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# Preserved striate cortex is not sufficient to support the McCollough effect: Evidence from two patients with cerebral achromatopsia

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Caitlin R Mullin, Jean François Démonet§, Robert W Kentridge#, Charles A Heywood#, Melvyn A Goodale‡, Jennifer K E Steeves¶

Centre for Vision Research and Department of Psychology, York University, Toronto, ON, Canada M3J 1P3; e-mail: steeves@yorku.ca; §INSERM U825, Hôpital Purpan, Toulouse, France;

# Department of Psychology, Durham University, Durham, UK; ‡ Department of Psychology, The University of Western Ontario, London, ON, Canada

Received 18 February 2009, in revised form 4 August 2009; published online 20 November 2009

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**Abstract.** The McCollough effect (ME) is a colour aftereffect contingent on pattern orientation. This effect is generally thought to be mediated by primary visual cortex (V1) although this has remained the subject of some debate. To determine whether V1 is in fact sufficient to subserve the ME, we compared McCollough adaptation in controls to adaptation in two patients with damage to ventrotemporal cortex, resulting in achromatopsia, but who have spared V1. Each of these patients has some residual colour abilities of which he is unaware. Participants performed a 2AFC orientation-discrimination task for pairs of oblique and vertical/horizontal gratings both before and after adaptation to red/green oblique induction gratings. Successful ME induction would manifest itself as an improvement in oblique-orientation discrimination owing to the additional colour cue after adaptation. Indeed, in controls oblique grating discrimination improved post-adaptation. Further, a subdivision of our control group demonstrated successful ME induction despite a lack of conscious awareness of the added colour cue, indicating that conscious colour awareness is not required for ME induction. The patients, however, did not show improvement in oblique-orientation discrimination, indicating a lack of ME induction. This suggests that V1 must be connected to higher cortical colour areas to drive ME induction.

## 1 Introduction

The McCollough effect (ME) is an orientation-contingent colour aftereffect where a grating pattern is paired with a colour, typically red or green, and, after prolonged viewing of the coloured grating, adaptation results in a negative colour aftereffect when viewing the black and white grating of that same orientation alone (McCollough 1965). For example, after adaptation to vertical red and black gratings, a vertical white and black grating will appear tinged with the complementary colour, green. Rotating the head or the grating eliminates the perceived complementary colour tinge in the grating, therefore demonstrating its orientation contingency (McCollough 1965). Its discovery has generated considerable interest and a large body of research (see Humphrey 1998 for a review). Nonetheless, the nature of its underlying mechanisms and the locus of the interaction between colour and orientation in the visual pathway remain a matter of debate.

The original interpretation suggested the ME arises from a single group of neurons coding both colour and orientation early in the visual pathway (V1) (McCollough 1965). In fact, its wavelength dependence (Thompson and Latchford 1986) and lack of interocular transfer (Murch 1972; Savoy 1984) also suggest that it is primarily a lower-level visual process (subserved by area V1 or earlier). The ME can be induced preconsciously by presenting the adaptation stimuli at a rate of 10 or 20 ms (Vul and MacLeod 2006). Since colour-sensitive cells in V1 have been shown to track colour faster than can conscious perception, this suggests the ME is located early in the visual pathway. Time scales of the ME also implicate early visual areas (Vul et al 2008).

Studies of neurological patients with specific visual-pathway damage also suggest that the neural substrates of the ME are early in the visual pathway. Humphrey et al (1991) attempted to induce the ME in patient DF, whose damage is primarily in the lateral occipital region of the ventral stream (mainly areas V2 and V3) and who exhibits a profound impairment in visual form and orientation perception (Goodale et al 1991). However, in this patient V1 as well as area V4 and other medial ventral-stream structures are largely intact, as is her ability to detect and discriminate colours (Milner and Heywood 1989). Instead of simply reporting whether or not the colours in the achromatic test grating were perceived—the typical procedure for measuring the ME—Humphrey et al (1991) designed an odd-man-out test based on grating orientation. Before adaptation, DF performed at chance when asked to discriminate between either achromatic test patches of cardinal (vertical and horizontal) or oblique ( $45^\circ$  and  $135^\circ$ ) gratings, consistent with her inability to discriminate orientation. After adaptation, ME induction changes the odd-man-out task from that of orientation to colour discrimination for the oblique gratings. Following adaptation to highly saturated green and magenta oblique gratings, DF could now discriminate between the oblique gratings but continued to fail to discriminate between the cardinal gratings. DF's ability to induce the ME despite damage to higher-order areas of the visual pathway is consistent with the theory that the ME reflects adaptation of early visual mechanisms.

