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Pig genetics : a review[1]

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Summary

A general review on pig genetics is given. The main topics covered are : karyotype and chromosomal abnormalities, coat colour, hereditary defects and disorders, disease resistance, immunogenetics, biochemical polymorphisms, linkages and syntenies, and quantitative traits of economic importance. As regards the latter field, special emphasis is put on the known influences of marker genes on performance traits. Lists of genes controlling coat colour, blood groups (erythrocyte antigens), and electrophoretic variants of blood serum proteins and blood cell enzymes are tabulated. A list of 29 genetic abnormalities which can be considered as probably monogenic in the pig is also given. The total number of presently identified loci is around 100.

I. Introduction

Domestication of the pig dates back to the Neolithic age and, recently, remains of domestic pigs dated 7 000 B.C. have also been found in China. Pig was considered as sacred in the ancient world and it still is in some countries like New-Guinea. Pigs have also been used in England to point for game and in France to root for truffles. Since modern times the pig has mainly been used to produce lard, fresh meat (pork) or various kinds of processed meat (bacon, ham, etc.). The increased demand for meat has seen the recent development of breeds with high lean meat content. Pig is also nowadays used as a laboratory animal in biomedical research (POND & HOUPT, 1978), owing to its close physiological similarities to man, and several strains of miniature pigs have been developed for that purpose. The total world pig population was estimated to nearly eight hundred millions heads in 1981. Asia produces the largest number followed by Europe, North America, the U.S.S.R., South America, Africa and Oceania. The number of breeds or local varieties exceeds 300 : see MASON (1969).

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(1) This review was originally intended as a chapter of the *Handbook of Mammalian Genetics* to be published by Garland Publishing, New York. We are grateful to the Editor (Dr Roy Robinson, Ealing, U.K.) for his permission to publish a revised version of the chapter separately, in view of the delay in the preparation of the book.

Cette revue bibliographique devait constituer un chapitre du livre Handbook of Mammalian Genetics *(éditeur : Garland Publishing, New York). Par suite des retards survenus dans la préparation de ce livre, l'Editeur scientifique (Dr Robinson, Ealing, G.B.) nous a autorisés à publier séparément une nouvelle version du texte. Nous lui en exprimons ici notre reconnaissance.*

The present-day domestic pig (*Sus scrofa var. domestica*) has evolved from the European wild boar (*Sus scrofa*) that was crossed with the Chinese pig (*Sus vittatus*). The species is highly prolific. With a gestation length of 114 days and a litter size of 8-12 piglets, a sow is able to produce regularly around 20 slaughter pigs per year from the age of one year up to that of 6-7 years in the most favourable cases.

TABLE 1

A list of reviews on pig genetics (not including the reviews on quantitative performance traits).

Liste de revues bibliographiques sur la génétique du Porc (caractères quantitatifs exclus).

Year	Authors	Coverage
1918	WRIGHT	Colour
1931	KOSSWIG and OSSENT	Colour
1936	SCHMIDT and LAUPRECHT	Colour
1938	SMITH, ROBINSON and BRYANT	General
1940	CASTLE	Colour
1945	HETZER	Colour
1957	KOCH, FISCHER and SCHUMANN	Abnormalities
1959	HANSET	Colour
1961	BERGE	Colour
1962	ANDRESEN	Blood groups
1964	JOHANSSON	Abnormalities
1968	BUSCHMANN and SCHMID	Serum proteins
1968	SEARLE	Colour
1969	HULOT	Chromosomes
1971	LARSEN	Blood groups and polymorphic proteins
1974	WIESNER and WILLER	Abnormalities
1975	RASMUSEN	Blood groups
1975	MCDERMID, AGAR and CHAI	Red cell enzymes
1975	WIDAR, ANSAY and HANSET	Enzymes
1977	IVANYI	Histocompatibility
1978	HUSTON, SAPERSTEIN, SCHONEWEIS and LEIPOLD	Abnormalities
1978	AGERGAARD and NIELSEN	Blood groups
1978	HYLDGAARD-JENSEN and JØRGENSEN	Serum proteins and red cell enzymes
1979	GAHNE	Immunogenetics and biochemical genetics
1979	OISHI	Immunogenetics and biochemical genetics
1981	FECHHEIMER	Cytogenetics
1981	HRUBAN	Immunogenetics

As indicated in table 1, several reviews on pig genetics have already been published, many devoted to coat colour. A very detailed review of genetic abnormalities has been published by KOCH, FISCHER & SCHUMANN (1957), to which we shall frequently refer in this article. A number of reviews have recently been devoted to the newer fields of immunogenetics and biochemical polymorphism. No general review on pig genetics appears to have been published since *The genetics of the pig* by SMITH, ROBINSON & BRYANT (1938).

II. Karyotype

Up to 1960, the pig chromosome number was an object of controversy. SMITH *et al.* (1938) however accepted 2n = 38 as a working hypothesis, a number on which there is now complete agreement, as indicated by HULOT (1969), to whom we owe a detailed historical review of techniques and chromosome number estimations between 1913 and 1968. But there has been some confusion on the description and arrangement of chromosomes even after the advent of the « banding » techniques in 1970, which, in principle, permit a precise identification of each chromosome. An up-to-date description and identification of the pig chromosomes is given by HANSEN (1980) and LIN *et al.* (1980), on the basis of relative length and several banding patterns.

Two polymorphisms in C-banding pattern have recently been reported in the pig by CHRISTENSEN & SMEDEGÅRD (1978, 1979) : these variants are inherited in a regular Mendelian way, as most of similar variants in humans. Chromosomal polymorphism appears to be a widespread phenomenon in pigs (HANSEN-MELANDER & MELANDER, 1974 ; VEIJALAINEN & RIMAILA-PÄRNÄNEN, 1978 ; SYSA, 1980).

The chromosome complement of some european wild pigs, with 36 chromosomes, includes a submetacentric chromosome which is considered as homologous to two telocentric chromosomes of the domestic pig, these two chromosomes being involved in a Robertsonian translocation (e.g. MC FEE *et al.*, 1966 ; POPESCU *et al.*, 1980). MC FEE & BANNER (1969) have shown that this translocation behaves as a simple Mendelian character, without any incidence on fertility or visible traits. In the Netherlands, a particular strain of wild pigs has been shown to carry three different chromosome numbers, 36, 37 and 38, which would indicate the existence of a balanced chromosomal polymorphism, similar to that found in other mammalian species (BOSMA, 1976). BOSMA suggests that the original chromosomal number in the wild boar was indeed 38 and has been reduced to 36 in modern wild populations as a consequence of a centric fusion of two telocentrics into one submetacentric. In fact, two types of such a fusion, involving three different telocentric chromosomes, have been shown to occur in wild boars of the U.S.S.R. (TIKHONOV & TROSHINA, 1975).

Pig chromosomal abnormalities have been reviewed by HULOT (1969) and, more recently, by GUSTAVSSON (1980), FECHHEIMER (1981) and POPESCU (1982). Most studies have been done in connection with reproductive abnormalities and they will be reviewed below in the corresponding section.

III. Coat colour

A. *Description of the main coloured types and breeds*

1. *Wild type*

The wild colour, similar to agouti in rodents, is characterized by a yellow subterminal band on an otherwise dark dorsal hair and also often by a variable colour intensity according to the body region. A particularity of the wild pig is that piglets at birth exhibit longitudinal stripes which gradually disappear later in life. This may also occur in some domestic breeds (e.g. *Mangalitza*).

2. *Uniform black*

This type is exemplified by the English *Large Black* breed. It was also found in several local breeds which have by now disappeared, such as the German *Cornwall*, the French *Gascon* and a variety of the *Mangalitza* breed.

3. *Uniform red*

Two breeds show this colour, *Duroc-Jersey* and *Tamworth*, and also the new American breed *Minnesota n° 1*. A red variety has also been reported in *Mangalitza*.

4. *Black spotting*

Black spots generally occur on a white background, but red hair in a variable amount may be mixed with white, up to a uniformly red background. Such a pattern is found in *Spotted Poland-China, Gloucester Old Spot, Piétrain* and *Bayeux*.

5. *Black with white points*

The coat is uniformly black with the exception of six white points (feet, tail and snout) in *Berkshire* and *Poland-China*.

6. *Belt*

Several breeds show a white belt of variable width and generally centered on the shoulders, against a background which is black in the following breeds, *Wessex, Essex, Hampshire, Hannover-Braunschweig, Basque* and *Limousin*, and red in *Bavarian Landschwein*, also called *half-coloured* as most of the anterior part of the body is white.

7. *White*

Two types of white coat exist, a *shiny white* with usually a white skin as in *Large White (Yorkshire), Middle White, Chester White, Lacombe, Blanc de l'Ouest* and the *Landraces* of various european countries, and a *« dirty » white* on a pigmented skin as in *Mangalitza*.

The asiatic breeds of pigs, as described by EPSTEIN (1969), also appear to fall in the same categories. Rarer types of colour have occasionally been reported and may be worth mentioning : *sepia hair, sepia coat* which is a mixture of white, dark and banded hair, and *roan*, a mixture of white and black hair. The *blue* colour, which consists of white hair on a black skin, has also sometimes been referred to as roan.

B. *Genetics of coat colour*

Table 2 summarizes the experimental work pertaining to the previously described main colour types in pigs and it shows that almost all possible combinations have been investigated in crossbreeding experiments. These, unfortunately, were not usually designed for studying colour inheritance, and not carried on long enough nor on a sufficient number of animals to allow definite conclusions to be reached. Therefore several points still remain obscure in pig coat colour inheritance. The first overall

TABLE 2

Combinations of coat colour types investigated in crossbreeding experiments.

Combinaisons de patrons de coloration étudiées dans des expériences de croisement.

Type	Uniform black	Uniform red	Black spotting	Black with white points	Belt	White	
						Dirty	Shiny
Breed example	*Large Black*	*Duroc-Jersey*	*Piétrain*	*Berkshire*	*Hampshire*	*Mangalitza*	*Large White*
Wild		SMITH *et al.* (1938)*		SMITH *et al.* (1938)	SMITH *et al.* (1938)		DECHAMBRE (1925, 1929), SMITH *et al.* (1938)
Uniform black		SMITH *et al.* (1938)		SMITH *et al.* (1938)		CONSTANTINESCU (1934), SMITH *et al.* (1938)	SMITH *et al.* (1938), HETZER (1945d), GLIGOR *et al.* (1959, 1960)
Uniform red				SMITH *et al.* (1938)	SMITH *et al.* (1938), BUSHNELL (1943)	SMITH *et al.* (1938)	SMITH *et al.* (1938), DRY and COOPER (1945), HETZER (1946, 1947)
Black spotting					DONALD (1951), MILOJIC (1966)		LAUVERGNE and OLLIVIER (1966), MILOJIC (1966)
Black with white points							SMITH *et al.* (1938), HETZER (1945b, 1945c)
Belt							SMITH *et al.* (1938), HETZER (1948), DONALD (1951)
Dirty white							SMITH *et al.* (1938), CONSTANTINESCU (1935)

* The reference is SMITH *et al.* (1938) for the experiments reviewed by those authors.

view of the subject was given by WRIGHT (1918). Early conclusions reached in America by WENTWORTH & LUSH (1923) and in Germany by KOSSWIG & OSSENT (1931, 1932, 1934) and by KRONACHER & OGRIZEK (1932) have been summarized by SMITH *et al.* (1938). But the major body of experimental data so far remains the series of papers by HETZER, published from 1945 to 1954. SEARLE (1968) discussed possible homologies between the colour genes of the pig and those of other mammalian species and we shall follow his order of presentation (see table 3 for summary).

