Influence of age and sex on plasma lipid and lipoprotein concentrations and associated enzyme activities in cats

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Objective—To determine effects of age and sex on plasma lipid and lipoprotein metabolism in cats.

Animals—33 kittens and 16 adolescent, 23 adult, and 10 senior cats.

Procedure—Plasma concentrations of cholesterol, triglyceride, and lipoprotein-cholesterol and activities of lipoprotein lipase, hepatic lipase, and lecithin:cholesterol acyl transferase (LCAT) were measured and compared within and among groups.

Results—Plasma cholesterol and triglyceride concentrations were significantly higher in 5- and 7-week-old kittens, compared with the same kittens after weaning and cats in the other age groups. Cholesterol concentration was significantly less in 20-week-old kittens, compared with adolescent and adult cats. Lipid and lipoprotein-cholesterol concentrations were not significantly different among the adolescent, adult, and senior groups, nor did sex influence lipid and lipoprotein-cholesterol concentrations in these groups. Activities of lipoprotein and hepatic lipases were significantly less in senior cats, compared with the other groups. Activity of LCAT was highest in 20-week-old kittens and was greater in sexually intact adult and adolescent females, compared with their male counterparts. After castration, activities of hepatic lipase and LCAT significantly decreased in adolescent male cats.

Conclusions and Clinical Relevance—The upper limits of reference ranges for plasma cholesterol and triglyceride concentrations should be increased for kittens < 8 weeks of age. Low cholesterol concentrations in adolescent cats likely reflect high tissue demands for growth and steriodogenesis. Decrease in lipoprotein and hepatic lipase activity in senior cats could predispose this age group to hypertriglyceridermia, particularly in insulin-resistant cats or those fed a high fat diet. (Am J Vet Res 2001;62:331–336)

Plasma lipoproteins transport aqueous insoluble lipids, specifically cholesterol and triglycerides, through the blood to specific tissues for metabolism, storage, or excretion. The uptake and transfer of lipids by lipoproteins is facilitated by their interaction with several key enzymes and cell surface receptors that regulate the flux of lipids and the concentration of lipoproteins within plasma.1 Interest in human lipoprotein metabolism is fueled by the role that certain lipoproteins play in the etiopathogenesis of atherosclerosis and the fact that hyperlipidemia is related to disturbances in lipoprotein metabolism that develop as primary defects or secondary to diseases such as diabetes mellitus.2

Although lipoprotein metabolism has been studied in numerous animal species as models for atherosclerosis3 or hyperlipidemia,1 little is known about lipoprotein metabolism in cats. Demacker et al4 identified lipoproteins with the physical and chemical characteristics of very-low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL) in plasma from 12 cats from which food had been withheld overnight. Chylomicrons were found in plasma samples obtained after a fat-rich meal, and activities of lipoprotein and hepatic lipases were found to be similar to those in healthy humans.

We have described methods for the measurement of plasma lipoprotein concentrations and activities of lipoprotein lipase, hepatic lipase, and lecithin:cholesterol acyl transferase (LCAT) in feline plasma.5 Furthermore, we showed that cats, similar to several other animal species, lack cholesteryl ester transfer protein and appear to accomplish reverse cholesterol transport by direct tissue uptake of cholesteryl esters from HDL. This was recently confirmed by results of another study in which cats were found to have notable plasma phospholipid transfer activity.6

Interest in clinical aspects of feline lipoprotein metabolism has been stimulated by the description7 and genetic investigation8 of inherited hypercholesterolemia in cats and transient hyperlipidemia in kittens,9,10 together with awareness that conditions such as diabetes mellitus may be associated with disturbances in lipid metabolism.11,12 There are, however, few reports describing normal variation in plasma lipid and lipoprotein concentrations in healthy cats. In one study,13 plasma cholesterol and triglyceride concentrations were significantly higher in sexually intact male cats, compared with females. We have shown that plasma cholesterol and triglyceride concentrations are low during lactation.14 This was associated with a pronounced reduction in VLDL concentration attributable to induction of lipoprotein lipase activity in mammary
Results of these 2 studies have highlighted a need to better understand the impact of factors such as age, sex, and diet on lipoprotein metabolism in cats. This is particularly important in determining reference ranges for the interpretation of plasma lipid concentrations in clinical situations. For instance, mean plasma cholesterol concentrations in one of these studies were approximately 2 to 3 times higher than those in the other study. The purpose of the study reported here was to determine the effects of age and sex on plasma triglyceride and cholesterol concentrations in cats so that reference ranges could be amended where necessary. Furthermore, by measuring plasma lipoprotein concentrations and activities of the enzymes involved in their metabolism, we sought to understand the biochemical mechanisms underlying variations in plasma lipid concentrations in cats.

