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Contents

Mentalising impairment as a trait marker of schizophrenia?

One of the most controversial issues in 'theory of mind' research in schizophrenia in recent years has been whether theory of mind impairment may be seen as a trait marker or rather linked to particular symptoms. Sprong *et al*[1] conclude that evidence to date seems to favour the notion that mentalising impairment represents a possible trait marker. We believe that their meta-analysis is an excellent piece of scientific work but that this conclusion should remain tentative.

First, the existing evidence on theory of mind abilities in remitted patients is limited and difficult to interpret because of methodological shortcomings, such as non-explicit criteria for remission and poor control of cognitive abilities in the experimental design. A recent study by our group revealed that as a whole, stable patients did not show theory of mind impairment compared with carefully matched non-psychiatric controls. When standard consensus criteria for remission were applied to the sample, half failed to meet criteria for remission and showed a significantly worse theory of mind performance than remitted patients and controls. Specific theory of mind deficits in this group were associated with delusions. Thus, specific theory of mind impairment could go hand-in-hand with the presence of symptoms.[2]

Second, findings of theory of mind impairment in schizophrenia high-risk groups seem to support the assumption that theory of mind deficits represent a trait marker of the disorder. However, since these studies are mostly correlational, it is possible that the continuity of theory of mind deficits among 'at risk' groups may in fact derive from an intrinsic relationship between a psychotic symptoms continuum and theory of mind impairment. A review of the literature of theory of mind and schizotypal personality traits reveals that studies finding a positive significant relationship do so mainly with respect to schizotypal positive traits such as the cognitive-perceptual and unusual experiences dimensions of the schizotypy instruments.[3] Regarding investigations of first-degree relatives, evidence is controversial,[1] with findings of impaired performance on the more common types of theory of mind tasks but not on the 'eyes' test. However, it should be noted from these studies that those controlling for subclinical symptoms or schizotypal traits conclude that the association may be linked exclusively to the presence of subclinical positive symptoms.[4,5]

In our opinion, the existing evidence in theory of mind research is still limited but the possibility of a state-like association should not be ruled out. The most methodologically sound means to explore this would be to carry out longitudinal studies comparing theory of mind abilities in different phases of the illness, defined by explicit criteria. Future studies also need to differentiate between the affective and cognitive aspects of theory of mind, since it is possible that these show a different pattern of relationship with symptom clusters or schizophrenia profiles. Furthermore, it is possible that future research reveals that state–trait interactions may be occurring.

1 Sprong M, Schothorst P, Vos E, Hox J, van Engeland H. Theory of mind in schizophrenia: meta-analysis. *Br J Psychiatry* 2007; **191**: 5–13.

2 Pousa E, Duñó R, Brébion G, David AS, Ruiz AI, Obiols JE. Theory of mind deficits in chronic schizophrenia: evidence for state dependence. *Psychiatry Res* 2007; in press.

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4 Irani F, Platek SM, Panyavin IS, Calkins ME, Kohler C, Siegel SJ, Schachter M, Gur RE, Gur RC. Self-face recognition and theory of mind in patients with schizophrenia and first-degree relatives. *Schizophr Res* 2006; **88**: 151–60.

5 Marjoram D, Miller P, McIntosh AM, Cunningham Owens DG, Johnstone EC, Lawrie S. A neuropsychological investigation into 'Theory of Mind' and enhanced risk of schizophrenia. *Psychiatry Res* 2006; **144**: 29–37.

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Authors' reply: Pousa *et al* comment that our conclusion that theory of mind impairment represents a possible trait marker for schizophrenia should remain tentative for two reasons. Regarding their first argument, data on remitted patients are indeed limited and have methodological shortcomings. Only five studies in remitted patients were available, and the number of remitted patients in each of these studies was small. We also remarked that the criteria for remission used may have varied across studies, and that other factors may have influenced the results. Thus, we agree that the conclusion that theory of mind impairment represents a trait marker for schizophrenia should be tentative. In fact, we did describe it as a 'possible' trait marker. It is important to note that meta-analyses are about effect sizes rather than significance levels. By synthesising data of multiple studies there is more statistical power to detect smaller group differences. Thus, although in three out of five studies the theory of mind impairment in remitted patients was not statistically significant, when the studies were combined, the overall effect was significant (mean $d=-0.692$, $P<0.01$). So when Pousa *et al* do no find theory of mind impairment in stable remitted patients, we are not only interested in the *P*-levels, but also in the effect size. We also agree with the second point that there is evidence of an association between psychotic symptoms and theory of mind impairment, but do not see why this would argue against our conclusion. Frith[1] already proposed associations between specific schizophrenia symptoms (e.g. paranoid delusions) and mentalising impairment, and in their upcoming paper Pousa *et al* apparently also find significant associations between theory of mind impairment and psychotic symptoms. Perhaps we should have stated that theory of mind impairment is a possible trait marker for psychosis rather than schizophrenia. We believe that theory of mind probably does not represent an 'all or nothing' skill, and that schizophrenia should perhaps be studied using a dimensional instead of a categorical approach.

1 Frith CD. *The Cognitive Neuropsychology of Schizophrenia*. Psychology Press, 1992.

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Month of birth in relation to suicide

Salib & Cortina-Borja[1] find that persons born during the spring–summer season of April, May and June were significantly more likely to die by suicide than those born during other months: they find a peak for May and a trough for October.

However, they misreport our earlier results in this field when they state in the introduction that 'Chotai *et al*[2] reported that people born in winter in Sweden were significantly more likely than those with other birth seasons to have used hanging as a suicide method'. They further misreport earlier findings of ours when they state in the discussion that: '. . . winter variations in serotonin reported by Chotai & Åsberg[3] are inconsistent with the findings of this study, essentially the opposite of the Swedish findings'.

