Inhibition and Re-Appraisal within Emotional Disclosure: the embodying of narration

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Abstract:

The emotional disclosure paradigm (EDP) associates better health with repeated disclosure of emotional experiences. However, disclosure does not bring health benefits for all, and neither does the EDP adequately specify embodied mechanisms or neural pathways whereby benefits might be produced. This paper addresses these issues by offering more sophisticated notions of emotional inhibition and cognitive reappraisal. It then outlines aspects of the somatic marker hypothesis which supports a more comprehensive conceptualization of the processes that may enable both the positive and negative health effects of disclosure.

Introduction

It is argued that highly charged negative affect laden experiences can produce deleterious effects if these experiences are not “named, thought about and dealt with cognitively” (Pennebaker, Kiecolt-Glaser, & Glaser, 1988). This theoretical position is central to psychotherapy’s promotion of the “talking cure”. It is now widely accepted that talking about traumatic events are associated with positive health outcomes: both in various schools of psychotherapy and in studies conducted within the emotional disclosure paradigm (EDP). In EDP studies, participants talk or write about negative emotional experiences in controlled conditions on three to five occasions. Compared to control groups who address emotionally neutral experiences, it is suggested that experimental groups typically have significantly more positive health outcomes (Frisina et al., 2004; Pennebaker, 1997; Smyth, 1998).

A number of meta-analyses have been conducted to evaluate the efficacy of the EDP. Smyth (1998) looked at the effects of emotional disclosure upon healthy people. Thirteen studies were meta-analysed and it was suggested that emotional disclosure in these studies significantly improved health in four outcome types: reported physical health, psychological well-being, physiological functioning, and general functioning. Frinsina et al. (2004) looked at the effects of emotional disclosure upon people with physical or psychiatric disorders. Nine studies were meta-analysed and it was suggested that emotional disclosure significantly improved health. [p.320] However, it was pointed out that it is more effective on physical than on psychological health outcomes. Meads and Nouwen (2005) looked at the effects of emotional disclosure upon both healthy participants and participants with pre-existing morbidity. Sixty-one studies were meta-analysed and it was suggested that there was no clear improvement for emotional disclosure compared to controls in most outcomes measured and so therefore, they state that the paradigm is in need of reassessment. Pennebaker, however, insists that a wealth of EDP studies continue to support the efficacy of the paradigm; he cites over sixty on his website (see http://homepage.psy.utexas.edu).

Although the methodology of the EDP is still widely utilized, according to the meta-analyses cited above, there is as yet no clear consensus regarding the reliability of the paradigm. However, Chalmers and Altman (1995), Eysenck (1994), and Petitti (1994) remind us that we should be cautious of the results of systematic reviews and meta-analyses. For example, among some of the criticisms it is argued that they tend to have restricted coverage; the quality of the studies included is at times questionable; the homogeneity of the studies included is at times questionable; and at times there is a failure to relate the data to a theoretical framework.

This last point is taken up in this paper. Here we argue that the variation of effect is not adequately related back to the disclosure data due to the lack of a comprehensive theoretical framework within the paradigm. Thus, we go on to look at the present frameworks offered to theorise the variation of effect within emotional disclosure studies and then offer a more inclusive and comprehensive model of the underlying embodied mechanisms that may be associated with emotional disclosure.
Some reasons for the variation of effect within the EDP studies are discussed by Pennebaker, Mayne, and Francis (1997). They state that the form and content of an individual's disclosure narratives can be associated with the positive and negative outcomes. Two psychophysiological models are usually hypothesized to describe a correlation between the various disclosure narrative constructions and positive and negative outcomes. These are namely the inhibition confrontation model and the cognitive reappraisal model (Pennebaker, 1997).

