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Adaptive Memory in Contamination Contexts: Emotionality as a Proximate Mechanism

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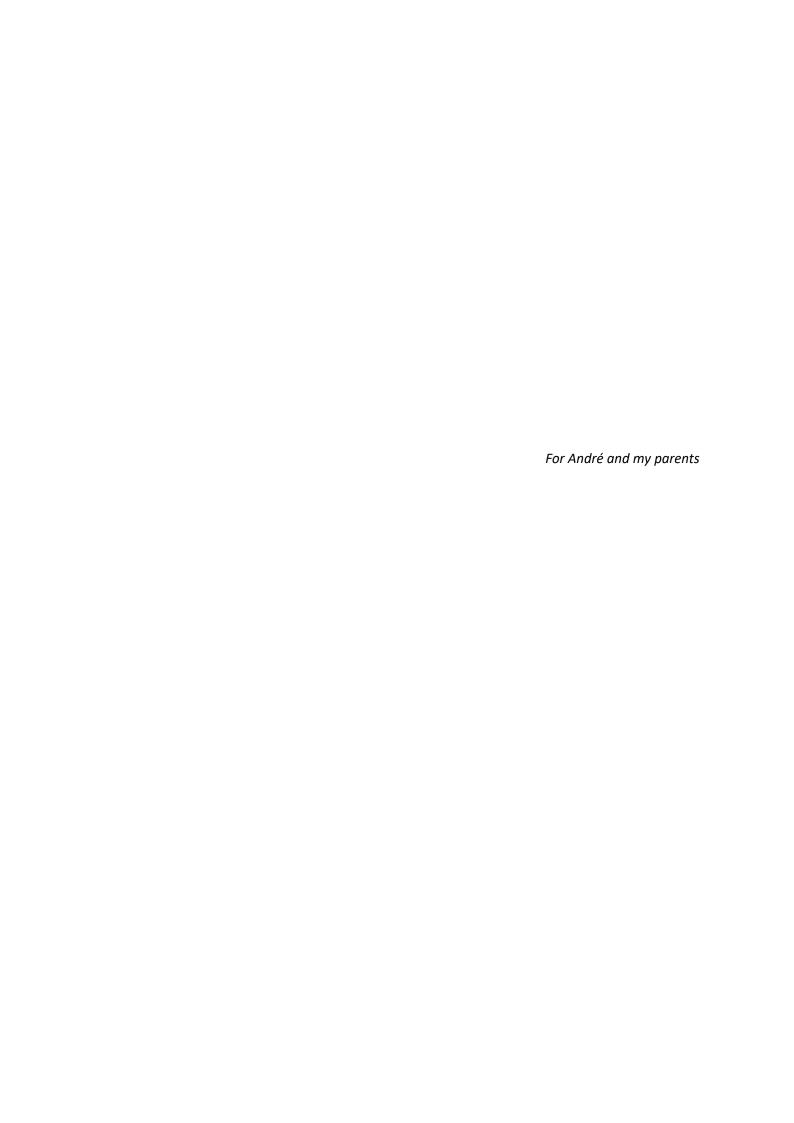
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Now that I have reached this academic milestone, I want to turn my attention to the people who have helped and encouraged me along the way. First, I am grateful to those who introduced me to the field of Social Psychology, where I have discovered my passion in life.

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Resumo

O sistema imunitário comportamental é um sistema de defesa desenvolvido através da seleção

natural. Este sistema coordena uma série de respostas com o principal objetivo de evitar o contacto

com agentes patogénicos. A memória é uma componente essencial deste sistema, e os indivíduos têm

melhor desempenho na recordação de pistas de contaminação. No entanto, o mecanismo proximal

desta vantagem mnésica ainda está sujeito a debate. Os estudos anteriores concentram-se

principalmente nos processos cognitivos como potenciais mecanismos proximais. Em contraste, a

presente investigação focou-se no papel da emocionalidade. Para esse fim, tivemos o objetivo de

replicar o efeito de contaminação e expandir estudos anteriores ao determinar de que forma as pistas

de contaminação impactam os indicadores psicofisiológicos. Os resultados revelaram que o

desempenho na evocação foi superior para objetos codificados como potenciais fontes de

contaminação do que para objetos neutros. Além disso, na exposição de pistas de contaminação, as

desacelerações da frequência cardíaca foram significativamente maiores do que na exposição a pistas

neutras. Estas evidências confirmam a robustez do efeito de contaminação e apontam para a

emocionalidade evocada por ameaças como um mecanismo proximal. Estes resultados têm

implicações na explicação de como o sistema imunitário comportamental opera e para o quadro-

teórico da memória adaptativa.

Palavras-chave: memória adaptativa, contaminação, emocionalidade, frequência cardíaca

Códigos de Classificação da APA:

2340 Processos Cognitivos

2343 Aprendizagem e Memória

2560 Psicofisiologia

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Abstract

The behavioral immune system is a defense system developed by natural selection and coordinates a

series of responses with the main objective of avoiding contact with pathogens. Memory is an essential

component of this system and is tuned for contamination-threatening cues. However, how this tuning

is achieved remains unclear. Previous research has focused mainly on cognitive processes as potential

proximate mechanisms. In contrast, the present research examines the role of emotionality. For this

purpose, we aimed to replicate the contamination effect and expand upon earlier studies by

investigating how contamination-threatening cues impact psychophysiological indicators. The results

demonstrated a significantly higher recall performance for objects touched by a sick person than by a

healthy person, thus replicating the effect. Furthermore, when contamination-threatening cues were

shown, the heart rate decelerated significantly more than when healthy cues were shown. These

findings confirm the robustness of the contamination effect and provide insight into the emotionality

elicited by threat as a proximate mechanism for memory tuning. These results have implications for

explaining how the behavioral immune system operates and for the adaptive memory framework.

Keywords: adaptive memory, contamination, emotionality, heart rate

APA Classification Categories:

2340 Cognitive Processes

2343 Learning & Memory

2560 Psychophysiology

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Introduction

From an evolutionary perspective, a single cue of a possible contamination threat being enough to trigger a mnemonic advantage constitutes a plausible adaptive way of coping because it increases the chances of survival and reproduction (Nairne, 2010). However, which underlying mechanism explains this advantage? The most established mechanisms suggest differential involvement or specific cognitive processes. Some examples are item-specific, richness, relational, and self-referential processing (Burns et al., 2011; Klein, 2012; Kroneisen & Erdfelder, 2011; Nairne et al., 2008).

The behavioral immune system (BIS), which was developed by natural selection, is paramount in a contamination-threatening context, as it activates a cascade of mechanisms to help avoid contact with contaminants and maintain the individual's well-being (e.g., Schaller, 2011). Disgust influences this defense system's activation and response magnitude (Nesse, 2015). Previous studies also demonstrated that disgust related to contamination threat benefits recall performance — the "contamination effect" (Fernandes et al., 2017). Notably, pathogenic disgust has been found to impact memory more than fear (Moeck et al., 2021). Yet, little research has focused on the emotionality related to threats as a potential proximate mechanism of the contamination effect. Moreover, most research in this domain has explored the role of emotionality through self-reported responses. However, participants in such studies only manage to report rationalizations for their interpretation of the stimuli, which inevitably results in the indirect measuring of emotionality with a certain degree of error. Others have used a control scenario inducing emotionality that was unrelated to threats, thereby not comparable to the manipulation scenario because not all emotions are equal in physiological correlates regardless of arousal levels (Bradley et al., 2001; Lang & Bradley, 2013).

The present study tackled this gap by examining emotionality with psychophysiological measures such as Heart Rate (HR). The few studies using comparable control conditions (Kazanas et al., 2021; Saraiva et al., 2021) or psychophysiological measures (Fiacconi et al., 2015) have found evidence supporting emotionality as a proximate mechanism of the memory advantage for stimuli processed in survival contexts. However, they only examined contexts that induce fear, not the ones that evoke pathogenic disgust. Here, we reason that pathogenic disgust would play a fundamental role in establishing emotionality as a proximate mechanism of the contamination effect. Furthermore, such evidence would support the view that disgust might be a critical component for processing threats and activating a cascade of defense mechanisms, such as those related to memory, designed to guide us in life-threatening contexts.

The goal of the current study was twofold. First, given that reproducibility is a cornerstone of scientific research (Open Science Collaboration, 2015), we aimed to replicate the contamination effect,

which was previously outlined as higher recall performance for objects encoded as sources of contamination than for objects encoded as sources of non-contamination. Second, we aimed to extend upon previous work by examining the role of emotionality in the effect, namely whether the HR responses differed when participants were exposed to contamination-threatening cues compared to neutral ones.

Given these objectives, Chapter 1 introduces the concept of BIS and briefly reviews its relation to pathogenic disgust and adaptive memory. Next, we review the main studies that explored the contamination effect. We also refer to studies on the survival processing effect to establish a parallel regarding potential proximate mechanisms. At the end of this chapter, we argue that emotionality is a possible proximate mechanism for the contamination effect. Chapters 2 and 3 present our empirical research, which is a replication and expansion of the experiments of Fernandes et al. (2017, 2021). Lastly, Chapter 4 summarizes the key findings, highlights the main contributions, and discusses the main limitations of the present investigation, as well as some possible avenues for future research.

CHAPTER 1

Literature Review

1.1. The Behavioral Immune System

The naked eye cannot see microbes, but they are usually in our bodies and all around us. While most microbes are harmless, some are pathogens (e.g., viruses) that can be dangerous to our survival and reproduction. Since the dawn of humanity, these invisible threats have been present (e.g., Spyrou et al., 2019), and infectious diseases have been one of the most common causes of morbidity and death over time (Dobson & Carper, 1996; Inhorn & Brown, 1990). Nowadays, people still suffer from these threats (World Health Organization, 2022). Therefore, pathogens are possibly one of the most potent selective pressures shaping human evolution (Fumagalli et al., 2011; Kelley et al., 2005).

Immunity against pathogens is thereby essential for our longevity and well-being. Notably, when we encounter a sick individual, enhancing the immune response from white blood cells and avoiding that person are two distinct processes by nature. Nevertheless, they operate together to increase our overall immunity. Schaller and Duncan (2007) proposed that natural selection designed two immune systems — one biological and one behavioral — to protect humans from pathogenic threats. Each one coordinates numerous mechanisms. Even though these systems are distinct and offer unique benefits, several researchers (e.g., Miller & Maner, 2011; Schaller et al., 2010; Schrock et al., 2020; Thiebaut et al., 2021; Tybur et al., 2020) claim that they are interlocked and function together in a concurrent and complementary way. For example, the mere perception of photographs or videos depicting realistic scenarios with higher contagion risk can trigger a rise in salivary antibody production (Keller et al., 2022; Schaller et al., 2010).

The biological immune system is a highly complex set of physiological mechanisms that primarily facilitate adaptive immune responses to detect and eradicate pathogens in the organism (e.g., Farmer et al., 1986). This system mainly acts as a reactive line of defense (Schaller, 2011, 2016). However, most pathogens reproduce in a matter of hours and quickly evolve to resist immune responses (Gootz, 2010). Moreover, biological immune defenses may entail metabolic costs to the organism (Lochmiller & Deerenberg, 2000; Murray & Schaller, 2016). Therefore, avoiding contact with pathogens might be crucial. Our bodies are provided with a BIS for that purpose (Schaller & Duncan, 2007). The BIS is a set of psychological mechanisms that primarily facilitate adaptive responses to detect and guide the organism's behavior toward avoiding exposure to potential sources of contamination so that it can prevent infections (Ackerman et al., 2018; Schaller, 2006, 2011, 2016; Schaller & Park, 2011). This system mainly acts as a proactive defense (Murray & Schaller, 2016).

The BIS is a unique motivational system that elicits and coordinates a cluster of affective, cognitive, and behavioral responses associated with the fundamental purpose of avoiding disease (Murray & Schaller, 2016; Neuberg et al., 2011; Schaller et al., 2017). The first step to activate the BIS is to prime disgust with perceptual or inferential cues of disease (e.g., a person with a runny nose). This process is sensitive to the point that we can detect facial and olfactory disease cues in others merely hours after their immune systems have been activated (Regenbogen et al., 2017). When the BIS is engaged, a cascade of affective and cognitive processes relevant to coping with pathogenic threats and avoidance behaviors is set in motion (Murray & Schaller, 2016; Tybur et al., 2013). For example, when people detect a cue of potential contamination, they experience disgust. Initially, bottom-up attention is deflected away from the cue to minimize exposure, but then top-down control mechanisms direct more attention to the cue to ensure a quick response (Liu et al., 2015; Zhang et al., 2017; Zimmer et al., 2015). People also retain those cues (Bonin et al., 2019; Fernandes et al., 2017, 2021; Gretz & Huff, 2019; Miller & Maner, 2012; Thiebaut et al., 2022). Subsequently, they inhibit contact with contaminated individuals or objects touched by them (Ackerman et al., 2018; Rozin et al., 1986; Schaller & Park, 2011; Tybur et al., 2016; Tybur & Lieberman, 2016).

The BIS possibly has deep evolutionary roots (Murray & Schaller, 2016; Schaller & Duncan, 2007; Tybur et al., 2013). This stance is supported by the large body of evidence showing that animals also manifest avoidance behaviors toward sources of contamination. For example, ants proactively line their nests with antibiotic resins due to latent infection risk (Chapuisat et al., 2007). Likewise, mice (Kavaliers et al., 2003), bullfrog tadpoles (Kiesecker et al., 1999), social lobsters (Behringer et al., 2006), and mandrill monkeys (Poirotte et al., 2017) reactively avoid conspecifics injected with pathogens.

Notably, we can sometimes interpret disease cues even in objectively innocuous situations (Michalak et al., 2020). However, Haselton and Nettle (2006) argue that since false positive errors are less costly than false negative errors, cognitive mechanisms associated with disease avoidance evolved to adaptively manage the fitness costs of appraisal errors. For that reason, the BIS, along with pathogenic disgust, functions like smoke detectors and heuristically infers threat even to stimuli that resemble infection risk but are objectively harmless (Nesse, 2015). For example, this overgeneralization of disgust occurs over chocolate fudge that just happens to be molded into the shape of feces (i.e., the law of similarity; Rozin et al., 1986) or photographs of objects touched by a sick person (i.e., the law of contagion; Bonin et al., 2019; Fernandes et al., 2017, 2019; Rozin et al., 1986; Thiebaut et al., 2022). The parallels and overlap between pathogenic disgust and BIS are evident.

1.2. The Role of Pathogenic Disgust in the Behavioral Immune System

Usually, disgust is described as a negatively valenced, high-arousing affective state composed of interconnected physiological, cognitive, and behavioral processes (Rozin et al., 1994). Disgust is a basic

emotion associated with a specific facial expression, a strong urge to withdraw from the triggering stimuli, and occasionally accompanied by a distinctive physiological manifestation of nausea, as well as a distinct feeling state of revulsion (Rozin et al., 2016).

Most researchers suggest that the role of disgust is originally related to a food rejection system and the innate aversion to bitterness (Curtis et al., 2004; Kelly, 2011). However, due to selective pressures, a pre-adaptation of that role may have occurred (i.e., co-opting an existing system for a new function). As a result, disgust evolved and expanded to a broad spectrum of elicitors (Kelly, 2011; Tybur et al., 2013). Disgust may have evolved when humans began to face new survival pressures as diets became more omnivorous or when they first started living in dense groupings (Rozin et al., 2016). Indeed, over 40,000 participants across several cultures reported that stimuli containing disease cues were more repulsive than equivalent stimuli without disease cues (Curtis et al., 2004). Furthermore, almost half of the variation in disgust sensitivity is closely linked to underlying genetic determinants (Sherlock et al., 2016), implying that disgust sensitivity is inherited to some extent. It is thought that infants cannot experience disgust. However, there is evidence that infants as young as 12 months old can acquire disgust-relevant information and subsequently avoid disgust-related stimuli (e.g., Moses et al. 2001). These findings support the notion that disgust is a powerful adaptation to selection pressures imposed by infection risk, encouraging people to avoid possible contaminants (Curtis et al., 2011; Tybur et al., 2013). It is so strong that people express pathogenic disgust even when they know that the disease threat is false and the target stimuli are innocuous (Rozin et al., 2016).

