therefore, that the authors failed to demonstrate larger ventricles in schizophrenic subjects, since their sample size (30 patients, 30 controls) gave them slightly less than a 50–50 chance of doing so.

The authors should be congratulated on a genuinely informative literature review, but it is a shame they felt unable to put their own prescriptions into practice. In illuminating the devil of the Type I error they have slipped into the deep blue sea of the Type II.

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## Schizophrenia: Recency Theory and Syphilis

SIR: Hare (*Journal*, October 1988, **153**, 521–531) argued that the historical origins of schizophrenia may have parallels with that of general paresis. He described the earlier global variation in the prevalence of paresis and the more recent increase in prevalence in non-Western countries, and judged from this that the changes in prevalence were real and probably attributable to changes in an environmental factor, i.e. the introduction of a new infectious agent.

The relative rarity of syphilis, and by extension of general paresis, in West Africa before 1950 may have been related to the endemicity of yaws; the rapid increase in the incidence of syphilis since then is thought to be related to the decrease in the prevalence of yaws following the WHO mass treatment campaign of the 1950s and 1960s (Perine, 1987).

Yaws is caused by *Treponema pertenue*, which is morphologically identical to *Treponema pallidum*, the cause of venereal syphilis. The increase in venereal syphilis since the mass treatment programmes may represent a decline in herd immunity to yaws, and thereby to syphilis. This thesis is not incompatible with the notion that venereal syphilis was a new disease. Venereal syphilis may well have derived from the endemic treponematoses (unitarian theory) which include pinta, yaws, bejel, and endemic syphilis (Csonka, 1987). However, the thesis suggests that rapid alterations in prevalence, especially in this case, may be as much to do with changes in herd immunity to a related pathogen as it is to do with introduction of a new pathogen.

I agree with Dr Hare that study of the historical origins of general paresis may throw some light on the schizophrenia debate and suggest that the curious relationship between rheumatoid arthritis and schizophrenia (if true) is reminiscent of that between yaws and venereal syphilis in the African population. Both rheumatoid arthritis and schizophrenia may be caused by related but different pathogens.

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## Tactile Extinction Phenomenon in Schizophrenia

SIR: With regard to Dr Cutting's comments (*Journal*, August 1988, **153**, 281) about our interpretation of the tactile extinction phenomenon in his review of *Etiopathogenetic Hypotheses of Schizophrenia: the impact of epidemiological, biochemical and neuromorfological studies*, the direct relationship between QET and "a disorder of some sort in the right hemisphere" is so far to be univocally demonstrated. Taking the neurophysiological pathway of tactile sensitivity into account, there is clinical evidence that both right contralateral and left frontal cerebral lesions cause a left-extinction phenomenon (Schwartz *et al*, 1977).

Attempts to clarify which of these (left homolateral or right contralateral) is responsible for our results have not given consistent results. Even if a neuropsychological interpretation of the QET suggests that left-extinguishing patients performed worse in right-hemisphere tasks (Gambini *et al*, 1986) the interpretation of these results still are not definitive. In fact, QET can be considered to be similar to other dichotic tasks (e.g. dichotic listening and, in general, simultaneous stimulations of the left and right sensorial pathways). Therefore, as Schwartz *et al* (1977, 1979) pointed out, it is reasonable to suggest that when a verbal response is needed, as in the QET paradigm, the two sensorial tactile pathways are quite different; in fact, the left-side stimulus has a longer indirect pathway to CNS in comparison with the stimulus from the right hand, which arrives directly to the left hemisphere. The longer way for the left-side stimulus includes fibres crossing from the right to the left hemisphere mainly through the anterior part of the corpus callosum. Thus it might be reasonably