In the inhibition confrontation (or catharsis) model, inhibition of trauma-related thoughts, feelings, and behaviours requires physiological work, leading to autonomic arousal in the short term and placing cumulative stress on the body in the long term (Pennebaker & Francis, 1996). Inhibition is thus an active process which increases the risk of both physical illnesses and mental distress. Talking or writing about emotionally laden traumatic experiences is a relatively safe way of confronting them: having done so, inhibition is no longer necessary so the stress it causes is reduced, with consequent health benefits. The other explanatory framework commonly used is the cognitive reappraisal model. Here, benefits flow primarily from the creation of a coherent narrative of the trauma an activity which might have multiple therapeutic effects. First, coherent narratives may increase perceived self mastery, enabling participants to understand their experiences as more controllable (Paez, Velasco, & Gonzalez, 1999). Lepore and colleagues suggest that “When people feel more control over their emotional experience, negative moods should dissipate, resulting in less chronic subjective stress” (Lepore, Greenberg, Bruno, & Smyth, 2002). Second, reappraisal may also change the meaning of events, reducing their associated negative affect. For example, a person’s narrative may come to include the statement “I considered how much worse things could have been” (Lazarus & Folkman, 1984), so emphasising positive aspects of a negative event. Finally, constructing a new narrative [p.321] of a traumatic episode may also generate insight into the experience (Meichenbaum, 1977), producing various health benefits (Traue & Pennebaker, 1993).

These two models are not mutually exclusive and can be deployed together to explain the beneficial effects of particular interventions. Both models address a range of findings from the various EDP studies via a focus on the activity of written or spoken re-narration, so deriving their explanatory force in part from an association with the “talking cure”. However, whether used singly or together, both models are flawed in three important respects. First, both rely upon an undifferentiated and relatively naive notion of the process of inhibition, even though there is evidence that inhibition is a diverse, multi-faceted phenomena. Second, neither model sufficiently acknowledges that the form and content of talking or writing within the EDP (and by implication, the kind of re-narration produced) might make a difference. And third, although both models claim to explain the positive health outcomes of the EDP neither specifies neural pathways or embodied mechanisms whereby these health benefits might be produced.

Attempting to address these problems in this paper we first formulate a distinction between active inhibition and passive inhibition and offer a more complex understanding of cognitive reappraisal. Second, in the light of these reconsiderations of inhibition confrontation and cognitive reappraisal, we theorize the effects that different forms of disclosure narratives may exert upon an individual. Third, there is an account of Damasio’s (1995, 2000) somatic marker hypothesis and in particular his notion of the as-if body loop, in an attempt to elucidate some of the neural particularities that may be involved in the positive and negative health outcomes of emotional disclosure.

**Inhibition**

Researchers in the EDP typically conceptualize emotional inhibition relatively unidimensionally as an active process of constraining thoughts, feelings, and behaviours (Pennebaker, 1997); a type of deliberate thought suppression (Petrie, Booth, & Pennebaker 1998). However, there are theoretical perspectives and empirical evidence which suggest that emotional inhibition is more complex. Emotional experiences may be excluded from consciousness in a number of ways. For example, psychoanalytic literature often distinguishes between suppression and repression. Although Sigmund Freud used the terms interchangeably, Anna Freud distinguished between them, describing suppression as a conscious process and repression as a separate,
unconscious process (Erdelyi, 1990). In a more recent paper by Erdelyi (2006), he puts forward a unified theory of repression, in which it is divided into two subclasses: “(1) inhibitory or subtractive processes (e.g., degrading the “signal”), and (2) elaborative or additive processes (e.g., adding “noise” to the signal)”. Although this paper includes both forms of repression, this section looks specifically at what Erdelyi calls inhibitory or subtractive processes, but, we add a further distinction to these processes which is distinguished as active inhibition and passive inhibition; Erdelyi’s unified theory does not include what is configured here as passive inhibition, when describing subtractive processes he focuses his attention on what is configured here as active inhibition. Later in this paper when looking at cognitive reappraisal there will then be a focus on a similar understanding to what Erdelyi calls elaborative or additive processes.

In what follows we use a similar distinction to what has been nominated suppression and repression, by distinguishing between active inhibition and passive inhibition. By active inhibition we mean both that such inhibition may be deliberate and also that it may operate on the edge of consciousness, placing the person in a simultaneous state of “knowing and at the same time not knowing” (Breuer & Freud, 1991, p. 236). Passive inhibition, by contrast, is the wholly non-conscious inhibition of affect.

