



Oxytocin improves compassion toward women among patients with PTSD



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ABSTRACT

Although impairments in social skills, including empathic abilities, are common in post-traumatic stress disorder (PTSD), the ability to feel compassion—a pro-social behavior that is based on empathy and drives us to help others—has never been assessed among these patients. The first aim of this study was to examine whether patients with PTSD suffer from deficits in compassion and to examine the association between the clusters of PTSD symptoms and these deficits. Furthermore, given that intranasal oxytocin (OT) has been suggested to possibly modulate social behaviors, the second aim of this study was to investigate whether intranasal OT may enhance compassion in these patients. Using a randomized, double-blind, placebo-controlled crossover design, we administered 24 IU of OT and placebo at a one-week interval to 32 patients with PTSD and to 30 matched healthy control participants. The results indicate that patients with PTSD exhibit deficits in compassion and that the numbing cluster emerged as the key predictor of those deficits. Moreover, the results indicate that a single intranasal dose of OT enhances compassion toward women (but not towards men), both in patients with PTSD and in controls. These results offer support for recent suggestions that intranasal OT may potentially be an effective pharmacological intervention for patients with PTSD.

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1. Introduction

Post-Traumatic Stress Disorder (PTSD) is a disorder that develops following one or more traumatic events and is characterized by intrusion memories, avoidance of trauma-related stimuli, negative alteration in cognition and mood and hyper-arousal (American Psychiatric Association, [APA] 2013). This disorder causes significant distress and impairs broad areas of functioning, including social and interpersonal skills (Charuvastra and Cloitre, 2008; Maercker and Horn, 2013). Studies among patients with PTSD have shown that this disorder is related to difficulties across several domains involving social relationships difficulties, including problems with partners, children and friends (see Maercker and Horn, 2013; Monson et al., 2009 for review). In particular, *emotional numbing* symptoms – a cluster of symptoms characterized by a restricted range of affect, anhedonia and detachment (e.g., Kashdan et al., 2006; Orsillo et al., 2007) – were significantly associated with

these relationship difficulties (e.g., Cook et al., 2004; Kuhn et al., 2003; Samper et al., 2004).

Recent studies have suggested that the social difficulties characterizing patients with PTSD are caused, at least in part, by impairment in *empathy*—the ability to understand the other's thoughts, desires and feelings (Nietlisbach and Maercker, 2009; Nietlisbach et al., 2010; Plana et al., 2013). In this study we take a further step in attempting to understand empathy deficits in PTSD and examine levels of compassion among these patients.

Recently it has been suggested that the ability to respond in a compassionate way is a key factor of the empathic response (Goetz et al., 2010). *Compassion* is a combination of salient pro-social feelings that drive us to help others. Compassion may be defined as the multi-dimensional feeling of warmth, understanding, sadness and kindness that arises in witnessing the distress and suffering of others and that motivates the desire to help and care for others (Goetz et al., 2010; Singer and Klimecki, 2014). It has been suggested that compassion is based on empathy, and that the compassionate response rely on two different components of empathy: *emotional empathy* – the ability to automatically experience affective reactions of others and to recognize their feelings, and *cognitive empathy* – the ability to cognitively understand and represent the thoughts, desires and feelings of others.

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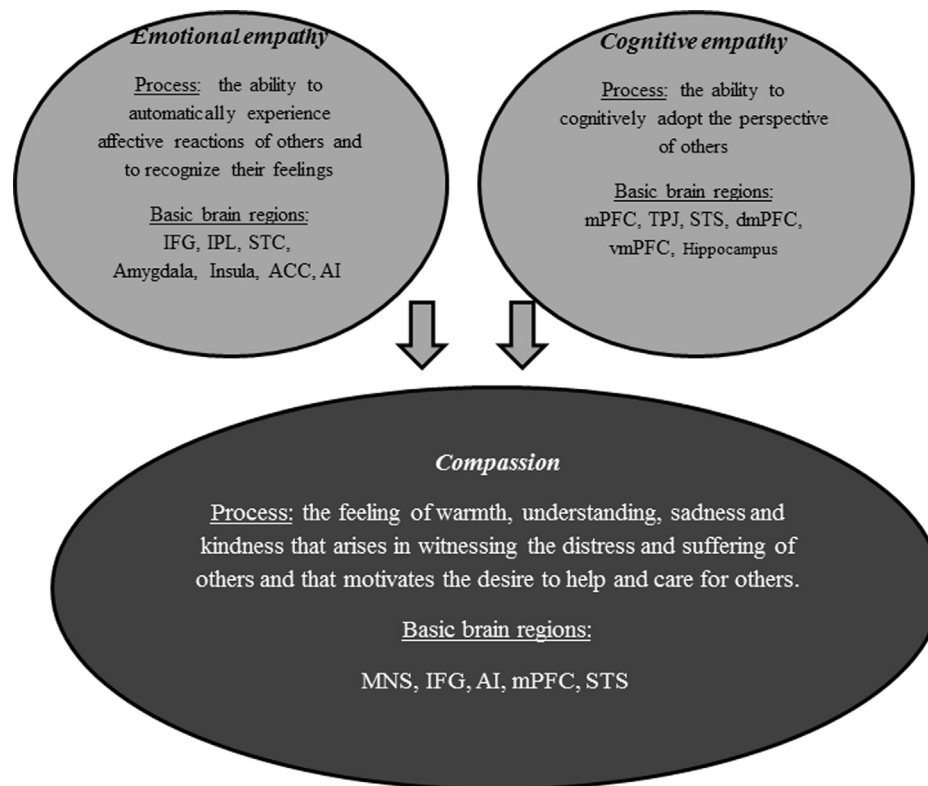