Materials and Methods

Animals and diet—Blood samples were collected from 4 cohorts of healthy cats kept at the WALTHAM Center for Pet Nutrition. These comprised kittens from 5 to 20 weeks old, adolescent cats from 9 to 12 months old, adult cats from 1 to 6 years old, and senior cats > 7 years old. All cats were domestic shorthairs and were nonobese, with body condition scores ranging from 2 (lean) to 4 (optimal) on a scale of 1 to 6. Adolescent, adult, and senior cats were fed a standard canned diet during and for at least 6 weeks prior to sample collection. The same diet was fed to the kittens after weaning. The nutrient composition of the diet was analyzed in triplicate by use of standard methods, including quantification of fatty acids by use of gas chromatography.

Blood samples were collected from 33 kittens (20 male, 13 female) belonging to 10 litters. Mean litter size was 3.3 kittens (range, 3 to 5 kittens). Samples were collected 5 weeks after birth (mean age, 38 days; range, 35 to 39 days), 1 week prior to weaning (ie, 7 weeks; mean age, 51 days; range, 48 to 53 days), 4 weeks after weaning (ie, 12 weeks; 87 days; 84 to 88 days), 8 weeks after weaning (ie, 16 weeks; 115 days; 112 to 116 days), and 12 weeks after weaning (ie, 20 weeks; 139 days; 138 to 140 days). Plasma cholesterol and triglyceride concentrations were measured in each sample, and samples from all kittens in each litter collected at 20 weeks of age were pooled to facilitate full lipid and lipoprotein analysis, including determination of enzyme activities.

The adolescent group comprised 8 sexually intact males and 8 sexually intact females with a mean age of 215 days (range, 211 to 218 days). Male cats were castrated soon after this date, and blood was again collected 8 weeks later. Female cats were neutered. The adult group comprised 23 adult cats (5 sexually intact males, 8 castrated males, 10 sexually intact females) with a mean age of 3 years (range, 2 to 6 years). The senior group comprised 8 sexually intact females and 2 castrated males with a mean age of 8.6 years (range, 7 to 11 years).

Sample collection—Blood samples from all groups other than 5-week-old kittens were collected from the cephalic vein after food had been withheld overnight for 16 hours. Food was not withheld from 5-week-old kittens before collecting blood samples. Blood was collected into tubes containing potassium EDTA for analysis of plasma concentrations of triglycerides, cholesterol, and lipoprotein-cholesterol and activity of LCAT. For measurement of plasma lipoprotein lipase and hepatic lipase activities, blood was collected into tubes containing lithium heparin 10 minutes after IV injection of 70 U of heparin/kg of body weight. Samples for measurement of lipid and lipoprotein concentrations were placed on ice, and the plasma was separated by centrifugation at 4 °C and stored on ice to the laboratory, where analysis was completed within 72 hours of collection. Samples for enzyme analysis were immediately placed on ice, and the plasma was separated by centrifugation at 4 °C and stored in aliquots at −20 °C before being shipped to the laboratory on dry ice for analysis.

Determination of plasma lipid and lipoprotein concentrations—Plasma cholesterol and triglyceride concentrations were measured, using commercially available reagents. Lipoprotein-cholesterol concentrations were determined by use of a combined ultracentrifugation-precipitation technique that has been adapted and validated for use in cats. Briefly, starting with 3 ml of plasma, VLDL were first separated by ultracentrifugation at a density of 1.006 g/ml. Low-density lipoproteins were then precipitated from HDL in the infranatant by the addition of 92 mM heparin-manganese chloride. The cholesterol contents before and after precipitation of LDL were measured to determine concentrations of HDL-cholesterol (HDL-C) and, by subtraction, LDL-cholesterol (LDL-C). The concentration of VLDL-cholesterol (VLDL-C) was calculated by subtraction of concentrations of HDL-C and LDL-C from total plasma cholesterol concentration.

