Lipoproteins form the most complex blood plasma macromolecules. This attribute of lipoproteins is the main reason that our knowledge of this biological system is still fragmentary in spite of great interest shown and progress made in lipoprotein investigations. Numerous investigations have been reported on composition, structure, metabolism and function of lipoproteins during the last three decades and are summarized in a number of reviews of which three adequately represent these studies (Tria and Scanu, 1969; Nelson, 1972; Scanu and Landsberger, 1980). The presence of lipids in clear solution in blood plasma of a normal lipemic mammal at the concentration of 200-900 mg/100 ml is made possible by their unique association in water-soluble complex proteins, the lipoproteins. Lipoproteins form a broad spectrum of macromolecules and micells which vary considerably in size, from 6000 Å in chylomicrons to an average of 86 Å in high density lipoproteins, and molecular weights from billions to 200,000 respectively. The amount and quality of lipids vary in lipoprotein molecules, whereas the protein entity, defined as apolipoprotein, seems to remain qualitatively constant. Depending on the relative proportions of lipid and protein in lipoproteins, from 98%:2% to 3%:97%, their hydrated densities vary from d 0.92 g/ml in chylomicrons to 1.30 g/ml in very high density lipoproteins, respectively. In short, lipoproteins form very dynamic, complex and heterogenous groups of blood plasma macromolecules with respect to metabolism, composition, size, hydrated density and immunogenicity. The primary physiological functions of lipoproteins have been defined as lipid transport proteins, and the regulation of lipid metabolism. More recent and incomplete reports seem to indicate that lipoproteins may be involved in other physiological processes as regulatory proteins controlling the synthesis and metabolism of certain biological compounds.

Two nomenclatures, physical and chemical (Alaupovic, 1978), are customarily used for classification of lipoproteins. The most commonly used is the physical nomenclature which subdivides lipoproteins into classes on the basis of their hydrated densities and electrophoretic mobilities (Havel et al., 1955; Kunkel and Slater, 1952). Using these methods, lipoproteins present in the blood plasma or sera are divided into three major classes: high density lipoproteins (HDL), d 1.063-1.2 g/ml, or α-lipoproteins (migrating to the α-position on paper electrophoresis); low density lipoproteins (LDL), d 1.006-1.063 g/ml, or β-lipoproteins; very low density lipoproteins (VLDL), d 0.94-1.006 g/ml, or pre-β-lipoproteins. The fourth class of lipoproteins, chylomicrons, d<0.94 g/ml, is not present in the fasted sera and is composed of very large particles which do not migrate in gel or paper electrophoresis. Chylomicrons are of intestinal origin and appear in the blood primarily after ingestion of dietary fat (Zilversmit, 1965). There is an additional minor class composed of very high density lipoproteins (VHDL), which is represented by molecules which do not float at d 1.21 g/ml. Numerous immunochemical and structural studies have presented evidence that lipids are not randomly distributed but rather associated with specific
apolipoproteins. With this discovery, it became apparent that apolipoproteins may prove to be important determinants of lipoproteins in their composition, physical and chemical properties, structure and biochemical and physiological function (Shore and Shore, 1972). To reconcile the two most widely used operational criteria (Nelson, 1972) with the new finding, Alaupovic and coworkers (Alaupovic et al., 1972) postulated that the lipoprotein system is composed of distinct lipoprotein families (LP). A lipoprotein family is defined as a polydisperse system of lipoprotein complexes characterized by the presence of a specific apolipoprotein, or its constituent polypeptides of a distinct isoantigenic specificity. There are at least ten recognized apolipoprotein families described in man (Alaupovic, 1978).

