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Anticipation of electric shocks modulates low beta power and event-related fields during memory encoding

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ABSTRACT

In humans, the temporal and oscillatory dynamics of pain anticipation and its effects on long-term memory are largely unknown. Here, we investigated this open question by using a previously established behavioral paradigm in combination with magnetoencephalography (MEG). Healthy human subjects encoded a series of scene images, which was combined with cues predicting an aversive electric shock with different probabilities (0.2, 0.5 or 0.8). After encoding, memory for the studied images was tested using a remember/know recognition task. Behaviorally, pain anticipation did not modulate recollection-based recognition memory per se, but interacted with the perceived unpleasantness of the electric shock [visual analogue scale rating from 1 (not unpleasant) to 10 (highly unpleasant)]. More precisely, the relationship between pain anticipation and recollection followed an inverted u-shaped function the more unpleasant the shocks were rated by a subject. At the physiological level, this quadratic effect was mimicked in the event-related magnetic fields associated with successful memory formation ('DM-effect') ~450 ms after image onset at left frontal sensors. Importantly, across all subjects, shock anticipation modulated oscillatory power in the low beta frequency range (13–20 Hz) in a linear fashion at left temporal sensors. Taken together, our findings indicate that beta oscillations provide a generic mechanism underlying pain anticipation; the effect on subsequent long-term memory, on the other hand, is much more variable and depends on the level of individual pain perception. As such, our findings give new and important insights into how aversive motivational states can drive memory formation.

1. Introduction

Rewards and punishments are motivating factors of goal-directed behavior in learning animals and humans. In line with the evolutionary need of a rapid learning system, electrophysiological studies (magnetoencephalography [MEG], electroencephalography [EEG]) show that both, nociceptive information and the anticipation of aversive stimuli, are signaled in various brain regions already at 100 ms after stimulus presentation (Garcia-Larrea, Frot, & Valeriani, 2003; Iannetti, Zambreanu, Cruccu, & Tracey, 2005; Pizzagalli, Greischar, & Davidson, 2003; Ploner, Gross, Timmermann, Pollok, & Schnitzler, 2006) (aversive anticipation: Dolan, Heinze, Hurlemann, & Hinrichs, 2006; Weymar, Bradley, Hamm, & Lang, 2013). Particularly, recordings in animals show that dopaminergic neurons in the substantia nigra/ventral tegmental area (SN/VTA) rapidly respond (onset latency of ~100 ms) not only to cues associated with reward (Schultz, 2007; Tobler, 2005) but also upcoming threat (Bromberg-Martin, Matsumoto, & Hikosaka, 2010; Ilango et al., 2014; Lammel et al., 2012). More specifically, in monkeys, SN/VTA activity increased linearly with the cue's probability to predict an aversive air puff to the eye and an appetitive juice drop, respectively (Matsumoto & Hikosaka, 2009).

While there are several findings in animals supporting the involvement of the dopaminergic midbrain in anticipating aversive events, there is only little evidence in humans (Bauch, Rausch, & Bunzeck, 2014; Fairhurst, Wiech, Dunckley, & Tracey, 2007). In an initial attempt to bridge this apparent gap between both species, we previously used fMRI in humans (Bauch et al., 2014). As a main finding, we could show that activity in the SN/VTA linearly increases as a function of shock probability during the anticipation of aversive events (electric shocks to the hand). However, the precise underlying temporal and oscillatory nature of shock anticipation remains unclear due to the sluggish properties of the BOLD signal.

In contrast, electrophysiological techniques such as EEG and MEG offer the possibility to investigate neural activity at the level

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of milliseconds, which enables us to dissociate between multiple processes and underlying neural mechanisms involved in pain anticipation. Previous studies showed that pain anticipation can influence early sensory components of electrical brain activity (Babiloni et al., 2003; Dillmann, Miltner, & Weiss, 2000; Miyazaki et al., 1994; Weymar et al., 2013) and nociceptive events modulate oscillatory power in a range of frequency bands (May et al., 2012; Mouraux, Guérit, & Plaghki, 2003; Ohara, Crone, Weiss, & Lenz, 2004; Ploner et al., 2006; Pomper et al., 2013; Raij, Forss, Stancák, & Hari, 2004). For instance, oscillatory power in the entire beta frequency range [low beta (13–20 Hz), high beta (20–30 Hz)] have repeatedly been shown to decrease (i.e. beta band suppression) in response to painful stimuli relative to non-painful or less painful events (Hauck, Lorenz, & Engel, 2007; Mancini, Longo, Canzoneri, Vallar, & Haggard, 2013; Ploner et al., 2006; Pomper et al., 2013; Senkowski, Kautz, Hauck, Zimmermann, & Engel, 2011; Stančák, Poláček, Vrána, & Mlynář, 2007). In contrast, beta power increases in response to tonic pain stimuli (Chang, Arendt-Nielsen, & Chen, 2002; Chang, Arendt-Nielsen, Graven-Nielsen, & Chen, 2003; Lalo et al., 2007) or long phasic stimulation (~400 ms stimulation duration, Worthen, Hobson, Hall, Aziz, & Furlong, 2011). Moreover, modulations in the lower frequency range (theta: 4–8 Hz; alpha: 9–12 Hz) have been linked to nociceptive processing (Domnick, Hauck, Casey, Engel, & Lorenz, 2009; Iannetti, Hughes, Lee, & Mouraux, 2008; Mouraux et al., 2003).

