# Megabacteriosis in Psittacine Birds

**Willa Harrison**[1]

**Summary**

*Megabacterium* spp is a pleomorphic, Gram-positive, rod-shaped bacterium. It is very large, almost fungal in appearance and is found in the gastrointestinal tract, mainly the proventriculus, of a number of bird species. *Megabacterium* spp is believed to cause proventriculitis and an associated chronic progressive weight loss syndrome, commonly known in budgerigars as "going light." The organism is of increasing importance in Australia, being identified as causing clinical disease in a number of bird species. Acute outbreaks of the disease in aviary situations allow no time for veterinary treatment, can result in large numbers dying, but both forms of the disease, acute and chronic, can be a cause of significant economic and genetic loss. Once a diagnosis has been achieved, treatment can be carried out but effective dose rates are still being clarified. As the prevalence is increasing, diagnosis of megabacteriosis is becoming a task for all veterinarians, not just the specialist, so I hope the following article will aid in the general understanding of the disease and it's pathological significance and help with its successful diagnosis and treatment.

## *Megabacterium* Spp - the Organism

*Megabacterium* spp is a large, Gram- positive, periodic acid-Schiff positive, often irregular, rod-shaped bacterium. When incubated on blood agar, the bacteria were found to be haemolytic, facultatively anaerobic and capnophilic (Scanlan and Graham, 1990). The bacteria were also noted to be highly pleomorphic (Baker, 1992; Scanlan and Graham, 1990; Perry, 1993) and when subcultured onto agar, showed marked changes in diameter and length (Scanlan and Graham, 1990).

Although the bacterium is more fungal in size and shape and responds to Amphotericin B treatment (an antifungal agent), not antibiotics (Filippich and Perry, 1993; Perry, 1993; Perry, 1995), it has been shown by Henderson, Gulland and Hawkey (1988), to have the electron microscopic characteristics of a bacterium. An extensive

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intracellular network has recently been identified using ultrastructural studies but it is not considered suggestive of a eukaryotic organism, as no DNA could be seen in the membrane-bound organelles (Bredhauer, 1996). In order to positively categorise the organism (ie. as a bacterium or fungus), Bredhauer (1996), attempted to study the organism's genetic code via molecular biological techniques but these were hindered due to the thickness of the bacterium's wall causing difficulty with cell lysis and release of DNA. When mechanical lysis of the cell was performed, the subsequently released DNA failed to amplify, perhaps damaged by the lysis technique (Bredhauer, 1996). *Megabacterium* spp has frequently been identified in association with the alimentary tracts of aviary and pet birds (Baker, 1992; Henderson, Gulland and Hawkey, 1988; Perry, 1993; Perry, 1995; Scanlan and Graham, 1990; Simpson, 1992). The majority is found in the proventriculus and the proventricular/gizzard junction (Perry, 1993; Henderson, Gulland and Hawkey, 1988), but it is also seen in the intestine and faeces and occasionally the crop (Perry, 1993; Perry, 1995).

The British exhibition/show budgerigar has most commonly been reported to be infected with *Megabacterium* spp, with the numbers increasing (Baker, 1992; Filippich, 1992). More recently in Australia there also appears to be a growing number of budgerigars (and other pet and wild bird species) infected with the bacterium (Filippich, O'Boyle, Webb and Fuerst, 1993; Perry, 1993). This increase in the bacterium's prevalence is possibly due to the large numbers of English budgerigars and other bird species being imported, and (as yet) the inability of quarantine to identify and treat infected birds (Perry, 1993; Perry, 1995).