TABLE 3

A list of coat colour genes in the pig, adapted from BERGE (1961) and SEARLE (1968).

Liste de gènes de coloration de la robe chez le Porc, adaptée de BERGE (1961) et SEARLE (1968).

Loci	Alleles
A	A^w (agouti white belly) (1) A^b (agouti badger face) a (non agouti) (1) a^s (sepia)
C	C (normal) (1) c^e (extreme dilution = dirty white)
D	D (normal) (1) d^s (sepia)
E	E^d (dominant black) E (normal) E^p (partial extension) e (red) (1) e^h (white face)
R	R (normal) r (red-eye)
I	I (inhibition of colour) I^d (roan) i (coloured) (1) i^m (dirty white)
Be	Be (belt) be (self) (1) be^b (half-coloured and possibly white face)

(1) Allelism not proven.

1. *Wild colour (Agouti locus)*

From crosses reported by SPILLMAN (1906), FRÖHLICH (1913), WENTWORTH & LUSH (1923) and KOSSWIG & OSSENT (1931), wild colour appears to be dominant to the *Berkshire* black, but recessive to *Hannover-Braunschweig* black and to white. However, the segregation results observed in crosses between wild and red or black breeds are complex and various hypotheses had to be put forward by KOSSWIG & OSSENT (1931) in order to explain them. This is a consequence of epistatic relations, not yet well understood, between the agouti locus (A) and the other colour loci.

It can reasonably be assumed, with BERGE (1961) and SEARLE (1968), that most domestic breeds carry the recessive non-agouti (*a*) allele, though the wild allele (*A*) may be present in some red breeds as the light-belly agouti pattern has been shown by LUSH (1921) to appear in an F_2 *Berkshire* × *Duroc-Jersey*. The *Mangalitza* breed is another exception in that respect, for wild type F_1 piglets may appear when *Mangalitza* is crossed with various other domestic breeds (OSSENT, 1929 ; KOSSWIG & OSSENT, 1931 ; CONSTANTINESCU, 1933 ; TEODOREANU, 1935). In view of this, KOSSWIG & OSSENT (1931) assume that in *Mangalitza* the *A* allele, which would be responsible for the juvenile striping pattern, is present at a high frequency. This is not accepted by CONSTANTINESCU (1933), TEODOREANU (1935) and HETZER (1945 a), who consider that the juvenile stripes, a constant characteristic of *Mangalitza*, and the wild pattern are determined by genes at two different loci. On the other hand, the presence of juvenile stripes in white breeds, often visible as a « ghost pattern » (DECHAMBRE, 1925), suggests that hair structure rather than pigmentation is involved in the phenomenon. Comparing wild coat colour between Papua New Guinea village pigs and the French wild boar, LAUVERGNE *et al.* (1982) think that another agouti pattern may exist besides the light-belly agouti of the previous authors : a kind of badger face pattern, with black belly and snout. The symbol they propose for light-belly agouti is A^w (*w* for white) and they prove that this allele is dominant on *a*. The tentative symbol for the badger face variant could be A^b (*b* for *badger face*).

2. *Brown and Albinism* (B *and* C *loci*)

No members of these two series were reported until recently, but according to LAUVERGNE *et al.* (1982) a brown variant of eumelanin could exist in Papua New Guinea village pigs. Albinism is unknown in the pig, but SEARLE (1968) considers the dirty white colour of *Mangalitza* as possibly due to an allele of the C series, homologous with extreme dilution c^e in other mammals.

3. *Dilution* (D *locus)*

A likely member of this series is, according to SEARLE (1968), the recessive sepia factor observed by MC PHEE *et al.* (1931), who describe this colour as due to a partial dilution of the black pigment and to a mixture of white, pigmented and banded hair. BERGE (1961) attributes it to an allele (a^s) of the agouti locus.

4. *Extension* (E *locus)*

A series of three alleles at the E locus is well established. The alleles are, in HETZER's nomenclature, *E* for uniform black, E^p for black-spotting and *e* for uniform red. The allelism of *E* and *e* has been shown in *Large Black* × *Duroc* crosses

(DETLEFSON & CARMICHAEL, 1921) and confirmed in *Hampshire* × *Duroc* crosses by BUSHNELL (1943). It is also compatible with observations involving the *Cornwall* and the *Bavarian* breeds by KRONACHER (1924). From those results, *Large Black, Hampshire* and *Cornwall* may be considered as *EE*, *Duroc* as *ee*, and *Bavarian Landschwein* as predominantly *ee*. Allelism between *E* and E^p may also be inferred from *Large Black* × *Berkshire* crosses (CARR-SAUNDERS, 1922), *Berkshire* × *Cornwall* crosses (KOSSWIG & OSSENT, 1931) and from comparisons between *Landrace* × *Large Black* and *Landrace* × *Poland-China* (or *Berkshire*) crosses by HETZER (1945 b, c, d), who also confirms (HETZER, 1946) the order of dominance $E/E^p/e$. It may thus be assumed that *Berkshire* and *Poland-China* are E^pE^p. The same genotype is found in the *Piétrain* breed (LAUVERGNE & OLLIVIER, 1966 ; MILOJIC, 1966). Thus the *Berkshire (Poland-China)* black is merely an extended form of black spotting. This was first suggested by WRIGHT (1918) and has later been confirmed by HETZER (1954) who showed that black spotting may be experimentally extended by selection. According to HANSET (1959), the origin of the *Piétrain* coat is the *Berkshire*, which was originally a spotted breed.

The existence of a fourth allele at the E locus is likely. KOSSWIG & OSSENT (1931) distinguish between a hypostatic black (*E*) in *Cornwall* (or *Large Black*) and a dominant black (E^d), epistatic over the other colours, in *Hannover-Braunschweig* (or *Hampshire*). This series of 4 alleles, $E^d/E/E^p/e$, is thus similar to that found in the guinea pig and the rabbit (SEARLE, 1968).

5. *Pink or Red-eye series (*P *and* R *loci)*

According to SEARLE (1968), no member of the P series is known, but the analogous (and linked) R locus (red-eye) of the rat seems to have an equivalent in the recessive autosomal gene (*r*) shown by ROBERTS & KRIDER (1949) to be responsible for the red-eye and dilution of the black pigment into a sepia colour, found in the *Hampshire* breed.

6. *White (*I *locus)*

White is the most frequent colour among the present domestic breeds of pigs, and, not surprisingly, crosses between white and coloured breeds have been the most investigated (see table 2). As early as 1906, SPILLMAN established the dominance of the white colour in a *Tamworth* × *Yorkshire* cross. On the basis of those results, WRIGHT (1918) assumed that white was due to two dominant genes, but, from later results, WENTWORTH & LUSH (1923) put forward the hypothesis of a single dominant gene. This was confirmed by HETZER (1945 b, c, d) who called the gene *I*. Later results also showed independence between the I and the E colour loci (HETZER, 1946). White breeds, such as *Yorkshire* and *Landrace*, are generally homozygous for *I*, a gene which inhibits both black and yellow pigment production, and coloured breeds, such as *Berkshire, Poland-China, Large Black*, etc., are homozygous recessive *ii*. A third allele, I^d, has been postulated by HETZER (1948) in order to explain the occurrence of roans in some crosses between *Landrace* and *Hampshire*. I^d would be recessive to and would have the same inhibitory effect on pigments as *I* when E^p is present, and it would give a roan phenotype (mixture of black and white hair) when *E* is present. The « sapphire hog » described by MC LEAN (1914) probably presented the same roan character, originating from the white breeds used in its foundation. This I^d gene, also present in the *Créole* pig of Guadeloupe (LAUVERGNE & CANOPE, 1979), is possibly homologous to the genes for roan found in cattle and in horses (SEARLE, 1968). A fourth allele i^m is assumed by BERGE (1961)

as responsible for the recessive white of *Mangalitza*, the order of dominance being then at the I locus $I/I^d/i/i^m$. This is not however accepted by SEARLE (1968) who considers the *Mangalitza* white as due to an allele at the C locus (see above).

7. *White Belt* (Be *locus*)

This pattern was first studied genetically by SPILLMAN (1907) who assumed it to be due to the complementary action of two genes. DURHAM (1921) is in favour of a major dominant gene, as also OLBRYCHT (1941) and DONALD (1951). The latter authors however disagree on the explanation for the colour polymorphism observed in the *Wessex Saddleback* and *Essex* breeds, where the belt is variable in width and black pigs are reported to remain at a noticeable frequency in spite of their regular elimination by the breeders. For OLBRYCHT (1944), belt width is essentially a polygenic character, whereas for DONALD the narrow belt selected for by the breeders is an « unfixable » heterozygous genotype ($Be^w be$), black piglets being mainly *bebe*. According to BERGE (1961), the wide belt of the *Hannover-Braunschweig* would correspond to the homozygous Be^wBe^w, and the extension of the belt towards the front found in the half-coloured *Bavarian Landschwein*, which is recessive to uniform colour, would be due to a third allele be^b at the same locus, the order of dominance being $Be^w/be/be^b$. SEARLE (1968) however considers the dominant white saddle of *Hampshire* (analogous to belt in cattle) and the recessive half-coloured pattern (similar to belted in mouse) as two entities not proven to be allelic. This opinion is somewhat reinforced by the suggestions of a close linkage between the E locus and the half-coloured pattern on one hand (KRONACHER, 1924) and of a loose linkage between the white belt factor and the E locus on the other hand (BUSHNELL, 1943). SEARLE (1968) also considers the white face pattern of the « Hereford hog » described by SMITH *et al.* (1938) as probably due to the same gene as the half-coloured pattern, whereas BERGE (1961) assumes an allele of the E series responsible for white face.

IV. Hair and skin

The hereditary basis of *hairlessness* (hypotrichosis) has been established by ROBERTS & CARROL (1931). This condition, which is to be distinguished from a similar one due to a deficiency of iodine, is due to a single autosomal recessive gene, which reduces the number of hair follicles.

Another type of hypotrichosis has been described by MEYER & DROMMER (1968). In their case an autosomal dominant gene is involved. This character is lethal, as homozygous hypotrichotic piglets die within 10 days. The vitality of heterozygous individuals is also reduced.