Although the importance of knowledge of lipoproteins is apparent, their structure, complexity, interaction and mechanism of action are the least understood of all blood serum proteins. In addition to complexity and heterogeneity, the characterization of lipoproteins is complicated further by variations observed between individual sera. These variations may result from genetic differences encoded in the apolipoprotein structure observed as polymorphic forms. Genetics lies at the base of the structure of all molecular systems and through them relates to fundamental physiological processes and functions. Therefore, genetic identification, characterization and elucidation of existing polymorphic forms of lipoproteins, and how they relate to physiological functions and interact with other molecular systems is of great significance. The most recent and extensive review on animal lipoproteins, however, has not surveyed reports on the genetic aspect of lipoproteins (Chapman, 1980). Our survey shows a relatively small number of genetic studies on lipoproteins. All of these reports are limited to immunogenetic studies, with a single exception of the E-lipoprotein in humans (Uterman et al., 1979). Two properties, immunogenicity and antigenicity of lipoproteins, are well known attributes. Immunological methods are used in studies of isotypic differences of lipoproteins, or their apoproteins; however, this great potential has barely been explored for investigation of genetic polymorphisms. This small number of studies on genetic polymorphism may stem from a lack of interest, in general, in genetic characterization of lipoproteins by the great majority of investigators engaged in studies of lipoproteins.

The original discovery of the lipoprotein polymorphism resulted from a search by Allison and Blumberg (1961) for alloprecipitins against any of the blood plasma proteins in sera of polytransfused patients. Later, lipoprotein polymorphisms were found in the rabbit, rhesus monkey, sheep, cattle, fish and swine (for review see Rapacz, 1978), and more recently in mink (Baranov et al., 1978) and chicken (Pesti et al., 1981). The most antigenic and polymorphic appears to be the LDL class, and specifically the LP-B family, or β-lipoproteins. Therefore, it is not surprising that the majority of the studies on lipoproteins in domestic animals have reported polymorphisms in this class.

Rabbits: In attempt to produce anti-immunoglobulin gamma (IgG) antibodies Kelus (1968) obtained alloprecipitins identifying the first lipoprotein allotype in rabbits, designated Lpl. Preliminary studies showed that the allotype migrated as human β-lipoproteins in the electrophoretic field and was inherited in a simple Medelian manner. Albers and Dray (1968, 1969) have identified four LDL markers: Lpq1, Lpq2, Lpq3 and Lpq4. Genetic studies seemed to indicate that the four antigens are controlled by three allelic genes: q.3, q.2.3 and q.2.4. Recently Rapacz (Rapacz, junior, in press) identified a rabbit lipoprotein allotype designated Lrbl (L-lipoprotein, r-rabbit, b-locus for β-apolipoprotein, 1-number of allele) which migrates as β-lipoproteins and is determined by an autosomal dominant gene, but has a distinctly different density distribution, d 1.002-1.045 g/ml, from those LDL allotypes (d 1.006-1.063 g/ml) reported by Albers and
Dray. Four allotypes, associated with the HDL class, identified by alloimmune sera and designated Hl-1, R67, Lhj-1 and Lhj-2 have been reported by Berg (1971), Boman et al., (1972) and Gilman-Sachs and Knight (1972), respectively. The alloantigens are determined by autosomal, dominant genes (Hl-1 and R67), and autosomal codominant alleles (Lhj-1 and Lhj-2). Loci for R67 and Hl-1 are not linked. The Lhj-1 and Lhj-2 showed α-1-globulin mobility in agar electrophoresis, and stained with β-naphtyl-acetate.

Sheep: An allotypic marker identified by alloprecipitins, designated Lps1, associated with lipoproteins of d 1.006-1.070 g/ml, migrating as α-2-globulin in agar gel, or as β-globulin on paper electrophoresis has been described in sheep by Rapacz et al., (1972). Genetic investigations revealed that Lps1 is coded by an autosomal dominant gene exhibiting variation in the gene frequency among five breeds tested.

Cattle: Injecting ammonium sulfate precipitate of cattle serum an alloprecipitin was obtained, which identified a lipoprotein antigen, designated Lpcl (Rapacz and Hasler, 1970). The allotype exhibited poor antigenicity and seemed to be controlled by an autosomal dominant gene. Wegrzyn (1973) obtained an alloimmune serum which identified a lipoprotein allotype, designated BA-1, determined by an autosomal dominant gene which migrated in agar gel immunoelectrophoresis as α-2-globulin. Iannelli et al., (1978) described another cattle lipoprotein allotype, LdlAl, associated with the LDL class.

