The Ferret in biomedical research. A review

by *Ricardo G. Fischer & Björn Klinge*
Lund University, Centre for Oral Health Sciences, Lab Animal Res.,
Carl Gustafs väg 34, S-214 21 Malmö, Sweden.

1. Introduction

The ferret, like skunks, otters, minks, weasels and badgers is a mammal belonging to the Mustelidae family of the carnivora order (*Wright* 1974, *Ryland et al.* 1983). In this order we also find the Canidae and Felidae families (*Wright* 1974). Two strains of ferret exist. One is the American black-footed ferret, Mustela nigripes, which is a native of western North America and is nearly extinct (*Willis & Barrow* 1971, *Ryland & Gorham* 1978, *Ryland et Fox* 1983, *Fox* 1987). The other is the domestic ferret, Mustela putorius furo, also known as the "polecat", of North America, Europe and Asia (*Willis & Barrow* 1971, *Ryland et al.* 1983, *Fox* 1987). It was imported into the United States around 1875 (*Ryland & Gorham* 1978, *Moody et al.* 1985). Two varieties of the domestic ferret can be recognized, the fitch ferret with a brown coat and a mask-like 'face' and the albino ferret, or English ferret, with a yellowish-white fur color and pink eyes (*Ryland & Gorham* 1978, *Ryland et al.* 1983, *Moody et al.* 1985).

The ferret was first used in Europe for the extermination of rodents and snakes and later for hunting rabbits (*Willis & Barrow* 1971, *Ryland et al.* 1983). Perhaps this is the reason why the ferret has a reputation for ferocity (*Hammond & Chesterman* 1972). It has become an increasingly popular pet even though the American Veterinary Medical Association considers the ferret an exotic and wild animal and is against its use as a pet animal (American Veterinary Medical Association Council 1986 a, b).

Both fitch and albino ferrets have been used as research animals in the medical field, especially in virology, immunology, physiology, pharmacology, toxicology, teratology, neuroanatomy, ophthalmology and gastroenterology (*Wright* 1971, *Beck et al.* 1976, *Ryland & Gorham* 1978, *Ryland et al.* 1983, *Moody et al.* 1985, *Fox* 1987, *Fox et al.* 1990). In odontology, the first reports of its use in periodontal research are from King in the 1940s (*King* 1944 a, b, *King & Glover* 1945, *King & Gimson* 1947 a, b, *King et al.* 1955). Later on, the ferret was also used in studies of gingival hyperplasia (*Steinberg et al.* 1972, *Hall & Squier* 1982), in endodontics (*Callis & Santini* 1987, *Holland* 1988) and more recently in investigations on calculus formation (*Harper et al.* 1990, *Mann et al.* 1990).

Their ease of maintenance, small size, generally friendly disposition, and the growing public pressure to reduce the use of dogs, cats and monkeys in research and some special characteristics have increased the interest in the use of the ferret as an experimental animal.

2. The animal

2.1. Growth and development

Newborn ferrets are hairless, deaf and blind and weigh 1 % of their adult weight (*Ryland & Gorham* 1978, *Shump & Shump* 1978). Both their eyes and ears begin to open at around 28 days (*Shump & Shump* 1978). The young ferrets begin to eat solid food regularly by 4 to 5 weeks of age when they also begin to defecate without stimulation from the females. Before this the mother licks the anal region to stimulate defecation and urination (*Shump & Shump* 1978). The young male and female ferrets increase their body weight and length at the same rate until 7 and 9 weeks of age, respectively. After this the male increases its weight more sharply than the female until their adult

weight is reached (*Shump & Shump* 1978). Males (1400–1500 g) are normally twice as large as females (800–900 g) (*Ryland & Gorham* 1978, *Shump & Shump* 1978, *Ryland et al.* 1985). Their body weight has a seasonal variation (30–40 %), due to fat accumulation in the autumn and its subsequent loss in the spring and summer (*Ryland & Gorham* 1978, *Ryland et al.* 1985). Females can also lose weight more rapidly after they become estrous (*Hammond & Chesterman* 1972). Body length ranges from 44 to 56 cm (nose to tip of tail) (*Ryland & Gorham* 1978). The mean life span is about 4–6 years, but occassionally survival to 8 to 9 years may occur (*Willis & Barrow* 1970, *Hammond & Chesterman* 1972).

