor a flight simulation, is sufficient for mock

³In Grush’s terminology, *emulator* is used as a synonym for *forward models* (see Grush, 2004, pp. 378–379).

sensory information”. Alternatively, in the words of Moulton & Kosslyn (2009), instrumental simulations (à la Jeannerod) can be thought of as *first-order* simulations that imitate the content of the simulated action, while emulative simulations can be thought of as *second-order* simulations that imitate both the content and the processes that change the content.

1.4 Electromyography of covert actions

1.4.1 Explaining the muscular activity during motor imagery

Motor imagery has consistently been defined as the mental rehearsal of a motor action without any overt movement. One consequence of this claim is that, in order to prevent execution, the neural commands for muscular contractions should be blocked at some level of the motor system by active inhibitory mechanisms (for a review, see Guillot, Di Rienzo, MacIntyre, Moran, & Collet, 2012). Despite these inhibitory mechanisms, there is now abundant evidence for peripheral muscular activation during motor imagery (for a review, see Guillot & Collet, 2005; Guillot et al., 2012). As suggested by Marc Jeannerod (1994), the incomplete inhibition of the motor commands would provide a valid explanation to account for the peripheral muscular activity observed during motor imagery. This idea has been corroborated by studies of changes in the excitability of the motor pathways during motor imagery tasks. Bonnet, Decety, Jeannerod, & Requin (1997) measured spinal reflexes while participants were instructed to either press a pedal with the foot or to simulate the same action mentally. They observed that both H-reflexes and T-reflexes increased during motor imagery, and that these increases correlated with the force of the simulated pressure. Using transcranial magnetic stimulation and motor evoked potentials (MEPs), several investigators observed muscle-specific increases of MEPs during various motor imagery tasks, while no such increase could be observed in antagonist muscles (e.g., Fadiga et al., 1999; Rossini, 1999)⁴.

Box 1.3: The motor inhibition problem

The problem of inhibition (page S106): how come that covert actions, in spite of activation of the motor system, do not result in muscular activity and overt movements? Two possibilities:

- * Subliminal activation during S-states, insufficient to fire spinal motoneurons
- * Or, activations would be blocked/attenuated before to reach the motoneurons by an inhibitory mechanism generated in parallel to the motor activation...

NB: these two possibilities are not necessarily incompatible...

Interestingly, the dominant interpretation of the muscular correlates of motor imagery at the beginning of the last century was that the peripheral muscular activity observed during imagined actions was the *source* of the mental content. However, as explained by Marc Jeannerod (2006), this interpretation of mental processes as a consequence of peripheral feedback is now disproved, for instance by the simple fact that many people

⁴As a side note, we should remark that these findings are consistent with both the simulation and the emulation views on motor imagery.

can experiment motor imagery, without any observable muscular activity⁵. In the most recent theoretical explanations of motor imagery (e.g., MST, emulation or internal models theories), the peripheral activity is rather assumed to be a consequence of an incomplete inhibition of motor output during the mental states involving motor simulation/emulation (i.e., these views adhere to a *centralist* interpretation of the physiological correlates of inner speech).

1.4.2 Controversial findings

As reviewed in Guillot, Lebon, & Collet (2010), although there are many observations showing a peripheral muscular activity during motor imagery, there are also many studies failing to do so, or reporting surprisingly high levels of inter-subject variability, with some participants showing no muscular activity at all. Putting aside the discussion on the exact nature and location of the inhibitory mechanisms during motor imagery (see Guillot et al., 2012), two main explanations have been advanced to resolve these discrepancies. First, the electromyographic activity recorded during motor imagery could be moderated by the perspective taken in motor imagery. We usually make a distinction between a first-person perspective or *internal imagery* (i.e., imagining an action as we would execute it) and a third-person perspective or *external imagery* (i.e., imagining an action as an observer of this action), that seem to involve different neural and cognitive processes. It has been shown that a first-person perspective generally results in greater EMG activity than motor imagery in a third-person perspective (Hale, 1982; Harris & Robinson, 1986). Second, some authors postulated that the intensity of the EMG activity recorded during motor imagery might be related to the individual ability to form an accurate mental representation of the motor skill (i.e., the vividness of the mental image). However, after reviewing the available evidence, Guillot et al. (2009) concluded that this is unlikely to be the case. Alternatively, discrepancies in experimental design and methodological choices (e.g., use of intramuscular versus surface electromyography) could also explain these different results (Guillot et al., 2010).

In the next section, we turn to a discussion of inner speech conceptualised as a kind of motor (and sensory) imagery of speech, and discuss the theoretical underpinnings of this proposition as well as the available empirical evidence in its support.

1.4.3 Electromyographic correlates of inner speech production

Various terms: internal speech, silent speech, covert speech, implicit speech, speech imagery... difference between generative component (mackay, 1992) / inner speaking / inner voice and perceptual or auditory component (MacKay, 1992) or inner hearing / inner ear...

A key question: what is the lowest level of specification of inner speech ? ... figure from oppenheim & dell (2010...)

See MacKay (1992, page 131) for discussion of speaking rate in inner and overt speech... faster rates also occur for other highly trained skills (e.g., tying a shoelace, Annett, 1988)... ref to review of Guillot...

⁵The *peripheralist* interpretation has also been disproved by the heroic experiment carried out by Smith, Brown, Toman, & Goodman (1947). Smith used d-tubocurarine (curare) to paralyse his own facial muscles in order to test this interpretation. He later reported that, while being paralysed, he was still able to think in words and to solve mathematical problems.

EMG during inner speech (from mckay, 1992): “interestingly, this same EMG activity invariably precedes by a few milliseconds the full blown muscle activity that occurs during normal movements” (e.g., Schmidt, 1982)...

Box 1.4: Proper control conditions in electromyographic studies of inner speech production

Guidelines from Garrity (1977) here...

While grasping the concept of a visual image appears as relatively straightforward, it seems more difficult at first to grasp the concept of a motor image, especially when it comes to verbal imagery. The subjective experience of the tension that results from a given position of the articulators and the covert production of an incompatible speech sound permits to substantiate what a motor image is. For instance, it is generally impossible to generate the image of the pronunciation of the sound “b” while keeping the mouth wide opened (e.g., Binet, 1886; Stricker, 1880). This simple experiment allows defining imagined speech as the simulation of the corresponding overt verbal content, where *simulation* is meant to be understood either as the off-line rehearsal of neural motor networks involved in the overt action (Marc Jeannerod, 2006), or in the terms of the emulation theories discussed previously⁶.

The model of wilful (voluntary) inner speech production introduced in Lvenbruck et al. (2018) goes one step further and, by building on the models of speech motor control (e.g., Houde & Nagarajan, 2011; Wolpert et al., 1995), describes inner speech as “multi-modal acts with multi-sensory percepts stemming from coarse multi-sensory goals”. In other words, the auditory and kinaesthetic sensations perceived during inner speech prediction are assumed to be the predicted sensory consequences of speech motor acts, emulated by internal forward models, that use the efference copies issued from an inverse model (this proposal shares similarities with the emulation model of motor imagery discussed earlier, Grush, 2004).

Fin de l’intro old de zygoto...

⁶Translated to speech, the MST is similar to previous proposals such as the *motor theory of voluntary thinking* (Cohen, 1986) or the hierarchical model of mental practice (Mackay, 1981).

METHODOLOGICAL FRAMEWORK

This chapter aims to provide the key concepts related to the methods we used. More precisely, in this chapter we cover the technical concepts related to speech production mechanisms, to electromyography and to the statistical approach we followed in our work. Finally, we give an overview of the following chapters.

2.1 Speech production mechanisms

2.1.1 Theoretical aspects of speech production

...

2.1.2 Mechanical aspects of speech production

...

2.1.3 Muscles of interest in the present work

In the current work, we were especially interested in the activity of the orofacial muscles (i.e., the muscles situated around the mouth). More precisely, we were particularly interested in the *orbicularis oris inferior* and *orbicularis oris superior* muscles. As can be seen from Figure XX, these two muscles are actually parts of a broad sphincter muscle that permits the contraction and aperture of the mouth...

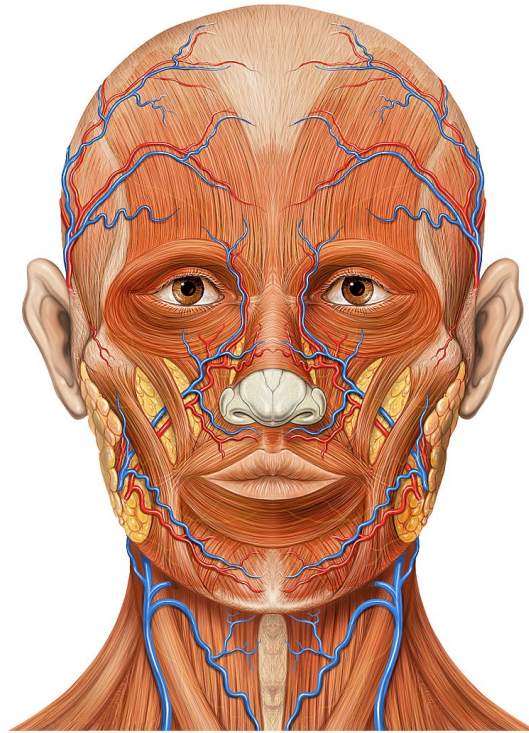


Figure 2.1: Illustration of a facial muscles of interest in the present work. Figure from Patrick J. Lynch, medical illustrator, <http://patricklynch.net>.

For sensors placement, we follow guidelines from...

- *Orbicularis oris inferior*: inferior portion of the *orbicularis oris* (mouth sphincter), serves to contract the lips and to compress them together and forward...
- *Zygomaticus major*: ...
- *Lateral frontalis*: ...
- *Flexor carpi radialis*: serves to raise the outer and middle brow... the electrode is placed on a vertical line that traverses the pupil of the eye (during centred gaze)...

2.2 A brief introduction to electromyography

Technically speaking, electromyography (EMG) is an experimental technique concerned with the recording and analysis of myoelectric signals (i.e., signals resulting from physiological variations in the state of muscle fibers membranes). Broadly speaking, EMG is a measure of the electrical activity generated during muscle contraction. It is used both as a basic tool in (for instance) biomechanical and psychophysiology studies and as an evaluation tool in applied research (e.g., physiotherapy, rehabilitation, human-computer interfaces). To facilitate the interpretation of the EMG signal, it is useful to briefly detail the meaning of its physiological components.

2.2.1 Nature of the EMG signal

2.2.1.1 Muscular anatomy and physiology

A muscle is a collection of fibers that can vary in length, orientation, diameter, and architectural characteristics. For instance, deeper muscle fibers are usually composed of a greater proportion of slow-twitch fibers (type I muscle fibers) whereas more superficial muscle fibers comprise a greater proportion of larger and fast-twitch fibers (type II muscle fibers, Kamen & Gabriel, 2010). On the basis of their structure and contractile properties, we can identify three types of muscle tissues: i) the skeletal muscles, ii) the cardiac muscles and iii) the smooth muscles. The skeletal muscles are the one we are recording the activity of when using EMG. These muscles are attached to the bones and are responsible for moving the skeleton.

The contraction of skeletal muscles is usually under voluntary control and is initiated by electrical impulses that propagate from the central nervous system to the muscle via the α -motoneurons. Interestingly, many (both larger and smaller) muscles are partitioned, with each portion having a specific role for the muscle function. Moreover, there is no one-to-one mapping between populations of motor neurons and muscle compartments. In other words, one population of motoneurons may innervate several compartments and several populations of motoneurons may innervate the same muscle compartment. Therefore, interpreting the EMG signal requires to be aware whether the recorded signal is characteristic of a whole muscle's activity or of a specific muscle compartment (Kamen & Gabriel, 2010).

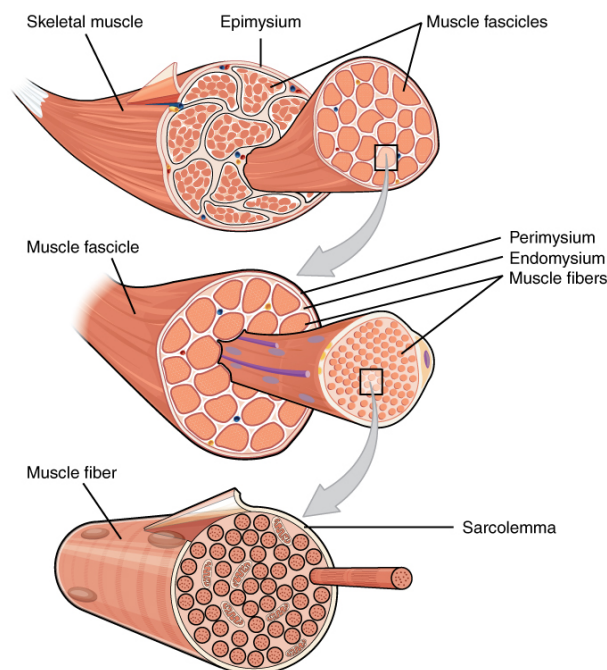


Figure 2.2: Illustration of a muscle fiber. Figure from the OpenStax *Anatomy and Physiology* Textbook. Download for free at <http://cnx.org/contents/14fb4ad7-39a1-4eee-ab6e-3ef2482e3e22@15.1>.

