IN THE NEWS

Gay genes boost fertility

The worldwide press has recently rekindled the debate on sexual orientation: is it down to nature or nurture? A familiar argument from the nurture camp is this: "If male homosexuality has a genetic component and homosexuals reproduce less than heterosexuals, then why is this trait maintained in the population?" (*The Independent*, *UK*, 13 October, 2004). A study[1] now answers precisely this question: "The genes that make men gay also help their female relatives to have bigger families" (*The Times*, *UK*, 13 October, 2004). The scientist interviewed the families of 98 homosexual and 100 heterosexual men — a total of 4,600 individuals — and found that mothers and maternal aunts (but not paternal relatives) of the homosexuals were more fertile than those of the straight men, and also produced more gay offspring. This indicates that the gene(s) that favours homosexuality also boosts female fertility and that homosexuality is passed through the maternal line, and so might be located on the X chromosome.

But 'gay genes' might not necessarily increase fertility itself. Neuroscientist Simon LeVay suggests that the genes might in fact be involved in sexual attraction to men. "They could predispose men towards homosexuality and women towards 'hyper-heterosexuality', causing women to have more sex with men and thus have more offspring" (*New Scientist*, *UK*, 13 October, 2004).

The research team that carried out the study point out that increased fertility linked with homosexuality in males "would not explain the majority (80%) of cases" (*BBC News Online*, *UK*, 13 October, 2004), and "stressed that there was ample room left for the influence of non-biological factors linked to culture and upbringing" (*Herald Sun*, *Australia*, 14 October, 2004).

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REFERENCE [1] Corna, F. *et al.* *Proc. R. Soc. Lond. B* 18 October 2004 (doi:10.1098/rspb.2004.2872)

SEX DETERMINATION

Platypus: the stranger sexxxxx

As if being an egg-laying mammal wasn't weird enough, the duck-billed platypus has now been shown to have a pretty unconventional way with chromosomes. Frank Grützner and colleagues have cleared up several unknowns surrounding the chromosome system of this small group of animals: their cytogenetic studies show that platypuses have not one, but five pairs of sex chromosomes, which link up to form a multivalent chain at male meiosis.

The Monotremata subclass of animals, to which the platypus belongs, split off from the mammalian lineage about 210 million years ago, and indeed the karyotype of this species departs in many ways from that of mammals. For one, several of the platypus' 52 chromosomes are known to be unpaired in males. What happens at meiosis is equally unusual, as the chromosomes, like those of some insects and plants but not of mammals, form a chain-like structure. And how sex might be determined with such a seemingly bizarre chromosome arrangement remained to be worked out.

Grützner *et al.* used a combination of chromosome painting and comparative genomics to shed light on all these questions. By hybridizing eight chromosome paints — which the authors created — to male and female platypus chromosomes, the authors show that males have five copies of an X-like chromosome — which are paired in females — and five Y-like chromosomes. They also establish that the ten sex chromosomes — $X_{1\text{–}5}$ and $Y_{1\text{–}5}$ — link up into a chain at male meiosis. This arrangement would spell disaster without a foolproof means of creating sperm that carry either five Xs or five Ys: as it happens, the chain contains an alternating set of X and Y chromosomes, allowing segregation to occur seamlessly.

The chromosomal arrangement in platypuses is like nothing else that came before it; how then, to explain the evolution of this peculiar sex-chromosome system? Although the X_1 chromosome shares many features with the mammalian X, none of the Y-like chromosomes carry an *SRY*-like gene, which is used to determine maleness in mammals. At the other end of the scale, the X_5 contains a homologue of the *DMRT1* gene, which is found on the bird Z chromosome and is thought to determine sex in the bird ZW system. So, just like their morphology and physiology, platypus chromosomes also show a mixture of mammalian and bird-like features: a mammalian-style X_1Y_1 pair and a bird-like X_5Y_5 pair. But it's the bird-like X_5Y_5 pair that probably holds the key to the evolution of this system. The meiotic chain is held together by limited homology between adjacent partners. The high degree of degeneracy of the Y_5 chromosome indicates that the sex chromosomes evolved from the X_5Y_5 sex-chromosome pair; the other four pairs would have been recruited from autosomes to join in, through a series of sex chromosome–autosome translocations.

PLANT DEVELOPMENT

Rooting out new signals

A recent study provides the first genetic insights into a root-derived signalling pathway that might coordinate plant development with environmental conditions.

Leslie Sieburth and colleagues first identified the gene *BYPASS 1* (*BPS1*) in a screen for *Arabidopsis thaliana* mutants with leaf-vein defects. *BPS1* encodes a novel protein needed for proper shoot and root development; however, when *BPS1* is mutated, root defects become apparent first, hinting that the shoot phenotype might be a secondary effect of faulty root development, owing to disrupted water or mineral uptake. But this was ruled out when mutants were grown in liquid culture — removing the need for uptake through the roots — as the same phenotype was seen.

To explore the relationship between the root and shoot defects further, the authors carried out grafting experiments. First, the apical parts of a *bps1* mutant were grafted onto the rootstock of a wild-type plant to test where *BPS1* function is required. This partially rescued the shoot defects, although the effect was only transient. An intriguing result came from control experiments: grafting a mutant apex onto a mutant rootstock also gave a partial rescue, implying that separating the developing shoot from the roots prevents the effects of *bps1* mutation. One explanation is that the roots produce a signal that inhibits shoot growth unless functional BPS1 is present. This was supported by the fact that continuous removal of roots during development rescues the *bps1* phenotype and that *bps1* roots are sufficient to arrest wild-type leaf growth in grafting experiments.

What is the inhibitory signal produced in the roots and how does BPS1 reverse its effects? The authors grew wild-type plants in the presence of a range of hormones that are key signalling molecules in plants. None of these mimicked the *bps1* phenotype, ruling them out as candidates for the root-derived signal. However, when *bps1* mutants were grown in the presence of fluridone, an inhibitor of carotenoid synthesis,