**Commensal Or Pathogen?**

*Megabacterium* spp is reported to be commonly seen in the proventriculus of clinically normal budgerigars (Scanlan and Graham, 1990; Filippich, 1992; Perry, 1993), where the bacterium exhibits an apparently intimate association with the glandular epithelial cells of the proventricular mucosa. This has led Scanlan and Graham (1990), to suggest that *Megabacterium* spp is a component of the normal flora of the budgerigars upper gastro-intestinal tract, and that a commensal or mutually beneficial symbiotic relationship may exist between the bacterium and the budgerigar. The suggestion of a mutually symbiotic relationship is not strongly supported (Perry, 1993; Simpson, 1992), but it is widely agreed that although many Megabacterium-infected birds show clinical signs (Baker, 1992; Filippich, 1992; Filippich and Perry, 1993; Perry, 1993; Simpson, 1992) there is a proportion of birds, which, though infected, are clinically normal (Baker, 1992; Filippich, 1992; Filippich and Perry, 1993; Perry, 1993; Perry, 1995; Simpson, 1992).

> "While the majority of cases which I have seen have shown the clinical signs and pathology described by Dr Baker, I have also seen cases where the organism was present and yet there was little or no pathology

(Simpson, 1992)".

There appears to be a carrier state, where the bacterium is carried in the bird's gastro-intestinal tract, mainly the proventriculus, for long periods of time without the bird exhibiting any clinical signs (Baker, 1992; Perry, 1993; Scanlan and Graham, 1990 ), needing only a stress or predisposing factor to initiate the associated disease (Perry, 1993; Perry, 1995). Although there is general support that an initiating factor is involved in the development of clinical disease, it has been reported that:

> "Virology studies, to date, on proventricular tissue or blood from birds with proventricular disease do not support the involvement of avian poxvirus, herpes virus, Newcastle disease virus or reticuloendotheliosis virus in the aetiology of proventricular disease (Filippich, 1992).

There are other situations were the associated disease and clinical signs of an infection with *Megabacterium* spp are seen soon after the bird has become infected. The bird may then recover from the clinical disease but remain infected with the bacterium, hence becoming a carrier (Perry, 1993; Filippich, 1992). During drug trials on budgerigars with megabacteriosis, it was found that birds which were 1+ and 2+ faecal megabacterium-positive were clinically normal and at necropsy, the proventriculus was only slightly affected or normal. On the other hand, the 3+ positive birds always showed moderate to severe proventriculitis. The faecal megabacterium status (1+, 2+ or 3+) correlated well with the megabacterium numbers seen at necropsy, that is light, moderate and heavy burdens (Filippich and Perry, 1993).

**Transmission**

The mode of transmission of *Megabacterium* spp is currently unknown but in a series of experiments reported by Filippich (1992) it was found that after 14 months of housing a pair of faecal megabacterium-positive budgerigars with a pair of faecal negative budgerigars, the faecal negative birds remained consistently negative. In breeding trials which took place in a *Megabacterium*-positive breeding house, 29% of chicks hatched and raised to faecal *Megabacterium*-negative parents, were faecal *Megabacterium*-positive. Forty six per cent of chicks hatched and raised by faecal *Megabacterium*-negative fosters but originating from eggs of faecal *Megabacterium*-positive parents, were faecal *Megabacterium*-positive and in 6 nests where faecal *Megabacterium*-negative fosters raised their own chick with an introduced *Megabacterium*-positive chick or egg, the foster chick tested faecal *Megabacterium*-negative (Filippich, 1992).

**Clinical Disease**

*Megabacterium* spp is reported to cause proventriculitis and associated signs like "going light" in budgerigars (Baker, 1992; Henderson et al, 1988) and other psittacine birds, such as grass parakeets, cockatiels, canaries, cockatoos plus a number of other species listed in the section "bird species affected" (Filippich, 1992; Filippich *et al*, 1993; Perry, 1993; Simpson, 1992). Going light is a disease syndrome which has been described as chronic fatal weight loss in the budgerigar despite an apparent ravenous appetite (Henderson *et al*, 1988). Such a broad syndrome could be associated with a number of causative agents affecting a number of body systems, all with the same outcome of chronic weight loss ending in death. The clinical disease caused by *Megabacterium* spp, megabacteriosis (Baker, 1992; Simpson, 1992), M.A.D. (*Megabacterium*-associated disease) (Perry, 1993) or proventricular disease (Filippich, 1992), has been described in detail by a number of persons (Baker, 1992; Filippich, 1992; Henderson *et al*, 1988; Perry, 1993). The clinical signs recorded are variable and range from:

**The acute form**:

Sudden death due to haemorrhage (Baker, 1992), or dying within 12 to 24 hours from the onset of clinical signs, which often go unnoticed. Filippich (1992), described an acute form of megabacteriois where birds in good condition became suddenly very depressed, lethargic and hypothermic. Many regurgitate blood or pass reddish, black droppings and all usually die within 12 to 24 hours. The acute form of the disease can be seen in the form of an outbreak in a budgerigar colony, where some birds in the colony are affected and die at around the same time or in a single individual (Filippich, 1992).

**The chronic form**:

The more common chronic form (Filippich, 1992), is characterised by progressive weight loss leading to concave pectoral muscles and emaciation, apparent polyphagia, depression, intermitted vomiting/regurgitation of crop contents and diarrhoea (Baker, 1992; Filippich, 1992; Henderson *et al*, 1988; Perry, 1993; Perry, 1995 ). Vomition or regurgitation , usually seen early in the disease process (Filippich, 1992; Perry, 1993), can often be identified by crop contents and sometimes blood, matting and staining the feathers on the top of the bird's head and around the commissures of the mouth (Perry, 1995). Neck stretching and mouth gagging may also be seen in an attempt by the bird to regurgitate (Filippich, 1992).

The droppings range from light khaki to dark green, to black in colour, with a consistency varying from being soft and mucousy, to watery diarrhoea, much of which

sticks to the tail feathers around the vent. Seed fragments and occasionally whole seed, can often be seen in the droppings (Baker, 1992; Filippich, 1992; Perry, 1993; Perry, 1995). The droppings are often bulky, being noticeably larger than normal droppings and whole seed may be visible to the naked eye (Perry, 1993; Perry, 1995).

Although the birds appear to exhibit polyphagia, it is reported that they only grind the seed up, allowing most to fall from their beaks and swallowing very little. It is therefore usual for the crop to be empty on palpation (Baker, 1992; Filippich, 1992;). Simpson (1992), reports to have noticed the same grinding of, but not swallowing seed in two budgerigars dying of *Leucocytozoon* infection. He suggests it may only be a sign of digestive discomfort, not a sign specific to megabacteriosis.

Birds with the chronic form of megabacteriosis often die in an emaciated state anywhere from 2-3 weeks, to several months from the onset of clinical signs (Baker, 1992; Filippich, 1992; Perry, 1993). During the end stage of the disease some birds lose their appetite and may exhibit paler legs and beak because of acute or chronic haemorrhage at the proventricular/gizzard junction (Perry, 1993; Perry, 1995). Once birds are showing the clinical signs of *Megabacterium*-associated disease, they rarely survive unless treated promptly and effectively (Perry, 1993). Some birds may recover but usually undergo a relapse several weeks or months down the track. These relapses occur when the bird is under the influence of a stressor, such as breeding , moulting or a concurrent disease (Filippich, 1992).

**Bird Species Affected**

Little is known about the extent of bird species which can become infected with *Megabacterium* spp, nor about the number which then go on to develop clinical disease . Perry (1993), using subjective assessment, reported that the most commonly infected birds in Australia were budgerigars (*Melopsittacus undulatus*), African lovebirds (*Agapornis* spp), and canaries (*Serinus canaria*). Simpson (1992) reported that he regularly diagnosed *Megabacterium* infections in budgerigars, grass parakeets (*Neophema* spp), cockatiels (*Nymphicus hollandicus*), and canaries. Filippich (1992) published a table (below), of bird species which have been faecal positive for *Megabacterium* spp:

| | |
|---|---|
| Gouldian finch (*Erythrura gouldiae*) | Rainbow lorikeet (*Trichoglossus haematodus*) |
| Scarlet-chested parrot (*Neophema splendida*) | Sulphur-crested cockatoo (*Cacatua galerita*) |
| Princess parrot (*Polytelis seainsonii*) | African lovebird (*Agapornis* spp) |
| Superb parrot (*Polytelis alexandrae*) | White-tailed black cockatoo (*Calyptorhynchus latirostris*) |
| Mulga parrot (*Psephotus varius*) | Border fancy canary (*Serinus canaria*)* |
| Red-crowned kakarikis (*Cyanoramphus novaezealandiae*) | Budgerigar (*Melopsittacus undulatus*)* |
| King parrot (*Alisterus scapularis*)* | Red-winged parrot (*Apromictus erythroterus*) |
| Zebra finch (*Taeniopygia guttata*) | Pistorella finch (*Heteromunia pectoralis*) |
| Bourke's parrot (*Neophema bourkii*) | Galah (*Eolophus roseicapilla*)* |

* **Birds with clinical or necropsy signs of proventricular disease.**

## Diagnosis

Megabacteriosis is diagnosed on the basis of the history, the clinical findings, and microscopic examination of crop and faecal samples. Necropsy along with histopathology and microbiology will supply a definitive diagnosis of proventriculitis and a concurrent *Megabacterium* spp infection. Culture and subsequent identification of the organism may be useful and was successfully carried out by Scanlan and Graham (1990) and Simpson (1992). More recently Filippich (1992), using the same techniques as Scanlan and Graham (1990), failed to successfully culture the organism leading to debate about the ease and usefulness of culturing the bacterium for diagnostic purposes.

**History and presenting signs**: The history is variable but for both forms of the disease an initiating stressor of some form can often be identified, such as moulting. The chronic form is often associated with birds which have been acquired from pet shops (usually crowded, badly managed and therefore highly stressful) and poorly managed, over-crowded aviaries. The birds usually present because they are listless and "sick-looking", that is they have a fluffed up/hunched appearance and are failing to act in what the owner perceives to be the birds "normal manner" (Perry, 1995). The weight loss and diarrhoea commonly pass unnoticed by the owner, as these signs are often considered subtle by the unfamiliar, being masked by feathers and "different" compared to larger animals.

**Clinical signs**: There appear to be two forms of megabacteriosis, the acute form and the more common chronic form. The acute form of the disease usually presents as sudden death, the clinical signs are short and sudden, and may go unnoticed (Baker,

1992; Filippich, 1992). If the disease follows a chronic course, the birds experience progressive weight loss and diarrhoea. The pectoral muscles become thin and concave, making the keel prominent and easy to feel. As the birds become sicker, they develop a fluffed-up appearance and may be seen resting on the floor of the cage/ground, instead of perching (Baker, 1992; Filippich, 1992; Henderson *et al*, 1988; Perry, 1993; Perry, 1995).

**Examination of faeces or crop contents**: Wet mount preparations of fresh faeces or crop contents (from vomitus or a crop wash) will usually reveal varying numbers of *Megabacterium* spp. Some birds, reported by Filippich (1992) to be around 15%, do not pass any or sufficient *Megabacterium* spp to detect on the slide (Baker, 1992; Filippich, 1992; Filippich and Perry, 1993; Perry, 1993). Repeated preparations are recommended especially if *Megabacterium*- associated disease is highly suspected or recently treated especially when the first faecal preparation, when examined microscopically, is *Megabacterium* negative(Filippich and Perry, 1993; Perry, 1993).

The bacterium is very large and easy to confuse with plant material also found in the faeces. Portions of plant plants usually sport one jagged end, whereas *Megabacterium* spp usually have both ends rounded in a cigar-like shape (Perry, 1993). Identification also can be made difficult by the pleomorphism exhibited by the bacterium, they can be straight, single or arranged end-to-end (more common), bent, Y-shaped or even constricted in a collar-like fashion (Bredhauer, 1996; Perry, 1993). The Y-shape especially, may be confused with branching or budding fungi, which can be distinguished by identifying yeasts or more filamentous fungal samples on the slide (Perry, 1993).

