CORTEX 47 (2011) 514–518

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Discussion forum

Mental imagery in congenital prosopagnosia: A reply to Grüter et al.

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Grüter et al. (this issue) provided a recent commentary with specific reference to the work of Tree and Wilkie (2010). Tree and Wilkie (2010) report on a case series of four congenital prosopagnosic (CP) cases, with whom face and object imagery testing was examined. The key finding of this work was that such cases can have co-occurring impairments of face recognition and face imagery – but that the mental imagery impairment in their group was *category specific* (i.e., not generalized to other non-face stimuli). One other finding was that CP testing of mental imagery using a 'vividness' questionnaire (i.e., a subjective measure of mental imagery in which participants are asked to introspectively rate vividness of different mental images) had little connection to CP performance on forced choice type (accuracy) measures of visual imagery – such that the former showed normal performance and the latter impaired performance. For this reason, Tree and Wilkie (2010) suggested caution in interpreting findings based purely on performance on a 'vividness' questionnaire. In earlier work using such testing, Grüter et al. (2009) reported in their large sample of CP cases that many showed very low scores on their 'vividness' questionnaire, implying that CP is largely linked to poor mental imagery. In their later commentary, Grüter et al. (this issue) have since taken issue with the work of Tree and Wilkie (2010), by levelling several criticisms which I will outline below. However, before doing so, I would suggest that the findings of both studies do not necessarily conflict, as we shall see – provided we can agree that (a) vividness testing and accuracy testing using forced choice measures can diverge from one another and (b) that CP cases can show dissociations of face and object imagery (a point that is never explicitly addressed in the work of Grüter et al. even though it is apparent from their findings).

Grüter et al. (this issue) make four criticisms of Tree and Wilkie (2010), summarised as follows: they take issue with, (1) the size of our case series, (2) the diagnostic 'criteria' for classifying cases as CP, (3) the visual mental imagery assessment methodology utilised in our study and (4) the control sample we used with respect to their vividness ratings. I will comment on each of these criticisms.

1. The size of our case series

Grüter et al. (this issue) take issue with the size of our case series – namely they imply that their 2009 study may be more valid as it has a larger sample, in that Tree and Wilkie (2010) report a case series of four CP individuals, rather than a large group study. In order to respond to the criticism, it is perhaps worth putting the research of Tree and Wilkie (2010) into the wider context of the field of cognitive neuropsychology, since this is my background. This will illustrate that the approach taken by Tree and Wilkie (2010) is by no means highly unusual, it is in fact a standard practice amongst many researchers.

The purpose of cognitive neuropsychology is not merely to 'classify' a case as having a face recognition impairment [or '*headache*' as Grüter et al. (this issue) put it], or to determine whether a face recognition impairment co-occurs with other 'symptoms', such as poor emotion recognition or gender recognition. The tradition of cognitive neuropsychology seeks to understand a face recognition impairment with respect to a cognitive model of face recognition. In other words, the work is theory grounded. A cognitive model of face recognition may suggest that several individuals may all be impaired at a particular test of face recognition such as a set of famous faces (that is to say they similarly score at a low levels relative to normal age matched controls) – but that the root cause of this problem can vary. In other words, simply focusing on

DOI of original article: 10.1016/j.cortex.2010.08.005.
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0010-9452/$ – see front matter
doi:10.1016/j.cortex.2010.11.005

a single outcome measure can be problematic. This particular issue is exactly why those in the cognitive neuropsychological tradition have avoided simply grouping individuals with specific cognitive impairments on the basis of a particular outcome measure and then attempting to make a generalization across the population.

As a consequence, it has been suggested that a more worthwhile approach is the *case series* method (e.g., Barton et al., 2009; Duchaine et al., 2007; Schmalzl et al., 2008), in this instance individual cases are closely scrutinsed via a battery of cognitive testing and each individual's profile is reported so that the range of abilities is transparent to the reader. Given a case series approach is in fact encompassed in elements of earlier work by Grüter et al. (2007), I would assume that these researchers would have some sympathies with the points I am making. The assumption here is that the CP population is by no means heterogeneous – and therefore the testing approach must reflect this assumption. In fact, in many instances it is *these very differences* that are so interesting to a cognitive neuropsychologist. Since they enable the sub-classification of the group of interest, and a degree of theory testing, given sub-classification should fall within a model's predictions. In Tree and Wilkie (2010) our case series were tested on a number of standardised face and non-face measures, and they were selected on the basis of the fact that their impaired performance patterns were very similar – in other words, far less heterogeneous than likely seen in a large sample of CP cases.