The muscle is a tissue, surrounded by a membrane, the *sarcolemma* (see Figure 2.2). Under resting conditions, the voltage inside the membrane is around -90mV , relative to the outside. This voltage results from a particular combination of sodium (Na^+), potassium

(K^+), chloride (Cl^-), and other anions. At rest, the concentration of Na^+ is relatively high outside the membrane and relatively low inside the membrane. The concentration of K^+ follows an opposite pattern, with a greater concentration inside the membrane, and a lower concentration outside the membrane.

2.2.1.2 The motor action potential

Because muscle membranes can change their electrical state, muscle fibers are excitable tissues. When a muscular fiber is depolarised, the *membrane potential* produces a response called the *muscle fiber action potential* or more generally the *motor action potential* (MAP). The generated action potential proceeds along the muscle fiber in both directions from the neuromuscular junction (Kamen & Gabriel, 2010)¹. In the first phase of the MAP, the Na^+ increases dramatically which induces a massive income of Na^+ into the cell, resulting in an inversion of the polarity of the cell (see Figure 2.3).

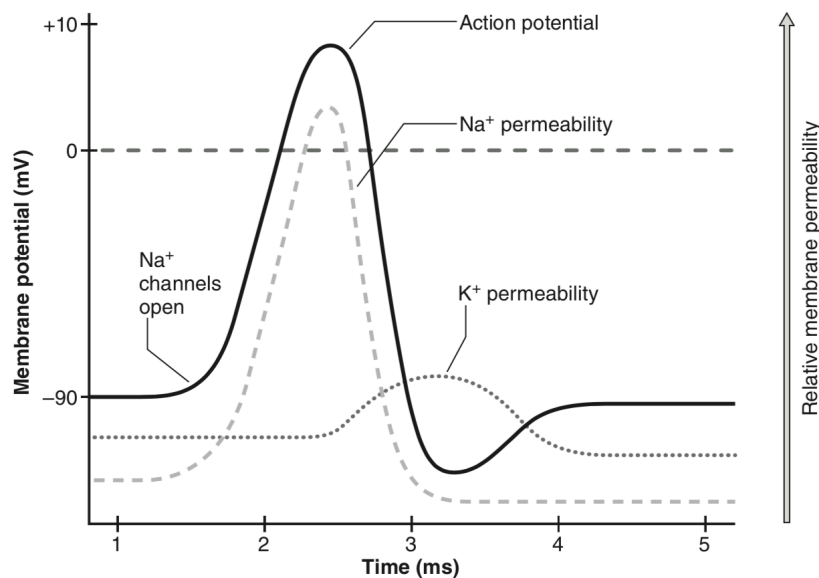


Figure 2.3: Time course of the muscle fiber action potential (Figure from Kamen & Gabriel, 2010).

The MAP is followed by a *refractory period*, during which there is a decrease in membrane excitability. This refractory period can be further decomposed in an *absolute* refractory period during which all Na^+ channels are closed, and a *relative* refractory period where some Na^+ channels are open (but to a lesser extent than before the MAP). Interestingly, this after-impulse hyperpolarisation limits the frequency of MAPs (Kamen & Gabriel, 2010).

2.2.1.3 The motor unit

The *motor unit* is the smallest controllable muscular unit. It consists of a single α -motoneuron, its neuromuscular junction, and all the muscle fibers it innervates. The number of motor units and their *innervation ratio* (i.e., the number of muscle fibers per

¹The *neuromuscular junction* is the site where the motoneuron's terminal meets the muscle fiber.

motor unit) can vary by muscle. For instance, large muscles have hundreds of motor units and an innervation ratio of a few thousands whereas small muscles have thousands of motor units but can have an innervation ratio of 10 to 20 per motor unit.

Because a single motoneuron can innervate multiple muscle fibers, the firing of a single motoneuron results in the simultaneous discharge of many muscle fibers. The *motor unit action potential* (MUAP) is the electric field resulting from the sum of the electric fields emitted by each fiber of the motor unit. In other words, it represents the spatiotemporal summation of individual MAPs originating from muscle fibers that are sufficiently close to a given electrode. This generates a *train* of MUAPs, called *motor unit action potential trains* (MUAPTs). The electric potential generated by this field is highly dependent of parameters such as the number of fibers, their length, the speed of conduction and position of the neuromuscular junction.

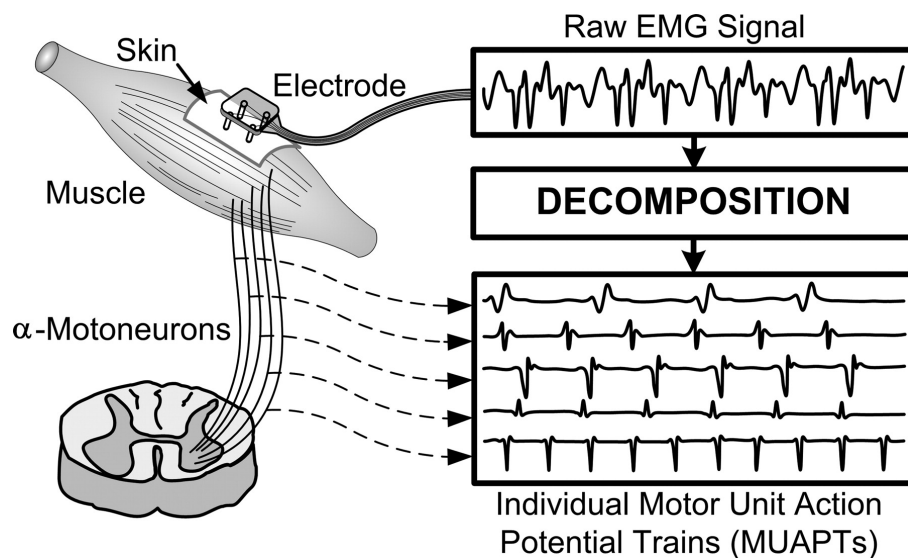


Figure 2.4: Motor unit action potential representation (Figure from De Luca et al., 2006).

In brief, the EMG signal results from a mixture of recruited motor units. It is composed of the sum of all MUAPs. This signal can vary considerably because of factors such as the muscle length (Babault, Pousson, Michaut, & Van Hoecke, 2003), the distance between the muscle fiber (of interest) and the electrode, the fiber length or the muscle temperature.

2.2.2 EMG instrumentation

Myoelectric measurements have a long history, starting in the XVII and XVIII centuries with the classical observations that muscle contraction can generate electricity and that electrical impulses can generate muscle contractions (for a more detailed historical perspective, see Raez, Hussain, & Mohd-Yasin, 2006). The term of electromyography and the first EMG measures were realised at the end of the XIXth century by Marey (1890).

Since then... Different types of electrodes: surface electrodes, active electrodes, indwelling electrodes, needle electrodes, wire electrodes... Fine-wire and Needle electrodes are mostly used in rehabilitation for diagnosis of muscle function and nerve conduction (Fridlund & Cacioppo, 1986)...

Many factors can affect the recorded EMG signals. These factors are usually described in three main categories (for more details, see De Luca, 1997):

- The *causative factors*, that have a basic effect on EMG signals. These factors can be further subdivided into two classes: i) the *extrinsic* factors including factors such as the type of electrode (e.g., size, shape, placement) or the inter-electrode distance and ii) the *intrinsic* factors such as physiological or anatomical factors (e.g., fiber type, fiber diameter, blood flow).
- The *intermediate factors*. These are the physiological phenomena that are influenced by one or more of the causative factors and that in turn influence the deterministic factors, such as the conduction velocity, spatial filtering or the signal *cross-talk*.
- Finally, the *deterministic factors* are influenced by the intermediate factors and have a direct effect on the EMG signal. These include the number of active motor units or the amplitude, duration and shape of the MUAPs.

All these factors contribute to modulating both the amplitude of the EMG signal and its spectral properties (e.g., its mean or median frequency). Fridlund & Cacioppo (1986) also emphasise the importance of six factors determining the quality of the recorded surface EMG signals. These factors are i) the proximity of the electrode to the underlying muscle mass, ii) the position of the electrode relative to muscle fibers size, location, and orientation...

(External) factors influencing the EMG signal: i) tissue characteristics (thickness, physiological changes and temperature), nb: vary greatly from subject to subject, ii) physiological cross-talk produced by neighbouring muscles but ECG spikes can also interfere with the EMG recording (especially in the upper trunk or shoulder muscles), iii) external noise in noisy electrical environments (e.g., electrical power), iv) electrodes and amplifiers...

The *crosstalk* phenomenon (De Luca, 1997)... In reason of the important... of facial muscles, the EMG activity of one recorded muscle generally does not represent the activity of a single muscle but rather a mixture of... L. Rapin (2011)... Placement of EMG sensors: we follow the atlas provided by Fridlund & Cacioppo (1986)...

Advantages of surface electrodes... they broadly detect the firing of aggregates of MUAPs that... however, they are nonselective (i.e., they cannot be interpreted as reflecting the activity of a single muscle), in comparison to inserted needle electrodes. As a result, it is usually inappropriate, when using surface EMG, to attribute the results to a single muscle (Fridlund & Cacioppo, 1986). Whereas for the sake of simplicity/clarity we designate sensors by the name of the underlying muscle (e.g., “FRO” for the *frontalis* site), it should be kept in mind that these sensors reflect the activity of “sites”, rather than single muscles.

2.2.3 EMG signal processing

The raw EMG signal is a stochastic train of MUAPs. As put by Fridlund & Cacioppo (1986), “when heard through a speaker, the raw EMG signal sounds like popcorn popping”. Therefore, it is usually unsuitable for immediate quantification. In order to illustrate what the EMG signal looks like, we simulated EMG data based on a standard algorithm implemented in the `biosignalEMG` package (Guerrero & Macias-Diaz, 2018), which is represented in Figure 2.5.

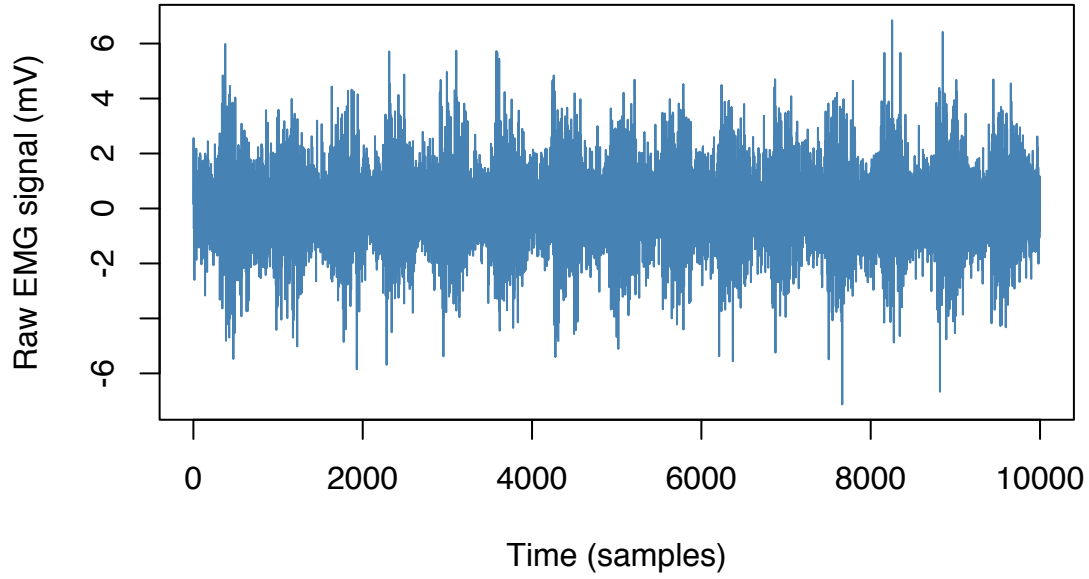


Figure 2.5: Simulated EMG signal.

We usually rectify the EMG signal by taking its absolute value and subtracting the mean in order to correct for any offset (bias) present in the raw data. This operation is represented in Figure 2.6.

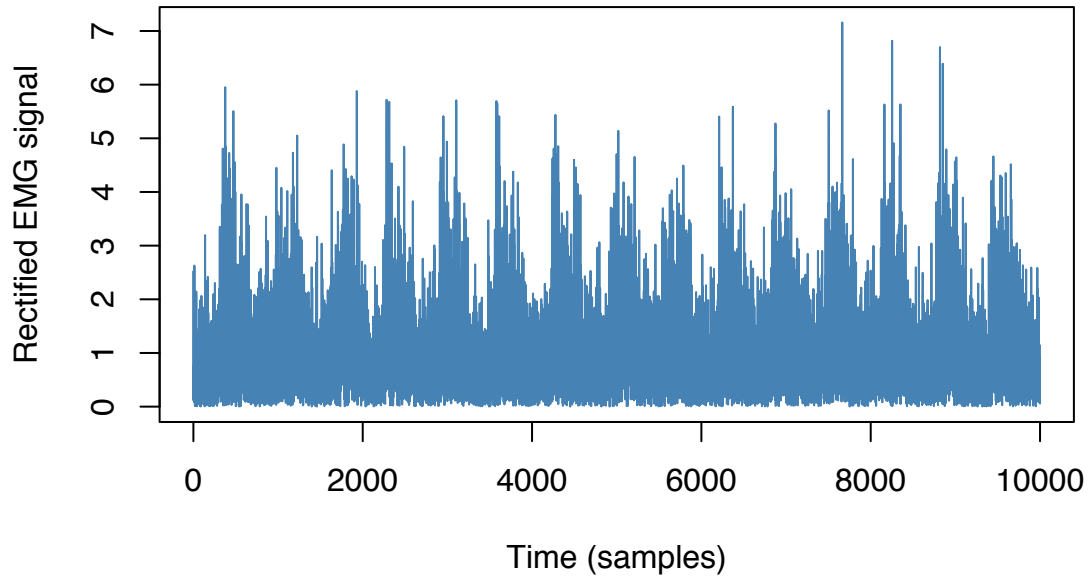


Figure 2.6: Rectified EMG signal.

