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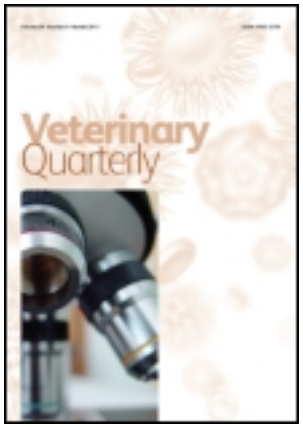

Veterinary Quarterly

Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/tveq20

Atherosclerosis in parrots. A review

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Available online: 01 Nov 2011

To cite this article: F.J. Bavelaar & A.C. Beynen (2004): Atherosclerosis in parrots. A review, Veterinary Quarterly, 26:2, 50-60

To link to this article: http://dx.doi.org/10.1080/01652176.2004.9695168

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Veterinary Quarterly 2004; 26(2): 50-60

Atherosclerosis in parrots. A review

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F.J. Bavelaar and A.C. Beynen

SUMMARY

Atherosclerosis is a common disease in parrots. The disease is found in all common parrot species, but especially in African Grey parrots and Amazons. It is a disease of older birds that is seen in both males and females. The most common sign is sudden death, but clinical symptoms that can be found include dyspnea, lethargy and nervous signs, such as paresis and collapses. Because the clinical signs are seldomly seen, it is difficult to diagnose atherosclerosis and therefore it is mostly an unexpected finding at necropsy. Age and species are determinants of atherosclerosis in parrots. Suggested risk factors include an elevated plasma cholesterol level, diet composition, social stress and inactivity, but research is needed to confirm this.

Keywords: *Arterial diseases; Atherosclerosis; Avian diseases; Cholesterol; Nutrition; Nutritional diseases; Parrot diseases; Risk factors; Vascular diseases*

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Introduction

Atherosclerosis is common among birds. Bohorquez and Stout (8) examined a group of 72 captive wild birds of 14 exotic avian orders that died in the Oklahoma City Zoo. In 90% of the birds, there were fatty streaks and fibrous plaques, whereas 24% showed real atheromatous lesions. Atherosclerosis appears to be more prevalent and severe among birds than among any species of mammals, except for humans. However, atherosclerosis is confined to certain species of birds, whereas it is virtually non-existent in others. A few examples of susceptible orders are Anseriformes (swan, geese, duck, etc.), Columbiformes (pigeons, doves, etc.), Galliformes (fowl, pheasants, etc.) and Psittaciformes (parrots, parakeets, etc.). In the group of Psittaciformes atherosclerosis is rarely seen in the smaller members, but it is very common in the larger members (17).

A lot of research has been conducted on atherosclerosis in domesticated birds. Examples are Japanese quails, chickens and pigeons. These animals are susceptible to both spontaneous and diet-induced atherosclerosis. Therefore, they have been widely used as experimental animals in atherosclerosis research (1,51). Less information is available on the occurrence of atherosclerosis in captive wild birds. This is particular evident in relation to the pathogenesis and risk factors of atherosclerosis in these birds.

One of the exotic avian orders susceptible to atherosclerosis are psittaciformes, especially the parrots. Since these birds are widely kept as pets, it is important to learn about their diseases, including atherosclerosis, and the clinical symptoms and etiology of these diseases. This paper gives a literature survey on atherosclerosis in parrots.

Description of atherosclerosis

Arteriosclerosis is the general term for thickened and hardened arteries. There are different types of arteriosclerosis and atherosclerosis is one of them. The word atherosclerosis is derived from the Greek words athero (gruel or porridge) and sclerosis (hardness). Atherosclerosis can be defined as a disease process that occurs when the influx and the deposition of cholesterol into the artery wall exceeds the egress of cholesterol (34). It is a chronic inflammatory condition, in which the artery wall, and specifically the intima, is thickened through accumulation of lipid and development of fibrous tissue (7,54). The pathogenesis involves deposition of fatty substances, cholesterol, cellular waste products, calcium and other substances in the intima. This build up is called plaque formation. The term atheroma is frequently used to describe an atherosclerotic lesion in which extracellular lipid is particular evident.

