Commentary on Synofzik, Vosgerau and Newen 2008
1. Introduction

Synofzik, Vosgerau and Newen (2008) offer a powerful explanation of the sense of agency. To argue for their model they attempt to show that one of the standard models (the comparator model) fails to explain the sense of agency and that their model offers a more general account than is aimed at by the standard model. Here I offer comment on both parts of this argument. I offer an alternative reading of some of the data they use to argue against the comparator model. I argue that contrary to Synofzik, Vosgerau and Newen’s reading the case of G.L. supports rather than contradicts the comparator model. Next I suggest how the comparator model can differentiate failures of action attribution in patients suffering parietal lobe lesions and delusions of alien control. I also argue that the apparently unexpected phenomenon of “hyperassociation” is predicted by the comparator model. Finally I suggest that as it stands Synofzik, Vosgerau and Newen’s model is not well specified enough to explain deficits in the sense of agency associated with delusions of thought insertion. Thus they have not met their second argumentative burden of showing how their model is more general than the comparator model.

2. Synofzik, Vosgerau and Newen’s Model

Synofzik, Vosgerau and Newen present a model of the sense of agency (SoA) over both bodily and mental actions. They argue that this model should replace one of the standard views in the field of self consciousness research. The particular position they take issue with is known as the comparator model (CM). This model holds that the SoA over bodily actions is elicited based on a represented match between the predicted sensory consequences of an action and the actual sensory consequences of that action (Frith et al. 2000b; Frith et al. 2000a). On this model deficits in the SoA observed in patients suffering delusions of control are explained by an inability to make this comparison, which in turn arises from deficits in
the formation of or access to predicted sensory consequences of action. This model holds that the SoA is elicited in the final stages of action execution by a single, low level, mechanism.

In contrast Synofzik, Vosgerau and Newen argue that the SoA is elicited by the interaction of two mechanisms. One of these is a low level mechanism operating around action execution. The other is a higher level concept or belief forming mechanism. On their view the SoA arises in two steps. First is the feeling of agency (FoA). The FoA is the “non-conceptual, low-level feeling of being the agent of an action” (Synofzik et al. 2008 pg 222). This is the feeling of oneself as the agent of action or not. It cannot, at this level, involve the feeling of someone else being the agent of action. They tell us that “at this level, an action is merely classified as self-caused or not self-caused” and “no external attribution is possible at this level” (Synofzik et al. 2008 pg 227). Second a judgement of agency (JoA) is needed. The JoA is the “conceptual interpretative judgement of being an agent” (Synofzik et al. 2008 pg 222). In contrast to FoA, JoA’s can involve the external attribution of agency to a specific agent (Synofzik et al. 2008 pg 228).

This is not merely a distinction between two notions of the term “sense of agency” rather it is the identification of two mechanisms that contribute to that sense. FoA is taken to arise from a variety of “agency cues” at the level of action execution. These cues are weighted in a context dependent way so as to elicit the FoA when appropriate. Such cues include efference copies of motor commands, predictions of sensory consequences of action based on these commands, actual sensory consequences of action and representations of matches between the later two (Synofzik et al. 2008 pg 226-227). In contrast JoA arise from the mechanisms of concept formation especially “ad hoc theorising” (Synofzik et al. 2008 pg 228) about ourselves involving metarepresentation (Synofzik et al. 2008 pg 227). These mechanisms
take the FoA as one input, but other factors, such as conceptions about oneself are also important (Synofzik et al. 2008 pg 228). We would also expect such things as attribution biases and the ability to recognise others as (potential) agents to play a role in generating JoA.

To argue for this two-step model they aim to show why the standard model in the field fails and why their model offers a better alternative. As to the first part of this argument they attempt to show that the CM fails to account for the SoA. As to the second part of this argument they attempt to show that their model provides a more general account than the CM aims at. In the next section I will examine their arguments and conclude that they have neither shown that the CM fails to explain its target nor that their two-step account is capable of offering a more general account of the SoA.

