Ethnic identity, perceptions of disadvantage, and psychosis
Findings from the AESOP study

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Many studies have shown that rates of psychosis are elevated in the Black and minority ethnic (BME) population in the UK. One important, but relatively less researched explanation of these high rates may be social adversity associated with acculturation processes. Strong identification with an ethnic minority group subjected to social disadvantage may exert adverse effects on individuals from BME groups. Using data from a large epidemiological case–control study of first-episode psychosis, we aimed to investigate whether strong ethnic identification is a factor contributing to the excess of psychosis in BME groups compared with the White British, after adjustment for perceptions of disadvantage. All cases with a first episode of psychosis presenting to specialist mental health services within tightly defined catchment areas in London and Nottingham, UK, and geographically matched community controls were included in the study. Data were collected on socio-demographic and clinical characteristics, perceptions of disadvantage, and identification with one’s own ethnic group. Analysis was performed on data from 139 cases and 234 controls. There was evidence that, as levels of ethnic identification increased, the odds of psychosis increased in the BME but not in the White British group, independent of potential confounders. However, the association between strong ethnic identity and psychosis in BME individuals was attenuated and non-significant when controlled for perceived disadvantage. Strong identification with an ethnic minority group may be a potential contributory factor of the high rates of psychosis in the BME population, the effects of which may be explained by perceptions of disadvantage.

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1. Introduction

Elevated rates of psychosis in the Black and minority ethnic (BME) population in the UK have been reported in numerous studies. Recent findings suggest a risk of between three- to six-fold for the Black Caribbean and Black African group compared to that of the White British. These findings extend to other BME groups, though rates of psychosis seem to be lower than in the Black Caribbean and Black African group (Cantor-Graae and Selten, 2005; Kirkbride et al., 2008). Variation in incidence in terms of ethnicity lends support to hypotheses that socio-environmental factors play a role in the aetiology of psychosis (Morgan, 2008).
One important, but relatively less researched socio-environmental factor potentially contributing to the high rates of psychosis in BME groups may be social adversity associated with processes at the interface between the host culture and the culture of origin, commonly referred to as ‘acculturation’ (Redfield et al., 1936). An important dimension of acculturation is ethnic identification, which results from a process of maintaining or relinquishing the characteristics of one’s own ethnic origin (Berry et al., 1986). There are only few empirical studies that have considered ethnic identification as a factor contributing to the risk for psychosis (Bhugra et al., 2010; Veling et al., 2010). Findings from these studies, taken together, remain equivocal with regard to the direction of effect. Stronger identification with a BME group subjected to discrimination and social disadvantage (Morgan et al., 2008; Veling et al., 2007) may be posited to exert adverse effects on BME individuals. By contrast, such adverse effects would not be expected to occur in the ethnic majority group (Rabinowitz et al., 2005; Bhugra, 2001, 2005). Previous reports did, however, not compare the associations found in BME to White native groups, making it difficult to draw any firm conclusions as to whether their findings may even partly explain the elevated rates of psychosis found in BME groups.

More recently, it has been argued that inconsistencies in empirical findings on ethnic identity and psychosis may be explained by contextual factors and perceptions of these (Bhui et al., 2005; Veling et al., 2010). Indeed, there are findings suggesting that contextual factors of the acculturation process such as ethnic density or perceptions of disadvantage (Cooper et al., 2008; Kirkbride et al., 2007; Veling et al., 2008) may partly contribute to the high rates of psychosis found in BME groups. Contextual factors may thus confound the relationship of ethnic identification and psychosis, but this has yet to be tested.

We aimed to (1) test the hypothesis that strong ethnic identification is associated with increased odds of psychosis in the BME group, but not in the White British group, and (2) investigate the extent to which any association between strong ethnic identification and psychosis is confounded by perceived disadvantage in the BME compared with the White British group.

