LETTER TO THE EDITOR

Reply: The Bayesian equation and psychosis

Valerian Chambon,1,2 Philippe Domenech,1,2 Guillaume Barbalat,3 Elisabeth Pacherie,4 Pierre O. Jacquet5 and Chloë Farrer6,7

1 Institut National de la Santé et de la Recherche Médicale, 75005 Paris, France
2 École Normale Supérieure, 75005 Paris, France
3 Department of Psychiatry, University of Cambridge, CB2 0SZ Cambridge, United Kingdom
4 Institut Jean Nicod, CNRS, DEC-ENS, 75005 Paris, France
5 Department of Psychology, University of Bologna, 40100 Bologna, Italy
6 Centre de Recherche Cerveau et Cognition, Université de Toulouse, UPS-CNRS, 31059 Toulouse, France
7 Faculté de Médecine de Rangueil, 31062 Toulouse, France

Correspondence to: Valerian Chambon, École Normale Supérieure, 29, rue d’Ulm, 75005 Paris
E-mail: valerian.chambon@ens.fr

Sir, In a recent study (Chambon et al., 2011), we investigated the ability of patients with schizophrenia to make accurate predictions about other people’s intentions. This ability has long been shown to be impaired in schizophrenia, and this impairment may be accounted for by an abnormal integration of two different sources of information: the sensory evidence conveyed by movement kinematics, and the observer’s expectations about how likely an intention is. In the task, these two types of information were systematically varied. Our results showed that patients with positive symptoms were prone to over-weight sensory evidence confirming their prior expectations and to disregard evidence that invalidated such priors. We hypothesized that this abnormal interplay of prior expectations and current sensory experiences—that normally guarantee accurate inferences—could result from a disturbance in prediction error signalling, possibly caused by alterations in the dopaminergic circuitry. We speculated that such aberrant prediction error signals might account for the formation, as well as for the update, of delusional beliefs as to how biological agents are most likely to behave.

In their comment, Garrett and Singh suggest that our results cannot be readily applied in vivo to psychosis ‘without distinguishing between pre-psychotic and post-psychotic prior experience’. Specifically, they argue that belief updating should be differentially impaired according to whether sensory evidence combines with a belief that has been formed prior to, or during, a psychotic episode. They argue that, in clinical psychosis, patients do not abnormally under- or over-weight information indistinctly, but that evidence disconfirming ‘psychotic’ beliefs only is under-weighted, whereas evidence confirming these beliefs is given too much credit. During acute psychotic episodes, confirmatory evidence is over-weighted because such evidence (e.g. ‘the fleeting glance of a stranger’) readily combines with previous confirmatory observations (e.g. ‘strangers are wearing blue coats’) to progressively inflate the veracity of the initial delusional belief (i.e. ‘FBI agents are watching me’).

This comment raises two important points. First, it suggests that the integration of sensory evidence and prior beliefs somehow differs according to whether these beliefs are ‘psychotic’ in essence, i.e. whether they involve a delusional content, or not. Second, the formation of delusional beliefs results from a deviation of this integration process relative to the ‘Bayesian norm’. In Garrett and Singh’s view, this process of delusion formation qualitatively differs from the process whereby formed beliefs (both delusional and non-delusional) are maintained during acute psychotic episode. As our task did not contrast delusional versus non-delusional beliefs, Garrett and Singh argue that our results pinpoint a disturbance in a non-specific mechanism of belief maintenance, but say little about how ordinary beliefs progressively convert into delusional thoughts.

We fully agree with Garrett and Singh that the formation of delusional beliefs calls for specific investigations, as it is still unclear whether it entails specific computational processes. For example, delusional beliefs might arise as a consequence of either misassessed prior probabilities (e.g. delusion-related priors may be...
abnormally inflated in patients), or misaggregated components of the Bayes’ rule (i.e. priors and likelihood may not combine according to this rule), or both (McKay, 2012). However, the fact that such processes specifically account for the emergence of delusional beliefs does not preclude their participation in the abnormal maintenance of beliefs. Indeed, both delusion formation and abnormal persistence of beliefs may just be explained by a single neurophysiological mechanism. Aberrant prediction error signalling in the context of dopamine dysregulation has been proposed as a probable candidate (Corlett et al., 2009; Fletcher and Frith, 2009).

In a nutshell, prediction error is defined as the discrepancy between what is expected and what is actually experienced. Such errors signal the need to update one’s beliefs about the world. Under physiological conditions, individuals update beliefs in an approximately Bayesian fashion, whereas pathological prediction error signals may yield significant deviations from normative Bayesian inference (Friston, 2010; McKay, 2012). It has been suggested that aberrant coding of prediction error in deluded patients may abnormally over-weight random events. Delusions would thus arise as a means of explaining, or accommodating, these odd, and abnormally salient experiences (Kapur, 2003; Corlett et al., 2009). Interestingly, the persistence of delusion, as well as the abnormal inflexibility of otherwise non-delusional beliefs, can be explained through the same mechanism. Indeed, in Bayesian hierarchical networks, persistent delusional beliefs are more likely as one goes up towards more abstract hierarchical levels, which depend less on external inputs and more on previous stage representations (Friston, 2010). Thus, misbeliefs can persist because of persistent abnormal prediction error throughout the whole hierarchy. A belief induced by the coincidental hypersalience of an event would thus be progressively reinforced and enriched as aberrant prediction error randomly occurs (Corlett et al., 2009). When the psychotic equilibrium is reached, persistent error signals would continuously reconsolidate this initial explanation, up to a point where the belief in this explanation becomes absolutely impervious to contradictory evidence, and ultimately creates the framework within which all perceptual experiences are interpreted.

In sum, we agree with Garrett and Singh that delusional beliefs may arise as a consequence of multiple deviations from normative Bayesian updating. However, we do not think there is a need to postulate a separate mechanism to explain why, at the same time, non-delusional (pre-psychotic) beliefs may prove highly inflexible and abnormally persist in the face of contradictory evidence.

Acknowledgements

We wish to thank Prof. Paul Fletcher for useful discussions on some of the issues addressed in this letter.

References