3. The Attack on the CM

3.1 The Feeling of Agency (FoA)/Judgement of Agency (JoA) Distinction

Synofzik, Vosgerau and Newen spend much time arguing that the CM can only explain FoA and not JoA (see especially Synofzik et al. 2008 pg 223, 226 and 232). This is correct and it seems to imply that we should take the explanatory target of the CM to be only the FoA and its break downs in delusions of alien control. This means that not all features of delusions of alien control will be explained by the CM. In particular those features for which a JoA is needed, such as the attribution of an action to some other specific agent, do not fall under the explanatory target of the CM.

This move usefully limits the explanatory target of the CM to FoA and its deficits in delusions of alien control. This means that alien control is not fully explained until an account
of JoA is in place. It also means, as Synofzik, Vosgerau and Newen point out, that operationalising FoA via JoA could be problematic, at least in some circumstances (Synofzik et al. 2008 pg 222). If the target of the CM is just the FoA then it is no objection to the CM that it fails to explain the external attribution of agency in delusions of alien control, as it is sometimes taken to be (the problem of "extraneity" in Proust 2006) as there is no possibility of external attribution at the level of FoA. Synofzik, Vosgerau and Newen have made an important contribution here by allowing us to more clearly state just what the CM is supposed to explain. I take it then that their argumentative burden to show the necessity of step 1 in their account is not to show that the CM can’t explain JoA, but rather to show that it can’t explain FoA. I will focus on their two main arguments for this claim in the next two sections.

3.2 The case of G.L.

Synofzik, Vosgerau and Newen argue that a comparison between the actual and predicted sensory consequences of an action and a representation of a match is not sufficient to elicit the FoA. The do this using the case of deafferented patient G.L.

...there is further evidence that the CM is insufficient to explain the SoA. For example, several studies showed that a deafferented person (patient GL) was not only unable to identify the visual consequences of her own movements... but also showed disturbances of her feeling of being in control over her movements when visual feedback was distorted: Under these circumstances, “she reported impressions of not controlling her movements, and not being aware [of] what she was doing”... If the feeling of being in control over her actions, however, was primarily based on efferent signals (as suggested in part by [advocates of the CM], patient GL should not experience feelings of not controlling her movements, since efferent signals- and pre-action processes in general- are readily available to her. Even if such a feeling was based on a comparison between a motor prediction and a sensory feedback modality, she should feel in control over her movements at least in cases when the visual feedback was congruent to her predictions. Hence, the lack of FoA in patient GL under circumstances of ambivalent
visual feedback emphasises the need for further sensory feedback information to experience SoA, particularly involving proprioception ... (Synofzik et al. 2008 pg 224 emphasis added).

It seems odd to use this data *against* the CM. Indeed Farrer and colleagues (2003) use this as evidence *for* the CM. This data does seem to support the CM as it bears out a prediction of that model. The CM predicts that G.L. will have some FoA over her bodily movements but only when she can see her movements and that visual feedback is not distorted. Recall that the CM explains the elicitation of the SoA in terms of a represented match between the predicted and actual sensory consequences of an action. Due to deafferentation many of the actual sensory consequences (in the proprioceptive and kinaesthetic modes for example) are not available. Thus the comparison used to elicit the FoA must only involve visual representations for G.L. When visual feedback is distorted the CM predicts that G.L. will experience distortions in her FoA. This is indeed what happens (Farrer et al. 2003 pg 616).

Far from posing a problem for the CM, the experiences of G.L. seem to be predicted by the model. That being said, Synofzik, Vosgerau and Newen are right to point out that this case shows that the formation of predictions *alone* is not sufficient to elicit the FoA. They usefully argue against any (potential) position that makes such a claim. However, the CM is not such a position. In contrast the CM tells us that predictions must be compared to actual sensory feedback and a match must be represented for FoA to be elicited. As such the case of G.L. cannot be used to show that the CM is insufficient to explain the FoA.

3.3 Alien Control and Parietal Lobe Lesions

Following this argument Synofzik, Vosgerau and Newen point to 2 other sources of data which they take to suggests that the CM can't explain the FoA. In particular they attack the
claim that the CM can explain the FoA deficits observed in delusions of alien control (but not, of course, the delusion itself). This attack has two stages.

