of asymmetry are shifted in typical directions when the hypothesised RS+ gene is present. The gene may be absent, or when present its expression may be reduced by factors that influence early growth. Among the variables associated with reduction in the shift of the chance distribution for handedness are male gender, twinning, low birth weight, poor phonological processing (occurs in many people with dyslexia) and early brain lesions. These reductions must be detected against a base rate of non-right-handedness in about one-third of the general population. Differences in asymmetry are not causal, but rather the results of changes in the frequency or expression of the RS+ gene. They are not likely to be useful markers for any specific clinical disorder.

In schizophrenia, I have suggested that the gene may lose its directional coding and become 'agnostic' for right or left. Symptoms of schizophrenia are hypothesised to occur when speech cortex is impaired on both sides of the brain, as expected in 50% of the relevant genotypes. Until the RS+ gene and its variants are found, however, the theory remains a hypothesis.

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Genetic variation in European suicide rates

The fact that Hungary and Finland had among the highest reported suicide rates in Europe has led to speculations about the possible involvement of a common genetic factor in this phenomenon (Marušič & Farmer, 2001). Both Finns and Hungarians, as some linguists believe, belong to the Finno-Ugrian family of ethnic groups, with certain similarities in their ancient language. The high suicide rates in the various groups of Finno-Ugrians suggested to Kondrichin (1995) that 'during the early stages of Finno-Ugrian ethnogenesis certain behavioural traits predisposing to suicide became fixed in the gene pool'.

We (Hrdina & Faludi, 2001) have examined the available molecular genetic data on serotonergic candidate genes and their allelic association with suicide (Nielsen *et al*, 1994; Du *et al*, 2000) for any similarities or differences in allelic frequencies between the various populations, particularly between Finns and Hungarians. A direct comparison between the findings of association between serotonergic gene polymorphism and suicidal behaviour is difficult, since in the reports of positive associations different phenotypes (suicide attempt, completed suicide) were investigated. However, if certain serotonergic gene variants increase the disposition for, or vulnerability to, suicide in some populations that share higher rates of suicide and that may share some similarities in their ethno-historical origins, then the frequency of these predisposing gene variants should be comparable in those populations.

Table 1 summarises the allelic distributions of serotonergic gene polymorphisms in some selected populations. It is clearly apparent that the allelic distributions of the two polymorphisms (5-HT transporter S/L polymorphism and tryptophan hydroxylase gene 218 A/C polymorphism) are remarkably different in Hungarian and Finnish populations. In fact, the frequencies of the S and L alleles of the 5-HT transporter in the Hungarian subjects are closer to those found in the British population.

The limited scientific evidence so far would suggest that there is no Finno-Ugrian 'suicide gene' or a shared genetic risk factor. It is unlikely that such a complex phenomenon as suicidal behaviour is genetically determined by a single gene or even a few gene variants. A more likely scenario is that the genetic contribution to suicide will be represented by small size effects of many gene variants associated with processes involved in suicidal behaviour, and by interaction of these genetic factors with environmental ones.

Table 1 Allelic distribution of serotonergic gene polymorphisms in selected populations

Population	5-HT transporter gene S/L polymorphism		TPH gene 218 A/C polymorphism	
	S allele (%)	L allele (%)	Allele A (%)	Allele C (%)
Hungarian	48.8	51.2	46.4	53.6
Finnish	33.0	67.0	35.0	65.0
British	45.6	54.4	39.1	60.9

5-HT, serotonin; TPH, tryptophan hydroxylase.

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Chronic fatigue syndrome or neurasthenia?

The interesting study reported by Hickie *et al* (2002) draws attention to the prevalence of ICD–10 neurasthenia (World Health Organization, 1992) in a large sample of the Australian general population. The authors' findings are of the utmost importance for clinicians concerned with the disabling effects of fatigue but also provide food for thought in the wake of the CFS/ME Working Group (2002) report to the Chief Medical Officer. In this report, the term chronic fatigue syndrome/myalgic

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