

The role of compassion in recovery

A neurobiological account

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I worked in London and Australia before becoming a full time GP in Edinburgh where I became interested in hypnotherapy. Along with Edinburgh University and Imperial College London I researched the impact clinical hypnotherapy had on patient health in primary care, from which I developed the idea of using positive mental training to treat depression. Since then I have conducted research into positive mental training in a general practice setting and am currently researching the effects of positive reappraisal with McGill University, Montreal.

Summary

Compassion has many neurobiological correlates.

Here we examine the different neurobiological pathways involved in compassion and how these influence and promote recovery.

'You can search throughout the entire universe for someone who is more deserving of your love and affection than you are yourself, and that person is not to be found anywhere. You yourself, as much as anybody in the entire universe deserve your love and affection.'

Buddha

Introduction

I once met a patient – aged over 60 – who had knee pain. She went for an X-ray, which not surprisingly revealed mild osteoarthritis, and, not wishing to wait for NHS treatment she went to a private specialist who did a knee operation which relieved her pain. She was very pleased, but six weeks later she returned to see me because the pain had come back. I wondered if I had been looking at her pain from the wrong perspective. So we started talking about her general stresses, and I asked her if anything significant had happened recently. She told me that her mother had just died.

My own mother had died two years before. I talked about my own bereavement problems and my difficult relationship with her; how I had felt that I hated her, and how eventually I had come to feel that this

was OK because I loved her as well. But before coming to this more compassionate (to myself) conclusion I had, for a time, hated myself for hating her; with all sorts of repercussions. My patient then told me about how when she was six her mother had once kept her home from school to help with the housework, and how she had been made to kneel and scrub the front step. When she missed an area her mother beat her hands with a cane until they were bleeding. She cried quietly as she told me this. I saw her sometimes after this. Her knee pain went away and did not return.

Later, I was at a four-day workshop with John Watkins, who together with Helen Watkins developed the field of ego-state therapy¹ and I told him the story of this lady. John, who was 92 at the time, suggested to me that I had been 'resonating' with my patient. I realised that 'mirror neurones' (which had been discovered shortly before) were a possible neurobiological explanation for my patient's pain having chimed with my own emotional experience.

Anatomy

The brain can be viewed as a biological instrument for assimilation, reflection

and action (see figure 1). In simple terms the back of the brain behind the central sulcus collates all the information from our senses, the front of the brain analyses this in relation to our needs, decides our priorities, and then designs and executes actions to achieve them. Our emotions are the mental fuel that drive our actions. At the very front of the brain the frontal lobes collate the information with our needs and priorities and at the 'back of the front' is the motor cortex whence the individual neurons cause the contraction of our voluntary muscles. The area in between the frontal lobes and the motor cortex is the pre-motor cortex (PMC), which processes the decisions and prepares the movement. The brain can be seen as an instrument of Bayesian logic: relying on past experience, the brain predicts what will happen next. It observes what is going on and continually modifies future predictions driven by the mismatch between expectation and observations.²

Mirror motor neurons

Until the 1990s the function of the PMC was known about only through experimental stimulation of patients' brains during surgery. It appeared to cause co-ordinated movements. Techniques were developed which allowed the recording of electrical activity in single brain cells. A neuroscientist in Milan, Giacomo Rizzolatti, worked extensively in this field and, by implanting electrodes in PMC neurons, his team demonstrated that individual pre-motor cortex neurons do not, as was assumed, code for muscle groups, but for whole patterns of co-ordinated goal-directed movements. They discovered for example that while one PMC neuron might provoke a monkey to put a piece of apple in its mouth, another PMC neuron would cause it to scratch. Even though both movements

involved many of the same muscles, the goal was different and could be triggered wholesale by a specific PMC neurone.