2. Material and methods

2.1. Sample and procedure

This research forms part of the ÆSOP (Aetiology and Ethnicity in Schizophrenia and Other Psychoses) study, a multi-centre epidemiological study of first-episode psychosis conducted over a 3-year period. The inclusion criteria for cases were as follows: age 16–64 years; residence within defined catchment areas in south-east London and Nottingham; the presence of a first episode of psychosis (ICD-10 F20–F29, F30–F33) (WHO, 1992) within the time-frame of the study; and no previous contact with health services for psychosis. Exclusion criteria were as follows: evidence that psychotic symptoms had an organic cause; and transient psychotic symptoms resulting from acute intoxication as defined by ICD-10 (WHO, 1992). All patients, who presented to mental health services within the catchment areas in south-east London and Nottingham, were screened for inclusion using the Screening Schedule for Psychosis (Jablensky et al., 1992).

A random sample of population based comparison participants, aged 16–64 years, were selected using a procedure adapted from that used by the Office of Population and Census Statistics Psychiatric Morbidity Survey (Jenkins and Meltzer, 1995). This was used as a sampling frame to generate a random sample of ten target addresses in relation to each case, from which controls were recruited. Each address was contacted three times (morning, afternoon, evening). If an eligible control was not recruited the procedure was repeated. All adults in each household were invited to participate, and where more than one occupant was willing to take part an amended Kish grid was used to randomly select one. Black Caribbean controls were purposefully oversampled to ascertain sufficient numbers. All potential controls completed the Psychosis Screening Questionnaire (Bebbington and Nayani, 1995) and, if screened positive, were excluded from the study.

During the study period, 390 cases and 390 controls were recruited to the case–control arms of the ÆSOP study in the south-east London and Nottingham sites (Morgan et al., 2007). The adapted version of the Culture and Identity Schedule-1 (CANDID-1-A) was completed by 373 subjects (139 cases, 234 controls), which were included into the current study. Included subjects were from the following four ethnic groups: White British, Black Caribbean, Black African, and Asian. Those who completed the CANDID-1-A did not differ significantly from those who did not in terms of basic demographic characteristics (data available from the corresponding author).

2.2. Measures

Detailed symptom information was gathered through face-to-face interviews with the Schedule for Clinical Assessment in Neuropsychiatry (WHO, 1994) to determine ICD-10 diagnosis on the basis of consensus meetings involving one of the ÆSOP study’s principal investigators (Julian Leff, Robin Murray, Peter Jones) and other members of the research team. More detailed information is available in the study of Kirkbride et al. (2006).

Data on age, gender, employment status, and place of birth were collected using the MRC socio-demographic schedule (Mallet, 1997). Ethnicity was self-ascribed and standardized employing the 11 categories used by the UK census in 2001. If there was ambiguity in assigning subjects to ethnic groups, a consensus rating was made by experienced members of the research team. We also used a dichotomous ethnicity variable of White British vs. BME (Black Caribbean, Black African, Asian) groups.

Identification with one’s own ethnic group was assessed using the CANDID-1-A (Bhugra et al., 1999, 2010) (see Appendix A). CANDID-1-A items assessing ethnic identification in major life domains ask participants to rate their answers on scales with three to five response options. On this basis, binary codings of strong (coding of 1) and weak (coding of 0) ethnic identification are assigned. Given our aim to compare ethnic identification across ethnic groups, we selected a small number of key items that assessed ethnic identification with sufficient underlying commonality across ethnic groups from the original CANDID-1. There was evidence from multiple-group confirmatory factor analysis
that a unidimensional model with factor loadings constrained to be equal across ethnic groups had an acceptable model fit ($\chi^2 = 81.1$, $df = 34$, $p < .001$ vs. baseline $\chi^2 = 428.9$, $df = 30$, $p < .001$, TLI = .90, RMSEA = .07), suggesting that the CANDID-1-A measures the same construct in both White British and BME individuals (Brown, 2006). IRT discrimination ($\alpha$) and difficulty ($\beta$) parameters estimated under a two-parameter normal ogive model were moderate to high (range, $\alpha_{\text{item6}} = .76$ to $\alpha_{\text{item5}} = 1.31$) and showed a good spread throughout the measurement range (range, $\beta_{\text{item2}} = -1.02$ to $\beta_{\text{item4}} = 2.14$), respectively. This indicated a good ability of CANDID-1-A items to discriminate between subjects from lower and higher levels of ethnic identification throughout the measurement range (Embreton and Reise, 2000). Sum scores were computed as a continuous measure of ethnic identification, with higher scores indicating stronger identification with one's own ethnic group. Additionally, median split was used to classify subjects into categories of strong and weak ethnic identification as a binary measure to increase comparability of findings to measures used in previous studies (Velinger et al., 2010).