Such effects of false contamination threats might be manifestations of the sympathetic magical law of contagion (Frazer, 1922; Mauss, 2001; Tylor, 1871), which states that once in contact, always in contact (Rozin & Nemeroff, 1990). Objects that have been in touch with disgust-eliciting sources are perceived as more disgusting than before the contact. Indeed, empirical research has demonstrated that people are unwilling to touch objects that have come into contact with disgust-eliciting sources (e.g., Gérard & Helme-Guizon, 2018; Rozin et al., 1986). People seem to anticipate that some contaminating attributes of the disgust-eliciting sources will be transmitted to other objects through contact (Rozin & Fallon, 1987). This belief that physical touch between two items often transfers qualities between them is known as "magical contagion". The more a person believes that the source of contamination is harmful, the more this contamination effect is going to elicit disgust. Even if it is shown that the contamination source was not dangerous after all, individuals will remain disgusted for as long as they recall this contagion (Rozin & Nemeroff, 1990). This effect matches the evolutionary perspective since organisms that successfully avoid contamination risks are more likely to survive and reproduce.

Disgust is a key component of the BIS (Oaten et al., 2009). However, some researchers (e.g., Lieberman & Patrick, 2014) argue that immediate arousal elicited by disgusting stimuli is not always

required for the BIS to function (e.g., proactive prophylactic behaviors; Schaller, 2014). Nevertheless, disgust powerfully shapes attentional mechanisms. Several studies have shown that disgusting stimuli draw more attention than fearful ones (Chapman, 2018; Chapman et al., 2013; Perone et al., 2021). Initially, disgust diverts bottom-up controlled attention from the stimuli, which is congruent with the avoidance reaction. Then, disgust holds top-down controlled attention to the cue for a longer duration, maybe because people need to do a more careful risk evaluation of the disgusting items (Liu et al., 2015; van Hooff et al., 2013; Zhang et al., 2017; Zimmer et al., 2015). In contrast, fearful stimuli attract attention for less time and are more linked with bottom-up rather than top-down attention. Disgust also significantly impacts memory. For instance, disgust-eliciting stimuli, such as vomit, are recalled better than fear-eliciting stimuli, such as predators (Chapman, 2018; Chapman et al., 2013; Schienle et al., 2021). In this respect, Croucher et al. (2011) demonstrated that disgust-eliciting images were better remembered than fearful ones, despite the disgust-eliciting images being reported as less arousing and the pleasantness of the images being equivalent across conditions.

Enhanced attention to and recall of contamination sources are crucial for preventing infection as people navigate their milieu. To effectively elicit disgust responses, which will activate the BIS and encourage avoidance behavior, one must be sensitive to cues that connote contamination threats, even when those cues are nuanced. Enhanced recall of disgust-eliciting stimuli may also help people avoid future contact with individuals or situations that have previously posed a danger of infection. Thus, memory is a component of the BIS.

1.3. Adaptive Memory and Contamination

The whole range of mechanisms associated with the BIS is not exclusively specific to this system and did not only evolve when there were selective pressures related to infectious risks (Murray & Schaller, 2016; Woody & Szechtman, 2011). For example, Schaller (2011) argues that the BIS relies on the eyes to detect sensory cues with contamination connotations. However, this function does not imply that the eyes evolved explicitly for the BIS and are a unique resource of this system. By the same logic, memory functions are adaptive response patterns that belong to several systems, including the BIS (Murray & Schaller, 2016). This argument about the adaptiveness of memory systems is reasonable because humans need to be able to process and remember better fitness-relevant information, such as the location of food or source of contamination, to make decisions that will help them reach their fitness goals in the present and the future (Klein et al., 2002).

According to the adaptive memory theoretical framework, memory is a product of natural selection, the process which aims to increase the probability of survival or reproduction. In other words, the pressures of evolution during the Pleistocene (Klein et al., 2002) shaped the systems that control memory to retain information relevant to our fitness instead of non-relevant information (Nairne, 2010). For

example, across cultures, the locations of high-calorie foods are better recalled than those of low-calorie foods (de Vries et al., 2021, 2022). Therefore, the adaptive memory framework hypothesized that memory systems (e.g., procedural, semantic, working memory, etc.) are functionally oriented to solve particular adaptive problems, like remembering sources of contamination or the places where food can be found (i.e., domain specificity; Tooby & Cosmides, 1992), and are functionally tuned to fitness-relevant content (Nairne, 2014).

This tuning assumption is controversial. Some researchers accept that higher memory retention depends on diverse factors, namely how encoding occurs (e.g., Kroneisen & Erdfelder, 2011; see Section 1.6). However, they maintain that higher retention is independent of whether the information is survival relevant. This standpoint implies a rejection of memory's functional roots and stipulates a domain-general mechanism to explain enhanced retention. Nevertheless, we concur that mnemonic processes have functional roots (Nairne, 2016; Nairne & Coverdale, 2022). For instance, significantly better recall performance was observed for a scenario inducing survival needs (e.g., gathering food to survive) than for the exact same scenario without inducing survival (e.g., gathering food for a contest, Nairne et al., 2009). Hence, memory systems most likely have developed to promote survival and reproduction; and, as a result, they are probably tuned to the retention of fitness-related aspects. Indeed, people can retain memories about a wide variety of non-fitness-related elements, and there are specific cognitive procedures that can increase retention regardless of the content (e.g., elaboration). Nevertheless, this does not falsify the argument that memory systems were designed by natural selection to help people remember information that is important to their fitness.

Content matters because humans have limited cognitive resources (e.g., Marois & Ivanoff, 2005), and some information is inherently more valuable to learning and creating associations than others (Nairne, 2022). Therefore, it is acceptable to claim that a top-down control that differentiates between non-relevant and relevant events whose associations should be maintained is required. In this sense, memory tuning can be viewed as a top-down control over the learning process for fitness-relevant dimensions (Nairne et al., 2017). Some researchers also refer to it as a memory-based "crib sheet" because it is embedded in the biological architecture (Ermer et al., 2007) and enables people to focus on and remember the stimuli that are essential when learning about how to improve the chances of survival and reproduction (Nairne, 2016).

For example, Öhman and Mineka (2003) demonstrated that it is easier to link an aversive event with a snake than with a flower. Similarly, it is considerably simpler to apply Pavlovian conditioning to food, shock, or disgust as unconditioned stimuli (i.e., stimuli that elicit a response without previous learning or conditioning) than to neutral stimuli. For example, bells are easier to condition to food or excrement since people do not need to learn to drool in response to food or withdraw from disgust-eliciting stimuli for the conditioning. Furthermore, since fear and disgust often co-occur in phobias and

anxiety pathologies, the same learning processes that lead to fear may also lead to disgust acquisition (Olatunji & Sawchuk, 2005; Olatunji & Tomarken, in press). Likewise, conditioned fear and disgust rely on the same brain network, which incorporates the nucleus accumbens and the cingulate, orbitofrontal, and occipital cortices (Klucken et al., 2012). Notably, disgust is even more resistant to extinction than fear (Olatunji et al., 2007).

However, saying that a tuning exists for survival-related information is inconsistent with the notion that human cognitive resources are limited (e.g., Marois & Ivanoff, 2005). What is plausible to assume is that there is a tuning, not to specific survival information per se, but to survival processing itself. For example, food is no longer survival relevant after a meal. Food is not survival relevant in absolute; such attribution results from processing the stimulus and its context (Nairne & Coverdale, 2022). This is not to say that memory tuning is an evolutionary trait per se because the enhanced retention might be a co-opting of general mnemonic processes. What can be assumed is that the mechanisms activated by the survival processing are products of evolution. The survival processing may act as a "front-end" adaptation that coordinates the response to threats, just like the flight-or-fight response (Nairne, 2016).

To deeply understand the functional nature and evolutionary stance of these memory systems, the adaptive memory framework adopted the forward engineering technique (Nairne, 2015). This approach draws empirical predictions a priori based on considerations of historical selection pressures. Their general prediction is that memory systems evolved to enhance the chances of survival and reproduction. Therefore, memory systems are sensitive to fitness content (i.e., enhanced retention when the content is survival related). Indeed, it was also found that animated items are better remembered than non-animated ones (Nairne et al., 2013). The same occurs for the faces of potential mating partners (Pandeirada et al., 2017). In general, items with properties inherently related to fitness or items encoded in a fitness-relevant context (e.g., the presence of predators) exhibit a retention benefit.

This variety of findings suggests that memory systems are activated by processing general fitness content rather than a particular type of fitness. Indeed, according to Nairne (2014), mnemonic advantages are domain-specific in that these advantages occur whenever fitness-related content processing is activated, regardless of whether the content is explicitly associated with contamination, predators, mating, or animacy. The preventative systems were activated based on the content's fitness-related relevance.

1.4. Contamination Effect

Fernandes et al. (2017; Experiment 1a and 1b) demonstrated that, in a fictional context of deadly infection risk, people tend to remember objects touched by potentially infected people more than those touched by potentially healthy people. In this paradigm, participants are shown pictures of objects along with short descriptions of the people who touched those same objects. For example, a

picture of a cup is displayed with the phrase "person with brown hair" or "person with a high fever". After every third stimulus, participants see the previous three objects again without descriptors. For each object, they are asked to evaluate if the person who touched it was potentially sick or healthy. This immediate memory test is to ensure that participants are paying attention. After a distraction task, the participants are surprised with a free recall task about the objects they have seen.

The immediate memory test results showed no differences between conditions, and most participants consistently scored correctly. However, in the surprise free recall, a significant difference was observed. People recalled objects displayed with descriptors of potentially sick people more than those presented with descriptors of potentially healthy people. Furthermore, these results have been replicated using photographs instead of drawings (Fernandes et al., 2021). Therefore, the research of Fernandes et al. (2017) is an example of a successful application of the forward engineering technique. Before, Nairne (2014) made the empirical prediction for the contamination effect. Given nature's fitness criterion, people should be especially effective at recalling contamination-related information because items connoting potential infection risk are more fitness-relevant than neutral items. Since then, Fernandes et al. (2017) have demonstrated evidence supporting this prediction.

The contamination effect has also been observed without descriptors. Other research used photographs of dirty hands holding objects versus clean hands (Fernandes et al., 2021); videos in a household setting depicting an infected person sneezing and then touching objects versus a person with cancer and a healthy person (Gretz & Huff, 2019); and photographs of objects in parallel with faces with signs of contagious diseases versus healthy persons (Bonin et al., 2019; Fernandes et al., 2017, 2021; Thiebaut et al., 2022). Notably, Fernandes et al. (2017) demonstrated that the same infected faces that previously elicited a memory tuning in a contamination context did not elicit that same effect when the context was set with no contamination potential — participants were told the faces with signs of potential infection were actors wearing makeup. Overall, these studies provide a large body of evidence highlighting how memory systems — in this case, episodic memory — are functionally tuned to fitness-relevant content.

Still, the demonstration of memory-based contamination does not reveal how this trait actually works. The ultimate mechanism of the contamination effect (i.e., "why" the trait evolved) is explained by humans' need to avoid pathogenic threats and increase the chances of survival and reproduction, assuming that nature has a fitness criterion. Nonetheless, how is the contamination effect generated, and under what conditions is this effect likely to manifest? For that, it is necessary to discuss the proximate mechanisms, which are still unclear (e.g., Thiebaut et al., 2022). Both types of explanations are necessary to completely explain for the contamination effect's evolutionary standing; thus, these are significant questions (Nairne, 2014).

Within the scope of adaptive memory, there are other known tuning effects besides the

contamination effect. Different proximal processes producing similar tuning effects are improbable, since redundant processing is incompatible with evolutionary principles (Burns et al., 2013; Hunt & Einstein, 1981). Therefore, a possible answer for the proximate mechanisms of the contamination effect may come from another similar tuning effect. We suggest that the most analogous is the so-called *survival processing effect*.

1.5. Proximate Mechanism of the Survival Processing Effect

The survival processing effect refers to a memory advantage for information processed in a survival scenario as opposed to other processing contexts. Nairne et al. (2007) elaborated a paradigm to study this memory advantage. Participants in the manipulation condition were instructed to imagine themselves stranded in the grasslands of a foreign country, without access to food or water, and with the directive to avoid predators. Then, common nouns were presented sequentially, and after each one, participants had to rate how relevant those nouns were for the scenario they were in. Following a digit distraction task, participants performed a surprise free recall task. The performance was compared with a control scenario of moving to a new home in a foreign land; or other established deep processing tasks, such as rating the pleasantness of the nouns.

Nairne et al. (2007) used this paradigm to demonstrate that words presented in the survival scenario were recalled better than words presented in the control conditions. This effect has been replicated extensively (for a review, see Scofield et al., 2017) and was found even in similarly complex control scenarios (e.g., planning a bank heist; Kang et al., 2008) or other deep processing tasks (e.g., forming a visual image; Nairne et al., 2008). Moreover, this effect is not dependent on ancestral contexts because it occurs in survival contexts with modern settings (e.g., escaping from an attacker in a city; being lost in outer space; surviving a zombie attack; Kostic et al., 2012; Nairne & Pandeirada, 2010; Soderstrom & McCabe, 2011); or even in the absence of any defined scenario, such as when participants are simply asked to imagine that they are attempting to stay alive (Klein, 2013). Thus, the memory tuning is domain-specific to fitness-relevant content but not to a specific type of threat (Nairne & Pandeirada, 2016).

However, the proximate mechanisms of the survival processing effect are controversial (Howe & Otgaar, 2013; Kazanas & Altarriba, 2015). Several mechanisms known to facilitate memory were suggested as explanations, such as self-referential, relational, and richness processing (e.g., Burns et al., 2011; Klein, 2012; Kroneisen & Erdfelder, 2011). These are domain-general mechanisms. Some researchers assume that the memory tuning should reflect fitness-tailored and not general operating characteristics. If general mechanisms are responsible for the survival processing effect, then the data supporting the memory tuning is attributable to artifacts or processing confounds associated with the rating task or the scenarios, according to these researchers. They propose that higher retention is

independent of whether the information is relevant for survival or not (see Section 1.3; Kroneisen & Erdfelder, 2017).

There is substantial support for the *richness of encoding hypothesis* (Kroneisen & Erdfelder, 2011), which states that survival processing merely induces participants to generate a higher number of distinct and rich ideas during encoding because the relevance rating task implicitly encourages participants to consider different uses of the objects in a survival context. Richly encoded information results in memory representations that are easier to retrieve due to the availability of several more retrieval paths. For example, Röer et al. (2013) requested participants to write down any ideas that occurred to them while considering the usefulness of the nouns in the rating task. The results suggested that participants created more ideas in the survival condition than in the fitness-irrelevant control conditions, and the number of ideas produced tracked how well the rated words were later recalled.

However, Nairne and Coverdale (2022) argued that if domain-general mechanisms are involved in the survival processing effect, this does not negate any evolutionary account since memory tuning can involve a co-opting of basic memory processes. So much so that adaptations often co-opt other basic processes, such as the fight-or-flight response. Besides, their standpoint does not consider the functional roots of the memory systems (see Section 1.3) and does not answer why survival situations especially engage these forms of encoding compared to control situations. In addition, the research conducted by Nairne et al. (2009) negates the claim that the tuning is an artifact of a particular scenario or rating task, since the control was a matched scenario. Participants were asked to rate the relevance of the nouns for the same hunting activity in a context of survival versus a competition context. Significantly better recall performance was observed for the survival condition compared to the control condition, and the only aspect that differed between the two scenarios was the fitness relevance.

In contrast, some researchers follow the evolutionary accounts and suggest that emotionality might be a proximate mechanism for the survival processing effect. Notably, Fiacconi et al. (2015) applied the survival paradigm and heart rate measurements to assess the existence of bradycardia, an indicator of the behavioral freezing response pattern and threat-related attentional orienting responses. The results replicated the survival processing effect and demonstrated that the HR decelerated more in the survival scenario compared to the house-moving scenario. This finding supports the hypothesis that emotionality contributes to the survival processing effect.

Furthermore, Saraiva et al. (2021) found no survival processing advantage when the survival and control scenarios were presented in a second language. There is evidence that emotionally charged stimuli are processed with higher emotional activation when provided in the native language (e.g., Garrido & Prada, 2018), owing to their deeper roots and stronger emotional associations, than when delivered in a second language. Psychophysiological approaches have previously demonstrated this effect (e.g., Baumeister et al., 2017). In line with this literature, Saraiva et al. (2021) demonstrated that

participants better recalled words in the survival context than in the house-moving scenario, but this only occurred in the native language condition. Consequently, Saraiva et al. (2021) suggested that the absence of the survival processing effect in a second language indicates that emotionality has a role in the processing of a survival context. These results were replicated by Kazanas et al. (2021).

Therefore, the findings of Fiacconi et al. (2015) and Saraiva et al. (2021) support the hypothesis that emotionality may be a mechanism that could explain how changes in the autonomic nervous system result in enhanced cognitive processing, presumably to mobilize more cognitive resources and avoid danger. Although these findings are related to the survival processing effect, we emphasize the parallel with the contamination effect considering that, in both cases, memory tuning occurs in threatening contexts.