First they argue that those with parietal lesions and no deficit in FoA display deficits which are commonly explained by a deficit in FoA for those with delusions of alien control (Synofzik et al. 2008 pg 225). Daprati and colleagues (1997) examined the ability of those suffering delusions of control or verbal hallucinations to attribute actions to their appropriate source. In this study subjects were required to observe a movement and report whether or not it was their movement they saw. Subjects held their right hand below a mirror; the image in the mirror was recorded and sent via closed circuit television to a screen. The image on the screen was reflected by another mirror such that it appeared to the subject to be in the same place as their hand. The monitor could also display an experimenter’s hand (recorded from a different room) such that the experimenters hand appeared where the subjects hand was. Subjects were required to perform a series of movements of their hand. They saw either their own hand or the experimenters perform an action and were required to say whose hand they thought they saw moving (Daprati et al. 1997 pg 77). Subjects were tested under three conditions. They saw i) their own hand moving, ii) an experimenter making the same movement they had made or iii) an experimenter making a different movement.

Overall patients suffering delusions of control or verbal hallucinations made more recognition errors on this task than schizophrenic patients without these symptoms or healthy controls (Daprati et al. 1997 pg 79). This performance could be explained by the patient’s lack of FoA. In order to recognize whether or not the viewed action is one’s own on this task one must compare visual and non-visual feedback of the movement. If the movement looks the same as it feels then it is one’s own. If not then it is someone else’s (Jeannerod 2006 pg
176-178). In order to recognize that it is oneself that one sees moving then one must recognize that the actions performed have something to do with oneself (Povinelli 2001 pg 85). This is the function of the FoA in this context. The CM explains the performance of those suffering delusions of control in terms of a failure to detect a match between predicted and actual sensory consequences of action. This is usually taken to be due to an inability to form or access predictions (Frith et al. 2000b).

The problem that Synofzik, Vosgerau and Newen raise for this evidence is that some subjects fail at this task without displaying a deficit in their SoA. They note that:

…patients with parietal lobe damage show an impairment in detecting mismatches between those movements they had actually performed and those movements that were visually displayed to them …

Yet these patients do not report abnormal agency judgments (Synofzik et al. 2008 pg 225).

Patients who suffer parietal lesions fail this task but there is no reason to think they have a deficit in either JoA or FoA. These patients have problems identifying the agent of action without developing delusions of control. As such it seems that problems with identifying the agent behind an action cannot be sufficient for the development of delusions of alien control. Thus Synofzik, Vosgerau and Newen infer that a deficit in being able to compare predicted and actual sensory consequences of action (which may explain these patients performance on this task) does not necessarily lead to a deficit in FoA or JoA (Synofzik et al. 2008 pg 225). So it seems that the CM fails to explain how the SoA is elicited.

This problem disappears when we note that there are two ways one could fail on this task. The subject may lack a SoA over the movements as discussed above. Alternatively the subject may maintain a SoA over the movements, but fail to match the seen and felt sensory
feedback of the movement. In this case the failure of self attribution has nothing to do the SoA.

It seems that those suffering parietal lesions fail this task for the second reason. There is evidence that those with parietal lesions lack access to non-visual actual sensory feedback of actions. Those with parietal lesions often perform the required gestures clumsily on these tests (Sirigu et al. 1999 pg 1870). However, when these patients see the experimenter performing the action accurately, these patients do not use this fact to judge that they did not perform the action they saw. At times patients remarked that they had performed the actions better then they had on previous trials (Sirigu et al. 1999 pg 1870). In other words, in the condition where the patient and the experimenter attempted the same movement those with parietal lesions could not use the fact that they failed to perform the correct movement to judge that the hand they saw doing the movement accurately was not their own. This suggests that these patients can access the visual feedback of the action but not the proprioceptive feedback. In contrast those with delusions of control and healthy controls could use mistakes in movements as additional cues as to the agent behind the movement they saw (Daprati et al. 1997 pg 78). As such it seems that those with parietal lesions have a deficit in accessing the non-visual feedback of their movements that is not shared by those with delusions of control. Thus, the poor performance of those with parietal lesions on this test can be explained without needing to suppose that they have a deficit in their FoA. As such it is not clear that these data threaten the CM in the way required by Synofzik, Vosgerau and Newen.

