

Bonding after trauma: on the role of social support and the oxytocin system in traumatic stress

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This paper outlines the state of affairs in psychobiological research on psychotrauma and PTSD with a focus on the role of the oxytocin system in traumatic stress. With a high prevalence of trauma and PTSD in the Netherlands, new preventive and therapeutic interventions are needed. The focus is on the role of social support and *bonding* in coming to grips with psychological trauma, about the oxytocin system as a basis for reducing the stress response and creating a feeling of *bonding*, about *binding* words to painful emotions in psychotherapy, and about the *bonds* between researchers and clinicians.

Keywords: *trauma; PTSD; oxytocin; social support; bonding; psychobiology*

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In 2006¹, I visited Belfast to attend a meeting of the ESTSS, the European Society for Traumatic Stress Studies. As you well know, Northern Ireland was burdened for many years by what was known as “the troubles”, an awfully understated term for years of exposure to traumatic stress by many. What has stayed with me most from that visit are the many murals in the city: gigantic, stabbing images that reflect the menace and suffering generated by many years of violence. I was deeply affected by one image in particular. It was a mural depicting a rifle pointed directly at you. From whatever angle you looked at it, you would always look straight into the barrel of the gun.

Later, after listening to the stories patients told me, I came to understand that this is exactly how it felt to them: there was no escape. These patients recounted how, despite their efforts to suppress the danger, the memory was always there and would surface in all its intensity at the most inconvenient of times. While you may need a photo album to evoke the memories of vacations past,

a traumatic memory is indelibly etched in the mind. The memory remains so vivid that it seems as if the trauma takes place anew each day, whether it is in the broad light of day or in the quiet darkness of the night. Nightmares are disturbing your sleep and you wake up to find yourself drenched in sweat. You are no longer able to concentrate during the day. Constantly watchful for any sign that fate will strike again, you are irritable and short-tempered. Everyday life is completely disrupted. Social contacts disintegrate, emotions flatten, a sense of alienation from others sets in, and all faith in mankind seems to be lost. All this while feeling connected to others is of such vital importance to reducing the psychobiological stress response after trauma—as we will see below.

Prevalence of trauma and PTSD

Trauma can happen to each and every one of us: young or old, male or female, black or white. Until 2007, we actually had no idea how many Dutch people had ever faced a traumatic event, involving actual or threatened death, serious injury, or threat to physical integrity. Extensive research by our department has now shown that 80% of all Dutch people encounter a traumatic event in their lifetime (De Vries & Olf, 2009,

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also high functioning politicians, Nijdam, Gersons, & Olf, 2010). That is, four out of every five of us. Many Dutch people have completely unexpectedly lost a family member, been in a serious car accident, lived through a major fire or some other kind of calamity—such as a natural disaster, a shipwreck or an airplane crash—or have been exposed to work-related trauma (members of the police force, the fire department or the ambulance services, for example). Others have experienced violence of a more interpersonal nature, such as armed robbery, rape, domestic violence, sexual and physical abuse in childhood, a terrorist attack, or torture and other war-related traumas (De Vries & Olf, 2009).

In approximately 10% of these cases, that kind of shocking event results in a posttraumatic stress disorder, a PTSD (De Vries & Olf, 2009), characterized by re-experiencing the traumatic event, avoidance of anything that is reminiscent of the trauma, emotional numbness, and hyperarousal symptoms including having a difficult time falling or staying asleep. In addition, in nearly the same percentage of cases, trauma leads to depression, panic disorder, fatigue syndromes, or addiction—disorders that are also regularly comorbid to PTSD.

In our well-organized country, inhabited by people who are among the happiest in the world, 900,000 adults—aged 18–80—have at some point had to contend with PTSD; women twice as often as men. As such, PTSD ranks fourth among the most common psychological disorders and with similar rates of PTSD as in e.g., the United States (Breslau, Davis, Andreski, & Peterson, 1991; Kessler, Sonnega, Bromet, & Hughes, 1995).

