7 Summary

Investigation of Osteodystrophia Fibrosa in guinea pigs *(Cavia aperea f. porcellus)* the satin type phenotype – development of a radiographic scoring validating the presumption diagnosis.

Between 1997 and 2002 seventy-six satin type guinea pigs suspected to have fibrous osteodystrophy were presented at the Clinic for Small Domestic Animals, Freie Universität Berlin. 15 out of 76 satin guinea pigs were examined pathologically.

In the present study 52 satin type guinea pigs (sasa; 25 ♀, 27 ♂) were analysed statistically. Two control groups out of healthy, non-satin, normal guinea pigs were established, one to compare radiological results (n = 25) and one to compare blood chemistry and blood count values (n = 36). In addition, seven carriers of the satin gene (sa-, normal fur; 6 ♀, 1 ♂) were examined.

Twenty out of fifty-two satin type guinea pigs were established to be clinically sick, in the other 32 there were no pathological findings. Fifteen animals had lost weight, ten animals had not eaten and had wet fur around their mouths. Four animals were dehydrated and the state of the teeth in three had deteriorated. Two others had grown elongated cheek teeth and twelve guinea pigs had misshapen mandibles. Fifteen satin type guinea pigs were reluctant to walk, four had painful knees and a painful hip, two had crooked backs. Twelve guinea pigs were weak and apathetic. Fibrous osteodystrophy was diagnosed in eleven out of fifteen guinea pigs by pathological examination. One out of seven carriers had vaginal discharge, the others were healthy.

A radiographic scoring out of nine parameters was developed. Alterations in the course of fibrous osteodystrophy were assessed at the os parietale, os frontale, ramus mandibulae, arcus zygomaticus, vertebrae lumbales, articulatio coxae, os femoris, os tibiae and the articulatio genus. Between 9 and 27 points were possible. Less than and equal to 13 points were defined as being ‘without pathological findings’, more than 13 points as ‘pathologic’. None of the control group and none of the carriers got more than 13 points. Scoring 52 satin guinea pigs showed that the younger animals (≤ 25 months) reached less points than the older animals (> 25 months; $p < 0.05$; U-test). Sick animals reached more points than those without pathological findings ($p < 0.05$). Sex was nonsignificant.

We found osteopenia in the os mandibulae, arcus zygomaticus, os femoris and os tibiae, vertebrae lumbales and in the pelvic bone as well as osteosclerosis in bones like the os parietale, os frontale, vertebrae lumbales and in long bones.

Satin type guinea pigs were hypocalcemic in comparison to control animals ($p < 0.05$; U-test). Younger satin guinea pigs had a higher phosphorus level than older ones ($p < 0.05$). Younger satin guinea pigs showed higher alkaline phosphatase levels than younger control animals ($p < 0.05$). The same was true for older animals. Sick satin guinea pigs had higher levels of the alkaline phosphatase than satin guinea pigs without pathological findings ($p < 0.05$). Radiologically suspected satin guinea pigs (> 13 points) had a higher alkaline phosphatase level than those without radiological changes. Creatinine in sick satin guinea pigs was higher than in animals that showed no clinical signs of disease ($p < 0.05$; U-Test). Carriers and controls had higher levels of creatinine than healthy satin type guinea pigs ($p < 0.05$). Clinicially inconspicious satin guinea pigs had less platelets than controls ($p < 0.05$). There were no significant differences in sex within all parameters.

Fibrous osteodystrophy caused by secondary hyperparathyroidism was diagnosed in sick satin type guinea pigs. The cause of the disease is unknown. We suggest a genetic disorder. This should be clarified in further studies.