Atherosclerosis is a disease that usually affects the large and medium sized arteries. It is a slow, progressive disease that often starts at young age and progresses with age (39). The thickening of the artery wall decreases the diameter of the artery. Narrowing of an arterial lumen decreases

the blood flow through this artery and thereby reduces the oxygen supply. This can lead to ischemia in the area receiving blood from the artery. The lesions can furthermore lead to hemorrhage or complete occlusion of a vessel (54). The lesions that cause clinical signs are advanced lesions that are usually located in the aorta and the arteries supplying the heart, brain and lower limbs.

There are several different atherosclerotic lesions, namely the fatty streak, the fibrous plaque, and the complicated lesion. The fatty streak is believed to be the earliest lesion in atherogenesis (35,38,52, 53). Stary (52) observed fatty streaks in children and young adults. He found that fatty streaks in the coronary arteries were located at the same anatomical sites as advanced lesions in adults. The author suggested that over time fatty streaks at particular sites are converted by a series of changes into more advanced lesions, while fatty streaks at other anatomical sites either remain the same or regress and disappear. McGill (35) demonstrated that with time, fatty streaks occupy increasing surface areas of the coronary arteries and that these sites also precede the formation of advanced lesions. Thus, it is plausible that fatty streaks are the precursor lesions that convert into the advanced lesions (46).

Fatty streaks are superficial fatty patches, which are only slightly raised into the lumen of the artery and do not cause narrowing or clinical signs (54). They are characterized by an accumulation of cholesteryl-ester loaded cells, so-called foam cells, just beneath the endothelium in the intima. The foam cells are mainly derived from circulating monocytes, but some arise from smooth muscle cells in the artery wall (53). The focal accumulation of lipid in the arterial intima can be seen as red streaks or patches after gross staining with Sudan IV (17).

Fatty streaks can develop into more advanced lesions, namely fibrous plaques (7,53). These plaques are raised areas in the artery wall, and consist of an accumulation of smooth muscle cells, fibrous connective tissue and lipids (54). The lipids, which are located both intracellularly and extracellularly, are derived from the plasma. The fibrous connective tissue is produced by smooth muscle cells.

Finally, complicated lesions are derived from the fibrous plaques as they become hemorrhagic, ulcerated, calcified or involved in thrombosis (46). They are the cause of occlusion of the arteries and of clinical signs (54). The form and composition of advanced lesions point at three different biological processes. First, there is the accumulation of intimal smooth muscle cells and a variable number of macrophages and T-lymphocytes. Secondly, there is the formation of large amounts of connective tissue matrix, such as collagen, elastic fibres and proteoglycans. This matrix is formed by the proliferating smooth muscle cells. Finally, there is the accumulation of lipids, mostly cholesterol esters and free cholesterol within muscle cells, and in the surrounding connective tissues (46).

Atherosclerosis in parrots

Already in the beginning of the 20th century, alterations in the arteries of parrots have been described that closely resembled atherosclerosis in humans (22). Since then, many studies have been conducted to investigate the incidence of atherosclerosis in parrots. The incidence reported by different authors is given in Table 1.

Table 1. The incidence of atherosclerosis for different parrot species as reported in the literature

Author(s)	Species	Incidence*
Bavelaar and Beynen (2)	Amazons	78.4 (40/51)
	African Grey parrots	92.4 (97/105)
	Macaws	77.8 (7/9)
	Cockatoos	71.4 (15/21)
	Eclectus parrots	50.0 (4/8)
Bohorquez and Stout (8)	Macaws	100 (1/1)
	Amazons	66.7 (2/3)
Dorrestein et al. (14)	Psittacini	8.3 (6/72)
Fox (19)	Psittaciformes	2.3 (27/1157)
	Parrots	2.5 (27/1098)
Griner (20)	Cockatoos	8.3 (5/60)
	Platycerini	2.1 (7/340)
	Lorini	1.3 (5/387)
	Psittacini	9.6 (11/115)
	Arani	5.0 (8/160)
Grünberg (22)	Psittaciformes	29.9 (29/97)
Kempeneers (32)	Amazons	9.9 (15/152)
	African Grey parrots	12.6 (19/151)
	Macaws	2.2 (1/45)
	Cockatoos	0 (0/46)

*In percentage with number of animals between brackets

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Grünberg (22) examined 838 birds, including 97 psittaciformes. Atherosclerosis was diagnosed in 29 parrots. Bohorquez and Stout (8) investigated 72 birds, but only 4 of these birds were parrots. Three out of four parrots showed atherosclerotic lesions. Fox (19) reported the results of autopsies of 1,157 birds from the Philadelphia Zoological Garden. He found an incidence of 2.3% in the psittaciformes and of 2.5% in the parrot group. Furthermore, he observed that the incidence was a 4-fold higher in males than females. Griner (20) looked at the results of a 14-year-during period of necropsies at the San Diego Zoo. She found an incidence of atherosclerosis of 9.6% in Psittacini, 5.0% in Macaws and 8.3% in Cockatoos.