While some believe that trauma and PTSD are American inventions designed to put a label on the flashbacks and the aggressive behavior by veterans of the Vietnam War, and although PTSD was only recognized as a psychological disorder in 1980, PTSD has been around since time immemorial of course. Greek tragedies already tell of it, Sigmund Freud wrote extensively about it, and Vincent van Gogh already spoke of it in one of his letters (see Jansen, Luijten, & Bakker, 2009a): “*It is and will remain a wound which I live above but which is there deep down and cannot heal – years from now it will be what it was the first day.*” (beautifully describing how traumatic memories do not fade away over time as do normal memories).

Not all of these 900,000 persons with PTSD end up in the mental health care system. For some people, the PTSD wears off spontaneously after a couple of weeks, as most psychological disorders do (e.g., Meewisse, Olf, Kleber, Kitchiner, & Gersons, 2011). Still, that leaves a large segment of PTSD patients who could benefit greatly from effective treatment.

What causes PTSD, how does it work and what can we do about it?

Now that I have been appointed professor of “*Neurobiological mechanisms of prevention and treatment in trauma and Posttraumatic stress disorders (PTSD)*” I will be able to fully explore the psychological and biological mechanisms that underlie the traumatic experience and the PTSD, as well as its prevention and its treatment.

In this paper entitled *Bonding after Trauma* I will guide you along the path leading from trauma to PTSD, describe the psychobiological research on psychological trauma and sketch out the research agenda for the years to come. I will discuss:

- (1) What exactly is psychological trauma and what resources do we have for *binding* the wounds it inflicts?
- (2) The importance of social support and interpersonal *bonding* in coming to grips with psychological trauma,
- (3) The psychobiological changes caused by PTSD, in particular the role that the oxytocin system plays in subduing the stress response and creating a feeling of *bonding* and togetherness,
- (4) Finding words that *bind* them to their past and to the anguished emotions that are addressed in therapy,
- (5) Researchers *bonding* with clinicians,
- (6) And finally about the ties that *bind* the Academic Medical Center (AMC) and Arq Psychotrauma Expert Group in the areas of treatment and research.

The AMC Psychiatry has a long history in trauma and PTSD with a strong focus on research. The Arq Foundation supports and *binds* together organizations that specialize in psychological trauma, such as Centrum ‘45, the Institute for Psychological Trauma, Psychotrauma, Impact, and others. Arq is not an abbreviation. Arq stands for something, for connection (Arq meaning Arch in old Latin).

Stress model

Why is it that one person develops a psychiatric disorder such as PTSD while someone else does not?

Ten years ago, when I started my research on posttraumatic stress, I made an attempt to create a heuristic model that would capture the various factors that contribute to developing or maintaining a PTSD. The model describes how experiencing a trauma may ultimately lead to a psychological disorder (Olf, Langeland, Gersons, 2005) and what factors (of the event itself, the appraisal and coping styles, the psychological and biological responses to it mediate and moderate this path. Within this pathway there are gender differences in all these factors which help explain the higher prevalence

rates of PTSD in women (see Olff, de Vries, Güzelcan, Assies, & Gersons, 2007).

Trauma

By definition it starts with the trauma. Literally, “trauma” is Greek for “wound”. However, in case of a psychological wound a simple band-aid far from suffices.

As we will see, the bindings that are needed to tend to trauma consist of psychosocial care much more than physical treatment. Incidentally, physical injury *does* often occur as part of the trauma (Haagsma et al., 2012). I have in mind particularly the accident victims who are referred to us by the emergency and traumatology units, or the intensive care, with whom we cooperate closely. Our colleagues in those units know that physical and psychological recovery go hand in hand.

The word trauma can often be heard in daily life. Rather arbitrarily, it is used to describe a range of very different events. Everything is labeled a trauma nowadays. However, a lost World Championship in soccer, or a fumbled kiss by Ali B (the first Moroccan-Dutch star in Dutch pop culture), is *not* a trauma.

Even animals can be traumatized, no matter how thick their skin is—remember how Astro, the mouse that was taped to a fireworks rocket by two misguided miscreants, ultimately succumbed to the stress.

A trauma, or strictly speaking: a potentially traumatic event, is a shocking event that one is exposed to through personal experience or through the witnessing of such an event. It is an event that is generally life-threatening, or at least has the potential to physically or psychologically damage people to a severe extent. The subjective response to this event, i.e., the appraisal of it as e.g., as threat vs challenge, activates the stress response and may so ultimately lead to PTSD (Olff et al., 2005).

