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RESILIENCE AND RECOVERY: DUMPING DUALISM

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ABSTRACT

Neurobiology and experimental psychology are discovering the faults in the analytical model of therapy for emotional distress, and increasingly recognize a monistic model of brain/body synergy, i.e. the effect of the body—and hence emotion—on cognition, and the effect of pre-conscious triggers on physiology. Psychology services within the NHS use a formulaic dualistic Cartesian logico-rational approach to recovery, despite the implicit contradiction of accepting mindfulness-based treatments. We explore the research base of mind/body medicine to elucidate a new therapeutic understanding.

Key words: hypervigilance, reappraisal, overgeneral, experiential, decentring, rumination

INTRODUCTION

Body is a kind of mind; mind is a kind of body.

Thich Nhat Hanh

Beck proposed a model of how cognitive-behavioural therapy (CBT) works in depression in his treatment manual (Beck et al., 1979): that interventions aimed at cognitive structures or core schemas are active change mechanisms; that engagement in logico-rational Socratic discussion enables the patient to change their core beliefs and effect a change in their feelings and behaviour, based on a Freudian/Adlerian concept of talking therapy. Analyses of CBT have shown that it does 'work' as a therapy, but that the active change has little to do with the changing of core beliefs. CBT is a complex intervention and the other factors involved, such as therapist–client psychodynamics and behavioural activation, are the vehicle for change (Blatt et al., 1996; Jacobson et al., 1996; Zuroff et al., 2000; Longmore & Worrell, 2007). Kroenke and Manglesdorff (1989) found only 16% of referrals to medical outpatient departments had a recognizable organic cause; the rest had symptoms which were called medically unexplained symptoms (MUS)—otherwise known as functional disorders encompassing many conditions such as irritable bowel syndrome (IBS), chronic pelvic pain (CPP), non epileptic seizures, swallowing problems, and fibromyalgia. Advances in neurobiology in the past 20 years suggest that the Cartesian brain/body separation is unhelpful and redundant.

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NEUROBIOLOGICAL PERSPECTIVE: BRAIN/BODY LINKS

DUALISM/MONISM

The science of medicine in the West is dominated by an implicit *dualism*. Dualism is a view that the brain and the body are separate entities, that the body is a machine that is largely self-supporting, with the brain working the levers and causing excitation of the body as necessary, and that thinking is independent of the body. *Monism*, more favoured in Eastern philosophy, is a view that the brain and the body are inseparable and that the terms 'mental' and 'bodily' are unhelpful concepts. Buddha proposed the pathway to enlightenment as 'the contemplation of the body in the body'.

BRAIN TO BODY

The biological sciences today increasingly recognize that the brain and body are inseparably linked. The neurologist William James (1842–1910) first proposed that emotions are defined by changes in the body rather than mental activity; if we see a bear we run away before we think; we feel fear *because* we run. The sequence of events is thus counterintuitive: 'we feel sorry because we cry, angry because we strike, afraid because we tremble, and not that we cry, strike, or tremble because we are sorry, angry, or fearful' (James, 1884). It is the 'fight or flight' response and the changes it brings about in the body that *tell you that you are afraid*. Joseph LeDoux in *The Emotional Brain* (1996) updated and clarified the neural pathways that James' observations implied, showing that pre-conscious mechanisms bypass the conscious mind to excite and alter the body (see Figure 1).

LeDoux found that there were two pathways for the perception of threat, in this example the visual image of the snake. The image is first processed pre-consciously in the *thalamus*, then the pathway splits—one part goes to the *amygdala* (where noxious stimuli become associated with sensory input giving rise to conditioned fear), the other to the *visual cortex* where it reaches consciousness. However, before this conscious signal can be processed, the amygdala has already alerted the body to danger, via the *sympathetic* branch of the *autonomic nervous system* (as shown in Figure 1) signalling the body to prepare for fight or flight. The stomach and bowels contract causing nausea, diarrhoea, pain, vomiting, difficulty swallowing; elsewhere the pupils dilate, tension in the muscles increases, heart rate and blood pressure increase, the hands sweat, and adrenaline floods the body.

These are signals we are all familiar with ('sick' with fear); equally we can all probably remember them coming on suddenly, even with no real threat, as a response to ambiguous information—a stick that looks like a snake, a sound that is unfamiliar, or a movement in a dark alley. In these circumstances your body will suddenly jolt into action; a fraction of a second later your conscious mind will tell you that it's just a stick and harmless, but by then your body is fired up for action and the bodily responses will take a while to die down. Such is the price of thousands of millennia avoiding jaguars and snakes; it is better to react quickly with action than await a more precise conscious assessment of danger, hence the thalamic pathway to the body via the amygdala is called the 'quick and dirty' pathway.

