Mild hearing impairment and psychotic experiences in a normal aging population

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Abstract

Background: Hearing impairment (HI) in the elderly may be a risk factor for psychosis, but associations between HI and psychotic disorder or psychotic experiences have been reported more consistently in younger than in older populations. The aims of this study were to replicate the positive association between hearing impairment and psychotic experiences and to clarify any differences between groups of young and old individuals in a non-clinical, normal aging general population sample.

Methods: HI, assessed at baseline and at 3-year follow-up, and psychotic experiences, assessed at 3-year follow-up, were analysed in a group of 848 individuals aged 33 to 89 years between 1999 and 2004. HI was determined on the basis of both self-report and audiometric examination. The “psychoticism” and “paranoid ideation” subscales from the SCL-90-R were used to assess level of psychotic experiences.

Results: Self-reported hearing problems expressed as conversational HI ($\beta=0.080$, 95% CI: 0.23, 7.90, $p=0.038$) and subjective HI ($\beta=0.087$, 95%CI: 0.70, 10.30, $p=0.025$), but not audiometric objective HI, were associated with psychotic experiences. In those with hearing aids, associations with psychotic experiences were only present if accompanied by self-reported hearing problems that persisted in spite of the hearing aid. In addition, HI increased the risk for psychotic experiences specifically in younger rather than older individuals.

Conclusions: Self-reported hearing problems rather than audiometric or remediated hearing loss may contribute to the development of psychotic experiences in younger rather than in older individuals.

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Keywords: Hearing impairment; Psychotic experiences; Depressive feelings; General population

1. Introduction

Reviews of the literature show that approximately 10% to 18% of individuals in the general population report one or more psychotic experiences (Krabbendam et al., 2004; Laroi et al., 2006; Tien, 1991; van Os, 2003). Prevalent subclinical psychotic experiences likely have a degree of continuity with more severe psychotic states. For example, many of the factors that contribute to the risk of subclinical psychosis in the general population have been found to also predict the risk for clinical psychotic states such as schizophrenia.
(Johns and van Os, 2001; Peters et al., 1999) and individuals with subclinical psychotic experiences have a much higher probability of later making the transition to clinical psychotic disorder (Poulton et al., 2000). It has been suggested that hearing impairment (HI) increases the susceptibility to develop psychotic symptoms (Cooper, 1976; Cooper and Curry, 1976; Corbin and Eastwood, 1986; David et al., 1995; Livingston et al., 2001; Stefanis et al., 2006; Stein and Bienenfeld, 1986; David et al., 1995; Livingston et al., 2001; Stefanis et al., 2006; Stein and Bienenfeld, 1992; Thewissen et al., 2005). Hearing-impaired people may have a tendency to withdraw from social interaction, which could lead to social isolation, oversensitivity or hostile reactions towards the environment (Corbin and Eastwood, 1986). Degenerative diseases of the ear impair responsiveness to the interpersonal and social environment may increase feelings of vulnerability and cause hallucinations in the affected sensory modality in some individuals (Sadock and Sadock, 2000). Misperception of auditory stimuli can occur and these may lead to apparently inappropriate associations that may constitute the basis of paranoid ideations (Cooper and Curry, 1976). Although such associations between psychotic experiences and HI are widely hypothesised, evidence is scarce and results to date are contradictory. In addition, it remains unclear to what degree HI increases the risk for psychosis in people of different ages. While some studies suggest that HI-associated psychotic disorder is particularly prevalent in older persons (Cooper and Curry, 1976; Howard et al., 1994; Rodriguez-Ferrera et al., 2004), others have reported that HI acts as a risk factor for psychotic experiences in samples of young people (Stefanis et al., 2006; Thewissen et al., 2005). In a comparison between 109 hearing-impaired and 137 non-hearing-impaired individuals, Thewissen and colleagues (2005) showed that HI originating early in life constituted an independent risk factor for the development of psychotic experiences later in life in this population-based sample. More recently, these results were replicated and extended to the age range of late adolescence (Stefanis et al., 2006). The evidence to date suggests that particularly long-standing HI in the young will predispose to psychotic symptoms later in life. In line with this is the report by David et al. (1995), who investigated the association between HI and schizophrenia in a large cohort (N=50,000) of 18-year-old male Swedish conscripts and found that schizophrenia was 1.8 times more prevalent among the hard of hearing.

