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Complementarity, Brain~Mind, and Pain

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Acknowledging Complementarity: From Neuroscience to a Neurophilosophy of Pain

A neuroscientific definition of pain involves both the physiological event of the peripheral and central nervous systems, and the psychological phenomenon subtended by the hierarchical networks conjoining the internal and external environments impacting the body and brain, that give rise to conscious processes (i.e. the ‘mind’) [1]. This definition illustrates that one of the notable achievements of contemporary neuroscience has been to ‘... bring together ... knowledge by the linking of facts and fact-based theory across disciplines to create a common groundwork for explanation’, and in so doing, make inroads toward achieving what biologist E.O. Wilson has termed ‘consilience’ [2]. Such consilience involves intellectual fusion, both within the sciences, and between the sciences and the humanities, and has been instrumental in reconciling somewhat divergent perspectives, so as to allow an enhanced appreciation of complementarity – most simply defined as an interrelation of reciprocity whereby one thing (e.g. science) supplements or depends on the other (e.g. the humanities) [3]. On a somewhat deeper level, such complementarity speaks to the entirety that results when two opposite states or principles are brought together. In this last sense, complementarity refers to properties and/or characteristics that manifest in seemingly opposite or contradictory ways at different times, depending upon the conditions of observation [3]. In other words, complementarity entails a ‘both-and’, rather than an ‘either-or’ epistemic framework. Kelso and Engstrøm maintain that this reflects a change in an overall worldview that is part of a Kuhnian paradigm shift, for which we have only recently begun to develop ways of knowing, expressing and symbolizing [4]. To assist in bridging this explanatory gap, these authors have suggested that the tilde symbol (~) should represent complementarity, and consequently this symbol will be used throughout this essay to reflect these relationships. Thus, while perspectives may differ, the blending of these dis-

tinct orientations, and reconciliation of either/or dichotomies provides a basis for dialectical synthesis and intellectual progress. The rejection of Cartesian dualism reflects this complementary orientation, and this has been evidenced in a broader, yet more comprehensive approach to studying the brain, mind (i.e. brain~mind), and pain.

In this issue, Frauke Musial provides a thorough description of the neural mechanisms and pathways of pain [5]. Musial depicts the complicated structural and functional neuraxis through which pain – as physiological event of the peripheral and central nervous systems – is expressed and experienced as a psychological event, evoked by the co-activation of integrated neural networks that subserve cognition, emotion and behaviors. In this way, the complementarity that is inherent to pain becomes evident; as Musial notes, ‘... it is unlikely that these events can be separated from each other’. Explanation of the neural event of pain cannot ignore the experiential dimension(s), and description of pain as experiential phenomenon must account for the role of brain~mind in consciousness.

Such complementarity is fortified through contributions from various disciplines that have resolved prior peripheralist-centralist, and cell-reticularist distinctions, and have shown pain to involve both peripheral and central mechanisms, functioning as part(s) of a complex, networked hierarchy [6]. Similarly, older classifications of pain have given way to newer taxonomies that reflect the involvement of heterogeneous molecular and anatomical substrates, and which classify pain as sign and symptom, disease and illness, and in objective and subjective terms [7, 8].

Complementarity, Complexity, Brain~Mind, Self, and Pain

We have posited that pain may be considered as a spectrum disorder that reflects dynamical, complex-systems’ effects that

involve genetic elements that interact with particular environmental factors to express definable endo- and exo-phenotypes to produce particular pain syndromes [9–12]. These variations may establish bases for potential changes in neural function and structure that (1) de-construct non-linear, adaptive pain modulating networks, (2) incur progressive, aberrant linearity within pain transmitting pathways, and (3) produce alteration(s) at higher loci to affect multiple substrates of the core neural networks of conscious process(es) [13]. In this way, pain can alter the brain~mind, and ~self. Furthermore, such geno- and phenotypes may be related to the (co-)morbidity of other CNS disorders, and explain, at least in part, individual, familial and/or community patterns of susceptibility to chronic pain, as well as sensitivities to certain therapeutics.