Despite the abundance of evidence supporting the role of early visual areas such as V1 in the ME, some recent evidence suggests its mechanism may not be confined to V1 alone (Humphrey 1998), and that there could be a network of cortical areas involved. Several functional magnetic resonance imaging (fMRI) studies have found consistent activation for the ME in an area of the lingual and fusiform gyri corresponding to area V4 (Barnes et al 1999; Humphrey et al 1999; Morita et al 2004). Previous research shows area V4 is associated with complex aspects of colour perception, such as the conscious perception of colour (Zeki 1993). These neuroimaging findings, coupled with previous research on colour regions in the visual pathway, suggest that, although V1 is responsible for the production of the ME, area V4 is required for its conscious perception (Humphrey and Goodale 1998). However, left-posterior V4 shows activation for the ME that is distinct from that associated with conscious colour processing (Morita et al 2004). Participants who are unaware of their experience of McCollough induction still show activation in this region. This distinct activation may reflect the association between the achromatic gratings and the specific colour, suggesting that components of area V4 may be necessary for generating the ME, and that V4 may be involved in more than simply conscious colour perception. Although a strong case has been made for the role of V1 in ME induction, it is possible that it may not be sufficient.

In the present study, we attempted to induce the ME in two patients with achromatopsia who have intact V1 but limited connections to the higher cortical colour areas, in an effort to verify whether V1 is sufficient to drive McCollough adaptation. Both patients suffered severe damage to the fusiform and lingual gyri (V4), but have spared V1. This is unlike the previous patient research on the ME where both V1 and V4 were intact. These patients also had some residual colour processing of which they were not explicitly aware (Heywood et al 1991; Lé et al 2002).

If these two patients do not experience the ME despite having intact V1, this would suggest that connections between V1 and higher cortical visual areas, possibly the fusiform and lingual gyri which are damaged in our patients, or other connections beyond V1, are required to generate this aftereffect. Considering, however, that both patients have previously exhibited residual colour processing of which they are not explicitly aware and that the current McCollough procedure does not require the conscious report of colour, we expected the patients might exhibit the ME since its induction and

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conscious colour perception can be dissociated (Morita et al 2004). If these patients showed evidence of an ME, this would suggest that V1 is sufficient for its induction and no connections to higher cortical colour-processing areas are necessary.

## 2 Methods

### 2.1 Participants

2.1.1 *Control participants.* Eighteen control participants were tested (eleven female and seven male; mean age = 24.3 years, SD = 4.45 years). All control participants had normal or corrected-to-normal vision, and no known colour anomalies.

2.1.2 *Patient SB.* SB is a 38-year-old male who suffered from meningoencephalitis at 3 years of age. His primary symptoms include profound object agnosia (inability to recognise objects) and prosopagnosia (inability to recognise faces) (Lé et al 2002). MRI revealed right-hemisphere damage to areas V2, V3, V4, and V5 (MT), sparing V1 (primary visual cortex). Left-hemisphere lesions were more limited but resulted in the complete destruction of the fusiform gyrus (see Lé et al 2002 for more details). These lesions lead to a left lateral homonymous hemianopia, sparing the macula. Further testing revealed that SB is also achromatopsic (cortical loss of colour vision); however, he has demonstrated the ability to identify highly saturated coloured targets in an odd-man-out paradigm, likely on a subconscious level (Lé et al 2002).