Our earlier findings are in fact similar to and consistent with the results of Salib & Cortina-Borja. In Chotai *et al*[2] we clearly show that those who preferred hanging rather than poisoning or petrol gases were significantly more likely to be born during February–April. In Chotai & Åsberg[3] we demonstrate that those born during February–April had significantly lower levels of 5-hydroindoleacetic acid (5-HIAA).

We have also published cosine analyses of our data,[4] in which we found that the minimum of the month-of-birth curve for 5-HIAA was obtained for the birth month April (*t*-min 3.4, Table 1, where the interval 3–4 depicts April) and the maximum was obtained for October (*t*-max 9.4). We also reported that the maximum of the month-of-birth curve for preferring hanging was for March–April and the minimum was for September–October.

Low serotonin turnover has been implicated as a risk factor for suicidal behaviour, particularly with violent or lethal methods of suicide, as discussed by Salib & Cortina-Borja.[1] Thus, our findings are in line with those of Salib & Cortina-Borja regarding suicidality, since we obtained a peak for the birth month April comparable to their peak for May, and found a trough for 5-HIAA for the birth month April.

In another epidemiological study,[5] we report that season of birth association with suicide methods is found in those without a history of psychiatric contacts, but not in those with such a history. We have argued that season of birth associations for suicide methods are likely to be mediated to a large extent by a suicidality trait independently of specific major psychiatric disorders, with serotonin as the likely underlying neurotransmitter.

In our studies, the season of birth variation was found for hanging as the suicide method, but not for other methods often denoted as violent, for example firearms or drowning. Hanging is a more universal method of suicide, and gender differences in the proportion of hanging are much lower than for other methods. In this light, it would be of interest to analyse the data of Salib & Cortina-Borja, specifically with regard to whether there is a month of birth variation in suicide by hanging.

1 Salib E, Cortina-Borja M. Effect of month of birth on the risk of suicide. *Br J Psychiatry* 2006; **188**: 416–22.

2 Chotai J, Salander Renberg E, Jacobsson L. Season of birth associated with the age and method of suicide. *Arch Suicide Res* 1999; **5**: 245–54.

3 Chotai J, Åsberg M. Variations in CSF monoamine metabolites according to the season of birth. *Neuropsychobiology* 1999; **39**: 57–62.

4 Chotai J, Adolfsson R. Converging evidence suggests that monoamine neurotransmitter turnover in human adults is associated with their season of birth. *Eur Arch Psychiatry Clin Neurosci* 2002; **252**: 130–4.

5 Chotai J, Salander Renberg E. Season of birth variations in suicide methods in relation to any history of psychiatric contacts support an independent suicidality trait. *J Affect Disord* 2002; **69**: 69–81.

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Salib & Cortina-Borja[1] describe a disproportional excess of people who kill themselves when born in early winter and between late spring and midsummer, and a disproportional deficit when born in late autumn. This month of birth effect can be interpreted in the context of another unexplained characteristic, namely the increasing south–north gradient (i.e. the geographical latitude effect, as shown in different countries).

Optimal maturation of the oocyte in animals and humans has been proposed to occur during the prime time of the seasonally-bound ovulatory seasons and to lead to optimal development of the zygote leading to less morbidity during pregnancy, birth and adulthood. In contrast, non-optimal maturation would occur during the inherent transitional stages leading to errant early neural migration and/or developmental differentiation.[2] This seasonally-bound month of birth effect is recognised in the presented data, particularly in females (violent and non-violent methods) and males (non-violent methods), and in anencephalia, schizophrenia and related diseases such as eating disorders.[3] This concept also explains the shorter life expectancy for people born during the first part of the year *v.* the longer expectancy during the second part, and its mirror image on the southern hemispere.[4]

Seasonality of the ovulatory pattern as cause of month of birth effect on suicide can easily be connected with the geographical latitude effect. In fact, the consistent relation between timing of mating seasons in different animals and humans causes stronger transitional stages the further distanced from the equator and, thus, higher frequency of non-optimal maturation of the oocytes. This biological phenomenon explains the mentioned geographical latitude effect on suicidality, schizophrenia and congenital anomalies of the nervous system, diverging between both hemispheres. The highly biased tertiary gender ratio in both suicidality and schizophrenia, and other high-risk factors such as teenage motherhood, multiparity and intrauterine growth retardation,[5] are quite compatible with this concept. This month of birth factor, therefore, does not need to be interpreted in terms of the 'foetal origins' hypothesis, nor the 'maternal–foetal origins' hypothesis, as suggested by the authors, but rather of the 'oocyte origins' hypothesis.

1 Salib E, Cortina-Borja M. Effect of month of birth on the risk of suicide. *Br J Psychiatry* 2006; **188**: 416–22.

2 Jongbloet PH. The effects of preovulatory overripeness of human eggs on development. In *Aging Gametes. Their Biology and Pathology* (ed RJ Blandau): 300–29. Karger, 1975.

3 Jongbloet PH, Groenewoud HMM, Roeleveld N. Seasonally-bound ovopathy versus 'temperature at conception' as cause for anorexia nervosa. *Int J Eat Disord* 2005; **38**: 236–43.

4 Doblhammer G, Vaupel JW. Lifespan depends on month of birth. *Proc Natl Acad Sci USA* 2001; **98**: 2934–9.

5 Mittendorfer-Rutz E, Rasmussen F, Wasserman D. Restricted fetal growth and adverse maternal psycholosocial and socioeconomic conditions as risk factors for suicidal behaviour of offspring: a cohort study. *Lancet* 2004; **364**: 1335–40.