The *woolly hair* condition, frequently occurring in the Brazilian native breed *Canastrao*, has been studied by RHOAD (1934), who showed it to be determined by a single autosomal dominant gene, segregating independently from genes for coat colour and pattern. The data were insufficient to establish its independence from the recessive type of hairlessness, a factor which also was present in the same breed.

Disturbances in the arrangement of the hair, known as *whorls* or « roses », occur mainly along the spinal column. They have been explained by the complementary action of two dominant genes (NORDBY, 1932).

Several kinds of skin defects, of a hereditary nature, have been reported in the pig. A condition known as *epitheliogenesis imperfecta* is characterized by areas of missing epidermis of variable and irregular size. The condition is semi-lethal, as affected piglets usually die within three days but may survive if the abnormal area is small and the animal properly handled. The first case was reported by NORDBY (1929) who considered the abnormality to be probably genetic. This has been confirmed by segregation results obtained by SAILER (1955), which correspond to a single autosomal recessive gene.

The occurrence of *melanotic skin tumours* was first studied by NORDBY (1933), who came to the conclusion that the defect is inherited, but with an unclear mode of inheritance. This has later been confirmed by HOOK *et al.* (1979) who were able to increase the frequency of the defect by selection in a line of miniature pigs. Their data suggest that the hereditary basis is polygenic and similar to that reported in humans.

A hereditary basis of the transient skin disease known as *pityriasis rosea* has for the first time been suggested by WELLMANN (1953), who was able to exclude infectious agents as possible causes. The disease begins with the apparition of a few hyperhaemic patches on the underside of the animal. These rapidly spread in a circular fashion and then join together to form large circular marks similar to those found in ringworm. The disease appears in the first weeks of life and generally lasts until 3-4 months of age. Observations made in several countries (HEUNER, 1957 ; FLATLA *et al.*, 1958 ; LARSSON, 1961 ; CORCORAN, 1964) show that the condition is widespread, of a hereditary origin and may go unnoticed by the breeder, as general health is not impaired. Its mode of inheritance is complex according to WELLMAN (1953) and FLATLA *et al.* (1958), whereas LARSSON (1961), from his observations in Sweden, assumes a monogenic autosomal recessive inheritance.

Skin lesions of a different origin may also appear in the first or second week of life. This disease, known as *dermatosis vegetans*, differs from the preceding one in that it also affects the feet and the lungs. The pig usually die within four to six weeks from either pneumonia or secundary bacterial infections. FLATLA *et al.* (1961) showed dermatosis vegetans to be a semi-lethal hereditary disease due to a single autosomal recessive gene, a hypothesis which also fits the observations of DONE *et al.* (1967). The disease has also been reported in Austria by GLAWISCHNIG *et al.* (1974) who tried unsuccessfully to obtain affected animals from matings between parents known to have given defective progeny. This genetic defect seems to affect mesodermal tissue selectively (dermis, intestinal, lymphoid tissue, tonsils and pulmonary lymph nodes) as shown by the histological observations of JERICHO (1974). The *club-foot* syndrome, reported by LARSSON (1953) and shown to be inherited as a single autosomal recessive gene, is likely to be the disease later described as dermatosis vegetans.

Abnormal mammae, called « inverted » or « cratered » teats, were first investigated by NORDBY (1934 a), who suggested a simple recessive mode of inheritance. A recessive inheritance was also assumed by FISCHER (1957), HAMORI (1962 a) and PARIZEK *et al.* (1965), but more recent studies by HOLMQVIST (1971), MOLÉNAT & THIBAULT (1977), VENEV (1977) and CLAYTON *et al.* (1981) are in favour of a polygenic inheritance, with heritability estimates ranging from 0.10 to 0.76.

V. Skeleton

A. *Axial skeleton*

A *shortened vertebral column,* due to the absence of four cervical and two dorsal vertebrae and to the fusion of the remaining ones, has been described by GLUHOVSCHI *et al.* (1967), in a *Large White* herd. As the parents are normal and no karyotype abnormality exists, the authors conclude that the defect is due to a single autosomal recessive gene.

The « *kinky tail* » condition characterized by rigid angles due to irregular vertebral fusions, which is one of the various tail anomalies of genetic origin found in mice, has also been shown to be hereditary in pig by NORDBY (1934 b), who assumed a single recessive gene as the main cause of the defect. A similar tail defect, sometimes associated with uro-genital disorders, has been reported by DONALD (1949) and RYLEY *et al.* (1955). DONALD assumes an incompletely dominant gene, with variable expressivity due to recessive modifying factors. For RYLEY *et al.*, the genetic basis is likely to be complex and a threshold may be involved. No conclusion as to the inheritance of « kinky tail » could be reached by RICHTER & SCHARRER (1959), whereas FISCHER (1960) postulated a single dominant gene with low penetrance.

Rudimentary tail has been observed in connection with hydrocephalus, due to a recessive lethal gene (see below), and a *tailless* condition of hereditary origin associated with vertebral abnormalities has been reported by BROOKSBANK (1958).

B. *Head and appendages*

A common skull defect in pigs is *brain hernia*, which is due to a cleft in the skull, through which meninges may protrude (meningocele) or meninges and brain tissue (encephalocele). The early work reviewed by KOCH *et al.* (1957) points towards a sublethal recessive gene, with variable penetrance and expressivity according to COHRS *et al.* (1963). GILMAN (1956) suggests that a maternal dietary influence may affect the penetrance of the gene. Later results by MEYER & TRAUTWEIN (1966), STEWART *et al.* (1972) and WIJERATNE *et al.* (1974) confirm that no simple genetic explanation exists for this defect.

Hydrocephalus is an enlarged head condition which results from an excess of cerebrospinal fluid either in the brain ventricles (internal hydrocephalus) or in the cranial cavities (external hydrocephalus). The defect is variable in expression and has been found associated with rudimentary tail and sometimes light-coloured hair and skin, in the *Duroc* breed, by BLUNN & HUGHES (1938) and WARWICK *et al.* (1943), who showed it to be due to a single autosomal recessive gene. Observations by SMITH & STEVENSON (1973), on sire x daughter matings within a *Yorkshire* × *Landrace* herd, confirm the single recessive hypothesis.

According to the review by KOCH *et al.* (1957), the exact mode of inheritance for *cleft lip, jaw* and *palate (cheilognathopalatoschisis)* is uncertain. A simple recessive gene has been suggested by NORODD (1958) and a recessive gene with incomplete penetrance by LABIK (1972). Non-genetic factors may also be involved as shown by the breeding experiment of BUTZ & MEYER (1960).

In Swedish *Large White*, a condition characterized by *shortened lower jaw* (*brachygnathia inferior*) together with hind leg malformations has been observed by IDVALL (1952), who assumes a simple recessive mode of inheritance.

Complete *absence of the lower jaw* (*agnathia*) has been reported by KELLER (1941), HOLZ & FORTUIN (1956) and KRÜGER (1965), but the inheritance of this lethal condition has not been investigated.

Investigations on other *jaw malformations* (crooked or shortened jaw) with an uncertain hereditary basis have been reviewed by KOCH *et al.* (1957). DONE (1977) suggests that many facial deformities, including those due to atrophic rhinitis, have a genetically multifactorial aetiology.

A lethal factor, supposed to be recessive, is held responsible for the occurrence of *bilobed ears*, an abnormality which is sometimes accompanied by cleft palate and hind leg malformations (ANNETT, 1938).

Reduction in *size of the ears* has been reported to occur either alone or in connection with skull defects (NORDBY, 1930). The condition is considered to be hereditary, but external causes may also produce it (HORNEFF, 1967).

The presence of *tassels* or « bells » in the neck region of the pig has been reported in several breeds (see KOCH *et al.*, 1957). From the genetic investigations of KRONACHER (1924) and ROBERTS & MORRILL (1944) it can safely be concluded that a single autosomal dominant gene is responsible for the development of those appendages.

C. *Limbs*

Absence of one, two or four legs has been reported in pigs. The absence of the four legs (*legless*) is a lethal condition, which has been described by JOHNSON & LUSH (1939) and shown to be due to a single autosomal recessive gene. The absence of one or two legs has several times been described in grown pigs. From the work reviewed by KOCH *et al.* (1957) it can safely be concluded that the *three-legged* condition is due to a single autosomal recessive gene, whereas the less common *two-legged* condition (apodia) is genetically unclear.

The *absence of toes* (adactylia) has also been described in pigs, either alone or associated with several other abnormalities (BUTZ & SCHNELLE, 1951 ; BEER, 1962). A hereditary basis is probable but has not yet been clearly established.

Syndactyly and *polydactyly* have been extensively studied. The early genetic hypothesis of a single dominant gene responsible for syndactyly (SIMPSON's, 1908) has been later on confirmed by other workers with few exceptions (see KOCH *et al.*, 1957). The inheritance of polydactyly is much less clear, owing to the irregular expression of the defect, and so far no clear conclusion can be reached, according to KOCH *et al.* (1957). However, MALYNICZ (1982) has recently described a case of autosomal dominant complete polydactyly in Papua New Guinea village pigs. Homozygous piglets are lethal monsters with club foot and otocephaly.

A hereditary defect leading to *unequal toes* has been described by NORDBY (1939). Its exact hereditary basis however remains to be explained.

A *short-leg* syndrome has been shown to be inherited as a single recessive gene by SWIGER (1981).

D. *Multiple abnormalities*

Various skeletal anomalies may occur on the same animal. Such is the case of the *Pulawska lethal factor* described by DABCZEWSKI (1949) and inherited as a single autosomal recessive gene. Various malformations affect the cranium bones, the vertebral column (vertebral fusion) and the ribs, and several internal organs (liver, pancreas, kidney and intestine are larger than normal, and lungs are rudimentary).

Other cases of multiple anomalies (involving skeleton and ears) have been reported by RYLEY *et al.* (1955), BEER (1962) and TROLLDENIER (1964). Their hereditary basis remains unclear, but they remind of the case, already mentioned, of ANNETT (1938).

As previously mentioned, *rudimentary tail* has been shown to be associated with *hydrocephalus* by BLUNN & HUGUES (1938) and WARWICK *et al.* (1943), *agnathia* with several malformations by KELLER (1941) and HOLZ & FORTUIN (1956), and *polydactyly* with *club foot* and *otocephaly* by MALYNICZ (1982).

In many cases the genetic study of skeletal anomalies is made difficult because some of them arise as a result of metabolic disturbances of non-genetic origin occurring during embryonic development, as indicated in particular by FREDEEN & JARMOLUK (1963). These authors give several examples of skeletal defects not reported previously and for which no genetic explanation can be offered. This is also the case for a vertebral column malformation (lordo-scoliosis) reported by CELA & COLOMBANI (1972).

Lameness (or the so-called *leg weakness syndrome*) occurs frequently in the modern pig. Various forms of skeletal lesions (osteochondrosis, epiphysiolysis, arthritis) are usually at the origin of this defect. An extensive literature is devoted to the subject, and many authors have suggested that the disease is partly of hereditary origin. A moderate heritability is usually found for leg weakness, when it is assessed either visually (SMITH, 1966 ; GRÖNDALEN, 1974 ; BRING-LARSSON & SUNDGREN, 1977 ; BERESKIN, 1979 ; WEBB & RUSSELL, 1981) or radiologically (NAGEL & SEIFERT, 1980).