The stress response

What happens in the human body immediately after the occurrence of a stress factor or trauma? Every stress factor, regardless of whether it is a negative or positive one activates the stress system. Stress is not necessarily bad for a person. The acute stress reaction is in fact a healthy, natural response, essential to human survival. The heightened heart and respiratory rates are needed to jump out of the way in time when a car is coming straight at you. The stress factor activates important nuclei in the brain, such as the paraventricular nucleus (PVN), in the hypothalamus) and with them the amygdala: the almond-shaped neural structure known as the alarm center of the body. Stress hormones are released and the peripheral nervous system becomes active. The body is geared up to engage in primal responses to danger: *fight or flight*, or sometimes *freeze*, seem to be the preferred options to survive (Olff et al., 2005).

Based on the theory of evolution, it is easy to understand why a “danger memory” is stored carefully and can be retrieved quickly under the influence of stress hormones. At one time, this would have increased our chance of survival. Now, however, this mechanism can sometimes seriously get in our way. One aspect of PTSD is exactly the inability to allow the trauma to fade to a normal memory.

Once the danger is over, another part of the stress system, in which the hormone oxytocin plays one of the major roles, sees to it that the system returns to its normal state (Amico, Mantella, Vollmer, & Li, 2004; Gulpinar & Yegen, 2004). The heart rate returns to normal and the level of cortisol decreases. After a period of alertness it is now safe to lean back again. The social context is of great importance in this respect since the biological system that produces feelings of calm and attachment can only be activated in socially favorable circumstances.

So far, stress research has mainly focused on the systems that underlie the *fight or flight* response. The oxytocin system, however, is considered at least as important to the stress response and the ability to recover from a stress factor (e.g., Kubzansky et al., 2012). In coordination with other chemical mediators (like vasopressin and dopamine) it is what enables us to enjoy life, to open ourselves up to others, to fall in love, to become attached to others, and to find repose and respite.

For years, oxytocin was mostly known as a hormone that facilitates breastfeeding and induces labor contractions. Women take a pinch to stimulate lactation. I also know from personal experience that administering oxytocin to cattle has been commonplace for decades in farming. One injection into a cow’s tail and after only a short time the milk comes squirting out of the udder. By now, the oxytocin system is considered to be essential to social contact: it provides a greater confidence in others as well as a feeling of bonding, hence the name *love hormone*.

Most neurobiological stress research is still limited to animal research. If research on humans is done at all, it often only involves one kind of humans, namely men. After all, women are much more complicated due to the hormonal changes that are part of their menstrual cycle. They may, however, also be more interesting—especially with regard to the oxytocin response.

The stress response on the rampage

A dysfunctional state after trauma occurs as a result of insufficient recovery from the stress response after the actual danger has long been over. The alarm bell in the brain continues to sound and the stress reaction is not suppressed. The working memory stays “charged” with sensory memories of the traumatic event: images, sounds, smells.

In PTSD, fear extinction is diminished. Normally, fear gradually disappears after repeated exposure to a variety of cues that are reminiscent of the stress factor. In case of PTSD, however, a sensitization takes place instead: the stress system becomes hypersensitive and will fire at full blast at the slightest trigger.

Neuroimaging studies have consistently shown that three major structures, i.e., the amygdala, hippocampus (the “seahorse”) and medial prefrontal cortex (mPFC), in the front of the brain, are involved in the pathophysiology of PTSD (Rauch, Shin, & Phelps, 2006). The amygdala is found to be very sensitive in people with PTSD (e.g., Rauch et al., 2000; Shin & Liberzon, 2010). In addition, we, and others, showed that patients with PTSD have smaller hippocampal volumes, important for learning and memory (Lindauer et al., 2004a). We have also established that the prefrontal areas function insufficiently (Lindauer et al., 2004b; Quidé et al., 2012; Vermetten & Bremner, 2002). In case of a PTSD, the hippocampus and the medial prefrontal cortex appear inadequate to reduce the fear response and restrain the PVN and amygdala’s activity, crucial to the phasing out of fear (Quirk & Mueller, 2008) and the regulation of emotions (Ochsner, Bunge, Gross, & Gabrieli, 2002; Phan et al., 2005). In other words, the *connectivity, or bonding*, between these parts of the brain is not optimal.