The ferret possesses a relatively short digestive tract, which is characteristic of carnivores. The large intestine is unique, as there is no external anatomical division between the ileum and the colon (*Ryland et al.* 1985). In common with the mustelids, ferrets have two anal sacs at the base of the tail, which secrete when the animal is angry or excited and, in females, in estrus (*Ryland & Gorham* 1978, *Ryland et al.* 1985). An usual anatomical variation is a single central artery instead of the more common bilateral carotid arteries (*Ryland & Gorham* 1978, *Ryland et al.* 1985). It is postulated that this configuration might be a functional adaptation to help the ferret maintain adequate cerebral flow as it turns its head 180 degrees (*Ryland & Gorham* 1978, *Wen et al.* 1985). Ferrets have good hearing and an acute sense of smell for hunting, and although they do not have colour vision, they can be as useful as dogs in ophthalmological research (*Wen et al.* 1985).

2.2. Reproduction

Ferrets normally reach sexual maturity in the spring after their birth, or at approximately 1 year of age (*Ryland & Gorham* 1978). The natural breeding season lasts from March–April to July–August (*Hammond & Chesterman* 1972, *Willis & Barrow* 1971, *Beck et al.* 1976, *Ryland & Gorham* 1978). However, they can be induced to breed under certain regimes of light manipulation. To obtain pregnant ferrets at other times of the year it is necessary to have a "short day" room (8 hs of artificial light) and a "long day" room (14 hs of artificial light). The basic principle is to keep the females in the short day room and transfer them to the long day room 3 months before they are required for breeding (*Beck et al.* 1976). They cannot be kept in a long day room for more than 6 months because they may become refractory after this period (*Beck et al.* 1976).

An alternative way of having males and females available for breeding is to keep them under a constant short day photoperiod. The males will nearly always be available for breeding and the females will breed at times determined by their dates of birth, after the first estrous at 7–8 months of age (*Hammond & Chesterman* 1972).

Estrous is indicated by the progressive swelling of the vulva to full size within a month, caused by rising estrogen levels. The animal will ovulate after coitus (*Hammond & Chesterman* 1972, *Beck et al.* 1976, *Ryland & Gorham* 1978). The testes enlarge during the breeding period and the scrotum loses much of its hair when the males are fertile (*Beck et al.* 1976). Coitus is protracted and may take from half an hour to several hours (*Willis & Barrow* 1971, *Hammond & Chesterman* 1972, *Ryland & Gorham* 1978). To decrease the failure rate, females can be mated twice (*Beck et al.* 1976 *Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Moody et al.* 1985).

After a gestation period of 42 ± 2 days (*Willis & Barrow* 1971, *Ryland & Gorham* 1978), a litter varying from 5 to 15 (mean of 9) is delivered. Fertility will decline after 3 to 4 years of age (*Ryland & Gorham* 1978).

2.3. Housing and handling

Ferrets can be housed outdoors very much like mink, provided that measures are taken

to avoid exhaustion in warm weather (*Hammond & Chesterman* 1972, *Moody et al.* 1985). Indoor housing is recommended for experimental ferrets or where breeding cycles are controlled artificially (*Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Moody et al.* 1985). Optimal room temperature is around 15°C (*Moody et al.* 1985). Ferrets do not tolerate well temperatures over 32°C, as their sweat glands are not well developed (*Ryland et al.* 1978). Optimal humidity is 40–65 % (*Moody et al.* 1985).

Different types of cages have been described. Cat or rabbit cages can be used, with different sizes depending on the number of ferrets that are to be housed together (*Hammond & Chesterman* 1972, *Moody et al.* 1985). The cages may be constructed from heavy wire mesh (*Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Moody et al.* 1985) or wood (*Hammond & Chesterman* 1972). Wire mesh will permit most waste materials to fall into collecting troughs or trays below (*Ryland & Gorham* 1978). A solid floor, covered with shavings or newspaper, is also satisfactory (*Hammond & Chesterman* 1972, *Moody et al.* 1985). Also, if ferrets are fed pellets, a solid floor is better (*Hammond & Chesterman* 1972).

Ferrets have a propensity to use one corner of their cage for droppings, which makes cage cleaning easier (*Moody et al.* 1985). The cage should also have an enclosed area where the ferrets can rest undisturbed (*Moody et al.* 1985).