Fish: A lipoprotein antigen, Lpfl, was identified by a heteroimmune reagent in sera of sexually mature carps (Rapacz et al., 1971). Its level in female sera exceeded many folds the level in males. The time of appearance and development of this allotype and another alloantigen, Ufl, in the carp, and the Sm allotype in salmon were correlated and labeled as "female factors", which seem to be associated with female sex hormones.

Mink: Seven mink serum allotypes, Lpm-1, -2, -3, -4, -5, -7 and Lpm-8 associated with the VHDL class (Baranov et al., 1978) and one, Ldl, found in fractions of d 1.006-1.1 g/ml (Baranov and Savina, 1979) have been described using alloimmune sera produced against normal mink sera. The seven VLDL allotypes were reported to be controlled by the following eight "genetic units": Lpm8, Lpm4, Lpm4,8, Lpm4,7, Lpm3,4,8, Lpm1,8, Lpm1,2,7 and Lpm2,4,5,7 which seemed to behave as alleles. The Ldl allotype which exhibits esterase activity is sensitive to storage and thawing and is controlled by an autosomal dominant gene. Both the Ldl and Lpm allotypes migrate as α-2-globulins in agar gel electrophoresis.

Chicken: Two lipoprotein allotypes, designated Lcpl and Lcp2, have been described in the chicken (Pesti et al., 1981) using alloprecipitin reagents. Originally, the alloantigens were classified as LDL markers on the basis of their distributions in the preparative ultracentrifuge fractions derived from plasma of males, or sexually immature birds of both sexes. Later, each blood plasma obtained from sexually maturing pullets, laying and non-laying hens showed the Lcp allotypes being present in the LDL and VLDL classes. The shift was gradual reaching a peak, with 100% of Lcp in VLDDL, a few days prior to laying of the first egg. With the exception of plasma from genetically controlled restricter ovulators in which the distribution-shift became permanent, the reversal process, expressed by the increase level of Lcp in LDL and the decrease in VLDDL, followed as hens progressed with laying. Parallel to the change in the Lcp class distribution, plasma lipids (triglyceride and cholesterol), Lcp allotypes and β-lipoprotein levels started to increase and rose at a considerable rate reaching their peaks prior to laying.

Further developmental studies (Pesti et al., in press) revealed that sexual maturity in male was not accompanied by the changes observed in the females. Investigations on plasma samples collected from younger chickens, as
early as 11 days of incubation until 100 days of age, have shown identical pat­
terns in the Lcp lipoprotein levels in both sexes; marked elevations were ob­
served during the last period of incubation and at hatching, with a gradual de­
crease, by 25-40%, reaching stable levels within three weeks of age. The or­
igin of the increased level of lipoproteins during the incubation and at hatch­
ing was investigated by testing sera of birds from specific Lcp matings. The results have shown that the elevated level of Lcp lipoproteins were not derived from the egg yolk, but were synthesized by the embryo. Segregation data from the Lcp1, 2 x Lcp0 type of mating indicated that Lcp allotypes are coded by allelic, or closely linked autosomal codominant genes. Serological studies, suggested that the Lcp2 antigen behaves as a "common specificity", which most likely is coded by more than one Lcp gene. These observations resembled those early data on swine lipoproteins (Rapacz et al., 1970) suggesting a more com­
plex genetic code and molecular form for the Lcp lipoproteins.