1.6. Emotionality as Proximate Mechanism for the Contamination Effect

Fear and disgust responses are frequently regarded as separate, with different impacts on attention and memory. For biological purposes, fear and disgust differ because fear is likely a survival reaction to immediate danger, and disgust is a rejection of pollution (e.g., Tybur et al., 2013). Nevertheless, fear and disgust have many similarities and are strongly linked (e.g., Woody & Teachman, 2000). Conditioned fear and disgust reactions rely on the same neuronal network (Klucken et al., 2012). Further, fear and disgust often co-occur and are seen as defensive emotions that produce high arousal to protect oneself from danger or injury (Davey, 2011). For example, both emotions influence the startle response, which can be viewed as a marker of the defensive system (Bradley et al., 2005; Yartz & Hawk, 2002). Therefore, it seems plausible that the same proximate mechanism that operates in fear-eliciting contexts also occurs in situations that induce pathogenic disgust.

1.6.1. Emotional Processing of Disgust

The processing of emotional stimuli is divided into several steps. First, people are oriented to sensory input and process contextual cues for information gathering. The autonomic system then causes bodily changes, such as in HR. Finally, relevant information about the stimulus is retrieved from memory, and people implicitly prepare to act appropriately (Bradley et al., 2001).

In James-Lange theory (1884), the conscious emotional experience is the interpretation of bodily responses, and different emotional stimuli elicit different bodily responses. Although some ideas presented on this theory may no longer be supported (Laird & Lacasse, 2014), it is becoming clear that visceral feedback has profound modulatory effects on emotional as well as cognitive processes. For example, the perception of bodily states has been shown to correlate with the intensity of responses to emotional stimuli (Critchley, 2009). Further, Levenson (2003) argues for autonomic specificity and states that emotions can be distinguished in terms of autonomic nervous system activity patterns. This view is

derived from an evolutionary perspective on emotionality, which claims that emotions were selected for their capacity to assist the organism in coping with challenges to survive. Notably, this perspective assumes the existence of an emotion system in which a central mechanism continuously scans the incoming stream of information from the external and internal realities in search of specific configurations that represent challenges with significant consequences for survival and well-being.

For example, when a disgust-eliciting stimulus is detected, the perceptual orienting reaction is more extended than fear because it takes longer to determine whether the disgust-eliciting stimuli are dangerous. Then, among several changes, a HR deceleration occurs (Levenson et al., 1990). These changes are designed to prepare the body for the avoidance reaction, such as moving away from or removing the source of contamination. The reaction also depends on the information recalled about the stimulus and previous experiences. The conscious experience of disgust elicited by the stimulus stems from the perception and interpretation of these bodily changes (Woody & Teachman, 2000).

1.6.2. Cardiovascular Indicator of Disgust Processing

Electrocardiography (ECG) uses non-invasive electrodes to measure the echoes of the electrical activity associated with heartbeats. The pacemaker cells in the sinoatrial node generate spontaneous action potentials that initiate the electrical process of depolarization and repolarization of the heart (Berntson et al., 2007). The sympathetic and parasympathetic divisions of the autonomic nervous system modulate the strength of the cardiac contractions. The sympathetic division boosts the sinoatrial node's automaticity and excitability (i.e., properties responsible for controlling heart rhythm), thus increasing heart rate. Notably, the sympathetic system dominates the control of cardiac contractility. The parasympathetic division reduces the automaticity and excitability of the sinoatrial node, lowering the heart rate, a process termed bradycardia (MacDonald et al., 2020).

One of the measures of cardiovascular activity is HR, which refers to the number of times the heart muscle contracts or beats per minute (BPM). The QRS complex (i.e., a combination of the Q wave, R wave, and S wave) is one of the ECG components. The R peak, which corresponds to the start of ventricular depolarization, is the most pronounced peak of the wave complex, and thus the intervals between R peaks are used to calculate the HR.

The cardiovascular response differs between the major categories of emotions (Sinha et al., 1992). Moreover, the neural process associated with HR deceleration is both connected to attentional and emotional processing. Several areas of the forebrain that project nerves to the medullary and spinal nuclei that control heart function modulate the activity of the autonomic cardiac nerves. Some of these are the insular cortex, the anterior cingulate cortex, the central nucleus of the amygdala, and various nuclei in the hypothalamus. The anterior cingulate and insular cortices are both part of the salience network. This part of the brain responds to behaviorally important stimuli and combines information

from outside the body with information from inside the body (Uddin, 2015). In particular, the amygdala and insula play key parts in disgust processing and its memory consolidation (McGaugh, 2004; Woolley et al., 2015). Therefore, HR is a versatile tool for analyzing the physiological response to pathogenic disgust.

1.6.3. Claims Against Emotionality in Memory Tuning

Although emotionality has evolutionary roots, is linked to memory, and plays a vital role in threat response, little research has examined it as a possible proximate mechanism for memory tuning. In light of this discrepancy, it appears worthwhile to understand why emotionality has often been overlooked in studies on memory tuning.

Several researchers employed the survival processing paradigm and applied control scenarios with equal emotionality and arousal characteristics, but the mnemonic advantage remained significant in the survival scenario. For example, Bell et al. (2013) contrasted the survival scenario against a suicide scenario. Participants remembered more words from the survival scenario than from the suicide scenario. Likewise, Yang et al. (2014) compared the survival scenario with a winning lottery scenario. The results were the same. On this premise, both authors and many other researchers (e.g., Kazanas & Altarriba, 2015; Nairne et al., 2017) concluded that negative or positive emotions are not proximate mechanisms and have no influence on the survival processing effect. However, not all emotions are associated with defensive motivational systems (Bradley et al., 2001). Most positive emotions do not display pronounced autonomic nervous system responses compared to negative emotions (Kop et al., 2011). Disgust and intense sadness are two negative emotions that induce cardiac deceleration by increasing parasympathetic activity and decreasing sympathetic impact (Kreibig et al., 2010). However, the origin of heart deceleration during these two emotions is likely distinct. Disgust is a powerful emotional response designed to protect oneself from the threat of disease (e.g., Curtis et al., 2004). Sadness, on the other hand, may be characterized by complex emotions including fatigue, helplessness, and sorrow (Kreibig et al., 2010). In addition, disgust improves episodic memory (e.g., Chapman et al., 2013) and modifies early attentional processes (e.g., van Hoof et al., 2013), but happiness and sadness have no impact on executive functioning or attention (Finucane et al., 2010).

Even when the activities in the survival and control situations are identical (e.g., hunting to gather food for survival vs. hunting for a game; Nairne et al., 2009), the emotion evoked by the survival scenario will vary from the control scenario due to the presence of a fitness-relevance feature: the threat of food deprivation. Likewise, Kroneisen et al. (2022) designed two similar scenarios alluding to the threats of COVID-19. One scenario focuses on emotional priming, and the other on strategies. However, the strategy scenario alluded to a direct threat to the individual (i.e., "imagine that you suffer from chronic lung disease. The novel coronavirus has broken out in your hometown, and you are a

high-risk patient who would most likely die from infection with the virus."). In the emotional scenario, the direct threat was absent (i.e., "You too are severely affected, financially as well as mentally, due to deaths in your family."). Therefore, the scenarios cannot be compared, and the role of emotionality cannot be evaluated.

The emotionality elicited by the control scenarios in these studies does not belong to a dimension of defensive emotions such as fear and disgust, which acutely use attentional resources and share facilitated learning pathways to avoid life-threatening situations. According to the *defense cascade model* (Bradley et al., 2001), fear and disgust are arranged within a motivational defense system, which should facilitate the processing of threatening cues and provide a reaction. In the initial phases, emotions are processed mainly perceptually through orienting, with little overall activity. When the threat becomes more imminent, the system changes to overt defensive behavior and increases activation. This model resonates with Levenson's (2003) evolutionary perspective on emotionality.

Within this reasoning, reducing the emotional intensity of the life-threatening cues in the survival context by limiting the activity to finding potable water in the grasslands (Kroneisen & Erdfelder, 2011), without mentioning predators or food deprivation, may reduce the threshold and not be sufficient to activate the defense system. Therefore, this might be the cause for the elimination of the survival processing effect, and not necessarily less rich encoding.

Other researchers controlled for arousal, disgust, and fear ratings in statistical analysis, and the mnemonic advantage was still substantial (e.g., Gretz & Huff, 2019). On this premise, some have argued that emotionality is not a proximate mechanism and does not influence the memory advantage. However, the participants' responses to self-reported emotional ratings may constitute rationalizations for their interpretation of the stimuli, thus inevitably measuring emotionality indirectly with a degree of error. Self-report measurements are useful for emotion verification but limited if used without the measurement of psychophysiological responses, especially when examining emotionality associated with automatic defensive responses. Indeed, people are only able to self-report on emotionality that exceeds the threshold of conscious awareness. In contrast, psychophysiological measurements can identify autonomic nervous system activity associated with specific emotional responses and provide information unknown to the individual.

Furthermore, Fernandes et al. (2021) noted that females are more prone to disgust than males, referring to a meta-analysis conducted by Sparks et al. (2018) that did not include studies with psychophysiological indicators. Since there were no gender differences in the memory advantage observed, Fernandes et al. (2021) claimed that emotionality is not a proximate mechanism involved in the contamination effect. Several researchers (e.g., Rohrmann et al., 2008; Schienle et al., 2005) did not find gender differences in autonomic responses. Therefore, the conclusion of Fernandes et al.

(2021) may be challenged, since subjective rationalizations of emotional responses are susceptible to degrees of error when attempting to access automatic emotional responses.

Notably, Forester et al. (2020) contended that an increase in emotional arousal is unlikely to be the primary factor underlying the survival processing effect, because they found no significant group differences in the overall amplitude of the P300 (500-700 ms) between survival and control. However, Zhang et al. (2020) observed increased positivity associated with survival processing in the P300 time period. Therefore, these neurobiological findings are still unclear.

In conclusion, the findings mentioned above do not rule out that emotionality may play a role in memory advantage for the survival processing effect and, therefore, the contamination effect.

1.7. Aim of the Present Study

Given the above considerations, emotionality may be a proximate mechanism for the contamination effect. The BIS is deeply intertwined with pathogenic disgust (e.g., Keller et al., 2022), which has a vital role in processing threats (Bradley et al., 2001; Lang & Bradley, 2013). Memory is a key component of emotional processing (Laird & Lacasse, 2014), and the memory tuning is a front-end adaptation that coordinates several processes to respond to threats (Nairne, 2016). The research on the contamination effect found that contamination-threatening stimuli are better remembered than neutral stimuli (Fernandes et al., 2017, 2021). From an evolutionary perspective, it is plausible to argue that this effect occurs because avoiding pathogenic threats increases the chances of survival and reproduction. But what proximate mechanisms explain how this memory benefit actually works?

It is evident from the findings provided by Saraiva et al. (2021) and Fiacconi et al. (2015) that emotionality is a strong candidate to be a proximate mechanism for the survival processing effect. However, they only investigated contexts that elicit fear and not those that elicit pathogenic disgust. Although we argued that the survival processing and the contamination effects share several characteristics, there is still a need to determine whether emotionality underlies the contamination effect or not.

Therefore, in the present research, we expected to replicate the contamination effect (Fernandes et al., 2017, 2021; Experiment 1a). We hypothesized that participants would exhibit higher recall for objects displayed with descriptors of potentially sick people than with descriptors of potentially healthy people. This is an approximate replication because some used stimuli were not part of the original studies of Fernandes et al. (2017, 2021). Notably, our primary goal was to investigate the role of emotionality in the contamination effect. We hypothesized that participants would exhibit significantly more HR deceleration while perceiving objects displayed with descriptors of potentially sick people than potentially healthy people.

CHAPTER 2

Method

2.1. Participants

We established a target sample size of 80 participants by using the study of Fiacconi et al. (2015) as a reference. These authors also investigated the role of emotionality in adaptive memory using psychophysiological measures. Eighty-one adults participated in this experiment. Data from one participant were excluded due to insufficient correct answers in the immediate memory task (< 60% accuracy; Fernandes et al., 2017, 2021). The final sample consisted of 80 participants (55 females; M_{age} = 22.60, SD_{age} = 8.06, ranging from 18 to 61 years). The inclusion criteria for participation were age (\geq 18 years old) and native language (European Portuguese). Participants were recruited by convenience sampling (i.e., flyer distribution on campus and online advertising on social media platforms) and were rewarded with a \leq 5 commercial voucher; or through the university's research participation system and were compensated with course credits. Before beginning the experimental task, all participants gave their informed consent. All research activities were conducted following the Declaration of Helsinki. Due to technical difficulties, one participant was removed from the HR analysis.

2.2. Materials

Each stimulus consisted simultaneously of a photograph depicting an object being held by hands and, below, a description of the person holding the object (see Table 1 from Appendix A).

2.2.1. Objects

We selected twenty-four photographs of handled objects, along with three additional photographs to be used in the practice trials, from the high-quality standardized Objects-on-Hands Picture Database (Fernandes et al., 2019). The photographs were selected from the "clean hands" and "frontal viewpoint" categories. For the experimental trials, the total set comprised 24 objects, and each set of four belonged to the following subcategories: fruit, vegetable, office supplies, kitchen utensils, toys, and accessories. Moreover, aspects of the objects that could affect recall were controlled, such as name agreement (e.g., Hovhannisyan et al., 2021) and familiarity (e.g., Nickerson & Adams, 1979). According to the Portuguese norming data (Fernandes et al., 2019), the selected experimental objects had a similar high name agreement (i.e., low number of different names participants use to refer to a given object; mean H = 0.09, SD = 0.16 [range: 0.00-0.66]), and similar high familiarity (i.e., participants frequently think or encounter the concept depicted by the object; M = 4.82, SD = 0.18 [range: 4.37-5.00]; on a scale ranging from 1 to 5).

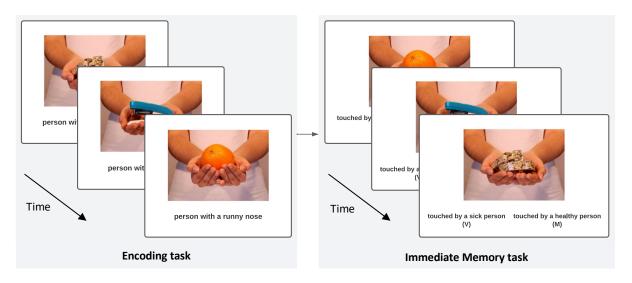


Figure 2.1. Procedure of the encoding and immediate memory tasks.

2.2.2. Descriptors

We selected 12 sentences for the experimental manipulation, 10 from Fernandes et al. (2017; Experiment 1a and 1b) and two from Garrido et al. (in prep) for counterbalancing purposes. Six of these sentences described signs and/or symptoms of disease to be used in the manipulation condition as potentially sick persons (e.g., pessoa com tosse constante [person with a constant cough]). The remaining six described neutral physical characteristics to be used in the control condition as potentially healthy persons (pessoa com olhos verdes [person with green eyes]). The length of the descriptors was not significantly different across conditions, t(10) = -1.01, p = .337.

2.3. Design

We used a within-subject design in which the same participants were exposed to both types of descriptors. The stimuli were divided into eight experimental blocks, each consisting of three trials. The counterbalancing (see Table 1 from Appendix A) followed the instructions of previous research (Fernandes et al., 2017). The objects were randomly separated into two lists to ensure that the same object would appear with sick and healthy descriptors. The objects of these two lists had the same number of categories (i.e., six; see section 2.2.1.); similar name length, U = 62.00, p = .571; name agreement, U = 58.00, p = .370; and familiarity, U = 64.50, p = .686. To counterbalance the order of the descriptors, a total of four lists were created. The first two trials of each block always had different conditions to prevent participants' attempts to guess the last stimuli of the block (Fernandes et al., 2017).

2.4. Measures

2.4.1. Behavioral Measures

Immediate Memory Test. To ensure that participants were paying attention to the stimuli, they were

asked to respond with a key press whether the previous objects were touched by a sick person (V) or a healthy person (M). After every third trial, this test was administered. Whether the participant responded or not, the screen shifted to the next object after 5 seconds (see Appendix B).

Distractor Task. Participants were asked whether each random single-digit number from 1 to 9 was even or odd. Each number was shown for two seconds or until a response was obtained. There were 47 single-digit numbers presented in total. This cognitively challenging task was included between the encoding and free recall phases to limit participants' rehearsal of the information and guarantee that the objects and descriptors were no longer stored in working memory. Data from this task were not analyzed.

Free Recall. Participants were asked to type the names of as many objects as they could recall, regardless of their decision about who touched them. This task lasted 5 minutes, without the possibility of ending earlier. After that time, the screen would automatically advance. Only responses consisting of objects from the experimental task were deemed valid (see Appendix C).