Ferrets can be housed individually or in groups of the same sex. However, during the breeding season males may become aggressive (*Ryland & Gorham* 1978, *Moody et al.* 1985). Females in estrous should not be placed together, as ovulation and pseudopregnancy may be induced by their play (*Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Moody et al.* 1985).

Ferrets are generally tame and may be handled without gloves, once they have been adapted to their environment (*Willis & Barrow* 1971, *Hammond & Chesterman* 1972). Light leather gloves may be worn if there is doubt about the disposition of the animal (*Moody et al.* 1985), although this may make it more difficult to hold the ferret firmly without hurting it (*Hammond & Chesterman* 1972). Many ways of restraining the ferret have been described. One method is to grasp the ferret over the shoulders and squeeze the forelimbs together using the other hand to support the hindlimbs (*Moody et al.* 1985). Another way is to place the hand across the animal's shoulders with the thumb and forefinger around the neck and the other fingers around the chest behind the forelimbs (*Hammond & Chesterman* 1972). Rarely, the animal may bite. If this happens, it is suggested that rather than trying to force the mouth open, hold its head under cold running water (*Hammond & Chesterman* 1972).

2.4. Nutrition

The nutritional requirements of the ferret have not been determined (*Ryland et al.* 1983, *Moody et al.* 1985, *Fox* 1988), but it is necessary to establish these to increase the efficiency and the validity of using this animal as a model for diverse biomedical applications (*Fox* 1988). Most of the information that is available has come from studies with mink (*Hammond & Chesterman* 1972, *Ryland et al.* 1983, *Moody et al.* 1985). Ferrets may consume 200–300 kcal/kg body weight for maintenance, but for growth and reproduction this level should be increased to 5000 kcal/kg diet (*Fox* 1988). The composition of the diet is dependent on the different phases of the life cycle. In mink, it has been found that for optimal growth 42 % of protein is necessary. For maintenance this amount may be decreased to 26–36 % (*Fox* 1988). The minimum level of dietary fat depends on its fatty acid composition. If high concentrations of unsaturated fats are used that may lead to rancidity and Vitamin E deficiency in the animal (*Moody et al.* 1985, *Fox* 1988). In this case supplementation with at least 10 mg of

Vit. E daily will avoid the development of yellow fat disease (*Moody et al.* 1985, *Fox* 1988). It is probable that the ferrets can be maintained without carbohydrates as long as the diet provides enough fat or protein (*Fox* 1988). There are many diet formulations. Natural formulations may include horsemeat, milk and rib-bone (*Willis & Barrow* 1971) or Wayne dog food cereal, Agway dog food cereal, beef tripe, beef lung, beef liver and cod liver oil (Marshall Farms, USA) (*Fox* 1988). Commercial formulations eliminate the labour of food preparation and the daily cleaning of food dishes, and they reduce costs (*Hammond & Chesterman* 1972, *Fox* 1988). Mc Lain Marshall Purified, Ralston Purina 5280, Agway Feed and Ralston Purina Cat Chow are some of the commercial formulations available (*Fox* 1988). These diets vary considerably in composition, containing from 34–47 % protein, 10–28 % fat and 22–42 % carbohydrate. Their energy value has a range from 3.98–4.90 kcal/g (*Fox* 1988). Milk is a good source of supplementary calcium (*Ryland & Gorham* 1978, *Moody et al.* 1985). At our laboratory commercial cat food has been used.

The ferret's milk has a high fat content and if necessary, young ferrets may be fed with cream enriched cow's milk or a milk and egg yolk mixture (*Hammond & Chesterman* 1972). Fresh water should be available at all times (*Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Moody et al.* 1985).

2.5. Diseases

Ferrets are susceptible to a variety of viral, bacterial and mycotic infections.