**Active inhibition**

Active inhibition involves an active constraint of thoughts, feelings, and behaviours or “ …a semi-conscious decision to defer paying attention to a conscious impulse of conflict” (Vaillant, 1990). For example, a child who was sexually abused stated “[W]hen I remember it, I keep trying to think about good things like Christmas and it goes away” (MacCahill, Meyer, & Fischman, 1979). Thus active inhibition, at times, may be viewed as a defence mechanism and represents a continuum of cognitive avoidance proportional to the psychological pain experienced by the individual (Christianson & Engelberg, 1997). This pain avoidance can in various contexts be adaptive to the functioning of the individual. The trauma may be too taxing for an individual to re-member, depending upon for instance: the nature and age of the trauma, the social support available, and the person’s general psychological state. However, prolonged active inhibition is usually associated with negative health. The continued effort to suppress highly charged negative experiences challenges the homeostatic regulation of the immune system. Numerous studies, addressing a range of arousal types, provide evidence for this assertion. For example, deception or suppression of the truth has been shown to increase sympathetic nervous system activity as measured through skin-conductivity (DePaulo & Rosenthal, 1979). Suppression of exciting thoughts, specifically sex, has a similar effect (Wegner, Shortt, Blake & Page, 1990). Likewise, psychophysiological differences are found between participants asked to suppress or not to suppress affect aroused due to emotionally arousing films (Gross & Levenson, 1993, 1997).

The notion that psychological activities and conduct can effect the physical functioning of the body (such as health) is well established in psychoneuroimmunological studies. Many of these studies have looked at active inhibition. For example, personal coping styles which suppress negative affect increase the risk of cancer by the down-regulation of natural killer cells which target cancerous cells and destroy them (Shaffer, Graves, Swank, & Pearson, 1987; Gross, 1989; Kune, Kune, Watson, & Bahnson, 1991). Booth, Petrie and Pennebaker (1997) found a proliferation of circulating lymphocytes in the blood of participants after writing about emotional topics. They then looked at the effects of suppression by examining participants’ blood before and after they wrote about either emotional or nonemotional topics, with or without deliberate attempts at thought suppression. They found that suppression caused a significant down-regulation in circulating T lymphocytes (CD3) as well as a marginal down-regulation in CD8 (T suppressor) cells and total lymphocyte numbers. By contrast, emotional writing proliferated circulating CD4 (T helper) cells and the number of total lymphocytes (Petrie et al., 1998). These studies are consistent with other work which associates [p.323] suppression with an increase of sympathetic nervous system activity (Gross & Levenson, 1993, 1997; Wegner et al., 1990).
Passive inhibition

Passive inhibition is problematic because, being a non-conscious internal process, it is difficult to define, measure, and observe. It may be related to notions of desynchrony whereby a response can be manifest at one level but not on another (Hugdahl, 1981; Turpin, 1991) and thus implicated in various anxiety and dissociative disorders. The pathogenic process may be summarised as follows, “when affect is disconnected from cognitions the two processes are no longer mutually regulating, and a state of “disorder” or “disease” may develop” (Schwartz & Kline, 1995). Understandings of the distinction between declarative memory and other memory systems help highlight the complexities of passive inhibition. Erdelyi (1990) observes that it is widely acknowledged in studies of neurological amnesia that there may be amnesia for declarative facts whilst other fragments of the experience are retained, “[T]he patient, then, may reflect all-too-good memory for past traumas, maladaptive interpersonal skills, irrational fears, inappropriate styles – with no recall of how the pathogenic procedural knowledge was acquired” (Erdelyi, 1990).

In some cases, this may be the result of faulty encoding caused by excessive levels of cortisol in the hippocampus, a brain structure implicated in the formation of new memories. Cortisol is released in response to stressful events. Moderate levels of cortisol within the hippocampus, facilitate memory encoding, since cortisol heightens perceptual and discriminatory abilities; this may be the origin of so-called flash-bulb memories (Brown & Kulik, 1977). However, extreme or prolonged trauma may induce an aggressive build up of cortisol, which in turn can lead to atrophy and partial temporary dysfunction of the hippocampus, with the consequence that episodic memories are not adequately encoded (Bremner et al., 1995).