A question remains with regard to the mechanism by which HI may increase the risk for psychosis, in particular with regard to possible underlying mediation by other variables. For example, HI may also increase the risk for other domains of psychopathology, in particular depression. Indeed, acquired HI has been considered a risk factor for depression (Heine and Browning, 2002; Tsuruoka et al., 2001), possibly because sensory loss is often followed by a period in which adaptation to the loss occurs, resulting in feelings of grief and depression. Given the fact that the pathogenesis of psychosis is thought to involve, among others, an emotional pathway (Garety et al., 2001; Hanssen et al., 2005; Krabbendam and van Os, 2005; Krabbendam et al., 2005; Myin-Germeys and Os, 2007), one hypothesis is that emotional dysfunction arising in the context of HI may contribute to the onset of psychotic experiences.

The aims of the study were threefold. The first aim was to replicate the association between HI and psychotic experiences in a non-clinical general population sample. The second was to examine the hypothesis of possible mediation by emotional dysfunction. The final aim was to examine possible interaction of HI with age. It was hypothesised that (1) hearing-impaired individuals would have higher levels of psychotic symptoms and paranoid delusions, (2) this effect could be explained in part by the mediating effect of depressive feelings, (3) the association between HI and psychosis would be more prominent in younger individuals.

2. Methods

2.1. Participants

Subjects were participants in the Maastricht Aging Study (MAAS) (Jolles et al., 1995), a longitudinal study devoted to the age-related decline of memory and other cognitive functions in the normal aging population (Jolles et al., 1995; van Boxtel et al., 1998). Participants were recruited from a patient register of family practices (Registration Network Family Practices, R Nh; Metsemakers et al., 1992). Medical exclusion criteria were previous coma, cerebrovascular pathology, tumours or congenital malformations of the nervous system, multiple sclerosis, Parkinsonism, epilepsy, dementia, organic psychosis, schizophrenia, affective psychosis or mental retardation. Data were collected at four points in time; at baseline (F0), and at 3 (F1), 6 (F2) and 9 years (F3) after baseline assessment. The reference measurement for the present study, F2, consisted of more male participants (z = 2.73, p < 0.001), was younger (t = 4.95, p < 0.001) and had higher SES (t = − 4.99, p < 0.001) as compared to the group at baseline. Since only data collected at F2 and F3 were used, these will be referred to as T0 (or
baseline) and $T_1$ (3 years after baseline), respectively. The group at $T_0$ consisted of 1031 individuals. Over the 3-year follow-up period, 210 subjects were lost to follow-up due to various reasons: no contact ($n=54$), deceased ($n=51$), too old or illness ($n=30$) and loss of interest ($n=75$). Socioeconomic status (SES) was defined as monthly level of income, divided into twelve categories which were collapsed into three main groups for the final analyses (‘low’ $<1361\€$, ‘medium’ $1362–2268\€$ and ‘high’ $>2269\€$).

### 2.2. Measurement of hearing impairment

Objective assessment of hearing function was done at $T_0$ using pure tone audiometry at four different frequencies (500, 1000, 2000 and 4000 Hz) which are relevant for speech comprehension. Detection thresholds were determined for each ear separately in steps of 5 dB (Jolles et al., 1995; van Boxtel et al., 2000). Objective hearing function was divided into ‘0’, non-impaired, and ‘1’, impaired, when a loss of more than 35 dB at the best ear was present (Davis, 1995). Subjective evaluation of hearing function was assessed at $T_1$ by asking each participant to judge his or her own auditory function (hereafter: subjective HI), in addition to a question about whether one experienced problems in following conversations in groups of four to five people (hereafter: conversational HI), with value labels ‘0’ no and ‘1’ yes. These questions have been shown to be effective in identifying hearing loss in different age groups and were found to be insensitive to language or culture (Nondahl et al., 1998; Torre et al., 2006; van Boxtel et al., 2000). Information on the use of hearing aids was added to create dummy variables; ‘1’ impaired, and ‘0’ no impairment set as reference group. This was done as the additional presence of hearing aids confers a greater level of validation to the exposure (Thewissen et al., 2005).