VI. Eye

Lack of pigmentation of the iris leads to the « glass-eye » defect also called *heterochromia iridis*. An account on the heredity of this condition has been given by GELATI *et al.* (1973). In the first genetic work, DÜRR (1937) concluded to an incompletely dominant gene, as only about 50 percent of the heterozygous individuals exhibit the trait. GELATI *et al.* (1973) conclude that, in their herd of miniature pigs, the defect is due to an autosomal recessive gene (*het*), whose expression is variable and in particular is influenced by the white colour gene (*I*). The two hypotheses can be reconciled if one assumes, as suggested by GELATI *et al.* (1973), that pigs with bilateral heterochromia are homozygous at the Het locus and unilateral or partial heterochromia, very frequent in DÜRR's data, may appear in heterozygous individuals. The gene may thus be considered as truly recessive for bilateral heterochromia.

Congenital blindness is of frequent occurrence in pigs and generally the result of various degrees of microphtalmia. Non-genetic causes may be implied in eye defects, especially lack of vitamin A. Several workers however have shown that a genetic basis exists for blindness in pigs. The various cases studied have been reviewed by KOCH *et al.* (1957) who observe that their own investigations, on the progeny of a blind boar, do not always confirm previous ones and conclude that further research is needed in order to clarify the heredity of the condition.

Cyclopia is an abnormality found in several species and shown to be hereditary in guinea pig and rabbit. In the pig, cyclopia often goes along with several other abnormalities and its heredity remains to be clarified (KOCH *et al.*, 1957).

The *red-eye* mutation (also known as *albinismus oculi*) has been referred to in the section on coat colour.

VII. Neurological and neuromuscular disorders

Congenital tremor (or *myoclonia*) is a frequent disease affecting the central nervous system of pigs, and it may have several different causes. In the taxonomy proposed by DONE (1976 a), those forms of tremor in which morphological lesions are found are called type A. Two of these are of genetic origin, namely AIII, which is due to a sex-linked recessive gene (HARDING *et al.*, 1973) and AIV, which is due to an autosomal recessive gene (PATTERSON *et al.*, 1973). Type AIII is similar to the « jimpy » mutant in mice and type AIV to the murine leukodystrophy designated « quaking » (DONE, 1976 b). Neurochemical techniques allow distinguishing the hereditary types from those due to transplacental infections (PATTERSON & DONE, 1977). There are also several observations indicating a possible role of the boar in a non-genetic transmission of myoclonia (STROMBERG, 1975). A favourable association in fattening pigs between tremor and growth and carcass quality has been shown by GEDDE-DAHL & STANDAL (1970), in contrast with the unfavourable effects of the disease in early life.

Epilepsy has been investigated by SONNENBRODT (1944), who considered it unlikely to be monogenic and postulated two or more factors responsible for the disease.

A *congenital motor defect*, demonstrated by clinical signs of ataxia and perverse movements, with no morphological defect in the central nervous system at birth, but dysplasia of the cerebellar cortex in older pigs, is inherited as a single autosomal recessive trait in *Large White* and *Wessex Saddleback* pigs (DONE, 1978). This has recently been confirmed in *Yorkshire* pigs in Finland (RIMAILA-PÄRNÄNEN, 1982).

Several cases of *hind leg paralysis* have been reported in Norway, in the U.S.S.R. and in Germany. KOCH *et al.* (1957) consider those cases as possibly all due to the same recessive lethal gene. The case reported by LUDVIGSEN *et al.* (1963) in the *Danish Landrace* may be due to a different recessive gene, this condition being associated with abnormal lumbar vertebrae.

In 1933, HALLQVIST described an abnormality of the forelegs in *Swedish Landrace* which he called *bent-stiff-legged* and showed to be due to an autosomal recessive gene. This defect, known as *arthrogryposis* in several other domestic species, may affect the four limbs as in the cases reported by ELY & LEIPOLD (1979) and also shown to be due to a single autosomal recessive gene. SWATLAND (1974) considers the general name *congenital articular rigidity* (CAR) as more appropriate, and in his review on porcine CAR he classifies the observed cases into hereditary, as the previously mentioned ones by HALLQVIST (1933), and environmentally-induced or sporadic due to an unknown cause. As to the primary cause of rigidity, it may be muscular, nervous (neuroaxonal dystrophy in ELY & LEIPOLD's cases) or skeletal as in the *thickleg syndrome* where both connective tissue and bone developments are abnormal. This defect, which preferentially affects the forelegs, is inherited as an autosomal recessive gene. It is not certain that this gene is different from the one responsible for bent-stiff-leg.

Splayleg is a condition of newborn piglets in which the hind legs and sometimes the forelegs tend to splay sidewards and forwards as a result of muscular weakness. In their reviews of the subject SWATLAND (1974) and WARD (1978) conclude that splayleg has a genetic basis, probably polygenic, though sometimes it occurs only in males and may be due to a sex-linked gene with variable penetrance (LAX, 1971) Myofibrillar hypoplasia has sometimes been observed in splayleg (though it may also be found in normal pigs) and indicates a retardation in the development of the leg muscles as a consequence of a possibly general neuromuscular dysmaturity. Splayleg accompanied by tremor has been reported to occur in Hungary by BERTHA (1975). SELLIER & OLLIVIER (1982) show the genetic basis of the character to be of an additive polygenic type, with an estimated heritability of 0.47. They also find a higher frequency of the defect in males (the character is sex-influenced but not sex-linked) and a higher incidence in larger litters.

Other disturbances in muscle development may result in the so-called *asymmetric hindquarter syndrome* (AHQS). A variety of degenerative and dystrophic changes have been seen in the muscles of AHQS pigs. The precise cause of the syndrome is still unresolved further than the observation of familial occurrence, which suggests a genetic liability (DONE *et al.*, 1975). This however could not be confirmed in a breeding experiment at the Hannover Veterinary School (Institut für Tierzucht, 1975).

A genetic basis is also assumed for the condition known as *acute back muscle necrosis* by BICKHARDT *et al.* (1975), according to whom this myopathy is one among several possible manifestations, induced by bodily exertion or stress, of a latent condition due to an insufficient muscle energy metabolism, other possible manifestations being the post mortem pale soft exudative (PSE) meat or the stress-induced sudden death or porcine stress syndrome (PSS). BRADLEY *et al.* (1979) also found the defect associated with PSS.

PSE and sudden death are also connected with a genetic defect known as *malignant hyperthermia* (MH), a syndrome found in several other species including Man and which is triggered by halothane anesthesia. A rapid rise in body temperature, muscular rigidity and blotchy cyanosis of the skin are the most obvious symptoms. Death normally ensues if anesthesia is continued. A genetic basis was first suggested by HALL *et al.* (1966). CHRISTIAN (1972) assumed a single autosomal recessive gene with incomplete penetrance, and this has been later on subtantiated by the experimental results of OLLIVIER *et al.* (1975), SMITH & BAMPTON (1977), ANDRESEN & JENSEN (1977), SCHWÖRER & BLUM (1977), MC PHEE *et al.* (1979), HRADECKÝ *et al.* (1980) and MABRY *et al.* (1981). However, MINKEMA *et al.* (1977), in the *Dutch Landrace*, consider the gene to be fully penetrant. Possible breed differences in gene penetrance (along with important differences in gene frequency) have been suggested by OLLIVIER *et al.* (1978). Hypotheses involving more than one gene have been postulated by BRITT *et al.* (1978) and WILLIAMS *et al.* (1978). The relationships between malignant hyperthermia, PSE meat condition, porcine stress syndrome and various forms of myopathy, such as the « creeper » trait in *Piétrain* described by WELLS *et al.* (1980), need further clarification. Well established connections however exist, according to several investigators, between MH, liability to sudden death and muscular hypertrophy. In fact, the major gene responsible for muscular hypertrophy in the *Piétrain* pig may well be the MH gene itself — Hal^s (or Hal^n) as opposed to the normal allele Hal^+ (or Hal^N) — as suggested by OLLIVIER (1980).

A muscle disease, known in human as *myositis ossificans*, has been shown to occur on 34 out of 115 progeny of a boar who himself developed the condition at the age of 9 months (SEIBOLD & DAVIS, 1967).

More detailed information and references pertaining to this section may be found in general reviews on nervous or muscular disorders by DONE (1968, 1976 b), SWATLAND (1974) and BRADLEY & WELLS (1978).

VIII. Haematological disorders

Abnormal haemoglobin synthesis in the bone marrow is thought to be at the origin of congenital *porphyria* (found in human, cattle and pigs) in which excessive amounts of porphyrin are deposited in the bones and teeth and excreted in the urine and faeces. In pigs, the condition has been studied in detail by JØRGENSEN (1959) who considers it to be hereditary and due to one or more dominant genes, contrary to cattle where it depends on a recessive gene. However, YAMASHITA *et al.* (1980) suggest that porphyria in pigs is also due to a single recessive gene.

A *hemophilia-like disease* was reported and studied genetically by BOGART & MUHRER (1942) in an inbred line of *Poland-China* swine and more recently by FASS *et al.* (1979). The disease was shown to be due to a single autosomal recessive gene. It is analogous to the human *von Willebrand's disease*, a type of hemophilia due to the lack of a plasma antihemophilic factor (factor VIII). Normal porcine plasma also contains a platelet aggregating factor (factor W) for human platelets and, as this W factor is associated with factor VIII, a quantitative assay of W makes it possible to detect heterozygous carriers of the hemophilia gene (GRIGGS *et al.*, 1974).

A hereditary *lymphosarcoma* (leukemia) has been described in a herd of *Large White* pigs, and shown to be due to an autosomal recessive gene (MC TAGGART *et al.*, 1979).

Anemia may be genetically determined in piglets, as various blood parameters have been shown by several authors to be under genetic control, likely polygenic in nature (see GABRIS, 1973, for references).

The *hemolytic* disease of the newborn piglets is similar to *icterus neonatorum* in humans. SZENT-IVANYI & SZABO (1953) assume an antigen, which they call Su, due to a dominant gene similar to the Rhesus factor in man, responsible for this sow-piglet incompatibility, which, owing to the particular pig placentation, can only express itself when the piglet has absorbed the antibodies present in the colostrum. The disease can also be produced experimentally, as shown by ANDRESEN & BAKER (1963), who used an antigen of the B blood group system. HIMENO *et al.* (1969) were able to identify one red cell antigen responsible for the disease and confirmed that it was due to a single gene. Antibodies against erythrocytes may also arise from the vaccination of the sow by the hog cholera crystal violet vaccine and several blood cell antigens may be involved (DUNNE, 1975).