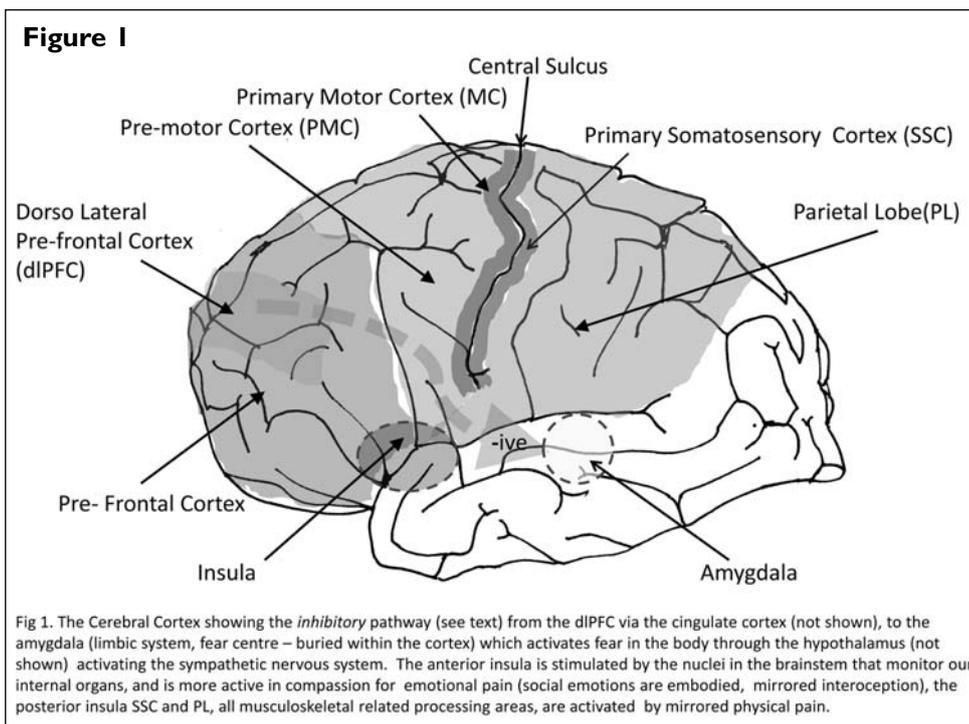
Then one day in 1992 Rizzolatti was filming his latest experiment which involved isolating the PMC neurons that made a macaque monkey grasp a piece of apple. Every time the monkey did this a buzzer linked to the neurone would sound. Rizzolatti's assistant Fogassi, who was at the other end of the laboratory where the monkey could see him, happened to be eating an apple. Quite unexpectedly, whenever Fogassi picked up a piece of apple he heard a buzz! It seemed the same PMC neuron that caused the monkey to hold a piece of apple was responding to Fogassi's own apple-eating movements.

This was the first time a neuron in any motor area had been shown to have sensory input, so because the cell responded to a visual stimulus they were called visuo-motor cell.³ In 1997 Alvin Goldman an expert in 'theories of mind' (ToM) was attending one of Rizzolatti's lectures. ToMs seek explanations of how we predict other peoples' intentions, and Goldman immediately saw the significance of these visuo-motor 'mirror neurons' (MNs). He suggested that by 'acting out' in our own minds and bodies the intentions of others, we gain insight into their mindset and so are more able to predict their intentions, because when we observe someone making a movement, our own corresponding muscles are 'primed' for action.

We now know that these processes are under the control of PMC neurons⁴ and that the person observing does not actually move because the final action pathway is prevented by an inhibitory network. But sometimes such mirrored movements do 'break through', as we have all experienced for example when we are sitting at a meeting table, and someone is absent-mindedly drumming their fingers during a boring speech, our own fingers may start

moving too, without any conscious awareness of this happening; or when we see a child struggling to untie a knot and our own fingers fidget, as if 'itching' to help.

Goldman and Gallese's thinking led to the idea that these mirror neurons are part of a predictive network.⁵ There is of course a huge survival advantage in being able to predict the intentions of others. A 'Bayesian brain' would indeed be expected to include pathways that enabled predictions about what others were thinking, planning and doing.⁶ Since motor pathways were already present in the evolving brain, it did not need to create a totally new network; rather it



'piggybacked' this advantageous ability on to an existing network with a simple mechanism to distinguish 'self' and 'other'.