### 2.3. Measurements of psychotic experiences and paranoid ideation

The psychoticism (SCL-psy) and paranoid ideation (SCL-par) subscales of the SCL-90-R were used to assess level of psychotic experiences and paranoid ideation at $T_1$. The SCL-90-R is a multidimensional self-report inventory consisting of 90 items (Arrindell and Ettema, 1986, 2003; Derogatis, 1983). The instrument has been designed to measure psychopathology as a continuous dimension of human experiences and enables screening of nine dimensions of psychopathology. The two scales consisted of ten and six questions respectively. The SCL-psy subscale included questions such as: “Do you ever have the feeling that other people can control your thoughts?” The SCL-par subscale included questions such as “Do you ever have the feeling that you are watched or talked about by others?”

Participants were asked to rate these questions on a 5-point Likert scale, with ‘1’ representing ‘not at all’ and ‘5’ representing ‘very much’. Sum scores were calculated and a previously described (Henquet et al., 2005; Spauwen et al., 2006) single outcome measure was created by combining the two subscales in a single psychosis dimension reflecting psychotic experiences (hereafter: PS; range: 16–80). The SCL-90-R depression subscale was used to measure the presence of depressive symptoms (hereafter: DS; range: 15–75).

### 2.4. Statistical analyses

All analyses were performed using Stata version 9.0 (StataCorp, 2005). Associations between HI as exposure and PS as response variable were assessed using multiple regression. The sample size was considered sufficiently large to use regression analysis on the expected skewed distribution of the SCL-90-R subscales outcome (Ross, 1976). A priori defined confounding factors were adjusted for: age, sex and SES. Mediation by emotional dysfunction was investigated by adjusting for DS. Effect sizes of the association between HI and PS on the one hand, and between HI and DS on the other were compared using the MVREG routine in STATA. Effect modification by age was examined by including the Age×HI interaction term. Associations at the two-sided $p\leq0.05$ level were considered statistically significant.

### 3. Results

#### 3.1. Descriptive statistics

Included in the analysis were 848 individuals with non-missing data on HI and PS (Table 1). There was a small but significant association between age and PS (Pearson $r=0.093$, $p=0.007$). $T_0$ prevalence of objective HI was 9.4%. At $T_1$, 11.0% reported having hearing problems (subjective HI) and 10.8% had problems in following conversations in groups consisting of more than four people (conversational HI). Of the total sample 8.2% used a hearing aid. $T_0$ objective and $T_1$ conversational HI (OR 9.44, 95% CI 5.57, 16.00, $p<0.001$), $T_0$ objective and $T_1$ subjective HI (OR 9.93, 95% CI 5.83, 16.92, $p<0.001$) and $T_1$ subjective and $T_1$...
Table 1
Characteristics of the sample at T1 (n=848)

<table>
<thead>
<tr>
<th>Age distribution, n (%)</th>
<th>Mean age in years (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young (&lt;50 years)</td>
<td>57.2 (14.3)</td>
</tr>
<tr>
<td>Middle aged (50–70 years)</td>
<td>21.77 (7.82)</td>
</tr>
<tr>
<td>Old (&gt;70 years)</td>
<td>23.80 (8.87)</td>
</tr>
<tr>
<td>Male gender, n (%)</td>
<td>437 (51.5)</td>
</tr>
</tbody>
</table>

Table 2
Zero-order correlations (Pearson) between variables that were included in the regression model (N=848; numbers available for pairwise correlations may be smaller)