Descriptors' Evaluation. Participants were asked to rate the descriptors in five dimensions on a 9-point scale, including valence ("How negative or positive is this description for you?", 1 = Very negative to 9 = Very positive); arousal ("How calm or activated do you feel when reading this description?", 1 = Very calm to 9 = Very activated); fear ("To what extent do you feel fear when reading this description?", 1 = Not scared at all to 9 = Very scared); disgust ("To what extent do you feel disgusted when reading this description?", 1 = Not disgusted at all to 9 = Very disgusted); and potential of contamination ("If you interacted with a person with this characteristic or symptom, how likely would you be to get sick?", 1 = Not likely to 9 = Very likely).

2.4.2. Heart Rate Measure

The disposable EL500 pre-gelled electrodes were linked via LEAD110 cables to a MEC110C adaptor and affixed to an ECG100C amplifier. The electrode measuring the positive potential was placed on the inner left ankle, the electrode measuring the neutral potential (i.e., grounding) was placed on the inner right ankle, and the electrode measuring the negative potential was placed near the right clavicle, as per the Lead II configuration. Before attaching the sensors, these locations were cleaned with alcohol to guarantee low impedance.

The signal was sampled at 1000 Hz and filtered with a high-pass of 0.5 Hz and a low-pass of 35 Hz. We sampled at a high rate to acquire valid data after smoothing and removing artifacts (Boucsein et al., 2012), and we did not downsample during pre-processing, since higher sampling rates are recommended to maximize the precision of heartbeat intervals (Pham et al., 2021).

The ECG signal was FIR band-pass filtered (1-35Hz; Fiacconi et al., 2015) with Hamming window (see Appendix D). Compared to other window functions, the Hamming window function has a relatively

narrow transition width and a higher stopband attenuation (An & Stylios, 2020). R peaks were detected automatically with the NeuroKit2's method (Makowski et al., 2021), which uses the steepness of the absolute gradient of the ECG signal to detect R-peaks as local maxima in the QRS complexes. Thus, segments with a high noise level are not considered by the algorithm. The signal was visually inspected, and only one peak was corrected. Signal inversions were not necessary. We employed density-based spatial clustering of applications with noise (DBSCAN) to identify artifacts based on the morphology of the waveforms, since the human evaluation of the signal is error-prone.

Next, physiological and technical artifacts such as long, short, missed, extra, or ectopic beats (i.e., irregular beats due to premature atrial or ventricular contraction) were detected using the automatic algorithm designed by Lipponen and Tarvainen (2019). This method uses the differences between successive RR intervals and their deviation from the median to separate outliers from the normal sinus rhythm. Ectopic, long, and short beats were corrected by replacing new RR values through interpolation. Only 0.2 % of the peaks were corrected. Then, the HR was computed through the heart periods and submitted to a monotonic cubic interpolation (see Appendix D) because this interpolator does not overshoot if the data is not smooth.

Considering the findings suggesting that HR is sensitive to anticipatory processing (Graham & Clifton, 1966; Merscher et al., 2022), we determined the baseline by identifying the area with an overlapping plateau of the grand average waveforms of both conditions. Gatchel and Lang (1973) argue that when the difference between the initial HR acceleration peak and the subsequent deceleration peak is used as an index, larger magnitude responses can be associated with the exposed stimuli. However, the initial acceleration peaks in our data did not overlap in the time frame; thus, an average of the baseline at 4,000-2,000 milliseconds before stimulus onset was more appropriate to maintain equivalence between conditions. The change in HR scores was calculated by subtracting the baseline value. Then the HR was reduced for each trial by averaging into 500 milliseconds bins (Fiacconi et al., 2015). We only considered the HR during the encoding trials.

2.5. Procedure

When recruited, participants were told that the purpose of the study was to examine how individuals evaluate descriptors. They were further informed that their cardiovascular activity would be recorded and where the sensors would be attached. They came to the lab for an individual session that lasted approximately 30 min.

After participants signed the informed consent, we applied the sensors, and the cardiovascular activity was recorded simultaneously and continuously throughout the experimental session. These measurements were recorded using BIOPAC MP150 (Biopac Systems, Goleta, CA) and the AcqKnowledge software (Version 4.1; Biopac Systems). The task was conducted with the software E-

prime 2.0 Professional (Psychology Software Tools). We assigned participants sequentially to one of the four lists (see Table 1 from Appendix A). After reporting their gender and age, they were requested to sit still and relax until a 3 min rest period was completed for the sensor's stabilization.

The experiment was conducted using the same instructions as Fernandes et al. (2017). Participants were asked to recall objects that had been touched by several people, as they would need to judge for each object whether the person who touched it was healthy or sick with a fatal contagious disease. The instructions did not specify the final free recall task (see Appendix B).

After the instructions, participants completed a practice phase. Then, the experimental phase began. Participants were exposed to three trials and then were required to complete an immediate memory test asking if the objects were touched by a sick or healthy person. This procedure was repeated until the eight blocks of stimuli were completed. In total, participants saw 24 different objects (paired with the descriptors). Each trial was separated by 12 seconds of white screen to allow the registration of psychophysiological changes after the stimuli onset (e.g., Bradley et al., 1996).

Next, participants were asked to perform a short numerical judgment task. Then, they were asked to complete a free recall task concerning the objects they had seen, in any order and regardless of the judgment previously made about them. For 5 min, participants typed their responses into the computer.

To avoid interaction with the manipulation effect (Hauser et al., 2018), the last task was the manipulation check of the descriptors. In random order, participants assessed them on a 9-point scale for arousal, valence, fear, disgust, and contamination potential.

2.6. Data Analysis

Statistical analyses were performed using R software version 4.2.1 (R Project for Statistical Computing) within RStudio statistical software version 2022.07.2+576. The statistical level of significance was defined at p < .05. The interpretation of effect sizes (d) was conducted using the criteria defined by Lovakov and Agadullina (2021), which is based on the actual distribution of effect magnitudes in the social psychology literature. For R^2 and odds ratio values, we used Cohen's criteria (1988).

The evaluations of the descriptors were examined for manipulation check and emotional verification. The immediate memory test performance and reaction times were examined to ensure that participants were paying attention to the stimuli.

We used mixed effects logistic regressions to analyze the free recall and immediate memory performances (Chatterjee & Hadi, 2012; see Appendix C). The 95% confidence intervals (CI) and p-values were computed using a Wald z-distribution approximation. Since the original studies (Fernandes et al., 2017, 2021) used different analyses from the present study, we conducted conversions of odds

ratio (OR) to Cohen's d (Sánchez-Meca et al., 2003). We also applied a meta-regression to compare findings equitably (see Appendix E for the replication using the original method).

For the psychophysiological data, we opted to correct outliers instead of removing parcels of the signal or removing the entire trial. This decision was based on Lipponen and Tarvainen's (2019) argument that cardiac measures, such as HR, are highly impacted when calculated from data with outliers. Hence, not handling outliers would introduce distortion to the results. Secondly, removing segments in the HR within the trials would introduce distortion because the data would correspond to the removing threshold and not to an actual or interpolated peak of a corrected RR interval. Lastly, removing the entire trial containing at least one outlier was not viable considering that, in our case, it would damage the balance between conditions and drastically reduce our data.

A grand average cardiac waveform was calculated, and time windows were established by visual inspection. We adopted the methodology of Ruiz-Pardial et al. (2018) and Fiacconi et al. (2015) given that, as previously stated, we anticipated differences between disgust-eliciting and neutral stimuli in the bradycardia response. Therefore, we focused our analysis on the observed maximum HR deceleration of the grand average, and compared between condition and their baseline. Following the methodology of Fiacconi et al. (2015), we first analyzed the data in aggregate and then split the participants into advantage and non-advantage groups based on whether they displayed the contamination effect. We used paired sample t-tests, Wilcoxon W, and one-way ANOVA.

CHAPTER 3

Results

3.1. Behavioral Data

3.1.1. Descriptors' Evaluation

Sick descriptors were evaluated as significantly more negative, arousing, fear-inducing, disgust-inducing, and potentially contaminating than healthy descriptors (all p's < .001; see Table 2 from Appendix A).

3.1.2. Immediate Memory Performance

The performance on the immediate memory test was almost perfect. In the healthy condition, participants provided 96% (SD = .20) of correct responses, compared to 92% (SD = .27) in the sick condition. We fitted a logistic mixed model to predict the performance with the conditions. We included participants as a random effect due to the within-subjects design. The model's total explanatory power was substantial ($R^2 = .37$). The effect of healthy condition was also positive and statistically significant (b = 0.71, 95% CI [0.29, 1.13], p < .001; see Table 3 from Appendix A). In the healthy condition, participants had a significantly better probability of answering correctly than in the sick condition (OR = 2.04, 95% CI [1.34, 3.10]).

We expected an absence of effect in the immediate memory test between conditions, as observed in the original study (Fernandes et al., 2017, 2021). Therefore, we proceeded to examine if this effect was similar between the participants who displayed the contamination effect (advantage group; n = 44) and those who did not (non-advantage group; n = 36). In the advantage group, the effect of healthy condition was statistically significant (b = 0.69, 95% CI [0.15, 1.23], p = .013, OR = 2.00, CI [1.16, 3.43]), with 95% (SD = .21) of correct answers in healthy, and 92% (SD = .27) in sick condition. In the non-advantage group, the effect of healthy condition was statistically significant (b = 0.75, 95% CI [0.09, 1.41], p = .027, OR = 2.11, 95% CI [1.09, 4.08]), with 96% (SD = .19) of correct answers in healthy, and 93% (SD = .25) in sick condition.

In both conditions, participants took an average of 2 seconds (SD = 0.86) to decide if the object was touched by a healthy or sick person ($M_{healthy} = 1.80$, $SD_{healthy} = 0.88$; $M_{sick} = 1.74$ s, $SD_{sick} = 0.85$), and the results were statistically equivalent, t(79) = 2.10, p = .019.

3.1.3. Free Recall

We fitted a logistic mixed model to predict performance with the conditions (see Figure 3.1). We included participants as a random effect due to the within-subjects design. The trials were also entered as random effects to account for object specificities and presentation order. The model's total

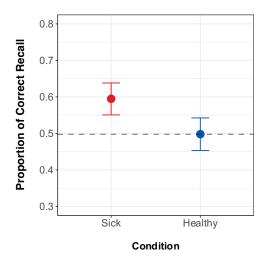


Figure 3.1. Proportion of Correct Recall as a Function of Condition. To adequately compare the proportions of the two conditions, we plotted the correlation and difference-adjusted 95% CI (Cousineau et al., 2021). The dashed line corresponds to the proportion of the control condition.

explanatory power was moderate (R^2 = .24). The effect of the sick condition was positive and statistically significant (b = 0.47, 95% CI [0.27, 0.66], p < .001, OR = 1.59, 95% CI [1.31, 1.94]). Participants had a significantly higher probability of remembering objects associated with sick descriptors (59%, SD = .49) than healthy descriptors (50%, SD = .50; see Table 4 from Appendix A). Previous studies by Fernandes et al. (2017; 2021) observed moderate and small effect sizes of d_z = 0.47 and d_z = 0.33, respectively. Our model had a small effect size of d_z = 0.26 after conversion. However, the meta-regression based on the proportions of these three studies revealed that our study had the largest OR compared to the original studies (see Table 5 from Appendix A).

3.2. Heart Rate Data

First, we analyzed the aggregated data. The deceleration peak in the sick condition (1,000-1,500 ms; M = -0.76, Mdn = -0.55, SD = 2.15) was significantly deeper than the baseline, W = 928, p < .001, r = -0.41. The deceleration peak in the healthy condition (1,500-2,000 ms; M = -0.30, Mdn = -0.62, SD = 2.32) was also significantly deeper than the baseline, W = 1186, p = .027, r = -.25 (see Figure 3.2). HR did not differ when compared directly between peaks, W = 1378, p = .162.

To assess the fast changes in HR deceleration, we compared the HR at 1,000 ms before the peaks in each condition. Results showed an effect of time on HR for the sick condition, as HR was significantly deeper at the peak (1,000-1,500 ms; M = -0.76, SD = 2.15) than at onset (0-500 ms; M = -0.41, SD = 2.03), t(78) = -2.22, p = .015, d = -0.25. However, this effect did not occur in the healthy condition's peak (1,500-2,000 ms; M = -0.30, SD = 2.32) when compared with the HR at 500-1,000 ms (M = -0.12, SD = 2.48), t(78) = -1.13, p = .132.

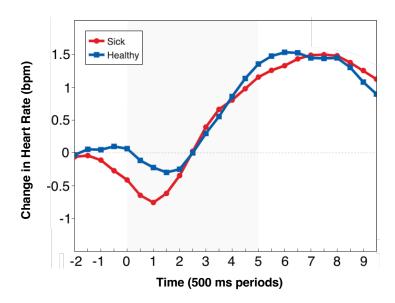


Figure 3.2. Grand Average Cardiac Waveform. Baseline-corrected. All participants aggregated. The dotted line represents the prestimulus baseline level of the response. The shadow area marks the time window of the stimulus exposure.

Next, we followed Fiacconi et al. (2015) approach by grouping the participants depending on whether they displayed the contamination effect (n = 43) or not (n = 36); and separating the trials by whether the object shown was later remembered or forgotten in the free recall. We extracted the HR average between 1,000-3,000 ms of the remembered trials in each group and condition. In the advantage group, the deceleration in the sick condition (M = -0.73, SD = 2.33) was significantly deeper compared with the healthy condition (M = 0.71, SD = 4.80), t(43) = -1.86, $M_{\text{difference}} = -1.44$, p = .035, d = -0.28. In the non-advantage group, the deceleration in the sick condition (M = -1.07, SD = 2.96) was not significantly deeper compared with the healthy condition (M = -0.37, SD = 3.09), t(35) = -0.97, $M_{\text{difference}} = -0.69$, p = .170.

As an exploratory analysis, we examined gender effects within the advantage group over the deceleration in the sick condition, and no significant differences were observed, F(1,42) = 0.74, p = .394.

CHAPTER 4

Discussion

4.1. Contamination Effect

The BIS system evolved in order to assist our ancestors in detecting and avoiding fitness-relevant risks to boost their chances of survival and reproduction. This system is defined by affective, cognitive, and behavioral processes collaborating to be the first line of defense against pathogenic threats (Schaller & Duncan, 2007; Schaller & Park, 2011). Potential sources of contamination elicit disgust, which in turn activates the BIS system. The responsiveness of this system is so geared toward self-protection that, according to some studies, the BIS operates as a "smoke alarm" that directs behavior even when the threat is seemingly subtle (e.g., Nesse, 2005). Similarly, multiple studies have shown that disgust responses are associated with increased allocation of attention (e.g., Ackerman et al., 2009; Schienle et al., 2021; van Hoof et al., 2013) and enhanced memory (e.g., Chapman et al., 2013; Schienle et al., 2021). For instance, to safely handle objects in a setting posing a risk of infection, it is vital to learn if they are contaminated. Indeed, Fernandes et al. (2017, 2021) found that, according to the law of contagion (Rozin, 1990), even visually neutral objects paired with sentences describing infection cues elicit disgust and are better remembered than those touched by a non-contaminated source. Our findings replicated this contamination effect, as participants were more likely to recall objects touched by a potentially sick person than a healthy one. According to Chapman et al. (2013), humans consolidate disgust more effectively because accurate memories of disgust-eliciting stimuli aid in preventing future contamination. Consequently, our results provide more evidence of the memory advantage in fitness-relevant contexts, such as a contamination threat (Bonin et al., 2019; Gretz & Huff, 2019; Thiebaut et al., 2022).

As seen in the original research (Fernandes et al., 2017, 2021), we anticipated a lack of effect in the immediate memory test across conditions. Although the performances were near-perfect in both conditions, we observed an enhanced performance for the healthy condition. After further examination, we concluded that this enhanced performance occurred in the participants who exhibited the contamination effect and who did not. Therefore, it cannot be claimed that attentional confounds impacted the contamination effect.

Several studies have previously shown that memory for contamination threats or disgust-eliciting stimuli is better than memory for other types of information (e.g., Ackerman et al., 2006). However, this line of research diverges from the Fernandes et al. (2017) paradigm. Due to the counterbalancing design, participants recalled the same objects in both conditions, thus addressing item-selection

problems; most importantly, the fitness-relevance of the stimuli was acquired through contagion with other threatening factors (e.g., a sick person).