Subsequently, these visuo-motor cells were shown to respond to auditory cues:⁷ PMC neurons involved in walking for instance respond not only to watching someone walk, but also to the sound of another's footsteps. This means that we can understand the gestures and speech of others and 'feel' their feelings about what they are saying, their own emotional engagement (see affective pathways).

Sensory mirroring

The discovery of mirror neurons in the PMC soon led to the discovery of other areas in the brain which share similar vicarious stimulation arising from observing others. There are areas in the somatosensory cortices (where neurons respond to pain, touch and position) which show similar levels of activation when observing another's actions; first observed during grasping and manipulating objects.⁸ Indeed, areas in the parietal lobe (PL) that integrate sight, sound and other sensory information are a major source of such inputs into the PMC.

When viewing a film of someone breaking their leg, one-third of viewers actually say they felt the pain in their own leg, while two-thirds of them reported negative feelings but without experiencing any pain as such. In this experiment, functional magnetic resonance imaging (fMRI) showed that the subjects experiencing the mirrored pain had increased activity in their parietal sensory cortices.⁹

However, all this empathic mirror activity in the sensory cortex does not entirely explain compassion. For example, when viewing a clip of someone having a needle inserted in their hand, the sensory mirror neurons of non-compassionate individuals who have cold-hearted psychopathic traits appeared to be *more sensitive*.¹⁰ Psychopaths are often charming and highly manipulative; something only made possible by having a very advanced ability to mirror the mental states of others. By contrast people with autistic traits have abnormally insensitive mirror neuron systems, which might partly account for their greatly reduced empathic sensitivity to others.¹¹

The *expression* of emotional distress recruits many of the same pathways as those involved in registering physical pain. It seems that in evolutionary terms mammals 'piggybacked' the pathways for social distress on to the existing pain pathways: so we vocalise and behave in much the same way whether our pain is emotional or physical.¹² But there are significant differences in the neural pathways elicited by experiencing and observing pain and emotional distress, which come together in compassion. A study using stories designed to activate specific social emotions towards others showed there were some important differences. Compassion stories of both emotional distress and physical pain recruited brain-stem nuclei usually activated by bodily sensations, in turn activating the hypothalamus and finally the insula. The

insula while not experiencing pain gives it *significance*, the anterior insula (AI) monitors all internal signals from the viscera (internal organs, heart, stomach) the posterior insula (PI) from the muscles and the ligaments. But while empathy for physical pain preferentially activated networks which process and integrate muscular information (PI and PL), mirroring of emotional distress involved networks concerned with visceral bodily perception the AI and the rostral Anterior Cingulate Cortex (rACC).¹³ The insula is closely connected to the fear association centre, the amygdala (fig 1) which is why fear is embodied; experienced within our body as nausea, vomiting and a racing heart.

It seems we have two systems involved in experiencing pain – the somatosensory cortex which deals with position and type of pain, and the insula which deals with its emotional significance, its unpleasantness. In a recent review of the experience of watching another's pain, Keysers, Kaas and Gazzola noted that systems are activated depending on whether we see the part affected or not.¹⁴ If we *do not*, we recruit the pathways involved in general distress perception (notably the AI and the rACC). But if we do, we vigorously recruit our own somatosensory cortex as well. So, when we see a loved one cut their finger we experience a strong impulse to grab our own finger, mediated by the somatosensory cortex, but our perception of how unpleasant or aversive an experience depends on visual facial and auditory vocal cues recruiting the AI and the rACC. We can infer from fMRI studies such as those conducted by Keysers *et al*¹⁴ and Saarela *et al*¹⁵ that the key brain structure involved in compassionate resonance is the anterior insula, and that we feel compassion for others *in our own visceral organs*. A further study has established such mirroring of the affective component of another's distress, and also that our own memories are activated by the observation of another's pain.¹⁶