A potential reply here is that a lack of access to non-visual actual sensory feedback seems like it should be enough prevent successful comparisons between actual and potential sensory feedback being made. This suggests that those with parietal lesions should experience a
deficit in FoA after all. When evaluating this concern it is important to keep in mind the importance of visual representations of the actual and predicted sensory consequences of action. We have already seen from the case of G.L. that when actual sensory consequences in the non-visual mode are not available performing the comparison in the visual mode alone is sufficient to elicit the FoA. Something similar seems to happen in patients suffering parietal lesions. Without non-visual actual sensory feedback, those suffering parietal lesions can use only matches in the visual mode to elicit the FoA.

Arising from the same type of experiment is the second stage of Synofzik, Vosgerau and Newen’s attack on the CM as an explanation of the deficits in FoA present in delusions of alien control. The problem those suffering delusions of alien control seem to have in these studies is in a sense the opposite of the problems that seem to be predicted by a deficit in FoA. For all groups, recognition errors occurred largely in condition ii, that being where the subject saw an experimenter perform the same movement they had made. That is they tended to attribute the actions of the experimenter to themselves. All subjects made very few errors in the other conditions (Daprati et al. 1997 pg 81). This problem is not exactly the problem we may expect given the content of the delusion of alien control. The content of the delusion of alien control suggests that those suffering the delusion tend to attribute their own actions to an external source. They feel as though someone else acts through their body. However, that is not the problem they have in this study. Synofzik, Vosgerau and Newen sum up this concern in this way:

This behavior, however, which was termed “hyperassociation” or “overattribution”, cannot be explained by the CM. If the patients were not able to form representations of the predicted sensory consequences of their actions, they should misattribute self produced sensory information to external sources rather than exhibit hyperassociations (Synofzik et al. 2008 pg 230).
This appears to be a serious worry for the CM. An immediately obvious reading of the CM does seem to suggest that it makes exactly the wrong prediction about this study. However, on closer inspection it seems that the model makes the right prediction after all. Frith and colleagues have suggested that the predicted sensory consequences of movement contribute significantly to representations of the nature of the movement performed in order to allow for faster responses (Frith et al. 2000b pg 1772; see Synofzik et al. 2006 for experimental evidence for this claim). If this is the case then the CM can explain why patients suffering delusions of control tend to overattribute the actions of others to themselves in this study. If the problem underlying the loss of the SoA in delusions of control is a lack of access to the predicted sensory consequences of action (as suggested above) then those suffering this symptom would have a reduced ability to represent the nature of the movements they perform (this may also explain the difficulties patients have with self monitoring, as in Fournieret et al. 2002; Franck et al. 2001; Frith and Done 1989; Stirling et al. 1998). They would therefore have a reduced ability to compare visual and non-visual representations of the movement. As such, they may take these representations as representations of the same movement when they are not. This leads them to overattribute actions to themselves despite having a deficit in their SoA. In other words the performance of those suffering delusions of control in this study is not to be explained by a deficit in FoA after all. Rather the deficit in FoA and the performance on this task have a common cause, namely an inability to access predicted sensory consequences of action. The deficit in FoA arises because a lack of access to predicted sensory consequences means that the right comparisons cannot be made. It seems that the overattribution of action displayed by those suffering delusions is consistent with the CM after all, thus it cannot form the attack on the CM required by Synofzik, Vosgerau and Newen.
3.4 The Extension to Delusions of Thought Insertion

The above challenges to Synofzik, Vosgerau and Newen’s reading of the data suggest that they have not established that the CM cannot explain the FoA over bodily actions. In this section I will examine their further claim that their two-step model, but not the CM can account for deficits in SoA associated with delusions of thought insertion.

There is some reason to suppose that SoA for bodily actions and SoA for mental actions (i.e. thoughts) arise from separate mechanisms. We may think this due to the double dissociation between deficits in this sense. SoA for bodily actions seems to be deficient in delusions of alien control, whereas SoA for thoughts seems to be deficient in delusions of thought insertion, thought withdrawal, made emotions, made impulses and verbal hallucinations. Delusions of alien control can occur without any of these other symptoms and vice versa (Mellor 1970). This being said if the two-step model could account for all of these deficits at once that would seem to be a major advantage for that model. However, we could not consider the two-step model to be complete until the double dissociation between SoA for bodily actions and SoA for thoughts had been explained.