Determination of lipoprotein lipase and hepatic lipase activities—Lipase activity was measured, using a triglyceride emulsion containing glycerol tri[1-14C]oleate. Total lipase activity was measured in the presence of 0.1M NaCl and human serum as a source of apoprotein C-II for the activation of lipoprotein lipase. Hepatic lipase activity was measured in 1M NaCl without serum activator, and lipoprotein lipase activity was calculated as the difference between total and hepatic lipase activities. The intra- and interassay coefficients of variation for lipoprotein lipase were 18.9 and 20.4%, respectively, and for hepatic lipase, 4.6 and 10.7%, respectively.

Table 1—Composition of feline milk and a canned food diet fed to 33 kittens and 16 adolescent, 23 adult, and 10 senior cats

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Canned food</th>
<th>Feline milk*5,10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moisture (g/100 g)</td>
<td>73.5</td>
<td>77–82</td>
</tr>
<tr>
<td>Protein (g/100 g)</td>
<td>11.4</td>
<td>7.0–9.5</td>
</tr>
<tr>
<td>Fat (g/100 g)</td>
<td>10.6</td>
<td>4.0–6.5</td>
</tr>
<tr>
<td>Ash (g/100 g)</td>
<td>2.8</td>
<td>0.6–0.8</td>
</tr>
<tr>
<td>NFE (g/100 g)</td>
<td>12.3</td>
<td>75–116</td>
</tr>
</tbody>
</table>

NFE = Nitrogen-free extract. ME = Predicted metabolizable energy.

Figure 1—Mean (±SD) plasma cholesterol and triglyceride concentrations in 33 kittens at 5, 7, 12, 16, and 20 weeks of age. *Significantly (P < 0.05) different from values at 12, 16, and 20 weeks.
Table 2—Effects of age on plasma lipid and lipoprotein-cholesterol concentrations in cats

<table>
<thead>
<tr>
<th>Group</th>
<th>Triglycerides (mmol/L)</th>
<th>Cholesterol (mmol/L)</th>
<th>VLDL-C (mmol/L)</th>
<th>LDL-C (mmol/L)</th>
<th>HDL-C (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kitten* (10)</td>
<td>0.33 ± 0.03</td>
<td>4.91 ± 0.59</td>
<td>0.23 ± 0.10</td>
<td>0.96 ± 0.25</td>
<td>3.72 ± 0.47</td>
</tr>
<tr>
<td>Adolescent (16)</td>
<td>0.36 ± 0.05</td>
<td>6.03 ± 1.22</td>
<td>0.23 ± 0.08</td>
<td>1.58 ± 0.59</td>
<td>4.21 ± 0.71</td>
</tr>
<tr>
<td>Adult (23)</td>
<td>0.35 ± 0.01</td>
<td>6.23 ± 1.20</td>
<td>0.25 ± 0.12</td>
<td>1.78 ± 0.76</td>
<td>4.30 ± 0.71</td>
</tr>
<tr>
<td>Senior (10)</td>
<td>0.38 ± 0.09</td>
<td>5.65 ± 1.12</td>
<td>0.21 ± 0.07</td>
<td>1.42 ± 0.93</td>
<td>4.00 ± 0.78</td>
</tr>
</tbody>
</table>

Data reported as mean ± SD. *Samples from 20-week-old kittens in each of 10 litters were pooled before analysis. 132 weeks old. 12 to 6 years old. 97 to 11 years old.

VLDL-C = Very-low-density lipoprotein-cholesterol. LDL-C = Low-density lipoprotein-cholesterol. HDL-C = High-density lipoprotein-cholesterol.
*Within a column, values with different superscripts are significantly (P < 0.05) different.

Effects of age—Plasma triglyceride and VLDL-C concentrations were similar among all ages of cats (Table 2). Total cholesterol concentrations were significantly less in 20-week-old kittens, compared with adolescent and adult cats. Cholesterol concentration was less in senior cats than in the other age groups, but this difference was not significant.

The low cholesterol concentration in kittens was primarily attributable to low LDL-C concentration; LDL-C concentration in 20-week-old kittens was significantly less than in the adolescent group. However, concentration of LDL-C did not differ significantly among kittens, adults, and senior cats. This may have reflected the large variation of data in the latter 2 cohorts. Concentration of HDL-C was significantly less in kittens, compared with adults, but was not significantly different from concentrations in adolescent and senior cats (Table 2).