The affective pathway

The rACC is where our current stream of consciousness is linked to our emotional circuitry – particularly to our fearful associations from previous experiences, and to fearful conditioned stimuli stored in the amygdala (see figure 1). For instance if at some time we have been stung, we later associate seeing a wasp with pain; or if we associate a spoken mistake with a teacher's or parents' anger we might later hesitate to say something in a workplace meeting for fear of rebuke. Such linkings let us predict (but can also make us falsely pessimistic about) the impact of what we are thinking and planning. In such situations our amygdala has sent a pulse of sympathetic nervous activity in our bodies, and the consequent bodily response (sweating, rapid heart rate) informs the brain of possible negative consequences. Damasio found that patients with damage to the amygdala/insula/ACC area from stroke or tumour (most famously Phineas Gage), were very poor at gambling (not careful enough of negative outcomes) but were also unable to predict the

effect of their behaviour in social situations, and so behaved inappropriately.^{17, 18} This became the basis of the ‘somatic marker’ hypothesis that our mind/body links are the final pathway of compassion.¹⁷

Damasios’s patients, like Phineas Gage, were socially dysfunctional because they could not respond appropriately to others; could not predict the feeling of another’s emotional discomfort by feeling it in their own bodies – which is how normal people operate. So they were unable in this way to sense the distressing feelings or negative events they themselves *or others* might be going to suffer as a result of their own behaviour. The ‘somatic marker’ hypothesis suggests that the ability to predict the level of distress in others by experiencing it *through our own distributed brain-body networks and body-brain networks*, is what enables empathy and compassion,¹⁷ a neurobiological process that literally puts us in their shoes, so that at some level we are sharing their visceral feelings.¹⁹ One of Damasio’s patients who had lost the ACC/insula/amygdala link (Dave) in a recent TV series (*The brain: A secret history*) said ‘It’s almost how I imagine a serial killer would feel, not that I could ever become a serial killer, but I have the feeling...it doesn’t bother me’.

What can this tell us about the cultivation of compassion? Vissipana meditators have been shown to have increased cortical thickness in the right AI.²⁰ Expert compassion-based meditators show increased activity in their amygdala.²¹ The discovery that the size of the amygdala is directly related to the level of social play in primates²² and also to the size of humans’ social networks²³ – a key element in social and biological success – strongly validates Damasio’s somatic marker hypothesis.

Solution mirroring: Compassion and emotional distress

Everyone experiences episodes of emotional distress at some time in their life. And psychologists name many ways of dealing with them – strategies such as distraction, avoidance and suppression are all too commonly employed as ‘solutions’. However there are other more resilient options, and they are of great importance.

Suppression is a problematic strategy. Because, having lived for millions of years in social groups, alert for danger, the human visual and auditory systems are very finely attuned to signals of alarm and distress. So while listening to someone and watching them speak, we are also sensing their bodily cues and mirroring their emotional state. This is a two-way process. Our patients are doing it at the same time that we are, so they sense and respond to our emotions even if we are suppressing them. They know whether we are stressed, fatigued or activated by our negative feelings toward them. And, as a result of what they sense they will be more or less likely to engage (eg with our advice) or *feel* that we can help them.

Reappraisal is a core concept in Buddhist philosophy. But it is also an important and commonly employed

strategy in our everyday interactions with others, and one that often crops up in conversation – for example ‘one door closes; another opens’. Goldin, McRae, Ramel and Gross²⁴ categorised such thoughts as a ‘cognitive-linguistic strategies that alter the trajectory of emotional responses by reformulating the meaning of a situation’ and entailing ‘early selection and implementation of a cognitive strategy that is efficient because it diminishes emotion without the need for sustained effort over time’ (pp577). The suppression of emotional reaction to images of amputation produced significant increases in cardiac reactivity²⁵ which was not seen when participants were told to think of it as a life saving operation.