From there, two main measures can be used to represent the magnitude of a muscle activity². The first one is the *mean absolute value* (MAV), which is computed over a specific interval and where $|x_n|$ is the absolute value of a datum of EMG in the data window.

$$MAV = \frac{1}{N} \sum_{n=1}^N |x_n|$$

²But see for instance Phinyomark, Nuidod, Phukpattaranont, & Limsakul (2012) for a brief overview of other features that can be extracted from the surface EMG signal.

The unit of measurement is usually the mV and the MAV calculation is generally similar to the numerical formula for integration (Kamen & Gabriel, 2010). The second one is the *root-mean-square* (RMS) amplitude:

$$RMS = \sqrt{\frac{1}{N} \sum_{n=1}^N x_n^2}$$

where x_n^2 is the squared value of each EMG datum and has both physical and physiological meanings. Put broadly, the RMS is taken to reflect the level of the physiological activities in the motor unit during contraction. Both the MAV and the RMS are illustrated in Figure 2.7.

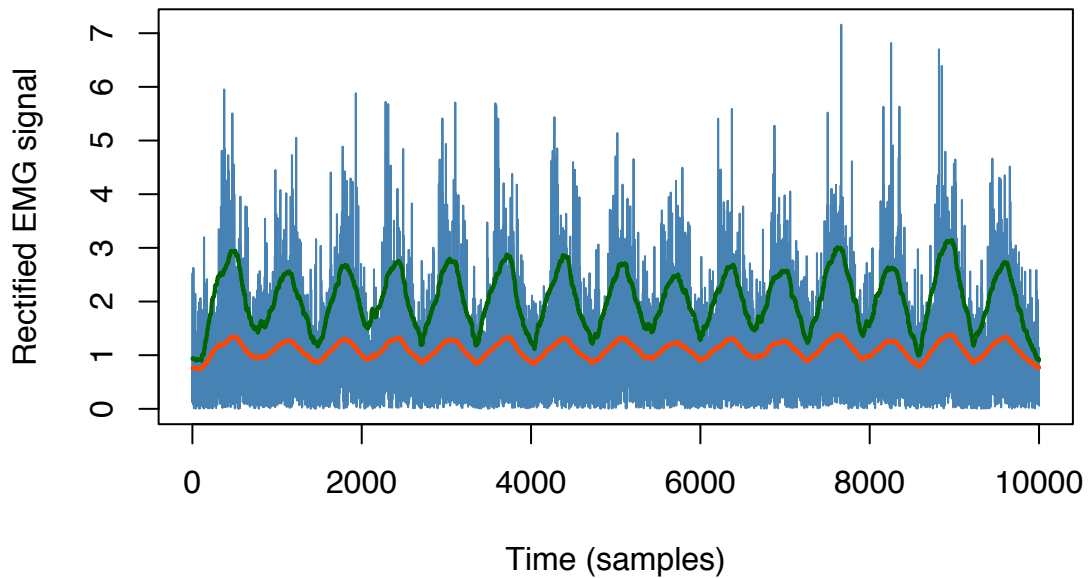


Figure 2.7: Illustration of the MAV (in orange) and RMS (in green) values. These two features are usually highly correlated but differ in magnitude.

For facial EMG studies, it is also recommended to filter... Studies have generally shown that the primary energy of surface EMG signals lie between 10 and 200Hz (Fridlund & Cacioppo, 1986). For facial surface EMG studies more specifically... See van Boxtel 2001... (Chowdhury et al., 2013; for more details on EMG signal processing, see for instance Ræz et al., 2006)...

2.3 Statistical modelling and statistical inference

2.3.1 Limitations of the standard statistical approach in Psychology

Numerous authors have highlighted the limitations inherent to the Null-Hypothesis Significance Testing (NHST) approach and the (exclusive) reliance on p -values and significance testing (e.g., Bakan, 1966; Gigerenzer, 2004; Kline, 2004; Lambdin, 2012; Meehl, 1967; Trafimow et al., 2018). Considering these limitations, some authors have recommended to push away significance testing and to develop the use of effect size estimates and confidence intervals in order to favour accumulation of evidence and a meta-analytical mode of thinking (e.g., Cumming, 2012, 2014).

However, the apparent superiority of confidence intervals over p -values is an illusion. Indeed, as noted by many observers, confidence intervals are simply inverted significance

tests. In other words, the confidence interval represents the range of values that are significant at some α level. Therefore, compared to a p -value, a confidence interval does not bring *any* new inferential value. Moreover, its interpretation might be as hard as the interpretation of p -values. For instance, contrary to a widely shared belief, confidence intervals do not contain the $(1 - \alpha) \cdot 100\%$ most probable values of the parameter (e.g., Morey, Hoekstra, Rouder, Lee, & Wagenmakers, 2015; Nalborczyk, Bürkner, & Williams, 2019).

That being said, it is fair to acknowledge that using confidence intervals (instead of or in addition to single p -values) do shift the emphasis from a mechanical (mindless) point-hypothesis testing procedure to a more careful consideration of the range of values that are *compatible* with some hypothesis. More importantly, it emphasises the uncertainty that accompanies every statistical procedure. Indeed, we think that most of the caveats that are attributed to a specific statistical procedure (e.g., to NHST) are really caveats of the way it is used. Namely, the fact that is used in a categorical and absolute way. This tendency has been coined as *dichotomania* (i.e., the tendency to consider that results are either present –if significant– or absent –if non-significant–), or *trichotomania* (e.g., when considering evidence ratios thresholds).

These biases have been highlighted by many statisticians in the past (e.g., Wasserstein & Lazar, 2016). Very recently, *The American Statistician* published a special issue on *Moving to a World Beyond “ $p < .05$ ”*, with the intention to provide new recommendations for users of statistics (e.g., researchers, policy makers, journalists). This issue comprises 43 original papers aiming to provide new guidelines and practical alternatives to the mindless use of statistics. In the accompanying editorial, Wasserstein, Schirm, & Lazar (2019) summarise these recommendations in the form of the ATOM guidelines: “Accept uncertainty. Be thoughtful, open, and modest.” We describe below how our statistical approach might be understood in the light of these core principles.

- **Accept uncertainty:** we try to represent and to acknowledge uncertainty in our analyses and conclusions. For instance, we do not conclude and/or infer that an effect is either “present” or “absent”, but we report the *estimated* magnitude of the effect and the uncertainty that comes with this estimation. Additionally, when relevant, we report probabilistic statements based on the posterior distribution.
- **Be thoughtful:** for each analysis opportunity (i.e., for each dataset to analyse), we consider what would be the most appropriate modelling strategy but we also acknowledge that there is no unique best way. In most empirical chapters, we clearly distinguish between confirmatory (preregistered) and exploratory (non-preregistered) statistical analyses. We routinely evaluate the *validity* of the statistical model (and of its assumptions) and we are suspicious of statistical *defaults*. We try to consider the *practical* significance of the results, rather than their *statistical* significance. We use a variety of statistics (e.g., effect sizes, interval estimates, information criteria) to obtain a more diverse picture of the meaning of the results.
- **Be open:** the soundness of a statistical procedure (and more generally, of an inferential procedure) can only be evaluated if it is made transparent to peers and readers for critical examination. Therefore, we take some space in the next section (but also in each experimental chapter) to motivate our statistical modelling decisions. We also make all R scripts available to ensure the reproducibility of the analyses. We try to be

exhaustive in the way we report our analyses and we beware of shortcuts than can hinder important information for the reader.

- **Be modest:** we recognise that there is no unique “true statistical model” and we discuss the limitations of our analyses and of our conclusions. We also recognise that scientific inference is much broader than statistical inference (e.g., a degenerative research program is much more informative than a non-significant p -value). We try not to conclude anything from a single study without the warranted uncertainty.

To sum up, we try to acknowledge the uncertainty that accompanies every (statistical) inference. In the next section, we present in more details what our approach does entail and we introduce some key technical concepts.

2.3.2 Current statistical approach

In brief, we tried to move from the point-hypothesis mechanical testing to an approach that emphasises parameter estimation, model comparison, and continuous model expansion (e.g., Cumming, 2012, 2014; Gelman & Hill, 2006; Gelman et al., 2013; Kruschke, 2015; Kruschke & Liddell, 2018a, 2018b; McElreath, 2016). In other words, our approach can be defined as a *statistical modelling* approach rather than a *statistical testing* approach. It means that we try to *model* the data (or rather the process that generated the data), rather than to “test” it. We carefully consider what could be the process that generated the data and we try to model it appropriately. For instance, we do not fit reaction time data, likert data, or electromyographic data using the same model (e.g., an ANOVA), as this practice would lead to high rates of erroneous inferences.

Throughout this work, we use Bayesian statistical modelling, not by dogmatism, but because we think the Bayesian approach provides richer inferences than the frequentist one (while the later can be subsumed under the first under particular assumptions). The main advantage of the Bayesian approach is the explicit use of probability to model the uncertainty (see Box 2.1). By doing so, the Bayesian approach permits to evaluate the probability of a parameter (or a vector of parameters) θ , given a set of data y :

$$p(\theta|y) = \frac{p(y|\theta)p(\theta)}{p(y)}$$

Using this equation (known as Bayes’ theorem), a probability distribution $p(\theta|y)$ can be derived (called the *posterior distribution*), that reflects knowledge about the parameter, given the data and the prior information. This distribution is the goal of any Bayesian analysis and contains all the information needed for inference.

The term $p(\theta)$ corresponds to the *prior distribution*, which specifies the prior information about the parameters (i.e., what is known about θ before observing the data) as a probability distribution. The left hand of the numerator $p(y|\theta)$ represents the *likelihood*, also called the *sampling distribution* or *generative model*, and is the function through which the data affect the posterior distribution. The likelihood function indicates how likely the data are to appear, for each possible value of θ . Finally, $p(y)$ is called the *marginal likelihood*. It is meant to normalise the posterior distribution, that is, to scale it in the “probability world”. It gives the “probability of the data”, summing over all values of θ and is described by $p(y) = \sum_{\theta} p(\theta)p(y|\theta)$ for discrete parameters, and by $p(y) = \int p(\theta)p(y|\theta)d\theta$ in the case of continuous parameters.

All this pieced together shows that the result of a Bayesian analysis, namely the posterior distribution $p(\theta|y)$, is given by the product of the information contained in the data (i.e., the likelihood) and the information available before observing the data (i.e., the prior). This constitutes the crucial principle of Bayesian inference, which can be seen as an updating mechanism. To sum up, Bayes' theorem allows a prior state of knowledge to be updated to a posterior state of knowledge, which represents a compromise between the prior knowledge and the empirical evidence.

Box 2.1: Probability theory as extended logic

Probability theory has been argued to be nothing more than *extended logic*. In other words, it generalises the rules of logic that apply to discrete events (e.g., TRUE or FALSE events) to continuous cases. By doing this move, probability theory gives a way to represent and to quantify the *uncertainty*. Importantly, the rules of probability have the same status as the rules of logic: these rules can be used to derive statements (conclusions) that are guaranteed to be correct given that some premises are correct.

Bayesian statistics can simply be presented as an application of probability theory to statistical analysis. Whereas the dependence of the inferential conclusions on prior assumptions is usually presented as a downside of this type of analysis, it is precisely what makes them *optimal* or *coherent* (in the sense of respecting the rules of probability). As put by Vandekerckhove (2018), the claim that Bayesian methods would be invalidated by their reliance on prior assumptions would be akin to conclude that logical deductions are somehow invalidated because they depend on premises.

We also use *multilevel models* (also known as *mixed-models*) to handle complex dependency structures and to obtain more precise estimates. A more accurate description of Bayesian multilevel models is outside the scope of this introductory section but the interested reader is redirected toward several existing tutorial papers (e.g., Nalborczyk, Batailler, Lvenbruck, Vilain, & Bürkner, 2019; Nicenboim & Vasishth, 2016; Sorensen, Hohenstein, & Vasishth, 2016). Throughout this work, we also make use of several tools with very distinct properties and uses. For instance, we use Bayes factors (BFs) to quantify the relative evidence for a statistical hypothesis (see Box 2.2), we use information criteria to assess the predictive abilities of our models (see Box 2.3), we use posterior predictive checks as well as a diagnostics tools (e.g., convergence indexes, trace plots) to assess the validity of our models, and we use summary statistics when appropriate to convey the meaning of the main results.

Box 2.2: What is a Bayes factor ?

