



Schema therapy for Dissociative Identity Disorder (DID): further explanation about the rationale and study protocol

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We respond to the letter to the editor authored by Brand and colleagues titled ‘Cautions and Concerns about Huntjens et al.’s Schema Therapy for Dissociative Identity Disorder’. We thank the authors for the comments raised and the associate editor for the opportunity to respond in order to explain our rationale and protocol in more detail.

Brand and colleagues argue that we misunderstand the staged model of DID treatment as trauma and its effects are always a focus of this treatment. In their view, a premature focus on trauma memories frequently causes DID patients to develop acute symptom exacerbations with increased suicidal and self-destructive behaviour. It was not our intention to argue that the staged approach to DID treatment does not always focus on trauma. However, in the first stage of this treatment, with its focus on maintaining personal safety, controlling symptoms, modulating affect, building stress tolerance, enhancing basic life functioning, and building or improving relational capacities (ISSTD, 2011), patients learn to deal with *the effects* of trauma, but this phase does not include active trauma memory treatment. Although we consider the use of stabilization techniques throughout the therapy process important for DID patients, we consider a separate stabilization phase as a prerequisite for phase two trauma-focused treatment as potentially harmful to patients for several reasons: 1) Usually, phase one treatment may take many years to complete in which patients are thus denied the access to effective trauma memory treatment (Groenendijk & van der Hart, 1995; Myrick, Webermann, Langeland, Putnam, & Brand, 2017); 2) Moreover, the majority of patients does not reach phase two; 3) Besides relieving posttraumatic complaints, trauma-focused treatment might also ameliorate the dissociative symptoms which are a reaction to the traumatic memories; thus trauma-focused treatment may in fact stabilize patients; 4) By keeping patients ‘fixed’ in a stabilization phase, they consistently receive the direct or indirect message from their

therapist that they are not ‘ready’ to deal with their traumata, inadvertently strengthening their avoidance behaviour instead of empowering the patient.

Most importantly, the question whether stabilization is a prerequisite for trauma-focused treatment is an empirical one, and cannot be answered by clinical expert consensus alone. Indeed, our study is a first attempt to challenge this view. Note that we might also fail to find evidence that skipping a stabilization phase is possible. Exploring this in a systematic way is important as data on this in DID samples is missing. At the moment, the most relevant empirical data on this matter come from studies of survivors of child sexual abuse, and studies that include PTSD dissociative subtype patients (i.e. clinically the PTSD dissociative subtype group may partly or completely overlap with the Other Specified Dissociative Disorder). A meta-analysis on studies including PTSD patients who were victims of childhood sexual abuse indicated more symptom improvement after trauma-focused treatment compared to non-trauma-focused treatment, such as stabilization-only treatment (Ehring et al., 2014). Moreover, a systematic review investigating the effect of prolonged exposure in PTSD on comorbid symptoms such as dissociative experiences, concluded that also these symptoms, both self-reported and clinician-rated, decreased alongside the PTSD symptoms (van Minnen, Harned, Zoellner, & Mills, 2012 also see Hagedaars, van Minnen, & Hoogduin, 2010). Furthermore, a recent study in a sample of patients with the PTSD dissociative subtype yielded large effect sizes for trauma-focused treatment without stabilization (Zoet, Wagenmans, van Minnen, & de Jongh, 2018). Taken together, so far there is no scientific evidence which indicates that a phase of stabilization is a *conditio sine qua non* for trauma memory confrontation in complex trauma-related disorders such as adult patients with a history of childhood sexual abuse, or that trauma memory treatment is less well tolerated and accepted (e.g. Neuner, 2008). That being said, we wish to emphasize that we do recognize that DID patients can be highly

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vulnerable and have low stress tolerance. Therefore, in the current study protocol we gradually introduce, step by step, from early on in therapy imagery rescripting as a trauma memory intervention, starting with neutral to mild experiences and gradually building up to more severe traumas. So, in comparison to, for example, the treatment of borderline personality disorder, the process is slower, using the same interventions, but in a more graded way. Moreover, we also developed adaptations to specifically deal with dissociative reactions and avoidance. Thus, treatment is tailored to the core symptoms of DID and it is well recognized that these patients are crisis prone, and are severely hindered by their dissociative features. Hence, there may be more similarities between our approaches than thought at first sight, especially as some therapists using a phase-based approach may flexibly use the different phases without unnecessarily delaying or giving access to phase two trauma treatment.

Secondly, Brand et al. state that we failed to mention a critical finding in the study of Jepsen, Langeland, Sexton, and Heird (2014). In this study inpatient trauma treatment was delivered in a group with and a group without complex dissociative disorders, resulting in a significant improvement in both groups (i.e. the symptom trajectories of both groups ran parallel). The authors point out that we did not pay attention to Jepsen et al.'s finding that general trauma treatment did not result in changes in *pathological* dissociation such as amnesia and identity fragmentation which are hallmarks of DID. However, we could not find any data in the article that substantiate this reasoning. Jepsen et al. used the DES-II, which is a general measure of dissociation, hence no statements can be made on specific dissociative features like amnesia or identity fragmentation. In order to grasp the reason why there was no clear improvement on the DES-II, Jepsen and colleagues *speculated* in their discussion about possible qualitative differences between the dissociative experiences that are reported by highly dissociative individuals compared to non-dissociative individuals, and wondered whether they are in need of another therapeutic approach. However, we could not find any data in the article referenced that directly substantiate this (ad hoc) interpretation.

Thirdly, the authors claim that our schema therapy approach should be viewed as a staged treatment and think that we offer 16 sessions of psychoeducation followed by trauma treatment. This is a misconception of our protocol. The naming of this condition as an 'education phase' may, on second thought, have contributed to this confusion. A better term would have been 'exploration phase'. This initial 8 week exploration condition was not meant nor devised as a phase one treatment. As mentioned in our paper, this condition was added purely for research purposes (i.e. to increase the power by including an additional within-subject

comparison condition in order to control for the effects of attention for the patients' problems). In these 8 weeks an idiosyncratic case-conceptualization is made, and the patient is educated on the schema mode model. Importantly, no active schema therapy strategies nor any skills training are being used, therefore no specific treatment effect is expected in this phase. This contrasts to a phase one treatment in which for example stabilization work or emotion regulation training is predicted to result in a decrease of symptoms. Only after this initial 8 week condition, the active treatment condition starts, and the therapist can use schema therapy strategies. Brand et al. have possibly misunderstood active schema therapy for active trauma memory treatment.

With regard to further details of our study protocol, we agree that sound diagnostic assessment is important, given issues related to differential diagnosis, but also the risk of a diagnosis resulting from socio-cognitive reinforcement including iatrogenesis induced by self-declared 'experts' of DID treatment. As was mentioned in our design paper, the follow-up assessment will be at 6 months after treatment. Indeed, we agree that the exclusion criteria will limit the generalizability of our findings, however, these criteria were chosen as a means to optimize the internal validity of the study (i.e. by excluding obvious confounders). We welcome the interest of the authors in the adaptations made in the schema therapy protocol for DID patients and we are planning to write, in case of successful treatment results, a detailed treatment protocol explaining all the relevant information needed for further training and dissemination of our approach.

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