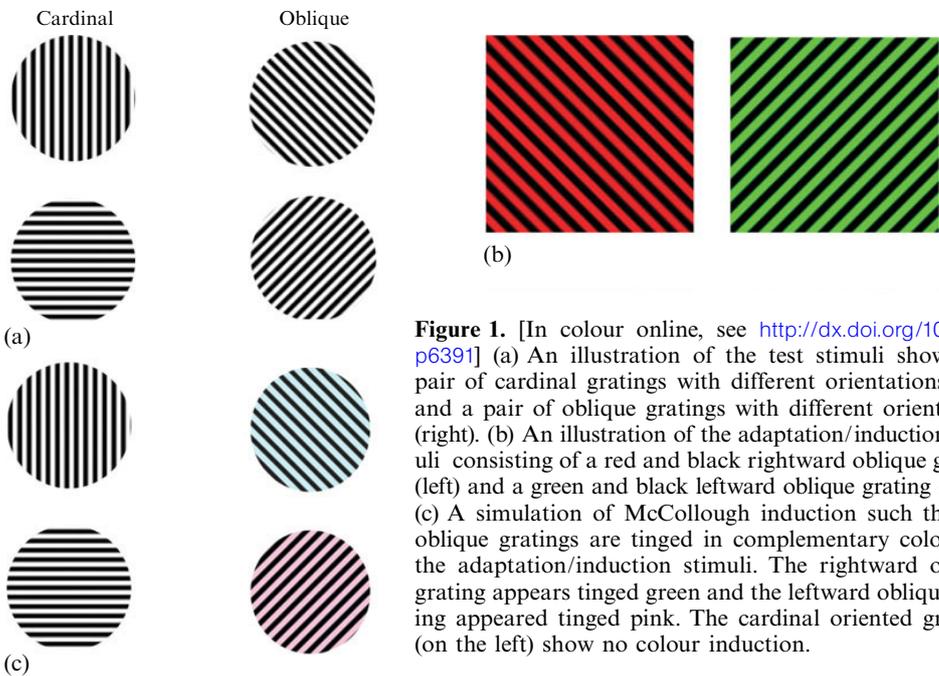
2.1.3 *Patient MS.* MS is a 59-year-old male who suffered from idiopathic herpes encephalitis in his early 20s which resulted in profound lesions to both hemispheres. MR imaging (Heywood et al 1991) revealed bilateral ventral and ventromedial damage to temporo-occipital regions. The left hemisphere suffered damage to the temporal pole, the parahippocampal and fourth temporal gyri of the temporal lobe, the collateral sulcus, and the mesial occipito-temporal junction. The right hemisphere suffered damage to the same regions but the striate cortex was additionally destroyed, producing a left homonymous hemianopia (see Newcombe and Ratcliff 1975 for more details). These lesions resulted in complete achromatopsia, prosopagnosia, and visual object agnosia. Despite his achromatopsia, MS has retained the ability to detect isoluminant chromatic boundaries composed of colours which he fails to tell apart (Heywood et al 1991).

### 2.2 Stimuli

All stimuli were presented on a 20-inch CRT monitor, with VPixx (VPixx Inc.) on a Macintosh computer. Test patterns were two 1 cycle  $\text{deg}^{-1}$  achromatic square-wave gratings presented within a 10 deg circular aperture. The two test patches were presented one above the other (separated by 2 deg vertically) at 4.5 deg to the right of a 0.5 deg fixation cross in order to present the stimuli to the patient's intact visual field. Test patterns were displayed in two orientation conditions: cardinal (vertical or horizontal) and oblique (leftward or rightward). Within each orientation condition, grating orientation was varied so that two test patterns could be of the same or different orientation.

There were 20 trials for each test condition, resulting in 160 trials total (20 trials  $\times$  2 orientations  $\times$  4 display durations). The order of presentation was fully randomised. The mask consisted of a full screen of rectangular patches of 1 cycle  $\text{deg}^{-1}$  square-wave gratings in randomly oriented cardinal and oblique orientations.

Adaptation stimuli were full-screen (25.4 deg  $\times$  32.3 deg) 1 cycle  $\text{deg}^{-1}$  oblique square-wave gratings. Right-oblique gratings were red and black while left-oblique gratings were green and black. For all stimuli, the luminance of the green, red, black, and white bars was 98, 30, 1.5, and 140  $\text{cd m}^{-2}$ , respectively. See figure 1 for illustrations of test and McCollough induction stimuli.