Johnson et al. (31) examined the results of necropsies of 12,072 birds from 1981 through 1992 in a retrospective survey. They found atherosclerosis in 53 birds, 42 of them being parrots. Because the total number of examined parrots was not given, no incidence could be determined. Atherosclerosis was found in 18 Amazons, 7 Cockatoos, 3 African Grey parrots, 3 Macaws, 2 Conures and 3 Eclectus parrots. It is clear that atherosclerosis occurs in all the common parrot species. The incidence was evenly distributed between male and female birds (20 males, 17 females, 6 unknown). Furthermore, the authors observed that the youngest animal with atherosclerotic lesions was younger than 1 year, while 10 birds were older than 10 years and 89% was older than 5 years.

Dorrestein et al. (14) examined the results of autopsies performed in 745 birds in 1975 at the Faculty of Veterinary Medicine, Utrecht. They found an incidence of 8.3% in Psittacini. Amazons were most common among the parrots with atherosclerosis. Kempeneers (32) studied the results of autopsies conducted between 1981 and 1985. He found an incidence of 9.9% in Amazons, 12.6% in African Grey parrots and 2.2% in Macaws. From the 46 Cockatoos examined no cases of atherosclerosis were diagnosed. Kempeneers (32) also found that the incidence of atherosclerosis did not differ between male and female birds and that atheromas were mainly seen in older birds.

Bavelaar and Beynen (2) studied the presence of atherosclerosis in parrots brought in for autopsy in 2001. They found that only 16.3% of all 202 examined birds were completely free of sudanophilic staining. The most susceptible species appeared to be African Grey parrots with an incidence of severe atherosclerosis of 24.8% and Amazons with an incidence of 15.7%. No severe atherosclerosis was seen in Macaws and Eclectus parrots. Gender was not related to the occurrence of atherosclerosis, but age was. Under 1 year of age, no plaque formation was found. However plaques did occur in the age group 1-5 years. The severity and incidence of atherosclerosis further increased with age.

A large variation exists between the incidences reported by different authors. Several explanations can be given. First of all, the examined parrots came from different populations as to feeding and housing habits. Some parrots were derived from zoological gardens, whereas others were kept as pets. The reports covered the time interval of 1933 to 2001. Over time, husbandry and feeding practices of parrots have changed, possibly influencing atherosclerosis incidence. Furthermore, the criteria used to diagnose atherosclerosis were different. Most authors scored atherosclerosis macroscopically after staining with Sudan III and IV. This method colors fat depositions in the artery wall. However, many authors did not report the criteria used, and some also used microscopical evidence of atherosclerosis. Furthermore, in some studies the parrots were only examined for the occurrence of atherosclerosis, whereas in other studies no special attention was paid to atherosclerosis. For example, the incidences reported by Dorrestein et al. (14), Kempeneers (32) and Bavelaar and Beynen (2) have all been determined in parrots brought in for autopsy to the Department of Pathology of the Faculty of Veterinary Medicine in Utrecht. However, both Dorrestein et al. (14) and Kempeneers (32) conducted a retrospective study using necropsy reports. During necropsy, the main attention is focused on the cause of death and not on the occurrence of atherosclerosis. Furthermore, autopsies were performed by different people. On the other hand, Bavelaar and Beynen (2) collected all parrots brought in for a period of 6 months and paid special attention to atherosclerosis. This approach is likely to result in a higher incidence of atherosclerosis. In conclusion, due to differences in experimental procedures and in populations studied the reported incidences show a large variation. However, it is clear that atherosclerosis is common among parrots.

