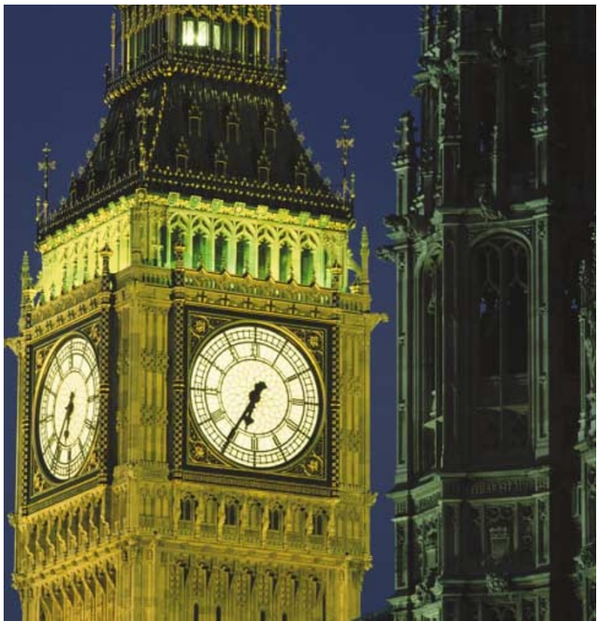

BEHAVIOURAL GENETICS

Smelling the time

Whether by compelling us to follow the latest fashion or to practise good table manners, the company we keep is known to have a strong influence on our behaviour. But the effect of society, it seems, can reach even deeper. Joel Levine and colleagues have found that internal biological clocks — those that regulate our sleeping rhythm, for example — can be reset by social interactions. Although this work was done in fruitflies, and the external cues were found to be olfactory, it is possible that the clocks of other species respond to the social environment in a similar way.

In all experiments, flies were exposed to a light–dark regime for five days, then placed in constant darkness for two weeks, after which time their clock rhythm — based on locomotor activity — was scored. The clocks of grouped flies were more synchronous after this treatment than were those of flies treated in isolation. If, as this result implies, company keeps flies in time, then genetically asynchronous flies would be expected to disrupt the harmonized clock when added to a wild-type group. This was tested by mixing flies mutant for the *period* gene (*per*), which have no sense of time, with wild-type flies, which, indeed, lost their synchronicity. Curiously, *per* mutants with early activity peaks ('early birds') were able to influence the activity of 'later-rising' *per* mutants, but not the other way around.

So, what is it about company that synchronizes the clocks of these flies? Simply exposing individual animals to the air from a chamber in which a group of flies was kept, was enough to synchronize their clocks. As the effect was abolished when the receiving flies had no sense of smell (because of a specific class of mutation in the *paralytic* gene), the authors settled on olfactory cues as being a good explanation.

Clocks and social interactions have been linked in many species, from humans to bees, but this is the first study to sniff out — genetically — the underlying sensory cause.

Tanita Casci

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WEB SITE

Jeff Hall's lab: http://www.bio.brandeis.edu/faculty01/hall.html

EPIGENETICS

Now you see it … now you don't!

Like a magician's cloak, heat shock protein 90 (Hsp90) hides the molecular variation contained within *Drosophila* and *Arabidopsis* populations — it is only when the masking effect of Hsp90 is lifted that a range of underlying morphologies appear. Favourable phenotypes revealed by the absence of Hsp90 activity can be fixed in the population and, therefore, persist even when Hsp90 function is restored. As with every conjuring trick, the question arises: how is it done? Fixation was thought to depend on the selection of pre-existing genetic variation in the population. Now, Sollars and colleagues report that, in *Drosophila*, phenotype fixation can also be caused by epigenetic alterations.

In their studies, the authors concentrated on the ectopic bristles seen in the eyes of flies in which Hsp90 function had been reduced through mutation or pharmacological inhibition. Offspring from flies fed an Hsp90-inhibiting drug were bred and selected for the eye bristle phenotype, over 13 generations, in the absence of the drug. The penetrance of the phenotype increased in response to selection and remained even when Hsp90 function was restored. However, as the strain of flies used in the experiment was nearly isogenic, genetic variation was unlikely to account for the fixation of the ectopic eye-bristle phenotype, as was previously thought. Instead, it was more likely that epigenetic changes affecting chromatin structure had occurred. In support of this idea, when flies from the F6 generation were treated with a histone deacetylase inhibitor — resulting in an increase in histone acetylation — the percentage of individuals showing ectopic eye bristles decreased significantly.

The authors also identified a further link between chromatin structure and ectopic eye bristles. A similar bristle phenotype to that seen in flies with compromised Hsp90 function was found in mutants of nine different *trithorax* group (*trx*) genes, which show hypoacetylation. Once again, the phenotype was reduced by histone deacetylase inhibitor treatment. Selection studies on the most severe *trx* mutant showed that, although the mutation is required initially in the mother to generate the ectopic eye bristles, after selection it is not required to maintain the phenotype. It seems, therefore, that *trx* mutations induce ectopic outgrowths by altering chromatin structure in the egg and then an unknown epigenetic mechanism results in their fixation. This indicates that Hsp90 removal might induce epigenetic changes, possibly through an interaction with Trx proteins.

This study indicates that epigenetic changes might lead to the fixation of phenotypes that are normally buffered by Hsp90 and that are exposed by, for example, environmental stress. This mechanism for evolutionary change has important implications. First, evolution might proceed more rapidly when fixation occurs by epigenetic mechanisms. Second, an epigenetically determined phenotype is likely to be less stable than one fixed through genetic changes, and so it might be reversed more easily.

Catherine Baxter

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