**Figure 1.** [In colour online, see <http://dx.doi.org/10.1068/p6391>] (a) An illustration of the test stimuli showing a pair of cardinal gratings with different orientations (left) and a pair of oblique gratings with different orientations (right). (b) An illustration of the adaptation/induction stimuli consisting of a red and black rightward oblique grating (left) and a green and black leftward oblique grating (right). (c) A simulation of McCollough induction such that the oblique gratings are tinged in complementary colours to the adaptation/induction stimuli. The rightward oblique grating appears tinged green and the leftward oblique grating appeared tinged pink. The cardinal oriented gratings (on the left) show no colour induction.

### 2.3 Procedure

Before beginning the experiment, control participants completed a pre-test form that assessed their familiarity with the ME to ensure an unbiased control group. Participants were given no indication that they would likely experience a coloured afterimage after adaptation to avoid biasing those who were not familiar with the effect.

**2.3.1 Experimental design.** All participants completed the pre-adaptation orientation-discrimination task, followed by adaptation, and then the post-adaptation orientation-discrimination task. The pre- and post-adaptation orientation-discrimination tasks were identical. Participants sat 57 cm from the display and viewed a fixation cross which appeared centrally for 500 ms followed by two test patterns which were followed by a mask. Each pair of test patterns was presented at four different exposure durations (0.3, 1, 2, and 4 s). The mask remained visible until the participant responded. Participants indicated whether the orientation of the two test patterns was the same or different by pressing designated keys on a keyboard. During adaptation, participants viewed full-screen leftward green adaptation gratings, which alternated with rightward red obliques every 10 s for a total of 20 min. Immediately after adaptation, participants were retested on the orientation-discrimination task.

A self-report questionnaire was completed by each control participant after completion of the experiment in order to determine whether the participant was aware of McCollough induction. The participants indicated whether they had experienced any coloured aftereffects in either the pre- or post-adaptation conditions. If the participants indicated that they did not experience colours in the post-adaptation condition, they were shown a screen of randomly oriented lines, some of which were of the same angle as the adapting stimuli, at which point the researchers asked if they saw any colours. If they answered “yes” they were then instructed to point to the sections of the mask where they saw colours.

## 2.4 *The oblique effect*

According to the well-known oblique effect, performance is superior for stimuli aligned in cardinal orientations compared to those in oblique orientations (Appelle 1972). Therefore, before adaptation, according to the oblique effect we expected participants to be worse at discriminating the orientation of oblique compared to cardinal gratings. After adaptation to coloured oblique gratings, ME induction should improve oblique discrimination because it will result in an additional ‘pop out’ colour cue for these gratings. In effect, after adaptation, oblique grating discrimination is a simple colour-discrimination task compared to a more difficult orientation-discrimination task. Evidence for a successful ME induction would be significant improvement in orientation discrimination from pre- to post-adaptation for the oblique orientations and no change in discrimination ability for the cardinal orientations.