**Necropsy Findings**: At necropsy the carcass is emaciated, with very prominent keel bones and the crop is usually empty (Baker, 1992; Filippich, 1992; Perry, 1993). The proventriculus is distended (Baker, 1992; Filippich, 1992), with its diameter up to three times the normal size, and is separated from the gizzard by a narrow neck (Baker, 1992). The proventriculus is most commonly empty except for a thick, cloudy film (of what is usually mucus) covering the mucosa, concentrated at the distal end of the proventriculus and the proventricular-gizzard junction. This area is usually raised and often encircles the inside of the organ. The lesions are reported to be seen in most clinical cases and are described as ulcerations and petechiae, causing the overlying mucous cover to be stained black from blood (Baker, 1992; Filippich, 1992; Perry, 1993).

The gizzard is also affected, becoming very soft and smooth, often with no visible koilin lining. The kaolin lining, when present, may look devitalised and the luminal surface of the gizzard is usually brown in colour and roughened. In those birds with proventricular bleeding the intestines may be darkened or even black in colour (Baker, 1992; Filippich, 1992; Perry, 1993). Baker (1992) also found that birds which died

suddenly had melaenic intestinal contents, pale tissues and organs and thin, watery blood.

Histological examination of the proventricular mucosa carried out by Baker (1992) showed an inflammatory reaction with mononuclear cells, mostly lymphocytes, plus macrophages and the occasional heterophil. The ulcers, usually shallow, contained extravasated erythrocytes and showed a local inflammatory reaction sporting mainly heterophils. The mucosal architecture of the effected area was disrupted and the normal tubular glandular structure was absent. The mucosa was also often thicker than normal, with a notable increase in goblet cell numbers.

Microscopic and histologic examination of the affected area of the proventriculus showed large numbers of *Megabacterium* spp arranged parallel to one another and extending in finger-like projections from the mucosal surface into the lumen. The bacterium were superficial, rarely invading the mucosa but in severe cases they did extend into the lumen of the proventricular glands (Baker, 1992; Filippich, 1992).

Scanlan and Graham (1990), described what is believed to be the same bacterium, routinely seen in the superficial mucosa of the distal end of the proventriculus in clinically normal budgerigars. They did not indicate wether any significant pathological necropsy findings (visual, microscopic or histological) were associated with this area.

Haematological findings: *Trichomonas gallinae* infection of the crop causes the same "going light" syndrome as *Megabacterium* spp and therefor they cannot be distinguished from one another on clinical grounds. Henderson *et al* (1988) attempted to distinguish between the causative agents using haematology and found that birds infected with *Megabacterium* spp showed anaemia, heterophilia, lymphocytosis, monocytosis, thrombocytosis and basophilia.

Those birds suffering from trichomoniasis had white cell values within the normal range but did show thrombocytosis and also had a tendency toward hypochromic anaemia (Henderson *et al*, 1988).

Trichomoniasis can also be diagnosed via microscopic examination of crop contents, obtained by a crop wash. The wet mount preparations should be examined immediately after the sample is taken, as the protozoa have a tendency to stop moving as they cool. Unfortunately, a negative sample is not a 100% guarantee that the bird is trichomonas-free, as much is dependent on the veterinarian's skill in obtaining crop washes (it is important to apply negative pressure as the crop needle is being withdrawn because the protozoa are concentrated round the throat area).

**Treatment**

Scanlan and Graham's (1990) *in vitro* experiments indicated a sulpha/trimethoprim resistance but found *Megabacterium* spp to be sensitive to ampicillin. All 8 *Megabacterium* isolates in these experiments were also sensitive to bacitracin, carbenicillin, cephaloridine, chloramphenicol, novobiocin, penicillin and vancomycin. At least one of the 8 isolates showed either moderate or complete resistance to all other antimicrobials trialed in this study. It should be noted that based on carbohydrate utilization profiles (Scanlan and Graham, 1990), the eight isolates examined represented 7 different biotypes, and the antimicrobial sensitivity testing was carried out *in vitro*, not *in vivo*.