LeDoux found that the thalamo-amygdala pre-conscious pathway is the pathway that promotes fear conditioning and, on later exposure to the conditioning stimulus, the subsequent expression of such conditioning; thus fear conditioning can be acquired and threats perceived

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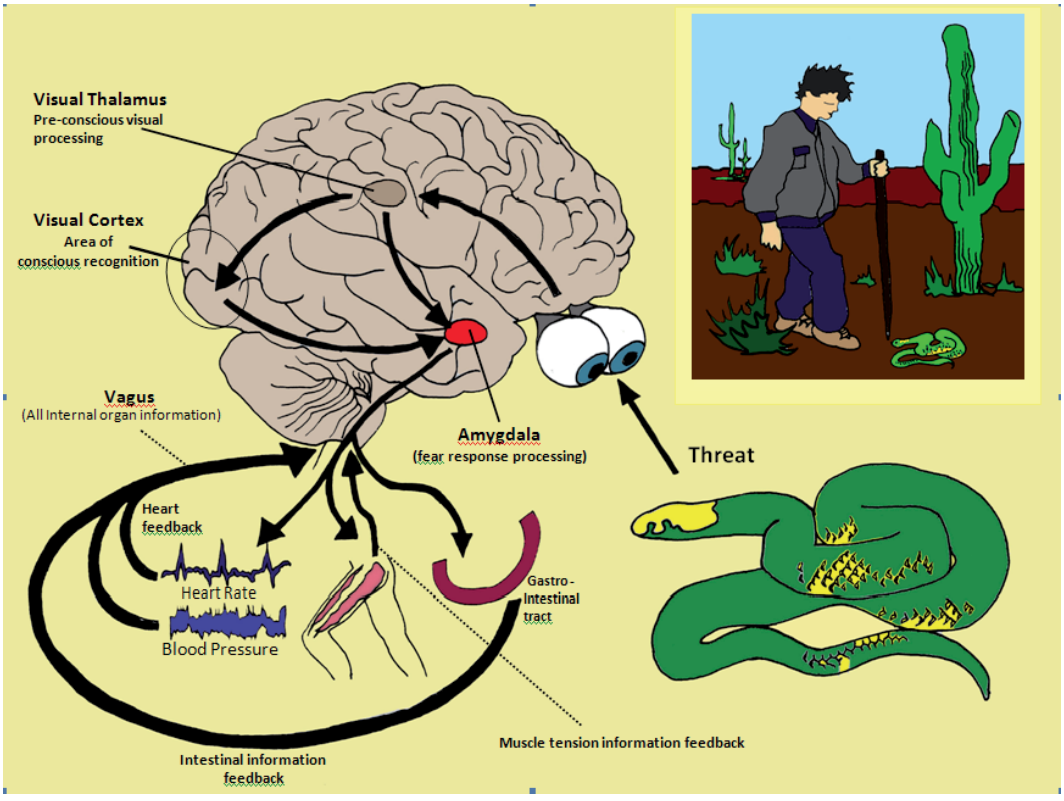

Figure 1. Conscious and pre-conscious threat pathways

entirely outside conscious awareness. LeDoux concluded that, after a pre-conscious threat affects your body, 'you may find yourself in the throes of an emotional state for reasons you do not quite understand' (LeDoux, 1996: 203). The conscious visual pathway had no impact on the perception of threat, the sense of fear, or its bodily expression. Where there is no conscious recognition of a threat, this leads to a persisting background emotion of fear.

The challenge in overcoming conditioned fear is that many things *associated* with fear can later cause pre-conscious fear conditioning. There are two different types of fear inducers, *primary* inducers and *secondary* inducers (Bechara et al., 2003). Primary inducers comes from direct stimulation of the amygdala and relate to large moving objects, snakes and spiders, loud noises, aggressive animals and people; secondary inducers come from the coincidental exposure during the action of primary inducers to external environmental factors, creating external triggers linked to fear expression in the amygdala. In Figure 1, the cactus may become pre-consciously associated with the fear of the snake (primary inducer), causing the man to feel anxious whenever he sees a cactus (secondary inducer) and, as LeDoux's work suggests, as he will not be consciously aware of the source of his feeling, it is difficult to change (for an explanation of primary and secondary inducers see also Damasio, 1994: 131–139).

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LeDoux suggests that in early childhood fear conditioning in the amygdala is present before the narrative memory circuits develop in the *hippocampus*. This would mean that in our early years, possibly up to 6 or 7 years, the amygdala will acquire and hold fear associations without an accessible conscious memory of events which could modulate the effects. LeDoux says that such associations are particularly challenging to shift. Thus instances of trauma—for example, neglect, bullying, abuse, the sound of anger or distress from a carer—set up associations with sounds, sights, and contexts that may later trigger pre-conscious fear via the thalamo-amygdala pathway. Vicarious fear (witnessing fear) is a particularly powerful pre-conscious trigger in children; witnessing fear/anger in parents is as powerful in conditioning as experiencing it yourself: 'fear learning following observation, like Pavlovian conditioning, need not be accompanied by explicit awareness of the Conditioned Stimulus (CS) for an emotional response' (Ollson & Phelps, 2004: 827). Highly stressful events will disrupt normal memory formation and access; LeDoux specifically compares the manifestations of post-traumatic stress disorder (PTSD) to physical and sexual abuse (1996: 130) (possible mechanisms are discussed below under 'Post-CBT Perspective'). Once there is an established pattern of withdrawal in specific situations, and a negative cognitive bias is established, there is no impetus in the brain or body of the person to change their habitual reactions, although we have found it can be surprisingly simple in practice.

BODY TO BRAIN

Other researchers have identified feedback mechanisms *to* the brain *from* the body, again outside of conscious awareness. The *vagus* nerve, the tenth cranial nerve, which enters/leaves the brain at the level of the brainstem and connects with a large number of brain nuclei, is often seen only as a top-down pathway for control of the internal organs (heart, intestines, etc.), but it is increasingly recognized to carry signals to and from these organs. Some 80% of the information in the cardiac vagus (e.g. how fast the heart is beating, how much stretch and tension there is in the heart muscle) is travelling from the heart *to* the brain (Porges, 2001) and similar information about the stomach and intestines travels up the enteric vagus (Foley & Dubois, 1937). This is now used therapeutically: external electrical vagal stimulation has an effect on cortical function and is established as a treatment for refractory epilepsy (for a review see Groves & Brown, 2005). Peripheral administration of adrenaline increases the number of impulses travelling up the vagus nerve from autonomic peripheral beta adrenergic receptor afferents on the blood vessels (Miyashita & Williams, 2006) and the peripheral administration of adrenaline (which does not cross the blood–brain barrier) has long been shown to increase the encoding of emotional memory (Gold & van Buskirk, 1975).