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>2. Sex</td>
<td>0.017</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>3. Income</td>
<td>–0.271*</td>
<td>–0.136**</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>4. PS</td>
<td>–0.085*</td>
<td>0.009</td>
<td>–0.216**</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>5. DS</td>
<td>0.079*</td>
<td>0.116*</td>
<td>–0.209**</td>
<td>0.850**</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>6. Hearing aid</td>
<td>0.127**</td>
<td>–0.069</td>
<td>–0.116*</td>
<td>0.094*</td>
<td>0.099*</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>7. Conversational HI</td>
<td>0.250**</td>
<td>–0.021</td>
<td>–0.186**</td>
<td>0.065</td>
<td>0.104*</td>
<td>0.394**</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>8. Subjective HI</td>
<td>0.246**</td>
<td>–0.068</td>
<td>–0.099*</td>
<td>0.023</td>
<td>0.055</td>
<td>0.370**</td>
<td>0.508**</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>9. Objective HI</td>
<td>0.365**</td>
<td>–0.078*</td>
<td>–0.116*</td>
<td>0.025</td>
<td>0.016</td>
<td>0.453**</td>
<td>0.390**</td>
<td>0.404**</td>
<td>–</td>
</tr>
</tbody>
</table>

Note: *p<0.05 and **p<0.001. PS=psychotic symptoms; DS=depressive symptoms.

3.2. Hearing impairment and psychotic experiences at T1

Logistic regression with age as predictor revealed that objective HI (OR 1.16, 95% CI: 1.12, 1.19, p<0.001) at T0, and subjective (OR 1.06, 95% CI: 1.04, 1.08, p<0.001) and conversational HI (OR 1.06, 95% CI: 1.04, 1.08, p<0.001) at T1 were all positively associated with age. Table 2 presents zero-order correlations between predictors and outcome variables. Regression analysis showed significant associations between T1 conversational HI and T1 PS (β=0.080, 95% CI: 0.23, 7.90, p=0.038) and between T1 subjective HI and T1 PS (β=0.087, 95% CI: 0.70, 10.30, p=0.025). Both associations were only present if self-reported hearing problems persisted in spite of the use of hearing aids. The same pattern was present for the association between T1 conversational HI and T1 DS (β=0.079, 95% CI: 0.24, 9.73, p=0.040), and T1 subjective HI and T1 DS (β=0.084, 95% CI: 0.59, 12.55, p=0.031). The presented associations were more prominent for DS than for PS (F=4.27, p=0.04) and disappeared after testing for possible mediation in the model (Table 3). A negative interaction was found between age and conversational HI in the model of PS which was solely present in the group without hearing aids (β=−0.745, 95% CI: −0.49, −0.13, p=0.001).

4. Discussion

As expected, HI was more prevalent among the elderly in this normal aging population sample. Not only did older people complain more about hearing loss, the prevalence of objective HI was also much higher. Participants with self-reported hearing loss (subjective and conversational HI) scored higher on the PS subscale than those participants without complaints. However, this association with psychotic experiences was only present if the self-reported hearing problems persisted in spite of the use of a hearing aid. On the contrary, objective HI was not associated with higher scores on the PS subscale. This suggests that not the objective impairment, but the perception of impairment contributed to the development of psychotic experiences in this population sample. Although all HI variables used in this study were strongly interrelated, self-reported HI assessment probably indexes a different aspect of hearing loss than...
objective HI. Self-reported hearing difficulties are an accepted indicator of true hearing loss, but additionally has also been found to measure the psychosocial problems associated with HI (Hashimoto et al., 2004). And likewise, conversational problems will often be the presenting complaint in individuals with HI, but may not yet be consciously appraised as evidence of sensory impairment.

Although numerous studies have reported evidence for a contributory role of emotional processes in symptom formation, pathogenesis and maintenance of psychosis (Hanssen et al., 2005; Krabbendam and van Os, 2005; Krabbendam et al., 2005), no evidence for a mediating role of depressive feelings in the association between self-reported hearing problems and psychotic experiences was found. Although individuals with conversational and subjective HI had stronger evidence of depressive feelings than psychotic experiences, these were unlikely to mediate the presented associations. The strong association between HI and depressive feelings is in accordance with earlier research in which acquired HI has been considered a risk factor for depression. Sensory loss often results in feelings of grief and depression (Heine and Browning, 2002; Tsuruoka et al., 2001). However, in our data this association also disappeared after testing for possible mediation by psychotic experiences.