Filippich (1992), and more recently Filippich and Perry (1993), conducted a number of clinical therapeutic drug trials. During these trials different drugs were tested by administering them to faecal megabacterium positive budgerigars, which were checked weekly to assess any change in their *Megabacterium* status. The drugs were given via addition to the drinking water or by the crop gavage method. Faecal megabacterium status (using 100 x magnification), was recorded as 1+; 2+ or 3+ burdens with 1+ representing less than 5 *Megabacterium* per 22mm x 22mm preparation, 2+ as greater than 5 but less than 144 and 3+ as greater than 144 megabacterium per preparation. The birds were necropsied at the end of the trial and examined for evidence of a megabacterium infection and proventricular disease.

**Water medication trials**: (Filippich, 1992)

**Drugs used**:

Amoxycillin trihydrate - 1.8 g/L
Trimsu l- 250mg sulphadiazine and 50mg trimethoprim/L
Metronidazole - 2g/L
Ketokonazole - 333mg/L
Sodium carbonate-bicarbonate buffered water, pH 9.1
Hydrochloric acid water, unbuffered with a pH of 0.91
Chloramine - 1g/L
Chlorhexidine - 10mg/L.

At necropsy, 36% of the birds showed evidence of proventricular disease, these birds had been treated with either amoxycillin; sulfa/trimethoprim; metronidazole; bicarbonate buffered water; unbuffered acid water, chloramine and chlohexidine.

> "During the study, all but 8 of the birds, remain consistently faecal positive for *Megabacterium* spp although in some birds their megabacterium status fluctuated. Eight of the 1+ Megabacterium-positive birds became faecal negative at least once during the study and in one bird (bird 4, chloramine) proventricular scrapings were negative

at necropsy (Filippich, 1992)"

It should be noted that all other birds (3), being treated with chloramine remained faecal positive for Megabacterium spp and showed evidence of proventricular disease at necropsy. Results from drug trials (using the above drugs and method) reported by Filippich and Perry (1993), show none of the in water experiments were consistent in eliminating or even lowering the faecal *Megabacterium* status of the majority of birds in each group.

Crop gavage medication trials: (Filippich, 1992; Filippich and Perry, 1993)

The drugs used, in addition to those use in the water trial, were ampicillin sodium, nystatin suspension, doxycycline hydrochloride iodine, enfloxacin, potassium permanganate, itraconazole, bromhexine, sucralfate, cimetidine and amphotericin B.

These trials were carried out on both clinical and experimental budgerigars, with only the experimental birds euthanased and necropsied at the end of both studies. The results obtained on all the drugs tested showed success, a faecal *Megabacterium*-negative sample, in 1/3 birds treated with itraconazole , 1/3 given potassium permanganate and 28/30 birds treated with Amphotericin B (Filippich and Perry, 1993). Of all the drugs experimentally tested, given via crop gavage or intramuscular injection (Perry and Filippich, 1993), amphotericin B (Fungilin, Squibb), is the only drug effective against *Megabacterium* Spp. Results from the study carried out by Filippich and Perry (1993), indicated that 0.05ml *Fungilin*, twice a day, for 10 days, at 100mg/ml in drinking water, was inadequate as a treatment. This is supported by Perry (1993) who suggested that even doses of 0.15ml-0.25ml twice a day are insufficient to reliably elicit complete clinical recovery by the end of the 10 day treatment period.

Investigations into the ideal dose rates for the most effective and safe treatment of megabacteriosis in different bird species are still being carried out, but due to the possibility of drug resistance (7% of birds appeared refractive to treatment) it is suggested to dose at a higher rather than lower rate (Filippich and Perry, 1993; Perry, 1993).