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Species and age are related to atherosclerosis incidence. The most susceptible species are Amazons and African Grey parrots. Atherosclerosis is found in other species such as Cockatoos, Macaws and Eclectus parrots, but the incidence is lower. Both the incidence and severity increase with age. The effect of age could cause a part of the variation in incidence, because longevity may differ between populations. Bavelaar and Beynen (2) reported that the mean age of Amazons was significantly higher than that of other parrots. This higher age could be responsible for the higher incidence of atherosclerosis. The above-mentioned studies indicate that gender does not influence the development of atherosclerosis. However, Fox (19) reported that males had a 4 times higher incidence of atherosclerosis. The number of males investigated was also much higher than the number of females.

Atherosclerotic lesions in parrots

The location of atherosclerosis in parrots has been well described. It appears that atherosclerosis is limited to the central portion of the systemic circulation, and that the descending aorta is usually free of atherosclerotic lesions (22,23). Fox (19) described that lesions were more prominent at the beginning of the aorta, but that they could extend into the brachiocephalic arteries, down the thoracic and abdominal vessels, around and into the celiac branches and at times into the iliac branches. Furthermore, he found that parrots do not exhibit deforming ulcerating surfaces and that thrombosis does not seem to occur.

Bohorquez and Stout (8) reported that mural thrombi were scarce in birds. They suggested that this could explain the lack of ischemic complications. Fiennes (17) found that the thick thoracic segment of the aorta and the brachiocephalic arteries were severely affected in psittaciformes. No lesions were present with a particular frequency in the smaller muscular vessels. In his retrospective autopsy survey, Kempeneers (32) found that the aorta and the main arteries were the preferred locations for atherosclerosis. Johnson et al. (31) also conducted a retrospective survey. They found atherosclerosis in 42 psittaciformes. In 34 birds lesions were found in the aorta, in 7 birds in the brachiocephalic arteries, in 8 birds in the myocardial vessels, in 5 birds in the splenic arteries, in 3 birds in the pulmonary artery. Lesions in the gastric artery, meningeal arteries and the vasa vasorum were all found in one case. The authors stated that in parrots the large elastic and muscular arteries are most commonly involved and most severely affected. Furthermore they found that coronary and other cardiac arteries are affected, but not as generalized and common as in humans, and that atherosclerosis in cranium vessels is uncommon to rare.

It may be concluded that parrots have a central form of atherosclerosis, mainly limited to the thoracic part of the aorta and the brachiocephalic arteries. However, lesions can be found in a wide range of arteries in individual parrots.

Clinical signs of atherosclerosis in parrots

Based on lesion distribution, signs related to atherosclerosis would be expected to be related to a generalized reduction in peripheral perfusion and a gradually increasing cardiac work load (31). However, parrots usually do not show clinical signs of atherosclerosis, and the most common sign is believed to be sudden death (31). Thus, atherosclerosis is usually observed as an unexpected lesion at necropsy (31). It is therefore important to identify clinical signs which might accompany atherosclerotic disease in parrots.

There are some case reports of parrots suffering from atherosclerosis. Phalen et al. (40) described the case of a female Macaw of at least 11 years of age. The animal had persistent dyspnea and exercise intolerance. On examination, a large cardiac silhouette and signs of pulmonary disease were found. At necropsy, the proximal portion (1 cm.) of the aorta and the brachiocephalic arteries were narrowed by extensive raised plaques. When the arteries were examined histologically, severe atherosclerosis was found in the aorta and brachiocephalic arteries. The authors gave a list of possible clinical symptoms, namely dyspnea, muscle wasting, progressive paresis of the hind limbs and seizure-like activity. Vink-Nooteboom et al. (56) reported a case of severe atherosclerosis in a 16-year-old Cockatoo. The symptoms of the bird were lethargy, decreased appetite and falling of the perch. At necropsy, all large vessels including the coronary arteries showed severe atherosclerosis.

Johnson et al. (31) described two cases. The first case was a 7-year-old Amazon parrot. This bird

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had a 2-month history of daily regurgitation and a peculiar aura-like behavior of holding the right leg in front of the body while appearing to go into a semiconscious state. At necropsy, pronounced atherosclerosis was observed in many vessels, including the entire length of the aorta and brachiocephalic arteries. The neurological signs may have been the result of reduced blood flow to the brain through stenotic carotid arteries. The authors had no explanation for the regurgitation. The second case was an 18-year-old African Grey parrot. The parrot suffered of rhinitis, dyspnea and weight loss. At necropsy, vascular changes including wall thickening, yellow discoloration and increased rigidity were seen in the entire length of the aorta and brachiocephalic arteries.