The observation that the contamination effect was triggered by subtle contamination signals through the law of contagion (i.e., contamination cues that are not visually disgusting), as opposed to the powerfully disgust-eliciting stimuli frequently used in the research, may indicate the effectiveness of this effect as a protective response. Furthermore, several studies have shown that memory for disgust-eliciting stimuli is better than memory for frightening stimuli (e.g., Moeck et al., 2021; Schienle et al., 2021).

In the present research, we used sentences to transmit the contamination cues. Other studies by Fernandes et al. (2017) showed faces with disease indicators (2017; Experiment 2) or dirty hands (2021; Experiment 3). All these paradigm variations demonstrated the contamination effect. However, the induction of pathogenic disgust may not rely on the availability of explicit information about contamination threat if the BIS of the participants is already engaged. For example, Miller and Maner (2011) found that those who had been sick in the previous week allocated more attention and displayed higher avoidance to faces with potential disease markers when compared to a group that had not recently been ill. In this study, no overt disease-related cues or scenarios were provided. It would be informative if future replications of the Fernandes et al. (2017) paradigm investigate whether the BIS increases, even more, the memory advantage for contamination cues in recently ill individuals than in a non-recently ill group. This gradient response would be extremely adaptive to protect oneself from potential contamination threats. Therefore, if supported, this perspective, would not only add weight to the evolutionary account of the memory advantage, but also oppose the view that the memory advantage is nothing more than the richness of processing.

In short, it important to emphasize the notion that recently ill participants already have the BIS activated. So, the results of Miller and Maner (2011) do not imply that a lack of contamination threat is viable to activate the BIS or the contamination effect. In fact, Bonin et al. (2019) found that the memory advantage did not emerge when the intensity of the contamination threat was reduced by providing a scenario of objects possibly touched by individuals who did not wash their hands after using the restroom.

Additionally, the perspective that BIS has a gradient activation rather than an all-or-nothing activation process is consistent with the view that pathogenic disgust is a proximate mechanism for the contamination effect. This is the case because, on the one hand, emotionality is a gradient response, and, on the other hand, disgust, along with fear, are potentially critical components of a defensive motivational system that activates more when facing immediate threats (Bradley et al., 2001).

4.2. Emotionality as Proximate Mechanism for the Contamination Effect

The main goal of the current research was to examine emotionality as a proximate mechanism for the contamination effect by using psychophysiological measures such as HR. We hypothesized that the memory advantage of the contamination effect is explained, at least in part, by the activation of autonomic emotional responses to pathogenic threats that emerge when the individual evaluates the objects' contamination potential in a lethal scenario. Notably, psychophysiological assessments during encoding indicated a higher HR deceleration for potentially contaminated objects. This response is consistent with the patterns of the defense-motive circuit activated in threat contexts, which include bradycardia (e.g., Bradley et al., 2001; Lang et al., 2000).

According to Lang et al. (1990), human emotions are considered action dispositions that arrange behavior along an appetitive/defensive axis. Valence determines which one of two systems is engaged (defensive vs. appetitive), while arousal determines the level of activation. According to this model, when negatively valenced and highly arousing stimuli are encountered, the defensive motivational system is engaged in preparation for the rapid execution of a repertoire of self-protective acts. What constitutes a threat might be biologically based (e.g., an instinctual response to the sight of a snake) or socially learned. Both animal and human research indicate that when the possibility of threat triggers the defense motivational system, physiological responses to the threat-congruent stimuli are enhanced, whereas when threat-incongruent stimuli are encountered, the physiological responses are diminished. In other words, our organism was designed to detect signals consistent with a state of danger and to respond when confirmatory signs are observed.

Although there is a substantial body of research demonstrating the significance of emotion in threat processing, previous studies on the role of emotionality in the contamination effect are scant. Several behavioral studies have suggested that emotionality does not contribute to the associated memory advantage. For example, Fernandes et al. (2017) displayed objects paired with the face of the person who touched them. The authors found a contamination effect for the faces with infection cues. However, the effect did not occur when participants were informed that certain faces were actors wearing makeup for a medical television show. The researchers concluded that emotional arousal was not responsible for the effects since they assumed the level of disgust was the same in both studies. Instead, they ascribed the effect to the contamination threat itself. Alternatively, we propose that the makeup scenario lowered the pathogenic disgust by removing the contamination potential. On the other hand, the defense motivational model (Bradley et al., 2001; Lang et al., 2000) argues that threats, such as contamination ones, elicit an emotional response without the individual needing time to think.

In contrast, two findings support our standpoint about emotionality. The findings of Saraiva et al. (2021) demonstrated that the memory advantage did not emerge when participants used a language with fewer emotional associations. Those of Fiacconi et al. (2015) indicated an HR deceleration

associated with the memory advantage. However, both studies addressed the survival processing effect. We argue in the present research that the survival processing effect is similar to the contamination effect, since both influence memory and require a context with threat cues. Additionally, the memory advantage of the contamination effect is not exclusive to the BIS, given that a whole range of used mechanisms is not specifically designed for this system (Murray & Schaller, 2016; Woody & Szechtman, 2011).

Indeed, we found a link between psychophysiological markers of defensive activation and subsequent memory performance. Participants who demonstrated the contamination effect in behavior were distinctively linked with heart deceleration while recalling objects from the sick condition. These results are in line with the findings of Fiacconi et al. (2015) related to the survival processing effect. Moreover, our results demonstrated that the significant HR deceleration had no relationship with gender, which challenges Fernandes et al. (2021) conclusions. The authors noted that the lack of gender effects on the memory advantage indicated that emotion does not play a role, assuming that females are more susceptible to disgust than males. Due to the complexity of emotional experience and the fact that the conscious threshold comes in the later phases, not all emotional responses reach the level at which participants are able to report.

To our knowledge, the present findings offer the first psychophysiological evidence that links the memory advantage of the contamination effect to the functioning of the autonomic nervous system. Taking the increase in contamination potential, perceived disgust, fear, arousal, and negative valence together, the decrease in HR observed in the sick condition corresponds to the description of bradycardia, which is thought to be dominated by parasympathetic control and has been associated with defense-motivated behavior (Bradley et al., 2001). Our results revealed the role of emotionality in defensive engagement not only by contrasting the two encoding conditions but also by establishing a relationship between trial-by-trial variance in the HR response during encoding and future recall performance. We observed that emotionality is the mechanism driving this memory advantage, since the HR deceleration was deeper during the trials of remembered contaminated objects than healthy objects in the group of people who exhibited the contamination effect.

Previous research manipulated degrees of threat and found a gradient in the psychophysiological markers (Bradley, 2009; Bradley et al., 2001). On this premise, it can be suggested that the difference in HR deceleration may indicate an enhanced attentional-orienting response. Indeed, the amplification of the orienting response induced by disgust may, from an evolutionary standpoint, skew attention toward a deeper examination of the source of contamination and ease the preparation of an action that promotes fitness (Levenson, 2003; Liu et al., 2015; Löw et al., 2008). In a threatening situation, the attentional processes may enhance memory because they induce greater elaboration, thereby making the memory of that experience more accessible (Kroneisen & Erdfelder, 2011; Röer et al.,

2013). Higher attentional allocation is associated with disgust-eliciting stimuli, even more than fear (Moeck et al., 2021); and a hyperscanning pattern was shown to correlate with the number of correctly recalled disgust stimuli (Schienle et al., 2021). But such attentional processes do not promptly invalidate the emotionality account in explaining the memory advantage because attention is also a component of the emotional processing (Levenson, 2003). So much so that the impairment of the amygdala affects preattentive processing of threat-related stimuli, emotional memory, and disgust recognition in patients (e.g., Phelps, 2006; Woolley et al., 2015). Additionally, Moeck et al. (2021) demonstrated that participants paid more attention to images of disgust than to images of fear at encoding, but logistic mixed effects models found that attention did not contribute to disgust memory enhancement. Although this study did not have a contamination or survival paradigm, these findings suggest that the disgust category and not attention that predicted recall. Our results in the immediate memory test align with this view, although indirectly. Since participants' working memory was not enhanced for the sick condition, we conclude that their attentional allocation was likewise not enhanced for stimuli that elicited disgust. It can be argued that the immediate memory results might be explained by the sick descriptors being less efficient for signaling disease, but our results from the manipulation check do not support this argument.

Interestingly, in our rating task, the descriptors of the sick condition were evaluated as highly inducing of disgust and fear. Both disgust and fear activate the amygdala by being highly arousing, but disgust also activates the insula (Vytal & Hamann, 2010). For example, in studies where participants viewed images of contamination, the insula appeared to be one of the primary brain regions of activation (Wright et al., 2004). The disgust-specific insula activation may augment the effects of amygdala activation, which might underpin a higher enhanced memory (e.g., Yonelinas & Ritchey, 2015). Future studies could replicate the contamination paradigm using event-related potentials to examine if specific brain potentials respond to the sick condition in the insula when compared to a healthy condition, such as Krolak-Salmon et al. (2003) and Liu et al. (2015) observed in similar paradigms involving disgust. Future studies could also use functional magnetic resonance imaging to investigate if the sick condition in the advantage group demonstrates an enhanced activation of the insula (i.e., feeling disgust; Wicker et al., 2003) when compared to healthy conditions and to the non-advantage group.

Notably, HR deceleration is a parasympathetic index that also occurs during attention and orienting processes, with increased response to emotional stimuli (Bradley et al., 2001; Lang et al., 1993). In threatening situations, bradycardia results from vagal nerve activation initiated by the amygdala and its projections to the brainstem. This process is thought to facilitate the organism's sensory processing of its surroundings to assess threats. Along the same line, Olatunji et al. (2007) argue that defense responses controlled by the sympathetic and parasympathetic systems, such as

disgust, influence the brain and behavior because they are incorporated into a feedback loop about the status of the environment. For example, when the individual sees a disgust-eliciting stimulus, an autonomic response is generated, and indexes such as HR are associated with bradycardia patterns. Such an experience may be considered by the brain as an anticipatory stimulus to guide behavior quickly. Therefore, the role that emotional processes play in solving fitness-related problems would be adaptive because they enhance the possibility that future behavior choices would be better guided by higher attentional allocation, enhanced memory consolidation of threat-congruent information, and retrieval of past threat experience.

In our findings, the deceleration peak occurred early, consistent with previous research demonstrating that disgust induces a quicker and deeper HR deceleration than fear (Moeck et al., 2021). However, this earliness can also be influenced by anticipatory processes because the conditions of the first two trials in each block might be predictable for attentive participants. Nevertheless, disgust has a distinct response compared to fear. Instead of the typical fast and sympathetically driven fightor-flight response, disgust-eliciting stimuli call for a more cautious reaction, which might explain why disgust-eliciting stimuli produce greater initial HR deceleration compared to fear and neutral stimuli (Cisler et al., 2009; Moek et al., 2021). In comparison, the findings of Fiacconi et al. (2015), related to the deceleration peak, occurred after 2 s of stimulus onset, which was later than our peak. Moreover, the authors observed carryover effects into later stages of the HR. In our study, we did not formulate a hypothesis for later stages because our stimuli induced pathogenic disgust, which does not systematically produce an HR acceleration (Cisler et al., 2009), whereas fear-eliciting stimuli are characterized by an HR acceleration in later stages (Woody & Teachman, 2000).

4.3. Limitations and Future Directions

It is important to acknowledge some limitations of the present work to help and guide future research. First, our study was conducted right after the COVID-19 pandemic peak. Even though everyone has a BIS, this system is activated to different degrees based on individual dispositions and/or circumstances — and its sensitivity consequently varies. Therefore, the higher effect sizes of our study than those in Fernandes et al. (2017, 2021) may be the result of our data collection being carried out in such a timeframe, an issue that might have increased the sensibility of the BIS toward contamination threats (Makhanova & Shepherd, 2020).

Additionally, some individuals are more chronically attuned to the threat (actual or perceived) of infections, and their BIS is more likely to be activated than others (Makhanova, 2022). Future studies could administer questionnaires such as the Disgust Scale (Haidt et al., 1994), the Three Domain Disgust Scale (Tybur et al., 2009), or the Perceived Vulnerability to Disease (Duncan et al., 2009) to account for individual differences in the activation of the BIS.

Our results did not add information about the role of sympathetic arousal in the mnemonic benefit associated with the contamination effect. In paradigms investigating memory effects induced by emotional stimuli, the SCR has shown an increase (e.g., Phelps, 2006). Our results only supported the role of greater parasympathetic activation, observed as HR deceleration, which is associated with the initial stage of the defensive response.

Future research should examine whether the effects described here are influenced by gender. According to Fernandes et al. (2021), if disgust is the main emotion underlying contamination effects in memory, a bigger impact should be detected in females than in males. In opposition, our psychological findings supported the role of emotionality in memory advantage, and we did not find gender influence over the HR deceleration. However, our sample was predominantly female.

Future studies should perform a data reduction of the HR through weighted averages (Graham, 1978) instead of arithmetic averages because the latter wrongly assumes an equal interval between heartbeats. The baseline identification should also consider the best overlapping area in each group of advantage and non-advantage. Additionally, our manipulation checks focused on the descriptors. Still, future studies should also measure the participants' perception of the objects to measure the impact of the stimuli as a whole (i.e., picture of the object plus the descriptor) and to check the occurrence of the law of contagion. Future studies could also use more descriptors to avoid repetition and habituation effects; or longer stimuli, such as videos (Bonin et al., 2019), to investigate further the parasympathetic responses of contamination threats; or the BIS involvement by using antibody indexes in the saliva production (Keller et al., 2022). Lastly, eye-tracking could also be informative about the attentional modulation of disgust related to contamination threats in the advantage group versus the non-advantage group during the encoding in the sick condition.

4.4. Conclusion

The ultimate aim of the BIS is to keep humans from coming into contact with biological threats, and memory is an important component of this system. In line with prior research (Bonin et al., 2019; Fernandes et al., 2017, 2021; Gretz & Huff, 2019; Thiebaut et al., 2022), we observed that contaminated objects were remembered better than non-contaminated objects, even when presented in visually clean hands. Additionally, the findings of the present investigation support the hypothesis that the emotional response of pathogenic disgust, a key component of the defensive motivational model (Bradley et al., 2001; Lang et al., 2000), contributes to the contamination effect. Concerning the precise mechanism of mnemonic enhancement, we propose that disgust-related bradycardia may contribute to the memory advantage by raising the degree of cognitive activation in the contamination-threatening context. Consequently, our findings suggest the existence of a potentially crucial relationship between autonomic changes and cognitive processing in adaptive

memory. The exact interaction between cognitive and emotional systems is thus likely to be the most crucial aspect of future research on the contamination effect.