A similar fMRI study showed a pattern suggesting that frontal lobe activity was suppressing the amygdala.²⁶ Women tasked to discuss a film with a listening partner were secretly told to suppress or reappraise their feelings. Extraordinarily, when *they* suppressed emotion, the blood pressure of the *listener* increased. It was observed that the suppressors showed less positive animation when they did this, so it can be assumed that changes in their voice and gesture communicated themselves pre-consciously, and so triggered a mirrored activation of the listeners’ insula.²⁷ This in turn caused autonomic arousal in the listener, which raised their BP. We are beginning to discover the mind/body pathways for such interpersonal, pre-conscious sharing of visceral emotional and physical distress.

Various fields of research support the idea that there is an ‘overlapping set of prefrontal regions support the cognitive regulation of feelings and thoughts’.²⁶ (pp1222). The dorso-lateral pre-frontal cortex (dlPFC) is a phylogenetic development of the frontal eye fields. Consequently, when someone is imagining and manipulating objects in 3-dimensional space (visualisation) or even just ‘manipulating’ concepts and changing abstract ‘perspective’ (as happens during ‘reappraisal’) there is a surge in activity in this area of the brain. A number of neurobiological imaging studies have outlined an **inhibitory pathway** which ameliorates the fear response. It runs from this dorsolateral zone to an area called the ventromedial pre-frontal cortex (vmPFC, which is part of the rACC) and thence to the amygdala. It seems this pathway by inhibiting the fear centre diminishes the expression of fear in the body (see figure 1). And we know too that this dlPFC and amygdala pathway also is less active in people who are depressed rather than it is in those who have never been depressed.²⁸ Moreover, this same cortico-thalamic pathway can be activated by different cognitive strategies, including mindfulness,²⁹ reappraisal,^{24, 26} change of observer perspective³⁰ and hypnosis.³¹ It is probably activated by many other therapeutic modalities which have not yet been examined for this effect. So here we have evidence of an important fear-reducing brain pathway that can be significantly activated by psychotherapy.

Desensitisation is part of a normal process whereby fear (ie of a wasp) is diminished; therapists utilise this

process to help those whose fear has become a problem. Repeated exposure to the conditioned stimulus (the wasp) without the unconditioned stimulus (the pain of the sting) leads to the gradual reduction of fear; Pavlov called this extinction, and it is present in all animals. We now know that this process involves the vmPFC – amygdala part of the **inhibitory pathway** (see above), and Delgado *et al*³² have suggested that this *human* cognitive control has piggybacked on this pre-existing *animal* pathway. Research into mindfulness-based cognitive therapy (MBCT) seems to show that This same pathway may underlie the distress-relieving effect of standard CBT in depression; that the experience of therapy leads to the client ‘stepping outside’ themselves (experiential thinking) sharing with a therapist their experiences and feelings from an external perspective. Arguably, this is the essence of mindfulness practice, and therefore it may well be this cognitive shift that mediates recovery rather than CBT’s ability to change dysfunctional beliefs. Indeed this pathway may be even more universal, if as some have said, it is the basis of the ‘therapist effect’ itself.³³ If so we may be nearer to a neurobiological explanation for why the non-specific affects of the ‘therapeutic alliance’ (ie interpersonal rapport) is five to seven times more effective than the specific techniques of any one particular type of therapy.

On the other hand, the same pathways should make us take very seriously the fact that interpersonal sharing of distress and physical pain has biological foundations mediated through the insula and amygdala and on into the autonomic nervous system. The shadow side of practitioner-client visceral-emotional resonance is its cumulative impact on healthcare workers. It results in defensive cynicism, exhaustion and that loss of sense of personal efficacy we know as the burnout triad. So this is our predicament: that what we need for effective social emotional interaction is a responsive amygdala, but what we need too, if we are constantly exposed to other peoples distress, is to have mechanisms for ameliorating the effects of that shared visceral/emotional compassion. The evidence from meditation studies suggests that it can produce a sort of cortico-limbic calming which may allow us to resonate and empathise while simultaneously protecting ourselves from the ill effects of emotional ‘cross-infection’ and exhaustion.