This resemblance has been confirmed further by showing that the Lcp lipo­
proteins in chicken, Lpp in swine, Lsp in sheep and Lmp in rhesus monkey repre­
sent homologous and the main lipoprotein in the LDL class - LP-B or β-lipopro­
tein. Therefore, to unify the nomenclature we have introduced a substitution, from "p" to "b", for the designation of the β locus in these four species from 1) Lcp to Lcb in chickens, 2) Lpp to Lpb in swine (Rapacz et al., 1976, Rapacz et al., 1978 and Rapacz, 1978), 3) Lsp to Lsb in sheep (Rapacz et al., 1972) and 4) Lmp to Lmb in rhesus monkey (Rapacz, 1978). Further preliminary investi­
gations on the Lcb polymorphism led to the identification of three new allo­
types, Lcb2, Lcb3, Lcb11. With this discovery there are now five Lcb allotypes, Lcb1, Lcb2, Lcb3, Lcb11 and Lcb12 (former Lcp2). Preliminary segregation data indicate that three Lcb genes: Lcb1, Lcb2 and Lcb3 control three complex alleles of Lcb1, Lcb2,11 and Lcb3,11,12; respectively. Plasma samples derived from 23 non-inbreeding lines, which originated from four breeds indi­
cate considerable variations in the frequency of the Lcb genes and can be sum­
marized as follows: all birds homozygous for; Lcb1 - six lines; Lcb2 - one line; Lcb3 - one line. Of the remaining 15 lines, five lines exhibited Lcb1 and Lcb2, three Lcb1 and Lcb3, one Lcb2 and Lcb3, and six lines carried Lcb1, Lcb2 and Lcb3 alleles. These studies are viewed as preliminary with regard to the number of alleles determining the Lcb polymorphism.

Swine: Studies on swine lipoproteins in this laboratory revealed large polymorphism in this species (Rapacz et al., 1970; 1972; Rapacz, 1974; Rapacz et al., 1976; 1978; Rapacz, 1978; Rapacz, Hasler-Rapacz, 1980). Using prepara­tive ultracentrifuge fractions of different densities, d 1.002-1.09 g/ml, 354 pigs were inoculated during the last 12 years. Alloprecipitins were identified in the immune sera of 241 recipients, using the immunodiffusion technique in agar gel. Further analysis of the immune sera showed that 117 of them were use­
ful for preparation of specific anti-swine lipoprotein reagents. By the use of these reagents, 35 patterns of precipitation reactions have been established. The majority of normal swine sera originated from four breeds and their crosses: Chester Whites, Hampshires, Poland Chinas and Yorkshires. In addition, 33 dif­
ferent groups or breeds of pigs (5-82 pigs per group or breed) have been tested with some of these reagents.

Segregation data indicated that each of the 35 patterns corresponded to a heritable lipoprotein marker, and each allotype seemed to be controlled by an autosomal codominant, or dominant gene. Genes controlling 28 of these allo­
types were assigned to five lipoprotein loci, b, r, s, t and u giving rise to five lipoprotein systems: Lpb, Lpr, Lps, Lpt and Lpu (Table 1). More detail data, although still preliminary, were reported on 11 Lpb allotypes. These studies, together with advanced investigations on Lpb, revealed at least eight autosomal codominant complex genes each coding for a very complex phenogroup.
<table>
<thead>
<tr>
<th>Temporary designation of systems</th>
<th>Number of alleles identified</th>
<th>Status of the systems</th>
<th>Linkage with other systems</th>
<th>Number of allotypic markers</th>
<th>Main lipoprotein - class distribution</th>
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<tr>
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<td>$b, t$</td>
<td>2</td>
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</tbody>
</table>

*L - lipoprotein, p - pig, b - locus with genes coding for $\beta$-lipoprotein markers (allotypes)
The allotypic markers in each phenogroup are divided into "individual" (Lpb1-Lpb8), and "common specificities" (Lpb11-Lpb18). The individual markers, assumed to arise as a result of single mutations, are unique entities restricted to one allele, phenogroup, or haplotype, whereas the common specificities occur in the alternative phenogroups forming together pairs of mutually exclusive specificities in the Lpb allelomorphs; e.g., Lpb2-Lpb12. An individual complex Lpb-code seems to control at least eight different Lpb alloantigens located in a single allelomorphic molecule as demonstrated by a specific serological test for molecular expression of Lpb genes using sera of Lpb homozygous and heterozygous pigs (Rapacz et al., 1976; 1978; Rapacz, 1978). Analysis of the progeny test involving 9,000 offspring, has not revealed a single irregularity in the transmission of the Lpb genetic information.