Ferrets are highly susceptible to the virus of canine distemper. The mortality rate may approach 100 % (*Hammond & Chesterman* 1972, *Ryland et al.* 1983, *Moody et al.* 1985, *Pecquet Goad* 1987). Moreover, they are used as test animals for the detection of this virus (*Ryland & Gorham* 1978). Ferret kits should be vaccinated against this infection at 6, 10 and 14 weeks of age (*Appel & Harris* 1988). After this primary vaccination one booster every year is sufficient (*Ryland & Gorham* 1978). Another vaccination scheme indicates a first dose of the vaccine at 6–8 weeks of age followed by a booster 2w later and another booster every 3 years (*Ryland et al.* 1983). Human influenza virus can also infect ferrets. It may cause initial signs similar to those of canine distemper, but the course of the disease is shorter and the recovery rate is high (*Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Moody et al.* 1985). Vaccination with attenuated live virus gives protection for 5 weeks, but killed vaccine is ineffective (*Ryland & Gorham* 1978) and is not recommemded routinely (*Ryland et al.* 1983). Other viral infections are Aleutian disease and rabies (*Ryland & Gorham* 1978, *Pearson* 1987). Vaccination against rabies should be administered annually, beginning at 3 months of age, and using only killed vaccine (*Ryland et al.* 1983).

Avian, bovine and human strains of mycobacteria can cause tuberculosis in ferrets (*Hammond & Chesterman* 1972, *Ryland & Gorham* 1978, *Ryland et al.* 1983). Lesions are located primarily in the alimentary tract and abdominal lymph nodes (*Hammond & Chesterman* 1972, *Ryland et al.* 1983, *Moody et al.* 1985). Ferrets are also susceptible to Botulism, types A, B and C (*Ryland & Gorham* 1978, *Ryland et al.* 1983). There is no treatment for the disease, but it can be prevented by excluding from the diet food items of unknown origin or by the prophylactic use of toxoid (*Ryland & Gorham* 1978). Staphylococcal, streptococcal and corynebacterial infections may cause abscesses arising from injuries to the mouth by bones in the diet or from bite wounds incurred during mating (*Ryland & Gorham* 1978, *Moody et al.* 1985).

Gangrenous mastitis is a rapidly progressive disease with high mortality (*Liberson et al.* 1983) and hemolytic Escherichia coli has been implicated as an important causative

agent in the domestic ferret (*Liberson et al.* 1983).

Proliferative colitis with a mortality rate of 100 % has also been described, where the causative bacterium was Campylobacter fetus subsp. jejuni (*Ryland et al.* 1983, *Moody et al.* 1985, *Fox* 1987). Recently, a Campylobacter-like organism has been associated with chronic gastritis (*Tompkins et al.* 1988). This microorganism was initially named Campylobacter pylori subspecies mustelae (*Fox et al.* 1988). C. mustelae was renamed later Helicobacter mustelae (*Goodwin et al.* 1989). H. mustelae associated gastritis in ferrets is considered a good animal model of Helicobacter pylori gastritis in humans (*Fox et al.* 1990, *Fox et al.* 1991).

Mycotic infections may include superficial mycoses such as 'ring worm' (Microsporum canis) and systemic infections including blastomycosis, cryptococcosis and histoplasmosis (*Moody et al.* 1985, *Fox* 1987). Parasites, as common as in dogs and in cats, include Sarcoptis scabei (pruritus), Ctenocephalides spp. (fleasmites), Toxoplasma, Giardia, Filaroides spp. (*Ryland & Gorham* 1978, *Ryland et al.* 1983, *Moody et al.* 1985). Diagnosis and treatment of these infections follow prescribed methods used for dogs and cats (*Ryland & Gorham* 1978, *Fox* 1987).

One of the most common syndromes in females ferrets is severe bone marrow depression associated with high estrogen levels in prolonged estrous. There is a great prevalence of this disease (50 %), mainly between April and July and in contact non breeding females (*Ryland & Gorham* 1978, *Moody et al.* 1985).

Litters of ferret kits show a variety of spontaneous congenital malformations, which include anaencephaly, neuroschisis, gastroschisis, cataracts and eye opacity (*Willis & Barrow* 1971, *Ryland & Gorham* 1978, *Moody et al.* 1985, *Dillberger & Altman* 1989).

Ferrets develop a number and variety of tumors similar to that seen in other domestic carnivores (*Pecquet Goad* 1987, *Dillberger & Altman* 1989). The most frequently occurring tumors are ovarian stromal tumors, haemangiomas and haemangio sarcomas (*Dillberger & Altman* 1989). The tumors may occur in all organ systems except the respiratory tract and central nervous system (*Dillberger & Altman* 1989).