LeDoux (1989, 1994, 2002) has studied the neuroanatomy of fear responses, showing that they are processed by a system which includes the bilateral amygdalae, brain structures which generate both autonomic and endocrine responses. LeDoux has found two distinct pathways by which sensory information can be generated from an amygdala response: a higher, slower, cognitive pathway involving conscious decisions about a stimulus (an emotional operant) and a lower pathway in which there is no conscious cognition but rather an involuntary autonomic response (an emotional respondent) (LeDoux, 1992). Hence, it is possible that on occasion the stimulus features of an object might trigger a fear response without any significant higher cognitive processing intervening, thus problematizing the formation of coherent episodic memories.

Another factor that might contribute to passive inhibition is exhibited in studies which show significant decreases in regional cerebral blood flow (rCBF) in Broca’s area during trauma (Rauch et al., 1996). Broca’s area is a left hemisphere region of the brain implicated in language production. Decreased rCBF in this area during trauma suggests that activity there is reduced, and Cozolino (2002) argues that the ensuing degraded linguistic ability might account for the “speechless terror” often reported by victims of emotional trauma.

Elsewhere, other problems concerning the recollection of emotional memories have been conceptualized. For example, some emotional memories are based within what Bucci (1995, 1997) nominates the subsymbolic order. Essentially these types of memories are not symbolised through imagery or language and so are particularly difficult to recollect. Bucci suggests that emotional memories tend to be of a different order to more cognitive memories in that they may at times be allied to neural processes rooted within pre-symbolic stages of intellectual development.

Thus, there are different forms of inhibition with correspondingly different effects. Active inhibition requires psychophysiological effort to withhold memories from consciousness, whereas passive-inhibition produces psychophysiological excitation through residual affective, implicit or feeling memories which non-consciously trigger somatic responses. Moreover, both forms may operate together, either simultaneously or sequentially, since as Erdelyi observes, “by inhibiting thinking about the to-be-remembered materials, we cause oblivescence to exceed reminiscence, and amnesia results” (Erdelyi, 1990). Affect laden memories that are continually pushed away from consciousness might, as a consequence, become harder to retrieve and result in a passive form of inhibition. Support for this notion is supplied by an fMRI study (Anderson et al.,...
2004) who stated that thought suppression (voluntary) conditioning may lead to thought repression (involuntary) forgetting.

Perhaps the phrase “passive inhibition” is imprecise as it implies, in the context of memory study, the inhibition of a memory which exists in an ontologically material sense. However, the way the notion is used in this paper is much wider. Some of the memories are not so much passively inhibited as they may have not existed in the first place, as there may have never been a memory of the experience appropriately laid down. For instance, it may not have been sufficiently encoded and thus in this sense is not inhibited but actually does not materially exist in an identifiable way; only fragments of the event exist in memory form, for example, a feeling memory. Although this imprecision is recognised, for want of a better phrase, the phenomenon in this paper will be continually referred to as ‘passive inhibition’ in order to distinguish it from active inhibition.

This more comprehensive elaboration of inhibition helps to illuminate some of the complexities associated with the EDP and may contribute to the understanding of some of the variability associated with its health effects that have been highlighted in the lack of consensus within the meta-analyses that have been conducted. So, central to the argument presented in this paper is associated with the paradigm’s restricted theoretical understanding of inhibition confrontation particularly with regard to what we have here nominated “passive inhibition”. It is argued here that inhibition confrontation may in fact lead to negative outcomes when memories are passively inhibited. As this paper has already alluded to and will go on to argue in more detail, attempting to confront passively inhibited affect may lead exacerbating somatic activities and in turn lead to reduced health. Further illumination is provided by a consideration of cognitive reappraisal, specifically with regard to the form of the re-narrations that produce it.