As a result of these studies on the Lpb system, the first immunogenetic model has been proposed (Figure 1). The current concept, based on immunogenetic evidence for Lpb lipoproteins in swine, assumes that the genetic code determining alloantigens of the β-apolipoprotein occupies a very complex locus, which evolved through individual mutations, each resulting in a replacement of the information for a common alloantigen by the information for an individual alloype. Available data on β-lipoproteins in man, rabbit, rhesus monkey and chickens do not exclude a similar hypothesis for the origin of the LP-B polymorphism in these species.

Analysis of the 37 breeds or groups of pigs tested have shown that the Lpb genes are not randomly distributed in this species but the various breeds have a characteristic complement of these genes. Preliminary report by Hojny and Duniec (1980), and the results of the first International Standardization Test on swine allotypes (Rapacz, in press; Animal Blood Groups and Biochemical Genetics) indicate the finding of an additional gene, Lpb9, in this system. The standardization test indicated that three, Ll, L2, and L5, of four swine lipoprotein antigens reported by Duniec et al., (1976) correspond in the reaction

### Table 1

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<tr>
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<tr>
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<td>1</td>
<td>LDL</td>
</tr>
<tr>
<td>Lpt</td>
<td>2</td>
<td>open</td>
<td>b, u</td>
<td>2</td>
<td>VLDL, LDL, HDL</td>
</tr>
<tr>
<td>Lpu</td>
<td>2</td>
<td>closed</td>
<td>b, t</td>
<td>2</td>
<td>LDL</td>
</tr>
</tbody>
</table>

*L - lipoprotein, p - pig, b - locus with genes coding for β-lipoprotein markers (allootypes)*
Figure I
IMMUNOGENETIC MODEL OF THE Lpb SYSTEM IN SWINE.
(a) Eight allelic genes (1-8) of the Lpb chromosomal region.
(b) Schematic representation of eight Lpb haplotypes (phenogroups) with their alloantigenic determinants (alloantigic specificities) designated by numerals. Each allele of the Lpb region determines a complex antigenic product bearing a set of distinct alloantigic specificities.

One of the best known functions of β-lipoproteins is transport of cholesterol. A recent study by Epstein et al., (1981) supports previous allegations that LP-B lipoproteins are directly involved in transport of sterols. Preliminary biomedical and nutritional studies on swine susceptibility or resistance to atherosclerosis have already implicated two Lpb genes; Lpb\(^3\) and Lpb\(^5\). All pigs which have been found susceptible to the development of aortic intimal lipidosis and fibrosis carried the Lpb\(^5\) gene (Rapacz, 1978), whereas all Lpb\(^3/3\) pigs were found the most resistant to elevation of cholesterol and Lpb lipoproteins in response to the atherogenic diet (Rapacz et al., 1977). Extended studies on pigs fed a standard low fat diet (fat<4%) confirmed previous observations that all pigs with high susceptibility to arterial lipidosis carry the Lpb\(^5\) gene, or its mutant, Lpb\(^5\)\(^b\), but not all pigs with these alleles are susceptible to the disease (Rapacz et al., unpublished). The degree of the susceptibility is strongly correlated with the Lpb and cholesterol levels, which seem to be heritable and can be detected at birth.