We also find a more sensitive hypothalamus-pituitary—adrenal gland-axis (HPA-axis) in people with PTSD, resulting in a dysregulated cortisol pattern, among other things. This disrupts the central and hormonal regulation of the stress response and the immune system—which in turn increases susceptibility to certain diseases.

While the level of cortisol usually rises in response to a stress factor, we encounter lower basal cortisol levels in case of a PTSD (e.g., Olf et al. 2007; Olf, Güzelcan, de Vries, Assies, & Gersons, 2006). This is the effect of an overly sensitive feedback mechanism (Yehuda, 2001; Yehuda, Bierer, Pratchett, & Malowney, 2010). Such changes are not consistently found (Meewisse, Reitsma, de Vries, Gersons, & Olf, 2007). They are found especially in women and when the trauma consists of sexual abuse.

Consequently, we can establish that in case of PTSD an external stress factor causes measurable biological changes. People with PTSD are more likely to get sick, suffer from cardiovascular diseases and cancer more often, and on average die at an earlier age (e.g., Boscarino, 2008; Boscarino & Figley, 2009; De Vries, Lok, Assies, & Olf, in prep). At the same time, we witness a great strength and creativity in people that have suffered psychological injury, or as Van Gogh put it: *“The more I become dissipated, ill, a broken pitcher, the more I too become a creative artist in that great revival of art of which we’re speaking”* (Jansen et al., 2009b)

Risk factors

On the path from trauma to psychopathology, who develops PTSD after trauma and who does not? Most people will be thinking: not me, that doesn’t happen to me. This is reflecting a healthy illusion of invulnerability. We will single out three important risk factors, namely: gender, genetics and social support.

Differences between men and women

Gender is a risk factor for developing a PTSD. Women are two to three times more likely than men to develop a PTSD after trauma (De Vries & Olf, 2009; Kessler et al., 1995). More generally, anxiety disorders are more common in women, while other psychological suffering (such as alcohol and drug addictions) is found more often in men (De Graaf, Bijl, Smit, Vollebergh, & Spijker, 2002).

The gender difference can not be explained by the number of traumatic experiences that men and women encounter. There is, however, a difference in the nature of the experienced trauma that carries in it a differentiation in conditional prevalence. Being subjected to interpersonal violence such as physical or sexual abuse, particularly at a young age, one has a greater chance of developing psychological problems than when one lives through a disaster or an accident. Such experiences are often events that have a direct impact on the attachment system (Nicolai, 2009) and undermine one’s trust in other people. If this occurs at a young age, it also disrupts the maturation of the biological stress system. The cortisol pattern becomes dysregulated and the oxytocin system develops in a way that is inadequate to deal with later hardships (Heim et al., 2008). In addition, women suffering from interpersonal trauma are less likely to engage in behavior that could stimulate their oxytocin system (such as resting, being massaged, sexual activity) precisely because these activities are associated with stress and anxiety (Nicolai, 2009).

Genetics

Could a greater risk of PTSD be a genetic predisposition? Based on the study of twins, heredity is estimated to make up approximately 30% of this risk (e.g., True et al., 1993). We now know that various polymorphisms—small differences in genes between people—and epigenetic processes are involved in the biological vulnerability to PTSD (e.g., Broekman, Olf, & Boer, 2007). Small but significant variants in genes constitute risk factors for PTSD symptoms (e.g., Goenjian et al., 2012). But that’s not the whole story. What is important is the interaction between genetic and environmental factors (Mehta & Binder, 2012). Our genes only then lead to an increased risk of PTSD when there are environmental risk factors in addition to the trauma, such as a lack of social support (e.g., Kilpatrick et al., 2007).

Social support

Social support proves to be an essential concept in relation to PTSD. A lack of social support and recognition by the environment is one of the most consistent risk factors for PTSD. Moreover, if the disorder *does* occur, the patient will recover faster through proper social bonding.

We have heard it countless times: patients telling us that the trauma was bad, but the response to it by their environment made it even worse—not being taken seriously by the official who is filling in the crime report with regard to a rape, the sense of loneliness and detachment when there is no-one left who will listen or who understands what you're going through, the trivial way in which your boss responds to the incident at work.

At the same time, I note from internal AMC referrals the positive effect of support by departmental managers who are actually showing great care for their employees. Also very important is the recent recognition of PTSD as an occupational disease for police officers by our minister of Security and Justice.

