Obesity and diabetes type II in cats belongs to the most increasing diseases within the last years and is thus one of the major problems in the future (Brennan at al. 2004). Besides food intake, food composition and lack of exercise also genetic factors influence the development of obesity in cats. We hypothesize that mutations within the genome are responsible for some forms of obesity in cats. In our experimental cat population a striking and apparently heritable obesity phenotype was observed. It is assumed that mutations in one or more unknown major genes are involved in the formation of obesity.

A segregation analysis clearly indicated an autosomal recessive major gene and a possible polygenic component as an underlying cause of the overweight phenotype in the investigated cat breeding family. In the next step the animals were analysed using the feline Illumina 62k SNP genotyping microarray and the genotyping results were analysed. This analysis identified markers on chromosome D3 and C2 to be associated with body condition score. Unfortunately, these results are not significant on genome wide level, which is likely to be caused by the small number of cats investigated. The associated regions on chromosome D3 contain MC4R and NPY1R as positional candidate genes while no obvious candidate gene could be identified in the associated region on chromosome C2.

In future finemapping will narrow the region of interest and the analysis of candidate genes of the regions associated to body condition to determine the mutations responsible for the phenotypic difference.

Besides the genetic differences in the experimental cat population a tendency to a lower the energy expenditure in tom cats of the overweight phenotype (GO) in the lean stage compared to always lean cats (GL) was found. It was assumed that a higher activity of the lean cats was causing this difference. However, food intake was significantly higher in group GO than in group GL and the eaten quantity per meal was larger. Unfortunately, it cannot be excluded that a preceding weight loss had an influence on food intake or energy expenditure. However, the clearly different food intake of the lean and overweight phenotype is concordant with results from the genome analyses, that showed associations for MC4R and NPY1R as both receptors are known to be involved in the regulation of food intake.

In future the hypotheses will be tested:

1. Cats with a genetic predisposition to overweight generally show a higher food intake compared to lean cats.
2. The energy expenditure of cats does not differ between individuals with or without overweight predisposition before obesity developed.

Publications / Publikationen


and impaired insulin sensitivity present in growing cats. J Anim Physiol Anim Nutr, 97: 813-819


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