Squamous cell carcinomas (*Ryland & Gorham* 1978, *Ryland et al.* 1983, *Moody et al* 1985, Pecquet Goad 1987), pancreatic adenocarcinomas (*Moody et al.* 1985, *Pecquet Goad* 1987), sebaceous adenoma, epithelioma of the skin (*Dillberger & Altman* 1989) and mast cell tumors (*Moody et al.* 1985, *Pecquet Goad* 1987) have also been observed in ferrets.

2.6. Anaesthetic procedures

Ferrets present no particular problem and the procedure chosen will depend on the time necessary for the chemical restraint and sedation of the animal (*Green* 1982, *Ryland et al.* 1983). Fasting, at least 6 hours before the induction of anaesthesia, is advisable, as ferrets frequently vomit when they are anaesthetized (*Green* 1982).

Ketamine, alone, or in combination with xylazine or diazepam may be used. The doses recommended vary from 10–60 mg/kg, im, of ketamine, when used alone (*Green* 1982, *Moreland & Glaser* 1985). In conjunction with xylazine (2 mg/kg, im) or with diazepam (3 mg/kg, im) the dose of ketamine used decreases to 25 mg/kg (*Green et al.* 1981, *Moreland & Glaser* 1985). The disadvantage of using ketamine alone is that the animals have muscle tremors, muscle rigidity, excessive salivation, paddling motions and an unstable ECG baseline (*Green et al.* 1981, *Moreland & Glaser* 1985, *Bone et al.* 1989). The duration of the anaesthesia varies between 40–60 min (*Green et al.* 1981, *Moreland & Glaser* 1985). The cardic arrhythmias that may be produced by xylazine, such as bradycardia and conduction disturbances, may be controlled by atropine (0.05 mg/kg, im or sc) (*Green* 1982, *Moreland & Glaser* 1985). Fentanyl-Fluanisone

(0.3–0.5 ml/kg, im) (*Green* 1978, *Green* 1982) produces a neuroleptanalgesia which is rapidly reversed with nalorphine, im. Tremors and excitability may be encountered occassionally 2–3 min. after administration (*Green* 1982).

Barbiturates can be used as an anaesthetic in ferrets, although the relative inaccessibility of peripheral veins may limit their use (*Green* 1982, *Ryland et al.* 1983). The recommended dose is 30–36 mg/kg, ip, and anaesthesia lasts for 50–120 min. Full recovery may take hours and be accompanied by excitatory behaviour, including twitching and limb paddling movements. These adverse signs can be diminished by sedative premedication (*Green* 1978, *Green,* 1982, *Ryland et al.* 1983).

For longer surgical procedures inhalation anaesthesia, delivered by mask or by endotracheal tube 3.0 is a good alternative. Methoxyfluorane or halothane with N2O:O2 (1:1) can be used at a flow rate of 1–2 liter/min (*Green* 1982, *Ryland et al.* 1983). Enflurane (*Green et al.* 1981), isoflurane (*Housmans & Murat* 1988) and fluothane with O2 (*Brown-Harcourt & Brown-Harcourt* 1978) can also be used.

In our laboratory, Ketamin (30 mg/kg, im) and Xylazine (2 mg/kg, im), associated with Atropine (0.05 mg/kg, s.c) has been the anaesthetic method of choice. It gives good relaxation for a maximum period of 40 minutes.

3. Oral cavity

The ferret has a deciduous dental formula of D.I 4/3, D.C 1/1 and D.M 3/3 and its permanent dental formula is I 3/3, C 1/1, PM 4/3 and M 1/2 (*Berkovitz & Silverstone* 1969). The first premolar is not present, thus, the D.M 2 in the deciduous dention and the PM 2 in the permanent dentition are the first cheek teeth (*Berkovitz & Silverstone* 1969. The carnasial teeth are the D.M 4, in the deciduous dention, and the P.M 4 (upper jaw) and the M1 (lower jaw) in the permanent dentition (*Berkovitz & Silverstone* 1969, *Berkovitz* 1973). The carnasial teeth together with the canines are considered to be the most suitable teeth for histological examination (*King* 1944 b, *Berkovitz & Silverstone* 1969). The carnasial teeth appear in the permanent dentition between 50 and 60 days, both in the upper and in the lower jaw (*Berkovitz & Silverstone* 1969, *Berkovitz* 1973). A high number of supranumerary incisors have been found in the deciduous and in the permanent dentitions (*Berkovitz & Thomson* 1973).