There are gender differences when it comes to social bonding as well. A lack of social support turns out to be more strongly related to the development of PTSD in women than in men (e.g., Ahern et al., 2004; Andrews, Brewin, & Rose, 2003; Weismann et al., 2005). Coping styles that are based on social orientation in particular can also be different for men and women in stressful situations. By nature, from prehistoric times, women are better equipped to give a so-called “Tend & Befriend” response (Taylor et al., 2000). Tending to the children and taking part in a social network was then beneficial to our species. To forgo this may hit women hard.

Early interventions

All this would seem to suggest that the solution lies in a well-organized provision of social support, immediate and proper care to all, having the victim express her or his emotions, sharing the suffering and talking the event through.

If only it were that simple. Our own research has revealed that early intervention for everyone, the so-called *debriefing*, a term that has its origins in the army, where emotions are expressed and information on potential symptoms is given, was not at all effective (Sijbrandij, Olf, Reitsma, Carlier, & Gersons, et al., 2006). Indeed, the very people who seemed to need it the most were better off with the control condition; the group who had received no intervention whatsoever (Sijbrandij et al., 2006). Just offering this type of immediate psychological assistance to everyone is useless then and can even be counterproductive at times. In addition, it does not do justice to the fact that posttraumatic stress symptoms after traumatic events naturally wear off in most people. What does seem to be very effective is a

number of cognitive behavioral therapy (CBT) sessions over a period of several weeks after a traumatic experience for people who are clearly exhibiting early PTSD symptoms (Roberts, Kitchiner, Kenardy, & Bisson, 2009; Sijbrandij et al., 2007).

So we were wrong. The support that was offered was certainly appreciated, people liked it, but in the end it did nothing to prevent PTSD. Even though new types of interventions are being studied (Mouthaan, Sijbrandij, Reitsma, Gersons, & Olf, 2011), this means we are still lacking an effective preventive intervention. Therefore, we must seek new ways to encourage the social support system.

We already know that we can positively affect health through social behavior. For example, increased social bonding is linked to a decrease in the level of stress hormones, a lower heart rate, lower blood pressure, lower cholesterol levels, improved immune reactions (e.g., Sherman, Kim, & Taylor, 2009). Also, people who are receiving a lot of social support not only feel more content, but also live longer (Croezen et al., 2010; Holt-Lunstad, Smith, & Layton, 2010).

The world of science is a world that is subject to fashion. There was a time when everything was considered from a psychological angle. Next came the era of neuroscience, in which all revolved around the brain. Right now, the focus is gradually shifting again towards cognitive neuroscience—the University of Amsterdam's Cognitive Science Center Amsterdam (CSCA) being a case in point. Simultaneously, however, there is a clear upsurge in the field of social neuroscience. It is fashionable again to examine the link between the brain and behavior; social behavior in particular.

Oxytocin

Oxytocin plays a leading role—interacting with a complex of other neuropeptides and hormones—in social neuroscience. Oxytocin is released during every imaginable form of pleasant social contact. A pleasant conversation, making love, massaging, cuddling, petting the dog, tweeting—all of it can stimulate the production of oxytocin. The satisfied feeling of a full stomach or following good sex is a good example of the effect of oxytocin interacting with systems that regulate feelings of reward and pleasure. On the Internet, this was definitely picked up on.

Bonding in voles

The basis of oxytocin research rests on the examination of another animal: the vole. Two species of voles that look very similar to each other have gained the most attention: the prairie vole and the montane vole. Genetically, they are nearly identical, but in terms of their social behavior they are as different as night and day. The socially monogamous prairie voles stay with the same

partner throughout their lives and care for their young together. The montane vole, however, does not enter into such close bonds. The pair-bonding prairie voles and the promiscuous montane voles form a perfect natural experiment to determine the neural substrate underlying bonding. Sue Carter, who is in Chicago and with whom we now have close ties as well, demonstrated that the two types differ as much in their oxytocin and vasopressin systems as they do in social behavior. Much of the variation in the “social exclusivity” could be attributed to species variation in the density of oxytocin’s receptors in brain regions that mediate reward and recognition (Carter, DeVries, & Getz, 1995; Yee, 2012).