At least on the issue of heterogeneity, it is of note that Grüter et al. (this issue) write "*A heterogeneous group of prosopagnosics may, of course, yield heterogeneous results* (Le Grand et al., 2006)." (p. 4). I would strongly agree with this point, and thus it isn't clear why these same authors don't draw the conclusion that if they do agree with me that any large sample of cases of CP will likely be heterogeneous, that their very approach (namely putting their sample into a single large group) can be problematic. How can we know if those that are impaired for the same reasons? For this reason, I would argue that the very point of suggesting one study may be more 'valid' than another just on the basis of the size of a group, is a rather strange point to make. Particularly, when one considers the following possibility: let us imagine that face recognition and face imagery are drawing upon two different underlying processes – which we might call process X (face recognition) and process Y (mental imagery). Under this proposal there are four different possibilities within a given population: (1) someone is unimpaired with X and Y, (2) someone is impaired with X but not Y, (3) someone is impaired with Y but not X and (4) someone is impaired with X and Y. If one classifies a large group of people who appear to have an impairment of X, that group could contain a varying number of individuals who fulfill the criteria for (2) or (4). The point being, the identification of cases with criteria (4) does not necessarily entail that cases with criteria (2) do not exist – since it may well be that impairments of X and Y can often co-occur, and this might be a general pattern when averaged across a group from the prosopagnosic population. In other words, the presentation of a number of cases with impairment X and Y (an association) remains difficult to interpret. Since presentations of such cases, cannot differentiate between an account in which X and Y are separate functional systems or otherwise. It is rather cases of impaired X and *not* Y (a dissociation) or vice versa which allow a much clearer interpretation [see Coltheart et al. (2010) for related discussion]. In other words, I would wish to stress that the findings of our study and theirs can quite happily co-exist, since it is possible that some sub-proportion of the prosopagnosic population do show co-occurring impairments of face recognition (X) and mental imagery (Y) – provided they are not arguing *everyone* with impairment X *must* have impairment Y (a point that Grüter et al. have never discussed explicitly, but would likely agree with given that their studies have shown CP cases with normal 'vividness' imagery testing). The overall point being made is that when Grüter et al. (2009) report a co-occurring 'symptom' (as they put it) of low 'vividness' ratings across their large group of CP cases, the research community is no wiser as to what such an association might imply with respect to functional mechanisms underpinning face processing and face imagery.

This is exactly why single cases (in the tradition of cognitive neuropsychology) have so much power, they are the 'black swans' (Tree and Kay, 2006) to accounts that wish to determine functional separability. If one follows this chain of logic, the claim that a sample size is in some manner a measure of 'validity' is as absurd as someone arguing they have seen 1000 white swans and insisting that all swans must be white because that number is so much larger than the 10 black swans someone else has reported seeing. I would therefore suggest, that the merit of our study has nothing to do with sample size – rather, it is demonstrating that *not all* prosopagnosics have general visual imagery impairments (although some might well do, and I would not take issue in principle with the findings of their study on this grounds), and our case series describes examples of these 'black swans'. The implication is therefore that X and Y must be functionally separable since that is the best account for the pattern of our findings – in accordance with the tradition of cognitive neuropsychology, these processes are *dissociable*. A conclusion which, as has already been stressed, does not appear to conflict with the findings of Grüter et al. (2009), since they also report CP cases with normal mental imagery. The difference between me and Grüter et al. is I am more interested in cases of dissociation, whereas they seem to stress an interest in the association (a co-occurring 'symptom').

2. The diagnostic 'criteria' for classifying cases as CP

Grüter et al. used a semi-structured interview form administered by a physician or psychologist to identify or 'diagnose' CP. Grüter et al. also report that this interview procedure has been verified by a face recognition battery in a subgroup of CP cases, and it is implied that such a procedure might be better than that used in Tree and Wilkie (2010). I would respond with the following comments.

Firstly, we never took issue with the primary 'diagnosis' of CP in their initial study, although they seem to be doing so for our study. Our point was primarily focused on the 'diagnosis' of visual imagery impairments in their population