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Appendix A – Tables and Figures

 Table 1

 Counterbalanced design used to present descriptors and pictures to participants

| <u></u> | -i- | | | List 1 | | List 2 | | List 3 | List 4 | | |
|----------|-------|------------|------------|---------------------------------------|------------|---------------------------------------|-----------------------------------|---------------------------------------|------------|---------------------------------------|--|
| Block | Order | Picture | Condition | Descriptor | Condition | Descriptor | Condition | Descriptor | Condition | Descriptor | |
| 0 | 0.1 | teacup | sick_p1 | pessoa com dores de garganta | healthy_p1 | pessoa com um nariz reto | sick_p1 | pessoa com dores de garganta | healthy_p1 | pessoa com um nariz reto | |
| Practice | 0.2 | puzzles | healthy_p1 | pessoa com um nariz reto | sick_p1 | pessoa com dores de garganta | healthy_p1 | pessoa com um nariz reto | sick_p1 | pessoa com dores de garganta | |
| <u>-</u> | 0.3 | tiara | sick_p2 | pessoa com erupções na pele | healthy_p2 | pessoa com o cabelo castanho | sick_p2 | pessoa com erupções na pele | healthy_p2 | pessoa com o cabelo castanho | |
| | 1 | belt | sick_1 | pessoa com tosse constante | sick_1 | pessoa com erupções na pele | healthy_1 | pessoa com pescoço comprido | healthy_1 | pessoa com o cabelo castanho | |
| 1 | 2 | stapler | healthy_1 | pessoa com o cabelo castanho | healthy_1 | pessoa com um rosto redondo | sick_1 | pessoa com corrimento nasal | sick_1 | pessoa com dificuldades respiratórias | |
| | 3 | orange | sick_2 | pessoa com erupções na pele | sick_2 | _2 pessoa com corrimento nasal he | | pessoa com um rosto redondo | healthy_2 | pessoa com um nariz reto | |
| | 4 | glass | sick_3 | pessoa com erupções na pele | sick_3 | pessoa com dores de garganta | healthy_3 | pessoa com dedos longos | healthy_3 | pessoa com olhos verdes | |
| 2 | 5 | gloves | healthy_2 | pessoa com pescoço comprido | healthy_2 | pessoa com o cabelo castanho | sick_2 pessoa com tosse constante | | sick_2 | pessoa com erupções na pele | |
| | 6 | balloons | healthy_3 | pessoa com um rosto redondo | healthy_3 | pessoa com um nariz reto | sick_3 | pessoa com febre alta | sick_3 | pessoa com corrimento nasal | |
| | 7 | pepper | healthy_4 | pessoa com o cabelo castanho | healthy_4 | pessoa com olhos verdes | sick_4 | pessoa com tosse constante | sick_4 | pessoa com dores de garganta | |
| 3 | 8 | dices | sick_4 | pessoa com febre alta | sick_4 | pessoa com dificuldades respiratórias | healthy_4 | pessoa com um rosto redondo | healthy_4 | pessoa com um nariz reto | |
| | 9 | ruler | sick_5 | pessoa com corrimento nasal | sick_5 | pessoa com dificuldades respiratórias | healthy_5 | pessoa com o cabelo castanho | healthy_5 | pessoa com um rosto redondo | |
| | 10 | mushrooms | sick_6 | pessoa com tosse constante | sick_6 | pessoa com dores de garganta | healthy_6 | pessoa com o cabelo castanho | healthy_6 | pessoa com olhos verdes | |
| 4 | 11 | apple | healthy_5 | pessoa com um rosto redondo | healthy_5 | pessoa com um nariz reto | sick_5 | pessoa com erupções na pele | sick_5 | pessoa com dores de garganta | |
| | 12 | knife | healthy_6 | pessoa com dedos longos | healthy_6 | pessoa com olhos verdes | sick_6 | pessoa com febre alta | sick_6 | pessoa com dificuldades respiratórias | |
| | 13 | pencil | healthy_7 | pessoa com um nariz reto | healthy_7 | pessoa com dedos longos | sick_7 | pessoa com dores de garganta | sick_7 | pessoa com tosse constante | |
| 5 | 14 | watch | sick_7 | pessoa com corrimento nasal | sick_7 | pessoa com febre alta | healthy_7 | pessoa com olhos verdes | healthy_7 | pessoa com dedos longos | |
| | 15 | watermelon | healthy_8 | pessoa com olhos verdes | healthy_8 | pessoa com pescoço comprido | sick_8 | pessoa com dificuldades respiratórias | sick_8 | pessoa com febre alta | |
| | 16 | bracelet | healthy_9 | pessoa com olhos verdes | healthy_9 | pessoa com dedos longos | sick_9 | pessoa com corrimento nasal | sick_9 | pessoa com febre alta | |
| 6 | 17 | marbles | sick_8 | pessoa com dores de garganta | sick_8 | pessoa com erupções na pele | healthy_8 | pessoa com dedos longos | healthy_8 | pessoa com pescoço comprido | |
| | 18 | spoon | sick_9 | pessoa com febre alta | sick_9 | pessoa com tosse constante | healthy_9 | pessoa com pescoço comprido | healthy_9 | pessoa com o cabelo castanho | |
| | 19 | lemon | sick_10 | pessoa com dificuldades respiratórias | sick_10 | pessoa com febre alta | healthy_10 | pessoa com olhos verdes | healthy_10 | pessoa com pescoço comprido | |
| 7 | 20 | onion | healthy_10 | pessoa com um nariz reto | healthy_10 | pessoa com um rosto redondo | sick_10 | pessoa com dificuldades respiratórias | sick_10 | pessoa com corrimento nasal | |
| | 21 | scissors | sick_11 | pessoa com dores de garganta | sick_11 | pessoa com tosse constante | healthy_11 | pessoa com um nariz reto | healthy_11 | pessoa com dedos longos | |
| | 22 | broccoli | sick_12 | pessoa com dificuldades respiratórias | sick_12 | pessoa com corrimento nasal | healthy_12 | pessoa com um nariz reto | healthy_12 | pessoa com um rosto redondo | |
| 8 | 23 | fork | healthy_11 | pessoa com pescoço comprido | healthy_11 | pessoa com o cabelo castanho | sick_11 | pessoa com erupções na pele | sick_11 | pessoa com tosse constante | |
| | 24 | cards | healthy_12 | pessoa com dedos longos | healthy_12 | pessoa com pescoço comprido | sick_12 | pessoa com dores de garganta | sick_12 | pessoa com erupções na pele | |

Table 2Summary of Descriptor Evaluation by Condition

| Dimension | Sick | | Healtl | hy | +/70\ | | ٦ |
|-----------------------------|---------|------|---------|------|-------------|-------|------|
| Dimension | М | SD | М | SD | t(79) | р | d |
| Paired T-Tests | | | | | | | |
| Arousal | 4.96 | 2.31 | 2.24 | 2.03 | 11.74 | <.001 | 1.31 |
| Fear | 4.25 | 2.57 | 1.44 | 1.29 | 12.04 | <.001 | 1.35 |
| Disgust | 3.68 | 2.44 | 1.44 | 1.26 | 11.17 | <.001 | 1.25 |
| Wilcoxon Signed Rank Tests | M(Mdn) | | M(Mdn) | | W(z) | | r |
| Potential for Contamination | 5.22(6) | 2.55 | 1.45(1) | 1.35 | 3095(-7.40) | <.001 | .59 |
| Valence | 3.41(3) | 1.93 | 5.74(5) | 2.26 | 218(-6.45) | <.001 | .51 |

Table 3Summary of Mixed Effects Logistic Regression Model for Immediate Memory Performance

| Effect | Estimates | SE | 95% CI | | - 7 | n | R^2 |
|----------------------------------|-----------|------|--------|------|-------|--------|-------|
| | Estimates | 3E | LL | UL | - Z | р | |
| All participants | | | | | | | .37 |
| Fixed effects | | | | | | | |
| Intercept | 3.21 | 0.25 | 2.72 | 3.71 | 12.77 | < .001 | |
| Condition [Healthy] ^a | 0.71 | 0.21 | 0.29 | 1.13 | 3.33 | < .001 | |
| Random effects | | | | | | | .02 |
| Subjects | 1.35 | | | | | | |
| Advantage Group | | | | | | | .35 |
| Fixed effects | | | | | | | |
| Intercept | 3.06 | 0.32 | 2.44 | 3.68 | 9.66 | < .001 | |
| Condition [Healthy] ^a | 0.69 | 0.28 | 0.15 | 1.23 | 2.49 | .013 | |
| Random effects | | | | | | | .02 |
| Subjects | 1.28 | | | | | | |
| Non-Advantage Group | | | | | | | .40 |
| Fixed effects | | | | | | | |
| Intercept | 3.41 | 0.25 | 2.61 | 4.21 | 8.36 | < .001 | |
| Condition [Healthy] ^a | 0.75 | 0.21 | 0.09 | 1.41 | 2.22 | .027 | |
| Random effects | | | | | | | .03 |
| Subjects | 1.43 | | | | | | |

Note. LL = lower limit; UL = upper limit.

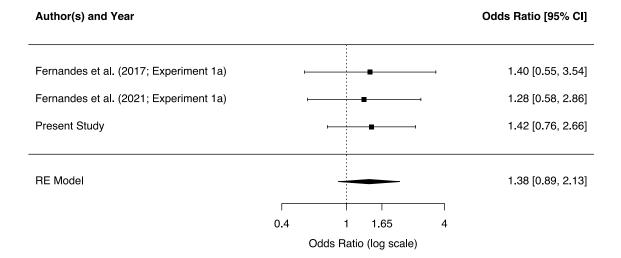
^a Immediate Memory Performance was coded as 0 = wrong answer, 1 = correct answer.

Table 4
Summary of Mixed Effects Logistic Regression Model for Recall Performance

| Effect | Estimate | SE | 95% | 6 CI | - | |
|------------------------------|----------|------|-------|------|-------|--------|
| Effect | Estimate | 3E | LL | UL | - Z | р |
| Fixed effects | | | | | | |
| Intercept | -0.01 | 0.20 | -0.41 | 0.39 | -0.05 | .958 |
| Condition[Sick] ^a | 0.47 | 0.10 | 0.27 | 0.66 | 4.62 | < .001 |
| Random effects | | | | | | |
| Subject | 0.33 | | | | | |
| Object | 0.92 | | | | | |

Note. Marginal R^2 = .01. Conditional R^2 = .24. $N_{Observations}$ = 1920.

Table 5Summary of Meta-Regression with Recall Performances of Previous and Present Studies



^a Recall Performance was coded as 0 = wrong answer, 1 = correct answer.

Appendix B - Flowchart of the Procedure



In this task, we ask you to recall objects that have been touched by different people; some of these people are infected with a deadly disease, others are healthy.

Throughout the experiment you will see pictures of objects presented together with a short description. This description will serve as a clue as to which person touched the object: the sick person or the healthy person.

You will have to decide whether the object was touched by the sick person or the healthy person and retain this information for a memory test.

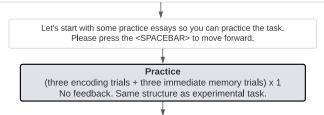
The objects and the corresponding description will be presented, one at a time, in sets of three. At the end of each set, each object will appear again and you will have to recall whether the object was touched by a sick person or a healthy person.

If the person who touched the object was the sick person, press the "V" key; if it was the healthy person, you should press the "M" key.

The description will not be displayed at the time you have to make this decision, so you will have to remember who touched each of the objects. After you answer each item, a new set of objects will appear, and this sequence of tasks will repeat.

ATTENTION: you will have only 5 seconds to see each object and its corresponding description. During the decision task, you will only have 5 seconds to decide whether the object has been touched by a sick or healthy person; you must decide within this time limit.

Please press the <SPACEBAR> to move forward.



The practice runs are over!

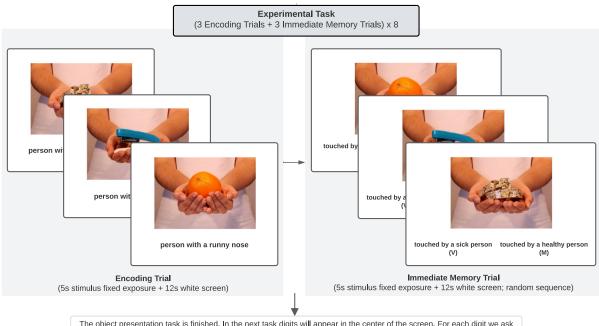
We remind you that in this task we ask you to recall objects that have been touched by different people; some of these people are infected with a deadly disease, others are healthy people. Throughout the experiment you will see pictures of objects presented along with a brief description. This description will serve as a clue as to which person touched the object: the sick person or the healthy person. You will have to decide whether the object was touched by the sick person or the healthy person and retain this information for a memory test.

The objects and the corresponding description will be presented one at a time, in sets of three. At the end of each set, each object will appear again and you will have to decide whether the object was touched by a sick person or a healthy person. If the person who touched the object was the sick person, press the key "V": if it was the healthy person, press the key "M". The description

healthy person. If the person who touched the object was the sick person, press the key "V": if it was the healthy person, press the key "M". The description will not be shown when you have to make this decision, so you have to remember who touched each object. After you answer each item, a new set of objects will appear and this sequence of tasks will repeat.

ATTENTION: you will have only 5 seconds to see each object and its corresponding description. During the decision task, you will only have 5 seconds to decide whether the object was touched by a sick or healthy person.

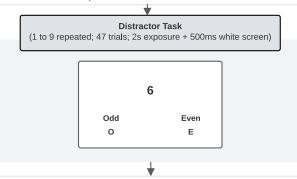
Please press the <SPACEBAR> to move forward.



The object presentation task is finished. In the next task digits will appear in the center of the screen. For each digit we ask you to press the 'O' key on your keyboard if the number is an odd number, or the "E" key on your keyboard if the number is an even number.

The numbers will appear very quickly, so you must pay attention.

Please press the <SPACEBAR> to move forward.



Now we would like you to recall the objects about which you made decisions during the first part of the experiment.

Please write the names of as many objects as you can remember in the space below. We would like you to remember all the objects about which you had to make a decision, regardless of what the decision was.

You have 5 minutes to complete this task. The program will automatically advance when that time is up.

Please spend the 5 minutes to remember as many objects as you can. It is normal that after a while you may feel that you cannot remember any objects anymore. However, our experience tells us that participants often remember more information when they persist with the task for the full time available.

Next the screen will be grayed out, and you should write down as many words as you can remember. To separate words you can use commas, periods, spaces, or the <Enter> key.

To move forward press the <SPACEBAR> key.



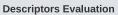
The experiment is almost over!

Next, we ask you to evaluate each of the descriptions that were presented to you earlier in a set of 5 dimensions. Each dimension will be accompanied by its response options. To answer, please use the 1 to 9 keys.

You have as much time as you need to answer each question; however, we ask you to answer quickly and intuitively respecting your reaction to the description presented.

ATTENTION: The order in which the dimensions to be evaluated are presented may vary from description to description, so it is important that you remain attentive.

To proceed to the evaluation of the words press the <SPACEBAR>



Potentital for Contamination + Disgust + Fear + Arousal + Valence (Self-paced)

To what extent do you feel fear when reading this description?

person with a runny nose

1 2 3 4 5 6 7 8 9

Not scared Very scared at all

Appendix C - R Script

0.1. Set Up

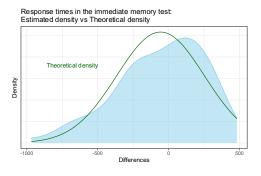
```
# set the working directory to the current folder
setwd(dirname(rstudioapi::getActiveDocumentContext()$path))
## load packages required in these analyses
new.packages <- list.of.packages[!(list.of.packages %in% installed.packages()[,"Package"])]</pre>
if (length(new.packages)) install.packages(new.packages) # install any new packages lapply(list.of.packages, require, character.only = TRUE) # library in all packages
rm(list.of.packages, new.packages)
# remove scientific notation
options(scipen = 999)
0.2. Main Data frame
# combine all E-PRIME txt files
listfile <- list.files(path = "raw_data", pattern = "txt", full.names = T, recursive = FALSE)
for (i in 1:length(listfile)) {
    if (i == 1) +
        assign(paste0("raw"), read.table(listfile[i], header = TRUE,
            sep = "\t", fileEncoding = "UTF-16"))}
    if (!i == 1) {
        assign(paste0("Test", i), read.table(listfile[i], header = TRUE,
        sep = "\t", fileEncoding = "UTF-16"))
raw <- rbind(raw, get(paste0("Test", i)))</pre>
        rm(list = ls(pattern = "Test"))}}
rm(i, list = ls(pattern = "listfile"))
# create a column with the version applied to each participant
raw <- raw %> mutate(Version = as.numeric(str_sub(ExperimentName, -1)),
                      descritor = tolower(descritor),
                      trigger = recode_factor(factor(trigger, exclude="7"), `5` = "sick", `3` ="sick",
                          `4` = "healthy", `2` = "healthy")) %>%
    relocate(Version, .after = Subject) %>%
arrange(as.numeric(Subject))
write_csv(raw, "0_raw.csv", na="")
1. Immediate Memory Test
df <- raw; names(df) <- gsub("\\.", "", names(df)) # clean columns' names</pre>
# gather variables relevant to the immediate task
imm <- df %>% dplyr::select(Subject,Version, ProcedureTrial,trigger,Imagem,JudgeACC, JudgeRT) %>%
dplyr::filter(ProcedureTrial == "judgetask") %>%
    mutate(ProcedureTrial = NULL, Imagem = str_sub(Imagem,6,-5))
# sum correct answers in the immediate task; one participant had 14 correct answers
imm_sum <- imm %>% group_by(Subject) %>%
  summarise(correct_total = sum(JudgeACC);
  correct_proportion = round(correct_total/24,2)) %>% arrange(correct_proportion)
write_csv(imm_sum, "1_immediate_memory_removed_participants.csv", na="
# 80 participants remained
imm <- imm[!(imm$Subject==58),]</pre>
df <- df[!(df$Subject==58),]</pre>
#---- Measure performance
imm_mean <- imm %>% group_by(trigger) %>%
  summarise_at(vars(starts_with("Judge")), list(mean=mean, sd=sd))
# correct answers
imm_t1 <- imm %>% dplyr::select(Subject, trigger, JudgeACC)
imm_fit <- glmer(JudgeACC~trigger + (1|Subject), data = imm_t1, family=binomial())</pre>
report(imm_fit)
# assumptions check
imm_fit_assumptions <- simulateResiduals(imm_fit)</pre>
svg("fig_1_immediate_memory_logistic_assumptions.svg")
plot(imm_fit_assumptions) # no over/underdispersion, no outliers
dev.off()
```