Plasma activity of lipoprotein lipase did not differ among kittens, adolescents, and adults but was significantly less in senior cats, compared with the other groups (Table 3). Hepatic lipase activity was also significantly less in senior cats. Lipoprotein lipase and hepatic lipase activities in senior cats were reduced by between 37 and 47% and 36 and 66%, respectively, compared with the other cohorts. Hepatic lipase activity was significantly greater in adolescent cats, compared with kittens, adults, and seniors. Activity of LCAT was highest in kittens and decreased significantly through adolescence into adulthood. Activity of LCAT in senior cats was not significantly different from that in adults.

Influence of sex and gonadectomy—In the adolescent cohort, we did not detect significant differences in plasma lipid and lipoprotein concentrations...
between sexually intact females and males or between sexually intact and castrated males. In the adult group, cholesterol, VLDL-C, and HDL-C concentrations were significantly higher in castrated males, compared with sexually intact males (Table 4).

Lipoprotein lipase activity was less in sexually intact adolescent and adult male cats, compared with sexually intact adolescent and adult females, but the difference was significant for the adolescent group only (Fig 2). Hepatic lipase activity was similar between sexually intact males and females in both the adolescent and adult groups, and differences in enzyme activities were not detected between sexually intact and castrated males in the adult group. Hepatic lipase activity was, however, significantly reduced in adolescent males following castration (Fig 3).

In the adolescent and adult groups, plasma LCAT activity was significantly higher in sexually intact females, compared with sexually intact males (Fig 4). In the adolescent group, LCAT activity decreased significantly in male cats after castration, but in the adult group, the activity of this enzyme was significantly higher in castrated males, compared with their sexually intact counterparts.

**Discussion**

In humans and several other animal species, plasma lipid and lipoprotein concentrations are influenced by factors such as genotype, age, sex, adiposity, diet, and exercise. To limit such variability in the present study, we used cats of a single breed type and fed a constant diet to all cats. Having done this, we found only minor variations in plasma lipid and lipoprotein concentrations among the 4 age groups. There were, however, certain findings that may have clinical implications. These include relatively high lipid concentrations in kittens < 12 weeks old, low cholesterol concentrations in weaned kittens, a decrease in lipoprotein and hepatic lipase activities in senior cats, and an apparent effect of sex steroids on LCAT activity.

Data from the present study suggest that reference ranges for plasma triglyceride and cholesterol concentrations in kittens prior to weaning should be modified from reference ranges for adults. Using our data, suggested upper reference limits (mean + 2 SD) for plasma triglyceride and cholesterol concentrations in suckling kittens are 1.70 and 13.50 mmol/L, respectively, and in kittens prior to weaning from which food has been withheld, 0.75 and 11.90 mmol/L, respectively. It is tempting to associate the high plasma triglyceride and

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**Table 4—Effects of sex on plasma lipid and lipoprotein-cholesterol concentrations in adult cats**

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>Triglycerides (mmol/L)</th>
<th>Cholesterol (mmol/L)</th>
<th>VLDL-C (mmol/L)</th>
<th>LDL-C (mmol/L)</th>
<th>HDL-C (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact female (10)</td>
<td>0.32 ± 0.05</td>
<td>6.20 ± 1.10</td>
<td>0.22 ± 0.06</td>
<td>1.60 ± 0.70</td>
<td>4.40 ± 0.85</td>
</tr>
<tr>
<td>Intact male (5)</td>
<td>0.34 ± 0.07</td>
<td>5.20 ± 0.59</td>
<td>0.15 ± 0.04</td>
<td>1.27 ± 0.71</td>
<td>3.80 ± 0.39</td>
</tr>
<tr>
<td>Castrated male (8)</td>
<td>0.40 ± 0.12</td>
<td>6.90 ± 1.31</td>
<td>0.36 ± 0.12</td>
<td>2.08 ± 0.87</td>
<td>4.40 ± 0.60</td>
</tr>
</tbody>
</table>

Data reported as mean ± SD.

