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An Explanation of Experienced and Empathic Pain

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Allometry is commonly used in both biology and engineering, but is significantly less familiar in neuroscience. This is so although the approach involves a few new ideas, it leads to vastly simpler, and more transparent interpretation of experienced and empathic pain than those provided by traditional explanation found in literature.

Keywords: pain, metabolic rate, allometric scaling

Singer *et al.* provided a new brain-imaging explain when we sympathize with another person's pain, we use many of the same brain areas that are activated by our own experience of pain[1]; Wager et al. show that anticipation of pain relief is closely tied to the placebo response[2]; Eisenberger et al. find that the brain bases of social pain are similar to those of physical pain[3]. The phenomenon is an intriguing and enduring problem, and we give an explanation of the phenomenon by comparing the metabolic activity of experienced pain with that of empathic pain or social exclusion pain in the "pain matrix" (bilateral anterior insula (AI), rostral anterior cingulate cortex (ACC), brainstem, and cerebellum).

Before explanation of the empathy for pain, we recapitulate the allometric scaling relationship for brain's metabolic rate. The overall cerebral metabolic rate for glucose, B_{brain}, can be expressed in the form[4]

$$B_{brain} \sim T_{brain}^{(3+N/6)/4} \tag{1}$$

where T_{brain} is its mass, N is cell's freedom of motion in the brain. For active brain, the cell's freedom is close to 3. Therefore, the prediction (1) yields $B_{brain} \sim T_{brain}^{0.87}$, which is in good agreement with experiment data[5].

For vegetative state patients, the cell's freedom of motion reduces up to zero, i.e. $N \to 0$. That means overall cerebral metabolic rates for glucose in vegetative state patients will be

massively reduced, this prediction is verified by positron emission tomography (PET) techniques[6]. In an extreme condition of a vegetative state patient, the cell's freedom of motion reduces to N=0, a global loss of neuronal function, and we predict a 32% decrease in brain metabolism for comatose patients[7], whereas the observed decrease[6,8] is about 45%.

The traditional allometry is the relationship between overall metabolic rate and the whole-body mass[9~12], the allometric scaling laws for organs are less studied, He et al. suggested an allometry in the form $B_{organ} \sim T_{organ}^{(D+N/6)/(D+1)}$, where B_{organ} is the metabolic rate of an organ, T_{organ} its mass, D is the dimension of the studied organ. This prediction agrees quite well with the experiment data for brain, liver, heart, and kidneys [4,13-18].

For better explanation of the empathic pain, we consider allometric scaling for "pain matrix" at the brain's components level:

$$B_{painmatrix} \sim T_{painmatrix}^{(3+N/6)/4} \tag{2}$$

where $B_{painmatrix}$ is the metabolic rate of the pain-related areas such as the secondary somatosensory cortex(SII), insular regions, the anterior cingulated cortex(ACC), etc., $T_{painmatrix}$ is its mass, and N, a pain-related parameter, is the affective dimension of experienced or empathic pain. For most pain experience, we choose N=3, the maximal value of N is 6, a condition in the most torturous pain, and it becomes painless when $N \to 0$.

Empathy broadly refers to being able to understand what others feel, be it an emotion or a sensory state[1,3]. Empathic experience enables the loved partners to harmoniously and automatically stimulate the brain activity of both persons, one experienced a painful stimulus and the other in a particular emotional state observed the signal indicating, leading to autonomic and somatic responses. As a result, pain-related cells in the almost same brain areas are activated by our own experience of pain. A particular cell motion pattern in "pain matrix" reflects a particular pain experience activated before, this leads us to understanding what it feels like when someone else experiences sadness or happiness, and also pain, touch, or tickling[1].

The experience of pain arises from both physiological and psychological factors, including one's beliefs and expectations [2,3]. Theoretically, pain depends upon cell's motion patterns in the "pain matrix", consequently we conclude that the decrease of cell's freedom of motion, N in (2), in the "pain matrix" reduces pain expectations through reducing the

scaling exponent in (2), and it will become painless if the exponent reduces to 3/4, which is associated with the vegetative state. Thus, placebo treatments that have no intrinsic pharmacological effects may produce analgesia by altering value of N in (2). In two functional magnetic resonance imaging (fMRI) experiments, Wager *et al.* [2] found that placebo analgesia was related to decreased brain activity (i.e. decrease the value of N in (2)) in pain-sensitive brain regions, and was associated with increased activity during anticipation of pain in the prefrontal cortex(i.e. increase the value of N in (2)), providing evidence that placebos alter the value , N, of the cell's freedom of motion in "pain matrix".

Eisenberger et al.[3] illustrates increased activity in anterior cingulated cortex(ACC) and right ventral prefrontal cortex during exclusion relative to inclusion, this results in a higher value of N in (2) than that during inclusion, leading to a painful feeling experienced before.

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