## 3 Results

### 3.1 *Self-report*

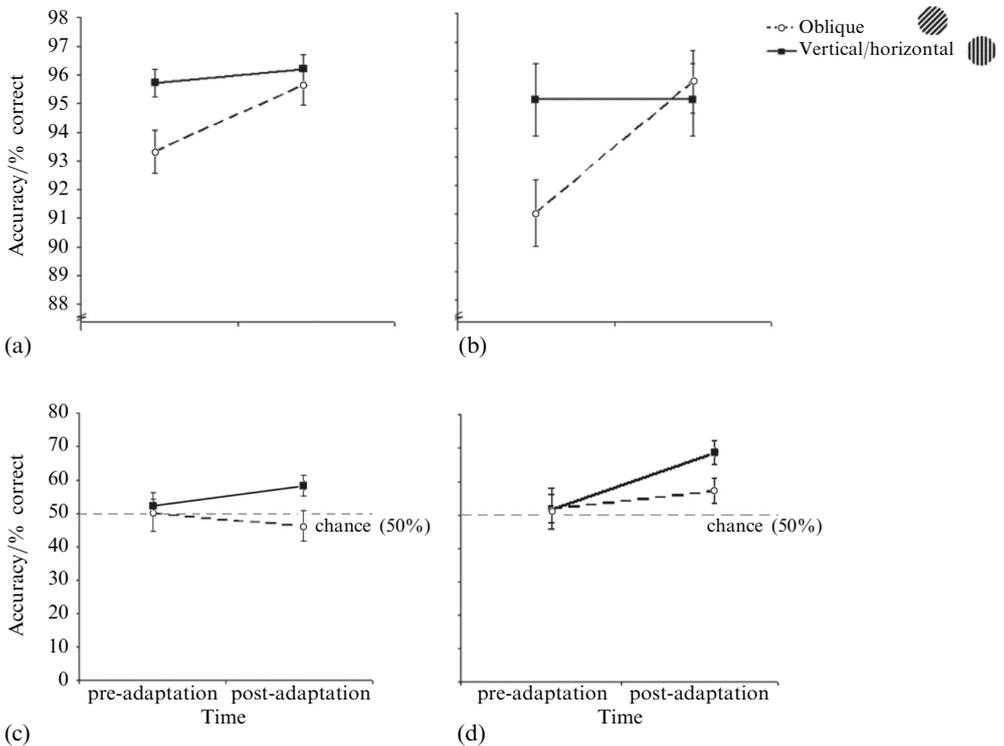
3.1.1 *Control participants.* None of the control participants reported experiencing colours in the pre-adaptation condition. Sixteen of the eighteen control participants reported experiencing coloured aftereffect after adaptation. The two control participants who did not report perceiving colour during the post-adaptation orientation-discrimination task became aware of the illusory colours during the post-test ME evaluation. Once the researchers instructed these participants to look for colours, they quickly were able to point to the oblique grating patches and indicated the perceived colours to be desaturated pink and green. Therefore, successful ME induction was confirmed by self-report in all control participants. The two participants who did not report perceiving colour until cued by the investigators comprised the ‘unaware group’, and their data are included in the control group but are also analysed separately to examine the effects of ME adaptation on participants who are unaware of the induced colours.

3.1.2 *Patients.* Neither patient SB nor patient MS reported experiencing any illusory colours during the post-adaptation orientation-discrimination task.

### 3.2 *Orientation-discrimination task*

3.2.1 *Data analysis.* Data were analysed with a  $2 \times 2 \times 4$  repeated-measures analysis of variance (ANOVA) [orientation  $\times$  time (pre/post)  $\times$  stimulus duration]. Separate paired *t*-tests were conducted on the data from the two participants in the ‘unaware group’.

3.2.2 *Overall controls.* The ME was successfully induced in the control participants (see figure 1a). There was a significant interaction between orientation and time ( $F_{1,17} = 11.31$ ,  $p = 0.004$ ,  $d = 0.04$ ). The main effects of orientation ( $F_{1,17} = 17.65$ ,  $p = 0.001$ ,  $d = 0.51$ ), and time ( $F_{1,17} = 7.44$ ,  $p = 0.014$ ,  $d = 0.31$ ) were significant. There were no significant main effects for stimulus duration and as a result all figures show data collapsed across the four stimulus durations. Pairwise comparisons with Bonferroni correction (adjusted  $\alpha = 0.025$ ) revealed that in the pre-adaptation condition participants were more accurate at vertical/horizontal compared to oblique discriminations ( $t_{34} = -2.07$ ,  $p = 0.021$ ,  $d = 2.07$ ), which is consistent with the oblique effect (see figure 2a). Further, participants’ accuracy improved significantly from pre- to post-adaptation for oblique-orientation discrimination ( $t_{17} = -3.48$ ,  $p < 0.001$ ,  $d = 0.86$ ). There were no significant differences in vertical/horizontal orientation discrimination from pre- to post-adaptation ( $t_{17} = -1.05$ ,  $p = 0.153$ ,  $d = 0.61$ ). The significant improvement in the oblique discrimination, post-adaptation, but not in the vertical/horizontal discrimination indicates successful ME induction.



**Figure 2.** Orientation-discrimination performance accuracy for oblique and cardinal gratings from pre- to post-adaptation. Each participant group's data are shown in separate panels: (a) Overall control participants, (b) unaware control participants, (c) patient SB, and (d) patient MS.

**3.2.3 Unaware control group.** The two control participants who reported being unaware of the illusory colours during post-adaptation orientation discrimination nonetheless exhibited successful ME induction (see figure 2b). A paired-samples  $t$ -test with Bonferroni correction (adjusted  $\alpha = 0.025$ ) showed significant improvement for oblique-orientation discrimination from pre- to post-adaptation ( $t_7 = -3.45$ ,  $p = 0.005$ ,  $d = 2.86$ ), while the vertical/horizontal condition remained the same ( $t_7 = 0$ ,  $p = 0.5$ ,  $d = 0$ ). The accuracy of the unaware participants was no different from that of the aware participants (mixed design ANOVA;  $F_{1,16} = 0.469$ ,  $p = 0.503$ ,  $d = 0.03$ ).