Dorrestein et al. (14) looked at autopsy reports of birds. They found that clinical nervous symptoms were frequently seen in birds suffering from atherosclerosis. The authors hypothesized that this was due to ischemia of the brain. Kempeneers (32) found that the most common symptoms were frequent faints, dyspnea, sudden death and lethargy. Johnson et al. (31) reviewed 32 cases in detail in order to correlate clinical signs with necropsy findings. Of these 32 cases, eight parrots had severe atherosclerosis and no signs of other unrelated diseases. Six out of the eight birds had a history of sudden death. One parakeet collapsed suddenly and showed dyspnea shortly before death. An Amazon was anorectic, lethargic, and had a five-day progressive course of hind-limb paresis developing into paralysis. The authors described the following symptoms: dyspnea, sudden collapses, anorexia, lethargy and hind limb paresis.

It appears that atherosclerosis only seldomly causes clinical signs. If parrots do show signs, the signs are related to a decrease of the blood flow to the central nervous system or to heart failure (40). However, because parrots are confined to a cage and have little exercise, they may be asymptomatic until they are stressed or until they acutely decompensate (31). Therefore, it is not clear how much impact the presence of atherosclerosis has on the parrot. To provide more insight into the effects of atherosclerosis in parrots the diagnosis in the living animal is essential.

Diagnosis of atherosclerosis in parrots

The diagnosis of atherosclerosis in living parrots is very difficult. Atherosclerosis usually does not cause overt clinical signs. The most common sign is sudden death. Therefore, when a bird is suffering from respiratory, circulatory of neurological signs or from lethargy, a comprised cardiac function due to atherosclerosis should be included in the differential diagnoses (31). That diagnosing of atherosclerosis is difficult becomes clear from the report of Phillips (41). He described the clinical findings of 112 cases of larger parakeets and parrots. No atherosclerosis was diagnosed, but based on the the high incidence of the disease a number of animals must have suffered from atherosclerosis.

A problem with the diagnosis of atherosclerosis is that the animals are often suffering from more conditions than atherosclerosis alone. Kempeneers (32) reported that in only 50% of the atherosclerosis-positive autopsies atherosclerosis was the only disorder found. Dorrestein et al. (14) described that in many cases other diseases, such as nephritis and pneumonia, were presented in combination with atherosclerosis. The fact that other conditions could be present makes the diagnosis of atherosclerosis even more difficult, because it is then easier to miss it.

There are diagnostic tools that can be used to diagnose atherosclerosis, such as auscultation and radiography. With the help of auscultation a heart murmur or irregularities in the heart rhythm can be found, although it is rarely reported for birds. A heart murmur can be confirmed with the help of phonocardiography and electrocardiography can be used to find irregularities in the heart rhythm (31). Furthermore, radiography and echocardiography can be used for diagnosing cardiac abnormalities (31,56). Because the anatomy of the cranial portion of the thorax is complex, it is difficult to observe changes in arteries and veins and abnormalities of the heart. Furthermore, the use of angiography is limited in birds. Echography could be effective for evaluation of heart size and function, but the heart is surrounded by air and bone and cannot always be seen adequately (40).

In conclusion, the diagnosis of atherosclerosis in the living parrot is very difficult and not always possible. However, atherosclerosis should always be included in the differential diagnosis of lethargy, weight loss, nervous symptoms, circulatory and respiratory signs.

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Risk factors of atherosclerosis in parrots

Little is known about possible risk factors of atherosclerosis in parrots. Since atherosclerosis in parrots closely resembles the lesions found in man (22), risk factors in humans may extend to parrots. An important risk factor of atherosclerosis in humans is an elevated plasma cholesterol level (11). Avian species, such as pigeons, quail and chickens, have been widely used in atherosclerosis research. Based on literature data, Bavelaar and Beynen (1) found a linear relationship between plasma cholesterol and the severity of atherosclerosis in pigeons, quails and chickens. Possibly, plasma cholesterol also is a risk factor in parrots. Finlayson and Hirchinson (18) induced hypercholesterolemia and severe atherosclerosis in female budgerigars by feeding 2.0% cholesterol during 6 months. Wolkoff (58) found severe atherosclerosis in a 40-year-old African Grey parrot that had been fed an egg daily for three years. The author suggested that the parrot suffered from cholesterol-induced atherosclerosis. Thus, psittaciformes might be susceptible to cholesterol-induced atherosclerosis. However, it is not known whether there is a relation between plasma cholesterol and the development of atherosclerosis in parrots. Plasma cholesterol values have been reported for different parrot species (Table 2). African Grey parrots and Amazons species have higher plasma cholesterol levels when compared to the other species. African Grey parrots and Amazons also are the most susceptible parrot species as to the development of atherosclerosis (2,14,32). If plasma cholesterol is indeed a risk factor of atherosclerosis in parrots, the high values in the susceptible species could explain their susceptibility.