The first reports of the use of the ferret as an experimental animal in periodontal research are by *King* (*King* 1944 a, b, *King & Glover* 1945, *King & Gimson* 1947 a, b, *King et al.* 1955, *Berkovitz & Silverstone* 1969). More recently gingival hyperplasia has also been studied in ferrets (*King & Gimson* 1947 b, *Steinberg et al.* 1972, *Hall & Squier* 1982). In endodontic research the ferret canine has been used in pulpectomy (*Holland* 1988) and apicectomy studies (*Callis & Santini* 1987).

Periodontal disease has been found to be similar to that in man (*King* 1944 a, b). The lesions appear first around the carnasial teeth, normally related to the presence of calculus deposits (*King* 1944 a, b, *King & Gimson* 1947 a, b). Even gingival hyperplasia has been described to occur around these teeth (*King & Gimson* 1947 b, *Steinberg et al.* 1972, *Hall & Squier* 1982). It may be of significance that the salivary glands of the ferret, as in the dog and cat, open into the mouth in the carnasial regions of the upper jaw (*King* 1944 b). As the tartar accumulations spread forward to the canine and incisor regions, gingival changes begin to appear (*King* 1944 a). Chemical analyses of the deposits show similarity to human calculus (*King et al.* 1955). It has been suggested that the content of carbonate in ferret calculus is more similar to human calculus than that in the dog (*King et al.* 1955, *Harper et al.* 1990, *Mann et al.* 1990). The main difference between animal and human calculus is the smaller degree of calcification on the

animal deposits (*King et al.* 1955). It has been suggested that periodontal disease is diet related (*King* 1944 a, b, *King & Gimson* 1947 a, b, *King et al.* 1955). *King* stressed that ferrets maintained on a diet of bread and milk, oat meal and milk or raw meat and milk developed periodontal disease in 8–12 weeks or less. The addition of short lengths of bone (plus muscular and other attachments) to these diets prevented the development of the lesions and also cured the established disease. The curative action of bone was attributed to a mechanical effect which prevented and removed deposits of salivary calculus (*King & Glover* 1945).

Clinically, the first sign of periodontal disease is redness of the gum margin. Hyperemia, hemorrhage, hypertrophy, ulceration, involvement of the alveolar bone and loosening of the teeth may follow (*King* 1944 a, b). Loss of the keratinous layer of the surface epithelium in the region of the gingival crest, dilation of the marginal capillaries and proliferation of the "subsurface gingival epithelial papillae" represent the earlier changes in the histological preparations. Later, leucocytic infiltration of the corium may appear (*King* 1944 a, b). At a late stage the alveolar bone may begin to atrophy, and resorption of the cementum and dentine of the tooth root may also occur (*King* 1944 a, b).

In our laboratory, histometric evaluation and the oral mucosal and gingival sulcus microflora related to experimental periodontitis induced by the placement of ligatures has been studied both in normal and immunosuppressed domestic ferrets. The results from these studies will be presented in subsequent papers in the dental literature.

Summary

Ferrets have been widely used as experimental animals in biomedical research. In periodontal research, increasing concerns about the use of dogs and monkeys as experimental animals has added to the interest in the use of the ferrets as an experimental animal. The aim of this review is to present aspects of the animals biology and husbandry, including reproduction, housing and handling, nutritional requirements, diseases and anaesthesia. Finally, the characteristics of dentition and periodontal diseases are presented.

Sammanfattning

Iller används i relativt stor omfattning som försöksdjur i biomedicinsk forskning. Inom paradontologisk forskning, där djurförsök är oundgängliga, strävar man efter att finna ersättning för hund och apa som används idag. Illern verkar erbjuda en möjlighet att i vissa fall ersätta dessa andra djurslag. Avsikten med denna översiktsartikel är att presentera illern i allmänhet, dess reproduktion, inkvartering och hantering, nutrition, sjukdomar av betydelse för illern samt olika anestesimetoder. Slutligen beskrivs mer detaljerat illerns tanduppsättning och dess paradontala sjukdomar.

Yhteenveto / K. Pelkonen

Frettejä on paljon käyletty koe-eläimenä biolääketieteellisessä tutkimuksessa. Kun koinien ja apinoiden käyttö koe-eläminä on tullul kasvavan julkisen arvostelun kohteeksi, on kännostus tretin käyttöö hammaslääketieteellisessä lutkimuksessa lisääntynøt. Tässä katsauksessa käsitellään frelin biologiaa, kasvatusta ja luolantoa, käsittelyä, ravilsemusta, tauteja ja anestesiaa. Lopuksi kuvailiaan frelin hampaisto ja hammasseiraudel.

