Int. Zoo Yb. (2010) **44:** 16–32
DOI:10.1111/j.1748-1090.2009.00097.x

Veterinary issues related to bears (Ursidae)

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A wide variety of infectious and non-infectious diseases has been described in bears. Some viral (e.g. canine distemper, infectious canine hepatitis), bacterial (e.g. salmon poisoning) and parasitic diseases (particularly skin mites and ascarid infections) are of concern. Non-infectious conditions, such as dental disease, degenerative joint disease and neoplasia, are very important in the management of captive bears. Appropriate anaesthesia is essential in both veterinary and biological interventions with bears.

Key-words: anaesthesia; *Baylisascaris*; canine distemper; degenerative joint disease; dental disease; infectious canine hepatitis; neoplasia; *Neorickettsia*; pseudorabies; Ursidae.

INTRODUCTION

Bears are large, charismatic mammals that have been the subject of numerous disease studies both in the wild and in captivity. This review emphasizes important infectious and non-infectious diseases, and provides information on anaesthesia. More complete listings of recorded diseases can be found in Bourne & Vila-Garcia (2007), Mainka (1999) and Ramsay (2003).

INFECTIOUS AND PARASITIC DISEASES

Viral diseases

Antibodies to a wide variety of viruses (e.g. avian influenza, bluetongue, canine parvovirus, eastern, western and Venezuelan equine encephalitis viruses) have been detected in bears, without associated clinical disease (Bourne & Vila-Garcia, 2007).

Bears are susceptible to canine distemper virus (CDV) but clinical disease has been recorded only rarely; fatal disease has occurred in neonatal cubs of Polar bears *Ursus maritimus*, Spectacled bears *Tremarctos ornatus* and Giant pandas *Ailuropoda melanoleuca* (Schönbauer *et al*., 1984; Deem *et al*., 2000). A canarypox-vectored recombinant CDV vaccine has been shown to be safe for use in Giant pandas, with inoculated pandas developing serum-neutralizing antibody titres that were interpreted as protective (Bronson *et al*., 2007).

Infectious canine hepatitis (canine adenovirus 1 infection) has been recorded on a few occasions, causing excessive salivation, vomiting, diarrhoea and signs of abdominal pain, as well as neurological signs; many affected bears died. Surviving bears may have prolonged (2–3 months) convalescence; corneal opacity has been reported but regressed spontaneously (Pursell *et al*., 1983; Collins *et al*., 1984; Kritsepi *et al*., 1996).

Bears should never be fed on pig meat because they are susceptible to fatal infection with Aujeszky's disease (pseudorabies), an important herpesvirus disease of pigs. Clinical signs have included lethargy and depression, anorexia, excessive salivation, nervousness and excitement, tremors, apparent inability to swallow in some individuals, localized or generalized pruritis leading to self-mutilation, diarrhoea, vomiting, haematuria or dark urine, dyspnoea and paralysis (Schultze *et al*., 1986; Salvelli & Zanin, 1996; Banks *et al*., 1999; Bourne & Vila-Garcia, 2007).

Rabid bears have been recorded only rarely (Bourne & Vila-Garcia, 2007). Natural infection with the West Nile virus caused severe disease requiring euthanasia in one Polar bear at a Canadian zoo (Dutton *et al*., 2009), but otherwise infection has been reported only from the presence of antibodies (Bourne & Vila-Garcia, 2007).

Bacterial diseases

Neorickettsia spp are responsible for the disease complex known as 'salmon poisoning/Elokomin fluke fever'. These organisms infect a trematode (fluke), which then develops through two intermediate hosts, a snail and a fish (usually salmonid), before the definitive mammalian host is infected when it eats an infected fish. The rickettsial organisms are released into the bloodstream after the adult fluke penetrates the mucosal lining of the gut (Reed, 2006). Originally, this disease was seen only in the Pacific northwest of North America (the snail intermediate host is endemic to this area) (Reed, 2006); however, it has occurred elsewhere following transplantation of hatchery salmonids from that area for sport fishing (Gai, 2007). Clinical signs include lethargy, anorexia, diarrhoea and sometimes vomiting (Farrell *et al*., 1973; Gai & Marks, 2005). Thorough freezing of fish kills the flukes and prevents transmission. Prompt treatment (e.g. oxytetracycline against the rickettsia plus a flukicide) is effective. Bears native to the Pacific north-west of North America appear to be less susceptible to development of clinical disease than non-native species (Gai & Marks, 2005).

As with many other species, bears are susceptible to clostridial organisms, both as enteric infections (*Clostridium difficile*, *Clostridium perfringens*) and causing myonecrosis (*C. perfringens*) (Bourne & Vila-Garcia, 2007); one case of myonecrosis followed intramuscular administration of anaesthetic drugs while another was associated with a facial wound (Barnes & Rogers, 1980; Rao *et al*., 1988). *Escherichia coli* infection is a cause of haemorrhagic enteritis in Giant pandas (Mainka, 1999) and has caused septicaemia in neonatal Polar bears, and fatal gastritis and acute catarrhal enteritis in Spectacled bear cubs (Wolff, 1989; Dollinger *et al*., 1996). Generalized dermatophilosis (*Dermatophilus congalensis* infection) has been seen in captive Polar bears, with hair yellowing, later greasy darkening, clumping of the coat, formation of crusty dark scabs, reddening of the skin under the scabs, pruritis and reluctance to bathe; alopecia may develop. The disease may be worse in summer and is thought to be spread by insects when wet conditions reduce the normal resistance of the skin and release zoospores (motile cocci) from pre-existing lesions. Antibiotic treatment may be effective, but the condition may recur and nutritional or environmental predisposing factors must be considered and addressed (Newman *et al*., 1975; Bourne & Vila-Garcia, 2007).

Tuberculosis is reported as a rare disease of bears (Bourne & Vila-Garcia, 2007). However, there appears to be a high incidence in ex-dancing bears at Wildlife SOS Sloth bear *Melursus ursinus* rescue facilities in Agra and Bannerghata, India, as confirmed at post-mortem examination (A. A. Shanmugam, pers. comm.).

Fungal diseases

Ringworm has been seen in a number of bears. *Trichophyton* sp infection was confirmed in wild-born American black bear *Ursus americanus* cubs being hand-reared (Convy, 2002) and rehabilitated, and *Microsporum canis* in Sun bears *Helarctos malayanus* in a zoo. In the Sun bears, lesions on the head and body were up to 8 cm in diameter, with moderate alopecia and scaling (Groves, 1969). Concurrent ringworm and sarcoptic mange sometimes occurs in *U. americanus* cubs during rehabilitation (J. R. Huckabee, pers. comm.). Other fungal diseases, including candidiasis, blastomycosis, pythiosis (*Pythium insidiosum* infection) and *Pityrosporum pachydermatis* infection, have been reported only rarely (Bourne & Vila-Garcia, 2007).

Parasitic diseases

Mite infections have been reported in several bear species but most commonly in American black bears; audycoptic and sarcoptic mange are considered to be particularly important. Audycoptic mange as a result of *Ursicoptes americanus* mites causes alopecia, pruritis and crusting (this may be seen around the head or may be more generalized), while sarcoptic mange may cause pruritis, alopecia, pustular dermatitis and crusting and thickening of the skin (Fowler, 1986; Bourne & Vila-Garcia, 2007). Demodicosis has been described in wild American black bears from one area of Florida (possibly related individuals) (Foster *et al*., 1998) and in Giant pandas (Janssen, Edwards *et al*., 2006).

Gastro-intestinal nematode infections are very common in bears. Hookworm (*Ancyclostoma* and *Uncinaria* spp) infections are most severe in young cubs. Diarrhoea, blood in the faeces, anorexia, weight loss, anaemia and debilitation can occur; infection may be fatal in juveniles. Appropriate treatments include ivermectin, $0 \cdot 3\,\mathrm{mg\,kg^{-1}}$ subcutaneously or orally once, repeated at 8 week intervals; levamisole $10\,\mathrm{mg\,kg^{-1}}$ orally or subcutaneously (note: toxic if the dose is doubled), fenbendazole or febantel orally $50\,\mathrm{mg\,kg^{-1}}$ daily for 3 days or $20\,\mathrm{mg\,kg^{-1}}$ daily for 5 days (Greenwood, 1992; Ramsay, 2003).

Ascarid (large roundworm), mainly *Baylisascaris transfuga*, infections are very common and can cause diarrhoea and anorexia. Severe infections can result in poor body condition, and in overwhelming infections worms may fatally block the small intestines. Recommended treatments include ivermectin ($0 \cdot 3\,\mathrm{mg\,kg^{-1}}$ subcutaneously or orally), milbemycin oxime ($1\,\mathrm{mg\,kg^{-1}}$ orally), fenbendazole ($50\,\mathrm{mg\,kg^{-1}}$ orally for 3 days or $20\,\mathrm{mg\,kg^{-1}}$ orally for 5 days) or mebendazole ($20\,\mathrm{mg\,kg^{-1}}$ daily for 3 days) (Ramsay, 2003; Meyerson, 2007). Anthelmintics are effective but reinfection is common. Ascarid ova are not destroyed by routine cleaning and disinfection; high-pressure hoses spread the ova around the enclosure and they can remain infective for several years. A blowtorch can be used to spot-treat appropriate surfaces to destroy ova (Abdelrasoul & Fowler, 1979; Ramsay, 2003). Repeated treatment with an effective anthelmintic keeping faecal egg counts to zero may allow eventual elimination of infection from an enclosure (Moran *et al*., 1994). In Giant pandas, intestinal infection with *Baylisascaris schroederi* has been described as a limited cause of mortality; recently, visceral larval migrans, owing to this or possibly another *Baylisascaris* sp, has been implicated as an important cause of mortality in wild pandas (Zhang *et al*., 2008).