Although studies on the other four lipoprotein systems (Table I) are very preliminary, it was possible to establish linkage between Lpb and two other systems, Lpt and Lpu. It seems interesting that the two implicated genes, Lpb\(^3\) and Lpb\(^5\) exhibit unique linkages with the Lpt and Lpu systems (Rapacz, 1978). This suggests that interaction between products of a specific Lpb-Lpt-Lpu haplotype may play an important role in lipid transport, metabolism and physiopathology. In addition, indirect observations suggest that Lpb and cholesterol are important contributors to the growth of young pigs; all runt pigs tested exhibited abnormally high Lpb and cholesterol levels, which may be interpreted as a lack of ability for utilization of these biological compounds. Studies underway show marked variations in the level of Lpb lipoproteins with regard to sex and age. Females exhibited higher Lpb levels than males, with variations during the estrus cycle and pregnancy; a marked decrease was observed in both sexes with the cessation of sexual activity in older pigs. Pigs are born exhibiting a low level of Lpb lipoproteins, which increase rapidly (+ 110 mg to + 360 mg/100 ml) until 16-24 days of age, and then decrease gradually reaching
adult levels (120–280 mg/100 ml between 6–8 months of age. There are two allelic genes, Lpu1 and Lpu2, identified in the Lpu system (Table 1). The Lpu2 allele is very common, whereas Lpu1 is a rare allele and was found only on the chromosome with the Lpb3 gene; however, only 20% of Lpb3 genes are associated with Lpu1. Two allotypes, Lpt1 and Lpt2, determined by two codominant genes, were identified in the Lpt system. Chromosomes carrying all Lpb genes but Lpb3 exhibit the Lpt1 gene, whereas Lpt2 is associated with all Lpb chromosomes except with the Lpb1 and Lpb6 genes. Only a single marker, Lps1, has been identified so far in the Lps system.

The remaining lipoprotein system, Lpr, is characterized by two allotypes, Lpr1 and Lpr2; controlled by two autosomal dominant genes, Lpr1 and Lpr2. Three of the four breeds studied, Hampshires, Poland China and Yorkshires have shown the Lpr1 gene frequency below the 0.02 level, whereas the remaining breed, Chester White, showed 0.18, all pigs being Lpb5/5. Further characterization studies on Lpr lipoproteins using sera and mesenteric lymph samples brought already interesting observations. The results of these observations can be summarized as follows: Lpr lipoproteins have a distinct physiological function—the transport of dietary lipids via the lymphatic system. Their origin of synthesis is not the liver as has been assumed for all lipoproteins, but the small intestine. During dietary lipid absorption, Lpr lipoproteins are entirely associated with chylomicrons. In the post absorptive state Lpr can be found in VLDL or in a class of smaller molecules after fasting. Change in density distribution of Lpr can be easily manipulated by the lipid content in the diet. In the blood, Lpr lipoproteins undergo rapid transformation, through all lipoprotein classes, and are found mainly in the VHDL class. With the exception of pigs with high Lpb and cholesterol levels, Lpr lipoproteins are absent in the serum at birth and develop gradually to the adult level during the first six months of life. The high level of Lpb lipoproteins in pigs of Lpb5 Lpr1 phenotype was found associated with a high level of Lpr lipoproteins.

It is clear from this presentation that the genetic knowledge of lipoproteins in domestic animals is very fragmentary and limited to the immunogenetic approach. Nevertheless, it seems appropriate to underline the significance and contribution of these investigations to a better understanding of the lipoprotein diversity and specificities which may represent essential components for consideration in studies of specific physiological roles of lipoproteins.

SUMMARY

Blood plasma lipoproteins in all animal species form very dynamic complex and heterogenous groups of macromolecules with respect to metabolism, composition, size, density and immunogenicity. These macromolecules, composed mainly of proteins and lipids in various ratios, are characterized by a specific protein moiety, apolipoprotein, which exhibits a distinct property to bind, solubilize and transport all lipids including sterols and fat soluble vitamins, and furthermore to regulate and interact with specific enzymes. Although there is great interest in elucidation of lipoprotein structure, composition and biological functions, investigations of the genetic aspects of lipoproteins, which underline a structure-function relationship, are so far limited to immunogenetic studies. Using this approach large genetic polymorphisms have been revealed in various families and classes of lipoproteins, especially low-density lipoproteins (LDL). This presentation reviews immunogenetic findings on lipoproteins in the following domestic animals; rabbits, cattle, sheep, swine, chickens, mink and fish, and discusses some new findings in swine and chicken lipoproteins.
ZUSAMMENFASSUNG


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