Perceptions of disadvantage were assessed using one item from the Culture and Identity Schedule-2 (CANDID-2), developed from an earlier questionnaire (Bhugra et al., 1999). This item asks subjects to rate their answer to the question ‘Do you believe that you experience any disadvantage when compared with other individuals in British society?’ on a 5-point Likert scale ranging from 1 (never) to 5 (very frequently).

2.3. Statistical analysis

Associations between basic sample characteristics and case–control status were assessed using chi-square and t-tests in Stata, Version 10 (Stata, 2007). Linear regression analysis was conducted to assess differences in levels of ethnic identification across ethnic groups stratified by case–control status. Binary logistic regression models were constructed to analyse associations between ethnic identification and ethnicity, as key independent variables, and case–control status as the outcome variable. Interaction effects for ethnicity and ethnic identification were assessed using likelihood ratio tests comparing logistic regression models with and without interaction term to address the first study aim. A more liberal value of $p < .10$ was used for assessing statistical significance of interactions to ensure that potentially important factors were not removed from the analysis. Age, sex, study centre, employment status, level of education, and place of birth were then added entering each variable in turn and likelihood ratio test conducted to assess potential confounding effects by these variables, only retaining significant variables in the final adjusted model. In a last step, we added perceived disadvantage to our models to address the second study aim.

3. Results

3.1. Socio-demographic and diagnostic characteristics

As can be seen in Table 1, cases were significantly younger, more often unemployed and of BME origin. There were significantly more controls from Nottingham than London. Most of the cases had a diagnosis of non-affective psychosis.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>Cases (n=139)</th>
<th>Controls (n=234)</th>
<th>Significance test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study centre, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>London</td>
<td>76 (54.7)</td>
<td>79 (33.8)</td>
<td>$\chi^2=15.71$, df=1</td>
<td>.001</td>
</tr>
<tr>
<td>Nottingham</td>
<td>63 (45.3)</td>
<td>155 (66.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>69 (49.6)</td>
<td>103 (44.0)</td>
<td>$\chi^2=1.11$, df=1</td>
<td>.29</td>
</tr>
<tr>
<td>Female</td>
<td>70 (50.4)</td>
<td>131 (56.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years), mean (sd)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>31.6 (11.1)</td>
<td>38.1 (12.5)</td>
<td>t=5.08</td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Employment status, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>63 (45.3)</td>
<td>149 (63.6)</td>
<td>$\chi^2=11.97$, df=1</td>
<td>.001</td>
</tr>
<tr>
<td>Other</td>
<td>76 (54.7)</td>
<td>85 (36.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level of education, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>School</td>
<td>81 (58.3)</td>
<td>115 (49.6)</td>
<td>$\chi^2=3.89$, df=2</td>
<td>.14</td>
</tr>
<tr>
<td>Further</td>
<td>37 (26.6)</td>
<td>64 (27.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher</td>
<td>21 (15.1)</td>
<td>53 (22.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnicity, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White British</td>
<td>75 (54.0)</td>
<td>181 (77.4)</td>
<td>$\chi^2=22.17$, df=1</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>BME</td>
<td>64 (46.0)</td>
<td>53 (22.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Place of birth, n (%)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>UK-born</td>
<td>114 (82.0)</td>
<td>206 (88.0)</td>
<td>$\chi^2=2.59$, df=1</td>
<td>.11</td>
</tr>
<tr>
<td>Non-UK-born</td>
<td>25 (18.0)</td>
<td>28 (12.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-affective psychosis</td>
<td>81 (58.3)</td>
<td>-</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Affective psychosis</td>
<td>58 (41.7)</td>
<td>-</td>
<td></td>
<td>-</td>
</tr>
</tbody>
</table>

Note: BME, Black and minority ethnic.