It is a rule in statistics that every statistics has already been suggested as the *new statistics*. Confidence intervals have been suggested as a replacement to p-values, being purportedly more informative and less difficult to interpret. Credible intervals have been suggested as a replacement to confidence intervals, for roughly the same reasons. Bayesian hypothesis testing through Bayes factors (BFs) has also been suggested as a replacement for frequentist hypothesis testing. It has been argued that they permit a richer inference and that they come with a more straightforward interpretation. Whereas this might be true, they nonetheless come with their lot of misinterpretations.

To highlight what BFs are and what they are not, it might be useful to write down the formula used to compute them. To this end, it is useful to write the Bayes rule in its *odds form*, making the BF explicitly visible:

$$\underbrace{\frac{p(H_0|D)}{p(H_1|D)}}_{\text{posterior odds}} = \underbrace{\frac{p(D|H_0)}{p(D|H_1)}}_{\text{Bayes factor}} \times \underbrace{\frac{p(H_0)}{p(H_1)}}_{\text{prior odds}}$$

This equation reveals that the *posterior odds*, the ratio of the posterior probability (i.e., how much more probable is hypothesis 1 (H_1) as compared to hypothesis 2 (H_2), after seeing the data D), is equal to the ratio of the probability of the data given the first hypothesis and the probability of the data given the second hypothesis, multiplied by the *prior odds* (i.e., how much more probable was hypothesis 1 (H_1) as compared to hypothesis 2 (H_2), before seeing the data D).

Importantly, what we consider as *evidence* in the Bayesian framework is also known as a *marginal likelihood* and represents the information contained in the data, weighted by the prior information. It is a sum when parameters are discrete or an integral when parameters are continuous.

$$\text{evidence} = p(D|H) = \int p(\theta|H) p(D|\theta, H) d\theta$$

Therefore, the BF does not indicate how much "probable" a hypothesis is, or how much more probable a hypothesis is, compared to another one (this would be to conflate the BF with the posterior odds). Instead, the BF can be (should be) interpreted either i) as a ratio of two *marginal likelihoods* (i.e., a ratio of *evidence*) or ii) as an updating factor, that indicates how we should reallocate credibility from prior knowledge (what we knew before seeing the data) to posterior knowledge (what we know after seeing the data).

Bayes factors are often said to have desirable asymptotic (when the number of observations is very large) properties. Indeed, they are *consistent* for model identification. It means that if a *true* statistical model is in the set of models that are compared, using a BF will usually lead to selecting this *true* model with a probability approaching 1 with increasing sample size. Whereas this seems an appealing property, it also poses a challenge to the underlying statistical philosophy. Indeed, one could question whether it is sensible to assume a "true model" (an oxymoron) in real life, especially in the social sciences (e.g.,

Burnham & Anderson, 2002, 2004). As Findley (1985) notes: “[...] consistency can be an undesirable property in the context of selecting a model”. A more realistic question is then not to look for the *true* model, but for the *best* model from some practical purpose.

The usefulness of information criteria comes from their being approximations of the *out-of-sample deviance* of a model (see Box 2.3). In the current PhD work, we used generalisations of the AIC (especially the WAIC and LOOIC) that also approximate the out-of-sample deviance and as such give an indication of how good/bad a model is to predict future (i.e., non-observed) data.

Box 2.3: Information criteria

Hirotsugu Akaike noticed that the negative log-likelihood of a model + 2 times its number of parameters was approximately equal to the **out-of-sample deviance** of a model, which lead to what is nowadays known as the *Akaike information criterion* (AIC):

$$\text{AIC} = \underbrace{-2\log(\mathcal{L}(\hat{\theta}|\text{data}))}_{\text{in-sample deviance}} + 2K$$

$\approx \text{out-of-sample deviance}$

Where K is the number of parameters of the model and the *deviance* is a measure of discrepancy between the data and the model. Interestingly, we can make a distinction between two types of deviances.

First, the **in-sample deviance** indicates how bad a model is to explain the current dataset (the dataset used to fit the model). Second, and more importantly, the **out-of-sample deviance** indicates how bad a model is to explain a **future** dataset issued from the same data generation process (the same population).

In brief, in the present work, we used various methods but coherently with a few (nuanced) guiding principles. Namely, we favoured a *model comparison* approach (e.g., Burnham & Anderson, 2002, 2004; Judd, McClelland, & Ryan, 2009), we used several statistics when they provide complementary information (e.g., using both posterior probabilities, information criteria or BFs), we assessed the validity of our models (e.g., via posterior predictive checks), we reported these analyses transparently and we tried to convey uncertainty in our conclusions.

2.4 Overview of the following chapters

The experiment ran during this PhD will be presented as five empirical chapters that can be grouped under two main axes. In the first couple of experiments, we used surface electromyography and muscle-specific relaxation to investigate the involvement of the speech motor system during induced verbal and non-verbal rumination (Chapter 3 & 4). In Chapter 5, we used surface electromyography and machine learning algorithms to decode the muscle-specific EMG correlates of inner speech production. In the last couple of experiments, we switched strategy from the “correlates strategy” to the “interference strategy”, where the goal was to directly interfere with the activity of the speech motor system. More precisely, we used articulatory suppression to disrupt induced rumination in Chapter 6, and we used articulatory suppression to disrupt either induced rumination

or problem-solving in Chapter 7. Finally, in Chapter 8, we summarise the main findings, discuss their implications and suggest ways forward from both a theoretical and an experimental perspective.

Part II

Experimental chapters

OROFACIAL ELECTROMYOGRAPHIC CORRELATES OF INDUCED VERBAL RUMINATION

Add a summary of the research and brief introduction to this first empirical chapter here...¹

3.1 Abstract

Rumination is predominantly experienced in the form of repetitive verbal thoughts. Verbal rumination is a particular case of inner speech. According to the *Motor Simulation view*, inner speech is a kind of motor action, recruiting the speech motor system. In this framework, we predicted an increase in speech muscle activity during rumination as compared to rest. We also predicted increased forehead activity, associated with anxiety during rumination. We measured electromyographic activity over the *orbicularis oris superior and inferior*, *frontalis* and *flexor carpi radialis* muscles. Results showed increased lip and forehead activity after rumination induction compared to an initial relaxed state, together with increased self-reported levels of rumination. Moreover, our data suggest that orofacial relaxation is more effective in reducing rumination than non-orofacial relaxation. Altogether, these results support the hypothesis that verbal rumination involves the speech motor system, and provide a promising psychophysiological index to assess the presence of verbal rumination.

3.2 Introduction

As humans, we spend a considerable amount of time reflecting upon ourselves, thinking about our own feelings, thoughts and behaviors. Self-reflection enables us to create and clarify the meaning of past and present experiences (Boyd & Fales, 1983; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). However, this process can lead to

¹This experimental chapter is a published paper reformatted for the need of this thesis. Source: Nalborczyk, L., Perrone-Bertolotti, M., Baeyens, C., Grandchamp, R., Polosan, M., Spinelli, E., ... Loevenbruck, H. (2017). Orofacial Electromyographic Correlates of Induced Verbal Rumination. *Biological Psychology*, 127, 53-63. <https://dx.doi.org/10.1016/j.biopsycho.2017.04.013>.

unconstructive consequences when self-referent thoughts become repetitive, abstract, evaluative, and self-critical (Watkins, 2008).

Indeed, rumination is most often defined as a repetitive and recursive mode of responding to negative affect (Rippere, 1977) or life situations (M. S. Robinson & Alloy, 2003). Although rumination is a common process that can be observed in the general population (Watkins, 2008), it has been most extensively studied in depression and anxiety. Depressive rumination has been thoroughly studied by Susan Nolen-Hoeksema, who developed the Response Style Theory (RST, Nolen-Hoeksema, 1991). According to the RST, depressive rumination is characterized by an evaluative style of processing that involves recurrent thinking about the causes, meanings, and implications of depressive symptoms. Even though rumination can involve several modalities (i.e., visual, sensory), it is a predominantly verbal process (Goldwin & Behar, 2012; McLaughlin, Borkovec, & Sibrava, 2007). In this study, we focus on verbal rumination, which can be conceived of as a particularly significant form of inner speech.

Inner speech or covert speech can be defined as silent verbal production in one's mind or the activity of silently talking to oneself (Zivin, 1979). The nature of inner speech is still a matter of theoretical debate (for a review, see Perrone-Bertolotti et al., 2014). Two opposing views have been proposed in the literature: the *Abstraction view* and the *Motor Simulation view*. The *Abstraction view* describes inner speech as unconcerned with articulatory or auditory simulations and as operating on an amodal level. It has been described as "condensed, abbreviated, disconnected, fragmented, and incomprehensible to others" (Vygotsky, 1987). It has been argued that important words or grammatical affixes may be dropped in inner speech (Vygotsky, 1987) or even that the phonological form or representation of inner words may be incomplete (G. S. Dell & Repka, 1992; Sokolov, 1972). MacKay (1992) stated that inner speech is nonarticulatory and nonauditory and that "Even the lowest level units for inner speech are highly abstract" (p.122).

In contrast with this *Abstraction view*, the physicalist or embodied view considers inner speech production as mental simulation of overt speech production. As such, it can be viewed as similar to overt speech production, except that the motor execution process is blocked and no sound is produced (Grzes & Decety, 2001; Postma & Noordanus, 1996). Under this *Motor Simulation view*, a continuum exists between overt and covert speech, in line with the continuum drawn by Decety & Jeannerod (1996) between imagined and actual actions. This hypothesis has led certain authors to claim that inner speech by essence should share features with speech motor actions (Feinberg, 1978; Jones & Fernyhough, 2007). The *Motor Simulation view* is supported by several findings. First, covert and overt speech have comparable physiological correlates: for instance, measurements of speaking rate (Landauer, 1962) and respiratory rate (B. Conrad & Schönle, 1979) are similar in both. A prediction of the *Motor Simulation view* is that the speech motor system should be recruited during inner speech. Subtle muscle activity has been detected in the speech musculature using electromyography (EMG) during verbal mental imagery, silent reading, silent recitation (Jacobson, 1931; Livesay, Liebke, Samaras, & Stanley, 1996; McGuigan & Dollins, 1989; Sokolov, 1972), and during auditory verbal hallucination in patients with schizophrenia (Rapin, Dohen, Polosan, Perrier, & Lvenbruck, 2013). Second, it has been shown that covert speech production involves a similar cerebral network as that of overt speech production. Covert and overt speech both recruit essential language areas in the left hemisphere (for a review, see Perrone-Bertolotti et al., 2014). However, there are differences. Consistent with the *Motor Simulation view* and the notion of a continuum between covert and overt speech, overt speech is associated with more activity in motor and premotor

areas than inner speech (e.g., Palmer et al., 2001). This can be related to the absence of articulatory movements during inner verbal production. In a reciprocal way, inner speech involves cerebral areas that are not activated during overt speech (Basho, Palmer, Rubio, Wulfeck, & Müller, 2007). Some of these activations (cingulate gyrus and superior rostral frontal cortex) can be attributed to the inhibition of overt responses.

These findings suggest that the processes involved in overt speech include those required for inner speech (except for inhibition). Several studies in patients with aphasia support this view: overt speech loss can either be associated with an impairment in inner speech (e.g., Levine, Calvanio, & Popovics, 1982; Martin & Caramazza, 1982) or with intact inner speech: only the later phases of speech production (execution) being affected by the lesion (Baddeley & Wilson, 1985; Marshall, Rappaport, & Garcia-Bunuel, 1985; Vallar & Cappa, 1987). Geva, Bennett, Warburton, & Patterson (2011) have reported a dissociation that goes against this view, however. In three patients with chronic post-stroke aphasia (out of 27 patients), poorer homophone and rhyme judgement performance was in fact observed in covert mode compared with overt mode. A limitation of this study, though, was that the task was to detect rhymes in written words, which could have been too difficult for the patients. To overcome this limitation, Langland-Hassan, Faries, Richardson, & Dietz (2015) have tested aphasia patients with a similar task, using images rather than written words. They also found that most patients performed better in the overt than in the covert mode. They inferred from these results that inner speech might be more demanding in terms of cognitive and linguistic load, and that inner speech may be a distinct ability, with its own neural substrates. We suggest an alternative interpretation to this dissociation. According to our view, rhyme and homophone judgements rely on auditory representations of the stimuli (e.g., Paulesu, Frith, & Frackowiak, 1993). Overt speech provides a strong acoustic output that is fed back to the auditory cortex and can create an auditory trace, which can be used to monitor speech. In the covert mode, the auditory output is only mentally simulated, and its saliency in the auditory system is lesser than in the overt mode. This is in accordance with the finding that inner speech is associated with reduced sensory cortex activation compared with overt speech (Shuster & Lemieux, 2005). In patients with aphasia, the weakened saliency of covert auditory signals may be accentuated for two reasons: first, because of impairment in the motor-to-auditory transformation that produces the auditory simulation, and second, because of associated auditory deficits. Therefore, according to our view, the reduced performance observed in rhyme and homophone judgement tasks in the covert compared with the overt mode in brain-injured patients, simply indicates a lower saliency of the auditory sensations evoked during inner speech compared with the actual auditory sensations fed back during overt speech production. In summary, these findings suggest that overt and covert speech share common subjective, physiological and neural correlates, supporting the claim that inner speech is a motor simulation of overt speech.

