TOUCHING BASE

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Mutant of the Month

Angabin Matin and Joe Nadeau

This month we feature the mouse *Ter* mutation as our August MoM. The *Ter* allele was discovered by Leroy Stevens at the Jackson Laboratory (*J. Natl. Cancer Inst.* 50, 235–242; 1973). Stevens originally found that the 129 mouse genetic background has a higher rate of development of spontaneous testicular germ cell tumors than other backgrounds. While searching for modifiers of this genetic predisposition, he and Tekehiko Noguchi identified the *Ter* mutation, which increases susceptibility to testicular germ cell tumors in the sensitive 129 background while causing primordial germ cell deficiency in all mouse genetic backgrounds. *Ter*-induced germ cell tumors are pictured here next to normal mouse testes and germ cell–deficient testes from *Ter* mice. Angabin Matin, Joe Nadeau and colleagues recently identified the genetic change responsible for the *Ter* allele (*Nature* 435, 360–364; 2005): a nonsense mutation that disrupts the coding region of the *Dnd1* gene, an ortholog of the zebrafish *dead end* gene. The DND1 protein is closely related to the apobec complementation factor, a component of an RNA editing complex. Although the consequences of the *Ter* mutation are as yet unknown, this mutant brings to light an intriguing connection between RNA editing, tumorigenesis and germ cell development. *EN*

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The season of the impact factor has come and gone, with the usual range of comment in the scientific and popular media on the ways in which these numbers can be manipulated and don't necessarily reflect the quality of individual articles. But one thing that gets less attention is the possibility that completely non-scientific factors may influence the citability of particular papers. A correspondence in the 6 July issue of the *New England Journal of Medicine* by Matthew Stanbrook and colleagues reports a study on the effect of assigning acronyms to clinical trials (*N. Engl. J. Med.* **355**, 101–102; 2006). They found that 34% (59 out of 173) of the published clinical trials they examined were named with an acronym, and these were cited more than twice as often as trials not so named, even though they were not more likely to report positive results. A subanalysis comparing trials published in the same journal yielded similar results. Although the authors say they cannot rule out the possibility that "exemplary investigators may generate both clever acronyms and important research," they prefer the explanation that acronyms serve as powerful mnemonic aids. As such, they exert an influence that "is not rational scientifically, even if it is understandable psychologically." The fact that acronym-named trials are four times as likely to be funded by pharmaceutical companies suggests the widespread use of acronyms is no accident. But what of research in, say, genetics? Are short, snappy titles sporting catchy gene names more citable, regardless of the importance of the results? Take sonic hedgehog, for example. Does the morphogen make the name, or does the name make the morphogen? Perhaps it's time we undertook our own analysis of the genetics literature: the *Nature Genetics* Names Undermining Metrics to Better Evaluate Research 1 study. We're above this sort of thing, you see, and think it's time this practice is exposed for what it is. *AP*

"I need to get more bone. I'll go to Russia with a pillowcase and an envelope full of euros and meet with guys who have big shoulder pads. Whatever it takes."

—Eddy Rubin, on plans to obtain additional Neanderthal tissue samples for ongoing DNA sequencing efforts (as quoted in *Wired*).

Accessibility of ENCODE regions

In the July issue of *Nature Methods*, two groups present analyses of DNase I hypersensitivity and chromatin accessibility of ENCODE regions using new methods with similar microarray platforms designed for eventual whole-genome analyses of hypersensitive sites. In one study, Francis Collins and colleagues present a method called DNase-chip, in which DNA flanking cleavage sites is isolated by attaching biotinylated tags to cleaved ends of 200- to 500-bp DNA fragments and then hybridizing the fragments to high-resolution microarrays (*Nat. Methods* **3**, 503–509; 2006). In the accompanying study, John Stamatoyannopoulos and colleagues present a similar approach called DNase-array, which also assumes that cleavage events are more likely to occur in close proximity within accessible regions (*Nat. Methods* **3**, 511–518; 2006). These two new methods, intended for whole-genome microarray-based analyses of hypersensitive sites, are tested in these initial studies on ENCODE regions. In addition to providing analyses of the hypersensitive sites of the ENCODE regions, these studies offer some initial general characterizations of the distribution of hypersensitive sites. In both of these studies, hypersensitive sites were found enriched at regions presumed to have active regulatory elements and within highly conserved gene-rich regions. Also, those genes with a hypersensitive site nearby showed higher levels of gene expression. Finally, over 80% of identified hypersensitive sites were located within 2.5 kb of another site, suggesting that clustering of DNase I hypersensitivity may reflect a common chromatin feature. *OB*

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