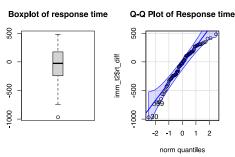
```
testCategorical(imm_fit_assumptions, catPred = imm_t1$Subject)
# response times
# assumptions check
imm_t2 <- imm %>% group_by(Subject, trigger) %>%
  summarise(correct = mean(JudgeACC), rt = mean(JudgeRT)) %>%
  pivot_wider(names_from = trigger, values_from = c(correct, rt)) %>%
  mutate(rt_diff = rt_sick - rt_healthy)
write_csv(imm_t, "2_immediate_memory_task_by_condition.csv", na="")
# compare estimated density and theoretical density
imm_p1 <- ggplot(imm_t2, aes(x=rt_diff)) +
  geom_density(fill="skyblue", color="skyblue", alpha=0.5) +</pre>
  stat_function(fun = dnorm, args = list(mean = mean(imm_t2$rt_diff), sd = sd(imm_t2$rt_diff)),
col="darkgreen") +
  theme_bw() + labs(x="Differences", y="Density") +
  theme(axis.text.y = element_blank(), axis.ticks.y = element_blank()) +
  ggtitle("Response times in the immediate memory test:\nEstimated density vs Theoretical density") +
annotate("text", color="darkgreen", x=-680, y=0.0009, label="Theoretical density")
ggsave("fig_2_density_immediate_memory_rt.svg", width = 6, height = 4)
# Boxplot and Q-Q Plot
svg("fig_3_distribution_immediate_memory_rt.svg", width = 6, height = 4)
par(mfrow = c(1, 2))
imm_p2 <- boxplot(imm_t2$rt_diff, main="Boxplot of response time")</pre>
imm_p3 <-qqPlot(imm_t2$rt_diff, line="robust", distribution = "norm", main="Q-Q Plot of Response time")</pre>
par(mfrow = c(1, 1))
dev.off()
# Anderson-Darling Test for normality
ad.test(imm_t2$rt_diff)
skew(imm t2$rt diff)/se.skew(imm t2$rt diff) # |-2| was = 2
# TOST paired samples t-test for response times
rm(list = ls(pattern = "imm"))
##---- Socio-demographic data
socio <- df %>% group_by(Subject,Version,Age,Sex,SPI, SessionDate) %>% summarise() %>%
  mutate(SPI = replace(SPI, SPI>0,1))
age <- c(summary(socio$Age), sd=round(sd(socio$Age),2));age</pre>
sex <- table(socio$Sex);sex</pre>
```

Assumptions for Immediate Memory Answers

QQ plot residuals Within-group deviation from uniformity n.s. Levene Test for homogeneity of variance n.s. 8 0.1 KS test: p= 0.86008 Deviation n.s. 0.8 simulationOutput\$scaledResiduals 0.75 9.0 st: p= 0.896 Dispersion 0.50 Deviation 0.4 0.25 0.2 Outlier test: n= 0.24493 0.0 0.333564734467199 1 0.0 0.2 0.4 0.6 0.8 1.0 Expected catPred

Assumptions for Immediate Memory Response Times





2. Descriptors

```
des <- df %>% dplyr::select(Subject,ProcedureBlock,descritor,ProcedureTrial,avaliacaoArousalRESP,
                                                  avaliacaoArousalRT,avaliacaoContaminacaoRESP,avaliacaoContaminacaoRT,
                                                 avaliacaoMedoRESP, avaliacaoMedoRT, avaliacaoNojoRESP, avaliacaoNojoRT,
                                                 avaliacaoValenciaRESP, avaliacaoValenciaRT) %>%
     dplyr::filter(ProcedureBlock =="avaliacaodescritores") %>%
     mutate(ProcedureBlock = NULL) %>%
     unite("rating", ends_with("RESP"), na.rm = TRUE) %>%
     unite("rt", ends_with("RT"), na.rm = TRUE) %>%
     na_if("") %>% na.omit %>%
     mutate(rating = as.numeric(rating),
                     ProcedureTrial = recode_factor(factor(ProcedureTrial),
                                                                                                   valencia = "Valence", arousa = "Arousal", medo = "Fear",
                                                                                                   nojo = "Disgust", contaminacao = "Contamination"),
                     condition = as.factor(case_when(str_detect(descritor,
                                                                           "erupções|dificuldades|febre|corrimento|dores|tosse") ~ "Sick",
                                                                           TRUE~ "Healthy")))
##---- Compare conditions
des_means <- des %>% group_by(ProcedureTrial, condition) %>%
    rating_sd = round(sd(rating), 2))
# assumptions check
des_p <- des %>% group_by(Subject, ProcedureTrial, condition) %>%
     summarize (rating_mean = mean(rating)) %>%
     pivot wider(names from = c(ProcedureTrial, condition), values from = rating mean) %>%
    mutate(valence_diff= Valence_Sick - Valence_Healthy,
                     arousal_diff= Arousal_Sick - Arousal_Healthy,
                     fear_diff = Fear_Sick - Fear_Healthy,
                     disgust_diff= Disgust_Sick - Disgust_Healthy,
                     contamination_diff = Contamination_Sick - Contamination_Healthy)
write_csv(des_p, "4_rating_descriptors_by_condition.csv", na="")
svg("fig_4_distribution_descriptors.svg", width=9, height=4)
par(mfrow = c(2, 3), mar= rep(2, 4))

qqPlot(des_p$valence_diff, line="robust", distribution = "norm", main="Valence")

qqPlot(des_p$arousal_diff, line="robust", distribution = "norm", main="Arousal")
qqPlot(des_p$fear_diff, line="robust", distribution = "norm", main="Fear")
qqPlot(des_p$disgust_diff, line="robust", distribution = "norm", main="Disgust")
qqPlot(des_p$contamination_diff, line="robust", distribution = "norm", main="Contamination")
par(mfrow = c(1, 1))
dev.off()
des_assump <- des_p %>% dplyr::select(Subject, ends_with("diff")) %>%
  gather(., key="trial", value="value", 2:6) %>% group_by(trial) %>%
     summarize(mean=mean(value),
                            A Statistic = ad.test(value)$statistic,
                            p_{value} = ad.test(value)p.value, # contamination and valence had p <.05 and skewness quocient
>|2|
                            asymmetry = ifelse(p value <0.05, skew(value)/se.skew(value), NA))</pre>
# valence and contamination were asymmetrical
des_p1 <- ggplot(des_p, aes(x=valence_diff)) +</pre>
    geom_density(fill="skyblue", color="skyblue", alpha=0.5) +
stat_function(fun = dnorm, args = list(mean = mean(des_p$valence_diff), sd = sd(des_p$valence_diff)),
col="darkgreen") +
     geom vline(xintercept = median(des p$valence diff), lwd = 0.6, col="darkblue") +
     \label{eq:theme_bw} \textbf{theme_bw}() + labs(x = "Differences", y = "Density") + theme(axis.text.y = element\_blank(), axis.ticks.y = theme_bw() + labs(x = "Differences", y = "Density") + theme(axis.text.y = element\_blank(), axis.ticks.y = theme_bw() + labs(x = "Differences", y = "Density") + theme(axis.text.y = element\_blank(), axis.ticks.y = theme_bw() + labs(x = "Differences", y = "Density") + theme(axis.text.y = element\_blank(), axis.ticks.y = theme_bw() + theme(axis.text.y = element\_blank(), axis.ticks.y = theme(axis.text.y = elem
element blank()) +
     ggtitle("Valence differences:\nEstimated density vs Theoretical density") +
    annotate("text", color="darkblue", x=-1, y=0.2, label="Median") + annotate("text", color="darkgreen", x=2.5, y=0.08, label="Theoretical density")
des_p2 <- ggplot(des_p, aes(x=contamination_diff)) +</pre>
    geom_density(fill="skyblue", color="skyblue", alpha=0.5) +
stat_function(fun = dnorm, args = list(mean = mean(des_p$contamination_diff), sd =
sd(des_p$contamination_diff)), col="darkgreen") +
     {\tt geom\_vline}({\tt xintercept = median(des\_p\$contamination\_diff), \ lwd = 0.6, \ col="darkblue") + lwd = 0.6, \ col="darkbl
     \label{eq:theme_bw} \texttt{theme_bw}() + \texttt{labs}(\texttt{x} = \texttt{"Differences"}, \texttt{y} = \texttt{"Density"}) + \texttt{theme}(\texttt{axis.text.y} = \texttt{element\_blank}(), \texttt{axis.ticks.y} = \texttt{theme\_bw}()
element_blank()) +
     ggtitle("Contamination potential differences:\nEstimated density vs Theoretical density") +
    annotate("text", color="darkblue", x=3, y=0.2, label="Median") + annotate("text", color="darkgreen", x=-1, y=0.08, label="Theoretical density")
ggarrange(des_p1, des_p2, ncol = 2, nrow = 1)
ggsave("fig_5_density_descriptors.svg", width = 9, height = 4)
```

```
svg("fig_6_boxplots_descriptors.svg", width = 6, height = 5)
par(mfrow = c(1, 2))
boxplot(des_p$contamination_diff, main="Boxplot of Contamination\npotential differences")
boxplot(des_p$valence_diff, main="Boxplot of Valence\ndifferences")
par(mfrow = c(1, 1))
dev.off()
# tests
des_t1 <- stats::wilcox.test(des_p$Valence_Sick, des_p$Valence_Healthy, paired=TRUE,</pre>
                                     exact=FALSE, alternative="two.sided", correct=TRUE)
des t1 z <-qnorm(des t1$p.value/2) # z value</pre>
\label{eq:des_t1_r} des\_t1\_r \ \leftarrow \ abs(des\_t1\_z) \ / \ sqrt(nrow(des\_p)*2) \ \# \ effect \ size
des_t2 <- ttestPS(des_p, pairs = list(list(i1 = 'Arousal_Sick', i2 = 'Arousal_Healthy')),</pre>
           bf=TRUE, hypothesis='different', effectSize=TRUE)
des_t3 <- ttestPS(des_p, pairs = list(list(i1 = 'Fear_Sick', i2 = 'Fear_Healthy')),</pre>
bf=TRUE, hypothesis='different', effectSize=TRUE)
des_t4 <- ttestPS(des_p, pairs = list(list(i1 = 'Disgust_Sick', i2 = 'Disgust_Healthy')),</pre>
             bf=TRUE, hypothesis='different', effectSize=TRUE)
des_t5 <- stats::wilcox.test(des_p$Contamination_Sick, des_p$Contamination_Healthy, paired=TRUE,
                                     exact=FALSE, alternative="two.sided", correct=TRUE)
des_t5_z <-qnorm(des_t5$p.value/2)</pre>
des_t5_r <- abs(des_t5_z) / sqrt(nrow(des_p)*2)</pre>
# gender differences related to reporting disgust
desc_disgust <- des %>% dplyr::filter(ProcedureTrial=="Disgust", condition=="Sick") %>%
 mutate(Sex = dplyr::recode(.$Sex, "female"= 0, "male" =1)) %>% dplyr::select(-rt)
#write_csv(desc_disgust, "4_rating_disgust_gender.csv")
desc_m <- lm(rating ~ Sex, data=desc_disgust)</pre>
report(desc_m)
##---- Descriptors' length
des_lenght <- des %>% group_by(descritor, condition) %>% summarise() %>% mutate(lenght =
str_length(descritor))
write_csv(des_lenght, "5_descriptors_lenght.csv", na="")
\mbox{\#} performed Shapiro-Wilk; p-values range between 0.121 and 0.225,
# normality is assumed (p > 0.05).
des_norm <- des_lenght %>% group_by(condition) %>%
  summarise(W_Statistic = shapiro.test(lenght)$statistic, p_value = shapiro.test(lenght)$p.value)
\# Pr(>F) ranges between 0.4556 and 0.5668, thus equal variances are assumed (p > 0.05).
des_levmean <- leveneTest(lenght ~ condition, des_lenght, center="mean")
des_levmed <- leveneTest(lenght ~ condition, des_lenght, center="median")</pre>
# perform an Independent Samples T-test
des_test <- apa(t.test(des_lenght$lenght ~ des_lenght$condition, var.equal=TRUE))</pre>
rm(list = ls(pattern = "des"))
                          Valence
                                                           Arousal
                                                                                              Fear
                                      43
            N
            0
                                              N
            Ņ
                                                                               0
                                              Ņ
                          Disgust
                                                         Contamination
                                              4
            N
                                              0
                                              0
            Q
                                              Q
             Valence differences:
Estimated density vs Theoretical density
                                                                                Boxplot of Contamination
                                                                                                     Boxplot of Valence differences
                                                                                     tial differences
           Density
                                                                                                   Q
                                                                               0
                                                                                                   4
                                                                               Q
                        Differences
                                                      Differences
```

3.1. Recall Task: Set Up

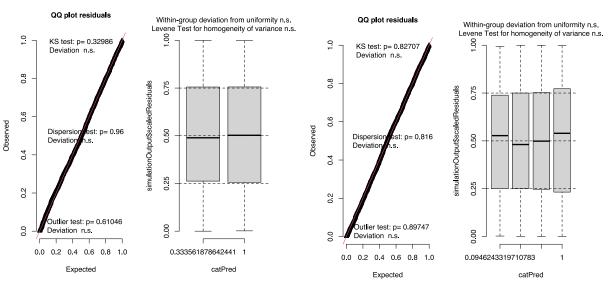
```
rec <- df %>% select(Subject,Sex,Version,ProcedureBlock,recallwordsRESP) %>%
      dplyr::filter(ProcedureBlock == "recall") %>%
      mutate(ProcedureBlock = NULL,
                       # clean the input
                       recallwordsRESP = str_replace_all(recallwordsRESP,
                                                                        recallwordsRESP = str_replace_all(recallwordsRESP,
\label{lem:ntrol} $$ NTROL \ \| (RIGHTARROW \) \| (ALT \) \| (CAPSLOCK \) \| (QUOTE \) \| (PICTURE \) \|
                       recallwordsRESP = str_trim(str_squish(tolower(recallwordsRESP)));
                       recallwordsRESP = str replace all(recallwordsRESP, "ma a", "maa"))
# make a separate column for each target object
for (i in loop_cues) {
    new <- str extract(rec$recallwordsRESP, i)</pre>
     rec[ , ncol(rec) + 1] <- new</pre>
names(rec)[5:28] <- loop_names</pre>
# make a column with incorrect words
rec$incorrect <- str_trim(str_squish(gsub(paste(unique(array(unlist(as.list(rec[, grep("^rec\\_+",</pre>
colnames(rec))])))), collapse = "|"), "", rec$recallwordsRESP)))
# make a column with total words
rec$total_words <- str_count(rec$recallwordsRESP, '\\w+')</pre>
write_csv(rec, "6_recall_validation.csv", na="")
### Summary of accepted and rejected words
rec_accepted <- data.frame(values = sapply(lapply(rec[5:29], unique), paste, collapse = ' '))
rownames(rec_accepted) <- sub("rec_", "", rownames(rec_accepted))
rec_accepted$values <- str_trim(str_squish(gsub("NA", "", rec_accepted$values)))
rec_accepted[25,] <- paste(sort(unique(unlist(str_split(rec_accepted[25,], " ")))), collapse=" ")
write.csv(rec_accepted, "7_recall_words_accepted.csv", na = "", row.names = TRUE)
rm(list = ls(pattern = "rec_accepted|loop|new"))</pre>
# make a separate column for each condition
rec_clean <- rec %>% dplyr::select(Subject, Sex, Version, starts_with("rec_")) %>%
    mutate_at(vars(matches("rec_")), ~ replace(., !is.na(.), 1)) %>% replace(is.na(.), 0) %>%
mutate(Sex = case_when(Sex=="female" ~ 1, Sex=="male" ~ 0)) %>%
     mutate if(is.character, as.numeric)
 objects\_a <- c("apple", "balloons", "bracelet", "cards", "fork", "gloves", "knife", "onion", "pencil", "pepper", "stapler", "watermelon") \\
for (i in objects_a) {
  rec_clean[[paste0(i,"_healthy")]] <- ifelse(rec_clean$Version < 3, rec_clean[[paste0("rec_",i)]], NA)</pre>
     rec_clean[[paste0(i,"_sick")]] <- ifelse(rec_clean$Version > 2, rec_clean[[paste0("rec_",i)]], NA)}
objects\_b <- c ("belt", "broccoli", "dice", "glass", "lemon", "marbles", "mushroom", "orange", "ruler", "marbles", "mushroom", "orange", "ruler", "marbles", "mushroom", "orange", "ruler", "marbles", "mushroom", "marbles", "mushroom", "orange", "ruler", "marbles", "mushroom", "mus
  "spoon",

"scissor", "watch")
for (i in objects_b) {
    rec_clean[[paste0(i,"_sick")]] <- ifelse(rec_clean$Version < 3, rec_clean[[paste0("rec_",i)]], NA)
    rec_clean[[paste0(i,"_healthy")]] = ifelse(rec_clean$Version > 2, rec_clean[[paste0("rec_",i)]], NA)}
rm(i,list = ls(pattern = "objects"))
3.2. Recall Task: Tests
###---- Main model
rec_t1 <- rec_clean %>% dplyr::select(Subject, Sex, apple_healthy:watch_healthy) %>%
     pivot_longer(cols=apple_healthy:watch_healthy, names_to = "condition", values_to = "performance") %>%
      drop_na() %>% mutate(object = factor(str_extract(condition, "[^_]+")),
condition = factor(gsub(".*_", "", condition),levels=c("healthy", "sick")))
trials <- data.frame(trials= c(11,6,16,24,23,5,12,20,13,7,2,15,1,22,8,4,19,17,10,3,9,18,21,14),
```

```
c('apple', 'balloons', 'bracelet', 'cards', 'fork', 'gloves', 'knife', 'onion', 'pencil',
'pepper', 'stapler', 'watermelon', 'belt', 'broccoli', 'dice', 'glass', 'lemon', 'marbles', 'mushroom', 'orange', 'ruler
', 'spoon', 'scissor', 'watch'))
rec_t1 <- left_join(rec_t1, trials)</pre>
write_csv(rec_t1, "8_recall_by_condition_regression.csv", na="")
rm(trials)
# model
rec_fit1 <- glmer(performance~condition + (1|Subject) + (1|trials), data = rec_t1, family=binomial())</pre>
report(rec_fit1); report_table(rec_fit1)
# assumptions
svg("fig_9_recall_main_assumptions.svg")
plot(simulateResiduals(rec_fit1)) # no over/underdispersion, no outliers, uniformity
dev.off()
# statistical power given sample size and effect size
wp.logistic(n = 80, p0 = 0.50, p1 = 0.59, family = "Bernoulli", alternative="greater")
## meta-regression
meta <- data.frame(author= c("Fernandes et al. (2017; Experiment 1a)",
                                     "Fernandes et al. (2021; Experiment 1a)",
                                     "Present Study"),
                        n = c(38, 48, 80),
                        ai = c(0.66, 0.52, 0.50), # healthy x outcome 0
                        bi = c(0.34, 0.48, 0.50), # healthy x outcome 1
                        ci = c(0.58, 0.45, 0.41), # sick x outcome 0
\label{eq:discrete} \begin{array}{rcl} & \text{di} = & c(0.42,0.55,0.59), \text{ \# sick x outcome 1} \\ \text{meta} & <- \text{ meta } \%\% \text{ mutate\_at(vars(ends\_with("i")), } & \sim \text{round}(.*n,0)) \end{array}
meta_reg <- rma.glmm(ai, bi, ci, di, n1i, n2i, measure="OR", data=meta, method="ML", test="z", slab=author)
svg("fig_12_meta-regression.svg", width=9, height=5)</pre>
forest(meta_reg,atransf=exp, xlim=c(-4.5,3.5), header="Author(s) and Year", at=log(c(.4, 1, 1.65, 4)))
dev.off()
###---- Gender
 rec\_fit2 <- glmer(performance \sim condition*Sex + (1|Subject) + (1|trials), \ data = rec\_t1, \ family=binomial()) 
report(rec_fit2); report_table(rec_fit2)
svg("fig_10_recall_assumptions_gender.svg")
plot(simulateResiduals(rec_fit2))
dev.off()
```

