

between *ceh-20* and *PBX2*, making this the most conserved region. One possible function for this region could be to provide additional DNA contacts, since the 5' end of the homeodomain has been shown to make contacts in the minor groove of the DNA<sup>12-14</sup>.

Our detection of the PBC domain in the PBX proteins has implications for the oncogenic mechanism of the E2A/PBX1 fusion protein. This chimaeric protein is missing the N-terminal 46 amino acids of the PBC domain. Thus, it cannot have the same transcription factor properties as *PBX1* if it lacks a presumably essential region that has been conserved from nematodes to humans. This argues against a simple model in which the regulatory elements of *E2A* inappropriately express a protein with normal *PBX1* transcription factor activities in pre-B cells, where it is normally absent. Rather, other features of this fusion protein, for example, the missing PBC N-terminal region or the fused *E2A* sequences, might cause its oncogenic activity.

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## BABI in dispute

Sir — We were concerned to read your editorial, *Nature Genetics* 1, 77–78; May 1992, “From genetics to revelation”, which we feel portrayed an unrealistic view of the place of *in vitro* fertilization in the field of prenatal diagnosis.

As the UK collaborators of Mark Hughes, to whom your editorial presumably refers, we do not feel that the neologism ‘BABI’ (Blastomere analysis before implantation) is helpful. There is a perfectly good and accepted term in the literature already, that is, ‘preimplantation diagnosis’; a term that covers screening at all the early stages of human development.

We are surprised to read in your

report that 660 embryos have been reimplanted with a remarkably high pregnancy rate. This is not true. As far as we are aware, our team at Hammersmith, London, is the only group to have successfully produced live babies after embryo biopsy and preimplantation diagnosis. In two years only seven babies have been born after preimplantation diagnosis, and much more work is required before we can judge the efficacy or safety of these difficult techniques. Until then, it is surely unwise for IVF units to set up a commercial service as is mentioned in your editorial.

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### Correction

In ‘No CFTR: are CF symptoms milder?’ contributed by Jeffrey J. Wine (*Nature Genet.* 1, 10; April 1992) the final two sentences of the second paragraph should have read: One of the earlier homozygous stop codon patients (R553X/W1316X)<sup>6</sup> has neither CFTR mRNA<sup>9</sup> nor CFTR protein<sup>10</sup>, and it was suggested that the  $\Delta F508$  mutation may cause a generalized processing or trafficking defect<sup>10</sup>. In short, the surprising notion that  $\Delta F508$  mutations are worse than the complete absence of CFTR protein has caught on<sup>4,6-10</sup>.

The discussion of 660 embryos referred to a sample of *in vitro* fertilization studies for infertility, and not blastomere analysis, which is still in its infancy. Preference for the neologism ‘BABI’ may simply reflect the cultural divide across the Atlantic (for example, see S. Fogle, *J. NIH Res.* 4, 46–48, June 1992). The Editor