A similar disease, *thrombocytopenia*, may arise from the development of maternal antibodies against the platelets of the piglets, with pathological changes characterized by paleness of various tissues and scattered hemorrhages in various parts of the body (STORMORKEN *et al.*, 1963). The simultaneous occurrence of hemolytic disease and thrombocytopenia has been reported (see DUNNE, 1975).

IX. Endocrine and metabolic defects

Only three cases of *dwarfism* in the pig have been mentioned in the literature according to PETROV (1974), who himself obtained a female dwarf in the F_2 of a black *Mangalitza* × *East Balkan* cross. A subsequent full-sib mating between normal parents gave 8 F_3 piglets of which 2 were dwarfs. The author considers the anomaly as similar to achondroplasy in cattle and similarly inherited as a recessive.

Investigations on the disease of newborn pigs called *oedema*, myxoedema or hydrops, have been reviewed by KOCH *et al.* (1957). Their conclusion is that a thyroid deficiency is at the origin of the disease, which appears to be inherited as a single autosomal recessive gene. In the pig oedema appears to be quite distinct from hemolytic disease, whereas in human and rabbit hydrops has been assumed to be a consequence of mother-fetus incompatibility due to a Rhesus-like factor.

A new syndrome, characterized by *acute respiratory distress* and by abnormalities of the skin, hair and thyroid, has been described in piglets by GIBSON *et al.* (1976). The syndrome, similar to the respiratory distress syndrome of the new born infant and the « barker » syndrome of the foal, is inherited as a single autosomal recessive gene. WRATHALL *et al.* (1977) suggest that the manifestations of the syndrome may all be more or less directly related to a fetal hypothyroidism.

A disturbance of calcium metabolism, causing severe *rickets* in pigs after the age of five weeks, has been shown to be due to an autosomal recessive gene by MEYER & PLONAIT (1968) and confirmed by HARMEYER (1982). In piglets with inherited rickets, the rachitic lesions do not respond to dietary vitamin D and the Ca intestinal resorption is markedly reduced, as in the vitamin D-resistant form of the disease in children. HARMEYER (1982) offers some evidence that this hereditary defect may also in pigs be due to an insufficient renal production of the vitamin D_3 hormone.

Obesity has been reported to occur in piglets and to be a semi-lethal condition with an unclear genetic aetiology (KOCH *et al.*, 1957). Recently, an *obese syndrome* has been described in a native breed of Georgia by MARTIN *et al.* (1973), and shown to be comparable to human diabetes. In fact, the pig has a natural propensity to excess adiposity and fat tissue development is a complex polygenic character which has been intensely selected by the breeder (see performance traits). A review on genetic and metabolic aspects of swine adipose tissue development has been given by HENRY (1977). Variations in *glucose tolerance* have been shown to exist in pigs and some attempts are being made at producing a genetically diabetic pig for biomedical purposes, by exploiting presumably polygenic variations in the rate of glucose utilization (PHILLIPS *et al.*, 1979).

X. Internal organs

A. *Digestive tract*

Atresia ani (closure of the anal outlet) is one of the most frequent abnormalities encountered in pigs : 0.14 p. 100 in Germany according to TRIEBLER *et al.* (1974), 0.14 and 0.31 p. 100 respectively for *British Large White* and *Landrace* from REED's survey (1976). Various genetic hypotheses have been proposed : see KOCH *et al.*

(1957) and WIESNER & WILLER (1974) for reviews. Either one recessive gene with incomplete penetrance or two incompletely dominant genes have been proposed as the most likely genetic explanations. NEETESON (1964), from observations on 36 litters, ruled out six different genetic explanations and retained as a provisional conclusion a two dominant genes hypothesis. The frequency of the defect in affected litters observed by TRIEBLER *et al.* (1974) agrees with the hypothesis of a recessive gene with 50 p. 100 penetrance. The linkage mentioned between atresia ani and thickleg by WALTHER *et al.* (1932) awaits further confirmation.

A different abnormality with similar consequences, *aplasia of the anal sphincter*, has been reported by HAMORI (1965) who considers it as a semilethal hereditary defect whose transmission still remains to be clarified.

Scrotal (or *inguinal*) *hernia*, a protrusion of the intestine into the scrotum (or through the inguinal canal), is probably the most frequent hereditary abnormality in the pig. In an externe case, 5 p. 100 of the male pigs were affected (MAGEE, 1951). Scrotal hernia is a sex-limited defect, whereas inguinal hernia is found in both sexes. Inguinal hernia in females has been reported to occur in conjunction with abnormal ovaries (COLENBRANDER & WENSING, 1975). According to WARWICK (1926), scrotal hernia is dependent on two recessive genes, whereas BERGE (1941) considers it to be monofactorial with incomplete dominance and HAMORI (1962 b) suggests two sex-linked recessive genes. After reanalyzing published data, SITTMAN (1973 a) came to the conclusion that scrotal (inguinal) hernia is fully penetrant in males homozygous recessive at two loci. These are normally viable but homozygous recessive females have a lowered viability, which may produce a deficiency of sisters of affected males. However, MAGEE (1951) assumes the character to be polygenic (with a threshold) with low heritability, and under the same assumption a high heritability has been estimated by MIKAMI & FREDEEN (1979).

Umbilical hernia is a less frequent type of hernia ; its frequency is higher in females than in males. KOCH *et al.* (1957) mention studies showing the condition to be hereditary, possibly of a dominant type.

A « baby pig disease », appearing as a degeneration of the myenteric *stomach plexus*, has been reported to occur in the progeny of two boars in a Danish experimental farm by STEINICKE & NIELSEN (1959) who suggest that hereditary as well as exogenous factors are probably involved.

A condition called *diverticulosis* has been observed with a high frequency in an experimental herd in New-Zealand by HANCOCK (1950). The abnormality appears as a series of pockets formed by the intestinal mucous membrane and it is associated with a thickening of the gut. The mode of inheritance is not clear but a recessive gene may be implied.

B. *Uro-genital system*

A bilateral *renal hypoplasia* has been observed in the progeny of one *Large White* boar by CORDES & DODD (1965) who state a strong evidence that the defect is due to a single autosomal recessive gene.

An *obstruction of the ureters*, which occurs shortly after birth and leads to death by uremia within a few days, is at the origin of the baby pig disease described by LUKAS (1953) and considered by him as hereditary.

Recently, the existence of an autosomal dominant gene responsible for *renal cysts* has been shown by WIJERATNE & WELLS (1980).

Various *uro-genital disorders* have previously been mentioned in connection with a hereditary tail defect (DONALD, 1949 ; RYLEY *et al.*, 1955).

The most common abnormality in the male reproductive system is *cryptorchidism*, for which a recent and very detailed review by SITTMAN & WOODHOUSE (1977) is available. By reanalyzing five sets of published data, they come to the conclusion that those data agree with the hypothesis of completely penetrant recessive genes at two autosomal loci, except in the *Lacombe* breed where a multifactorial threshold model is more plausible. With this same model, MIKAMI & FREDEEN (1979) obtain a heritability of 0.5 for the underlying variable. TRIEBLER *et al.* (1974), from observations in German Democratic Republic, assume one autosomal recessive gene.

Boar sterility may be due to either incapacity to copulate (*sexual impotence*) or incapacity to fertilize. Among 22 sons of a slow breeding boar, HOLST (1949) found 12 that had to be discarded as sexually impotent and were the sons of 6 different sows. His data suggest, as those of AAMDAL & NES (1958) and WIERZBOWSKI (1959), a hereditary basis for sexual impotence but they do not corroborate the hypothesis suggested by FUNKQUIST (1929), on a rather limited sample, of a recessive sex-linked mode of inheritance.

Incapacity to fertilize may be due to *testicular hypoplasia* for which a hereditary basis has been suggested by HOLST (1949), WOHLFARTH (1964) and WOHLFARTH & SEFFNER (1968). A case due to an abnormal sex chromosome constitution (XXY) has been reported by HANCOCK & DAKER (1981). Fertility disorders may also result from *epididymal aplasia* which has been found by KÖNIG *et al.* (1972) in a fertile boar (with unilateral aplasia) and three of his sons. These observations are in favour of a recessive mode of inheritance, as for a similar and much more frequent defect in bulls.

Abnormal spermiogenis may also affect fertility. The *knobbed acrosome* defect, due to an autosomal recessive gene in cattle, is likely to have a different genetic basis in pig. BISHOP (1972) rightly remarks that the recessive mode of inheritance suggested by WOHLFARTH (1961) is not supported by his own data. A new defect of the sperm head, appearing as a nuclear cyst and associated with lowered fertility, has been described by BLOM (1973) who called it « *SME-defect* » from the name of the boar carrying it and it is considered as probably hereditary by ANDERSEN & FILSETH (1976) and BLOM & JENSEN (1977), the latter authors assuming a simple recessive mode of inheritance. It cannot yet be ascertained whether this sperm defect is related or not to the preceding one.

Finally, it must be mentioned that various *chromosomal abnormalities* may reduce boar fertility as a consequence of unbalanced gametes produced at meiosis which increase embryonic mortality (HENRICSON & BACKSTRÖM, 1964 ; VOGT *et al.*, 1974). Thus a reduction of about 50 p. 100 is generally observed in the size of the litters produced by a boar carrying a *translocation*, as it is the case for the reciprocal translocation found by POPESCU & LEGAULT (1979). Ten different types of translocations have so far been reported in the pig, as reviewed by POPESCU (1982).

Among *gross genital abnormalities* observed in gilts by WIGGINS *et al.* (1950), the most frequent are tubal abnormalities (1.4 p. 100), cystic follicles (1.7 p. 100) and missing parts (0.7 p. 100), if one excludes intersexuality and immature genital tract.

Such abnormalities, which explain about one quarter of the reproductive failures, have no established hereditary basis, except for a segmental aplasia of the uterus shown to be due to one autosomal recessive gene (KING & LINARES, 1980).

Intersexuality is a frequently occurring defect in pigs, the reported frequencies ranging from 0.1 to 1.4 p. 100 (BACKSTRÖM & HENRICSON, 1971). In general intersexes may be either *true hermaphrodites*, which have gonads of both sexes, or pseudohermaphrodites, these having either female or male gonads and being called accordingly *female* or *male pseudohermaphrodites*. The great majority of pig intersexes belong to the latter category and they have been shown to possess the normal female XX chromosome constitution, and may be regarded as masculinized females (BREEUWSMA, 1970 ; BACKSTRÖM & HENRICSON, 1971). Most authors consider this type of intersexuality as hereditary : see KOCH *et al.* (1957), BREEUWSMA (1970) and BISHOP (1972) for reviews. The simplest hypothesis, proposed by several authors, of a recessive autosomal gene with sex-limited expression, is excluded by BACKSTRÖM & HENRICSON (1971) who imply several genetic causes on the grounds of a higher frequency of abnormalities among intersexes and also of a reduced sex-ratio due to an increased female embryonic mortality. BREEUWSMA (1970) came to the conclusion that intersexuality is controlled both paternally (as shown by differences between boars with regard to its incidence among their offspring) and maternally (through an early contact between male and female blastocysts due to crowding of the uterus). However, when the same data were reanalyzed by SITTMANN *et al.* (1980), maternal and other environmental factors appeared to be unimportant, and intersexuality was held to be essentially genetic, either with polygenic inheritance or more likely due to recessive genes at very few autosomal loci.