Initially, the bond between a baby and a mother was believed to only be owing to the fact that babies were just plain hungry and that the bond between mother and child was consolidated through breast milk. Even Freud, the psychoanalyst, and B.F. Skinner, the behaviorist—avowed enemies—agreed on this (Konner, 2004). This turned out to be incorrect. Classic experiments by Harlow using infant monkeys that had been separated from their real mothers showed that the little monkeys did not prefer the metal surrogate mothers that were offering delicious milk, but favored the soft mock-up mother that allowed them to experience a kind of cuddly contact.

The softer terry-cloth mother more effectively leant itself to being utilized for the regulation of the infant’s emotional state since so many mammals use physical touch as an analog of security/safety. The lack of maternal security and touch likely disrupted the oxytocin system as more recent research has shown that early maternal separation and other early disruptions to social stability are capable of dysregulating the oxytocin system in animals (Bales, Boone, Epperson, Hoffman, & Carter, 2011), but also in, for instance, maltreated orphans (Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005).

We now know that administering oxytocin to monkeys leads to more social behavior (Chang, Barter, Becket Ebitza, Watson, & Platt, 2012).

Oxytocin and PTSD

Would it be possible to stimulate the oxytocin system in people with PTSD as well? Endogenous, through the provision of social bonding? Or exogenous, via a nasal spray? Could we return a sense of safety and confidence to people that way, to replace the fear, avoidance and hyperarousal that are so characteristic of PTSD?

We now know that administering oxytocin to healthy volunteers affects the stress response (e.g., Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Kirsch et al., 2005). Healthy test subjects react less fearful in response to scary images after having been given a sniff of oxytocin. That could be determined by monitoring the activity in the amygdala, that alarm center in the brain,

which was a lot lower than that of test subjects who had been given a placebo. The prefrontal cortex seems to be functioning better and also the HPA-axis reacts to oxytocin (Heinrichs et al., 2003). And psychophysiological people show improved recovery (Kubzansky et al., 2012). Therefore, we expect that oxytocin may be able to offer some relief to people with a PTSD and a corresponding overly active alarm center and stress reactivity.

It has already been established that oxytocin reduces the psychophysiological stress response in veterans with PTSD, and the first results of research directed specifically at PTSD patients show that a sniff of oxytocin seems to at least cause the acute PTSD symptoms to subside (Pitman, Orr, & Lasko, 1993; Yatzkar & Klein, 2009).

In addition, oxytocin may also have a positive effect on the social interaction and reduce emotional numbing that is experienced by PTSD patients (see Fig. 1). Oxytocin stimulates the nucleus accumbens, the part of the brain part that has to do with “social rewards” (Sailer et al., 2008). This way, we hope that people with a PTSD will be able to benefit more from social contacts and support again, will seek out such interaction once more, and as a result will hopefully break the vicious circle. It is possible that the feeling of safety during a therapy session as well as the sense of trust in the therapist will also be augmented, making it easier to recover traumatic memories.

Cautionary note

However, oxytocin is no panacea. The system is incredibly complicated and subtle. Oxytocin exerts its effects only in specific circumstances, interacting with a large number of other systems (Bao et al., 2005, 2006). The social context is a significant factor in this, as our Amsterdam colleagues have already demonstrated (De Dreu et al., 2010) and studies by foreign colleagues on Borderline patients, for example, have also shown (Bartz et al., 2011).

Rather than uniformly increasing prosocial behavior and positive feelings, oxytocin may well increase sensitivity to and willingness to engage with the social context by attenuating a sense of threat (Kubzansky et al., 2012; Shamay-Tsoory et al., 2009).

Also, although oxytocin seems to facilitate a positive psychobiological stress response for both men and women, some studies indicate different responses in women (Domes et al., 2010; Kubzansky et al., 2012; Lischke et al., 2012).

Interventions

I believe that, despite these limitations, oxytocin can indeed play an important role in the treatment of PTSD (Olf, Langeland et al., 2010). In essence, every PTSD treatment focuses on the disrupted anxiety system and on restoring the patients trust in the world.