A wide variety of other parasitic infections has been recorded in bears, including cestodes (both adult tapeworms in the intestines and larval forms), trematodes (flukes), acanthocephalans, *Dirofilaria ursi*, toxoplasmosis (sometimes fatal), trichinellosis, lice, fleas, ticks and others (Rogers & Rogers, 1976; Kiupel *et al*., 1987; Bourne & Vila-Garcia, 2007). Appropriate therapy against trematodes is part of treating the rickettsial disease complex 'salmon poisoning/Elokomin fluke fever' as indicated above.

NON-INFECTIOUS DISEASES

Dental disease

Dental pathology is almost ubiquitous in captive bears and should therefore be of primary veterinary concern. In one study, $>70\%$ of zoo bear skulls studied had broken or open-tipped canine teeth (Kitchener, 2004). Dental disease is a significant welfare issue and may also predispose to secondary local or systemic bacterial diseases (DeBowes *et al*., 1996) affecting the myocardium, kidneys or bones.

The primary bear dental disease syndromes are fractured teeth, caries and calculus build-up. Calculus deposition and periodontal disease may be prevented and managed through dietary and management changes: minimizing high-sugar items and feeding a diet containing dry bear kibble, vegetables and fruit, plus enrichment items, such as rawhides and bones, including the use of log and puzzle feeders, are all beneficial.

Bears kept in inadequate environments, or with severe behavioural problems, may resort to 'bar-biting'. This stereotypy, where bears chew compulsively on their cage bars, primarily causes erosion of the enamel from the lingual surfaces of the canine teeth and wearing of incisors, weakening teeth and predisposing to fractures. Additionally, fractures may occur as a result of trauma. Tooth fracture exposes the pulp, causing severe pain (although bears often do not show obvious external signs of this pain), and allows tracking of bacteria to the tooth apex, causing tooth root abscessation and alveolar osteomyelitis. Fractured canines usually have associated apical infection. Draining tracts from bilateral infected canine teeth can eventually weaken the rostral jaw bones, predisposing to pathological fractures.

There is no standardized treatment for fractured canine teeth; both endodontic and extraction techniques have been used in large numbers of Asiatic black bears *Ursus thibetanus* and Indian Sloth bears *M. ursinus* with apparent success. Preliminary data from the International Animal Rescue/Wildlife SOS Agra Bear Rescue Facility and Animals Asia's China Bear Rescue Centre indicate that these treatments may improve the activity and sociability of bears kept in groups (International Animal Rescue/Wildlife SOS Agra Bear Rescue Facility, pers. comm.; Animals Asia's China Bear Rescue Centre, pers. comm.). Endodontic therapy should be undertaken only by qualified personnel, and only after clinical and radiographic evaluation confirms that the tooth is suitable for such a treatment. There are theoretical concerns that ennervation of these teeth may result in their overuse with possible further damage to the tooth or the jaw. Extraction of ursid canine teeth is relatively straightforward and, when thorough clinical and radiographic examinations are performed to ensure any dental fragments are removed, post-operative complications are rare.

Good analgesia is vital. Ideally, a suitable combination of analgesia and anaesthesia should be used to provide neuroleptanalgesia. The protocol for dental work followed at the China Bear Rescue Centre consists of medetomidine–toletamine–zolezepam for anaesthetic induction and isoflurane in oxygen for anaesthetic maintenance, with bupivacaine mental, maxillary or infraorbital nerve blocks administered depending on the location of the tooth to be extracted, presurgical parenteral carprofen and buprenorphine, and post-operative oral carprofen. This has been used for >50 bears. No post-operative complications have been noted, with all bears eating softened food within 8 hours of surgery and returning to their social groups and normal diet without incidence within 24 hours post-surgery. No self-mutilation has been noted after facial numbing.

In the author's (H. J. B.) experience, there are no significant behavioural differences between bears with extracted canine teeth and those with a healthy dentition. Following dental extractions, bears appear to be able to maintain their social status, utilize enrichment items, and feed, play and fight as normal. Bears with no canine teeth have been observed dominating fully toothed bears using an open-mouth threat display and no long-term husbandry changes are required after dentistry. Canine teeth are important as a defensive tool in lower-ranking animals when challenged by higher-ranking individuals. However, it is debatable as to how effective fractured, infected and chronically painful canine teeth are; in such cases, maintenance of tooth structure through endodontic therapy may be the preferred option.

Degenerative joint disease (DJD) and mobility problems

DJD is an insidious but significant syndrome, usually developing over a number of years, primarily in aged animals. While it may be a natural consequence of ageing, inflammatory and infectious aetiologies may be involved. Rather than specific lameness, bears with DJD often become less active, sleep more, climb less and are generally slower or more irritable. Stiffness is almost always an indicator of underlying pathology. DJD includes osteoarthritic changes, joint trauma or sepsis and osteo- and spondyloarthropathies. Studies

have shown clinical demonstration of osteoarthritic changes in 56% of bears (Föllmi, 2005) and an incidence of spondyloarthrosis or osteoarthrosis in 27–96% (Kitchener, 2004; Nunn *et al*., 2007) of bear skeletons of various ages. As populations of captive bears are becoming increasingly geriatric (Kitchener, 2004), we have a responsibility to develop medical and husbandry strategies to meet their requirements, including managing DJD.

Joint problems can be minimized by a well-balanced diet, weight monitoring and adequate exercise facilities. Wild bears spend much of their time foraging and climbing. An ideal enclosure should allow these activities; the restrictive captive environments of many bears may contribute to DJD. It is vital that enclosures allow the expression of natural behaviours, to maintain both physical and mental health. Enrichment programmes allow bears to engage in natural behaviours; keeping staff should be encouraged to devise comprehensive and imaginative programmes maximizing the amount of time their bears are active (see also Law & Reid, 2010).

Obesity and mobility issues are common in captive bears, and are interrelated. Reducing body mass reduces the load on joints; maintenance of a healthy body mass is essential in preventing and managing lameness. As bears are very food motivated, any reduction in the diet should be carefully monitored to prevent aggression occurring within a bear group, and should ideally be timed with a natural seasonal reduction in food; for example, throughout winter.

Aged mammals are at particular risk of spondylarthropathies and these lesions are well described especially in species with a large body mass (Kolmstetter *et al*., 2000; Nunn *et al*., 2007). Regular visual assessment, physical examinations and radiographic evaluations help monitoring of disease progression.

DJD is known to cause pain. Because pain transmission involves multiple mechanisms, pathways and transmitters, it is advisable to apply the theories of multimodal analgesia to bears suffering from DJD, as probably no single therapy will provide complete pain relief (Lascelles, 2007). A variety of medical, surgical (Witz *et al*., 2001) or alternative therapies may be used.

Neutraceutical therapy may be helpful, although efficacy is not yet proven in bears. Supplements containing polysulphated glycosaminoglycans, such as chondroitin sulphate, reduce catabolic enzyme activity and inhibit serine proteinases, thus reducing collagen degradation. Glucosamine sulphate, an amino monosaccharide, may provide the 'building blocks' of cartilage and theoretically help to repair joint damage.

Non-steroidal anti-inflammatory drugs (NSAIDs) are very effective in the treatment of arthritic pain. Carprofen (Bourne & Vila-Garcia, 2007), meloxicam and tepoxalin may be used depending on individual response to treatment. Suggested drug doses for the treatment of DJD in bears are provided in Table 1.

Steroids should not be used as a first-line treatment because of their potent side-effects, but are used when mobility disease is caused by a neurological problem or where other therapies have failed. They are used orally or intraarticularly to relieve arthritic pain. They should not be used concurrently with NSAIDs and may be contraindicated when infectious disease is present.

Tramadol binds to m receptors and inhibits noradrenaline and serotonin reuptake, providing effective analgesia. It can be used in combination with both NSAIDs and steroids; therefore, it is useful in multimodal analgesia, but it should not be used with mood-altering drugs, such as selective serotonin reuptake inhibitors or monoamine oxidase inhibitors.

Other drugs that may be considered in a multimodal approach are gabapentin and amantadine, both of which are reported to be effective in the control of chronic or neurogenic pain.

Geriatric bear facilities, including non-slip flooring, low nesting areas and sympathetically designed drains, should be provided to reduce the strain on diseased joints. Keeping staff should be aware of the potential for sudden collapse in bears with mobility problems. In a group situation, the collapsed bear may be mauled by conspecifics that no longer recognize their denmate.