The information from the autonomic nervous system's peripheral receptors is fed into brainstem nuclei (i.e. vagal afferents to the *nucleus tractus solitarus* (NTS) and spinal afferents to the *locus coeruleus*). The locus coeruleus influences the patterns of activity and the functional (as opposed to physical) structure of the brain by links to the brainstem nuclei. It is a key neuro-modulatory centre (McGaugh & Roozendahl, 2002), feeding noradrenaline to multiple brain areas including the hippocampus, amygdala, and pre-frontal cortex, and changing the conductivity of neurons by affecting chloride pumps through metabotrophic membrane-based G-protein receptor activation. This results in changes in processing patterns throughout the brain from diffuse to focused attention, mediating wakefulness in the pre-frontal cortex; dif-

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ferent states of wakefulness are associated with additional increases in the neuromodulators glutamate or dopamine in pre-frontal cortices (Kodama et al., 2002), i.e. increase in dopamine in the prefrontal cortex increases creative problem solving, sociability, and flexibility (Isen & Labroo, 2003). There is also an observed difference between the pattern of brain interaction and emotional memory which is dependent on the sequence of arrival at the amygdala of noradrenaline from the locus coerulus and circulating adrenal corticosteroids from the adrenals, produced when stress activates the hypothalamus (which in turn activates the adrenals; see van Stegeren et al., 2010).

There are numerous neuro-modulatory systems with differing effects (mostly unexplored) with similar feedback paradigms; all can potentially change the functional structure, the 'state' of the brain, and all have inputs from the autonomic afferents from the body. There is thus a potentially infinite repertoire of afferent autonomic effects of the body via the brainstem on cortical processes, which, if activated by pre-conscious triggers, act entirely outside conscious awareness by changing cognition and action. There is also a network of sensors in the body's muscles which convey continuously updated information about the level of body muscle tension into the cortex through the spinal cord. At the same time information is fed from autonomic afferents via the vagus directly to the cortex, specifically the anterior cingulate cortex and the insular cortex (Craig, 2003). These areas drive broad behavioural categories, such as approach and withdrawal, and identify specific action repertoires.

INTEGRATED THEORETICAL PERSPECTIVES

THE SOMATIC MARKER HYPOTHESIS

The idea of the body being an inseparable part of cognition has been given an interesting theoretical basis by the 'somatic marker' hypothesis of Antonio Damasio and described in his book *Descartes' Error* (1994). Damasio's team analysed the accounts of Phineas Gage, who had a part of his brain sliced through cleanly by an explosion in 1848. Although intellectually unimpaired, he would swear and behave completely inappropriately in social situations as well as gambling heavily (and losing). Damasio's research team looked at current patients in whom stroke, accident, or tumour had damaged similar pathways to Gage's within the brain. These pathways convey autonomic information from the body, viscera, and muscles via the anterior insula to the place where affect is linked with current cognition—the anterior cingulate cortex (ACC). It is here that the autonomic signals from the body become the driver for change of strategy when a current goal becomes unrewarded (Shima & Tanji, 1998). Damasio's team found these patients' primary inducer fear circuitry was intact (the pre-conscious thalamo-amygdala pathway in Figure 1): the patients would react appropriately to loud noises and snakes but, unlike normal controls, they showed no sympathetic autonomic response (measured by skin conductance which increases with sweating); that is, no limbic response to secondary conditioned fear such as gruesome pictures or remembered fearful events.

Bechara and colleagues (1997) invented a gambling task where subjects had to make the choice of which of four piles to draw a card from: some packs paid high rewards but had higher losses, others paid lower gains but had smaller losses. Over time it was better to choose from the packs with lower gains, but this was not explained to the players who were just told to maximize their profits. Normal people playing this game, once they had made a few initial

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losses, would avoid risky choices even before they explicitly understood the rules, and this avoidance was accompanied by increased sweating (a purely sympathetic bodily response) *just before they made their choice*—their gut feeling of fear was guiding their decision. The brain-damaged or compromised patients, however, were not guided by the fear they had just experienced with initial losses, so would lose heavily and showed no early skin conductance response. Although they reacted after their losses (a primary fear response) they could not subsequently remember the association of the 'dangerous' pack and their losses and apply the embodied learning (gut feeling) from it. Without this seamless entirely unconscious integration of emotion and current cognition, the patients were severely disadvantaged in decision making in all spheres. They were unable to make sensible cautious predictions in situations where caution was required, and they could not experience a sympathetic fearful response to objects/situations presented in working memory (which pack to choose from) which, previous to their brain insult, would have elicited such a response. An object/situation could also be a social encounter in which they would not react appropriately. Being unable to use their bodies to recall the distress they had felt in social situations (i.e. a social 'gaffe'), the patients could not model the consequences of their words or actions on others. Thus they lacked empathy—a very serious social deficit.

Most interesting is what this reveals about the importance of emotion in everyday cognition for normal people and how they use their bodies to predict the negative outcome of their actions. If A needs to speak to B about B's work and needs to tell B that something is unsatisfactory, A will project himself forward in his own body to 'feel' for himself any possible distress B will feel to what A is thinking of saying, putting himself in B's shoes. If A's body feels distressed from this he may change tack and perhaps delay the discussion or try a different approach. We experience levels of distress in others through our own distributed brain–body and body–brain networks.