However, the *Motor Simulation view* has been challenged by several experimental results. Examining the properties of errors during the production of tongue twisters, Oppenheim & Dell (2010) showed that speech errors display a lexical bias in both overt and inner speech. According to these researchers, errors also display a phonemic similarity effect (or articulatory bias), a tendency to exchange phonemes with common articulatory features, but this second effect is only observed with overt speech or with inner speech accompanied with mouthing. This has led Oppenheim & Dell (2010) to claim that inner speech is fully specified at the lexical level, but that it is impoverished at lower featural (articulatory) levels. This claim, related to the *Abstraction view*, is still debated however, as a phonemic similarity effect has been found by Corley, Brocklehurst, & Moat (2011). Their

findings suggest that inner speech is in fact specified at the articulatory level, even when there is no intention to articulate words overtly. Other findings however, may still challenge the *Motor Simulation view*. R. Netsell, Ashley, & Bakker (2010) have examined covert and overt speech in persons who stutter (PWS) and typical speakers. They have found that PWS were faster in covert than in overt speech while typical speakers presented similar overt and covert speech rates. This can be interpreted in favour of the *Abstraction view*, in which inner representations are not fully specified at the articulatory level, which would explain why they are not disrupted in PWS speech. Altogether, these results suggest that full articulatory specification may not always be necessary for inner speech to be produced.

The aim of this study is to examine the physiological correlates of verbal rumination in an attempt to provide new data in the debate between motor simulation and abstraction. A prediction of the *Motor Simulation view* is that verbal rumination, as a kind of inner speech, should be accompanied with activity in speech-related facial muscles, as well as in negative emotion or anxiety-related facial muscles, but should not involve non-facial muscles (such as arm muscles). Alternatively, the *Abstraction view* predicts that verbal rumination should be associated with an increase in emotion-related facial activity, without activity in speech-related muscles and non-facial muscles.

There is strong interest in the examination of physiological correlates of rumination as traditional assessment of rumination essentially consists of self-reported measures. The measurement of rumination as conceptualized by Nolen-Hoeksema (1991) was operationalized by the development of the Ruminative Response Scale (RRS), which is a subscale of the response style questionnaire (Nolen-Hoeksema & Morrow, 1991). The RRS consists of 22 items that describe responses to dysphoric mood that are self-focused, symptom-focused, and focused on the causes and consequences of one's mood. Based on this scale, Treynor, Gonzalez, & Nolen-Hoeksema (2003) have offered a detailed description of rumination styles and more recently, Watkins (2008) has further characterized different modes of rumination. The validity of these descriptions is nevertheless based on the hypothesis that individuals have direct and reliable access to their internal states. However, self-reports increase reconstruction biases (e.g., Brewer, 1986; Conway, 1990) and it is well known that participants have a very low level of awareness of the cognitive processes that underlie and modulate complex behaviors (Nisbett & Wilson, 1977).

In order to overcome these difficulties, some authors have attempted to quantify state rumination and trait rumination more objectively, by recording physiological or neuroanatomical correlates of rumination (for a review, see Siegle & Thayer, 2003). Peripheral physiological manifestations (e.g., pupil dilation, blood pressure, cardiac rhythm, cardiac variability) have been examined during induced or chronic rumination. Vickers & Vogeltanz-Holm (2003) have observed an increase in systolic blood pressure after rumination induction, suggesting the involvement of the autonomic nervous system in rumination. Moreover, galvanic skin response has shown to be increased after a rumination induction, in highly anxious women (Sigmon, Dorhofer, Rohan, & Boulard, 2000). According to Siegle & Thayer (2003), disrupted autonomic activity could provide a reliable physiological correlate of rumination. In this line, Key, Campbell, Bacon, & Gerin (2008) have observed a diminution of the high-frequency component of heart rate variability (HF-HRV) after rumination induction in people with a low tendency to ruminate (see also Woody, McGeary, & Gibb, 2014). A consistent link between perseverative cognition and decreased HRV was also found in a meta-analysis conducted by Ottaviani et al. (2015). Based on these positive results and on suggestions that labial EMG activity may accompany inner speech and therefore rumination, our aim was to examine facial EMG as a potential

correlate of rumination and HRV as an index to examine concurrent validity.

In addition to labial muscular activity, we also recorded forehead muscular activity (i.e., *frontalis* muscle) because of its implication in prototypical expression of sadness (e.g., Ekman & Friesen, 1978; Kohler et al., 2004), reactions to unpleasant stimuli (Jäncke, Vogt, Musial, Lutz, & Kalveram, 1996), and anxiety or negative emotional state (A. Conrad & Roth, 2007)². Our hypothesis was that *frontalis* activity could be an accurate electromyographic correlate of induced rumination, as a negatively valenced mental process.

In this study, we were also interested in the effects of relaxation on induced rumination. Using a relaxation procedure targeted on muscles involved in speech production is a further way to test the reciprocity of the link between inner speech (verbal rumination) and orofacial muscle activity. If verbal rumination is a kind of action, then its production should be modulated in return by the effects of relaxation on speech effectors. This idea is supported by the results of (among others) Cefidekhanie, Savariaux, Sato, & Schwartz (2014), who have observed substantial perturbations of inner speech production while participants had to realize forced movements of the articulators.

In summary, the current study aimed at evaluating the *Motor Simulation view* and the *Abstraction view* by using objective and subjective measures of verbal rumination. To test the involvement of the orofacial motor system in verbal rumination, we used two basic approaches. In the first approach, we induced verbal rumination and examined concurrent changes in facial muscle activity (Experiment 1). In the second approach, we examined whether orofacial relaxation would reduce verbal rumination levels (Experiment 2). More specifically, in Experiment 1, we aimed to provide an objective assessment of verbal rumination using quantitative physiological measures. Thus, we used EMG recordings of muscle activity during rumination, focusing on the comparison of speech-related (i.e., two lip muscles *orbicularis oris superior* and *orbicularis oris inferior*) and speech-unrelated (i.e., forehead *frontalis*- and forearm *flexor carpi radialis*) muscles. Under the *Motor Simulation view*, an increase in lip and forehead EMG activity should be observed after rumination induction, with no change in forearm EMG activity, associated with an increase in self-reported rumination. Alternatively, under the *Abstraction view*, an increase in forehead activity should be observed, associated with an increase in self-reported rumination, and no changes in either lip or forearm activity should be noted.

In Experiment 2, in order to assess the reciprocity of the rumination and orofacial motor activity relationship, we evaluated the effects of orofacial relaxation on rumination. More specifically, we compared three kinds of relaxation: i) Orofacial Relaxation (i.e., lip muscles), ii) Arm Relaxation (i.e., to differentiate effects specific to speech-related muscle relaxation) and iii) Story Relaxation (i.e., to differentiate effects specific to attentional distraction). If the *Motor simulation view* is correct, we predicted a larger decrease of lip and forehead muscle activity after an Orofacial Relaxation than after an Arm Relaxation (associated with a larger decrease in self-reported rumination), which should also be larger than after listening to a story. We also predicted that forearm activity should remain stable across the

²The *corrugator supercilii* was another potential site, as it is sensitive to negative emotions. However, it has been claimed to be mostly activated for strong emotions such as fear/terror, anger/rage and sadness/grief (Ekman & Friesen, 1978; Sumitsuji, Matsumoto, Tanaka, Kashiwagi, & Kaneko, 1967). The rumination induction used in this study was designed to have participants self-reflect and brood over their failure at the I.Q. test. It was not meant to induce such strong emotions. Several studies have reported increased activity in the *frontalis* muscle at rest in anxious or generalized anxiety disorder patients (for a review, see A. Conrad & Roth, 2007). We expected the type of emotional state induced by rumination to be closer to anxiety or worry than to strong emotions like fear, anger or grief. It was therefore more appropriate to record non-speech facial activity in the *frontalis* rather than in the *corrugator*.

three conditions (i.e., should not decrease after relaxation). Alternatively, if the *Abstraction view* is correct, we predicted that none of the relaxation conditions should have an effect on lip or arm activity, because none of these should have increased after induction. However, we expected to observe a decrease in forehead activity and self-reported rumination after Orofacial or Arm relaxation, this decrease being larger than after listening to a Story. Importantly, we predicted that, under the *Abstraction View* no superiority of the Orofacial relaxation should be observed over the Arm relaxation.

3.3 Methods

3.3.1 Participants

Because of the higher prevalence of rumination in women than in men (see Johnson & Whisman, 2013, for a recent meta-analysis), we chose to include female participants only. Seventy-two female undergraduate students from Univ. Grenoble Alpes, native French speaking, participated in our study. One participant presenting aberrant data (probably due to inadequate sensor sticking) was removed from analyses. Final sample consisted of seventy-one undergraduate female students ($M_{\text{age}} = 20.58$, $SD_{\text{age}} = 4.99$). They were recruited by e-mail diffusion lists and participated in the experiment for course credits. They did not know the goals of the study. The cover story presented the research as aiming at validating a new I.Q. test, more sensitive to personality profiles. Participants reported having no neurologic or psychiatric medical history, no language disorder, no hearing deficit, and taking no medication. Each participant gave written consent and this study has been approved by the local ethical committee (CERNI, N° 2015-03-03-61).

3.3.2 Material

EMG signals were detected with TrignoTM Mini sensors (Delsys Inc.) at a sampling rate of 1926 samples/s with a band pass of 20 Hz (12 dB/ oct) to 450 Hz (24 dB/oct) and were amplified by a TrignoTM 16-channel wireless EMG system (Delsys Inc.). The sensors consisted of two 5 mm long, 1 mm wide parallel bars, spaced by 10 mm, which were attached to the skin using double-sided adhesive interfaces. The skin was cleaned by gently scrubbing it with 70% isopropynol alcohol. EMG signals were then synchronized using the PowerLab 16/35 (ADInstrument, PL3516). Raw data from the EMG sensors were then resampled at a rate of 1 kHz and stored in digital format using Labchart 8 software (ADInstrument, MLU60/8). As shown in Figure 3.1, bipolar surface EMG recordings were obtained from two speech-related labial muscles: *orbicularis oris superior* (OOS) and *orbicularis oris inferior* (OOI), as well as from one non speech-related but negative-affect-related facial muscle: *frontalis* (FRO) and from one non-facial and non speech-related muscle: *flexor carpi radialis* (FCR) on the non-dominant forearm. The latter pair of electrodes was used to check whether the rumination induction would cause any muscle contraction, outside of the facial muscles. The same sensor layout was used for all participants. Asymmetrical movements of the face have been shown in speech and emotional expression. As reviewed in Everdell, Marsh, Yurick, Munhall, & Paré (2007), the dominant side of the face displays larger movements than the left during speech production, whereas the non-dominant side is more emotionally expressive. To optimise the capture of speech-related activity, the OOS and OOI sensors were therefore positioned on the dominant side of the body (i.e. the right side for right-handed participants). To optimise the capture of emotion-related activity,

the FRO sensor was positioned on the non-dominant side. To minimise the presence of involuntary manual gestures during the recording, the FCR sensor was positioned on the non-dominant side. Each pair of electrodes was placed parallel with the direction of the muscle fibers, at a position distant from the innervation zones and the muscle tendon interface, following the recommendations of De Luca (1997). The experiment was video-monitored using a Sony HDR-CX240E video camera to track any visible facial movements. A microphone was placed 20–30 cm away from the participant's lips to record any faint vocal production during rumination. Stimuli were displayed with E-prime 2.0 (<http://www.pstnet.com>) on a 19-inch color monitor.

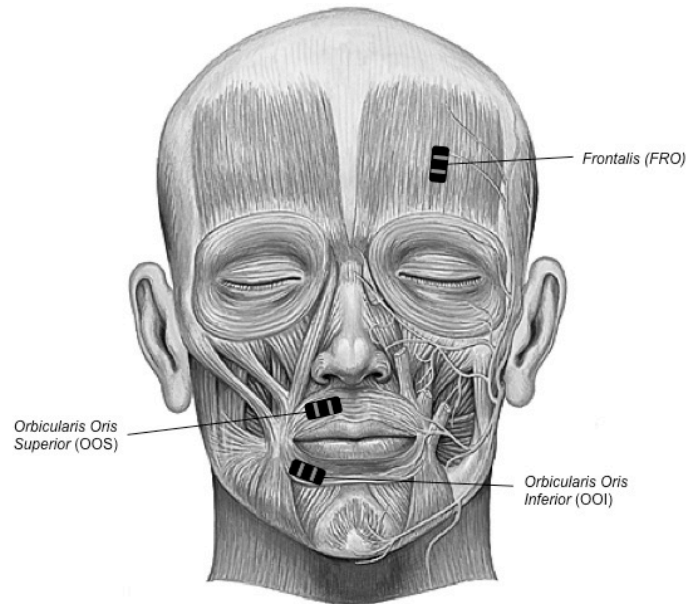


Figure 3.1: Facial muscles of interest. Two speech-related labial muscles: *orbicularis oris superior* (OOS) and *orbicularis oris inferior* (OOI); as well as one non speech-related but sadness-related facial muscle: *frontalis* (FRO).

