Megabacteriosis is a disease condition that we have been diagnosing at our practice quite often. It is difficult to say if it has become more common or if the means of diagnosis and recognition of the condition have improved. There are many aviculturists who have the organism in their collection of their birds and are totally unaware of its presence. Another consideration is that birds purchased from various sources could be carrying the organism with the resultant risk of transfer to birds in your collection.

Megabacteriosis is a poorly understood infection believed to be caused by megabacteria, a large gram-positive rod. Megabacteria are found in the proventriculus (acid stomach) and ventriculus (gizzard), particularly in the transitional zone between the two stomach portions in symptomatic and asymptomatic psittacine and non-psittacine birds.

Although originally thought by most researchers to be a bacterium, recent studies suggest that this organism is actually yeast. As fungi, the megabacteria are unusual because they mimic bacteria. Their fungal nature had been suspected because fungal medications appear to be more effective than antibiotics in treatment of megabacteriosis. Due to the evidence of its fungal nature, it has been suggested that megabacteria is a misnomer and it should be referred to as avian gastric yeast (AGY).

The question of pathogenicity is not clear. Megabacteria have low invasiveness and elicit a poor inflammatory reaction. Megabacteria have been detected in birds, particularly budgerigars, which fail to demonstrate any clinical signs of disease. The vast majority of birds in the collection at Texas A&M University that were diagnosed with megabacteriosis failed to display any clinical signs of ill health, nor any gross lesions on necropsy. It is suspected that either the organism is immunosuppressive or the host has been immunosuppressed at the time of exposure, by factors such as breeding, molting, infectious and non-infectious disease, transport, malnutrition, and other stressors.

An important factor in the pathogenicity might be the decrease in the acidity of the GI tract. The organisms are thought to reduce acid production in the proventriculus, which changes the pH levels, making the environment less acidic and interfering with normal digestion. The megabacteria altered the pH of the proventricular contents from a normal range of 0.7 - 2.4 in unaffected canaries, to 7.0 - 7.3 in severely affected birds. It remains to be determined if the alkalization is caused by the organism itself or by the mucus production of the host, thus inhibiting the release of acid from the storage form.

In the past, the infection was commonly seen in budgerigars (a chronic wasting condition termed “going light”), cockatiels, lovebirds, finches, and canaries, but it has now been
described in many species, including large psittacines. Diseases in chicks are rare, and the average age of affected birds is between 1 to 5 years.

Clinical signs are non-specific and indicative of a chronic, debilitating disease. Commonly reported signs of disease include chronic weight loss, apathy, anorexia, vomiting/regurgitation, and the passage of whole or partial seeds in soft, watery droppings. Digested blood may also be seen in the droppings. In the advanced stages of the disease, there may be vomiting of slimy material. Chronic emaciation occurs over a long period of time. The course of the disease may take months and there may be intermittent periods of recovery and relapse. Differential diagnoses include proventricular dilatation disease, lead, zinc or copper toxicoses, trichomoniasis, bacterial, mycotic or parasitic infections, and neoplasia.

An acute form has been described in budgerigars, where birds in good condition suddenly become severely depressed, show ruffled plumage, and die within 12 to 24 hours. They often regurgitate blood and the cause of sudden death may be excessive bleeding in the proventriculus.

Because of their size, megabacteria can be easily recognized under the microscope so that diagnosis can be made through the identification of megabacteria in wet mounts or stained smears from crop swabs, proventricular scrapings, and droppings. Periodic fecals should be examined, as there may be intermittent shedding of the organism. Because a fecal is negative for the presence of megabacteria, it does not entirely indicate that the bird is free from infection. In severely affected birds, Gram staining of the droppings may reveal the organism. Blood work reflects poor physical condition with anemia and low total protein. Megabacteria can be difficult to isolate on culture.

Radiographs may reveal proventricular dilatation as well as an hourglass-like constriction between the proventriculus and ventriculus. Thickening of the proventricular wall can also be seen with contrast (barium) studies.

Necropsy findings will indicate proventriculitis and proventricular dilatation. The proventricular wall may be thickened with small hemorrhages, and a thick mucus layer may cover the mucosa, particularly in the lower part of the proventriculus. The organism will be apparent in scrapings taken from the proventricular mucosa. The koilin layer of the ventriculus may be loosened, becoming brown in color and have a rough surface. In birds with more excessive bleeding, the intestinal loops may contain black ingesta.

Elimination of the disease can be difficult, as no effective treatment is known. Megabacteria have been shown to be resistant to tested antibiotics. In addition, control of the disease has not been adequately described. Treatment protocols that have been described as having some promise attempt to lower the pH of the proventriculus (often with Lactobacillus) to reverse the effects of the disease and make the environment less habitable for the megabacteria. Acidification of the drinking water with hydrochloric acid may help in some cases of megabacteriosis, as well in cases of enteritis that are not responsive to traditional therapy. For this treatment, a 1mol/L solution of HCl is mixed at
a rate of 30ml/pint of drinking water. Acidifying the GI tract with apple cider vinegar, white vinegar, or grapefruit juice has also been suggested. The pH of the water should not fall below 2.5 and should be continued for several weeks. Chlorhexidine (Nolvasan) in the drinking water has been suggested as a treatment, but it has not been shown to be effective.

Fungicides have also been tried and it has been reported that amphotericin B has been effective in treatment. We have great success in our practice treating these cases with oral amphotericin B. Oral amphotericin B at 100mg/kg PO SID for 10 days exhibits the highest rate of success. Periodic fecals are checked over several weeks to be certain that the bird has been cleared of the megabacteria. Amphotericin B is not absorbed systemically from the GI tract after oral administration so higher doses can be given in contrast to the IV dose. Amphotericin B can be difficult to obtain in the oral formulations, but it can be made at certain compounding pharmacies.

All clinically ill birds should be treated; however, there is a debate whether non-clinical birds displaying megabacteria in their droppings should be treated. Factors in making this decision include the difficulty in treating the bird, the exposure to other birds, ability of the owner to recognize signs of disease if they develop and owner compliance.

Megabacteriosis is a controversial condition, to say the least. Although there is agreement presently as to its fungal nature, questions still exist as to whether megabacteria are normal flora or are indicative of disease and the manner of treatment that is most effective. More research is needed to gain a better understanding of the condition. Be cautious with your purchases from uncertain sources. If there have been unexplained deaths of birds in your collection which have “gone light” and passing undigested food in the droppings, Megabacteriosis may be a consideration.