**Cognitive reappraisal**

Cognitive reappraisal can be allied to Erdelyi’s (2006) second subclass division of his unified theory of repression. He describes memory as also incorporating elaborative or additive process, “e.g., adding ‘noise’ to the signal” he relates Bartlett’s (1932) [p.325] understanding, of the revisionistic nature of memory, to Freud’s notions around the distortions of memory that are typical in part of Freud’s notion of repression, for example, through such defence mechanisms as: rationalisation, condensation, displacement, and reaction-formation etc. However, one of the main differences which is not explicitly pointed out by Erdelyi, is that Freud saw these forms of repressions as resulting from a need to withhold certain painful information from consciousness, whilst Bartlett was more inclined to suggest that memory is fundamentally revisionistic.

In contrast to Bartlett’s position, advocates of the cognitive reappraisal model, within the context of the EDP, tend to suggest that the reordering of memories in various ways, particularly to facilitate coherence, may have multiple therapeutic effects. This notion draws on the assumption that memory is relatively fixed and stable and thus available to be reworked through re-narration. However, alongside Bartlett many theorists today emphasize the revisionistic character of memory (Edwards & Middleton 1992; Middleton & Brown, 2005). Bartlett emphasized the cultural and social dimensions of memory and saw reappraisal as an inevitable part of narration. Participants asked to retell stories would often supply their own information, adding new details, transforming or inventing characters and revising plots to produce stories more sensible to them and to the social world, in light of their experiences. So, Bartlett was primarily concerned with how the past is constructed in the present to serve the needs of the actions of the current engagement.

A bottom line presentation of Bartlett’s argument suggests that any form of accurate recollection of an experience is simply not possible. However, here it is important not to throw the baby out with the bath water. Clearly there are many problems with the representation of reality which have historically been outlined by philosophers as far back as Plato to more contemporary deconstructionist arguments concerning epistemological relativism and language. This paper argues however, that although accurate recollection may be a psychological and philosophical impossibility, linguistic representations offer us the chance to communicate our experiences more or less accurately but quite often, due to varying degrees of biological,
psychological, socio-cultural constraints, accurate accounts of experiences can be overly limited with negative consequences.

An example of some of the socio-cultural constraints upon accurate recall is put forward by Gergen (1994). He suggests that individuals narrate memories in a certain way, adhering to and constrained by specific cultural norms. Gergen and Gergen (1988) signify these norms in five categories: valued endpoint, selection of related events, temporal ordering of events, causal linkage and demarcation signs. They state that these categories are drawn from the work of literary theorists, semiologists and philosophers of history and constitute the “rules of narrative” (Gergen, 1994). These rules are seen to be influenced by folktales, parables, myths and legends that constitute our “narrative heritage” and that they are essential to the narrative if it is to be trusted by others. According to Gergen, this notion was exemplified in a reanalysis of a study by Bennett and Feldman (1981) in which research participants were exposed to 47 testimonies that were either genuine attempts to recount the past or fabricated stories. The stories that were judged to be true were primarily those conforming to the rules of the well-formed narrative (Gergen, 1994) not those that were genuine. Gergen concludes that cultural expectations are placed upon the individual to tell a certain kind of story in a particular way. Expressions of what [p.326] really was perceived to occur are at times not permissible to communicate as the narrator may experience the story as being untellable due to social norms and demands. Thus, cognitive reappraisal is not simply about the adjustment of memory traces. Where traumatic memories of significant events are at stake it may also involve relatively profound reconfigurations of identity and some degree of accompanying existential anxiety, since notions of self are bound up with and partially emerge from the narratives that also constitute the traumatic event: change one, and you must also at least consider changing the other. Cognitive reappraisal is thus influenced by many factors, some of which are both external to the individual and subject to more-or-less gradual change. For example, both relationship changes and cultural shifts will condition the relative legitimacy and currency of the narratives people can deploy.