CLASS	DRUG	SPECIES	DOSE
NSAID	carprofen	Asiatic black bear *Ursus thibetanus*	2–4 · 4 mg kg^{-1} p.o., s.c., s.i.d.
	meloxicam	Brown bear *Ursus arctos*	0 · 5 mg kg^{-1} s.c. followed by 0 · 1 mg kg^{-1} p.o., s.i.d
		Asiatic black bear *U. thibetanus*	0 · 2 mg kg^{-1} p.o. for 5 days followed by 0 · 1 mg kg^{-1} p.o., s.i.d
		Andean bear *Tremarctos ornatus*	0 · 2 mg kg^{-1} p.o., s.i.d.
	tepoxalin	Asiatic black bear *U. thibetanus*	20 mg kg^{-1} p.o., s.i.d. once, followed by 10 mg kg^{-1} p.o., s.i.d.
Steroids	prednisolone	Polar bear *Ursus maritimus*	25–80 mg p.o., s.i.d. for treatment of allergic dermatitis with noted improvement in mobility also
	prednisolone/ cinchophen	Asiatic black bear *U. thibetanus*	prednisolone 0 · 125 mg k^{-1} plus cinchophen 25 mg kg^{-1} (one Predno-leucotropin tablet per 16 kg) p.o., b.i.d.
Tramadol	tramadol	Asiatic black bear *U. thibetanus*	4–6 mg kg^{-1} p.o., b.i.d.
Nutraceuticals	glucosamine/ chondroitin	All bear species	used in combination, preferred ratio of 500 mg glucosamine:400 mg chondroitin; dose according to the manufacturer's instructions and scale up to appropriate dose for body mass

Table 1. Bear mobility drug chart: b.i.d., twice a day; NSAID, non-steroidal anti-inflammatory drugs; p.o., *per os* **(by mouth); s.c., subcutaneous; s.i.d., once a day.**

Finally, euthanasia is an unpopular but necessary tool for the management of bears with mobility disease, whose welfare cannot be ensured. The bear's quality of life should always be the primary consideration when making this decision; pre-emptive euthanasia is preferable to collapse or to chronic pain caused by inadequate management.

Neoplasia

The exact cause or causes of cancer in bears are unknown. Several different neoplasias are recorded in bears (see Table 2) and their aetiologies are likely to be varied as in other species, with genetic and environmental factors, such as infection, inflammation, trauma, nutrition and toxins, all contributing.

In captive bears, neoplasia commonly involves the hepatobiliary and gastrointestinal systems (Ramsay, 2003; Bourne & Vila-Garcia, 2007). Hepatic neoplasia is particularly common in bears from bile farms: in one rescue centre 35% of Asiatic black bear deaths are caused by hepatic neoplasia and 38% of bear deaths are attributed to some form of neoplasia ($n = 247$) (Animals Asia Foundation, China Bear Rescue Centre, mortality records). Clinical cholecystitis as a result of bile extraction was present in all these bears before death and cholecystectomies had been performed because of the extensive biliary inflammation, infection and choleliths present. It is hypothesized that the long-term trauma induced in the biliary system by the bile extraction process predisposed these bears to the development of hepatocellular carcinoma.

In Sloth bears, high levels of dietary fat and low levels of vitamin A and selenium may

SPECIES	NEOPLASIA
Asiatic black bear	
Ursus thibetanus	hepatic adenocarcinoma
	nasal adenocarcinoma
	oesophageal squamous cell carcinoma
	lymphoma
	pancreatic adenoma
	lymphosarcoma
	biliary adenocarcinoma
	hepatocellular carcinoma
	pancreatic adenocarcinoma
Sloth bear	
Melursus ursinus	oral squamous cell carcinoma
	mesothelioma
	cementifying fibroma
	pancreatic adenocarcinoma
	biliary adenocarcinoma
	extrahepatic biliary carcinomas
	cholangiocellular carcinoma
	metastatic adenocarcinoma
	adenocarcinoma of the gall bladder
Andean bear	
Tremarctos ornatus	mesothelioma
	thymoma
	cardiac rhabdomyosarcoma
	carcinoma involving the lung and heart
	unspecified anterior mediastinal mass
	squamous cell carcinoma
	pyloric leiomyoma
	right testicular tumour
	mammary adenoma
	spindle cell thymoma
	cholangiosarcoma
	transitional cell carcinoma of the urinary bladder
American black bear	
Ursus americanus	carcinoma of the urinary bladder
	mammary neoplasia
	carcinoma of the tongue
	laryngeal squamous cell carcinoma
	gastric leiomyoma
	lipoma
	squamous cell carcinoma
Sun bear	
Helarctos malayanus	mandibular squamous cell carcinoma
	intestinal adenocarcinoma
	papillary cystadenocarcinoma
	pyloric leiomyoma
	squamous cell carcinoma of the left eye
	extrahepatic biliary carcinomas
	biliary adenocarcinoma
Brown bear	
Ursus arctos	skin fibroma
	adrenal gland adenocarcinoma
	kidney adenoma
	lymphoblastic lymphosarcoma
	mammary neoplasia
	malignant melanoma
	osteoblastoma-like osteosarcoma
	malignant melanoma of the hard palate

Table 2. Continued

SPECIES	NEOPLASIA
	intestinal adenocarcinoma
	disseminated abdominal adenocarcinoma
	intestinal lymphosarcoma
	lymphocytic lymphoma
	gastrointestinal argentaffin carcinoma
	intestinal lymphosarsocoma
	pancreatic carcinoma
	pancreatic parenchymal cell carcinoma
	tubopapillary mammary carcinoma
	biliary adenocarcinoma
	cholangiocellular carcinoma
Polar bear	
Ursus maritimus	small intestinal leiomyoma
	unipolar renal tubular adenoma
	skin fibroma
	pancreatic β-cell carcinoma
	pancreatic islet cell adenomas and carcinomas
	hepatocellular carcinoma
	seminoma of the testis
	spindle cell thymoma
	biliary adenocarcinoma
	hepatocellular carcinoma
	cholangiocellular carcinoma
	liver adenocarcinoma
	benign hepatoma
Panda	
Ailuropoda melanoleuca	cutaneous haemangioma
Unspecified	thyroid gland carcinoma
	pancreatic carcinoma
	adrenal carcinoma

Table 2. Recorded neoplasias in bears (Mauroo *et al*., 2006; Murray *et al*., 2006; Bourne & Vila-Garcia, 2007; Nak *et al*., 2008).

predispose to the development of hepatic neoplasia. In collections where old or mouldy bread or cereals are fed, the consumption of aflatoxins may be implicated in the development of cancer.

HAND-REARED CUBS

Cubs hand-reared from birth following maternal rejection are particularly susceptible to infection. Administration of serum (7 · 5–15 ml per 100 g body mass suggested, based on Domestic cat *Felis catus* data), ideally from the mother, can be given, subcutaneously to increase systemic IgG levels and orally to improve local gastro-intestinal tract immunity (Bourne & Vila-Garcia, 2007; Hedberg, 2007).

PATHOLOGY IN BILE-FARMED ASIATIC BEARS

Across Asia, bear bile is prized as a constituent of traditional Chinese medicine. Bear bile is marketed as a general tonic and cure-all. Its active ingredient ursodeoxycholic acid (UDCA) is commonly used in western medicine to treat cholestasis but, as purified UDCA is produced by a number of pharmaceutical companies and herbal alternatives are also available, significant doubt exists regarding the rationale for the maintenance of bear

farms producing unregulated, contaminated bile products. The species primarily used for bile extraction is the Asiatic black bear, although Sun bears and Brown bears *Ursus arctos* are also used in areas where wild populations are accessible.

A wide range of pathologies has been recorded in bears used for bile extraction that either have not been described in non-farmed bears or have been recorded with a much greater frequency in farmed bears than non-farmed bears. These include hernias (30%), cholecystitis (97%), gall bladder polyps (68%), gallstones (25%), abdominal abscesses (28%), peritonitis (6%) and hepatic/biliary neoplasia (35%), as well as dental disease (69%) ($n = 247$) (Animals Asia Foundation, China Bear Rescue Centre, mortality records).

The poor condition of bears rescued from bile farms, in conjunction with these multiple pathologies, provides a strong indication of the ethical and welfare issues surrounding bear bile farming. Additionally, the culture of organisms such as *E. coli*, *Enterococcus* spp and *Pseudomonas* spp from bear bile raises concern over the potential public health implications associated with bile consumption.

BEHAVIOURAL MEDICINE

Many bears demonstrate abnormal behaviours in captivity. Stereotypy is often considered to be a negative behavioural manifestation of mental stress. Stereotypy is defined as the maintenance of one attitude for a long period or repetition of meaningless behaviours or movements (Editors of The American Heritage® Dictionaries, 2007).

Bears affected by stereotypical behaviour for long time periods may become clinically ill. Common stereotypies observed in bears include pacing, headswaying, head rolling and paw sucking. Less common stereotypies include catatonia and regurgitation.

The aetiology of behavioural problems is often complex and multifactorial. The first step in addressing any behavioural problem is to ensure that there is no medical aetiology for the stereotypy. Headswaying has been observed in a bear later diagnosed with a nasal adenocarcinoma and reductions in stereotypical behaviour post-dentistry have also been seen.