3.2. Ethnic identity, ethnicity, and psychosis

BME cases and controls reported significantly weaker ethnic identification than their White British counterparts (see Table 2). These differences remained significant after adjustment for confounders. The same pattern of findings was observed when comparing White British to Black Caribbean subjects. The number of Black African (n=29) and Asian (n=12) subjects was too small for comparisons to be made.

The continuous measure of ethnic identification and ethnicity was entered into a logistic regression model, with case–control status as the outcome variable (see Table 3). We assessed whether the association between stronger ethnic identification and psychosis varied by ethnic group by adding an interaction term to a logistic regression model. As can be seen in Table 3, there was evidence of interaction, in that the association held in the BME but not in the White British subjects. When adjusted for confounders, the association between ethnic identity and psychosis remained significant in the BME and non-significant in the White British group. These findings largely held when comparing White British to Black Caribbean subjects.

Findings on the binary measure of ethnic identification and case–control status by ethnicity are summarised in Table 4. This shows, again, evidence of interaction, in that BME cases were significantly more likely to report strong identification with one’s own ethnic group compared with BME controls. In
contrast, there was no significant association between ethnic identification and psychosis in the White British group. When confounders were adjusted for, the estimated odds ratios increased in the BME group due to the inclusion of study centre into the model. There was also an increase in the White British group, but odds ratios remained non-significant. In the adjusted model, BME cases were more than 2.5 times more likely than BME controls to report strong ethnic identification. A similar pattern of findings was evident when comparing the White British and Black Caribbean group.

### 3.3. Confounding by perceptions of disadvantage

When perceived disadvantage was entered into the adjusted logistic regression model for ethnic identification and case–control status by ethnicity, the strength of the association between the binary measure of ethnic identification and psychosis was attenuated and ceased to be statistically significant in the BME group (\(p = .088\)) (see Table 4). When this step in the analysis was repeated using the continuous measures of ethnic identification, the strength of the association was again attenuated, but remained just significant (\(p = .046\)) (see Table 3). By comparison, the estimated odds ratios for this relationship were attenuated, but remained non-significant in White British subjects. Again, these findings largely held when comparing the White British and Black Caribbean group.

### 4. Discussion

#### 4.1. Main findings

This is the largest case–control study to date to investigate acculturation as a risk factor contributing to the high rates of psychosis in the BME population in the UK. We found evidence that, as levels of ethnic identification increased, the odds of psychosis increased in the BME group, independent of potential confounders. In contrast, no significant differences in ethnic identification were observed between White British cases and controls. There was also evidence that the relationship of ethnic identification and psychosis in the BME group was confounded by perceived disadvantage. This pattern is suggestive of ethnic identification being a potential contributory factor of the elevated rates of psychosis in the UK BME population, the effects of which may at least partially be explained by perceived disadvantage.

### Table 2

Ethnicity and ethnic identification, by case–control status.

<table>
<thead>
<tr>
<th>Ethnic identification</th>
<th>Standardized coefficient b</th>
<th>Unstandardized coefficient B</th>
<th>95% Cl</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>BME vs. White British</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>-.65</td>
<td>-3.14</td>
<td>-3.61 to -2.66</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Adjusted &lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.65</td>
<td>-3.13</td>
<td>-3.67 to -2.59</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cases</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>BME vs. White British</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>-.55</td>
<td>-2.20</td>
<td>-2.76 to -1.63</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Adjusted &lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.54</td>
<td>-2.16</td>
<td>-2.90 to -1.42</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Controls &lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black Caribbean vs. White British</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>-.63</td>
<td>-3.32</td>
<td>-3.87 to -2.77</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Adjusted &lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.61</td>
<td>-3.19&lt;sup&gt;c&lt;/sup&gt;</td>
<td>-3.77 to -2.61</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cases &lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Black Caribbean vs. White British</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>-.55</td>
<td>-2.38</td>
<td>-3.06 to -1.71</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Adjusted &lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.49</td>
<td>-2.12</td>
<td>-2.97 to -1.26</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

<sup>a</sup> Adjusted for potential confounding by age, sex, study centre, employment status, level of education and place of birth.

