Introduction

ANDREW T. A. CHENG and BRIAN COOPER

The title of this supplement is that chosen for the Eighth Congress of the International Federation of Psychiatric Epidemiology, held in Taipei, Taiwan, on 6–9 March 1999 and co-sponsored by the Institute of Biomedical Sciences, Academia Sinica, and the Taiwanese Society of Psychiatry. In addition to the Federation's main aim of encouraging population-based research in different world regions, and especially in developing countries, the Taipei Congress was intended to improve communication between disciplines that in general still pursue such research quite independently of one another. The papers included here represent a small part of the scientific programme, selected for their special relevance to the central topic of the meeting.

The term *genome*, though comparatively recent, is now widely understood to refer to the total genetic complements of living organisms, and *genomics* to mean the study of their structure and functions. *Envirome* (or *environome*) has been proposed as a corresponding term for the totality of environmental conditions and processes that impinge upon human health and influence the risks for mortality and morbidity, and *enviromics* by analogy to denote the study of such influences. *Psychiatric enviromics* refers more specifically to studies of environmental conditions and processes that either promote mental health or increase the risk of mental disorder, whether early developmental influences comprising 'nurture' or subsequent exposures throughout the life span. *Gene–environment correlation* and *interaction* thus constitute aspects of the interplay of nature and nurture although, as several contributors emphasise, genetic and environmental factors do not act on one another directly (induced mutation apart), but in each case exercise their effects upon the individual phenotype at cellular, organic or individual level.

The special fascination of this research nexus stems from its position at the crossroads of psychiatry, medical and behavioural genetics, environmental science and medical sociology. Medical genetics, being concerned with genetic anomalies as causes of human disease, has traditionally concentrated on single-gene Mendelian disorders whose transmission could be mapped and were little affected by surrounding conditions. Today, however, genetic epidemiology deals increasingly with complex polygenetic disorders, in which inherited tendency and pathogenic exposures are jointly implicated. Among these must be numbered, on the evidence reviewed here, psychiatric illness categories such as schizophrenia, major affective disorder, alcohol dependence and late-life dementia.

In behavioural genetics, which explores the significance of inherited variation for manifold features of human behaviour, the notion of qualitative abnormality recedes into the background or even disappears. The discipline is none the less relevant in psychiatric research, whose range of inquiry extends from disease aetiology to the quantitative genetics of cognitive function and personality traits such as neuroticism and extraversion. Environmental medicine is concerned with natural and artificial hazards to human health, whether traumatic, toxic or biological in nature, and medical sociology with causal or predictive factors such as low social status, social deprivation, family dysfunction and stressful life events. Individual studies have confirmed the importance of all these various types of risk.

Gene–environment research in this field is, however, still in its infancy, and will take time to produce substantive findings. Three main areas of difficulty can be delineated. To begin with, although researchers have nominated many candidate genes for the major psychoses, few findings have been replicated and none as yet definitely confirmed. In each case lack of knowledge about the pathogenetic basis of these conditions renders the endeavour akin to searching for needles in the proverbial haystack. Moreover, some disorders may be related to anomalies of gene expression, which occur at distinct stages of development and are not detectable by cross-sectional studies. Second, research into the socio-environmental risk factors of mental illness has suffered increasing neglect since the 1980s and is in urgent need of revival. There is no overall frame of reference for assembling and classifying the great variety of hazards, known or putative, in different populations and cultures. Indeed, in many instances it is unclear whether the subject of investigation has any direct causal link with psychiatric risk, or simply serves as a convenient marker for some as yet unidentified hazard. Third, progress in this field will require the testing of specified forms of interaction occurring at different stages of the life cycle. Thus genetic inheritance combined with early developmental (including prenatal and perinatal) exposures may result in a latent sensitivity or vulnerability, so that later encounters with relatively mild or even ubiquitous forms of environmental stress can then provoke the clinical symptoms of illness.

While much more research will be necessary before the results of gene–environment studies can find practical application in treatment or prevention, encouragement is to be found in the beginnings of inter-disciplinary dialogue and co-operation, for which the contributions included here provide some evidence. Being based on conference presentations, these papers do not conform to any predefined schema, but are widely disparate in content and style. To ensure a degree of cohesion, and to emphasise the underlying thread of argument, they have been grouped into four sections. First, Steven Rose (2001, this supplement), James Anthony (2001, this supplement) and Jenae Neiderhiser (2001, this supplement) discuss from different standpoints the underlying conceptual and methodological issues. Next is a group of papers in which epidemiological research on genetics, neurobiology and environmental exposures is reviewed with respect to schizophrenia (Ming Tsuang *et al*, 2001, this supplement; Hiroshi Kunugi *et al*, 2001, this supplement; Povl Munk-Jørgensen & Henrik Ewald, 2001, this supplement), alcohol dependence (Andrew Heath *et al*, 2001, this supplement) and cognitive development (Robert Plomin & Ian Craig, 2001, this supplement; Te-Jen Lai *et al*, 2001, this supplement). In a third cluster of studies, predisposing or provoking

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social factors such as family adversity, low social status and stressful life events are introduced into the causal models for early-onset Alzheimer's disease (Lawrence Whalley, 2001, this supplement), functional psychosis in an immigrant population (Mandy Sharpley *et al*, 2001, this supplement) and the non-psychotic mental disorders common in survey populations (David Goldberg, 2001, this supplement; Jim van Os *et al*, 2001, this supplement; Angela Fan & William Eaton, 2001, this supplement; Jennifer Ritsher *et al*, 2001, this supplement). Finally, an attempt is made to review progress and assess future prospects for interdisciplinary research in this field (Brian Cooper, 2001, this supplement).

The Taipei Congress succeeded in its objective of bringing together clinicians and scientists from many countries who believe that this is the right direction for future advance, and we hope that this volume will help to reinforce the trend.

ANDREW T. A. CHENG, FRCPsych, Division of Epidemiology, Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan; BRIAN COOPER, FRCPsych, Institute of Psychiatry, King's College, London, UK

Correspondence: Professor Andrew Cheng, Institute of Biomedical Sciences, Academia Sinica, Taipei 11529, Taiwan. Fax: +88 62 27853569; e-mail: bmandrew@ccvax.sinica.edu.tw

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