

Prepubertal and post castrational development of bones in prenatally dexamethasone-treated marmosets (*Callitrix jacchus*)

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The marmoset serves as a primate model for many human diseases. Whether it develops osteoporosis following castration is not known. This was tested in the first set of experiments in adult female and male castrated animals. From experiments in rats it is known that prenatal dexamethasone (DEX) treatment results in some but not all symptoms reminiscent of the metabolic syndrome of the human. To test whether the marmoset is an even better model for this disease pregnant animals were either treated at day x or day y after conception with 5 mg DEX. In the male offspring and in the castrated adult animals total surface and bone mineral density (BMD) of the cortex and of the trabecular structures of the metaphysis of the tibia and of the 5th lumbar vertebra (L5) were determined by quantitative computer tomography in 6 months intervals either after castration or during pubertal life respectively. Surrogate parameters of bone metabolism (osteocalcin = OC and n-terminal breakdown products of collagen 1 α 1 = crosslaps) were measured. Male and female marmosets lost between 5-20 % of their initial trabecular BMD in the metaphysis of the tibia and this was statistically significant 6 months after castration. No loss of BMD was observed in L5. Both OC and the crosslaps in the serum were increased at this timepoint. 6 months after birth, BMD of the metaphysis of the tibia but not L5 was significantly decreased in both prenatally DEX treated animals. At this timepoint serum crosslaps but not osteocalcin was increased. It is concluded that castration of male or female marmoset results in substantial loss of BMD in the metaphysis of the tibia but not in the vertebra. Prenatal DES treatment appears to increase osteoclast but not osteoblast activity. Taken together the results indicate that marmosets may be a good more human like experimental model for bone research.

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