Table 2. Plasma cholesterol levels for the different parrot species as reported in the literature

Authors	Number	Species	Cholesterol (mmol/l)*
Bavelaar and Beynen (3)	30	African Grey parrot	8.4 ± 2.6
Bavelaar and Beynen (5)	3	Yellow-headed Amazon	8.2 ± 2.8
	3	Blue-fronted Amazon	8.8 ± 4.6
	4	Blue-and-yellow Macaw	4.5 ± 0.8
	3	Scarlet Macaw	5.1 ± 0.2
	14	Red fan parrot	4.2 ± 1.0
	4	Long-billed Cockatoo	6.0 ± 1.3
Polo et al. (42)	6	Yellow-crowned Amazon	7.1 ± 2.5
	11	African Grey parrot	6.8 ± 0.7
	30	Blue-and-yellow Macaw	4.2 ± 0.9
	12	Scarlet Macaw	4.3 ± 1.1
	13	Hyacinthine Macaw	3.1 ± 0.6
	16	Green-winged Macaw	4.2 ± 0.9
	16	Military Macaw	4.2 ± 1.3
	8	Palm Cockatoo	3.6 ± 0.5

*Values are mean ± SD

Atherosclerosis research in humans and experimental animals has focused on the role of the diet; especially the amount and type of dietary fat, in the development of atherosclerosis. Isoenergetic replacement of saturated fatty acids by either polyunsaturated fatty acids or carbohydrates decreases plasma cholesterol levels and thus decreases the risk for atherosclerosis (21,28,36).

Bavelaar and Beynen (3) conducted a feeding experiment with African Grey parrots and found that a high-fat diet rich in saturated fatty acids significantly increased plasma cholesterol levels when compared to feeding either a low-fat diet or a high-fat diet rich in the n-6 polyunsaturated fatty acid linoleic acid. These results could imply that dietary fatty acid composition influences the risk

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of atherosclerosis in parrots. However, differences in commercial parrot feeds probably are too small to have a significant impact. Bavelaar and Beynen (4) collected commercial parrot feeds available on the Dutch market and analyzed the fat content and fatty acid composition. With the help of a formula established by Mensink and Katan (36) the effect of dietary fatty acid composition on plasma cholesterol levels in humans can be predicted. When this formula was used for the commercial parrot feeds, Bavelaar and Beynen (4) found only small differences in effects on plasma cholesterol levels, ranging from about -0.25 to 0.20 mmol/l. This small range is due to the fact that even though the fat level is high in some feeds, they are all rich in polyunsaturated fatty acids (4).

The intake of n-3 polyunsaturated fatty acids may protect against the development and progression of atherosclerosis in humans and experimental animals, such as quails and monkeys (12,16,24,30, 48). For parrots, the main n-3 fatty acid is α-linolenic acid (C18:3n-3), which can be found in plant oils such as soybean oil, rapeseed oil, perilla seed oil and linseed oil. Eicosapentaenoic acid (C20:5n-3, EPA) and docosahexaenoic acid (C22:6n-3, DHA) are present in appreciable amounts in the fat component of fish products. The effect of n-3 polyunsaturated fatty acids on the development and progression of atherosclerosis is multifactorial. It is believed that n-3 polyunsaturated fatty acids have anti-thrombotic properties, that they have a favorable effect on endothelial function, and that they decrease platelet-derived growth factor, which is the key chemoattractant and mitogen for smooth muscle cells and macrophages, cell types involved in plaque formation. Furthermore, n-3 polyunsaturated fatty acids have a strong anti-inflammatory action, which is important because atherosclerosis is an inflammatory disease (47). Polyunsaturated fatty acids of the n-3 family have hypolipidemic properties (10,25,47,49). Since parrots usually do not consume fish products, α-linolenic acid is the major n-3 polyunsaturated fatty acid for parrots. Most research on the effects of n-3 polyunsaturated fatty acids has been conducted with fish products and therefore it is uncertain whether α-linolenic acid exerts the same effects. While EPA and DHA generally lower plasma triglycerides in humans, it is believed that α-linolenic acid has no strong hypotriglyceridemic properties (10,26). In animal studies, EPA and DHA also have no consistent effect on plasma triglycerides (27). On the other hand, it is believed that α-linolenic acid has the same cholesterol-lowering capacity as does linoleic acid (6,10,26,50).

