splice variant may abolish the DNA binding activity of the N-terminal part and unmask the binding potential of the C-terminal part of the paired domain[17]. If this is the case, the phenotype–genotype correlation in this family suggests that the exon 5a variant has a role in retina formation, while the protein without exon 5a plays a part in the formation of anterior segments. This should be tested experimentally.

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# Research in India

Sir — I agree with your recent editorial (Indian incense and sensibility; *Nature Genet.* **6**, 1996) about the need to prevent exploitation of Indian subjects for genetic research by non-Indian scientists. Collaborations that benefit both Indian and foreign collaborators are also laudable. However, the Indian response to recent misdeeds — the imposition of cumbersome rules by an inflexible bureaucracy — is unlikely to prevent further exploitation. It may even hinder equitable collaboration, as my experience shows.

As an Indian expatriate, I have long been interested in fostering psychiatric genetics, my specialty, in India. My initial application to investigate the feasibility of genetic epidemiological studies in schizophrenia, which explicitly stated that I would not remove tissue samples from India, was processed for 13 months by no less than five Indian agencies. These included the Ministries of Health, Education and Home, as well as the Indian Council of Medical Research and the Intelligence Bureau. By the time the application was approved and I arrived in India, my Indian collaborator had decided to retire. Although finding another Indian collaborator was easy, obtaining approval for the alternative arrangement was not, even though the research project had not changed. My application languished in the Ministry of Health for over 12 weeks. No objections were raised during my meetings with concerned officials, but it seemed that no one wished to sign the letter of approval. With a few notable exceptions, most of the officials appeared disinterested or unaware of the importance of such research.

In view of these difficulties, it is not surprising that certain individuals are tempted to bypass the regulatory authorities entirely.

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# CNTF and psychiatric disorders

Sir — Thome and coworkers[1] suggest that a null mutation in the gene coding for the ciliary neurotrophic factor (CNTF)[2] is an important risk factor for the development of endogenous psychoses. They reported that the prevalence of the null mutation was significantly increased in psychiatric patients when compared to controls. This finding is of special interest since evidence has accumulated that a significant proportion of schizophrenia cases may neurodevelopmental origin. Neuropathological studies have found a reduction in hippocampal volume and cell numbers[3–5]. Possible causal factors include a genetic control of neurodevelopment[6].

We have studied 139 patients suffering from schizophrenia (49 females and 90 males) and 98 patients with bipolar affective dis-