A negative interaction was found between age and conversational HI in the association with psychotic experiences at $T_1$. Younger age increased the susceptibility of developing psychotic experiences in the presence of conversational HI. However, no evidence was found for a risk-increasing effect of conversational HI on psychosis outcome in older participants. These results are in agreement with previous research, even though different age categories were used to define “young” and “old”. The association between HI and psychotic experiences is probably an age-dependent one which tends to be absent in older persons. Cooper (1976) postulated that especially HI with early onset and long duration and severity would be of aetiological significance. More recently, it was shown that individuals with long-standing HI were more likely to develop psychotic symptoms and paranoid ideation (Stefanis et al., 2006).

Interpretation of the results should be made with some caution given the fact that not all variables could be measured at the same period in time. Conditions may have changed in 3 years, which could have attenuated associations between the variables that were measured, leading to a potential underestimation of the observed associations. Furthermore, bias may have occurred if participants who remained in the study differed systematically from those individuals who were lost to follow-up with respect to key exposures and outcomes. The possibility that those individuals who were lost to follow-up had low rates of HI and more experiences of psychopathology, thus biasing the results, cannot be ruled out by the present data set. It must also be kept in mind that the study design does not allow us to draw any conclusions about causality since information on level of psychotic experiences at baseline is absent. As such, conversational HI might be a consequence rather

### Table 3
Overview of the multiple regression analyses with PS and DS as dependent variables ($N=848$)

<table>
<thead>
<tr>
<th></th>
<th>DS</th>
<th></th>
<th>PS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>95% CI</td>
<td>$\beta$</td>
<td>95% CI</td>
</tr>
<tr>
<td>1.</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Conversational HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without HA</td>
<td>0.064</td>
<td>-0.41, 5.02</td>
<td>0.037</td>
<td>-1.13, 3.26</td>
</tr>
<tr>
<td>With HA</td>
<td>0.079</td>
<td>0.24, 9.73*</td>
<td>0.080</td>
<td>0.23, 7.90*</td>
</tr>
<tr>
<td>Subjective HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without HA</td>
<td>0.031</td>
<td>-1.50, 3.53</td>
<td>0.022</td>
<td>-1.44, 2.60</td>
</tr>
<tr>
<td>With HA</td>
<td>0.084</td>
<td>0.59, 12.55*</td>
<td>0.087</td>
<td>0.70, 10.30*</td>
</tr>
<tr>
<td>Objective HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without HA</td>
<td>-0.035</td>
<td>-4.41, 1.62</td>
<td>-0.040</td>
<td>-3.73, 1.13</td>
</tr>
<tr>
<td>With HA</td>
<td>0.029</td>
<td>-2.35, 5.32</td>
<td>0.028</td>
<td>-1.92, 4.25</td>
</tr>
<tr>
<td>2.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conversational HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without HA</td>
<td>0.032</td>
<td>-0.18, 2.49</td>
<td>-0.019</td>
<td>-1.64, 0.52</td>
</tr>
<tr>
<td>With HA</td>
<td>0.010</td>
<td>-1.74, 2.94</td>
<td>0.011</td>
<td>-1.33, 2.45</td>
</tr>
<tr>
<td>Subjective HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without HA</td>
<td>0.012</td>
<td>-0.84, 1.62</td>
<td>-0.005</td>
<td>-1.12, 0.85</td>
</tr>
<tr>
<td>With HA</td>
<td>0.008</td>
<td>-2.34, 3.52</td>
<td>0.014</td>
<td>-1.50, 3.25</td>
</tr>
<tr>
<td>Objective HI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without HA</td>
<td>0.000</td>
<td>-1.44, 1.48</td>
<td>-0.010</td>
<td>-1.49, 0.86</td>
</tr>
<tr>
<td>With HA</td>
<td>0.004</td>
<td>-1.64, 2.07</td>
<td>0.003</td>
<td>-1.38, 1.62</td>
</tr>
</tbody>
</table>

Reported are standardised regression coefficients (beta), 95% confidence interval and level of significance ($^{*}p<0.05$ and $^{**}p<0.01$).