Thus, since cognitive reappraisal is the outcome of re-narration, it is relevant that there are significant links between memory, language, narrative and selfhood. Narratives confer structure upon experiences (McAdams, 1993): locating them within webs of meaning, making them this kind of event rather than that. They facilitate both re-membering and re-telling, allowing us to become accountable and acceptable to ourselves and others, such that “the human being is understood as that agent which constructs itself as a self through giving its life the coherence of a narrative” (Rose, 1998). This does not mean we must reduce the self to language and narrative; rather, that narratives embedded in language are a primary means whereby material events and embodied experiences get situated within local moral and social orders, so acquiring their full human significance. Moreover, the significance of language and narrative are not confined to the social world. Following Vygotsky (1962) it is widely accepted that the skills of metacognition (e.g., not being able to remember, but being able to reflect upon memory and deploy effective strategies for retention, retrieval and ordering) are primarily acquired in social interaction. Suggestions made by others (together with their accompanying affective tone) get rehearsed as private speech (where we speak out loud to ourselves) before being internalized as inner speech, the abbreviated, condensed form that guides our activities. This understanding is the basis of Luria’s view that language influences the development of higher cortical structures, becoming the “chief mechanism of voluntary action” (Luria, 1960) and “the highest regulator of human behaviour” (Luria, 1966). Since Luria and Vygotsky there has been much work conducted in looking at the importance of inner speech; for example, it has been associated with the regulation and structuring of a substantial amount of human activity (Clowes, 2007).

Hence, reformulating a story, in order for it to be a socially acceptable narrative, may influence a wealth of biopsychosocial phenomena. Of course similar to some of the positives associated with inhibition, re-narrating in order to further suppress the actual memory of the event may have beneficial payoffs. As was stated above the trauma may be too taxing for an individual to re-member, depending upon for instance: the nature and age of the trauma, the social support available, and the person’s general psychological state. This function then may lead to a lowering of stress and anxiety in the short term which may have otherwise been induced due to the narrative incurring a negative reception, yet in the long term, stress levels are likely to
increase, according to the stress theory that was adhered to earlier in this paper (Pennebaker & Francis, 1996).

[p.327] Associating narrative style with inhibition and reappraisal

Thus, focusing on the narrative processes that produce reappraisal, Pennebaker and colleagues suggest that particular combinations of inhibition confrontation and cognitive reappraisal are associated with positive health outcomes (Pennebaker et al., 1997). EDP studies have suggested that participants who confront otherwise inhibited negative emotional episodes through disclosure and increase their use of cognitive-mechanistic words (e.g., understand, know, realise) across repeated disclosures (indicative of cognitive reappraisal) tend to have increased health benefits. Further, Bucci (1995) reanalysed a disclosure study and identified distinct subgroups of participants whose health both improved and worsened. Those whose health improved structured their experiences using narratives that followed a “referential cycle”, wherein affect laden memories are increasingly linked to an evolving coherent narrative; by contrast, those whose health deteriorated failed to make such integrations. On this view, cognitive reappraisal and inhibition confrontation is driven by the increasing integration of narrative and affect, achieved through re-telling and indexed by an increase in cognitive-mechanistic language and affect language (indicative of inhibition confrontation) this produces positive health outcomes. However, as Bucci suggests, if experiences are repeatedly rehearsed without such integration and re-narration of the event remains static or increases the misrepresentation of the actual experience, affect is continually evoked but not integrated into a new, more functional narrative.

Thus, through this more complex view of inhibition it appears that if the inhibition is active, effort may then be expended inhibiting it, if the inhibition is passive the negative affect associated with the initial trauma is re-evoked: in either case the net result is an increase in ANS activity that produces negative health outcomes. Similarly, if the degree of cognitive reappraisal due to re-narration unduly misrepresents the event, there is also likely to be an increase in ANS activity and associated negative health outcomes. To further illustrate the neuropsychophysiological compositions underlying these processes, we have employed Damasio’s understanding of as-if body loop processes described in his somatic marker hypothesis.

Somatic markers and the as-if body loop

Damasio and colleagues (Bechara, Damasio, & Damasio, 2000) describe a neural system that integrates feelings, affect and cognitions. They propose that much decision making is influenced by marker signals that arise in bioregulatory processes; these influences may operate both consciously and non-consciously. The ventromedial (VM) sector of the frontal lobes is a critical node in this system; they show that VM lesions significantly disrupt decision making in social settings, whilst leaving intellectual faculties such as learning, memory, language and attention relatively unaffected.