In some cases, as those reported by GLUHOVSCHI *et al.* (1969) and LOJDA (1975), male pseudohermaphrodites show a male chromosomal constitution (XY). This condition, much rarer than the XX hermaphroditism, is comparable to the human syndrome of *testicular feminization*. Two possible genetic hypotheses have been proposed, in Man as well as in pig, either an autosomal dominant gene or a sex-linked recessive, between which it is not yet possible to decide (LOJDA, 1975).

Freemartinism as another cause of intersexuality has been reported in pigs. BOSMA *et al.* (1975) have reviewed such cases and their own ones, in which an XX/XY mosaicism is found in lymphocytes and an XX pattern in kidney cells. This suggests that placental cross-circulation between embryos of different sexes is the primary cause of intersexuality, as in cattle free-martins.

Abnormal chromosome pattern of the Klinefelter type (XXY) and various mosaics have occasionally been reported in pig intersexes (see the review by HULOT, 1969) and also in a boar with testicular hypoplasia (see above).

To summarize the above sections on genetic abnormalities, a list is given in table 4 of 29 defects which may be considered as probably monogenic.

XI. Disease resistance

Resistance to disease is not in general simply inherited, apart from the purely genetic diseases which have been described before, like pneumonia in dermatosis vegetans, tremor, hemophilia, leukemia, hemolitic disease, oedema, respiratory distress,

TABLE 4

A list of probably monogenic abnormalities in the pig.

Liste des anomalies porcines probablement monogéniques.

Abnormality	Mode of inheritance	Degree of lethality
Hairlessness	Autosomal recessive	
Hairlessness	Autosomal dominant	Homozygous lethal
Woolly hair	Autosomal dominant	
Epitheliogenesis imperfecta	Autosomal recessive	Semi-lethal (C_{17}) (*)
Dermatosis vegetans	Autosomal recessive	Semi-lethal (C_{13})
Hydrocephalus	Autosomal recessive	Lethal (C_8)
Tassels	Autosomal dominant	
Legless	Autosomal recessive	Lethal (C_9)
Three-legged	Autosomal recessive	
Syndactyly	Autosomal dominant	
Polydactyly	Autosomal dominant	Homozygous lethal
Pulawska factor	Autosomal recessive	Lethal (C_{16})
Heterochromia iridis	Autosomal recessive	
Congenital tremor A III	Sex-linked recessive	Semi-lethal
Congenital tremor A IV	Autosomal recessive	Semi-lethal
Congenital ataxia	Autosomal recessive	Semi-lethal
Hind leg paralysis	Autosomal recessive	Lethal (C_2)
Bentleg	Autosomal recessive	Lethal (C_6)
Thickleg	Autosomal recessive	Semi-lethal (C_5)
Malignant hyperthermia	Autosomal recessive	
Hemophilia (von Willebrand's disease)	Autosomal recessive	Semi-lethal (C_{15})
Leukemia	Autosomal recessive	
Hemolytic disease	Autosomal dominant	Lethal (C_{14})
Œdema	Autosomal recessive	Lethal (C_{12})
Acute respiratory distress ("barker")	Autosomal recessive	Lethal
Rickets	Autosomal recessive	
Renal hypoplasia	Autosomal recessive	Lethal
Renal cysts	Autosomal dominant	
Uterus aplasia	Autosomal recessive	

(*) Reference in the international list of lethal defects given by WIESNER and WILLER (1974).

rickets, etc. However, a resistance to *neonatal diarrhoea* due to a single autosomal recessive gene has been reported by RUTTER *et al.* (1975) and further investigated by GIBBONS *et al.* (1977). Resistance to *leptospirosis* has been shown to be associated with various genetic markers by PRZYTULSKI & PORZECZKOWSKA (1976, 1979), and *lung lesions* with adenosine deaminase by HYLDGAARD-JENSEN (1978).

More frequently, disease resistance behaves as a polygenic character with one (or several) threshold, and the concept of « liability » should in this context be preferred to « susceptibility », as liability includes all circumstances, internal as well as external, which can influence the manifestation of the threshold character (FALCONER, 1965 ; DONE & WIJERATNE, 1972). Such a kind of genetic resistance has been shown to exist

for several specific diseases, namely *swine fever* and *foot-and-mouth disease* (SMITH *et al.*, 1938) *brucellosis* (CAMERON *et al.*, 1943), *pigling dysentery* (ENGELHARDT, 1951), *lactation failure* (RINGARP, 1960 ; MARTIN & MC DOWELL, 1975), *tuberculosis* (GEDYMIN *et al.*, 1964), *gastric ulcer* (BERRUECOS & ROBISON, 1972), *atrophic rhinitis* (e.g. JONSSON, 1965 ; SEIFERT *et al.*, 1971 ; PLANCHENAULT *et al.*, 1978 ; KENNEDY & MOXLEY, 1980), *respiratory diseases* (LUNDEHEIM, 1979) and *leptospirosis* (PRZYTULSKI & PORZECZKOWSKA, 1980). In their review of early selection experiments for resistance against swine fever, SMITH *et al.* (1938) conclude that a certain degree of resistance is inherited, the most likely mode of inheritance being polygenic, with recessive factors. They also state that inbreeding reduces and crossbreeding enhances disease resistance, which confirms the existence of recessive factors. This has since been shown in numerous investigations (see the review by SELLIER, 1970) to be a rather general phenomenon.

XII. Immunology

For a long time the research on pig immunogenetics has been confined to *erythrocyte antigens* or *blood groups*. The first steps in this field are the discovery of the A blood factor by SZYMANOWSKI *et al.* (1926) and the genetic study of KAEMPFFER (1932) hypothesizing that the A factor is inherited as a Mendelian dominant character. A detailed review of the earlier work on pig blood groups has been made by ANDRESEN (1962) : at that time, 22 blood factors had been assigned to 10 genetic systems (A, B, E, F, G, H, I, J, K, L). Since then, more than 40 other blood group factors belonging to 8 of these 10 systems or to 5 new systems (C, D, M, N, O) have been genetically described, as successively summarized by DINKLAGE (1970 a), LARSEN (1971), IMLAH (1972 a), RASMUSEN (1975 a), AGERGAARD & NIELSEN (1978), GAHNE (1979), OISHI (1979) and HRUBAN (1981).

The current status of the 15 blood group systems controlling the polymorphism of pig erythrocyte antigens is given in table 5, with a list of references for each of them. A large number of reports being devoted to the most polymorphic systems (E, H, K, L and M), one or two recent references where these reports are quoted are only given in addition to the original report. From 2 to 16 « alleles » or « phenogroups » are presently known in each system. The dash superscript « - » in a gene symbol refers to a « silent » allele for which no antigenic substance is yet detected and the corresponding systems, i.e. C, H, J, K and M, are called « open ». In contrast, the 2-allele B, D, G, I, and O, the 3-allele F and N, the 6-allele L and the 15-allele E systems are « closed » systems. The existence of recombinations within the complex E system has been postulated by RASMUSEN (1963) and BAKER (1971).

The two-factor A system has serological and genetic properties which give it a special position. Two types of A-positive red blood cells, i.e. A(Ac) and Aw(Ap), are recognized (HOJNÝ & HÁLA, 1965 a ; HOJNÝ & STRATIL, 1978). Moreover, while genes are codominant for the other blood group systems, the gene A^A controlling the factor A is completely dominant on its allele a^O controlling the factor O. The existence of A-O negative individuals and the occurrence of A-positive offspring with allegedly A-negative parents have led several authors to the hypothesis that the expression of the A and O antigens is influenced by genes at a different locus (ANDRESEN, 1962 ; SAISON & INGRAM, 1962 ; RASMUSEN, 1964 ; HOJNÝ & HÁLA, 1965 a). The results of

TABLE 5

The current status of the blood group systems in the pig.

Etat actuel des systèmes de groupe sanguin du Porc.

System	Number and designation of blood factors	Minimum number of "alleles"	Allelic symbols	References
A	2 A(Ac,Ap),O	2	A^A, a^O	HOJNÝ and HÁLA (1965a), HOJNÝ and STRATIL (1978)
B	2 Ba,Bb	2	B^a, B^b	BAKER and ANDRESEN (1962, 1964)
C	1 Ca	2	C^a, C^-	ANDRESEN and BAKER (1964)
D	2 Da,Db	2	D^a, D^b	SAISON *et al.* (1967), HRADECKÝ and LINHART (1970)
E	16 Ea,Eb,Ed,Ee,Ef, Eg,Eh,Ei,Ej,Ek, El,Em,En,Eo,Ep, Er	15	$E^1 = E^{bdgkmp}, \ldots,$ $E^{15} = E^{abgmnop}$	ANDRESEN (1962) and many subsequent reports quoted by LINHART and ROMANOV (1975)
F	4 Fa,Fb,Fc,Fd	3	F^{ac}, F^{bc}, F^{bd}	ANDRESEN (1957), HRADECKÝ and HOJNÝ (1970), VORON and SOKOLENKO (1971)
G(*)	2 Ga,Gb	2	G^a, G^b	ANDRESEN and WROBLEWSKI (1961)
H	5 Ha,Hb,Hc,Hd,He	7	$H^a, H^b, H^{ab}, H^{cd}, H^{bd}, H^{be}, H^-$	ANDRESEN and WROBLEWSKI (1961) and subsequent reports quoted by HOJNÝ (1973)
I	2 Ia,Ib	2	I^a, I^b	ANDRESEN (1962, 1964)
J	2 Ja,Jb	3	J^a, J^b, J^-	ANDRESEN (1962), HOJNÝ and HRADECKÝ (1972)
K	7 Ka,Kb,Kc,Kd,Ke, Kf,Kg	6	$K^{acef}, K^{acf}, K^{ade}, K^{adeg}, K^{bf}, K^-$	ANDRESEN and IRWIN (1959), BRUCKS (1966), HOJNÝ *et al.* (1979b), and other reports quoted by NIELSEN and VÖGELI (1982)
L	12 La,Lb,Lc,Ld,Lf, Lg,Lh,Li,Lj,Lk, Ll,Lm	6	$L^{adhi}, L^{bcgi}, L^{bdfi}, L^{adhjk}, L^{adhjl}, L^{agim}$	ANDRESEN (1962) and subsequent reports quoted by HOJNÝ *et al.* (1966) and LINHART (1971)
M	11 Ma,Mb,Mc,Md, Me,Mf,Mg,Mh, Mi,Mj,Mk	16	$M^{ae}, M^b, \ldots, M^-$	NIELSEN (1961) and other papers quoted by HOJNÝ *et al.* (1979a)
N	3 Na,Nb,Nc	3	N^a, N^b, N^{bc}	HÁLA and HOJNÝ (1964), HOJNÝ *et al.* (1966), SAISON (1967)
O	2 Oa,Ob	2	O^a, O^b	HOJNÝ and HÁLA (1965b), HOJNÝ *et al.* (1966)

(*) A factor (Gc) controlled by a G^{bc} allele is likely to exist (ANONYMOUS, 1981).