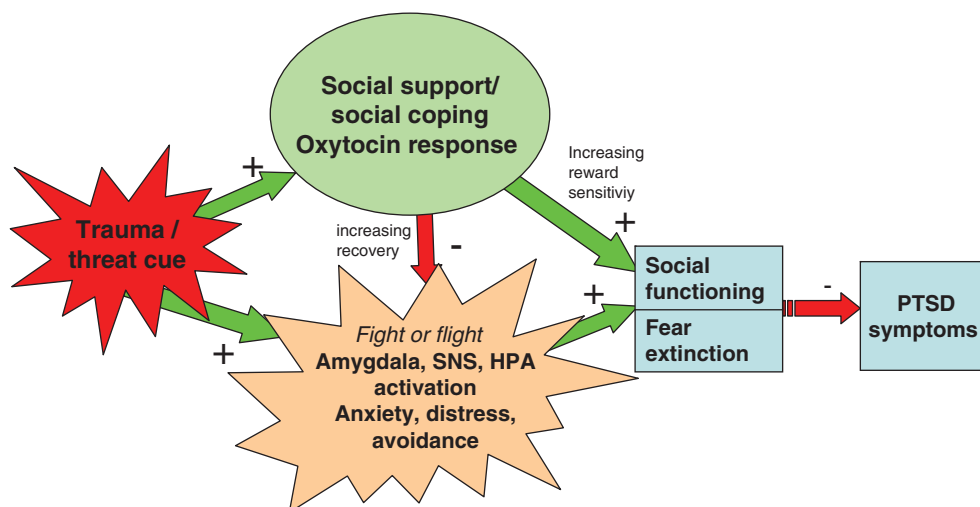


Fig. 1. The oxytocin system for increasing fear extinction and social functioning after trauma.

However, many patients are not responding optimally yet; some do not even respond to it at all (Bradley, Greene, Russ, Dutra, & Westen, 2005; Cloitre, 2009). Furthermore, no adequate preventive intervention has yet been devised to preclude PTSD symptoms after trauma.

PTSD symptoms can be treated through medication. The SSRI (Selective Serotonin Re-uptake Inhibitors) in particular have been found to be effective and will certainly be indicated in cases of severe comorbidity. Oxytocin interacts with many systems that are affected by the SSRI.

As far as psychotherapy is concerned, there are a great many approaches to trauma: they involve re-exposing the patient to the trauma in some way and—however difficult that may be—teaching the patient to attach words to their painful emotions and overwhelming sensory experiences, *binding* them to their personal history (Gersons & Olf, 2005). This can be done in several ways: through Cognitive Behavioral Therapy (CBT), Exposure treatment, EMDR (Eye Movement and Desensitization Reprocessing), Narrative Exposure therapy, much used at Centrum '45, and Brief Eclectic Psychotherapy (BEP), as developed at the Academic Medical Center by Berthold Gersons. On average, two-thirds of the patient group recovers following these forms of treatment (Bradley et al., 2005).

In our own trial, in which we compared BEP and EMDR, we also determined both to be effective, whereby it should be mentioned that EMDR possibly got faster results (Nijdam, Gersons, Reitsma, de Jongh, & Olf, 2012). Especially interesting, however, are the effectiveness predictors. How can we target the treatment more accurately? Who has the most benefit from what treatment? We now have a number of careful pointers for biomarkers that could indicate successful treatment, in the area of the cortisol response and neuropsychological indicators (Polak, Witteveen, Reitsma, & Olf, 2012;

van Zuiden et al., 2011). But we want to further increase the chances of success.

We still do not know how medication relates to psychotherapy (Witteveen et al., in prep). What happens in the brain when we are able to attach words to traumatic images remains immensely intriguing. How to strengthen the connections in the brain again that make sure that the prefrontal cortex inhibits the amygdala's activity is a question that will require a lot more research. Interestingly, pharmacotherapy and psychotherapy each seem to have their own “normalizing” influence on (functional) brain abnormalities in patients with PTSD (Quidé, Witteveen, El-Hage, Veltman, & Olf, 2012), top down vs. bottom up. These results call for further research into the effects of successful treatment aimed at normalizing the stress response, the *bonding* (connectivity) between different parts of the brain, and the role of oxytocin in this process.