Damasio (1995, 2000) suggests that there is a constant representational mapping within the brain of the bioregulatory state of the body. This body-state mapping is coupled with monitoring of the external world. As new stimuli are encountered, maps previously associated with similar stimuli get reconstituted, and the feelings associated with previously experienced stimuli are re-evoked. These feelings then act as a covert biasing system which facilitates rapid decision-making, marking options [p.328] with valences according to whether the re-evoked feeling is negative or positive. Evidence for this “somatic marker” hypothesis is derived both from clinical studies which reveal a “double dissociation” between VM lesions, impaired decision making and flattened affect, and from experimental work with brain injured participants. In one study, participants tried to win money in a card gambling task. A control group without lesions developed higher levels of skin conductivity before proceeding with high risk gambles, indicative of heightened ANS arousal, and subsequently developed effective strategies. However, participants with bilateral VM lesions did not develop such changes and were also unable to find appropriate gambling strategies and avoid losses.

Damasio and colleagues thus propose that decision making may be dependent on the generation of somatic states, configurations of the “internal milieu, visceral and musculoskeletal” activity (Bechara, Damasio,
Damasio, 2000). The VM prefrontal cortex is necessary to form such links between factual knowledge and body-states although it is emphasised that these links are dispositional, they do not hold the representation of the facts or of the emotional state explicitly, but hold rather the potential to reactivate an emotion [body-state] by acting on the appropriate cortical or subcortical structures (Bechara et al., 2000). Importantly however, following repeated associations the actual body-state is not always actuated, but instead may be merely re-presented as a somatosensory map: Damasio names this the as-if body loop, since the brain creates the feeling of a body-state without there being any actual change.

The association between a certain mental image and the surrogate of a body state would have been acquired by repeatedly associating the images of given entities or situations with the images of freshly enacted body states. To have a particular image trigger the “bypass device,” it was first necessary to run the process through the body theatre, to loop it through the body, as it were (Damasio, 1995).

Where formerly a somatic profile was evoked, now a cortical representation of that profile is evoked instead, accelerating the process. Because of the advantages this acceleration provides, Damasio suggests that this as-if body loop will usually come to supersede the somatic state response, so that:

the fundamental component of the somatic marker hypothesis is that bioregulatory signals, including those that constitute feeling and emotion, provide the principle guide for decisions and are the basis for the development of the as-if body loop mode of operation (Bechara et al., 2000).

Clearly, the movement from somatic states to the as-if body loop may reduce bodily changes and lower ANS activity. A stimulus which previously initiated a highly aroused body-state now calls out its surrogate instead, using this to inform executive activity. Aside from the cognitive advantage gained by the increased speed of processing due to the as if body loop, this reduction of body-state and ANS activity is likely to also be conducive to positive immunological functionality.

Conclusion

In the light of this more complex understanding of inhibition and of narrative formation and reconstruction, Damasio’s as-if body loop theory helps to illuminate a possible number of neurophysiological mechanisms concerning the effects of the EDP. Primarily, what is of interest here is the possibility that the confrontation of inhibited affect through repeated disclosure leads to an as-if body loop [p.329] and subsequently lowers body-state activity. Second, cognitive reappraisal may assist the integration of fragmented memories into as-if body loop processes, again facilitating the lowering of body-state activity. Third however, if a narrative is constructed in such a way that it does not integrate memories appropriately (for example, the narrative constructions fails to integrate feeling memories that are passively inhibited or the narrative constructions adversely misrepresent the actual experience through cognitive reappraisal), the participant will instead repeatedly re-evoke the inhibited associated affect within the disclosure period, and possibly engage in some active inhibition as a consequence; hence, ANS activity is increased (which depletes immune system functionality) and health is likely to deteriorate over the study period. Thus, this more complex understanding of inhibition confrontation and cognitive reappraisal used in conjunction with a more detailed and closer analysis of the disclosure narrative data is likely to enhance our understanding of thevariability of effect that is evident today within the paradigm.
References