*2 to 6 years old. See Table 2 for key.
cholesterol concentrations in kittens prior to weaning with the recent ingestion of a fat-rich meal (ie, milk). In support of this, Demacker et al showed that plasma triglycerides increase in young adult cats by approximately 2-fold (from a mean of 0.23 to 0.51 mmol/L) in the 4 hours after consuming a fat-rich meal. Although this may account for the high triglyceride concentrations in kittens at week 5, it does not explain why plasma triglyceride concentrations were still high in 7-week-old kittens from which food had been withheld.

It is possible that the relatively higher plasma lipids concentrations in kittens before weaning resulted from their milk-based diet. The decrease in plasma triglyceride and cholesterol concentrations from 5 to 7 weeks of age may have reflected a decreasing reliance on maternal milk and increased consumption of the canned diet in the week or so prior to weaning. Dietary factors that potentially increase plasma triglyceride and cholesterol concentrations are fat and carbohydrate content, fatty acid composition, and protein-to-energy ratio. The canned diet was richer in fat than milk but had a similar fatty acid composition and protein-to-energy ratio (canned food, 9.0 g/100 kcal; milk, [median] 8.6 g/100 kcal). The only dietary component that could explain the higher plasma lipid concentration in suckling kittens was the higher carbohydrate (ie, lactose) concentration in milk. Other factors may also be responsible for the high plasma triglyceride concentrations in young kittens, such as a slow clearance rate, which could be assessed by use of an oral fat tolerance test as well as by measuring plasma lipoprotein lipase activity.

The relatively low cholesterol concentrations in 20-week-old kittens likely reflected increased uptake of cholesterol to meet the needs of rapid tissue growth, sexual development, and steroidogenesis. During periods of high tissue demands for cholesterol, uptake of LDL by peripheral tissues increases and transport of cholesterol via HDL from tissues to the liver decreases. Consistent with this, both LDL-C and HDL-C concentrations were low in kittens, and concentrations of cholesterol, LDL-C, and HDL-C reached a plateau in adult cats after growth and development were complete. On the basis of the present data, the upper limit of high tissue demands for cholesterol, uptake of LDL by peripheral tissues increases and transport of cholesterol via HDL from tissues to the liver decreases. Consistent with this, both LDL-C and HDL-C concentrations were low in kittens, and concentrations of cholesterol, LDL-C, and HDL-C reached a plateau in adult cats after growth and development were complete. On the basis of the present data, the upper limit was 6.1 mmol/L, compared with 8.5 and 8.6 mmol/L in adolescent and adult cats, respectively.

Plasma activities of lipoprotein lipase and hepatic lipase were significantly less in senior cats, compared with the other age groups. Lipoprotein lipase is responsible for the hydrolysis of triglycerides carried in chylomicrons and VLDL, and a deficiency of this enzyme is associated with an inability to clear plasma triglycerides after a fatty meal and persistence of hypertriglyceridemia after withholding food. In the present study, there were no significant changes in plasma lipid or lipoprotein concentrations in senior cats, compared with the other age groups, to suggest that the metabolism of triglyceride-rich lipoproteins was impaired by low enzyme activities. However, these were healthy nonobese cats fed to meet their maintenance energy requirements. It is possible that our senior cats had an impairment of chylomicron clearance, which may have been detected as high plasma triglyceride concentrations if blood samples had been collected soon after eating or if dietary fat intake was increased. In addition, further reductions in lipoprotein lipase activity in this group of cats as a result of obesity, insulin resistance, or diabetes mellitus could result in fasting hypertriglyceridemia with clinical consequences.

The clinical importance of low hepatic lipase activities in senior cats is less clear. This enzyme acts on triglycerides and phospholipids in LDL and HDL and is responsible for intravascular remodeling of these lipoproteins. Immunologic blockade of hepatic lipase in cats has been shown to increase plasma triglyceride concentrations after a meal, indicating that the enzyme plays a key role in postprandial lipoprotein metabolism. In the same study, it was shown that complete blockade of hepatic lipase activity did not affect fasting plasma lipid concentrations. This could explain why we did not detect significant changes in plasma lipid and lipoprotein concentrations associated with low hepatic lipase activity in the senior cats in this study.