**3.2.4 Patients.** Both patient SB and patient MS performed near chance for vertical/horizontal and oblique orientations pre-adaptation consistent with their previous inability to discriminate orientation. After adaptation, both patients showed no significant change in accuracy for either orientation compared to pre-adaptation [SB: ( $F_{1,3} = 0.168$ ,  $p = 0.71$ ,  $d = 0.05$ —see figure 2c) and MS: ( $F_{1,3} = 0.059$ ,  $p = 0.824$ ,  $d = 0.02$ —see figure 2d)]. This suggests that neither patient SB nor patient MS could induce the ME.

## 4 Discussion

In this study we attempted to determine whether striate cortex (V1) alone is indeed sufficient for ME induction by testing two patients with intact V1 but damage to extrastriate visual areas. We found that control participants were able to successfully induce the ME, demonstrated by a significant improvement in oblique-orientation discrimination from pre- to post-adaptation. We also demonstrated that controls could induce the ME without explicit awareness of the added colour cue, indicating that conscious colour awareness is not a requisite property of ME induction. Neither of the

patients was able to successfully induce the effect. This cannot be attributed to their orientation processing deficit, since previous research has shown that attention to orientation is not required for successful ME induction (Houck and Hoffman 1986; Humphrey et al 1991, 1995). Subsequent to adaptation, discrimination of the orientation of the oblique gratings can be based entirely on the induced colour cue and explicit knowledge of the orientation of the grating is not necessary (Humphrey et al 1991).

It could also be argued that our patients were not able to induce the effect as a result of a lack of explicit colour processing (achromatopsia). We were able to show, however, that the ME can be induced without explicit awareness of its colours in our control participants. Two healthy participants in our study were unaware of the induced colours until after those colours were brought to their attention once the experiment was complete. Nonetheless, these two same control participants showed very robust ME induction. This confirms that the ME does not require conscious colour awareness. Our two patients have unusual specific residual colour abilities despite their achromatopsia suggesting that they may have some aspect of non-conscious colour ability. Patient MS can detect isoluminant chromatic boundaries composed of colours which he fails to tell apart (Heywood et al 1991) and patient SB can detect highly saturated colours (Lé et al 2002). Because the ME requires sensing colour alternating with black bars, the lack of colour contrast here would probably not have made the ME a 'conscious' experience for MS, even if it were induced. Further, because the ME results in desaturated colour afterimages, the effect would also not have been 'conscious' for SB. Therefore, even if the ME was successfully induced in these patients, they would not have been 'conscious' of its presence.

Together with our data from the two participants in the unaware control group, this study demonstrates that the absence of ME adaptation in our patients is not due to a lack of explicit colour experience but rather a result of damage to higher-order cortical areas. This conclusion is consistent with the recent neuroimaging data on the ME showing conscious and non-conscious McCollough adaptation in different regions of V4 (Morita et al 2004) and lends strong support to the idea that these higher cortical areas play a role in the ME. We speculate that the intact connections between V1 and higher cortical visual areas, possibly the fusiform and lingual gyri which are damaged in our patients (or other connections beyond V1), are required to generate this aftereffect. It is possible that the induction of the ME begins through the adaptation of separate colour and orientation systems in V1 leading to modified signals sent to cortical structures further downstream. These modified signals may interact in conscious and/or non-conscious regions within V4, which accounts for the fact that our patients who lack V4 did not show the ME. Finally, this is further evidence that V1 alone is not sufficient to support McCollough adaptation.

**Acknowledgments.** This research was supported by a grant from the Natural Sciences and Engineering Research Council of Canada (NSERC) and from the French Ministry of Foreign Affairs to JKES. We extend a special thank you to SB and MS for continuing to participate in our research. We thank Laurence Harris for helpful comments on the manuscript.

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ISSN 0301-0066 (print)

ISSN 1468-4233 (electronic)

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VOLUME 38 2009

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