3.3.3 Procedure

This study consisted of two parts. The first part was carried out a week before the EMG experiment and consisted in checking the inclusion criteria. We checked that participants did not exceed a threshold on a depressive symptoms scale. This was assessed using the French version of the *Center for Epidemiologic Studies Depression* scale (CES-D, Fuhrer & Rouillon, 1989), which evaluates the level of depressive symptom in subclinical population. We also collected information about any potential speech, neurologic, neuromuscular or cardiac disorders and about academic curriculum. Finally, the tendency to ruminate (i.e., trait rumination) in daily life was evaluated using the French version of the Mini-CERTS (Cambridge-Exeter Repetitive Thought Scale, Douilliez, Philippot, Heeren, Watkins, & Barnard, 2012). The second part included two EMG interdependent experiments related to *Rumination Induction* and *Rumination Reduction by Muscle Relaxation*. Specifically, Experiment 1 consisted of acquiring physiological EMG data during rest and induced rumination and Experiment 2 consisted of acquiring physiological EMG data after different kinds of relaxation (see below).

During both Experiment 1 and Experiment 2, momentary rumination was assessed using four different Visual Analogue Scales (VAS, the first two being adapted and translated to French from Huffziger, Ebner-Priemer, Koudela, Reinhard, & Kuehner, 2012) rated from 0 to 100: i) “At this moment, I am thinking about my feelings” (referred to as VAS “*Feelings*”), ii) “At this moment, I am thinking about my problems” (referred to as VAS “*Problems*”), iii) “At this moment, I am brooding about negative things” (referred to as VAS “*Brooding*”) and iv) “At this moment, I am focused on myself” (referred to as VAS “*Focused*”).

3.3.3.1 Experiment 1: rumination induction

Participants were seated in front of a computer screen in a comfortable and quiet room. EMG sensors were positioned as explained above (see Figure 3.1). Before the rumination induction, each participant underwent a non-specific relaxation session (i.e., without targeting specific muscles) in order to minimize inter-individual initial thymic variability (approximate duration 330 s). Immediately after, participants were instructed to remain silent and not to move for one minute to carry out EMG “baseline” measurements. Then, participants’ initial level of rumination was assessed using the four VASs.

Subsequently, participants were invited to perform a 15-min I.Q. test, which was presented on the computer screen facing them. They were instructed to correctly respond to three types of I.Q. questions (logical, mathematical and spatial-reasoning questions) in a very short time (30 s). Most of the questions were very difficult, if not impossible, to correctly answer in 30 s. We included ten different questions for each of the three types of I.Q. question: ten logical questions (e.g., finding the next number of a Fibonacci sequence), ten mathematical questions (e.g., “What is the result of the following calculus: (30/165) (70/ 66)”) and ten spatial-reasoning questions (e.g., finding the next figure of a series). Forced-failure tasks have extensively been employed in the literature to induce a slightly negative mood, ideal for subsequent rumination induction (e.g., Lemoult & Joormann, 2014; Randenborgh, Hüffmeier, LeMoult, & Joormann, 2010).

After the I.Q. test, participants were invited to reflect upon the causes and consequences of their feelings, during five minutes (rumination induction). This method is based on the induction paradigm developed by Nolen-Hoeksema & Morrow (1993). The classical paradigm uses a series of prompts. In order to avoid the potential confound in muscle activity induced by silent reading, we did not use the full paradigm. We simply summarised the series of prompts by one typical induction sentence. During this period, participants were asked to remain silent and not to move, while EMG recordings were carried out (i.e., EMG Post-induction measures). EMG signals of rumination were collected during the last minute of this period. Finally, participants were instructed to self-report momentary rumination on the four VASs.

3.3.3.2 Experiment 2: rumination reduction by relaxation

After Experiment 1, participants were randomly allocated to one of three groups. In the first group, participants listened to a pre-recorded relaxation session that was focused on orofacial speech-related muscles (“*Orofacial Relaxation*” condition). In the second group, relaxation was focused on the arm muscles (“*Arm Relaxation*” condition). In the third group, participants simply listened to a story, read by the same person, for an equivalent duration (“*Story*” condition, detailed content of the story can be found in the supplementary materials, in French). In summary, the first condition allowed us to evaluate the effects of targeted speech muscle relaxation on rumination. The second condition

allowed evaluating the effects of a non-orofacial relaxation (i.e., speech-unrelated muscles) while the third condition allowed controlling for effects of attentional distraction during relaxation listening.

The speeches associated with the three conditions, relaxation sessions and story listening session, were delivered to the participants through loudspeakers. They were recorded by a professional sophrology therapist in an anechoic room at GIPSA-lab (Grenoble, France) and were approximately of the same duration (around 330 s).

After the relaxation/distraction session, participants were asked to remain silent and not to move during one minute, during which EMG measurements were collected (EMG Post-relaxation measures). Finally, participants were instructed to self-report rumination on the four VASs.

3.3.4 Data processing and analysis

3.3.4.1 EMG data processing

EMG signal pre-processing was carried out using Labchart 8. The EMG data were high-pass filtered using a Finite Impulse Response (FIR) filter at a cut-off of 20 Hz, using the Kaiser window method with $\beta = 6$. Then, output of this first filter was to a low-pass filtered at a cut-off of 450 Hz (with the same parameters), in order to focus on the 20–450 Hz frequency band, following current recommendations for facial EMG studies (A. Boxtel, 2001; De Luca, 1997; De Luca, Donald Gilmore, Kuznetsov, & Roy, 2010).

Although we specifically asked participants to remain silent and not to move during EMG data collection, tiny facial movements (such as biting one's lips) or vocal productions sometimes occurred. Periods with such facial movement or vocal production were excluded from the analysis. To do this, visual inspection of audio, video, and EMG signal was performed. Specifically, for the EMG signals, we compared two methods of signal selection. The first one consisted of setting a threshold on the absolute value of the EMG signal and portions of signals above this threshold were removed. This threshold was empirically chosen using visual inspection of a few samples and set to the mean EMG value plus 6 SDs. The second method consisted of manually removing periods of time that included visually obvious bursts of EMG activity, corresponding to overt contraction (as in Rapin, Dohen, Polosan, & Perrier, 2013). Based on samples from a few participants, the comparisons between these two methods showed that the automatic threshold method was somewhat less sensitive to overt movements. Therefore, the second method was used, as it was more conservative and less prone to leave data related to irrelevant overt movements.

After pre-processing, EMG data were exported from Labchart software to Matlab r2014a (Version 8.3.0.532, www.mathworks.fr). For each EMG signal, mean values were computed under Matlab, using 200 ms sliding windows. The average of these mean values were calculated for each recording session (baseline, after induction and after relaxation/induction). This provided a score for each muscle of interest (OOS, OOI, FCR, FRO) in each Session (Baseline, Post-Induction, Post-Relaxation) for each participant³.

³Because of constraints attributable to the design of our experiment, we were not able to perform conventional control measures (e.g., time of the day, food consumption, sport activity, smoking habits, etc.). Moreover, in our study, periods of signal recording had to be shorter than usual HRV analysis time periods (cf. methodology section). Although recent studies suggest that “ultrashort term” HRV analysis seems to correlate quite well with HRV analysis performed on longer periods of time (Brisinda et al., 2013; Salahuddin, Cho, Jeong, & Kim, 2007), we cannot exclude that our measurements might be unreliable. For these reasons, we chose not to present HRV results in this report and to focus on EMG results as well as subjective reports of

3.3.4.2 Statistical analyses

Absolute EMG values are not meaningful as muscle activation is never null, even in resting conditions, due in part to physiological noise (Tassinari, Cacioppo, & Vanman, 2007). In addition, there are inter-individual variations in the amount of EMG activity in the baseline. To normalise for baseline activity across participants, we used a differential measure and expressed EMG amplitude as a percentage of baseline level (Experiment 1) or of post-induction level (Experiment 2).

To model EMG amplitude variations in response to the rumination induction (Experiment 1) and relaxation (Experiment 2), we used a bayesian multivariate regression model with the natural logarithm of the EMG amplitude (expressed in % of baseline level) as an outcome, in an intercept-only model (in Experiment 1), and using Condition (Orofacial, Arm or Story) as a categorical predictor in Experiment 2. We used the same strategy (two multivariate models) to analyse VAS scores (expressed in relative changes) along the two experiments.

These analyses were conducted using RStudio (RStudio Team, 2018) and the brms package (Bürkner, 2018), an R implementation of Bayesian multilevel models that employs the probabilistic programming language Stan (Carpenter et al., 2017). Stan implements gradient-based Markov Chain Monte Carlo (MCMC) algorithms (e.g., Hamiltonian Monte-Carlo), which allow yielding posterior distributions that are straightforward to use for interval estimation around all parameters. Two MCMC simulations (or “chains”) were run for each model, including 100,000 iterations, a warmup of 10,000 iterations, and a thinning interval of 10. Posterior convergence was assessed examining autocorrelation and trace plots, as well as the Gelman-Rubin statistic. Fixed effects were estimated via the posterior mean and 95% highest density intervals (HDIs), where an HDI interval is the Bayesian analogue of a classical confidence interval⁴.

This strategy allowed us to examine posterior probability distribution on each parameter of interest (i.e., effects of session and condition on each response variable). When applicable, we also report evidence ratios (ERs), computed using the hypothesis function of the brms package (Bürkner, 2018). These evidence ratios are simply the posterior probability under a hypothesis against its alternative (Bürkner, 2018). We also report summary statistics (mean and HDI) of Cohen’s d effect sizes, computed from the posterior samples.

3.4 Results

3.4.1 Experiment 1: rumination induction

The evolution of VAS scores (for the four assessed scales: Feelings, Problems, Brooding, and Focused) and EMG (for the four muscles: OOS, OOI, FCR and FRO) activity from baseline to post-induction were examined.

3.4.1.1 Self-reported rumination measures: VAS scores

Results for VAS relative changes based on the multivariate models described earlier are shown in the right panel of Figure 3.2. Thereafter, α represents the mean of the posterior rumination.

⁴While not suffering from the misunderstandings associated with frequentist confidence intervals (for more details, see for instance Morey et al., 2015).

distribution of the intercept. Raw pre- and post-induction scores are provided in the supplementary materials.

Mean VAS score on the Feelings scale was slightly lower after induction ($\alpha = 5.55$, 95% HDI [-10.89, 0.24], $d = 0.23$, 95% HDI [-0.46, 0.01]), while Problems score was slightly higher ($\alpha = 3.99$, 95% HDI [-2.04, 9.83], $d = 0.15$, 95% HDI [-0.08, 0.37]). We observed a strong increase of the score on the Brooding scale ($\alpha = 14.45$, 95% HDI [8.07, 20.72], $d = 0.50$, 95% HDI [0.26, 0.74]), and a strong decrease on the Focused scale ($\alpha = 11.63$, 95% HDI [-17, 6.07], $d = 0.48$, 95% HDI [-0.72, 0.24]). As we examined the fit of the intercept-only model, these estimates represent the posterior mean for each muscle.

In the following, we report the mean (indicated by the Greek symbol ρ) and the 95% HDI of the posterior distribution on the correlation coefficient (ρ). Examination of the correlation matrix estimated by the multivariate model revealed no apparent correlation neither between Feelings and Problems scales ($\rho = 0.01$, 95% HDI [-0.23, 0.22]), nor between Feelings and Brooding ($\rho = 0.08$, 95% HDI [-0.15, 0.30]). However, we observed a strong positive correlation between Problems and Brooding VASs ($\rho = 0.64$, 95% HDI [.49, 0.76]), a positive correlation between Feelings and Focused ($\rho = 0.30$, 95% HDI [.08, 0.50]), and a negative correlation between Problems and Focused ($\rho = 0.30$, 95% HDI [-0.49, 0.08]), as well as between Brooding and Focused ($\rho = 0.18$, 95% HDI [-0.39, 0.05]).

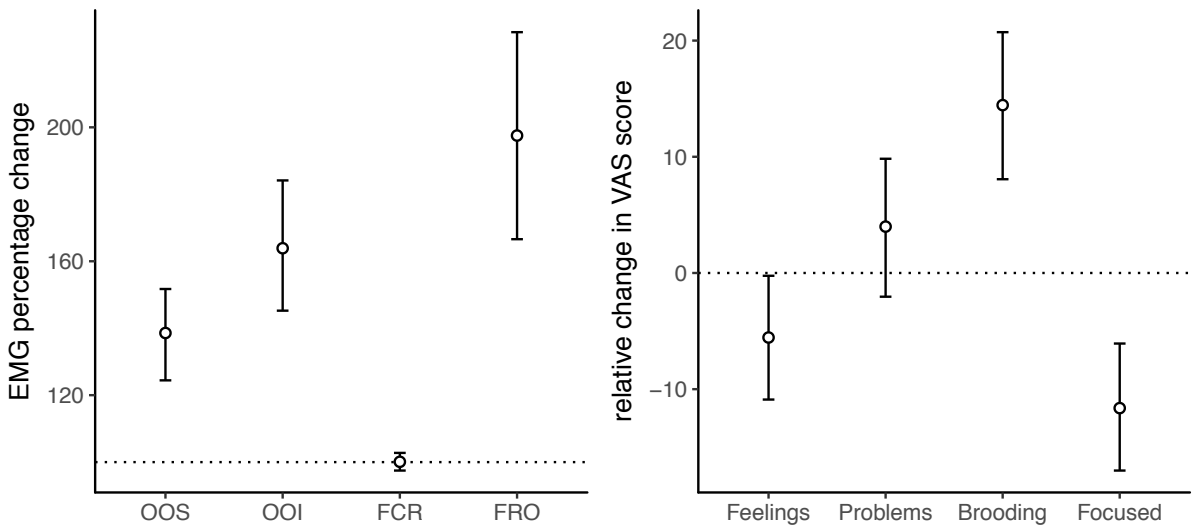


Figure 3.2: Posterior mean (white dots) and 95% credible intervals for the EMG amplitude (expressed in percentage of baseline level, left panel), and the VAS score (expressed in relative change from baseline, right panel). $N = 71$ (for each muscle and each VAS). Dashed line represents the null value (i.e., 100% for the EMG amplitude and 0 for the VAS scores).

