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Evolutionary mechanisms involved in the origin of a natural mutant rat from an inbred rat strain.

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Short Abstract: In order to investigate the evolutionary mechanisms involved in the origin of a natural mutant rat, a whole genome scan was performed along with its parent and two other normal strains, and observed genetic drift which may be attributable to the inbreeding like effects in the parent strain.

Long Abstract:
To investigate the possible evolutionary mechanisms (genetic drift / mutation / migration), that might have been involved in the origin of a natural mutant, the mutant strain along with its parent, a related strain to the parent and an unrelated strain were genotyped. A whole genome scan was performed spaning all the 20 autosomes and X chromosome using microsatellite DNA markers.

Microsatellite DNA are 2-4bp tandem repeats. They are relatively evenly distributed through out the genome and mostly present in the vicinity of the coding regions. Polymorphism at a microsatellite DNA locus is mainly due to difference in repeat number and hence are highly polymorphic, i.e, presence of more than two allelic variants may be visualized and they also reveal co-dominance. They can be easily analyzed using PCR and subsequent gel electrophoresis.

Inter-strain and intra strain polymorphism at few microsatellite loci were observed in standard strains, whereas mutant strain showed only intra strain polymorphism, i.e., a change in frequency of allelic variants with respect to its parental strains.

The fixed allelic variants, and the allelic variants with different frequencies in the normal rat strains, may speak about the stability of microsatellite markers and the genetic drift that has occurred in them.

The genetic drift that occurred in mutant strain, from parent strain may throw insights for inbreeding like effects / inbreeding depression that might have occurred in parent rat strain. The continuous inbreeding might have resulted in the appearance of a homozygous state at the candidate locus, the disease allele which might have been present in parent strain since an earlier date in heterozygous state, and hence not identified and because of deleterious effects, this allele might have been lost from parent population or present as a rare allele which is evident from allelic variants of few markers that show a high frequency in mutant population are either present at a very low frequency or even absent in parent population.