References

American Veterinary Medical Association Council. J. Am. Vet. Assoc. 1986, *188,* 121.

American Veterinary Medical Association Council. J. Am. Vet. Assoc. 1986, *188,* 1122.

Appel MJ & WV Harris: Antibody titers in domestic ferrets jills and their kits to canine distemper virus vaccine. J. Am. Vet. Med. Assoc. 1988, *193,* 332–333.

Beck F, H Schön, G Mould, P Swidzinska, S Curry & J Grauwiler: Comparison of the teratogenic effects of Mustine Hydrochloride in rats and ferrets. The value of the ferret as an experimental animal in Teratology. Teratol. 1976, *13,* 151–160.

Berkovitz BKB & LM Silverstone: The dentition of the albino ferret. Caries Res. 1969, *3,* 369–376.

Berkovitz BKB & P Thomson: Observations of the aetiology of the supranumerary upper incisors in the Albino ferret (Mustela Putorius). Arch. Oral Biol. 1973, *18,* 457–463.

Berkovitz BKB: Tooth development in the Albino ferret (Mustela Putorius) with special reference to the permanent carnasial. Arch. Oral Biol. 1973, *18,* 465–471.

Bone L, AH Battles, RD Goldfarb, CW Lombard & AF Moreland: Electrocardiograph values from clinically normal, anesthetized ferrets (Mustela Putorius furo). Am. J. Vet. Res. 1989, *49,* 1884–1887.

Brown-Harcourt NH & FM Brown-Harcourt: Anesthetizing ferrets. Vet. Rec. 1978, *102,* 269.

Callis PD & A Santini: Tissue response to retrograde root fillings in the ferret canine: a comparison of a glass ionomer cement and guttapercha with sealer. Oral Surg. Oral Med. Oral Pathol. 1987, *64,* 475–479.

Dillberger JE & NH Altman: Neoplasia in ferrets: Eleven cases with a review. J. Comp. Pathol. 1989, *100,* 161–176.

Fox JG: Introduction and history of ferrets. J. Am. Vet. Assoc. 1987, *190,* 1610.

Fox JG: Bacterial, Mycotic and parasitic diseases. J. Am. Vet. Assoc. 1987, *190,* 1610.

Fox JG: Biology and diseases of the ferret. Lea & Febger, Philadelphia. 1988, cap. 5, pg. 135.

Fox JG, NS Taylor, P. Edmonds & DJ Brenner: Campylobacter pylori subsp. mustelae subsp. nov. isolate from the gastric mucosa of ferrets (Mustela putorius furo), and an emended description of Campylobacter pylori. Int. J. Syst. Bact. 1988, *38,* 367–370.

Fox JG, P Correa, NS Taylor, A Lee, G Otto, JC Murphy & R Rose: Helicobacter mustelae-associated gastritis in ferrets. An animal model of helicobacter pylori gastritis in humans. Gastroenterology 1990, *99,* 352–361.

Fox JG, G Otto, NS Taylor, W Rosenblad & JC Murphy: Helicobacter mustelae-induced gastritis and elevated gastric pH in the ferret (Mustela putorius furo). Infect. Immun. 1991, *59,* 1875–1880.

Goodwin CS, JA Armstrong, T Chilvers, M Peters, P Edmonds, MD Collin, L Sly, W McConnel & WES Harper: Transfer of Campylobacter pylori and Campylobacter mustelae to Helicobacter gen. nov. as Helicobacter pylori comb. nov. and Helicobacter mustelae comb. nov., respectively. Int. J. Syst. Bacteriol. 1989, *39,* 397–405.

Green CJ: Anesthetizing ferrets. Vet. Rec. 1978, *102,* 269.

Green CJ, J Knight, S Precious & S Simpkin: Ketamine alone and combined with diazepam or xylazine in laboratory animals: a 10 year experience. Lab. Anim. Sci. 1981, *15,* 163–170.

Green CJ: Animal anaesthesia – Laboratory Animals LTD, London 1982, cap. 12, pg. 199.

