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The relevance of neuroscientific research for understanding clinical reasoning

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The relevance of neuroscientific research for understanding clinical reasoning

The current debate on clinical reasoning revolves primarily around Dual-Process Theory. This theory suggests that there are two distinctively separate cognitive systems underlying thinking and reasoning; commonly referred to as System 1 and System 2.1 System 1 is considered intuitive, fast and reliant on automatic activation of "illness scripts" stored in memory and leading to effortless pattern recognition. System 2 on the other hand is considered analytic, slow, deliberate, and systematic. The clinical reasoning literature is divided; one group of researchers defending System 1 reasoning as the hallmark of expert decision-making, whereas the other camp of researchers considers System 2 reasoning as superior and more likely to achieve diagnostic accuracy.^{2,3} Some also argue that System 2 is less prone to biases (premature closure, confirmation bias etc.).⁴

Although researchers in both camps acknowledge that System 1 and 2 reasoning processes are intertwined and overlap, in the end, the preference for one side appears to take prevalence and data are projected against their preferred System 1 or 2 background.⁵ There is a chance that this practice may result in a deadlock in which little progress is made in advancing our understanding of clinical reasoning other than trying to convince the opponents of the superiority of their findings.

Perhaps it is time to take a step back and re-examine the very premise of the argument. The premise is that there are two distinct reasoning systems in the brain. But what if there are not? What if there are more than two reasoning systems? Alternatively, what if there is only one system that relies on the same cognitive pathways but at different intensities, such as changing gears from automatic to controlled, from low effort to high effort, from implicit to explicit, from associative to rule-based etc.

This raises the question; what empirical evidence is there to support the dichotomy? One has to note that System 1 and 2 found a relatively recent introduction to the field of clinical reasoning and has a longer history in social psychology, philosophy, cognitive psychology, and economics. It is therefore that the bulk of evidence for Dual-Process Theory emanated from these disciplines. In addition, most of the research on this distinction is behavioural, and thus one has to infer the underlying cognitive brain processes from observational data.

For instance, a well-established line of research concerns the "belief-bias effect" in syllogistic reasoning tasks. Belief bias refers to the tendency of people to be more likely to accept the conclusion of a syllogism if they find it believable than if they disbelieve it, irrespective of its actual logical validity.6 A believable syllogism is "no mammals are birds and all dogs are mammals, therefore no dogs are birds." An example of an unbelievable conclusion of a syllogism is "no mammals are birds and all pigeons are mammals, therefore no pigeons are birds." It is argued that this belief bias results from the competition between System 1 and 2 reasoning when evaluating such arguments: Since we know that pigeons are birds we reject the latter (in itself correct) syllogism. De Neys manipulated working memory capacity during syllogistic reasoning.7 He did this by introducing a second-distractor-task. The assumption is that having to engage in two tasks at the same time makes reasoning more difficult. Results suggest that when a correct response could be produced by System 1 reasoning, a distractor task did not influence that correct response. Subsequently, belief bias was introduced by presenting an unbelievable syllogism, which resulted in reduced performance when the distractor task was initiated. These outcomes support the notion that System 1 is indeed automatic and is not dependent on the limited size of working memory, while System 2 is.

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A more direct manner to test Dual-Process Theory is to examine whether there are two autonomous regions in the brain that act independently. That would imply looking at the brain itself when physicians engage in clinical reasoning. It is then possible to directly examine if anatomically distinct parts of the brain are associated with the two distinct reasoning modes. With the availability of functional Magnetic Resonance Imaging (fMRI) and other scanning hardware, neuroscientific research seems to come into closer reach to researchers. This emerging trend is visible in the current literature on reasoning in which increasingly reference is made to neuroscientific evidence in support of the distinction between System 1 and 2. However, as with the behavioural studies cited above, "neuroscientific evidence" is produced in studies of syllogistic reasoning, rather than clinical reasoning. For instance, Tsujii and colleagues utilised Near Infra-red Spectroscopy (NIRS) while conducting belief-bias experiments.⁶ They found that only in the case of unbelievable syllogisms, the right inferior frontal cortex (IFC) was activated, an anatomical region associated with analytical reasoning (i.e., System 2).8-10

The critical question is whether these findings can a priori be translated to the field of clinical reasoning. Generic syllogism tasks do not require specialized knowledge. However, a characteristic of clinical reasoning is that large amounts of previously acquired knowledge must be used in order to solve clinical cases. In medicine, when trying to make sense of signs and symptoms in order to arrive at a correct diagnosis, physicians employ knowledge acquired through learning and experience. Therefore, one cannot exclude the possibility that clinical reasoning is qualitatively different from syllogistic reasoning.

To address this issue of ecological validity, it appears timely to conduct neuroscientific studies that investigate the complexity of clinical reasoning, through direct imaging of the brain. That is not an easy task to accomplish. How to present a clinical case with all its complexity? New experimental materials and procedures are needed that can simulate the complexity of clinical reasoning. Once we succeed, however, these data will help to clarify the status of Dual-Process Theory in the field of clinical reasoning and whether it is justified to think of it as a dichotomy between System 1 and System 2.

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