RASMUSEN (1972) and HOJNÝ (1974) indicated that alleles at the H blood group locus are involved in the determination of the A-O phenotypes. However, according to RASMUSEN *et al.* (1980), RASMUSEN (1981) and ANDRESEN (1981), there is evidence that inhibition of expression of A and O is not due to the H system itself but to a distinct locus (S, as designated by RASMUSEN, 1964) closely linked to H.

According to HOJNÝ & STRATIL (1978), more genetical evidence is required to classify the so-called Hel system (SCHMID & BUSCHMANN, 1966) as the 16th pig blood group system.

Immunogenetic studies in pigs have resulted in the detection of other markers than red blood cell antigens (GAHNE, 1980).

The pig's *major histocompatibility complex* (MHC), which plays a decisive role in acute transplantation reactions, has been first evidenced by VAIMAN *et al.* (1970, 1971). This system, called SL-A (or SLA), presents many functional similarities with the murine H-2 and the human HLA systems (e.g. VAIMAN, 1974 ; IVANYI, 1977 ; LEVEZIEL, 1979). Its complex genetic organization and polymorphism are now under investigation : the SLA chromosomal region is composed of a yet unknown number of closely linked loci and its total size is likely to be less than 1 map unit. Serologically defined (SD) or class I antigens are controlled by genes of three allelic series at very closely linked loci (SLA-A, SLA-B and SLA-C), with more than 25 specificities (allelic forms) presently identified by the lymphocytotoxic test (e.g. HRUBAN *et al.*, 1977 ; VAIMAN *et al.*, 1979). The mixed lymphocyte reaction (MLR) is controlled by a region (SLA-D) which is distinct from but closely linked to that controlling the class I antigens (VAIMAN *et al.*, 1973 ; BRADLEY *et al.*, 1974) : SLA-D is very likely to be localized outside the SLA-A, B, C region, as indicated by recombination studies of VAIMAN *et al.* (1979) and PENNINGTON *et al.* (1981 b). Class II (or Ia) antigens have also been found in the pig and are coded for by genes closely associated with those controlling the MLR (VAIMAN *et al.*, 1975 ; LUNNEY & SACHS, 1979 ; CHARDON *et al.*, 1981 ; PENNINGTON *et al.*, 1981 b). In addition, the SLA complex influences the level of serum hemolytic complement activity (VAIMAN *et al.*, 1978 b) and SLA-like antigens have been demonstrated on boar spermatozoa (VAIMAN *et al.*, 1978 a). Current views on the genetic organization of the SLA chromosomal region (with the five loci A, B, C, D, and DR), the specificities so far recognized (more than 40) and the strong linkage disequilibria exhibited by the SLA genes have been summarized by RENARD *et al.* (1982).

Besides SLA antigens, genetically determined *leucocyte antigens* with different designations have also been described in early studies (e.g. SIMON & HRUBAN, 1971 ; SCHMID & CWIK, 1972) and it is not known whether all or some of them belong to the SLA system. An alloantigen distinct from serologically determinable SLA antigens has been detected on lymphocytes by HRUBAN *et al.* (1978) and designated SLB : the SLB locus has been shown to be independent of the SLA complex of loci. Six different B-lymphocyte-specific antigens, provisionally designated Mü-SB 1, ..., Mü-SB 6, have been identified by CWIK *et al.* (1981) : they follow an autosomal and codominant mode of inheritance and are independent of the SLA system. Moreover, the presence of red blood cell antigenic substances from the A, E, G and N systems has been demonstrated on leucocytes and other tissue cells : e.g. HÁLA (1967), SIMON & HOJNÝ (1972), SIMON & HRUBAN (1972), and HRUBAN *et al.* (1972). The results of HRUBAN *et al.* (1974) indicate the survival of skin grafts to be apparently influenced

by differences in the E blood group system and this highly polymorphic system might represent a minor histocompatibility system according to IVANYI (1977) and PAZDERA *et al.* (1981). Furthermore, PENNINGTON *et al.* (1981 a) have presented evidence for one or two non-SLA-linked immune response gene(s) controlling the rejection of SLA-identical renal allografts.

The term allotype refers to genetic variants of soluble plasma proteins which can be detected by the application of immunological techniques. Several classes of *allotypes* are presently known in the pig. Two γ-globulin (immunoglobulin) allotypes, controlled by codominant alleles (Gl^a and Gl^b), have been first demonstrated by RASMUSEN (1965 a). In the study of NIELSEN (1972), four immunoglobulin allotypes (a, b, c, d) have been found and they appear to be simply inherited and controlled by multiple genes at a single autosomal locus or at very closely linked loci. The genetic control of nine antigenic markers of immunoglobulins (A_1, A_2, A_3, B_1, B_2, C_1, C_2, D_1 and D_2 allotypes) has been investigated by RAPACZ & HASLER-RAPACZ (1982). Among the three allotypic antigens detected by OISHI *et al.* (1979 a), two belong to the β-globulin electrophoretic fraction and are controlled by alleles of the PSA-I locus, while the third one belongs to the α-globulin fraction and is controlled by a gene of the PSA-II locus. Possible identities between these three allotypes and those previously reported by LANG (1970), TIKHONOV *et al.* (1970) and DUNIEC (1972) are discussed by OISHI *et al.* (1979 a). However, the most extensively studied class of pig allotypes involves blood serum low density β-lipoproteins (LDL) which exhibit a marked antigenic polymorphism in this species. Most of LDL allotypic specificities are determined by a series of codominant alleles at an autosomal locus, designated Lpb (formerly Lpp) : e.g. RAPACZ *et al.* (1970), RAPACZ (1974), RAPACZ *et al.* (1976), ANDRESEN *et al.* (1976), RAPACZ *et al.* (1978), and HOJNÝ & DUNIEC (1980). In addition to the Lpb system, four other serum lipoprotein systems (Lpr, Lps, Lpt and Lpu) have been identified by RAPACZ & HASLER-RAPACZ (1980). The current status of the immunogenetic polymorphism controlled by these five systems is summarized by RAPACZ (1982).

No additive genetic variation for the *immune responsiveness* of pigs to sheep erythrocytes could be detected within breed by RADZIKOWSKI *et al.* (1974), though significant differences between breeds have been found in this study. In later studies, evidence for within-breed additive genetic variation has been presented with respect to the immune response of pigs to bovine or human serum albumin (HUANG, 1978 ; HYLDGAARD-JENSEN, 1979) and to some *E. coli* antigens (EDFORS-LILJA *et al.*, 1982). In addition, MEYER *et al.* (1982) have successfully performed selection for increased antibody forming capacity to dinitrophenyl (DNP)-hapten in a pig line. Genes (Ir-Lys) located within or near to the SLA chromosomal region are involved in the humoral immune response against egg-white lysozyme according to VAIMAN *et al.* (1978 c).

XIII. Biochemical polymorphism

The majority of the polymorphic protein systems presently known in farm animals have been detected by means of starch gel electrophoresis as introduced by SMITHIES (1955). *Electrophoretic variants of proteins* have been first described in pig blood serum by ASHTON (1957, 1960) and KRISTJANSSON (1960 a, 1960 b, 1961) and thereafter our knowledge

on the pig protein polymorphisms and their genetic control has regularly extended as it can be seen in the successive reviews of OGDEN (1961), BUSCHMANN (1965 a), LUSH (1966), BUSCHMANN & SCHMID (1968), BOUW & OSTERLEE (1969), STORMONT (1970), LARSEN (1971), KLUCINSKI (1973), MC DERMID *et al.* (1975), WIDAR *et al.* (1975), HYLDGAARD-JENSEN & JØRGENSEN (1978), OISHI (1979) and GAHNE (1979).

Most of the polymorphic loci involve blood proteins and a list of *serum proteins* and *red cell enzymes* found to be polymorphic in the pig is given in tables 6 and 7, respectively. These protein variants are generally controlled by codominant genes so that the genotype of an animal can be established directly from the phenotype. Exceptions are the red cell adenosine deaminase and the serum albumin systems for which four phenotypes are governed by two codominant alleles (*A* and *B*) and a recessive « null » allele (*O*).

Several genetic polymorphisms of proteins have been found by electrophoresis in other body fluids than blood or in tissue extracts. Since the first observations of GLASNÁK (1966), genetic variants of the sow's *milk proteins* have been reported, as reviewed by LARSEN (1971) and LYSTER (1972). The three main casein fractions (α-Cn, β_1-Cn and β_2-Cn) have been shown to be polymorphic ; see GLASNAK (1966, 1968 a, 1968 b), GERRITS *et al.* (1969) and KEMMER *et al.* (1972). Genetic variants have also been identified for whey proteins of porcine milk : β-lactoglobulin (β-Lg) (KEMMER, 1969 ; KRAELING & GERRITS, 1969 ; BELL *et al.*, 1981 a), α-lactalbumin (α-La) (SCHMIDT & EBNER, 1972 ; BELL *et al.*, 1981 b), and the so-called $whey_2$ protein (ALTHEN & GERRITS, 1972). Several polymorphic proteins of the seminal plasma of boars have been detected by DOSTÁL (1968, 1970) and DOSTÁL *et al.* (1976). One of the systems of lactate dehydrogenase isoenzyme (LDH-C) is polymorphic in boar spermatozoa (VALENTA *et al.*, 1967). Apparently the same polymorphism of PHI enzyme as in red blood cells is demonstrable in extracts of liver (TARIVERDIAN, 1970) and in muscle (GEE & NOLTMANN, 1981). Sorbitol dehydrogenase has been studied with kidney as a source material and this enzyme (SDH) is polymorphic with two codominant alleles (OPT HOF *et al.*, 1972). Genetic variants of pancreatic proteinase, controlled by two loci (PPr_1 and PPr_2), have been identified by TAKAHASHI *et al.* (1974).

XIV. Linkages and syntenies

Systematic search for genetic linkages in the pig has essentially dealt with polymorphic blood group and biochemical systems.

The first case of close linkage has been discovered by ANDRESEN & BAKER (1964) and involves the C and J blood group systems. Additional data on the recombination frequency between genes at the C and J loci have been provided by RASMUSEN (1965 b), ANDRESEN (1966 a) and MUIR & RASMUSEN (1974) : the combined estimate of recombination frequency given by the latter authors is 5.7 ± 0.8 p. 100. Thereafter, linkage of the pig main histocompatibility complex (SLA) and the J blood group system has been demonstrated by HRUBAN *et al.* (1976), with a recombination frequency of 9.8 p. 100. As linkage between the C blood group locus and the SLA region has been further confirmed by HRUBAN *et al.* (1977), the linkage group including the C and J blood group loci and the SLA complex of loci (see above for more details on the SLA region) is established : the map order of these markers is SLA-J-C according to HRADECKÝ *et al.* (1982).