In humans, hepatic lipase activities are higher in women than in men as a result of modulation by sex steroids, particularly estrogens. Hepatic lipase activities generally decrease after menopause, and it is possible that decreasing reproductive activity in senior cats in the present study was responsible for the low plasma hepatic lipase activity in this group. This possibility is supported by comparison of hepatic lipase concentrations in sexually intact females from the adult and senior groups; senior females had significantly lower hepatic lipase activity (mean, 13.6 μmol of fatty acids/ml/h), compared with adult females (20.5 μmol of fatty acids/ml/h). However, we did not detect a difference in hepatic lipase activities between sexually intact adolescent and adult females and their male counterparts. This result is similar to that reported by Demacker et al, and suggests that other factors besides estrogen may regulate hepatic lipase activity in cats.

Our finding that hepatic lipase activities were similar in sexually intact male and female adolescent cats but decreased significantly in males after castration suggests that male sex steroids play a role in regulating hepatic lipase activity. We did not investigate the effects of ovariohysterectomy, which would have helped to clarify the role of female sex steroids on hepatic lipase activity. It is clear, however, that other factors must contribute to the expression of hepatic lipase activity because of the significant differences in plasma activity of this enzyme between sexually intact adolescents and adults of both sexes.

Castration had a significant effect on plasma lipid and lipoprotein concentrations that was apparent in adult but not adolescent cats. The apparent absence of any significant effect of castration in the adolescent group may have been attributable to the relatively brief period (8 weeks) between castration and sample collection. Alternatively, it is possible that factors in the adult group other than castration, such as differences in age, adiposity, and activity level, could have accounted for the higher plasma concentrations of cholesterol, LDL-C, and HDL-C in the castrated males. This is unlikely, however, as there were no significant differences in plasma hepatic lipase activities between the two groups.
no differences in age, body weight, or activity level between sexually intact and castrated adult male cats. This impact of gonadectomy on plasma lipid and lipoprotein concentrations could well be important in adult cats; adult cats had the highest plasma lipid and lipoprotein-cholesterol concentrations of any group examined. It has been shown that castration in cats leads to weight gain, which is associated with a reduction in basal metabolic rate.\textsuperscript{21,22} Weight gain has, in turn, been shown to induce insulin resistance and impair glucose tolerance,\textsuperscript{23} which can lead to alterations in lipoprotein metabolism and hyperlipidemia.

Lechtin:cholesterol acyl transferase activities were high in kittens and adolescent cats, low in adult and senior cats, lower in male cats than in females, and decreased in adolescent males after castration. This enzyme is not generally associated with hyperlipidemia. However, cell membrane abnormalities develop in humans with LCAT deficiency because of deficient cholesterol esterification.\textsuperscript{24} Little is known regarding factors that regulate LCAT activity; it is generally assumed that rate of cholesterol esterification is related to substrate rather than LCAT concentrations.\textsuperscript{25}

High plasma LCAT activity probably reflects a high rate of plasma cholesterol esterification that is, in turn, driven by high concentrations of HDL and triglyceride-rich lipoproteins. Increased plasma cholesterol traffic in young growing animals could potentially explain the difference in LCAT activity among groups; activity was higher in kittens, compared with adolescent and adult groups. In addition, LCAT activity was less in males than females, which suggests a potential role for sex steroids in modulating LCAT activity, either directly or through an effect on cholesterol flux. This was not, however, supported by our finding that LCAT activity was decreased, rather than increased, in male adolescent cats following castration. In adult cats, castrated males had higher plasma LCAT activity than did sexually intact males, which may reflect other factors such as adiposity and activity level.

Our results indicate that lipoprotein metabolism varies in cats as a result of age and sex. These results also highlight the need to investigate further the effects of exercise and adiposity as well as ovariectomy on plasma lipoprotein metabolism. Reference limits derived from the data reported here differ in several respects from values reported by others.\textsuperscript{3} Such differences may have a dietary component as well as reflect variation in type or breed of cats, age, sex, adiposity, and activity. Elucidation of the contribution of each of these factors to variations in plasma lipid concentrations is required to fully understand apparent abnormalities in lipid metabolism in cats.

\textsuperscript{3}WHISKAS feline concentration diet (canned), Masterfoods, Bruck, Austria.
\textsuperscript{4}Department of Pathological Biochemistry, Royal Infirmary, Glasgow, G4 0SF, UK.
\textsuperscript{5}Boehringer Mannheim UK, Lewes, UK.

References