3.4.1.2 EMG

Results for EMG data based on the multivariate model described earlier are shown in the left panel of Figure 3.2. Summary statistics were computed on posterior samples transformed back from log scale.

Mean EMG amplitude for OOS was higher after induction ($\alpha = 138.57$, 95% HDI [124.43, 151.71], $d = 0.66$, 95% HDI [0.49, 0.84]) as well as for OOI ($\alpha = 163.89$, 95% HDI [145.24, 184.14], $d = 0.77$, 95% HDI [0.61, 0.94]), and FRO ($\alpha = 197.55$, 95% HDI [166.59, 228.42], d

= 0.74, 95% HDI [0.59, 0.89]). Effects on the FCR were approximately null ($\alpha = 100.10$, 95% HDI [97.48, 102.76], $d = 0.01$, 95% HDI [-0.24, 0.23]).

Examination of the correlation matrix estimated by the bayesian multivariate model revealed a positive correlation between OOS and OOI EMG amplitudes ($\rho = 0.44$, 95% HDI [0.24, 0.61]), while no apparent correlations neither between OOS and FCR ($\rho = 0.09$, 95% HDI [-0.14, 0.31]), OOS and FRO ($\rho = 0.12$, 95% HDI [-0.11, 0.35]), OOI and FCR ($\rho = 0.02$, 95% HDI [-0.21, 0.25]), FRO and FCR ($\rho = 0.06$, 95% HDI [-0.28, 0.17]), nor OOI and FRO ($\rho = 0.07$, 95% HDI [-0.16, 0.29]). Scatterplots, marginal posterior distributions and posterior distributions on correlation coefficients are available in supplementary materials.

In order to check whether the propensity to ruminate could predict the effects of the rumination induction on EMG amplitude, we compared the multivariate model described above, with a similar model but with the score on the abstract dimension of the Mini-CERTS as an additional predictor. We compared these models using the widely applicable information criterion (WAIC; Watanabe, 2010), via the WAIC function of the *brms* package (Bürkner, 2018). Results showed that the intercept-only model had a lower WAIC (WAIC = 177.39) than the more complex model (WAIC = 182.01), indicating that there is no predictive benefit in adding the Mini-CERTS score as a predictor.

3.4.1.3 Correlations between EMG amplitudes and VAS scores

Correlations between EMG amplitudes and VAS scores were examined using the *BayesianFirstAid* package (Bååth, 2018), using 15,000 iterations for each correlation coefficient. Both estimated correlation coefficients (ρ s) and 95% HDIs are reported in Table XX.

3.4.2 Experiment 2: rumination reduction by relaxation

In the second experiment, we aimed at comparing the evolution in EMG activity and VAS scores from post-induction to post-relaxation in three different conditions: Orofacial relaxation, Arm relaxation, and listening to a Story.

3.4.2.1 Self-reported rumination measures: VAS scores

Posterior means and 95% HDIs of the VAS scores in each condition of experiment 2 are represented in Figure XX and Table XX.

In order to compare the effects of the two kind of relaxation on the VAS scores, we then used the *hypothesis()* function of the *brms* package that allows deriving evidence ratios (ER). These evidence ratios are simply the posterior probability under a hypothesis (e.g., the hypothesis that the Orofacial relaxation session would be more effective in reducing self-reported rumination than the Arm relaxation session) against its alternative (Bürkner, 2018).

Since the Problems and the Brooding scales seemed to be sensitive markers of rumination (as their scores increased after induction in Experiment 1), our analyses were focused on these two scales.

Concerning the Problems VAS, the decrease observed in the Orofacial condition was more pronounced than in the Arm condition (Est = 11.06, SE = 6.35, ER10 = 22.65), and slightly more pronounced compared to the Story condition (Est = 6.05, SE = 6.31, ER10 = 4.98). The observed on the Brooding VAS score in the Orofacial condition was larger than

in the Arm condition (Est = 9.98, SE = 6.07, ER10 = 18.85), and slightly more important compared to the Story condition (Est = 5.23, SE = 6.01, ER10 = 4.27).

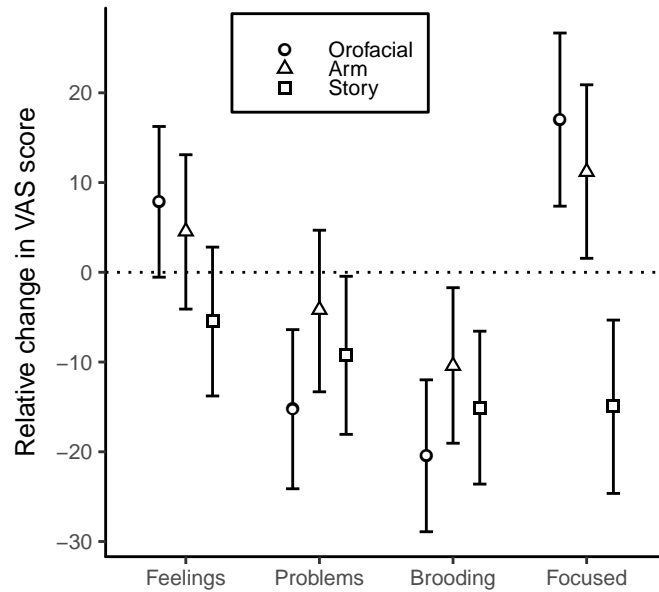


Figure 3.3: Posterior mean and 95% credible intervals for the VAS score (expressed in relative change from post-induction level).

3.4.2.2 EMG

Posterior means and 95% HDIs of the EMG amplitude in each condition of experiment 2 are represented in Figure XX and reported in Table XX. We used the same strategy as before to compare the effects of the two kinds of relaxation on the EMG amplitudes.

Concerning the OOS, the observed decrease in the Orofacial condition was more pronounced than in the Arm condition (Est = 0.34, SE = 0.14, ER10 = 140.73), as well as concerning the OOI (Est = 0.35, SE = 0.19, ER10 = 29.46), while we observed no noticeable differences between the two kinds of relaxation concerning the EMG amplitude of the FRO (Est = -0.04, SE = 0.14, ER10 = 1.53).

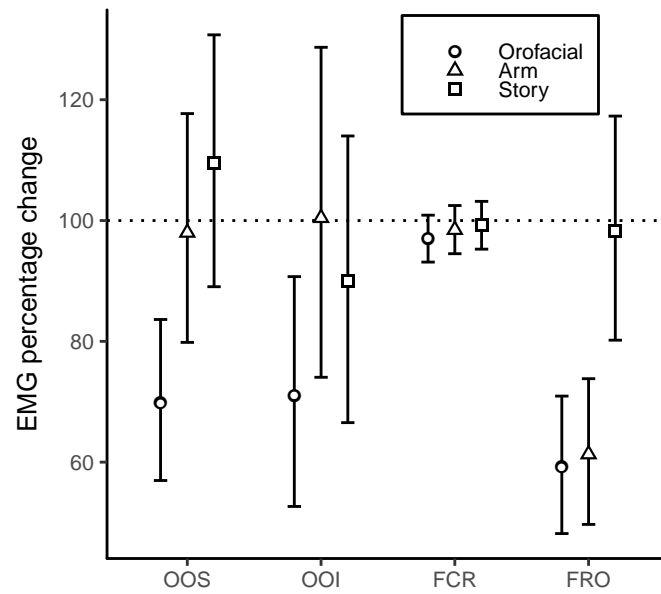


Figure 3.4: Posterior mean and 95% credible intervals for the VAS score (expressed in relative change from post-induction level).

3.5 Discussion

3.5.1 Experiment 1

In the first experiment, we examined electromyographic correlates of induced rumination in healthy individuals. According to the *Motor Simulation view*, we predicted an increase in the activity of all facial muscles after the rumination induction, associated with an increase in self-reported rumination. Alternatively, the *Abstraction view* predicted an increase in self-reported rumination associated with an increase in forehead activity with no changes in either lip or forearm activity.

To test the predictions of these two theoretical views, we compared EMG measures and VAS scores after induction to their values before induction. EMG activity was examined in four muscles: OOS and OOI, two muscles involved in speech production, FRO, a facial negative- affect-related but not speech-related muscle, and FCR, a non-facial control muscle on the non-dominant forearm.

As predicted by the *Motor Simulation view*, we observed an increase in the activity of the two speech-related muscles (OOS & OOI) as well as in the negative-affect-related muscle (FRO) and no change in FCR activity. The increase in facial EMG together with the increase in the subjective reports of rumination suggests that facial EMG increase is a correlate of verbal rumination. As supported by several studies results, the forehead muscle activity has been associated with unpleasant emotions (Jäncke et al., 1996) or anxiety (A. Conrad & Roth, 2007). The increase in FRO activity observed here is consistent with the increase in negative emotions induced by our negatively valenced induction procedure. *Orbicularis oris* lip muscles are associated with speech production. The increase in lip activity observed here suggests that the speech motor system was involved during the ruminative phase. The fact that the FCR remained stable after rumination induction suggests that the observed facial

activity increase was not due to general body tension induced by a negative mental state. These facial EMG results therefore support the hypothesis that rumination is an instance of articulatory-specified inner speech.

After the rumination induction, a larger increase in OOI activity was observed compared to the increase in OOS activity. This finding is consistent with previous findings of higher EMG amplitude in the lower lip during speech and inner speech (e.g., S. M. Barlow & Netsell, 1986; Regalo et al., 2005; Sokolov, 1972) or auditory verbal hallucinations (Rapin et al., 2013). Rapin et al. (2013) have explained the difference between the activities of the two lip muscles by muscle anatomy. The proximity of the OOI muscle with other speech muscles (such as the depressor angular muscle or the mentalis) could increase the surface EMG signal captured on the lower lip (OOI), as compared to the upper lip (OOS) during speech. An even larger increase in FRO activity was observed compared to the increase in lip muscle activity. As EMG amplitude is known to vary with muscle length (Babault et al., 2003), the greater increase in frontalis activity could be explained by its anatomical properties.

However, although a functional distinction can be drawn between the forehead and the lip muscles, one should acknowledge the fact that these two sets of muscles can be commonly activated during some behaviours. For instance, A. van Boxtel & Jessurun (1993) have shown that orbicularis oris inferior and frontalis were both activated during a two-choice serial reaction task in which nonverbal auditory or visual signals were presented. Moreover, there was a gradual increase in EMG activity in these muscles during the task, either when the task was prolonged or when the task was made more difficult. They interpreted this increase in EMG activity as associated with a growing compensatory effort to keep performance at an adequate level. An alternative interpretation is that the increase in task difficulty was dealt with by inner verbalization. Covertly rehearsing the instructions or covertly qualifying the stimuli might have helped the participants to perform adequately. Therefore, the increase in orbicularis oris activity might have been related to an increase in covert verbalization, whereas the increase in frontalis activity might have been related to increased anxiety or tension. The fact that the EMG increase was muscle specific, and that some facial muscles (*orbicularis oculi*, *zygomaticus major*, *temporalis*) did not show an increase in activity unless the task became too difficult, supports this interpretation. It cannot be ruled out, however, that orbicularis oris activity may in some cases be related to mental effort without mental verbalisation. Nevertheless, although the I.Q. test itself was designed to induce mental effort, no cognitively demanding task was asked to the participant during the period of EMG recording (i.e., approximately four minutes after the end of the test). Although we cannot absolutely exclude that rumination in itself could require cognitive effort, it seems unlikely that mental effort was the main factor of variation.

Scores on the VAS need to be discussed in further detail. We examined which VAS scales were most suitable to capture changes in state rumination to allow focused analyses. Due to the “pre-baseline” relaxation session, during which participants were asked to concentrate on their body and breathing cycles, participants reported a high level of attentional self-focus at baseline (“Feelings” and “Focused” VAS). Because of the high level of self-focused attention at baseline, it is likely that the scores on the “Feelings” and “Focused” VAS did not show the expected increase after rumination induction (ceiling effect). The scores on the scales “Problems” and “Brooding”, which are more representative of maladaptive rumination, did increase after our rumination induction paradigm, however. Interestingly, the “Brooding” VAS corresponded to a larger increase and seemed to be more sensitive to rumination induction than the “Problems” VAS. Given this greater sensibility and the strong positive correlation between the “Brooding” and the “Problems” VAS, it

thus make sense to consider the “Brooding” VAS as a better estimate of ruminative state, at least within our paradigm. We will therefore only use this scale to assess rumination in the following.

The fact that we did not observe any association between the propensity to ruminate (as measured by the Mini-CERTS questionnaire) and the effects of the induction is consistent with the results of Rood, Roelofs, Bögels, & Arntz (2012) who found that the level of trait rumination did not moderate the effects of a rumination induction.