<sup>b</sup> For the Black African (\(n = 29\)) and Asian (\(n = 12\)) group, the number of subjects was too small for comparisons to be made.

### Table 3

Ethnic identification<sup>a</sup> and case–control status, by ethnicity.

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Unadjusted OR&lt;sup&gt;b&lt;/sup&gt;</th>
<th>95% Cl</th>
<th>p</th>
<th>Adjusted OR&lt;sup&gt;c&lt;/sup&gt;</th>
<th>95% Cl</th>
<th>p</th>
<th>Adjusted OR&lt;sup&gt;d&lt;/sup&gt;</th>
<th>95% Cl</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>White British</td>
<td>.95</td>
<td>.80 to 1.12</td>
<td>.556</td>
<td>1.08</td>
<td>.89 to 1.30</td>
<td>.451</td>
<td>.90</td>
<td>.75 to 1.08</td>
<td>.257</td>
</tr>
<tr>
<td>BME</td>
<td>1.41</td>
<td>1.08 to 1.85</td>
<td>.012</td>
<td>1.45</td>
<td>1.08 to 1.96</td>
<td>.013</td>
<td>1.33</td>
<td>1.00 to 1.76</td>
<td>.046</td>
</tr>
<tr>
<td>White British</td>
<td>.95</td>
<td>.80 to 1.13</td>
<td>.556</td>
<td>1.02</td>
<td>.86 to 1.23</td>
<td>.797</td>
<td>.90</td>
<td>.75 to 1.08</td>
<td>.274</td>
</tr>
<tr>
<td>Black Caribbean&lt;sup&gt;e&lt;/sup&gt;</td>
<td>1.34</td>
<td>.99 to 1.82</td>
<td>.056</td>
<td>1.43</td>
<td>1.03 to 2.00</td>
<td>.034</td>
<td>1.21</td>
<td>.89 to 1.64</td>
<td>.217</td>
</tr>
</tbody>
</table>

<sup>a</sup> Continuous measure of ethnic identification with higher scores indicating stronger identification with one’s own ethnic group.

<sup>b</sup> Likelihood ratio test for interaction of ethnic identification × ethnicity: White British vs. BME, \(\chi^2 = 6.58, p = .010\); White British vs. Black Caribbean, \(\chi^2 = 4.24, p = .040\).

<sup>c</sup> Adjusted for potential confounding by age, sex, study centre, employment status, level of education, and place of birth.

<sup>d</sup> Adjusted for perception of disadvantage.

<sup>e</sup> For the Black African (\(n = 29\)) and Asian (\(n = 12\)) group, the number of subjects was too small for comparisons to be made.
Ethnic identity and psychotic experiences

4.2. Methodological considerations

The direction of causality cannot be determined from this case–control study. The Black Caribbean group was of modest sample size and the number of Black Caribbean and Asian subjects was too small for individual case–control comparisons to be made. Thus, caution must be taken in interpreting the results until replication in larger samples has been conducted. Perceived disadvantage was measured using only one item, which may have limited the reliability of findings and could, in theory, have been leading. However, given most participants responded negatively to this item, there is no evidence that this was the case. The CANDID-1-A allowed for assessing identification with one’s own ethnic group as an important dimension of acculturation. These items did not allow for measuring individuals’ outgroup orientation, i.e. identification with the other ethnic group, as a separate dimension, as has been suggested by some authors (Rabinowitz et al., 2005). Empirically, however, we observed reasonable internal and cross-cultural validity of the CANDID-1-A as a unidimensional measure of ethnic identification.