Some compassionate healthcare workers may already have effective ways of dealing compassionately with this predicament, but if they don’t, they can still develop it through practice, as a recent study conducted in the Scottish health service showed. A 12-week course of combined techniques based on these principles significantly improved a whole range of burnout measures and produced a fall in referrals to occupational health for mental stress.³⁴ So it seems GPs, nurses and therapists by understanding the cortico-limbic pathway and how this modulates their own distress, can reflect and transfer such understanding to patients through the emotional flow of their speech and gestures. If the therapist is rationally and emotionally able to recommend recovery pathways to

someone experiencing distress, this authenticity will be signalled through such flow. When this happens a therapeutically useful situation can come about, whereby the two parties mirror one another, and the distressed party will have an opportunity to resonate with the recovery pathway evoked by the client-therapist interaction. A brain-body pathway which as I hope I have shown, is underpinned by a neural resonance in the insula/amygdala *of both parties*. At the same time the therapist - by having the solution embedded in their own experience and learning - is protected from being overwhelmed by their compassion for the patient/client. This I believe forms a useful neurobiological basis for psychologically-minded practice.

I have described motor ‘goal-oriented’ mirroring, sensory mirroring, and emotional/affective mirroring. To these I would suggest we can now add ‘**solution mirroring**’, in which the goal is healing or recovery. This builds on the principles laid out in the concept of ‘social intuition’.³⁵ For example, had I not personally found a way of resolving my own bereavement distress, and understood both cognitively and emotionally my own ambivalent and complex relationship with my mother, I could not have ‘explained’ it (logically, but also in an embodied, emotionally articulate way) to my patient. Critically, had we not empathically mirrored this between us, there would have been no real engagement. In order to *allow* herself to alleviate her distress, my bereaved patient needed to *feel* that my experience and its resolution were authentic. This made it possible for her to engage *emotionally* with my cognitive understanding of her distress. I believe this sort of solution mirroring can not only help our patients escape from cycles of re-traumatisation, but that it may also protect us from compassion fatigue and burnout.

Conclusion

The pathways for empathy and compassion fall into two categories. One is based on sensing general levels of distress from facial expressions, and the patient’s voice and movements, all of which stimulate our own insula to mirror the other person’s. And so we share in common a distressing visceral emotional experience. The other is based on positional and modality information in our own sensory cortex. The former is now generally accepted as the main pathway for real empathy and compassion.

The quote from Buddha at the start of this piece is a tangential reference to compassion. And indeed neuroscience appears to confirm its basic premise that in order to be compassionate to others we need to have some solution for the client’s/patient’s distress that is authentically grounded in a cognitive *and emotional* understanding that we can mirror back to them. For this to happen, such a solution has to be intellectually and emotionally available to ourselves. If it is, then reappraisal or a shift to engagement will permit a calming of the client’s *and* the therapist’s amygdala fear centre. If not, our clients/patients will quite directly assess the authenticity of our solution by mirroring our distressed

mind/body state; they will experience instead our cognitive dissonance; they will know we can ‘talk the talk’ but not ‘walk the walk’.

We have developed and tested a 12-week supervised self-help programme for relieving emotional distress called Positive Mental Training. A critical part of the programme is the engagement of GPs/nurses and therapists in the understanding of the pathways of recovery, of embodiment and compassion. With over 50,000 patients treated and 650 NHS staff attending workshops, we have found a high proportion of those attending our workshops use the programme for their own benefit. The intervention, is based on the neuroscientific principles of adopting an experiential viewpoint under stress (mindful practice) while priming patients pre-consciously for positive re-appraisal. The programme significantly improves a range of mental health measurements, and is effective in depression and burnout of patients and NHS staff.^{34, 36}

Across Europe only a third of those in distress are treated, despite well-intentioned healthcare.³⁷ If we are serious about reducing distress across society every health worker needs to be able to access, understand, and deliver the neurobiological principles of empathy and compassion.

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