At present, for a budgerigar-sized bird, Ross Perry (Perry, 1993; 1995) recommended that *Fungilin* be given at a dose rate of 4-6 drops by mouth or 0.25-0.3 ml directly into the crop, via a crop needle. The drug should be given at least morning and night but 3-4 times a day is preferable, for a minimum of 10 days. Ideally, for maximum effectiveness, the crop should be empty at the time of treatment. *Fungilin* has a very wide safety margin in birds, probably due to the fact that very little of the drug is absorbed from the alimentary canal into the body and dose rates of 1ml, twice a day have been given to budgerigars without inflicting death. Care should be taken when administering the drug as many birds regurgitate at least some, especially if given by

mouth or if fungilin is coating the outside of the crop needle.

Supportive care is often indicated, such as soft food or crumbles, as seeds and grits are likely to be irritant to an inflamed, ulcerative and haemorrhagic proventriculus (Perry, 1995). Granivore (supplied by wombaroo) or Poly-aid (supplied by Vetafarm) can be administered by crop needle (15mins or so after a fungilin treatment), if the bird is to sick to eat or is uninterested in food. Any concurrent infections, such as a secondary bacterial infection or chlamidiosis, should also be treated and until the bird has regained its normal body weight. Ina ddition, a heat and light source should be constantly provided (Perry, 1995).

The bird should be isolated from other birds and the cage, plus contents, repeatedly cleaned and disinfected throughout the treatment. One to two days after the cessation of treatment, the birds should be re-checked for faecal megabacteria and if positive, should be re-treated with higher doses. For example, treat with 0.5-1 mL at 2-4 times a day, via a crop needle for another 5 days. They should then be re-tested. Follow up testing should be carried out in all birds, 2-4 weeks after treatment finishes, this includes birds which tested negative for their first follow-up test. It is advised to permanently separate treated and apparently cured birds from untreated birds and to follow quarantine procedures when introducing new birds (Perry, 1993; Perry, 1995).

**Discussion**

*Megabacterium* spp is an organism, which is increasing in prevalence and importance in the avian world. As the bacterium is seen more and more in a clinical situation, that is in association with proventricular disease, there an increasing need to define what allows *Megabacterium* spp to cause disease. The interaction between the bird and the bacterium is very complex, and investigation is needed into the state of the birds immune system, possible immunity from the parent birds or due to past exposure and the bird species involved. I believe comparison between *Megabacterium* isolated from clinically normal birds, from birds with chronic megabacteriosis and from birds with the acute form of the disease is necessary to assess if the bacterium exhibits different pathogenicities in each situation. There is a lot to still learn about *Megabacterium* and the controversy surrounding its significance as a pathogen, the treatments which are successful and the bird factors involved are indicative of a complex situation - if it was black and white everyone would agree!

**Conclusion**

*Megabacterium* spp has been identified as a bacterium using electronmicroscopy and no DNA containing intracellular organelles have been found, indicating the organism is

not eukaryotic but Amphotericin B (an anti-fungal drug), is the recognized treatment of choice, raising a number of questions needing to be investigated, involving the taxonomy of the organism and the action of the drug in this situation. Although *Megabacterium* has been identified in wild birds, it is not known wether the bacterium was introduced and increasing, or if it occurs naturally, with little to no clinical significance. The extent of the organisms clinical significance and the disease's importance in the bird world is currently unknown and more investigation is required in-order to effectively treat/cure infected and clinically ill birds and to control the spread of megabacteria through the bird population. What is known, is that the prevalence is increasing in caged and aviary birds, so it is therefor becoming increasingly important for veterinarians to recognize the clinical signs of the disease and be able to confidently diagnose and treat the infection.

Megan Bredhauer (Department of Small Animal Medicine and Surgery, University of Queensland) is currently involved in carrying out this much needed research on *Megabacterium*, so we may soon be able to categorise the organism and more fully understand it's significance.

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