The functional importance of the amygdala suggests it is not a vestigial organ from prehistoric survival. Although clearly it will protect us from threat, it also has an extremely important social and civilizing role. The discoveries that the size of an individual's amygdala is directly related to the level of social play in primates (Lewis & Barton, 2006) and the size of humans' social networks (Bickart et al., 2011)—a key element of social and biological success—gives strong validity to the somatic marker hypothesis.

The *insula* (insula cortex) is the final destination of autonomic information from the body; it appears to function as 'an encephalization of autonomic control … thus, our bodies have three interacting systems for the control of the gut: one in the gut itself, one in the hypothalamus for automatic homeostatic control, and one in the cortex closely linked to self-awareness, motivation, and decision-making' (Allman et al., 2010: 513). It is also implicated in the feeling of knowing and the recognition of social errors (Craig, 2009). The insula can be considered as the autonomic equivalent of the sensory cortex, and the ACC as the equivalent autonomic motor cortex (Craig, 2008). This makes it the destination of the signalling from the body to the brain which drives embodied decision making, as originally postulated by Damasio (1994) in the somatic marker hypothesis.

Activity in the right insula is positively correlated with sympathetic activation, increasing our perception of risk, while activity in the left insula is positively correlated with parasympathetic activity and activated 'mainly by positive and affiliative emotional feelings' (Craig, 2009: 61). Together they are seen as an interdependent system: activation of the right insula

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inhibits the left and vice versa. Functional magnetic resonance imaging (fMRI) studies show increasing co-activation of the amygdala by the right insula during puberty when it develops 'increased influence of negative feedback (i.e. error detection and/or anticipation) on cognitive control' (Allman et al., 2010: 513). Allman and colleagues propose that this growth period of co-activation supports the neurobiology of Damasio's somatic marker theory. The insula, the amygdala, and the ACC are seen as part of a network of brain structures monitoring danger (amygdala) and assessing risk (insula), with the ACC facilitating any change of strategy in a situation with a changing reward/risk base (Shima & Tanji, 1998), often with lightening speed in social interactions. If disturbed by fear or *the expectation of fear*, the organs (stomach, heart, intestines, blood vessels), along with the muscles, inform the brain, often entirely outside conscious awareness, of the presence of danger. The cortex then weighs up and quantifies the continuing need for metabolic and cortical vigilance in the insula (Singer at al., 2009).

RESILIENCE

An interesting correlation of these observations on the amygdala/insula/ACC complex comes from the field of resilience. Following on the work of Ashby et al. (1999) on the influence of positive affect on cognition, Fredrickson et al. (2003) found that the mediator of resilience is the ability to access positive emotion. A recent study on the neurobiology of resilience (Waugh et al., 2008) showed that in response to a cue that signalled an equal possibility of threat or no threat, low resilient individuals demonstrated prolonged activation in the right insula (signalling sympathetic activity) while high resilient individuals had no right insula response to the equal possibility *cue*; they only reacted when the *threat* was activated. This reinforces the key role of the insula in resilience.

What characterizes resilience is 'not a blunted response to affective stimuli'. High-resilient individuals 'do not differ from low-resilient individuals in the degree of experienced adversity while the threat is still present'; they just recover metabolically physiologically and cognitively more quickly (Waugh et al., 2008: 329). Waugh and colleagues propose that highly resilient individuals can simultaneously access positive and negative emotions which somehow modifies the right insula response, as suggested by Craig: 'Positive and negative affect interact in an opponent fashion; for example, it is well documented that social engagement (and oxytocin) can suppress arousal, stress, depression, and cortisol release' (2008: 285). These observations open up the field for methods of simultaneously increasing positive and decreasing negative affect to boost resilience, which the current authors are exploring.

NEGATIVE COGNITIVE BIAS

Feedback from the body to the brain will serve to concentrate attention on possible threats by increasing the scanning of incoming sensory information: 'once the amygdala detects danger it can influence sensory and cognitive processing in the cortex by activating modulatory systems' (LeDoux & Phelps, 2008). Perception of the outside world thus attains a *negative bias*: a neutral facial expression appears fearful; a letter is expected to be bad news, as is a telephone call; any ambiguity in an email tends to be interpreted as negative. An unrecognized pre-conscious threat has created a vicious cycle. This cycle of trigger–bodily fear–increased perception of threat–increased bodily fear is known as *hypervigilance*. This idea has been consolidated by Fales et al. (2008) who showed that depressed people have a particular difficulty with unat-

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tended threats stimulating their amygdala; the pre-conscious effect of threat is much stronger than for normal controls.

Patients with IBS (the commonest form of medically unexplained symptoms) demonstrate a tendency to process sensory information differently to normal controls, with increased selective attention to negative emotional material, and it is postulated that hypervigilance primes the attentional pathways to perceive normal visceral signals as painful (Musial et al., 2008). Work with animal models of IBS suggests that hypervigilance may happen due to a prolonged period with raised cortisol levels produced by a stressful and extreme event. This then alters the response of the amygdala, resetting it at a higher baseline level of activity while simultaneously reducing memory access (Myers & Greenwood-Van Meerveld, 2009). In humans this would be a good model for the cause of emotional distress (generalized anxiety, depression and PTSD—the commoner manifestations) or MUS such as whiplash, IBS, chronic back pain, fibromyalgia, and chronic pelvic pain (CPP) amongst others. Roelefs and Spinhoven (2007) found that in most studies abuse (sexual, physical, or psychological) is more common in IBS and CPP patients, and proposed that MUS originates from some traumatic incident which has not been assimilated or accepted by the person, although the precipitating event is often a physical illness. In this model, an attack of food poisoning could trigger IBS, or a viral illness could trigger fibromyalgia, but only in individuals already hypervigilant from previous trauma.