A complete overview of the current husbandry practices and enrichment programmes should be performed, including the development of a behavioural ethogram based on observations over a period of time. This may indicate a potential trigger for stereotypy, such as food preparation or visitor viewing. Basic keeper rapport-building or formal training programmes under experienced guidance may divert the bear's focus and provide mental engagement; enrichment should be an evolving process. If comprehensive management techniques do not resolve the issue, further veterinary intervention may be required.

Fluoxetine, a selective serotonin re-uptake inhibitor, has been used in both Brown bears (at $0 \cdot 62\,\mathrm{mg\,kg^{-1}}$ orally daily: Yalcin & Aytug, 2007) and Polar bears (at $1 \cdot 0\,\mathrm{mg\,kg^{-1}}$ orally daily: Poulsen *et al*., 1996) to manage stereotypical pacing without adversely affecting typical behaviours. In the Brown bear, administration of the fluoxetine was discontinued after 6 months (2 weeks after pacing ceased) and environmental enrichment was continued, and pacing behaviour did not resume (1 year follow-up). Zuclopenthixol is a neuroleptic dopamine D1/D2 receptor antagonist and has been used successfully to reduce aggressive interactions in two group-housed ♂ Asiatic black bears (at a dose of $0 \cdot 3$–$0 \cdot 5\,\mathrm{mg\,kg^{-1}}$ orally once daily), based on evidence extrapolated from other species (Manzaneque & Navarro, 1999). Zuclopenthixol has also been used extensively in human patients to alleviate aggression associated with dementia or psychosis (Nygaard *et al*., 1994). No side effects from its use have been recorded in bears.

ANAESTHESIA

Anaesthesia of bears is integral to most veterinary or biological interventions, including translocations, research and medical management. This applies to captive or wild situations, which require different approaches to the anaesthesia of ursid patients.

There is surprisingly little written on the anaesthesia of bears. The little that exists focuses on wild bears, including Polar, Brown and American black bears, with fewer papers on the five remaining species. An excellent overview of bear anaesthesia can be found in Caulkett (2007), and readers requiring further information are recommended to consult this reference.

SPECIES	BODY-MASS RANGE	DOSAGES
Spectacled bear *Tremarctos ornatus*	♂ up to 140 kg ♀ 60 kg	• tiletamine–ketamine 2 · 8 mg kg^{-1} i.m. (Bush *et al*., 1980) • tiletamine–ketamine 3 · 2–11 · 1 mg kg^{-1} i.m. (Nielsen, 1999) • medetomidine 0 · 035–0 · 075 mg kg^{-1}, ketamine 2 · 5–4 mg kg^{-1} and midazolam 0 · 05–0 · 09 mg kg^{-1} i.m. (Black & Whiteside, 2005)
Asiatic black bear *Ursus thibetanus*	♂ 110–150 kg ♀ 65–90 kg	• tiletamine–zolazepam 4 · 4 mg kg^{-1} i.m. (Kreeger, 1999) • tiletamine–zolazepam 5 mg kg^{-1} (hibernating) i.m. (Asano *et al*., 2007) • tiletamine–zolazepam 7 mg kg^{-1} i.m. (Kojima *et al*., 2001) • tiletamine–zolazepam 9 mg kg^{-1} i.m. (Asano *et al*., 2007) • tiletamine–zolazepam 18 mg kg^{-1} (high-dose trial) i.m. (Asano *et al*., 2007) • medetomidine 0 · 01 mg kg^{-1} and tiletamine–zolazepam 0 · 5 mg kg^{-1} i.m. (Cracknell, 2007) • xylazine 0 · 6 mg kg^{-1} and ketamine 4 mg kg^{-1} i.m. (Dutta *et al*., 1999) • ketamine 10 mg kg^{-1} (Dutta *et al*., 1999)
American black bear *Ursus americanus*	♂ 115–270 kg ♀ 92–140 kg	• tiletamine–ketamine 4 · 7 mg kg^{-1} i.m. (Bush *et al*., 1980) • tiletamine–zolazepam 7 mg kg^{-1} i.m. (Ramsay, 2003) • medetomidine 0 · 04 mg kg^{-1} and ketamine 1 · 5 mg kg^{-1} (Ramsay, 2003) • medetomidine 0 · 035–0 · 075 mg kg^{-1}, ketamine 2 · 5–4 mg kg^{-1} and midazolam 0 · 05–0 · 09 mg kg^{-1} i.m. (Black & Whiteside, 2005) • xylazine 2–4 · 5 mg kg^{-1} and ketamine 4 · 5–9 mg kg^{-1} i.m. (Addison & Kolenosky, 1979) • butorphanol 0 · 26 mg kg^{-1} plus azaperone 0 · 22 mg kg^{-1} plus medetomidine 0 · 087 mg kg^{-1} (Wolfe *et al*., 2008) • carfentanil 6 · 8–18 · 8 µg kg^{-1} p.o. (Ramsay *et al*., 1995) • etorphine 0 · 02 mg kg^{-1} i.m. (Ramsay, 2003) • isoflurane, oxygen/nitrous oxide (epileptic bear) (Clutton, 1987)
Brown bear *Ursus arctos*	102–780 kg depending on range	• tiletamine–zolazepam 3 mg kg^{-1} i.m. (Ishikawa *et al*., 1998) • tiletamine–ketamine 3 · 5 mg kg^{-1} i.m. (Bush *et al*., 1980) • tiletamine–zolazepam 7–9 mg kg^{-1} (Ramsay, 2003) • medetomidine 0 · 06 mg kg^{-1} and tiletamine–zolazepam 2 mg kg^{-1} i.m. (Ramsay, 2003) • xylazine 1–11 mg kg^{-1} and ketamine 10–11 mg kg^{-1} i.m. (Ramsay, 2003) • premed midazolam 0 · 25 mg kg^{-1} i.m., induction ketamine 5 mg kg^{-1} i.m. (Ishikawa *et al*., 1998) • carfentanil 7 · 6 µg kg^{-1} p.o. (Winter) (Mortenson & Bechert, 2001) • carfentanil 8 µg kg^{-1} p.o. (Mama *et al*., 2000) • carfentanil 12 · 7 µg kg^{-1} p.o. (Summer) (Mortenson & Bechert, 2001) • carfentanil 0 · 012 mg kg^{-1} and xylazine 0 · 3 mg kg^{-1} i.m. (Ramsay, 2003) • etorphine 0 · 02–0 · 06 mg kg^{-1} i.m. (Ramsay, 2003)
Polar bear *Ursus maritimus*	♂ 300–800 kg ♀ 150–300 kg	• medetomidine 0 · 035–0 · 075 mg kg^{-1}, ketamine 2 · 5–4 mg kg^{-1} and midazolam 0 · 05–0 · 09 mg kg^{-1} i.m. (Black & Whiteside, 2005) • xylazine 1 · 9 mg kg^{-1} and tiletamine–zolazepam 2 · 9 mg kg^{-1} i.m. (Cattet, Caulkett & Stenhouse, 2003)
Malayan sun bear *Helarctos malayanus*	27–65 kg	• tiletamine–zolazepam 4 · 1 mg kg^{-1} i.m. (Bush *et al*., 1980) • tiletamine–zolazepam 4–5 · 5 mg kg^{-1} i.m. (Nielsen, 1999) • medetomidine 0 · 05 mg kg^{-1} and tiletamine–zolazepam 2 mg kg^{-1} i.m. (Onuma, 2003)
Sloth bear *Melursus ursinus*	55–145 kg	• medetomidine 0 · 035–0 · 075 mg kg^{-1}, ketamine 2 · 5–4 mg kg^{-1} and midazolam 0 · 05–0 · 09 mg kg^{-1} i.m. (Black & Whiteside, 2005) • xylazine 1 · 4–2 · 4 mg kg^{-1} and ketamine 5 · 8–9 · 8 mg kg^{-1} i.m. (Page, 1986) • premed atropine 0 · 025 mg kg^{-1}, induction xylazine 2 mg kg^{-1} and ketamine 5 mg kg^{-1} i.m. (Shanmugam *et al*., 2008)

Table 3. Continued

SPECIES	BODY-MASS RANGE	DOSAGES
Giant panda *Ailuropoda melanoleuca*	75–160 kg	• tiletamine–zolazepam 6–6 · 6 mg kg^{-1} i.m. (Ramsay, 2003) • xylazine 0 · 43 mg kg^{-1} and ketamine 5 mg kg^{-1} i.m. (Ramsay, 2003) • atropine premed 0 · 01 mg kg^{-1} i.m., xylazine 0 · 6 mg kg^{-1} and ketamine 6 mg kg^{-1} i.m. (Mauroo *et al*., 2006) • xylazine 0 · 35 mg kg^{-1} and ketamine 5 · 7 mg kg^{-1} i.m. (Janssen, Edwards *et al*., 2006) • ketamine 5 · 7 mg kg^{-1} i.m. (Janssen, Edwards *et al*., 2006) • ketamine 10 mg kg^{-1} i.m. (Hildebrandt *et al*., 2006) • diazepam 0 · 13 mg kg^{-1} and ketamine 5 · 7 mg kg^{-1} i.m. (Janssen, Edwards *et al*., 2006)

Table 3. Reported anaesthetic doses used for treating bear species in captivity: i.m., intramuscular; p.o., ***per os*** **(by mouth).**

As with any species, pre-anaesthetic assessment of a patient is essential. In a captive situation, and to some degree in the wild, history, age, sex, reproductive status (especially pregnant or lactating ♀♀ with cubs present), medical history and weight should all be considered. Weight is especially important and, in a captive situation, all efforts should be made to obtain accurate weights as part of the pre-anaesthesia workup. In the wild situation, estimates of weights must be used and often dosing schedules are based on experience or knowledge based on age, sex and size (Arnemo *et al*., 2001). The time of the year is also an important consideration when considering the anaesthetic agents to use and the doses to be used (Mortenson & Bechert, 2001). Physiological differences between the seasons influence changes in fat stores and metabolic state, all of which are independent of hibernation but impact on dose selection for induction as well as consideration for location of darting or jab sticking, and the needle length required.

