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JOURNAL ARTICLE

# Deficiency of fucosidase results in acrosomal dysgenesis and impaired sperm maturation

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Although a variety of glycosyltransferases and glycosidases have been implicated in spermatogenesis and posttesticular sperm maturation, the biological role of these enzymes in these processes is largely unknown. We describe reproductive sequelae in a cohort of male dogs suffering from fucosidosis, a heritable lysosomal storage disorder caused by a severe deficiency of alpha-L-fucosidase. There was a reduction in the total number of sperm in the ejaculate. Only 3-5% of sperm were motile. None of the sperm were found to be morphologically normal. The predominant morphological defects observed were malformed acrosomes (56%) and retained proximal cytoplasmic droplets (92%), indicating that spermiogenesis and sperm maturation were impaired. The cytoplasm of all cellular components of the testis and excurrent ducts were vacuolated. The vacuolation resulted from enlargement of lysosomes caused by accumulation of compounds that are otherwise cleaved/degraded when lysosomal hydrolases are present normally. It is possible that impairment in spermatogenesis, particularly morphogenesis of the acrosome, is due to physical damage caused by anomalous enlargement of lysosomes. Although an unambiguous causal relationship could not be established, it is evident from the available information that the derangement in events associated with epididymal sperm maturation, namely acquisition of motility and shedding of the cytoplasmic droplet, is likely due to lack of fucosidase leading to impaired sperm membrane modification. This heritable condition in dogs may serve as a spontaneously occurring knock-out model for further elucidating the role of alpha-L-fucosidase in spermatogenesis and sperm maturation.



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