Hall BK & CA Squier: Ultrastructural quantitation of connective tissue changes in phenytoin induced gingival overgrowth in the ferret. J. Dent. Res. 1982, *61,* 942–952.

Hammond J & JR Chesterman: The ferret. The UFAW handbook on the care and management of laboratory animal. 4th ed. Edinburgh 1972.

Harper DS, PH Mann & S Regnier: Measurement of dietary and dentifrice effects upon calculus accumulation rates in the Domestic Ferret. J. Dent. Res. 1990, *69(2),* 447–450.

Holland GR: Periapical innervation of the ferret canine and the local retrograde neural changes after pulpectomy. Anat. Rec. 1988, *220,* 318–327.

Housmans PR & I Murat: Comparative effects of halothane, enflurane and isoflurane at equipotent anesthetic concentration on isolated myocardium of the ferret. II. Relaxation. Anesthesiology 1988, *69,* 464–471.

King JD: Experimental investigations of Parodontal disease. 1. The significance of the blood supply in gingival health and disease of the ferret. Br. Dent. J. 1944 a, *77,* 213–221.

King JD: Experimental investigations of Parodontal disease. 1. The significance of the blood supply in gingival health and disease of the ferret. Br. Dent. J. 1944 b, *77,* 245–254.

King JD & RE Glover: The relative effects of dietary constituents and other factors upon calculus formation and gingival disease in the ferret. J. Path. Biol. 1945, *57,* 353–362.

King JD & AP Gimson: Experimental investigations of parodontal disease in the ferret and related lesions in man. Br. Dent. J. 1947 a, *80,* 126–127.

King JD & AP Gimson: Experimental investigations of parodontal disease in the ferret and related lesions in man. Br. Dent. J. 1947 b, *80,* 148–149.

King JD, SL Rowles, K Little & J Thewlis: Chemical and X Ray examination of deposits removed from the teeth of golden hamsters and ferrets. J. Dent. Res. 1955, *34,* 650–660.

Liberson AJ, CE Newcomer, JI Ackerman, JC Murphy & JG Fox: Mastitis caused by hemolytic Escherichia coli in the ferret. J. Am. Vet. Med. Assoc. 1983, *183,* 1179–1181.

Mann PH, DS Harper & S Regnier: Reduction of calculus accumulation in Domestic Ferrets with two dentifrices containing Pyrophosphate. J. Dent. Res. 1990, *69 (2),* 451–453.

Moody KD, TA Bowman & M Lang: Laboratory management of the ferret for biomedical research. Lab. Anim. Sci. 1985, *35,* 272–279.

Moreland AF & C Glaser: Evaluation of ketamine, ketamine-xylazine and ketamine-diazepam in the ferret. Lab. Anim. Sci. 1985, *35,* 287–290.

Pearson RC: Viral diseases of ferrets. J. Am. Vet. Assoc. 1987, *190,* 1610.

Pecquet Goad ME: Neoplasia in the ferret. J. Am. Vet. Assoc. 1987, *190,* 1610.

Ryland LM & JR Gorham: The ferrets and its disease. J. Am. Vet. Assoc. 1978, *173,* 1154–1158.

Ryland LM, SL Bernard & JR Gorham: A clinical guide to the pet ferret. Compend of continu-

ing education for the practicing veterinarian. 1983, *5,* 25–32.

Shump AU & KA Shump: Growth and development of the European ferret. (Mustela Putorius). Lab. Anim. Sci. 1978, *38,* 89–91.

Steinberg AD, J Alvarez & H Jeffay: Lack of relationship between the degree of induced gingival hyperplasia and the concentration of diphenylhydantoin in various tissues of ferrets. J. Dent. Res. 1972, *51,* 657–662.

Tomkins DS, JI Wyatt, BJ Rathbone & AP West: The characterization and pathological significance of gastric Campylobacter like organisms in the ferret: a model for chronic gastritis? Epidemiol. Infect. 1988, *101,* 269–278.

Wen GY, JA Sturman & JW Shek: A comparative study of tapetum, retina and skull of the ferret, dog and cat. Lab. Anim. Sci. 1985, *35,* 200–210.

Willis LS & MV Barrow: The ferret (Mustela putorius furo) as a laboratory animal. Lab. Anim. Sci. 1971, *21,* 712–716.

Wright CA: Biochemical and immunological taxonomy of animals. Academic Press, London 1974, cap. 1 pg. 13.