Captive bears can be moved into a smaller cage for induction of anaesthesia. Several rehabilitation centres and zoological collections have made the use of honey as a distraction technique at darting. In some situations, target training can be used to facilitate calm inductions, and even allow pre-operative blood sampling (Janssen, Morris *et al*., 2006). Wild bears can be caught through either aerial captures or physical restraint with the use of culvert traps or snares. These different techniques can have a detrimental physiological impact on the patient that must be considered when choosing an appropriate capture method, including haemoconcentration, stress leukograms, muscle damage, lesions consistent with capture myopathy and potential fractures or other injury (Caulkett & Cattet, 1997; Cattet, Christison *et al*., 2003; Caulkett, 2007). Bears are monogastric and vomiting is not uncommon at induction; it is advisable to avoid anaesthetizing bears that have recently eaten (Caulkett & Cattet, 2002).

Selection of induction agents is primarily based on the suitability for the species. There is a considerable variation in the doses and species' responses to combinations of induction agents, and a summary of agents used for captive and wild bears can be seen in Tables 3 and 4, respectively. In general, increased doses tend to be used in the wild bears because of the need for quick and safe inductions, to counteract the stressful induction processes, and because of the risk of underestimating weights leading to failed inductions. Availability might also determine drug choice; for instance, xylazine–ketamine is readily available in India in contrast to tiletamine–zolazepam. Cost might also be a contributing factor, especially in the rehabilitation setting. Local traditions and the eating

SPECIES	BODY-MASS RANGE	DOSAGES
Spectacled bear *Tremarctos ornatus*	♂ up to 140 kg ♀ 60 kg	• not recorded
Asiatic black bear *Ursus thibetanus*	♂ 110–150 kg ♀ 65–90 kg	• tiletamine–zolazepam 4 · 4 mg kg^{-1} i.m. (Ramsay, 2003) • tiletamine–zolazepam 9 mg kg^{-1} i.m. (Okano *et al*., 2004; Asano *et al*., 2007)
American black bear *Ursus americanus*	♂ 115–270 kg ♀ 92–140 kg	• **tiletamine–zolazepam 7 mg kg^{-1}** (Kreeger, 1999) • tiletamine–zolazepam 7–9 mg kg^{-1} i.m. (Arnemo *et al*., 2001) • medetomidine 0 · 05 mg kg^{-1} and tiletamine 0 · 86 mg kg^{-1} and zolazepam 0 · 86 mg kg^{-1} i.m. (Caulkett & Cattet, 1997) • **medetomidine 0 · 04 mg kg^{-1} and ketamine 1 · 5 mg kg^{-1} i.m.** (Caulkett, 2007) • **xylazine 2 mg kg^{-1} and tiletamine–zolazepam 3 mg kg^{-1} i.m.** (Caulkett, 2007; Caulkett & Arnemo, 2007) • **xylazine 2 mg kg^{-1} and ketamine 4 · 4 mg kg^{-1} i.m.** (Kreeger, 1999) • xylazine 2–4 · 5 mg kg^{-1} and ketamine 4 · 5–9 mg kg^{-1} i.m. (Addison & Kolenosky, 1979) • butorphanol 0 · 26 mg kg^{-1} plus azaperone 0 · 22 mg kg^{-1} plus medetomidine 0 · 087 mg kg^{-1} (Wolfe *et al*., 2008)
Brown bear *Ursus arctos*	102–780 kg depending on range	• **tiletamine–zolazepam 7–9 mg kg^{-1} i.m.** (Arnemo *et al*., 2001) • tiletamine–zolazepam 8–10 mg kg^{-1} i.m. (Cattet, Christison *et al*., 2003) • **medetomidine 0 · 02–0 · 035 mg kg^{-1} and tiletamine–zolazepam 3 · 9–4 · 8 mg kg^{-1} i.m.** (Arnemo *et al*., 2001), dosing dependent on age and weight range • xylazine 2 mg kg^{-1} and tiletamine–zolazepam 3 mg kg^{-1} i.m. (Cattet, Christison *et al*., 2003) • **xylazine 2 · 5 mg kg^{-1} and tiletamine–zolazepam 3 · 8 mg kg^{-1} i.m.** (Caulkett, 2007) • xylazine 2 mg kg^{-1} and tiletamine–zolazepam 3 mg kg^{-1} i.m. (Caulkett & Arnemo, 2007) • etorphine 0 · 011–0 · 132 mg kg^{-1} i.m. (Herbert *et al*., 1980)
Polar bear *Ursus maritimus*	♂ 300–800 kg ♀ 150–300 kg	• tiletamine–zolazepam 3 · 8–12 · 2 mg kg^{-1} i.m. (Cattet, Caulkett, Polischuck & Ramsay, 1999) • tiletamine–zolazepam 5 mg kg^{-1} i.m. (Ramsay, 2003) • tiletamine–zolazepam 5 mg kg^{-1} (Haigh *et al*., 1984, 1985) • tiletamine–zolazepam 5–14 mg kg^{-1} i.m. (Semple *et al*., 2000) • **tiletamine–zolazepam 7–9 mg kg^{-1} i.m.** (Arnemo *et al*., 2001) • tiletamine–zolazepam 8–9 mg kg^{-1} i.m. (Stirling *et al*., 1989) • medetomidine 0 · 03–0 · 23 mg kg^{-1} and tiletamine–zolazepam 1 · 14–7 · 43 mg kg^{-1} (Cattet, Caulkett, Polischuck & Ramsay, 1999) • medetomidine 0 · 07 mg kg^{-1} and ketamine 3 mg kg^{-1} i.m. (Ramsay, 2003) • **medetomidine 0 · 07 mg kg^{-1} and tiletamine–zolazepam 2 · 3 mg kg^{-1} i.m.** (Cattet *et al*., 1997) • **medetomidine 0 · 075 mg kg^{-1} and tiletamine–zolazepam 2 · 2 mg kg^{-1} i.m.** (Caulkett *et al*., 1999) • medetomidine 0 · 08–0 · 35 mg kg^{-1} and ketamine 1 · 9–8 · 8 mg kg^{-1} i.m. (Cattet, Caulkett, Polischuck & Ramsay, 1999) • medetomidine 0 · 16 mg kg^{-1} and ketamine 4 mg kg^{-1} i.m. (Caulkett *et al*., 1999) • **xylazine 2 mg kg^{-1} and tiletamine–zolazepam 3 mg kg^{-1} i.m.** (Caulkett, 2007; Caulkett & Arnemo, 2007) • xylazine 2 · 2 mg kg^{-1} and tiletamine–zolazepam 3 · 3 mg kg^{-1} i.m. (Cattet, Caulkett & Lunn, 2003) • xylazine 6 · 8 mg kg^{-1} and ketamine 6 · 8 mg kg^{-1} i.m. (Lee *et al*., 1981)
Malayan sun bear *Helarctos malayanus*	27–65 kg	• not recorded

Table 4. Continued

SPECIES	BODY-MASS RANGE	DOSAGES
Sloth bear *Melursus ursinus*	55–145 kg	• tiletamine–zolazepam 6 mg kg^{-1} i.m. (Ramsay, 2003) • medetomidine 0·03 mg kg^{-1} and tiletamine–zolazepam 2 mg kg^{-1} i.m. (Cracknell, 2007) • medetomidine 0·05 mg kg^{-1} and ketamine 3 mg kg^{-1} (Cracknell, 2007) • xylazine 2 mg kg^{-1} and ketamine 7·5 mg kg^{-1} i.m. (Ramsay, 2003)
Giant panda *Ailuropoda melanoleuca*	75–160 kg	• not reported

Table 4. Reported anaesthetic doses used for treating bear species in the wild: bold font indicates recommended dosages used to facilitate capture of American black bears, Brown bears and Polar bears according to Caulkett (2007); i.m., intramuscular.

of bear meat may also impact on the timing of anaesthetics and the selection of induction agents (Semple *et al*., 2000). The author aims to ensure that the anaesthetic doses provided are correct but readers are advised to check with the current literature as anaesthetic techniques develop.

Human safety is paramount during any bear anaesthesia and care must be taken when approaching a bear to assess the depth of anaesthesia. The use of a broom handle or a similar device is advocated. Also of consideration is the location of any other wild bears or cubs. Cotton-wool earplugs and a blindfold are useful to reduce external stimulation. Suitable ophthalmic lubricants should be applied in all cases. When moving a bear, the anaesthetist should be well prepared with lifting equipment and the use of stretchers is preferred to that of net slings, which can result in acute hypertension, increased arousal and, potentially, hypoxaemia (Cattet, Caulkett, Streib *et al*., 1999).

