In the news

KEEPING UP WITH THE GREWCOCKS

Men who share the same surname have a 25% chance of being related, a British study reveals. The rarer the surname, the greater the chance of random pairs of surnames being related (*The Scotsman*, 22 February 2006). So there is little hope of building a large family tree for the Smiths, Joneses and Taylors, but there are much improved chances for the Attenboroughs and Grewcocks, for example.

For the study, which was published in *Current Biology* on 21 February 2006, researchers recruited 150 pairs of English men that shared the same surname but were not knowingly related. About a quarter of these men were genetically connected through their Y chromosome, meaning that they must have shared an ancestor more recently than 20 generations ago, or about AD 1300, when surnames were first used in the United Kingdom (*New Scientist*, 22 February 2006).

Y chromosomes are inherited from father to son, and surnames are inherited from father to children, so men who share surnames are also related. Has this work merely told us what we know already? Yes and no. Complicating factors such as illegitimacy, adoption and multiple originators would increase the chances that two people with the same surname would not be related (*Seed Magazine*, 27 February 2006).

That striking strong signal immediately suggests a practical application. Forensic scientists could use DNA retrieved from a crime scene to predict the surname of the suspect (*BBC News*, 21 February 2006) when used in combination with other intelligence. Indeed, Brian Sykes, from the University of Oxford, recommends that we create a Y-chromosome database (*The Scotsman*).

The bottom line is, if you think you've got criminal tendencies, you could do worse than change your name to Smith.

Tanita Casci

NEUROGENETICS

A male gene for a male brain

Male and female brains are different in important ways — not just in generating distinct sexual behaviours, but also in terms of cognition and other key functions. Until recently, these differences were thought to arise from the effects of sex hormones during brain development, but there have been several hints lately that genes in the adult brain have a role in maintaining this male–female divide. Work from Eric Vilain and colleagues now shows that the Y-chromosome gene *Sry* directly influences the function of the brain in adult males.

Sry is a transcriptional regulator and is best known as the master controller of male sexual development. However, previous studies have shown that, intriguingly, this gene is also expressed in the adult male brain. Vilain and colleagues confirmed this, showing that *Sry* mRNA is present at a low level throughout the brain cortex in male mice, and at higher levels in two regions: the mammillary bodies and the substantia nigra (SN). Focusing

CANCER GENETICS

From expression signatures to their regulators

Analyses of global gene-expression patterns have yielded molecular signatures that predict progression, prognosis and response to therapy for many tumour types. Adler and Lin *et al.* now take this approach a step farther — they have developed a genetic linkage-based method to identify key molecular players that regulate cancer-associated changes in transcriptional signatures.

With the aim of understanding the genetic determinants that underlie the characteristic and at times predictive changes in gene expression that occur in tumours, the authors developed a genome-wide genetic linkage method. In a procedure that the authors call SLAMS (stepwise linkage analysis of microarray signatures), the linkage of prospective regulator genes is first mapped to large chromosomal regions. This is achieved by looking for correlation between a specific expression signature and copy-number changes within particular genomic regions. This

> “…a general method that … can be adapted to identify linkage between expression signatures and other types of data, such as SNPs or DNA methylation.”

linkage is subsequently validated and refined by determining whether increased expression of candidate genes from within these regions is correlated with the signature, as would be expected if increased copy number of a gene had a regulatory effect. To improve the robustness of their analysis, Adler and Lin *et al.* considered the coordinated behaviour of many genes in an expression signature.

The authors previously identified a ‘wound response signature’, which is a powerful predictor of metastasis and poor prognosis in many tumour types. SLAMS was used to identify genetic regulators of this signature in breast cancer tissue. Using 37 samples, they identifed a region on 8q that strongly associated with the wound signature. Further refinement indicated that within this region it is the MYC oncogene and CSN5 (a catalytic subunit of the COP9 signalosome) that cooperate to generate the expression signature. Consistent with this prediction,

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