Bavelaar and Beynen (2) found a relation between the degree of atherosclerosis and the intake of α-linolenic acid in parrots presented for autopsy. Since it was not possible to assess fatty acid composition of the diets consumed by the parrots, fatty acid composition of breast muscle and adipose tissue, if available, was used as an index of fatty acid intake. It was observed that the content of α-linolenic acid in both breast muscle and adipose tissue was negatively associated with the severity of atherosclerosis. Thus, α-linolenic acid might play a protective role against the development of atherosclerosis in parrots. It is interesting to note that commercial parrot diets had α-linolenic acid contents ranging between 0.03 and 0.65 g/MJ metabolizable energy (4). Bavelaar and Beynen (5) fed the two commercial diets, with lowest and highest α-linolenic acid content to parrots, but did not find an effect on plasma cholesterol. However, it is possible that this lack of effect could be due to a too small contrast in α-linolenic acid in combination with the high levels of linoleic acid in the diets. If α-linolenic acid protects against the development of atherosclerosis in parrots, it may not be mediated through plasma cholesterol levels.

A role of the diet, if any, in the development of atherosclerosis in parrots so far is not clear. Fiennes (17) reported that susceptible groups of animals are those whose natural diet consists of fruits or fresh animal products. The author suggested that changes in the natural diet in captivity could play a role in the development of atherosclerosis. Ullrey et al. (55) pointed out that most seeds found in mixtures sold for caged parrots are foreign to their free-living relatives. Griner (20) mentioned the role of the diet in relation to the difference in incidence of atherosclerosis between Lories and parrots, both members of the psittaciformes. The incidence in Lories was 0.5% (1 out of 185 Lories), whereas the incidence in Psittacini was 9.6%. The author suggested that the species difference was related to diet composition, the Lories being fed a diet with low fat content. However, it is more likely that genetic factors play a role. Ratcliffe (45) reported results of necropsies conducted at the Philadelphia

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Zoological Garden, as from 1901. He found that after a diet change, the pattern of atherosclerosis changed too. Before 1935, parrots were fed sunflower seeds supplemented with small amounts of fruits, and after 1935 they were fed the Penrose Research Laboratory diet for omnivores. Along with the diet change, relative mass atheromas of the proximal aorta and the brachiocephalic arteries were replaced by less conspicuous lesions. This would suggest that diet can influence the development of atherosclerosis.

Further risk factors in humans are among others exposure to cigarette smoke, obesity, and inactivity (9,13,29,31,33,43,54). Warnock et al. (57) found that inactivity in chickens resulted in an increase in atherosclerosis, while forced exercise seemed to protect. Ratcliffe and Cronin (44) reported that after the diet was changed at the Philadelphia Zoological Garden, the parrots showed a 2-fold increase in age and a 6-fold increase in the incidence of atherosclerosis. A part of the increase in atherosclerosis could be a result of the increase in age, but that could not be the only cause. The frequency of atherosclerosis rose mainly in larger birds, such as the parrots, having little tendency or opportunity for appropriate natural behavior. The authors hypothesized that the increase in atherosclerosis could be due to both inactivity and social stress caused by the inability to exhibit natural behavior and due to stress as a result of the attempts to assemble and maintain breeding stocks. The social stress results in an imbalance of adrenal secretion. Ratcliffe (45) found that chickens exposed to social stress developed more atherosclerosis.

Some authors have mentioned other possible risk factors such as dietary deficiencies, thyroid disease and a viral agent (31,56). It is believed that the herpes virus that causes Marek's disease in chickens can cause atherosclerosis as well in these animals (15,37).

In conclusion, possible risk factors for atherosclerosis in parrots are age, genetics, plasma cholesterol levels, diet, inactivity, social stress and obesity. However, only the effects of age and genetics have been clearly demonstrated in parrots.

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