Intubation is simple with the bear in ventral recumbency and the use of ropes to open the mouth while simultaneously lifting the head. Head torches or laryngoscopes assist in the process. Care should be taken when intubating lightly anaesthetized animals as they tend to snap their jaws, especially when the α-2-agonist–ketamine combinations or tiletamine–zolazepam are used. Endotracheal tube size is variable depending on the species but ranges from 7 to 18 mm external diameter. If intubation is not performed, then supplemental oxygen should be provided via a facemask or an intranasal tube.

Monitoring of bears is relatively straightforward and excellent reports on the different responses to various induction combinations have been provided (Caulkett, 2007). Muscle tone, cranial reflexes, head or limb movement, and respiratory rate are useful indicators of the depth of anaesthesia. Cardiovascular assessment is essential and peripheral pulses can be felt at the lingual or the brachial artery. Heart rates vary according to the induction agent and the species. Intravenous cannulation is straightforward in the cephalic, medial saphenous or jugular veins and provides access for administration of intravenous fluids, analgesics or 'top-up' anaesthetic agents. The author (J. M. C.) has monitored blood pressure using both invasive and non-invasive means. The same techniques used in domestic canids can be extrapolated but non-invasive blood pressure cuffs need to be of a suitable size; often, utilizing human equipment is sufficient. Invasive monitoring requires percutaneous cannulation of an artery, typically the medial metatarsal artery, and attachment to a transducer or a manometer. Normal blood pressures vary depending on the anaesthetic regimens used. Pulse oximeters are a useful monitoring device in the field, and blood-gas data have been collected for a number of species

(Bush *et al*., 1980; Cattet, Caulkett, Streib *et al*., 1999). Capnography provides information on respiration and tissue metabolism as well as cardiovascular function, and can be easily applied to an endotracheal tube or directly into the nares of non-intubated bears. Body-temperature monitoring is vital in the larger species as hyperthermia can occur, especially with α-2-agonist-tiletamine–zolazepam combinations (Caulkett, 2007). Recovery should be monitored and antagonists should always be used if available, especially in wild patients. The use of anaesthetic records should be encouraged and all efforts should be made to advance and improve the techniques used in ursid anaesthesia.

TRAINING

The use of positive reinforcement training can enable many non- and minimally painful veterinary procedures, such as inspection of the teeth and feet, radiography, ultrasonography, cleaning wounds, injections and blood sampling, without the need for anaesthesia or physical restraint (Martínez, 2006; Perry & Stephens, 2006; Bourne & Vila-Garcia, 2007).

Bears are easily trained to take medications. A reliable method is to blend the medication with fruit and water ± jam and offer in a plastic jug from which the bear laps. Foul-tasting medication (e.g. tramadol, metronidazole, ivermectin) can be stuffed into the centre of marshmallows that are then smeared in honey. Bears swallow these without chewing as long as the marshmallows are offered in quick succession.

CONCLUSION

Bear medicine has developed considerably over the last 20 years, and as population management and conservation technologies develop, the knowledge of *in situ* and captive ursid health care will improve. The species differences and specialities within the Ursidae provide a variety of challenges for the interested clinician.

REFERENCES

Abdelrasoul, K. & Fowler, M. (1979): Epidemiology of ascarid infection in captive carnivores. *Proceedings of the Annual Meeting of the American Association of Zoo Veterinarians* **1979:** 105–106.

Addison, E. M. & Kolenosky, G. B. (1979): Use of ketamine hydrochloride and xylazine hydrochloride to immobilize black bears (*Ursus americanus*). *Journal of Wildlife Diseases* **15:** 253–258.

Arnemo, J. M., Brunberg, S., Ahlqvist, P., Franzen, R., Friebe, A., Segerstrom, P., Soderberg, A. & Swenson, J. E. (2001): Reversible immobilization and anesthesia of free-ranging brown bears (*Ursus arctos*) with medetomidine–tiletamine–zolazepam and atipamezole: a review of 575 captures. In *Proceedings of AAZV, AAWV, NAZWV Joint Conference, Orlando, FL*: 234–236. Kirk Baer, C. & Wilmette, M. W. (Eds). Yulee, FL: American Association of Zoo Veterinarians.

Asano, M., Tsubota, T., Komatsu, T., Katayama, A., Okano, T. & Nakamura, S. (2007): Immobilization of Japanese black bears (*Ursus thibetanus japonicus*) with tiletamine hydrochloride and zolazepam hydrochloride. *Journal of Veterinary Medical Science* **69:** 433–435.

Banks, M., Monsalve Torraca, L. S., Greenwood, A. G. & Taylor, D. C. (1999): Aujeszky's disease in captive Bears. *The Veterinary Record* **145:** 362–365.

Barnes, D. M. & Rogers, L. L. (1980): Clostridial myonecrosis in a black bear associated with drug administration. *Journal of Wildlife Diseases* **16:** 315–317.

Black, S. & Whiteside, D. P. (2005): Immobilization of captive sloth bear (*Melursus ursinus*), spectacled bear (*Tremarctos ornatus*), black bear (*Ursus americanus*), and polar bear (*Ursus maritimus*) with a medetomidine, ketamine, and midazolam combination. In *Proceedings of AAZV, AAWV, AZA/NAG Joint Conference, 14–21 October, Omaha, NE, USA*: 104–105. Kirk Baer, C. (Ed.). Yulee, FL: American Association of Zoo Veterinarians.

Bourne, D. C. & Vila-Garcia, G. (2007): *Wildpro® Bears: health and management*. London: Wildlife Information Network. Available at http://www.wildlifeinformation.org

Bronson, E., Deem, S. L., Sanchez, C. & Murray, S. (2007): Serological response to a canarypox-vectored canine distemper virus vaccine in the giant panda (*Ailuropoda melanoleuca*). *Journal of Zoo and Wildlife Medicine* **38:** 363–366.

Bush, M., Custer, R. S. & Smith, E. E. (1980): Use of dissociative anesthetics for the immobilization of captive bears: blood gas, hematology, and biochemistry values. *Journal of Wildlife Diseases* **16:** 481–489.

Cattet, M. R. L., Caulkett, N. A., Polischuk, S. C. & Ramsay, M. A. (1997): Reversible immobilization of free-ranging polar bears with medetomidine–zolazepam–tiletamine and atipamezole. *Journal of Zoo and Wildlife Medicine* **33:** 611–617.

Cattet, M. R. L., Caulkett, N. A., Polischuk, S. C. & Ramsay, M. A. (1999): Anesthesia of polar bears (*Ursus maritimus*) with zolazepam–tiletamine, medetomidine–ketamine, and medetomidine–zolazepam–tiletamine. *Journal of Zoo and Wildlife Medicine* **30:** 354–360.

Cattet, M. R. L., Caulkett, N. A., Streib, K. A., Torske, K. E. & Ramsay, M. A. (1999): Cardiopulmonary response of anesthetized polar bears to suspension by net and sling. *Journal of Zoo and Wildlife Medicine* **35:** 548–556.

Cattet, M. R. L., Caulkett, N. A. & Lunn, N. J. (2003): Anesthesia of polar bears using xylazine–zolazepam–tiletamine or zolazepam–tiletamine. *Journal of Wildlife Diseases* **39:** 655–664.

Cattet, M. R. L., Christison, K., Caulkett, N. A. & Stenhouse, G. B. (2003): Physiological responses of grizzly bears to different methods of capture. *Journal of Wildlife Diseases* **39:** 649–654.

Caulkett, N. A. (2007): Bears. In *Zoo animal and wildlife immobilization and anesthesia*: 409–415. West, G., Heard, D. & Caulkett, N. A. (Eds). Ames, IA: Blackwell Publishing.

Caulkett, N. A. & Arnemo, J. M. (2007): Chemical immobilization of free-ranging terrestrial mammals. In *Lumb and Jones' veterinary anesthesia and analgesia* (4th edn): 807–831. Tranquilli, W. J., Thurmon, J. C. & Grimm, K. A. (Eds). Ames, IA: Blackwell Publishing Professional.

Caulkett, N. A. & Cattet, M. R. L. (1997): Physiological effects of medetomidine–zolazepam–tiletamine immobilization in black bears. *Journal of Wildlife Diseases* **33:** 618–622.

Caulkett, N. A. & Cattet, M. R. L. (2002): *Anesthesia of bears*. Ithaca, NY: International Veterinary Information Service. Available at http://www.enr.gov.nt.ca/_live/documents/documentManagerUpload/anesthesia_of_bears.pdf

Caulkett, N. A., Cattet, M. R. L., Caulkett, J. M. & Polischuk, S. C. (1999): Comparative physiological effects of telazol, medetomidine–ketamine, and medetomidine–telazol in captive polar bears (*Ursus maritimus*). *Journal of Zoo and Wildlife Medicine* **30:** 504–509.

Clutton, R. E. (1987): Anesthesia in an epileptic black bear (*Ursus americanus*): a case report and discussion of a dilemma. *Journal of Zoo Animal Medicine* **18:** 66–69.