BRAIN–BODY (MIND) STATES

The vicious cycle of hypervigilance, excessive risk perception, and sympathetic autonomic activity is dependent on continuing fear signals feeding back from the body—nausea, tense muscles, rapid pulse. If these are absent, if the physical body is in equilibrium, the brain also reverts to a state of calm autonomic equilibrium and perception becomes balanced again (for an explanation of the primacy of bodily feedback see Damasio, 1994; Craig, 2008; Allman et al., 2010). This is not surprising if the perception of risk relies on autonomic feedback from the viscera. There is thus the potential for bodily feedback to act to *reduce* the state of hypervigilance by increasing positive autonomic feedback, as elucidated in Brown and Gerbarg (2005). Heart rate variability (HRV)—the level of variability of the heart rate due to respiratory sinus arrhythmia (RSA)—is a measure of parasympathetic activity and is inversely related to depression (Aizenberg et al., 2004). Breathing at six breaths a minute (associated with praying and meditation) maximizes HRV (Brown & Gerbarg, 2005).

The authors have personally observed in a study (in preparation) treating IBS with autonomic feedback, using real-time visual feedback of HRV, that after two minutes of slow diaphragmatic breathing there is a change in the signals *from the brain* via the autonomic nervous system. This is demonstrated by a decrease in the threat-based autonomic 'sympathetic' signal to the body and an increase in the calming vagal 'parasympathetic' efferent signal which cyclically inhibits the baseline rate of the sino-atrial node and increasing RSA. There is good evidence to suggest this change is partly due to autonomic visceral afferents.

POST-CARTESIAN LOGIC AND REASON

Damasio's research, particularly on gambling (Bechara et al., 1997), shows that in situations where normal undamaged people believe they are acting logically and rationally (as the normal subjects did) they are actually using the emotion of fear *expressed in their bodies* (sympathetic

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nervous activity) to weigh up alternatives, unconsciously incorporating emotional 'embodiment' with cognition. In this formulation something 'is' right because it 'feels' right. This may apply to such basic maths as 2 + 2 = 4; we know 5 is wrong because it feels wrong *in our bodies* (possibly the result of fear of social ostracism by classmates, teachers, and parents). Under hypnosis highly susceptible subjects can be made to make basic mathematical errors (negative hallucination of a number missing) which they would normally reject. If we assume the rejection is emotional, this fits with the dissociation of cognition and emotion in hypnosis described by Gruzelier (1998, 2006) and integrates this dissociation with the somatic marker hypothesis.

From LeDoux's work it is clear that emotions are initiated pre-consciously in the thalamus, but are *experienced* in the body, and thus, from Damasio's work, so are logic and reason. We describe something as rational because the integration of body and brain in the insula does not signal a mismatch between the emotional expectation and the autonomic bodily feedback, and therefore registers no error. When we think or speak 'rationally', it feels right to us *in our bodies*, not because of activity in some independent area of our brains which assesses 'levels' of rationality. Such an assessment will take into account our expectations which, in turn, depend on our perception (our memory) of risk and our level of bias.

In summary, in emotional distress and in MUS, pre-conscious threats are mostly related to past interpersonal relationships, abuse, bullying, neglect, or personal or carer distress, which will continually leave our bodies in a state of sympathetic autonomic excitation, feeding back to the brain and leaving us vulnerable to misinterpreting ambiguous signals, further affecting the body: a cycle of negative bias, a state of hypervigilance. Perceptions of threat *feel* logical and rational and so the misinterpretations seem real and cannot be changed by 'logical' argument.

POST-CBT PERSPECTIVE

DECENTRING AND MINDFULNESS

> *[A]n intervention primarily designed to increase metacognitive awareness by changing patients' relationship to negative thoughts and feelings without any attempt to change belief in the content of negative thoughts or underlying assumptions can significantly reduce relapse or recurrence in depression. (Teasdale et al., 2002: 285)*

From the late 1980s, the theoretical basis of 'dysfunctional thinking' as the mediator of vulnerability to, and recovery from, depression, has been inconsistent with studies on depressed people. An individual's dysfunctional attitudes score (DAS) was found to have poor predictive ability on the outcome or course of their depression (Longmore & Worrell, 2007). Instead, it was demonstrated in a number of studies that the mediating factor that most accurately predicts an individual's tendency to develop depression and relapse under stress is *overgeneral memory*, the inability to access specific memories (Williams & Dritschel, 1988; Williams, 1992; Brittlebank et al., 1993; Kuyken & Brewin, 1994; Hermanns et al., 2008). The exact cause of

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overgeneral memory is unknown. At present there are three possible theories: (1) loss of memory cells in the narrative memory circuits of the hippocampus (perhaps explained by excess cortisol reducing neurogenesis or new memory cell survival (Winocur et al., 2012); (2) an effect on the storage of memory—the interaction of cortisol and noradrenaline in hypervigilance will override and inhibit the normal narrative storage of memory (Myers & Greenwood-Van Meerveld, 2009); or (3) the strain on working memory from suppressing unpleasant memories/emotions; or a combination of any or all of these (Conway & Pleydell-Pearce, 2000; Williams et al., 2007). Suppression of unpleasant thoughts paradoxically leads to *increased* sympathetic activation (Gross, 2002) which, by increasing emotional expression in the body, increases negative bias, unconstructive thinking patterns, and access to negative memories (Dalgleish & Yiend, 2006).

