Nature vs. Nurture

by Pat Rock

October 2003 AKC Gazette

English Cocker Spaniel Column

Proof is emerging that nutrition, infection with microbes, and genetically determined production of important molecules are linked to one another in determining an individual's phenotypic makeup. In fact, the term phenotype (observable physical characteristics that are genetically determined) may need to be redefined somewhat in light of better understanding of the process of creating a new individual from a set of chromosomes in an embryo.

The implications for dog breeders are staggering. We were beginning to think that if we threw enough research money at the problem, the gene for *any* trait could be isolated and a gene or marker test found - and,, while the research and testing might be expensive, at least we'd have a cut-and-dried answer about the status of our breeding animals for that particular defect.

We should be grateful that some traits can be controlled by isolating a single gene. But for other traits, the situation is more complex. In the development of individual animals, the genes act like a set of house plans. If the designated materials are not all available, substitutions may be made. For example, when building a house a sub floor that is nailed down will pass inspection and, once it is covered by carpet, it will not be apparent that nails were used instead of screws. It may be years before the floor starts squeaking or warping. A similar scenario may occur in our dogs' bodies. Lack of a nutrient during gestation or growth may not result in any observable abnormality, but over time an organ system might fail. Obviously, such a failure is not a genetic problem.

Here's another scenario: Suppose most individuals in a populations (read dog breed) are genetically programmed to be able to manufacture an enzyme that can "cover" the effects of the lack of that nutrient, perhaps by allowing an alternate pathway to create needed molecules. The ones in the population that can't make the enzyme will only demonstrate the defect if they also are deprived of a particular nutrient during gestation r growth. You can remove the obviously defective animals from the gene pool, and maybe even their first-degree relatives (parents or siblings), but you still have those animals that are never identified as connected with the defect because they were not subjected to the nutrient deficiency.

Might not some bone and joint disorders fit this model? Look how many years of selection there have been against hip dysplasia, and in some breeds it is still rampant. The damage of HD can be artificially prevented by limiting exercise, but that only changes phenotype, not genotype. The real problem is growth rate and nutrient absorption. The former is strongly influenced by carbohydrate in the diet (dogs fed mostly protein and fat grow at a slower rate but reach the same mature height and bone mass). The latter is determined both by how the minerals are provided in the diet, and by the genetically determined ability of the animal to digest and absorb them. For all this we are using X-rays to determine what is right and wrong with this animals' genes?

Want to read more on the cutting edge of this research? See "Selenium Deficiency and Viral Infection," Beck. M.A., Levander, O.A., Handy, J., Journal of Nutrition, May 2003 (5Supp 1): 1463S-7S.