4.3. Comparisons with previous research

There has been a dearth of research into the role of ethnic identity in ethnic variations in the incidence of psychosis. In contrast to what we found, a matched case–control study of first-episode schizophrenia in non-Western immigrants in the Netherlands (Veling et al., 2010) showed that cases were more likely than controls to report weak ethnic identity. Variations in findings between the two studies are most likely due to methodological differences in terms of study design and measurement methods. It is, nonetheless, intriguing to speculate whether the impact of ethnic identity on risk for psychosis might vary by group and setting. While very speculative, it is possible that strong ethnic identity may be a proxy for access to social support networks in a particular BME group (e.g. Moroccan) and country (e.g. the Netherlands) and, by contrast, may not represent access to support, but compound risk in another group (e.g. Black Caribbean) and country (e.g. the UK). However, this remains speculative and can only be addressed by future research with consistent methodologies and sufficient power across countries and BME groups. The only study that we are aware of that has considered ethnic identity in relation to psychosis in the BME population in the UK is a small study by Bhugra et al. (2010). They found an ambivalence of attitude in the Black Caribbean and Asian group with psychosis, in that BME cases reported weaker ethnic identification in some life domains than BME controls, but stronger ethnic identification in others. Both the Dutch and British study did not examine ethnic identification in the White majority group and thus did not allow for conclusions as to whether ethnic identification is associated with elevated risk of psychosis in the BME but not the White majority group.

In line with previous studies, we found social experience of contextual factors, i.e. perceived disadvantage compared to other individuals in society, to be relevant to acculturation processes. This is consistent with reports suggesting that contextual factors and perceptions of these (Kirkbride et al., 2007; Veling et al., 2008; Cooper et al., 2008) may contribute to the high rates of psychosis found in BME groups.

4.4. Ethnic identification, perceptions of disadvantage and psychosis

One possible mechanism of how ethnic identification may increase risk of psychosis in BME groups is that strong ethnic identification may be associated with increased social distance to, and isolation from, other members of the multi-ethnic society (Boidell et al., 2001; Kent and Bhui, 2003; Morgan et al., 2008). On one hand, individuals with strong ethnic identification with a BME group may perceive greater social distance to the White British majority. On the other, given the considerably lower levels of ethnic identification that we found in the BME compared to the White British group, we may further speculate that stronger ethnic identification in BME individuals may also increase social distance to individuals of the BME group. Indeed, perceived disadvantage compared with other individuals in society did
explain at least some of the excess risk of psychosis in the BME group associated with increased ethnic identification. This interpretation of our findings would explain why rates of psychosis are elevated in the UK but not in the country of origin (Bhugra et al., 1996; Mahy et al., 1999), as in the country of origin, first, there is no White British majority, and, second, levels of ethnic identification may be expected to be higher in the general population, thus providing no grounds for adversity to occur in this social and cultural context.

In conclusion, the current study identified increased ethnic identification as a factor modifying the effect of ethnicity on psychosis, providing evidence suggestive, however, not conclusive of a potential causal pathway into psychosis. At least some of this pathway was explained by a higher prevalence of perceived disadvantage in the BME population. These findings require careful replication before firm conclusions can be drawn. Future research faces, then, the challenge to simultaneously test how individual- and area-level factors combine across countries and ethnic groups to more fully explain the excess risk of psychosis in BME groups.

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Contributors

R.M., J.L. and P.J. designed the study and wrote the protocol. R.M., J.L., P.J. and G.D. had managerial responsibility for the successful completion of the study. C.M., P.D., K.M., P.F. and G.H. were involved in sample recruitment and data collection. Under the direction of C.M., T.C. and H.F., U.R. conducted the research question, conducted the analyses, and wrote each draft of the manuscript. C.M., T.C. and H.F. provided guidance and interpretation of the analyses. All authors contributed to and have approved the manuscript.

Conflict of interest

All authors declare that they have no conflicts of interest.

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