Other studies of depression have found that differing ways of relating to your thoughts may affect depression. Watkins and Teasdale (2004) proposed that there are two ways of relating to your thoughts (or 'thinking style'): *analytical rumination*—being absorbed in your feelings (I'm depressed) and *decentred or experiential rumination*—'stepping back' outside yourself to observe thoughts and memories (I've noticed I'm feeling depressed). They discovered that this latter relationship (noticing feelings and tension) was very helpful to people who were or had been depressed, because only if you notice something can you let it go. There are many demonstrated beneficial effects of decentred thinking, such as reduced overgeneral memory and improved problem solving in depression (Watkins & Baracaia, 2002); reduced emotional vulnerability to failure (Moberley & Watkins, 2006); and a decrease in negative self-evaluation (e.g. I'm a failure) (Rimes & Watkins, 2005).

Mindfulness-based cognitive therapy (MBCT) was developed in the 1990s and comprises exercises in visualization and breathing, as in classical meditation practice. In this therapy there is no analytical logico-rational debate in which clients learn to relate to their thoughts and feelings from a different perspective. Teasdale and colleagues (2002) recognized the contribution of overgeneral memory to depression and observed that decentred thinking reduced this. They created a measure of decentring which they called *metacognitive monitoring*, and found that low levels of this measure predicted relapse while improvements in depression with MBCT treatment were correlated with higher levels of metacognitive monitoring. Interestingly, clinical improvement with traditional logico-rational CBT treatment was also correlated with an increase in metacognitive monitoring; by parsimony Teasdale et al. (2002) thus deduced that it was the change to decentred thinking that was the mediator and underlying mechanism for the beneficial effect and recovery with both CBT and MBCT. They thus proposed that in CBT it was not the *nature* of the therapy—the attempt to demonstrate dysfunctional thinking 'errors'—but the *coincidental* change of relationship to one's thoughts brought about by working with a CBT therapist that brought about recovery.

THINKING STYLES

Watkins (2008) examined 138 studies of the effects of decentred thinking and analytical ruminative 'thinking'. He proposed that:

1. Emotional dysfunction is underpinned by the inability, *under stress*, to swap between the analytical and the experiential mind/body state.

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2. Analytical rumination is beneficial when you are not challenged by stress; it is good to be absorbed in good feelings rather than to step away from them.
3. Analytical rumination is the default style for most of us most of the time, but under stress it is a hindrance, blocking the resources we need to resolve our situation.

He thus proposed that the timely adoption of experiential thinking under stress can break the vicious cycle of ruminative re-traumatization. A team at the University of Exeter have developed and tested a form of CBT based on this precept of swapping between the two states. This has enjoyed early success and initial signs are that it is more effective than standard logico-rational CBT (Watkins et al., 2011).

Fresco and colleagues (2007), including Teasdale, noted that decentred thinking is correlated with an increase in a change of perspective called *reappraisal*. Reappraisal is commonly employed in our everyday interactions with others and often crops up in conversation (e.g. one door closes another opens). It is a 'cognitive-linguistic strategy that alters the trajectory of emotional responses by reformulating the meaning of a situation' and involves 'early selection and implementation of a cognitive strategy that diminishes emotion without the need for sustained effort over time' (Goldin et al., 2007). It has been shown to be a most effective way of reducing the fight-or-flight effects on the body in a stressful situation (Gross, 2002). Accordingly, a situation that would otherwise cause fear from threatening events, for example an arm amputation, can be experienced calmly when instructions are given to see this as a life-saving operation. Similarly going to a party can be seen as an opportunity for friendship and fun rather than a test of personal worth. Over the long term, frequent employment of reappraisal leads to enhanced control of emotion, interpersonal functioning, and psychological and physical well-being.

Studies in the rat have also outlined an extinction pathway which ameliorates the fear response, running from the neurons in the ventromedial pre-frontal cortex (vmPFC, part of the ACC) to the amygdala where it stimulates intercalated inhibitory neurons (Quirk et al., 2003), diminishing the expression of fear in the body. In humans, for example, conditioned fear from a wasp sting, with repeated exposure to a conditioned stimulus (the wasp) without the unconditioned stimulus (the sting) leads to the gradual diminution of the fear (extinction). This pathway similarly involves the vmPFC–amygdala part of the inhibitory pathway as demonstrated by Gottfried and Dolan (2004). Activity in the dorso-lateral pre-frontal cortex (dlPFC), an evolutionary development of the frontal eye fields, is observed in imagining the manipulating of objects in three-dimensional space (visualization) and similarly in the manipulation of concepts and changes of abstract perspective typified by reappraisal (Ranganath & D'Esposito, 2005). Neural tracing in the macaque monkey has outlined a clear inhibitory pathway from the dlPFC to the amygdala via neurons in the vmPFC (Ghashghaei et al., 2007). In imaging studies this cortico-limbic emotional regulation pathway is seen to be activated by different cognitive strategies which dampen the expression of fear. These include mindfulness (Lutz et al., 2007), reappraisal (Oschner et al., 2002; Goldin et al., 2007), change of observer perspective (Lévesquea et al., 2007), hypnosis (Rainville et al., 2002), and probably many other therapeutic modalities as yet not examined by imaging for this effect. Delgado et al. (2008) hypothesize that the cognitive control of fear expression has piggybacked (phylogenetically) on the pre-existing vmPFC–amygdala extinction pathway. The idea of an 'overlapping set of