Fig. 1. Compassion integrates *emotional empathy* and *cognitive empathy* with pro-social motivation to help others. ACC—anterior cingulate, AI—anterior insula, dmPFC—dorsal-medial prefrontal cortex, IFG—inferior frontal gyrus, IPL—inferior parietal lobule, PFC—medial prefrontal cortex, STC—superior temporal cortex, STS—superior temporal sulcus, TPJ—temporal parietal junction, vmPFC—ventromedial prefrontal cortex.

Thus, compassion—the pro-social motivation to help others who are in distress, is an outcome of cognitive and emotional of empathy (Palgi et al., 2015) (see Fig. 1). In line with this theoretical framework, Zaki and Ochsner (2012) recently proposed, a model of empathy, which includes three components: (a) affective empathy and experience sharing, (b) cognitive empathy and mentalization ability, (c) empathic motivation and empathic concern. The third component includes the pro-social motivation to help others, e.g., compassion response, as a result of using one or both components of empathy (affective and cognitive). Thus, compassion appears to be based on both components of empathy: based on empathy that helps one to identify and understand the distress of other compassion encourages improving the other's wellbeing and motivates pro-social behavior (Singer and Klimecki, 2014). Accordingly, recent studies have found that compassion is mediated by brain areas associated both with emotional empathy [i.e., inferior frontal gyrus (IFG), anterior insula (AI) and dorsal anterior cingulate (ACC)] and with cognitive empathy [i.e., superior temporal sulcus (STS), medial prefrontal cortex (mPFC)] (Goetz et al., 2010).

A growing number of studies have recently shown that patients with PTSD exhibit impairments in pro-social behaviors (Charuvastra and Cloitre, 2008), including emotional empathy and cognitive empathy (also known as *Theory of Mind—ToM*) (see Plana et al., 2013; for review)—abilities that underlie the ability to respond compassionately. Moreover, accumulating neuroimaging studies have shown altered activity in the amygdala, mPFC and ACC, as well as in the hippocampus and insular cortex in patients with PTSD (Pitman et al., 2012; Zoladz and Diamond, 2013), all areas that moderate empathy (Shamay-Tsoory, 2011) and compassion (Goetz et al., 2010). For example, neuroimaging studies found that patients with PTSD have hyper-activation in the amygdala during the presentation of fearful stimuli (e.g., Rauch et al., 1996; Shin et al., 2005), a core region in the emotional empathy. Moreover,

other studies found that patients with PTSD have small volume (e.g., Rauch et al., 2003) and hypo-activation (e.g., Shin et al., 2004) of the mPFC, as well as small volume (e.g., Rauch et al., 2003; Schuff et al., 2008) and hypo-activation (e.g., Semple et al., 2000; Milad and Quirk, 2012) of the ACC, both core regions in the empathy network (for review see: Pitman et al., 2012; Zoladz and Diamond, 2013). Yet to the best of our knowledge, the impairments in compassion among these patients have never been assessed. Therefore, the rationale behind this study was to take a further step in understanding empathy deficits in PTSD by examining levels of compassion in these patients. Furthermore, since symptoms of emotional numbing are significantly associated with relationship difficulties among patients with PTSD (e.g., Cook et al., 2004; Kuhn et al., 2003; Samper et al., 2004), we hypothesized that the intensity of the symptoms would predict the intensity of the compassion deficit.

The neurochemical underpinnings of compassion are still largely unknown. Since compassion is a social emotion, it is reasonable to assume that neuropeptides such as oxytocin (OT), which has been found to mediate complex pro-social, affective and caring behaviors, should play a key role in mediating compassion. OT is a nine amino-acid cyclic neuropeptide produced in the brain that functions both as a neurotransmitter and as a hormone. During the past decade, ample evidence has shown that the OT system serves as a key mediator of complex social behaviors, including empathy (Kanat et al., 2013). The original speculation was that intranasal administration of OT would have general positive effects on social behaviors (Heinrichs et al., 2009). Nevertheless, recently it has been suggested that the effects of OT are more contextual and are affected by inter-individual factors, including gender and personal traits. Moreover, recent studies have shown gender differences in behavioral and neural responses to intranasal OT (Domes et al., 2010; Rilling et al., 2014). One leading hypothesis regarding these diverse effects of OT is the *social salience hypothesis* (Bartz

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