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Give the dog a clone

With the birth of an Afghan hound named Snuppy, which stands for Seoul National University puppy, the dog has become the latest addition to the list of mammals that have been successfully cloned, alongside the mouse, rat, cat, sheep, goat, rabbit, mule, and horse.

The quest to clone a dog has been particularly challenging. In this species, oocytes are immature at the time of ovulation and develop as they move through the oviducts to the uterus. Previous cloning attempts involving eggs taken from the ovaries have failed.

Byeong Chun Lee and colleagues at Seoul National University in South Korea had better success using a technique for flushing oocytes from the oviducts. They cultured these cells and then replaced the nuclei with nuclei taken from skin cells from the ear of a male Afghan hound. The success rate was incredibly low: Transfer of 1,095 embryos into 123 recipients resulted in three pregnancies and two births. One of the puppies died 22 days after birth. The surviving puppy, which has been shown to be genetically identical to the donor, was born to a yellow Labrador retriever surrogate and appears to be healthy (*Nature*, 4 August).

If the technique can be refined to improve the success rate, lines of genetically identical dogs may someday serve as models of human diseases.

Genetic basis for sweet blindness in cats

A pseudogene is at least partly to blame for cats' notoriously picky eating habits, according to a new study showing that cats lack a functional taste receptor for sweetness.

Cats are obligate carnivores that have long been known to be indifferent to sugary foods. Previous studies have shown that cats respond to salty, sour, and bitter stimuli but do not show a neural response to sugars. In most mammals, two proteins, T1R2 and T1R3 (encoded by the genes *Tas1r2* and *Tas1r3*, respectively), combine to form the receptor for sweetness.

A group led by Joseph G. Brand of the Monell Chemical Senses Center in Philadelphia compared the section of the cat genome encoding the *Tas1r2* and *Tas1r3* genes with equivalent regions from the human, dog, mouse, and rat. They found that a 247-base pair deletion in *Tas1r2* makes it an unexpressed pseudogene (*PLoS Genet.*, July); the absence of functional T1R2 prevents the formation of sweet receptors.

Brand's team speculates that this inability to taste sugar played a key role in the evolution of the cat's carnivorous eating habits.

Mouse food for thought

Choice of food source for research animals may be even more important than previously thought, according to the results of a new study in mice. New research shows that variations in phytoestrogen concentrations in commercially available rodent chow can have significant effects on experimental variables.

Plant-derived phytoestrogens, much touted for their apparent health benefits, can also act as endocrine disruptors, causing developmental and reproductive problems. When Sudhansu K. Dey's research program relocated to Vanderbilt University (Nashville, TN) from its longtime home at the University of Kansas Medical Center (Kansas City, KS), the researchers began noticing variations in uterine gene expression and reproductive function in their mouse models.

They suspected that the cause of these changes was related to diet, because mice at Vanderbilt are fed chow high in phytoestrogens (>400 p.p.m. isoflavones) while those at the University of Kansas eat chow with a much lower isoflavone concentration (<50 p.p.m.). They confirmed this hunch by determining that feeding female mice chow with the higher phytoestrogen concentration significantly increases uterine growth, changes uterine gene expression, and changes the timing of implantation compared with low-phytoestrogen food (*Proc. Natl. Acad. Sci. USA*, 12 July).

The knowledge that dietary phytoestrogen concentrations can affect experimental outcomes highlights the need for researchers to consider food choices carefully, and possibly indicates the need for standardization of phytoestrogen levels in commercially available rodent chow.