Collins, J. E., Leslie, P., Johnson, D., Nelson, D., Peden, W., Boswell, R. & Draayer, H. (1984): Epizootic of adenovirus infection in American black bears. *Journal of the American Veterinary Medical Association* **185:** 1430–1432.

Convy, J. A. (2002): Rehabilitation of black bear cubs. *Wildlife Rehabilitation Bulletin* **20:** 16–43.

Cracknell, J. M. (2007): *ZSL anaesthetic handbook*. London, UK: Zoological Society of London Internal Publication.

DeBowes, L. J., Mosier, D., Logan, E., Harvey, C. E., Lowry, S. & Richardson, D. C. (1996): Association of periodontal disease and histologic lesions in multiple organs from 45 dogs. *Journal of Veterinary Dentistry* **13:** 57–60.

Deem, S. L., Spelman, L. H., Yates, R. A. & Montali, R. J. (2000): Canine distemper in terrestrial carnivores: a review. *Journal of the American Veterinary Medical Association* **31:** 441–451.

Dollinger, P., Baumgartner, R., Pagan, O. & Weschler, B. (1996): Husbandry and pathology of polar bears (*Thalarctos maritimus*) in Swiss zoos. In *European Association of Zoo and Wildlife Veterinarians first scientific meeting, Rostock, Germany, May 16–18, 1996*: 47–54. Zwart, P., Dollinger, P. & Pagan, O. (Eds). Switzerland: European Association of Zoo and Wildlife Veterinarians.

Dutta, B., Kalita, D. & Lahon, D. K. (1999): Xylazine and ketamine anaesthesia in two black bears. *Indian Veterinary Journal* **76:** 553–555.

Dutton, C. J., Quinnell, M., Lindsay, R., DeLay, J. & Barker, I. K. (2009): Paraparesis in a polar bear (*Ursus maritimus*) associated with West Nile virus infection. *Journal of Zoo and Wildlife Medicine* **40:** 568–571.

Editors of The American Heritage® Dictionaries (2007): *The American Heritage® medical dictionary*. Boston, MA: Houghton Mifflin Harcourt Publishing Company.

Farrell, R. K., Leader, R. W. & Johnston, S. D. (1973): Differentiation of salmon poisoning disease and Elokomin fluke fever: studies with the black bear (*Ursus americanus*). *American Journal of Veterinary Research* **34:** 919–922.

Föllmi, J. (2005): *Symptoms, radiographic examinations and pathologies: development of a scoring system to evaluate physical condition and quality of life in geriatric zoo mammals*. Veterinary Medicine Inaugural Dissertation, University of Berne, Switzerland. Available at http://www.ths.vetsuisse.unibe.ch/lenya/housing/live/publications/Diss_Foellmi.pdf (accessed April 2009).

Foster, G. W., Cames, T. A. & Forrester, D. J. (1998): Geographical distribution of *Demodex ursi* in black bears from Florida. *Journal of Wildlife Diseases* **34:** 161–164.

Fowler, M. E. (1986): Carnivores (Carnivora): Ursidae. In *Zoo & wild animal medicine* (2nd edn): 811–816. Fowler, M. E. (Ed.). Philadelphia, PA: W. B. Saunders & Co.

Gai, J. (2007): Salmon poisoning disease – not just a problem in the Pacific northwest. In *BIERZS – bear information exchange for rehabilitators, zoos & sanctuaries, 24–26 August 2007, Pomona, CA, USA*: 26. Los Angeles, CA: Bear Information Exchange for Rehabilitators, Zoos & Sanctuaries.

Gai, J. & Marks, S. L. (2005): Salmon poisoning disease in two Malayan sun bears (*Helarctos malayanus*). In *Proceedings of AAZV, AAWV, AZA/NAG Joint Conference, 14–21 October, Omaha, NE, USA*: 175–176. Kirk Baer, C. (Ed.). Yulee, FL: American Association of Zoo Veterinarians.

Greenwood, A. G. (1992): Veterinary considerations. In *Management guidelines for bears and raccoons*: 153–169. Partridge, J. (Ed.). Bristol: The Association of British Wild Animal Keepers.

Groves, M. G. (1969): Griseofulvin treatment of *Microsporum canis* infection in Malayan sun bears (*Helarctos malayanus*). *Journal of the American Veterinary Medical Association* **155:** 1090–1092.

HAIGH, J. C., STIRLING, I. & BROUGHTON, E. (1984): Clinical experiences with telazol for polar bear (*Ursus maritimus* Phipps) immobilization. *Proceedings of the American Association of Zoo Veterinarians* **1984:** 130–131.

HAIGH, J. C., STIRLING, I. & BROUGHTON, E. (1985): Immobilization of polar bears (*Ursus maritimus* Phipps) with a mixture of tiletamine hydrochloride and zolazepam hydrochloride. *Journal of Wildlife Diseases* **21:** 43–47.

HEDBERG, G. (2007): Infant hand rearing ursid serum IgG supplementation protocol. In *BIERZS – bear information exchange for rehabilitators, zoos & sanctuaries, 24–26 August 2007, Pomona, California, USA*: 24. Los Angeles, CA: Bear Information Exchange for Rehabilitators, Zoos & Sanctuaries.

HERBERT, D. M., LAY, D. W. & TURNBULL, W. G. (1980): Immobilization of coastal grizzly bears with etorphine hydrochloride. *Journal of Wildlife Diseases* **16:** 339–342.

HILDEBRANDT, T. B., BROWN, J. L., GORITZ, F., OCHS, A., MORRIS, P. & SUTHERLAND-SMITH, M. (2006): Ultrasonography to assess and enhance health and reproduction in the giant panda. In *Giant pandas: biology, veterinary medicine, and management*: 410–439. Wildt, D. E., Zhang, A., Zhang, H., Janssen, D. L. & Ellis, S. (Eds). Cambridge: Cambridge University Press.

ISHIKAWA, A., MATSUI, M., TSURUGA, H., SAKAMOTO, H., TAKAHASHI, Y. & KANAGAWA, H. (1998): Electroejaculation and semen characteristics of the captive Hokkaido brown bear (*Ursus arctos yesoensis*). *Journal of Veterinary Medical Science* **60:** 965–968.

JANSSEN, D. L., EDWARDS, M. S., SUTHERLAND-SMITH, M., YU, J., ZHANG, G., WEI, R., ZHANG, C. L., MILLER, R. E., PHILLIPS, L. G., HU, D. & TANG, C. (2006): Significant medical issues and biological reference values for giant pandas from the biomedical survey. In *Giant pandas: biology, veterinary medicine, and management*: 59–86. Wildt, D. E., Zhang, A., Zhang, H., Janssen, D. L. & Ellis, S. (Eds). Cambridge: Cambridge University Press.

JANSSEN, D. L., MORRIS, P., SUTHERLAND-SMITH, M., GREENBERG, M., LI, D., MAUROO, N. F. & SPELMAN, L. (2006): Medical management of captive adult and geriatric giant pandas. In *Giant pandas: biology, veterinary medicine, and management*: 353–376. Wildt, D. E., Zhang, A., Zhang, H., Janssen, D. L. & Ellis, S. (Eds). Cambridge: Cambridge University Press.

KITCHENER, A. C. (2004): The problems of old bears in zoos. *International Zoo News* **51:** 282–293.

KIUPEL, H., RISCHER, D. & FRICKE, G. (1987): Toxoplasmose bei jungen Kodiakbaren (*Ursus arctos middendorffi*). In *Internationalen Symposium uber die Erkrankungen der Zootiere vom 20. Mai bis 24. Mai 1987, Cardiff. Verhandlungsbericht des 29*: 335–339. Ippen, R. & Schroder, H.-D. (Eds). Berlin: Akademie-Verlag.

KOJIMA, E., TSURUGA, H., KOMATSU, T., MURASE, T., TSUBOTA, T. & KITA, I. (2001): Characterization of semen collected from beagles and captive Japanese black bears (*Ursus thibetanus japonicus*). *Theriogenology* **55:** 717–731.

KOLMSTETTER, C., MUNSON, L. & RAMSAY, E. C. (2000): Degenerative spinal disease in large felids. *Journal of Zoo and Wildlife Medicine* **31:** 15–19.

KREEGER, T. J. (1999): *Handbook of wildlife chemical immobilization*. Fort Collins, CO: Wildlife Pharmaceuticals Inc.

KRITSEPI, M., RALLIS, T., PSYCHAS, V., TONTIS, D. & LEONTIDES, S. (1996): Hepatitis in a European brown bear with canine infectious hepatitis-like lesions. *The Veterinary Record* **139:** 600–601.

LASCELLES, B. D. X. (2007): Beyond NSAIDs for canine osteoarthritis patients: multimodal drug therapy. In *BVOA: spring scientific meeting: 11th April 2007*: 16–26. Moores, A. (Ed.). Grantham: British Veterinary Orthopaedic Association.

LAW, G. & REID, A. (2010): Enriching the lives of bears in zoos. *International Zoo Yearbook* **44:** 65–74.

LEE, J., SCHWEINSBURG, R., KERNAN, F. & HAIGH, J. C. (1981): Immobilization of polar bears (*Ursus maritimus* Phipps) with ketamine hydrochloride and xylazine hydrochloride. *Journal of Wildlife Diseases* **17:** 331–336.