But while it is tempting to depict ‘how’ and ‘where’ pain affects the brain, it is erroneous to assume that a given anatomical structure subserves a specific function, as this reflects the ‘mereological fallacy’ of attributing the function of the whole (i.e. the brain~mind) to a single part (i.e. an anatomical locus or structure) [14]. More appropriately, as Musial explains, it is the temporally and spatially patterned activity of hierarchical neural networks that actually produce the sensations, cognitions, emotions and behaviors of pain [15, 16]. A single site does not produce ‘pain’ per se, but rather the ‘feeling(s) of pain’ is/are evoked by the engagement of a myriad of microsystems within a complex, networked whole (i.e. the brain~mind). Thus the experience of pain (as any other) is potentially (and most likely) unique to each individual (i.e. the ‘self’). Given this uniqueness, the claim that ‘... no two pains are exactly alike’ relates a profound truth with regard to pain as (1) a phenomenal event, (2) a self-sustaining process within a (unique) nervous system, and ultimately (3) a manifest subjective illness.

Yet in many ways our study of pain has been somewhat constrained by the ‘hermeneutic circle’: to understand the parts requires an understanding of the whole, yet the whole cannot be understood without knowledge of its constituent parts [18]. As well, knowledge of pain is limited by problems of understanding and explanation: what is subjectively understood cannot be directly explained, and what can be explained does not directly reflect that which is subjectively understood [19]. Pain is subjective, and while we may have knowledge of the neural mechanisms, systems and networks that are involved in pain transmission, third-person assessment does not allow apprehension of what an other’s pain ‘feels like’, and hence the discernment, differentiation and diagnosis of particular pain syndromes is reliant, to a great extent, upon a complementary approach that meaningfully combines objective data with subjective information. We cannot state that pain is ‘either’ a neural event ‘or’ an event of consciousness, nor can we claim that we can understand how the neuraxis ‘creates’ pain without appreciating the complementarity of body~brain~mind (i.e. ‘bottom-up’) and mind~brain~body (i.e. ‘top-down’) systems that act in concert with environmental input(s).

Complementarity in, and of Pain Research and Clinical Therapeutics

Clearly, clinical issues focal to the treatment of pain should be the source and direction for pain research. But if research is to provide knowledge that informs clinical therapeutics, then *what* we learn about pain should direct *how* we study pain, and *how* pain studies are conducted should provide critical insight(s) to new types and domains of knowledge [20, 21]. If we consider pain to be a spectrum disorder, then we must recognize that contributory brain~mind functions can affect and be affected by internal and external environments, and therefore it is important to study these functions and effects across levels that range ‘... from synapses to social groups’ [22]. The methodologies utilized should optimize such inquiry, and must be valid, reliable, elucidative, applicable and effective [23]. In this light, it is critical to assess whether current research methods and protocols best address the problem of pain with respect to our contemporary knowledge of the complementarity of body, brain, and mind [24, 25].

Such knowledge provides the epistemic grounding of the philosophical and pragmatic understanding of both pain, and pain therapeutics. As Musial astutely states, we must investigate the ‘... neurobiological bases of pain ... from nociceptor to brain’, and I would add, the relationship of body~brain~mind, if neuroscience (and neurophilosophy) is to provide an explanation of pain that validates its mechanistic and phenomenal realities. This would allow heuristics to be wedded to a hermeneutic approach, enable explanatory constructs to relate to the existential dimensions of pain, and in this way would align clinically relevant scientific orientations to the subjective experience(s) of the pain patient. Musial’s assertion that ‘... chronic pain tends to (pre-)occupy the individual’... reinforces the need to apply these facts within the anthropologic, humanitarian, and ethical domains of a philosophy of pain and pain medicine. A meaningfully integrative pain medicine would be equally complementary, incorporating both curative and healing approaches to enable truly ‘patient-centered’ care [26].

For once we acknowledge pain – as neural mechanism, psychological event, and phenomenal experience that occurs in a unique sentient being – it becomes our incumbent obligation to develop improved research methodologies that more completely elucidate the multiple dimensions of pain, and utilize the knowledge gained from these studies to implement the most technically effective, and ethically sound clinical therapeutics.

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