Assumptions for Main Model

Assumptions for Model with Gender



3.3. Recall Task: Visualization

```
plot <- rec_t1 %>% dplyr::select(Subject,condition, performance) %>%
  mutate(trial = rep(1:12, 160)) %>% pivot_wider(names_from = condition, values_from = performance) %>%
  relocate(healthy,.after = last_col())
# Anscombe transformation
A <-function(v) {
  x \leftarrow sum(v)
  n <- length(v)</pre>
  asin(sqrt((x+3/8)/(n+3/4)))
SE.A <- function(v) {
  0.5 / sqrt(length(v+1/2))
CI.A <- function(v, gamma = 0.95){
 SE.A(v) * sqrt( qchisq(gamma, df=1) )
# proportion of success
prop <- function(v){</pre>
  x \leftarrow sum(v)
  n <- length(v)
 x/n
# de-transformed confidence intervals from Anscombe-transformed scores
CI.prop <- function(v, gamma = 0.95) {</pre>
     <- A(v)
 У
        <- length(v)
  n
  cilen <- CI.A(v, gamma)</pre>
 ylo <- y - cilen
yhi <- y + cilen
  # reverse arc-sin transformation: naive approach
  cilenlo <- ( sin(ylo)^2 )
  cilenhi <- ( sin(yhi)^2 )</pre>
  c(cilenlo, cilenhi)
}
superbPlot(plot,
            WSFactors = "Moments(2)",
variables = c("healthy", "sick"),
            statistic = "prop",
error = "CI",
            adjustment = list(purpose = "difference", decorrelation="CA"),
            plotStyle = "point",
            errorbarParams = list(color=c("#E31B23","#005CAB")),
pointParams = list(size = 4, color = c("#E31B23","#005CAB"))
) + theme_bw(base_size = 10) +
  theme(axis.text = element_text(size = 12), axis.title = element_text(size = 12, face="bold")) +
  labs(y = "Proportion of correct recall\n", x =  "\nCondition") +
  scale_x_discrete(labels=c("Sick", "Healthy")) +
  scale_y_continuous(limits = c(0.3, 0.8)) +
  geom_hline(yintercept=mean(rec_test$proportion_healthy), linetype="dashed", size=0.3)
ggsave("fig_12_recall_proportionplot.svg", width = 4, height = 4)
```

Appendix D - Python Script

The data was simulated in this script for illustration purposes.

0.1. Setup

```
participant = "x"
sampling_rate = 1000
%matplotlib widget
# Import the necessaries libraries
import numpy as np
import pandas as pd
import neurokit2 as nk
import bioread
import mne
import scipy.signal
import scipy.io
from sklearn.neighbors import NearestNeighbors
from sklearn.cluster import DBSCAN
import pywt
import matplotlib.pyplot as plt
import plotly.express as px
import plotly.offline as pyo
import plotly.graph_objects as go
pyo.init_notebook_mode()
```

0.2. Extract ECG data

```
datasource_acq = bioread.read_file("".join(["biopac/", participant, ".acq"]))
digital_input_a = datasource_acq.channels[2].data
digital_input_b = datasource_acq.channels[3].data
digital_input_c = datasource_acq.channels[4].data
ecg_raw = datasource_acq.channels[0].data
time = datasource_acq.channels[0].time_index
print('We have',len(time),'data points on the ECG signal from the AcqKnowledge file.')
```

We have 1825978 data points on the ECG signal from the AcqKnowledge file.

0.3. Extract Event Markers

```
window_after=12998
window_before=4000
# remove the overshoots of the digital input
 for i in range(len(t)):
            if t["input_b"].loc[i]==5 and t["input_a"].loc[i]==0 and t["input_a"].loc[i+1]==5:
            t["input_b"].loc[i]= 0
elif t["input_b"].loc[i]==5 and t["input_b"].loc[i+1]==0 and t["input_b"].loc[i-1]==0:
                        t["input_b"].loc[i]= 0
 # identify conditions
 val=[]
 for i in range(len(t)):
            if t["input_a"].loc[i]== 0 and t["input_b"].loc[i]==5 and t["input_b"].loc[i+1]==0 and
 t["input c"].loc[i]== 0:
                        val.append(2)
            elif \ t["input\_a"].loc[i] == 5 \ and \ t["input\_b"].loc[i] == 5 \ and \ t["input\_b"].loc[i+1] == 0 \ and \ t["input\_b"
 t["input_c"].loc[i]== 0:
                        val.append(3)
            else:
                        val.append(np.nan)
 t['marker'] = pd.DataFrame(val).astype("Int64") # 2 (healthy), 3 (sick)
events = t[["index","time","marker"]]
# add count of trials (e.g., 2_1, 2_2, ...)
events['trial'] = events['marker'].copy().apply(str).str.cat(events.groupby(events.marker,
dropna=True).cumcount().add(1).astype(str),sep='_')
events.loc[events['trial'].str.contains('<NA>'), 'trial'] = np.nan
# extend marker throught the trials duration
```

```
events['trial'] = events['trial'].ffill(limit = window_after)
events['trial'] = events['trial'].bfill(limit = window_before)

# count every time point of the trials
events['epoch'] = events['trial'].str.cat(events.groupby(['trial']).cumcount().add(1).astype(str),sep='_')

# identify condition
events["label"] = events["trial"].copy().replace(regex=[r'^2_*', r'^3_*'], value=["Healthy", "Sick"])
```

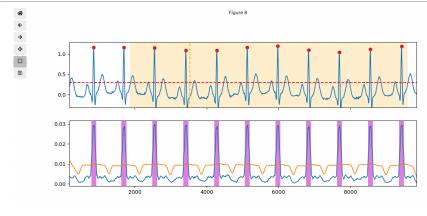
Filter

2. Cut Area of Interest

3. R-Peaks Detection

```
def _ecg_findpeaks_neurokit(signal, sampling_rate, smoothwindow, avgwindow, gradthreshweight, minlenweight,
mindelay, height, show):
    if show is True:
        __, (ax1, ax2) = plt.subplots(nrows=2, ncols=1, sharex=True)
    # gradients
    grad = np.gradient(signal)
    absgrad = np.abs(grad)
    smooth_kernel = int(np.rint(smoothwindow * sampling_rate))
    avg_kernel = int(np.rint(avgwindow * sampling_rate))
    smoothgrad = nk.signal_smooth(absgrad, kernel="boxcar", size=smooth_kernel)
avggrad = nk.signal_smooth(smoothgrad, kernel="boxcar", size=avg_kernel)
    gradthreshold = gradthreshweight * avggrad
    mindelay = int(np.rint(sampling_rate * mindelay))
    if show is True:
        ax1.plot(signal)
        for i in events_list["index"]: # code added
             ax1.axvline(i, color="orange", linestyle="--") # code added
        ax1.axvspan(i-1000, i+5000, facecolor="orange", alpha=0.2) # code added ax1.axhline(height, xmin=0, xmax=1, color="red", linestyle="--") # code added
        ax2.plot(smoothgrad)
        ax2.plot(gradthreshold)
    # identify start and end of QRS complexes.
    qrs = smoothgrad > gradthreshold
    beg_qrs = np.where(np.logical_and(np.logical_not(qrs[0:-1]), qrs[1:]))[0]
    end_qrs = np.where(np.logical_and(qrs[0:-1], np.logical_not(qrs[1:])))[0]
    # throw out QRS-ends that precede first QRS-start.
    end_qrs = end_qrs[end_qrs > beg_qrs[0]]
    # identify R-peaks within QRS (ignore QRS that are too short).
    num_qrs = min(beg_qrs.size, end_qrs.size)
    min_len = np.mean(end_qrs[:num_qrs] - beg_qrs[:num_qrs]) * minlenweight
    peaks = [0]
    for i in range(num qrs):
        beg = beg qrs[i]
        end = end_qrs[i]
        len_qrs = end - beg
```

```
if len_qrs < min_len:</pre>
            continue
       if show is True:
            ax2.axvspan(beg, end, facecolor="m", alpha=0.5)
       # find local maxima and their prominence within QRS.
       data = signal[beg:end]
       locmax, props = scipy.signal.find_peaks(data, height, distance=None, prominence=(None, None))
#modified
       if locmax.size > 0:
            # identify most prominent local maximum.
            peak = beg + locmax[np.argmax(props["prominences"])]
            # enforce minimum delay between peaks.
            if peak - peaks[-1] > mindelay:
               peaks.append(peak)
   peaks.pop(0)
    if show is True:
       ax1.scatter(peaks, signal[peaks], c="r")
        count_peaks = list(range(len(peaks))) # code added
        for i,txt in enumerate(count_peaks): # code added
            ax1.annotate(txt, (peaks[i], signal[peaks][i] + 0.5),color="red", fontsize = 7) # code added
    peaks = np.asarray(peaks).astype(int) # convert to int
    peaks_amp = np.asarray(signal[peaks]).astype(int)
    return peaks, peaks_amp
rpeaks, rpeaks_amp = _ecg_findpeaks_neurokit(ecg_filtered_cut, sampling_rate, show=True,
```



```
rpeaks_df = pd.DataFrame(rpeaks, columns = ["index"])
rpeaks_df["rpeaks"] = range(len(rpeaks_df))
data = data.merge(rpeaks_df, how="left", on="index")
data['rpeaks'] = data['rpeaks'].astype("Int64")
data['rpeaks_amplitude'] = data["ecg_filtered"][rpeaks]
data['rpeaks_index'] = data["index"][rpeaks].astype("Int64")
rpeaks_index = data[data['rpeaks_index']>0].index.to_numpy()
rpeaks_amplitude = data["rpeaks_amplitude"][data['rpeaks_amplitude']>0].to_numpy()
data[data["rpeaks_index"]>0].head(3)
```

4. Artifacts Detection

```
artifacts, rpeaks_corrected = nk.signal_fixpeaks(rpeaks, iterative=True, method="Kubios", show=True)

rpeaks_corr_df = pd.DataFrame(rpeaks_corrected, columns = ["index"])
rpeaks_corr_df["rpeaks_corr"] = range(len(rpeaks_corr_df))
data = data.merge(rpeaks_corr_df, how = 'left', on="index")
data["rpeak_corr_amplitude"] = data.loc[data["rpeaks_corr"] >= 0, "ecg_filtered"]
total_artifacts = np.array(artifacts['ectopic'] + artifacts['missed'] + artifacts['extra'] +
artifacts['longshort'])
location_artifacts = data[data["rpeaks"]>=0].iloc[total_artifacts]
prop_corr_rpeaks_events = len(location_artifacts[~location_artifacts["trial"].isnull()])/len(rpeaks)
```

```
events_artifacts = location_artifacts[~location_artifacts["trial"].isnull()]
"R-Peaks corrected inside events: {} ({}%)".format(len(events_artifacts),
round(prop_corr_rpeaks_events*100,4))
```

5. Rate

```
rate = nk.signal_rate(rpeaks, sampling_rate=1000, desired_length=len(ecg_filtered_cut),
interpolation_method="monotone_cubic"))
rate_corrected = nk.signal_rate(rpeaks_corrected, sampling_rate=1000, desired_length=len(ecg_filtered_cut),
interpolation_method="monotone_cubic")
rate_df = pd.DataFrame({"index":list(range(len(rate))), "rate": rate, "rate_corrected": rate_corrected})
data = data.merge(rate_df, how= "left", on="index")
```

6. Store Epochs

```
epochs = data[data['epoch'].notna()]
epochs["participant"] = participant
epochs['label_id'] = epochs['trial'].astype(str) + participant[2:]
size = len(epochs[epochs["trial"]=="3_1"])
epochs["window"] = (list(np.linspace(-3,14.999,size)))*24
```

```
epoch_pivot_rate_corrected = epochs.pivot(index='label_id', columns='window', values='rate_corrected')
len(epoch_pivot_rate_corrected[epoch_pivot_rate_corrected.isna().any(axis=1)]) # How many null values?
```

0

7. Save Results

pivot_log_out.to_csv('dataframes/eda/pivot_log_out_{0}.csv'.format(participant), sep='\t', encoding='utf-8')
dat.to_csv('dataframes/eda/processed/processed_{0}.csv'.format(participant), sep='\t', encoding='utf-8')

^{&#}x27;R-Peaks corrected inside events: 1 (0.0867%)'

Appendix E – Supplementary Analysis

Table 1Summary of Free Recall Performance Comparison Between Conditions

| Dimension — | Sick | | | Healthy | | +/70\ | _ | ا | DE |
|------------------------------------|------|-----|----------|---------|-----|-------|-------|----------|------------------|
| Differsion | М | SD | | М | SD | t(79) | р | d | BF ₁₀ |
| Paired T-Tests at Individual Level | | | | | | | | | |
| Immediate Memory | .92 | .13 | ≠ | .96 | .08 | -2.37 | .020 | -0.27 | 1.71 |
| Free Recall | .59 | .13 | > | .50 | .17 | 4.42 | <.001 | 0.49 | > 100 |
| Paired T-Tests at Item Level | | | | | | t(23) | | | |
| Free Recall | .59 | .21 | > | .50 | .21 | 3.79 | <.001 | 0.77 | > 100 |

Note. These data were derived from proportions. Therefore, t-tests were not our first choice. Since original studies of the contamination paradigm used t-tests, we still run those tests. The effect size of free recall performances in our data was larger than in the original studies.