Future research

In our new studies we will examine different aspects of the stress system, the brain with fMRI and EEG, also during sleep, the HPA-axis, genetics and epigenetics, receptor sensitivities, and oxytocin of course. We will also be using a new technique to determine the level of cortisol in a strand of hair. This provides marvelous opportunities to establish cortisol levels preceding the trauma as well. Per centimeter of hair, we can travel 1 month back in time—and so hopefully gain more insight into pre-trauma vulnerabilities versus post-trauma changes. Incidentally, measuring cortisol levels is no easy task. The Dutch working group Trauma & Neurobiology and the ESTSS task force Neurobiology have been working for years on developing the gold standard for this.

It will be no surprise to you that our aim will be to stimulate the oxytocin system, both in patients who are

taken to the hospital immediately after a traumatic experience and in patients who come to us with a pre-existing PTSD. In doing so, we will carefully map the stress reaction and brain activity. For this, we have received a considerable TOP grant from The Netherlands Organization for Health Research and Development (ZonMw), intended to support innovative research. Our research on oxytocin will have a wider scope than PTSD alone. I am therefore looking forward to cooperating with the colleagues from the Psychiatry Department on depression and psychotic disorders.

And within Arq we will investigate whether administering a low dose of oxytocin may improve the psychotherapy of chronic and complex PTSD.

In general, future research will need to continue the search for the psychobiological and neurobiological mechanisms that underlie PTSD. The goal in this respect is to make screening and diagnostics more efficient, in order to be able to better target treatments to specific cases. And, importantly, we need to improve preventive interventions as well as treatment of trauma related disorders, paying special attention to psychobiology. Since relatively little studies have addressed gender difference in neurobiology of PTSD or in treatment, future studies should take gender differences into account and put sufficient statistical power in the clinical trials to be able to determine whether men and women benefit to the same extent from intervention.

Research collaboration and clinical practice

The focus of this paper has been on scientific research. Internationally, the Netherlands scores high marks for the quality of PTSD care and research. It may be no coincidence that Dutch researchers in the field of psychological trauma are present in force, and particularly productive, at international conferences.

All these research projects have in common that they were conducted in collaboration with other, specialist departments within the Academic Medical Center or within Arq, nationally and internationally (e.g., Bisson et al., 2010; Eriksson et al., 2012; Martin-Pena, Rodríguez-Carballeira, Escartín, Porrúa, & Olf, 2011). I believe that the success of this research makes a powerful case for further cooperation. Steps in this direction are made in several new projects; a development that could hopefully lead to an even stronger international psychological trauma research field.

As chairperson of the European Society for Traumatic Stress Studies (ESTSS) I experienced firsthand how nurturing and valuable international connections are. As Editor-in-Chief of the *European Journal of Psychotraumatology (EJPT)* I am also very aware of the advantage of a good international network in attracting top authors and finding the right reviewers. Despite the fact that it has only been in existence for little over a year,

the journal has already been included in PubMed, one of the major databases for scientific articles in the field of health care (Olf, 2010; Olf & Bindselev, 2011).

Still, the practical application of our work remains important and I will therefore do my utmost to see that our research findings can be implemented in practical care. I am pleased that Arq, traditionally based on practice, is presently working so hard to link science to clinical practice. My chair is a part of this plan.

“Bonding after trauma” is the title I gave this paper. First and foremost, I wanted to outline the state of affairs in psychobiological research on psychological trauma, with a focus on the role of the oxytocin system in traumatic stress, and offer a research agenda for the years to come. With 80% of persons in the Netherlands experiencing trauma, and 10% of those developing PTSD, we need to pay attention to “*binding*” the psychological wounds inflicted by trauma. I have discussed the essential role of social support and *bonding* in coming to grips with psychological trauma, and the oxytocin system as a basis for reducing the stress response and creating a feeling of *bonding*. This type of research may hopefully lead to new preventive and better therapeutic interventions and create more insight into the mechanisms behind the *binding* of words to painful emotions in therapy. However, without the practical application of our studies, patients will not benefit from the research findings. Therefore, we need to strengthen the *bonds* between researchers and clinicians. I am looking forward to further *binding* and *bonding* between the Academic Medical Center with its strong research tradition and Arq with its large clinical and practice settings but with interest in innovation and providing evidence based care. This type of collaboration may inspire other centers in the Netherlands and abroad as it offers the best of opportunities for innovative patient research and improving clinical practice.

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There is no conflict of interest in the present study for any of the authors.

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