inhabitants can experience the joint benefits or deficits of genes and environment. Another type of gene–environment correlation occurs if different parental genotypes are associated with systematic differences in rearing styles. In the usual, non-fostered breeding schemes with inbred strains, strain-specific maternal behaviors and uterine environments are perfectly correlated with one another and with the genotype of their offspring. In the Francis *et al.* study[3], systematic cross-fostering at different developmental stages could dissociate the usual gene–environment correlations based on intrauterine factors and/or maternal rearing practices. However, because the behavior of only one strain of mouse was used to investigate the combined effects of pre- and postnatal cross-fostering, it was not possible to test for the effects of genotype–environment interaction. The data clearly show that additive effects of pre- plus postnatal environments could not account for the results, as there was virtually no effect of either environmental manipulation alone; both were required to turn a genetic B6 mouse into a behavioral phenocopy of a BALB. As the authors note, extension of these experiments to other genotypes will be crucial to solving the question of mechanisms of interaction between pre- and postnatal environment effects and genotype.

Non-genomic transmission of behavioral differences involves persistent modulation of gene expression, but how this is achieved and for which genes are questions just beginning to be explored[7]. We also have yet to understand the mechanisms underlying the three-way interaction involving the gene, the prenatal environment and the postnatal environment (G E1 E2). The most likely source of the transformation from B6 to BALB behavior in the current study is subtle effects of BALB-specific maternal behavior acting on a nervous system that was slightly altered during intrauterine rearing by other BALB-specific influences (according to studies in which the first such observations were made[5,6]). Although the end result may be a spectrum of differences in the adult expression of particular genes in such doubly cross-fostered pups, such as those involved in hypothalamic–pituitary–adrenal axis regulation, we are still looking at complex patterns. It will be valuable to explore a range of behaviors that show sensitivity to the G E1 E2 interaction.

Finally, we should not overlook the role of the father. It is certain that paternal contribution does not stop at fertilization. Though paternal effects are often ignored in animal models, they apparently have a role in family conflict resolution even in mice[14]. In many, if not most, mouse colonies, the male remains with the female and her pups until weaning (although this practice was likely not followed by Francis *et al.*[3] in their cross-fostering design), and male mice show nesting-related behaviors such as pup retrieval. In humans, there is a long history of work tracing early developmental influences of both male and female parents on adult behavior. So maybe it really is not all Mom's fault.

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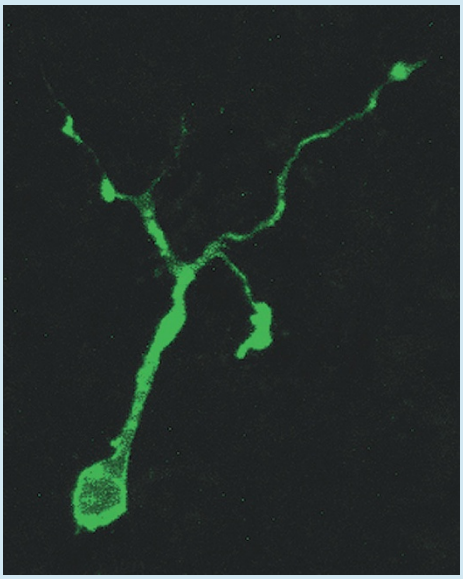
Young neurons in old brains



Neurons born in adulthood must develop in a far different environment from those born in young animals, particularly because the adult-born neurons must find their way through and integrate into functioning neural circuits. In this issue (pages 507–518), Pierre-Marie Lledo and colleagues report that the electrophysiological properties of adult-born neurons develop in a sequence different from that seen in young animals. New adult neurons born in the subventricular zone of the lateral ventricles migrate through mature neural tissue—first tangentially to the olfactory bulb and then radially to their final position. The authors used a replication-defective retrovirus expressing enhanced green fluorescent protein to identify these newborn cells in living brain tissue. Patch-clamp recordings showed that tangentially migrating neurons expressed extrasynaptic $GABA_A$ and AMPA receptors. NMDA receptors appeared later, in radially migrating neurons (shown in photo), in contrast to young tissue where NMDA receptors precede AMPA receptors. Spontaneous synaptic activity emerged soon after migration was completed. However, spiking activity did not occur until neurons were almost fully mature. This delayed maturation of excitability may serve to prevent the newborn cells from disrupting the function of circuitry already in place in the adult.

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