There is some reason to suppose that Synofzik, Vosgerau and Newen’s model, as it stands, does not have the theoretical resources to explain deficits in SoA observed in delusions of thought insertion and the like. The model is not well specified enough at the level of FoA or JoA to provide such an explanation. There is some confusion about what constitutes the FoA for thoughts. At times it seems that on their model all there is to SoA for thoughts is JoA. On the two-step model all the resources for eliciting FoA are bodily. For example on the diagram on page 228 we see that FoA arises from “feed forward cues”, “proprioception” and “sensory
feedback” which are jointly described as “action-related perceptual and motor cues” (Synofzik et al. 2008 pg 228). This limits FoA to being a FoA for bodily actions and not for thoughts. As such it seems that the two-step model explains the SoA for thoughts in terms of JoA alone.

This is contradicted later when it is asserted that delusions of thought insertion:

...have to be explained by at least two factors...First, there must be some kind of “strange feeling” on the level of FoA and second, there must be a different process that leads to stable misattribution on the level of JoA (Synofzik et al. 2008 pg 235).

It seems that this model does posit a FoA for thoughts after all. Yet on closer examination the first step of their explanation of delusions of thought insertion is not in terms of FoA. Similar to an account offered by Stephens and Graham (Graham and Stephens 1994; Stephens and Graham 2000), Synofzik, Vosgerau and Newen propose that delusions of thought insertion arise from the patients unusually strong emotional responses against normal intrusive thoughts (Synofzik et al. 2008 pg 235). These strongly unwanted thoughts are then judged to come from some other agent. Even though this is still a two-step model, the first step does not seem to involve a FoA at all. Rather the first step is an unusually strong emotional response to intrusive thoughts. So whilst the two-step model uses more than JoA to explain delusions of thought insertion, the first factor is not FoA.

This is a problematic position as it seems that SoA for thoughts can’t be explained in terms of JoA without FoA. In particular there seems to be more to SoA for thoughts than JoA. This problem is particularly robust if Synofzik, Vosgerau and Newen’s hypothesis is that JoA for thoughts involves the same mechanisms as our capacity to make other judgements about
thoughts (i.e. theory of mind), as they seem to suggest (Synofzik et al. 2008 pg 227, 228).

One reason to think this is a problem is that those who have problems forming judgements about thoughts in general (e.g. 3 year olds or those suffering autism) do not have delusions of thought insertion. Similarly those who suffer delusions of thought insertion and the like do not have deficits in forming judgements about thoughts more generally (Corcoran et al. 1995; Greig et al. 2004; Mazza et al. 2001; Pickup and Frith 2001). This suggests either that SoA for thoughts is not limited to JoA or that whatever mechanism is responsible for JoA it is not the same as our capacity to form other judgements about thoughts.

There seems to be two ways around this problem. The first would be to amend the two-step model such that it contains a specific hypothesis as to which mechanism in forming judgements about thoughts is deficient. Such a hypothesis would have to clearly differentiate the mechanism of forming JoA from other mechanisms of forming other judgements about thoughts (e.g. theory of mind). I suspect that this will be Synofzik, Vosgerau and Newen’s preferred move. It is, however, worth noting a second option; we may amend the first step in the two-step model such that FoA could be formed for thoughts. This move requires some account of the resources used to elicit FoA for thoughts. Either alteration would have the result that the two-step model wouldn’t predict that those suffering autism (or indeed healthy 3 year olds) would also have delusions of thought insertion or the like.

I take it that Synofzik, Vosgerau and Newen have offered a viable account of the SoA. However, they have not yet shown it’s superiority to the CM. The alternative readings of the data I present here suggest that they have not yet shown that the CM fails to explain its explanatory target, namely the FoA. Furthermore, the lack of specification of what
mechanisms are involved in SoA deficits for thoughts (as seen in delusions of thought insertion) means that they have not yet shown how their model explains this phenomenon.
References


