IN THE NEWS

Clone baby?

Doubts have started to surface over the truth of claims that the first human clone has been born, after Clonaid — the company that made the original 27 December announcement — backed away from an independent verification by genetic tests.

The *New York Times* reported that Clonaid's self-imposed one-week deadline expired with no evidence forthcoming. Clonaid's chief executive Brigitte Boisselier said "The parents told me that they needed 48 hours to decide yes or no — if they would do it" (*New York Times*).

The credibility of the claims took another hit when Michael Guillen — the freelance science journalist organizing the genetic tests — pointedly distanced himself from Clonaid after the tests did not go ahead. "It's entirely possible [that] Clonaid's announcement is part of an elaborate hoax to bring publicity to the Raelian movement," he said (*The Guardian*).

Claude Vorhilon (aka 'Rael'), leader of the pseudoscientific sect that funds Clonaid, suggests that a Florida court action aimed at placing the baby under the court's protection might explain the company's reticence. "…to take away this poor baby from a mother, I think this is completely crazy, just because she was cloned. So I called Doctor Boisselier, and I said, 'If I was you, I would not test anything.'" (*The Washington Times*).

The increasing scepticism of the media has not prevented Clonaid from expanding their claims: according to them a further three human clones will be born in the next month, in addition to the trio already born (*The Guardian*).

Perhaps the biggest concern for geneticists arising from the whole media furore is the spur it is likely to provide for efforts in the US Congress to ban human cloning (*The Guardian*).

Nick Campbell

Apart from the important biological insights, Kamath *et al.* have given the community an important resource — the bacterial RNAi library that can be used over and over again. Many more reports of screens such as those by Ashrafi *et al.* and Lee *et al.* are bound to follow, the results of which will provide a more complete picture of individual biological processes. The hope is that, as RNAi technology improves, similar systematic screens will also be feasible in mammalian cells.

Magdalena Skipper

References and links

ORIGINAL RESEARCH PAPERS Kamath, R. S. *et al.* Systematic functional analysis of the *Caenorhabditis elegans* genome using RNAi. *Nature* **421**, 231–237 (2003) | Ashrafi K. *et al.* Genome-wide RNAi analysis of *Caenorhabditis elegans* fat regulatory genes. *Nature* **421**, 268–272 (2003) | Lee, S. S. *et al.* A systematic RNAi screen identifies a critical role for mitochondria in *C. elegans* longevity. *Nature Genet*. **33**, 40–48 (2003)

FURTHER READING Dillin, A. *et al.* Rates of behavior and aging specified by mitochondrial function during development. *Science* **298**, 2398–2401 (2002)

EVOLUTIONARY GENOMICS

Compensation or innovation

Gene duplications are often seen as an opportunity to evolve new functions through the accumulation of mutations leading to functional diversification. But, they can also be thought of as a back-up or a buffer against loss-of-function mutations in one of the duplicates. Using yeast as a working example, Gu *et al.* have shown that gene duplications considerably contribute to genetic robustness against null mutations.

A previous study of genetic robustness — the ability to withstand null mutations — concluded that it was redundant metabolic pathways and networks, rather than duplicate genes, that mainly fulfil this function. However, these conclusions were based only on a few genes, so Gu *et al.* revisited this problem — this time addressing it on a genome-wide scale. They made use of a previous study in which almost all of the genes of *Saccharomyces cerevisae* were knocked out and the fitness of the mutants was assessed under five different conditions (see Highlights section in September 2002 issue). When the authors compared the fitness of strains deleted for unduplicated genes with those deleted for duplicates, they found a significant difference — deletions of duplicates were significantly less likely to cause lethality and more likely to have mild effects, or no effects, under each of the five experimental conditions. These observations indicated that duplicated genes compensate for each other, a conclusion that was supported by the fact that deletions of either gene from a duplicate pair showed similar fitness effects. Furthermore, the smaller the divergence between the duplicates, the better they compensate for each other. It also turns out that deleting duplicate genes with higher expression levels has a greater effect on fitness than deleting those that are not as highly expressed.

These data provide strong evidence for the role of gene duplication in genetic robustness against null mutations. Whether this contribution is more or less important than the interactions between unduplicated genes that function in alternative pathways remains to be seen. The role of duplicates in genetic buffering might explain why these genes do not 'decay' into pseudogenes as quickly as expected. But, Axel Meyer — the author of the accompanying *News and Views* — suggests that their incorporation into new networks and pathways also prevents such decay. So, does this mean that we were wrong about duplications being the prerequisite for innovation? Gu *et al.* believe that gene duplication is still the most common path for innovation, and that a duplicate gene is maintained because it might be able either to improve part of the original function or to perform a new function. The buffering effect of duplicates is largely caused by their partially overlapping function. Axel Meyer argues for a dual role for duplicate genes, but further whole-genome-sequence comparisons and functional genetic analyses are needed before we can really address this question.

Magdalena Skipper

References and links

ORIGINAL RESEARCH PAPER Gu, Z. *et al.* Role of duplicate genes in genetic robustness against null mutations. *Nature* **421**, 63–22 (2003)

FURTHER READING Meyer, A. Duplication, duplication. *Nature* **421**, 31–32 (2003)