Conclusions: There is a need to ensure that resilience programs are adapted to enhance their cultural safety and relevance. To articulate the impact of adapted programs, mixed methodologies are essential.

Autumn birth and the cognitive deficit schizophrenia subtype

D Rock, A Jablensky, S Howell

Clinical Research and Neurophysiology, Centre for Clinical Research in Neuropsychiatry, University of Western Australia, Perth, Australia

Background: Seasonal asymmetry of schizophrenia births is a well-documented phenomenon, with most studies finding a winter/early spring excess. This effect is consistent in the northern hemisphere but rare in the southern hemisphere. In Western Australia, using an endophenotype-based approach, we delineated a discrete, genetically distinct subtype of schizophrenia characterized by pervasive cognitive deficits (CDs), with the residual cases being cognitively spared (CS) (Hallmayer et al. 2005).

Purpose: We compared birth seasonality in Australianborn patients with CD and CS subtypes.

Methods: Data were grouped according to month of birth, with calendar adjustment. Season of birth was calculated using the Kuipers grouped method (Freedman 1979).

Results: The CD, but not the CS, subtype showed a significant season-of-birth effect, with an autumn peak (April) and a summer trough.

Conclusions: Analysis of northern hemisphere studies (Messias et al. 2004) found that the deficit syndrome of schizophrenia (Kirkpatrick et al. 2001), sharing some characteristics with our CD subtype, has a peak of summer births, at variance with the winter peak for all schizophrenia. Although the peak for CD is in autumn, it too contrasts with the spring peak of all schizophrenia births in Western Australia (Morgan et al. 2000). Despite numerous studies showing birth seasonality in schizophrenia, its cause remains elusive. Geographical differences in exposure to circannually fluctuating, nongenetic risk factor(s) may underlie this phenomenon (McGrath & Welham 1999). A challenge for future studies is to map the seasonal relationship between schizophrenia endophenotypes and risk factors such as low birth weight.

Using the semantic priming task in schizophrenia research: methodological and theoretical considerations

A Stefanovic, S Rossell

Mental Health Research Institute, Melbourne, Australia

Background: Schizophrenia is characterized by disturbances in language and thought. Semantic priming (SP) paradigms have been frequently used to investigate language function in schizophrenia. The SP effect is the reaction time advantage that is achieved by priming a target to which a participant is responding with a semantically or associatively related word. In schizophrenia, this area of research has produced many contradictory results.

Methods: This is a comprehensive up-to-date review of research on SP in schizophrenia. It considers the significance of the specific task parameters used and the characteristics of the patient sample as possible reasons underlying discrepancies.

Results: From this review, it has been established that there are two robust variables that produce different results in schizophrenia. First, the relatedness proportion effect, where low proportions of related primetarget pairs result in reduced or normal SP in people with schizophrenia, while higher proportions lead to increased SP. Second, using indirectly related primetarget pairs results in increased SP in schizophrenia. Further, in terms of patient characteristics, patients with thought disorder produce the most consistently abnormal SP results.

Conclusions: The results indicate that enhanced automatic spread of activation might be one of the causes of language deficits in schizophrenia. In the framework of distributed network models, it is possible that the patterns of representations overlap more between different or unusual concepts in people with schizophrenia compared with healthy people, especially those with the symptom of thought disorder.

Reduced dysbindin (DTNBP1) mRNA in hippocampus of patients with schizophrenia

CS Weickert¹, R Straub², J Kleinman², T Hyde², D Rothmond³

¹Neuroscience Institute of Schizophrenia and Allied Disorders (NISAD); ²CBDB, NIMH; and ³MiNDS Unit, NIMH, USA

Dysbindin has been implicated as an etiological factor in schizophrenia by genetic linkage studies, genetic association studies and molecular studies of postmortem brains of patients with schizophrenia. In this report, we