MAINKA, S. A. (1999): Giant panda management and medicine in China. In *Zoo & wild animal medicine: current therapy 4*: 410–414. Fowler, M. E. & Miller, R. E. (Eds). Philadelphia, PA: W. B. Saunders Company.

MAMA, K. R., STEFFEY, E. P. & WITHROW, S. J. (2000): Use of orally administered carfentanil prior to isoflurane-induced anesthesia in a Kodiak brown bear. *Journal of American Veterinary Medical Association* **217:** 546–549.

MANZANEQUE, J. M. & NAVARRO, J. F. (1999): An ethopharmacological assessment of the effects of zuclopenthixol on agonistic interactions in male mice. *Methods and Findings in Experimental and Clinical Pharmacology* **21:** 11–15.

MARTÍNEZ, G. (2006): Bear training: a tool that improves care. *The Shape of Enrichment* **15**(2): 9–10.

MAUROO, N. F., ROURKE, N. L., CHAN, W. K. & CHIR, B. (2006): Cutaneous hemangioma in a giant panda (*Ailuropoda melanoleuca*). *Journal of Zoo and Wildlife Medicine* **37:** 59–60.

MEYERSON, R. (2007): *Standardized animal care guidelines for polar bears (Ursus maritimus)*. Silver Spring, MD: American Zoo and Aquarium Association.

MORAN, J. F., FELIU, C. & GOMEZ, M. S. (1994): *Baylisascaris transfuga* (Rudolphy, 1819) (Nematoda, Ascarididae); a common parasite of bears. Ecological aspects and treatment. In *Proceedings from the international conference of aspects of bear conservation, Bursa, Turkey, May 31 to June 5, 1994*: 121–124. Dorrestein, G. M. & Kahraman, M. M. (Eds). Utrecht: Utrecht University.

MORTENSON, J. & BECHERT, U. (2001): Carfentanil citrate used as an oral anesthetic agent for brown bears (*Ursus arctos*). *Journal of Zoo and Wildlife Medicine* **32:** 217–221.

MURRAY, S., SANCHEZ, C. D., SIEMERING, G. H., ENQVIST, K. & DEEM, S. L. (2006): Transitional cell carcinoma of the urinary bladder in a spectacled bear (*Tremarctos ornatus*). *The Veterinary Record* **158:** 306–307.

Nak, D., Cangul, I. T., Nak, Y., Cihan, H. & Celimli, N. (2008): Tubulopapillary mammary carcinoma in a brown bear (*Ursus arctos*). *Journal of Wildlife Diseases* **44:** 505–508.

Newman, M. S., Cook, R. W., Appelhof, W. K. & Kitchen, H. (1975): Dermatophilosis in two polar bears. *Journal of the American Veterinary Medical Association* **167:** 561–564.

Nielsen, L. (1999): Species-specific recommendations. In *Chemical immobilization of wild and exotic animals*: 227–281. Nielsen, L. (Ed.). Ames, IA: Iowa State University Press.

Nunn, C. L., Rothschild, B. & Gittleman, J. L. (2007): Why are some species more commonly afflicted by arthritis than others? A comparative study of spondyloarthropathy in primates and carnivores. *Journal of Evolutionary Biology* **20:** 460–470.

Nygaard, H. A., Bakke, K., Brudvik, E., Elgen, K. & Lien, G. K. (1994): Dosing of neuroleptics in elderly demented patients with aggressive and agitated behaviour: a double-blind study with zuclopenthixol. *Current Medical Research and Opinion* **13:** 222–232.

Okano, T., Murase, T. & Tsubota, T. (2004): Electroejaculation and semen cryopreservation of free-ranging Japanese black bears (*Ursus thibetanus japonicus*). *Theriogenology* **66:** 1371–1376.

Onuma, M. (2003): Immobilization of sun bears (*Helarctos malayanus*) with medetomidine–zolazepam–tiletamine. *Journal of Zoo and Wildlife Medicine* **34:** 202–205.

Page, C. D. (1986): Sloth bear immobilization with a ketamine–xylazine combination: reversal with yohimbine. *Journal of the American Veterinary Medical Association* **189:** 1050–1051.

Perry, L. & Stephens, L. (2006): Positive reinforcement training for biomedical and reproductive management of giant pandas. *Animal Keepers' Forum* **34:** 394–401.

Poulsen, E. M., Honeyman, V., Valentine, P. A. & Teskey, G. C. (1996): Use of fluoxetine for the treatment of stereotypical pacing behavior in a captive polar bear. *Journal of the American Veterinary Medical Association* **209:** 1470–1474.

Pursell, A. R., Stuart, B. P., Styer, E. & Case, J. L. (1983): Isolation of an adenovirus from black bear cubs. *Journal of Wildlife Diseases* **19:** 269–271.

Ramsay, E. C. (2003): Ursidae and Hyenidae. In *Zoo & wild animal medicine: current therapy* **6:** 523–538. Fowler, M. E. & Miller, R. E. (Eds). St Louis, MO: W. B. Saunders Company.

Ramsay, M. A., Sleeman, J. M. & Clyde, V. (1995): Immobilization of black bears (*Ursus americanus*) with orally administered carfentanil citrate. *Journal of Wildlife Diseases* **31:** 391–393.

Rao, M. R. K., Char, N. L. & Ramanathan, S. (1988): Clostridial infection in a brown bear (*Ursus arctos*). *Indian Veterinary Journal* **65:** 167–168.

Reed, H. (2006): Salmonid poisoning and tapeworms. In *Polar bear nutrition guidelines*: 58–60. Lintzenich, B. A., Ward, A. M., Edwards, M. S., Griffin, M. E. & Robbins, C. T. (Eds). Sebastopol, CA: Polar Bears International, in association with the AZA Bear TAG.

Rogers, L. L. & Rogers, M. (1976): Parasites of bears: a review. In *Bears, their biology and management. Third international conference on bear research and management, Binghamton, NY, USA, and Moscow, USSR, June 1974. IUCN Publications New Series* No. 40: 411–430. Pelton, M. R., Lentfer, J. W. & Folk, G. E. (Eds). Morges: IUCN.

Salvelli, A. & Zanin, E. (1996): Pseudorabies in a group of brown bears (*Ursus arctos*) in captivity. In *European Association of Zoo and Wildlife Veterinarians first scientific meeting, Rostock, Germany, May 16–18, 1996*: 181–184. Zwart, P., Dollinger, P. & Pagan, O. (Eds). Switzerland: European Association of Zoo and Wildlife Veterinarians.

Schönbauer, M., Kölbl, S. & Schönbauer-Längle, A. (1984): Perinatale staupeinfektion bein drei eisbaren (*Ursus maritimus*) und bei einem Brillenbaren (*Tremarctos ornatus*). In *Internationalen Symposiums uber die Erkrankungen der Zootiere vom 2. Mai bis 6. Mai 1984, Brno. Verhandlungsbericht des 26*: 131–136. Ippen, R. & Schroder, H.-D. (Eds). Berlin: Akademie-Verlag.

Schultze, A. E., Maes, R. K. & Taylor, D. C. (1986): Pseudorabies and volvulus in a black bear. *Journal of the American Veterinary Medical Association* **189:** 1165–1166.

Semple, H. A., Gorecki, D. K. J., Farley, S. D. & Ramsay, M. A. (2000): Pharmacokinetics and tissue residues of telazol in free-ranging polar bears. *Journal of Wildlife Diseases* **36:** 653–662.

Shanmugam, A. A., Kumar, J. K., Selvaraj, I. & Selvaraj, V. (2008): Hematology of sloth bears (*Melursus ursinus ursinus*) from two locations in India. *Journal of Wildlife Diseases* **44:** 509–518.

Stirling, I., Spencer, C. & Andriashek, D. (1989): Immobilization of polar bears (*Ursus maritimus*) with telazol in the Canadian Arctic. *Journal of Wildlife Diseases* **25:** 159–168.

Witz, M., Lepage, O. M., Lambert, C., Couillerot, D., Fau, D., Rachail, M. & Troncy, E. (2001): Brown bear (*Ursus arctos arctos*) femoral head and neck excision. *Journal of Zoo and Wildlife Medicine* **32:** 494–499.

Wolfe, L. L., Goshorn, C. T. & Baruch-Mordo, S. (2008): Immobilization of black bears (*Ursus americanus*) with a combination of butorphanol, azaperone, and medetomidine. *Journal of Wildlife Diseases* **44:** 748–752.

Wolff, P. (1989): Selected medical aspects of the spectacled bear *Tremarctos ornatus*. In *Proceedings of the first international symposium on the spectacled bear, October 14–15, 1988*: 313–317. Rosenthal, M. (Ed.). Chicago, IL: Lincoln Park Zoo.

Yalcin, E. & Aytug, N. (2007): Use of fluoxetine to treat stereotypical pacing behavior in a brown bear (*Ursus arctos*). *Journal of Veterinary Behavior: Clinical Applications and Research* **2:** 73–76.

Zhang, J.-S., Daszak, P., Huang, H.-L., Yang, G.-Y., Kilpatrick, A. M. & Zhang, S. (2008): Parasite threat to panda conservation. *EcoHealth* **5:** 6–9.

Manuscript submitted 12 January 2009; revised 1 May 2009; accepted 14 October 2009