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prefrontal regions support[ing] the cognitive regulation of feelings and thoughts' (Oschner, 2002: 1222) is gaining credence from different areas of research:

> *The ventral ACC and mPFC might thus perform a generic negative emotion inhibitory function that can be recruited by other regions (e.g. dorsal ACC and mPFC and lateral PFC) when there is a need to suppress limbic reactivity. This would be a prime example of parsimonious use of a basic emotional circuitry, conserved between rodents and humans, for the purpose of higher-level cognitive functions; possible only in humans. (Etkin et al., 2011: 90)*

This inhibitory pathway is clearly important, but does not entirely explain certain observations from fMRI studies. There is an area in the ventro-lateral pre-frontal cortex (vlPFC) that accounts for 40% of the variability between individuals in the behavioural expression of conflict adaptation, and it is postulated that the dlPFC may be recruited only as required to ameliorate fear expression (Egner, 2011). It may be that some individuals are inherently able to access these cognitive dampening tools, while others need to practise and learn it to automatize the process; just as when learning new motor activity, extensive cortical activity in the practice phase is rapidly reduced to a few crucial synapses. In a study looking at the mediation of the effect of reappraisal in diminishing distress, Wager et al. (2008) found a secondary mediating pathway via the nucleus accumbens (where activity is associated with pleasure and reward), and postulated that the success of reappraisal may be partly related to positive emotional activation, rather than just negative amygdala suppression. This may lead eventually to an integrated understanding encompassing the ideas of Waugh et al. (2008) and Craig (2008). The authors are currently investigating the effects of hypnotically induced positive affect with McGill University.

NEUROBIOLOGY OF RECOVERY

Figure 2 illustrates the beneficial effects of reappraisal and decentred thinking, freeing the body from states of pre-conscious conditioned fear and hypervigilance, resulting in a calm, balanced, autonomic body state further reinforced by 'mindful' breathing and muscular relaxation which, in themselves, are 'experiential' forms of observation. We suggest this could be called the *experiential mind/body state*. This freeing can occur when dealing with a threat situation, thought, or feeling, and also fearful memories. We have observed that: (1) this experiential mind/body state can become automatic when learned through repeated practice and (2) it may also be considerably strengthened by positive affect. These factors result in a state of emotional stability, well-being, and resilience.

Studies of experiential processing have shown that detailed trauma memories are easier to access and are less overgeneral. We observe in our practice that when learning how to access the experiential mind/body state, individuals will sometimes describe spontaneously accessing specific details of emotionally charged memories, which previously were subsumed in an overgeneral memory, and re-experience them *without fear* from a third-person perspective. In this way the memory can become autonomous and provide powerful personal coping resources, incorporated into a personal narrative.

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This is particularly true in terms of childhood experiences of trauma, neglect, or abuse. Survivors of such incidents often blame themselves for what happened (van der Kolk & van der Hart, 1989); 'when a source of stability becomes a source of danger children attempt to re-establish some sense of safety by blaming themselves' (Frederick & Phillips, 1995: 252). However, the non self-evaluative stance of the experiential perspective frees them from such potential self-loathing. Adopting the experiential mind/body state then can be a learnt technique which can underpin recovery and resilience . Recent studies have signposted a way to achieve much more effective extinction than previous desensitization techniques using retrieval and extinction within a time window for reconsolidation (Monfils et al., 2009; Schiller et al., 2010). This should give rise to novel methods of psychological therapies, and we are following this idea with our theoretical and clinical formulation. In summary, the development of depression is mediated by overgeneral memory and the inability to access specific memories. The experiential mind/body state improves access to specific memories, and these memories become internal resources which help to resolve present difficulties and challenges. Therapies which increase the ability to swap from a ruminative state to an experiential state will therefore build individual coping and aid recovery from emotional distress. (Figure 2)

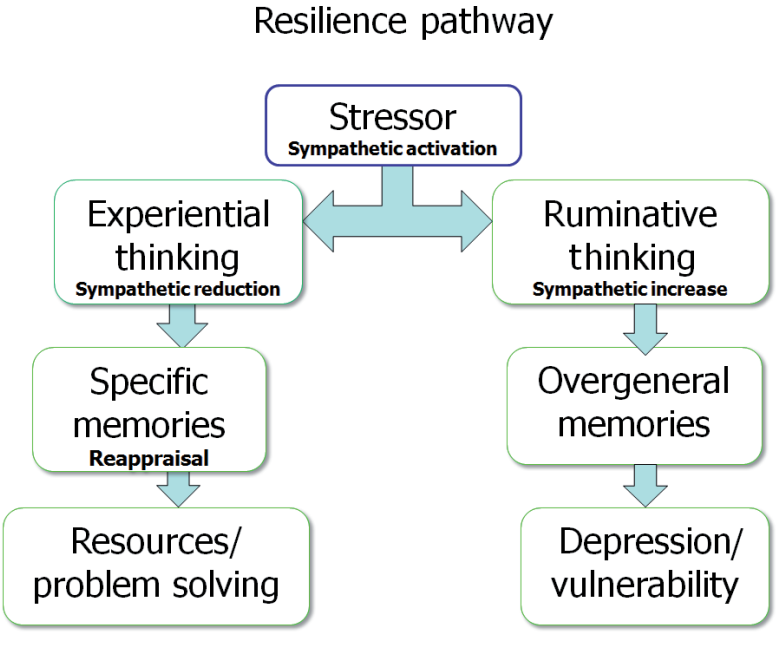

Figure 2. The resilience-recovery pathway

Hypervigilance, as manifested by emotional distress and MUS, is associated with overactivity of the amygdala and insula, resulting in sympathetic autonomic imbalance. The key factor in recovery is to find out how to reduce the link between such imbalance and cognition, thus

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preventing re-traumatization. The experiential mind/body state and positive reappraisal both involve similar neurobiological pathways which are easily automatized (Dobbin et al., 2009).