Note: HA = hearing aid; PS = psychotic symptoms; DS = depressive symptoms 1 = unadjusted effect sizes, 2 = effect sizes adjusted for DS as possible mediator for PS outcome and adjusted for PS as possible mediator for DS outcome, 3 = as in 2 and additionally adjusted for age, sex and SES.

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than a cause of psychotic symptoms. Although reverse causality cannot be excluded, empirical evidence to date suggests that this may not be a very likely mechanism (Cooper and Curry, 1976; Stefanis et al., 2006; Thewissen et al., 2005).

Another limitation concerns the assumption of the multidimensionality of the SCL-90-R. The SCL-90-R is developed as an instrument to measure distinct multidimensional subcategories of psychopathology (Arrindell and Ettema, 1986). This multidimensionality has recently been questioned. In a review (Hafkenscheid, 2004) it was argued that the SCL-90-R should be seen as a unidimensional screening instrument for general psychopathology, since all SCL subscales of the SCL-90-R were highly intercorrelated. Most of the variance of SCL psychotic experiences was explained by level of depression, and other predictors, such as income and sex, only showed minor contributions. Furthermore, symptoms of paranoia and psychotic experiences are much more attenuated in non-clinical samples, which could make it more difficult to measure this dimension of psychopathology in a sensitive fashion. Apart from these disadvantages, it has been shown that the SCL-90-R has good power to discriminate between patients and the community and many subscales show good convergent and divergent validity. It can be used very well in research settings to assess psychological functioning and distress (Kamphuis and Geurts, 2006; Schmitz et al., 2000).

It could be argued that auditory impairment and vulnerability to psychosis both share common precursors. Although we cannot exclude this based on our results, there is no evidence available to date to support this notion.

Finally, it has been argued that especially early onset, long-standing and severe hearing deficits (e.g., due to middle-ear infections in children) are of aetiological importance in the pathogenesis of psychotic experiences, rather than the quality of hearing loss. This suggests that the mode of action of deafness in paranoid psychosis is probably one in which changes in psychological functioning and social adaptation take place slowly and progressively over a prolonged period (Cooper, 1976; Cooper and Curry, 1976; Stefanis et al., 2006). Given this time frame, presbyacusia may not be as important in the aetiology of psychosis in the elderly (Cooper, 1976).

In the MAAS study, no distinction was made between different causes of hearing loss and age of onset. Variability in HI was even restricted at baseline, because HI that was incompatible with cognitive testing was used as an exclusion criterion. This might have weakened any existing association, in particular if exposure to HI over certain developmental periods is critical in relation to the development of psychosis.

5. Role of Funding Source

The study was funded by grants from the Dutch Government, Maastricht University Hospital and Maastricht University. These did not have a role in the design of this study or the results presented in the paper.

6. Contributors

Martin van Boxtel provided in part the rationale for the study, participated in the design of the study, data analysis and preparation of the manuscript. Jim van Os participated in formulating research questions and hypotheses to be addressed, analyses of the data, interpretation of the findings and writing up the results. Jelle Jolles is initiator and scientifically responsible for the MAAS study and member of the MAAS project group. Frans Verhey is a member of the MAAS project group and participated in the design of the study. Viviane Thewissen provided background information on the topic and helped writing up the results.

7. Conflict of Interest

Jim van Os is a speaker or member of the advisory board for Lilly, BMS, Janssen-Cilag, Lundbeck, Organon and AstraZeneca. He received grant or research support from Lilly, GSK, BMS and AstraZeneca. All other authors declare that they have no conflicts of interest.

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