3.5.2 Experiment 2

In the second experiment, we studied the effects of two muscle-specific relaxation sessions: Orofacial relaxation and Arm relaxation. We compared their effects to a third control condition (Story), which did not involve the deliberate relaxation of any specific muscle. Our predictions were that a decrease in facial EMG activity should be observed in each condition. If the *Motor Simulation view* is correct, we expected a larger decrease in the activity of all facial muscles in the “Orofacial relaxation” condition than in the “Arm relaxation” condition, associated with a larger decrease in self-reported rumination. Additionally, we expected a more pronounced decrease in the two relaxation conditions (orofacial and arm relaxation conditions) than in the control (“Story”) condition. We also expected no difference between relaxation conditions regarding the change in the forearm muscle activity.

The data indicated a decrease in self-reported rumination (“Brooding” VAS) in each condition. The “Orofacial” relaxation condition elicited a slightly larger decrease than the “Arm relaxation” or the “Story” condition. However, there was extensive individual variation in response to these conditions. As concerns EMG results, we observed a decrease in OOS and OOI activities in all three conditions but this decrease was more pronounced in the orofacial condition than in the other two conditions. The frontalis activity did not show the same pattern. A similar FRO activity decrease was observed in both the orofacial and the non-orofacial relaxation conditions. Therefore, in Experiment 2, the lip muscles and the forehead muscle follow differential evolutions. A dissociation was observed: whereas both orofacial and arm relaxations resulted in a decrease in forehead activity, only orofacial relaxation was successful at reducing lip activity.

Considering both VAS results and the dissociation in EMG patterns, several interpretations are possible. The first interpretation is that verbal production associated with rumination was more reduced by orofacial muscular relaxation than by non-orofacial relaxation. This interpretation is consistent with the fact that the “Brooding” VAS was slightly more decreased in this condition compared to the other two. The larger decrease in OOS and OOI amplitude after orofacial relaxation would thus reflect this reduction in verbal production, as hypothesised by the *Motor Simulation view*. The fact that FRO activity displayed a similar decrease in both orofacial and non-orofacial relaxation conditions could suggest that any means of body relaxation (be it orofacial or not) is appropriate to reduce negative affect and can therefore reduce forehead contraction. This suggests that the FRO activity increase presumably reflected negative affect and tension (such as observed in EMG studies on generalised anxiety disorder patients, see A. Conrad & Roth, 2007, for a review).

Alternatively, one could also argue that the larger decrease in lip muscle activity after orofacial relaxation finds a more trivial explanation in that it seems obvious to expect that orofacial relaxation will be more efficient to reduce lip muscle contraction than non-orofacial relaxation. Thus, the different impacts of the two relaxation sessions

on the lip muscles would not be related to reduced rumination per se but simply to a more anatomically targeted relaxation. However, several observations argue against such an interpretation. The larger decrease in the “Brooding” VAS in the orofacial relaxation condition compared with the other conditions suggests that the reduction in lip muscle activity is indeed related to the reduction in rumination. Moreover, an interpretation solely based on anatomical links does not explain why FRO activity displayed the same amount of reduction in both relaxation sessions. If reduction in muscle activity was merely related to the effect of facial muscle relaxation, then the decrease in FRO activity should have also been higher in the orofacial relaxation condition than in the other relaxation condition, which was not the case. Therefore the dissociation between forehead and lip patterns of activity, together with the differential effects of the two types of relaxation on subjective rumination reports strongly suggest that different processes underlie the activity of these two sets of muscles. We therefore consider that the first interpretation is more plausible: frontalis activity seems related to overall facial tension due to negative affect whereas lip activity seems to be related to the specific involvement of the speech musculature in rumination. These results thus seem to confirm the interpretation of decreased OOS and OOI activities in the orofacial relaxation condition as markers of rumination reduction.

Interestingly, we observed no changes of forearm EMG activity in any of the three conditions of Experiment 2. The fact that the relaxation session focused on the forearm was not associated with a decrease in FCR activity has a simple explanation: FCR activity had not increased after rumination induction and had remained at floor level. The forearm was thus already relaxed and the Arm relaxation session did not modify FCR activity. Another interesting conclusion related to this absence of modification of forearm activity is that relaxation does not spuriously decrease muscle activity below its resting level. One possible interpretation of the increase in lip EMG after rumination induction could have been that baseline relaxation artificially decreased baseline activity under its resting level. The facts that forearm activity did not decrease after arm-focused relaxation contradicts this interpretation.

Finally, the “Story” condition was also associated with a decrease in OOI and FRO activities. This could mean that listening to a story reduced rumination to the same extent as relaxation did. However, the discrepancy observed in “Focused” VAS between the two relaxation conditions on the one hand and the control condition on the other hand, suggests that the EMG decrease observed in the “Story” condition might be attributable to a different cause than that observed in the two relaxation conditions. Listening to a story could help reducing rumination by shifting attention away from ruminative thoughts. Relaxation sessions could help reducing rumination by shifting attention to the body in a beneficial way.

3.5.3 General discussion

We set out two experiments to examine whether rumination involves motor simulation or is better described as linguistically abstract and articulatory impoverished. We used labial, facial, and arm EMG measures to assess potential articulatory correlates of rumination. The patterns of results of our study seem to be in favour of the motor nature of verbal rumination. In Experiment 1, rumination induction was associated with a higher score on the scale “I am brooding about negative things” which is representative of abstract-analytical rumination, considered as verbal rumination. This maladaptive rumination state was associated with an increase in the activity of two speech-related

muscles, without modification of the arm muscle activity, which indicates that rumination involves activity in speech articulatory muscles, specifically. The concurrent increase in forehead muscle activity could be explained by an increase in negative emotions induced by our negatively valenced induction procedure. The results of Experiment 1 therefore show the involvement of the speech musculature during rumination. This is in line with the *Motor simulation view*, according to which inner speech is fully specified at the articulatory level, not just the lexical level.

In Experiment 2, guided relaxation resulted in a decrease in speech muscle activity. In the lip muscles, the activity decrease was stronger after orofacial relaxation than after arm-focused relaxation. In the forehead muscle, however the effect was the same for both types of relaxation. This decrease in speech muscle activity was associated with a decrease in self-reports of rumination and was most pronounced after orofacial relaxation. These findings suggest that a reduction in speech muscle activity could hinder articulatory simulation and thus limit inner speech production and therefore reduce rumination. This interpretation is consistent with the *Motor Simulation view* of inner speech. Brooding-type rumination was also diminished after the arm-focused relaxation as well as after listening to a story, although less than in the orofacial relaxation. This suggests that general relaxation or distraction are also likely to reduce negative rumination. To summarize, experiments 1 and 2 are consistent with the *Motor Simulation view* of inner speech, according to which speech muscle activity is inherent to inner speech production. Experiment 1 shows the involvement of the lip musculature during brooding-type rumination. Experiment 2 suggests that brooding-type rumination could be reduced by blocking or relaxing speech muscles.

These data support the utility of labial EMG as a tool to objectively assess inner speech in a variety of normal and pathological forms. We suggest that this method could be used as a complement to self-report measures, in order to overcome limitation of these measures.

Our results should be interpreted with some limitations in mind. First, our sample consisted exclusively of women. Although this methodological choice makes sense considering the more frequent occurrence of rumination in women, further studies should be conducted to ascertain that our results may generalize to men. Second, in Experiment 1, no between-subject control condition was used to compare with the group of participants who underwent rumination induction. Thus, we cannot rule out that other processes occurred between baseline and rumination induction, influencing responding. Thirdly, substantial inter-individual differences were observed concerning the size of the effect of rumination induction on facial EMG activity. The results of Jäncke (Jäncke, 1996; Jäncke et al., 1996) can shed light on this last result. Jäncke used a similar procedure (i.e., negative mood induction using a false I.Q. test and facial EMG measurements to assess emotions), except that the experimenter was not in the room while participants performed the test and acknowledged their results. The experimenter then came back to the room and analysed participants' behaviours. Jäncke observed an increase in facial muscular activity (assessed when participants were reading their results) only in participants who were prone to express their distress when the experimenter came back, while more introverted participants did not show any increased facial activity when reading their results. Jäncke interpreted these results in the framework of an ecological theory of facial expression, suggesting that facial expressions would not only be guided by underlying emotions, but also by their communicative properties. Considering these results, it seems likely that the proneness of participants to communicate their emotions could have mediated effects of the induction on their facial EMG activity. This could partially explain the observed

inter-individual variability in facial EMG activity associated with rumination. Moreover, even though rumination is a predominantly verbal process, one cannot exclude that some of our participants experienced rumination in another modality (e.g., imagery-based rumination), which would explain their lower than average lip activity.

Thus, a logical next step is to examine qualitative factors that mediate the link between rumination and facial muscular activity. These factors (among others) could be proneness to communicate emotion or proneness to verbalize affects. Additionally, recent studies suggest a link between verbal aptitudes and propensity to ruminate. Uttl, Morin, & Hamper (2011) have observed a weak but consistent correlation between the tendency to ruminate and scores on a verbal intelligence test. Penney, Miedema, & Mazmanian (2015) have observed that verbal intelligence constitutes a unique predictor of rumination severity in chronic anxious patients. To our knowledge, the link between verbal intelligence and induced rumination has never been studied. It would be interesting to examine whether the effects of a rumination induction could be mediated by verbal intelligence, and to what extent this could influence related facial EMG activity.

In conclusion, this study provides new evidence for the facial embodiment of rumination, considered as a particular instance of inner speech. Even if more data are needed to confirm these preliminary conclusions, our results seem to support the *Motor Simulation view* of inner speech production, manifested as verbal rumination. In addition, facial EMG activity provides a useful means to objectively quantify the presence of verbal rumination.

3.6 Acknowledgements

This project was funded by the ANR project INNER SPEECH. The first author of the manuscript is funded by a fellowship from Université Grenoble Alpes and a grant from the Pôle Grenoble Cognition. We thank Nathalie Vallet for recording the relaxation and distraction sessions. We thank our colleagues from GIPSA-lab: Marion Dohen for her help in the recording of the audio stimuli in the anechoic room at GIPSA-lab, as well as Christophe Savariaux and Coriandre Vilain for their advice in the audio setup associated with the EMG measures. We are also grateful to Rafael Laboissière and Adeline Leclercq Samson for their advice concerning data analysis. We sincerely thank two anonymous reviewers for their critical reading of our manuscript and their many insightful comments and suggestions. Access to the facility of the MSH-Alpes SCREEN platform for conducting research is gratefully acknowledged.

3.7 Supplementary data

Supplementary data associated with this article can be found at <https://osf.io/882te/>.

Summary of Chapter 3

In this first experiment we... Rumination is predominantly experienced in the form of repetitive verbal thoughts. Verbal rumination is a particular case of inner speech. According to the Motor Simulation view, inner speech is a kind of motor action, recruiting the speech motor system. In this framework, we predicted an increase in speech muscle activity during rumination as compared to rest. We also predicted increased forehead activity, associated with anxiety during rumination. We measured electromyographic activity over the orbicularis oris superior and inferior, frontalis and flexor carpi radialis muscles. Results showed increased lip and forehead activity after rumination induction compared to an initial relaxed state, together with increased self-reported levels of rumination. Moreover, our data suggest that orofacial relaxation is more effective in reducing rumination than non-orofacial relaxation. Altogether, these results support the hypothesis that verbal rumination involves the speech motor system, and provide a promising psychophysiological index to assess the presence of verbal rumination.

Blah blah...

DISSOCIATING FACIAL ELECTROMYOGRAPHIC CORRELATES OF VISUAL AND VERBAL INDUCED RUMINATION

Summary of the research...¹

Summary of Chapter 4

Blah blah ...

¹This experimental chapter is a submitted manuscript reformatted for the need of this thesis. Pre-registered protocol, preprint, data, as well as reproducible code and figures are available at: <https://osf.io/c9pag/>.

MUSCLE-SPECIFIC ELECTROMYOGRAPHIC CORRELATES OF INNER SPEECH PRODUCTION

Summary of the research...¹

Summary of Chapter 5

Blah blah ...

¹This experimental chapter is a submitted manuscript reformatted for the need of this thesis. Pre-registered protocol, preprint, data, as well as reproducible code and figures are available at: <https://osf.io/czer4/>.

ARTICULATORY SUPPRESSION EFFECTS ON INDUCED RUMINATION

Summary of the research...¹

Summary of Chapter 6

Blah blah ...

¹This experimental chapter is a submitted manuscript reformatted for the need of this thesis. Pre-registered protocol, preprint, data, as well as reproducible code and figures are available at: <https://osf.io/3bh67/>.

REFINING THE INVOLVEMENT OF THE SPEECH MOTOR SYSTEM DURING RUMINATION: A DUAL-TASK INVESTIGATION

Summary of the research...¹

Summary of Chapter 7

Blah blah ...

¹This experimental chapter is a submitted manuscript reformatted for the need of this thesis. Pre-registered protocol, preprint, data, as well as reproducible code and figures are available at: <https://osf.io/8ab2d/>.

Part III

Discussion and conclusions

DISCUSSION AND PERSPECTIVES

8.1 Summary of the results

...

8.2 Benchmarks for theories of inner speech

A revised version of MacKay (1992) constraints...

8.3 Limitations and ways forward

...

8.4 Conclusions

...

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