Further confirmation of this pathway, whereby experiential thinking affects the body, is suggested by the study of the neurophysiology of hypnosis. It has been proposed that hypnosis has elements of mindfulness (Wagstaff et al., 2004), and we have previously outlined a theory (Dobbin et al., 2009) that the hypnotic trance invokes an effective experiential mind/body state in which the subject becomes an external observer of physical and psychic events.

Hypnosis has been shown to change the response to fear conditioning, reducing the effects on the body as demonstrated by reducing sweating (Gruzelier 1998, 2006). Gruzelier described an 'executive' uncoupling *in the brain* of the cognitive and emotional processes, involving the vmPFC/ACC. During event-related potential recording he observed the specific loss of the error evaluation wave (Pe wave) in hypnotized individuals. The size of this wave is positively correlated with levels of subsequent sympathetic activity in response to conflict as measured by skin conductance, which represents the level of conflict generated by the error (Hajcak et al., 2004). In accordance with Allman et al.'s (2010) hypothesis, the insula is the source for this wave which is switched off by hypnosis. Rainville et al. (2002) observed that the process of hypnosis causes an increase in dlPFC activity and a decrease in right insula activity (potentially reducing sympathetic output). As a result of this executive dissociation, described by Gruzelier (1998), we suggest that depressed and traumatized individuals are able to view past events and rehearse future events without habitual fearful reactions, internal conflict, or negative self-evaluation.

Hypnosis and meditation simultaneously reinforce the other aspects of bodily relaxation and vagal stimulation. For instance, hypnosis as therapy invariably uses a calm voice, slow breathing (causing matching of breathing pattern by the patient), and suggestions of external noticing of muscular relaxation. Meditation uses recitations with breathing which also slow respiration. Hypnosis also offers the potential to stimulate the positive affect pathway—offering a system of 'mindfulness plus' by combining two pathways (positive affect *and* sympathetic decrease) to maximize parasympathetic output. This has multiple benefits on creativity, flexibility, sociability, and sense of inclusion (Isen & Labroo, 2003), widening the attentional field (Waldinger & Isaacowitz, 2006; Labroo & Patrick, 2009) and boosting emotional resilience.

FUTURE TRENDS

The discovery of the beneficial effects of mindfulness-based therapies and decentred thinking, alongside the recognition of the experiential nature of CBT and the establishment of the monistic nature of empirical neuroscience, all favour a neuro-physiological mind/body 'state' model of recovery, supporting a common underlying mechanism of techniques such as mindfulness, hypnosis, autogenic training, and others. The 'therapist effect' (Safran & Segal, 1990) has been shown to contribute substantially to the effects of all psychological therapies (Blatt et al., 1996; Zuroff et al., 2000). If this effect is the main component of the effects of any therapy it makes sense to explore any avenue that might maximize this effect.

The experiential mind/body state can be brought about with the practice of mindfulness but, based on our observations, *this is not necessary*. The beneficial changes Watkins (2008) described were brought about in most cases not with the full practice of mindfulness but instead with very simple exercises. He recognized that mindfulness brings with it other aspects such

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as acceptance and commitment which are separate from the experiential state. We would suggest that these attributes will follow on *automatically* from the physiology of the experiential mind/body state (autonomic sympathetic amelioration). In this context, 'acceptance' is the ability to experience memories in an experiential mind/body state without sympathetic activation, negative self-evaluation, or fearful emotions attached. We further suggest that acceptance, reappraisal, and enlightenment are all processes of cognitive adjustment to the physiological changes of the experiential mind/body state.

Equally, and encouragingly, we have found that the switch to the experiential state, described by some researchers as 'effortful', is in fact effortless, if rendered automatic by practice. Indeed, the innate automatic ability to do this, we propose, is what makes some individuals resilient; in others we observe it can be simply learned.

CONCLUSION

Dualism is intuitive to human observation. It is counterintuitive to believe that our minds are based on shifting emotional states rather than sound logic and reason; in the same way as it is counterintuitive to believe that the earth is spinning round at 1,000 mph and circling the sun at incredible speed when it seems solid and fixed. The discovery of the common mediator of MBCT and logico-rational CBT by Teasdale et al. (2002) has moved the goalposts in this debate, so that research on the process of CBT is now supporting state-based recovery. The schism between mind and body (analytical and physiological) treatments for emotional distress has come full circle, via mindfulness, back to a psychophysiological 'state'-based model.

It is our argument then that the pathway to recovery from emotional distress and medically unexplained symptoms is through enabling a 'swap' to the experiential mind/body state, which will facilitate, through autonomic rebalancing and extinction, both reappraisal and the transformation of overgeneral traumatic memories to autonomous specific memories from which solutions and positive emotions will come (Figure 2). We suggest that ruminative and experiential thinking styles are not solely mental states, but are mind/body states in which the expression of emotion is seamlessly and inseparably integrated in the mind and body. We suggest that well-being, a function of resilience, results from automatic access to the experiential mind/body state. As such, emotional distress represents an imbalance in mind/body processes and recovery will result from the facilitation of timely access to this state.

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