Explaining delusions of control: the comparator model 20 years on

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Many of the symptoms of schizophrenia, particularly those described by Schneider (Schneider, 1957) as first rank, seem to reflect a confusion between changes in sensation caused by actions of the self and changes with external causes. Examples include, *hearing thoughts spoken aloud, thought echo, made emotions, somatic passivity experiences,* and *delusions of control.* In all these cases the patient is attributing effects generated by the self to external forces.

Perhaps we should experience surprise, not at the strange ideas of the patients, but at the ease with which the rest of us make the distinction between self and other generated sensation. This was Helmholtz' concern (Helmholtz, 1866): When an image moves across our retina, how do we know whether this is the world moving across our eyes, or our eyes moving across the world? His answer was that an active movement, moving the eyes across the world, will be preceded by a motor command, which does not occur when the world moves in front of our eyes. Information about this motor command, subsequently termed *corollary discharge* or *reafference copy,* can be used to predict the sensory consequences of the eye movement. The predicted and

observed outcomes of the movement can be compared. If they match, then the changes were self generated.

Given this account, it seems plausible that first rank symptoms, such as delusions of control, might occur because something goes wrong with this comparator mechanism (Feinberg, 1978; Frith, 1987). Since this first suggestion, the comparator model has become far more sophisticated and has been supported by empirical studies (e.g. Frith & Done, 1989).

The first increase in the sophistication of the model came from the development of engineering approaches to the study of motor control (e.g. Miall & Wolpert, 1996). In order to overcome the inherent delays in feedback signals associated with movement, the system needs to generate a *forward model*. The forward model predicts the consequences of implementing motor commands in terms of both kinematics (where and when our hand will arrive) and in terms of sensations (what it will feel like). If we can predict where our hand is going to be after the motor command has been implemented, then we can check whether we have chosen the correct command before we actually make the movement. If we can predict the sensations we are going to feel, then they are unimportant and can be attenuated. Experiments suggest that patients with delusions of control have problems with both the kinematic and sensory aspects of the forward model (Blakemore, Smith, Steel, Johnstone, & Frith, 2000; Franck et al., 2001; Lindner, Thier, Kircher, Haarmeier, & Leube, 2005). But the precise nature of these problems remain to be specified.

Recently Synofzik and colleagues (2010) have shown that the problem lies in the precision of the prediction of the sensory consequences of action: the less the precision the greater the severity of delusions of control. As a result of this lack of precision, patients rely more strongly on external cues for agency, leading to better than normal adaptation to external feedback.

One problem with the proposal that patients with delusions of control fail to compute a forward model is that such a failure would be associated with major problems in motor control. This is clearly not evident in the every-day behaviour of patients with delusions of control. Indeed, there is evidence that the automatic predictions that occur well below awareness are intact in patients with delusions of control (Delevoye-Turrell, Giersch, & Danion, 2002). The problems arise at a higher level, in particular in relation to agency: the conscious experience of motor control. For example, the motor problems revealed in the experiment by Synofzik and colleagues were relatively subtle. The major problem related to the patients' subjective experience of movement as indicated by their report of the discrepancy between their actual movement and what they saw in the virtual reality setup. A much more specific account is required linking the function of the comparator with the experience of agency (Synofzik, Vosgerau, & Newen, 2008). Another problem with the proposal is that observations of a number of disorders, including schizophrenia, show that a mere failure of prediction is not sufficient to generate a delusion (Davies, Coltheart, Langdon, & Breen, 2001). Unlike healthy subjects, delusional patients are prepared to explain anomalous perception in terms of beliefs that

are highly implausible.

Our understanding of the cognitive basis of the sense of agency has been greatly enhanced by the discovery of the phenomenon of *intentional binding* (Haggard, Clark, & Kalogeras, 2002). The experience of agency is associated with binding, such that actions and their consequences are experienced as closer together in time as long as the action is experienced as the cause of the outcome. Studies of binding have shown that the experience of agency depends upon two components, first an advance prediction of the effects of the action and, second, a retrospective component whereby the nature of the outcome changes the experience of the action (Moore, Lagnado, Deal, & Haggard, 2009). Voss and colleagues (2010) have demonstrated that delusions and hallucinations are associated with a failure of the predictive component, while the retrospective component remains intact. Thus, the patients' experience of agency relies excessively on the sensory outcomes of their actions, confirming, from a very different perspective, the results of Synofzik and colleagues.

The series of studies I have outlined above have considerably advanced our understanding of the control and experience of action in the normal case as well as in patients with first rank symptoms. While the essence of Helmholtz' original idea remains intact, the new models emphasise prediction rather than monitoring. This emphasis provides an interesting link to the role of dopamine. We have known for many years that dopamine blocking drugs can reduce the

severity of symptoms such as delusions of control (e.g. Johnstone, Crow, Frith, Carney, & Price, 1978), but it is only more recently that evidence has emerged that dopamine has a critical role in prediction, or, more precisely, that dopamine release is a signal of reward prediction error (Schultz & Dickinson, 2000). So far dopamine's role in prediction has been explored mainly in classical learning paradigms concerned with which stimulus or which action will be rewarded. However, we should now examine whether dopamine has an equivalent role in the predictions associated with the control of action.

The framework in which prediction plays such an important role in the experience of action is essentially Bayesian. The prediction is the prior expectation and the sensory feedback is the new evidence on the basis of which the prior should be updated. Bayes' theorem indicates the extent to which such updating should occur. This framework is relevant for understanding the normal experience of agency, as well as all kinds of delusions and hallucinations, including symptoms of first rank such as delusions of control. Indeed, within this framework there is no real distinction between hallucinations (perceptions) and delusions (beliefs). Both depend upon appropriate combination of prior expectations and new evidence. This raises the possibility that an account could be developed, within this framework, that would apply to all the positive symptoms associated with schizophrenia (Fletcher & Frith, 2009). The comparator model would be seen as a special case within this framework and it is also possible that the two-factor account of delusions would be consistent with a Bayesian account.

There are two remaining problems. First, while a general theory of symptoms seems appropriate since some commonality between symptoms is required to justify the persistence of the idea of a unitary diagnosis of schizophrenia, there also needs to some account of why specific symptoms differ so markedly from one patient to another. Second, even a general, Bayesian approach does not provide a plausible account for the most striking of all first rank symptoms, *thought insertion*. This is ironic given that it was to explain this symptom that the comparator theory was first proposed (Feinberg, 1978). The problem is both theoretical and practical (see e.g. Vosgerau & Newen, 2007). Can we treat a thought like an action? In what sense can we predict the consequences of thought, since thinking seems to generate neither kinematics nor sensations? We need to develop objective measures of thinking analogous to all the ingenious measures of action and the experience of action that have so successfully been applied to the study of delusions of control.

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