However, compared to the delivery of the nociceptive stimulus, it is largely unknown whether beta and theta power also signal the anticipation of an aversive and painful event. Initial evidence of beta power (14–30 Hz) increases during the anticipation phase of nociceptive stimuli has been reported in an MEG study (Worthen et al., 2011), where beta power modulations have been interpreted as a binding mechanism of pain-associated processes between different brain regions.

In contrast to aversive anticipation, there is increasing evidence that the anticipation of appetitive stimuli, such as reward, modulates oscillatory power in the theta and beta frequency range (Bunzeck, Guitart-Masip, Dolan, & Düzel, 2011; Doñamayor, Marco-Pallarés, Heldmann, Schoenfeld, & Münte, 2011; van Wingerden, Vinck, Lankelma, & Pennartz, 2010). More specifically, in line with animal studies (Fiorillo, Tobler, & Schultz, 2003), the human brain quickly responds to cues that predict monetary rewards (i.e. ~100 ms after cue onset). Moreover, while oscillatory power in the theta (5–8 Hz) band linearly decreases as a function of reward probability, high beta power (20–30 Hz) increases with reward probability (Bunzeck et al., 2011). Here, following the rationale of similar coding strategies between reward and aversive processing (Bauch et al., 2014; Bromberg-Martin et al., 2010), we tested the hypothesis that oscillatory power in the theta and beta frequency range also signals the anticipation of aversive events in a linear fashion.

Behaviorally, aversive stimuli such as painful, electric shocks can have beneficial effects on long-term memory. More specifically, when aversive events are applied briefly after encoding, they can increase recognition memory possibly via enhanced arousal (Schwarze, Bingel, & Sommer, 2012; see also Dunsmoor, Martin, & LaBar, 2012; McCullough & Yonelinas, 2013). In a more recent study, we (Bauch et al., 2014) showed that even the anticipation of nociceptive events influences recognition memory. Recollection – recognition memory for contextual details of the studied episode (Tulving, 2002) – was modulated by shock probability following an inverted u-shape function. In other words, recollection was best for cues predicting an upcoming shock with a shock probability of 50%. This quadratic effect was mimicked by encoding-related activity in the posterior hippocampus. In contrast, familiarity – a general feeling of knowing the event in absence of contextual details (Tulving, 2002) – was linearly scaled as a function of shock probability, which was paralleled by a linear increase in the anterior parahippocampal gyrus.

Here, we used MEG to investigate the temporal and oscillatory dynamics of pain anticipation as a function of probability and its link to declarative memory formation. In a classical conditioning paradigm, participants initially learned to associate three different picture frames with three different probabilities (0.2, 0.5 or 0.8) to predict an electric shock. During an incidental encoding task, subjects categorized a series of indoor/outdoor scene images that were surrounded by these picture frames and were followed by an electric shock consistent with the frame's probability. Approximately 15 min after encoding, recognition memory for the scene images was tested using a modified remember/know recognition task (Fig. 1). We predicted that the anticipation of aversive electric shocks is processed rapidly and signaled in beta (low beta: 13–20 Hz; high beta: 20–30 Hz) and/or theta (4–8 Hz) oscillatory power as a function of shock probability, similar to the effects in animal studies (Matsumoto & Hikosaka, 2009) and the reward literature (Bunzeck et al., 2011). Finally, we expected temporal and oscillatory modulations of brain activity associated with successful memory encoding as a function of shock probability (Bauch et al., 2014). A common index for successful memory formation is the so called 'DM-effect' (difference due to later memory, Paller, Kutas, & Mayes, 1987), that refers to the difference between encoding-related brain activity of later remembered and forgotten items.

Previous M/EEG studies showed theta power increases (Duzel, Penny, & Burgess, 2010) and beta power decreases (Hanslmayr, Spitzer, & Bäuml, 2009; Hanslmayr, Staudigl, & Fellner, 2012) for remembered relatively to forgotten items (i.e. DM-effect). Based on these findings and our recent fMRI study (Bauch et al., 2014), showing increased hippocampal DM-activations for 0.5 shock probability, we expected increased theta power and decreased beta power for the DM-effect in the 0.5 shock probability condition relative to DM-effects associated with 0.2 and 0.8 shock probability.

2. Materials and methods

2.1. Participants

43 healthy young adults participated in the present study (27 female, age range 20–34 years, mean 25 years, age range: 20–34 years). All participants were right-handed and had normal or corrected-to-normal vision, no history of neurological, psychiatric, or medical disorders or any current medical problems. Each participant gave written informed consent according to the approval of the local ethics committee (medical association Hamburg).

2.2. Task

The entire experiment took place while the subjects were placed in the MEG-scanner. Individual pain thresholds were calibrated before the actual experiment started. Here, participants rated the intensity of the electric shock on a visual analog scale (VAS) ranging from 0 (i.e. electric stimulation is not perceptible) to 10 (i.e. electric stimulation is intolerable). A shock intensity of seven was used as nociceptive stimulus throughout the experiment. All participants took part in three consecutive phases: a conditioning phase, an encoding task and a memory recognition task.

In the conditioning phase, 20 green, 20 blue and 20 red colored, rectangular picture frames were randomly intermixed and presented in central vision for 1.5 s on a gray background (Fig. 1). Participants implicitly learned that the color of the cue (i.e. green, blue or red picture frame) predicted an aversive electric shock with