Table 1. *Mean BMI score and number of overweight/obese adolescents in the UHR group and control group*

	Controls	UHR
Mean BMI score, kg/m^2 (S.D.)	20.1 (3.1)	22.2 (3.2)
Overweight, *n*	1	3
Obese, *n*	1	1

BMI, Body mass index; UHR, ultra-high-risk; S.D., standard deviation.

Using these criteria we assessed the number of adolescents with overweight or obesity in our control sample and clinical sample. Results are shown in Table 1.

In order to assess whether the subjects with overweight or obesity were driving the group differences in salivary levels of testosterone, we reanalysed our data with obese and overweight subjects excluded. Results of this new analysis showed that mean levels of testosterone were 20.9 (S.D. = 14.2) pg/ml for the UHR group and 35.6 (S.D. = 29.7) pg/ml for controls, as compared with 20.0 (S.D. = 13.6) pg/ml in the UHR group and 33.6 (S.D. = 28.2) pg/ml for controls in our initial analysis. The degree to which testosterone levels were lower in the UHR group as compared with controls did not change, as the effect size, Cohen's *d*, remained 0.7. Our sample size was too small to compare frequency of overweight/obesity in both groups, so we were not able to assess whether adolescent boys with prodromal symptoms were at increased risk for overweight/obesity.

In addition to analysing these group differences, we also used a more dimensional approach and assessed the correlation between BMI score and level of testosterone across all subjects (UHR and control groups collapsed). Pearson correlational analysis resulted in $r = -0.27$, $p = 0.16$, Spearman correlational analysis in $r = -0.05$, $p = 0.78$.

Although the increased risk for the metabolic syndrome in individuals with schizophrenia and the relationship between the metabolic syndrome and lower testosterone do suggest that obesity may help explain reduced levels of testosterone in adolescents with prodromal symptoms, data in our sample do not convincingly support this hypothesis. Exclusion of those with overweight or obesity did not substantially change our findings and no significant relationship between BMI score and levels of testosterone was found. Based on data in our study, we have to conclude that reduced levels of testosterone in our sample of adolescents cannot be attributed to adiposity. However, more studies, with larger sample sizes and a wider range of measures, are needed to further test the hypothesis put forward by Brietzke & Bressan.

Declaration of Interest

None.

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Letter to the Editor

Poor childhood mental health may explain linkages between trauma, cannabis use and later psychotic experiences

An accumulation of evidence indicates that early adverse experiences could enhance the risk of developing psychosis (Read *et al*. 2005). In addition, several meta-analyses support the idea that exposure to Δ^9-tetrahydrocannabinol, the psychoactive component of cannabis, may produce an increased risk of psychosis (e.g. Arseneault *et al*. 2004; Fergusson *et al*. 2006; Large *et al*. 2011). However, for numerous reasons, children and adolescents with poor mental health may be more likely than others to experience trauma or to go on to use drugs (Hawkins *et al*. 1992). Thus, initial mental health must be taken into consideration when evaluating if environmental risk

factors such as childhood adversity or cannabis use contribute to psychosis.

Surprisingly, this is often not the case (MacLeod & Hickman, 2010). A recent article published in *Psychological Medicine* neglected to consider initial psychotic symptoms or mental health more generally when demonstrating a large association between early experiences of non-consensual sex and psychosis in adulthood, particularly amongst cannabis users (Houston *et al*. 2011). It is difficult to ascertain from the extant literature how omitting a measure of initial mental health may affect the findings reported by Houston *et al*. (2011). I therefore utilized secondary data to identify if adjusting for the presence of hallucinations during childhood might diminish associations between non-consensual sex, cannabis use and experiencing hallucinations in adulthood.

Of 3649 participants with usable data from the 1970 British Birth Cohort sample (Elliot & Shepherd, 2006), 58.9% of males and 40.2% of participants reported having tried cannabis by age 29 years. Few males had experienced non-consensual sex (0.1%) whereas 1.6% of the female sample indicated they had been forced to have sex by age 16 years. In logistic regression analyses, which adjusted for the participant's socioeconomic background, females who had experienced non-consensual sex by age 16 years were at an elevated risk of visual and auditory hallucinations at age 29 years [$\chi^2 = 3.8$, $p = 0.05$, odds ratio (OR) 8.51, 95% confidence interval (CI) 0.99–73.28]. No such link was found for males who had experienced non-consensual sex, nor was there any evidence for either males or females that cannabis use interacted with non-consensual sex to produce hallucinations in adulthood. As expected, females who experienced hallucinations by age 16 years were at high risk of experiencing non-consensual sex in the same period ($\chi^2 = 7.29$, $p = 0.007$, OR 28.81, 95% CI 2.52–330.38). This finding opens up the possibility that the association between early non-consensual sex and later mental health problems may be confounded by initial psychotic symptoms. In support of this idea, adjusting for initial psychotic experiences eliminated any link between non-consensual sex in females and later visual or auditory hallucinations ($\chi^2 = 0.29$, $p = 0.59$, OR 2.43, 95% CI 0.09–62.88).

The empirical illustration above, although limited to hallucinations, does indicate that the findings of Houston *et al*. (2011) should be interpreted with caution. Further research is needed to test if non-consensual sex in childhood combines with cannabis use to place people at enhanced risk of later psychotic experiences *over and above the presence of initial psychotic symptoms or poor mental health*. In the past, methodologically more robust studies have taken the approach of adjusting for initial symptoms of psychosis or excluding participants with a diagnosis of psychosis at baseline (e.g. see Moore *et al*. 2007). Whilst this step is useful, subclinical psychotic experiences and poor mental health should also be adjusted for. This is because subtle individual differences in mental health amongst those without psychosis at baseline could be the primary determinant of later psychosis.

The study by Houston *et al*. (2011) relied on retrospective accounts of environmental exposures and due to the cross-sectional nature of the data collected could not assess the subjective experiences of participants at baseline. However, an attempt could have been made to elicit retrospective information relating to psychotic symptoms (e.g. age of onset, age of first diagnosis if applicable). This information would have helped the researchers to gauge the likelihood that sexual abuse and cannabis use may combine to produce an independent risk of psychosis. Future research would contribute key policy-relevant insights by clarifying the temporal interrelationships between childhood trauma, cannabis use and early mental health and the role of each factor in the development of psychosis (Nelson & Mann, 2011).

Declaration of Interest

None.

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