TABLE 6

A list of electrophoretically detectable variants of blood serum proteins in the pig.

Liste de variants de protéines sériques du Porc détectables par électrophorèse.

Protein	Locus symbol	No of alleles	Usual allelic symbols	References
Alkaline phosphatase	Akp	3 (at least)	*Akp^A*, *Akp^B*, *Akp^C*, ...	DINKLAGE (1970b), KIEREK-JASZEZUK *et al.* (1978), BATRA and KHANNA (1981)
Albumin	Alb (Alb$_1$)	3	*Alb^A*, *Alb^B*, *Alb^O*	KRISTJANSSON (1966)
Amylase	Am (Am$_1$) Am$_2$	4 2	*Am^A*, *Am^{BF}*, *Am^B*, *Am^C* *Am$_2{}^A$*, *Am$_2{}^B$*	IMLAH (1965), HESSELHOLT (1970) JUMKOV and NIKONCHIK (1977)
Ceruloplasmin	Cp	3	*Cp^A*, *Cp^B*, *Cp^C*	IMLAH (1964), OISHI *et al.* (1980b)
Esterases	Es EsII	2 3	*Es^A*, *Es^B* *EsII^D*, *EsII^E*, *EsII^F*	KUBEK (1970) GRUNDER and KRISTJANSSON (1974)
Hemopexin	Hpx (Hp)	8	*Hpx^O*, *Hpx^{1F}*, *Hpx^1*, *Hpx^2* *Hpx^{3F}*, *Hpx^3*, *Hpx^4*, *Hpx^5*	KRISTJANSSON (1961), HESSELHOLT and HRISTIC (1966), OISHI *et al.* (1980b)
Prealbumin (Protease inhibitor-1)	Pa (Pi-1)	2	*Pa^A(Pi-1^A)*, *Pa^B(Pi-1^B)*	KRISTJANSSON (1963), JUNEJA and GAHNE (1981)
Protease inhibitor-2	Pi-2	(3)		GAHNE and JUNEJA (1982)
Postalbumin	Pa$_1$	2	*Pa$_1{}^A$*, *Pa$_1{}^B$*	KUBEK and MATOUŠEK (1970)
Postalbumin-1	Po-1	(6)		JUNEJA and GAHNE (1978), GAHNE and JUNEJA (1982)
Postalbumin-2	Po-2	2	*Po-2^F*, *Po-2^S*	JUNEJA *et al.* (1982)
Sα_2–macroglobulin	Sα_2	3	*S$\alpha_2{}^A$*, *S$\alpha_2{}^B$*, *S$\alpha_2{}^C$*	SCHRÖFFEL (1965)
Transferrin	Tf	6	*Tf^A*, *Tf^B*, *Tf^C*, *Tf^D*, *Tf^E*, *Tf^I (= Tf^P)*	KRISTJANSSON (1960b), IMLAH (1965), GLASNÁK *et al.* (1976), SKLADANOWSKA *et al.* (1979), STRATIL *et al.* (1982)

TABLE 7

A list of electrophoretically detectable variants of blood cell enzymes (*) in the pig.

Liste de variants d'enzymes des cellules sanguines du Porc détectables par électrophorèse.

Enzyme	Locus symbol	No of alleles	Usual allelic symbols	References
Acid phosphatase	Acp	(2)	*(Acp^A, Acp^B)*	MEYER and VERHORST (1973)
Adenosine deaminase (§)	Ada	3	*Ada^A, Ada^B, Ada^O*	ANANTHAKRISHNAN and WALTER (1973), WIDAR *et al.* (1974), HYLDGAARD-JENSEN and WEGGER (1977)
Carbonic anhydrase	Ca (Ca II)	2	*Ca^A, Ca^B*	KLOSTER *et al.* (1970)
Catalase	Cat	2 (at least)		BARANOV (1970)
Esterase-D	Es-D	2	*$Es\text{-}D^A$, $Es\text{-}D^B$*	TANAKA *et al.* (1980), HYLDGAARD-JENSEN and THORUP (1981)
Glucose-6-phosphate dehydrogenase	Gpd (G6pd)	2	*Gpd^A, Gpd^B*	VERHORST (1973)
Peptidase C	PepC	(2)	*($PepC^F$, $PepC^S$)*	SAISON (1973)
6-phosphogluconate dehydrogenase	Pgd (6Pgd)	2	*Pgd^A, Pgd^B*	DINKLAGE (1969), SAISON and GIBLETT (1969)
Phosphoglucomutase :				
erythrocytes	Pgm (Pgm_2)	2	*Pgm^A, Pgm^B*	SAFAROVA *et al.* (1972)
leucocytes	Pgm_3	2	*$Pgm_3{}^F$, $Pgm_3{}^S$*	PRETORIUS *et al.* (1977)
Phosphohexose isomerase (= Glucose phosphate isomerase)	Phi (Gpi)	2	*Phi^A, Phi^B*	KUBEK and DINKLAGE (1971), SAISON and O'REILLY (1971)

(*) Red cell enzymes if not specified otherwise.
(§) With regard to Ada phenotypic patterns in leucocytes, see WIDAR and ANSAY (1975) and HYLDGAARD-JENSEN (1981).

Linkage between the K blood group locus and the Hpx locus for hemopexin has been demonstrated by IMLAH (1965), ANDRESEN (1966 b) and HESSELHOLT & NIELSEN (1966). Combining segregation data from these three studies and their own study, HAGEN *et al.* (1968) have estimated at 3.8 ± 0.5 p. 100 the recombination frequency between K and Hpx.

Close linkage between the Am locus for serum amylase and the I blood group locus has been found by ANDRESEN (1966 c) and NIELSEN (1966) : recombination frequency was 0.8 and 2.5 p. 100, respectively.

The H blood group locus has been found to be closely linked to the Pgd locus for 6-phosphogluconate dehydrogenase and to the Phi locus for phosphohexose isomerase by ANDRESEN (1970 a, b). The recombination frequency between the two enzyme loci has been found higher than those between H and Phi and H and Pgd by the same author (ANDRESEN, 1971), according to whom the most likely order of the three loci is Phi-H-Pgd.

As first suggested by JØRGENSEN *et al.* (1976) on the basis of strong associations between halothane sensitivity and PHI phenotypes in several *Landrace* populations, close linkage between the Hal locus and the Phi locus has been demonstrated by ANDRESEN & JENSEN (1977, 1978 a) from segregation results in families. Mapping of the chromosomal region comprising the Hal, H, Pgd and Phi loci is currently under investigation. The following recombination frequencies (in p. 100) in this linkage group have been reported :

H-Pgd 3.4 (ANDRESEN 1970 a) ; 4.0 (RASMUSEN *et al.*, 1980) ; 5.2 ± 1.4 (JØRGENSEN, 1981).

H-Phi 2.6 (ANDRESEN, 1970 b) ; 1.4 (RASMUSEN *et al.*, 1980) ; 12.3 (IMLAH, 1980) ; 4.5 ± 1.7 (JØRGENSEN, 1981).

Pgd-Phi 8.1 (ANDRESEN, 1971) ; 6.25 (OISHI & ABE, 1979) ; 4.8 (RASMUSEN *et al.*, 1980) ; 12.4 ± 4.6 (JØRGENSEN, 1981) ; 0.56 (GUÉRIN *et al.*, 1983).

H-Hal 7.4 (IMLAH, 1980) ; 3.0 ± 2.3 (JØRGENSEN, 1981).

Pgd-Hal 9.1 ± 6.3 (JØRGENSEN, 1981) ; 1.18 (GUÉRIN *et al.*, 1983).

Phi-Hal 6.9 (IMLAH, 1980) ; no recombinant (JØRGENSEN, 1981) ; 0.73 (GUÉRIN *et al.*, 1983).

Note that the figures reported above for the study of RASMUSEN *et al.* (1980) correspond to the recombination frequencies pooled over the two sexes.

That the Hal locus is situated between the H and Phi loci has been deduced from relative linkage disequilibrium estimates by ANDRESEN (1979 a) and this hypothesis has been further accepted by RASMUSEN *et al.* (1980), IMLAH (1980) and JØRGENSEN (1981) : the most likely order is therefore Phi-Hal-H-Pgd according to these authors. However, GUÉRIN *et al.* (1983) have provided data in favour of the alternative order Hal-Phi-Pgd in this linkage group. In addition, as suggested by RASMUSEN *et al.* (1980), there might be a locus (S) closely linked to but separate from the H locus for inhibition of expression of A-O blood group phenotypes : according to ANDRESEN (1981) and RASMUSEN (1981), data obtained so far are consistent with the following map order for the five loci : Phi-Hal-S-H-Pgd.

The locus Po-2 responsible for electrophoretic variants of a blood serum protein (postalbumin-2) has recently been assigned to the latter linkage group, with a recom-

bination frequency of 3.2 p. 100 between Po-2 and Phi and with the Po-2 locus being probably located between the H and Pgd loci (JUNEJA *et al.*, 1982). On the other hand, evidence for a loose linkage between the H blood group locus and the C and J blood group loci has been presented by RASMUSEN (1982), the frequencies of recombination being 42.4 ± 3.0 p. 100 for H and C and 41.7 ± 3.5 p. 100 for H and J. All things together, it appears that nine polymorphic loci or systems, i.e. Hal, Phi, S, H, Po-2, Pgd, C, J and SLA, would be localized on the same chromosome : however, further data are needed to determine the exact gene order of this linkage group.

The loci for postalbumin-1 (Po-1) and protease inhibitor-1 (Pi-1) are closely linked according to GAHNE and JUNEJA (1982).

Possible linkage between two coat colour loci, E (extension) and Be (belt), has been reported by BUSHNELL (1943). Loose linkage between the B blood group locus and the Pa locus for serum prealbumin has been suggested by ANDRESEN (1968). There is some evidence of linkage between the genes controlling the different casein fractions of the sow's milk, according to GLASNÁK (1968 b). An early lethal factor has been found to be linked to the Tf locus for transferrin (IMLAH, 1970 a). Linkage between genes controlling different immunoglobulin (IgG) allotypes has been shown by RAPACZ & HASLER-RAPACZ (1974, 1982). Close linkage between the SLB locus and the L blood group locus has been demonstrated by HRUBAN *et al.* (1978). Among the five lipoprotein allotype loci identified so far, three, i.e. Lpb, Lpt and Lpu, are closely linked, as reported by RAPACZ (1982).

Somatic cell hybridization techniques have recently been used for pig gene mapping. The first synteny, i.e. location on the same chromosome, for the genes controlling glucose-6-phosphate dehydrogenase (G6PD), hypoxanthine guanine phosphoribosyltransferase (HPRT) and phosphoglycerate kinase (PGK) has been found by GELLIN *et al.* (1980) and this syntenic group is to be assigned to the X chromosome according to FÖRSTER *et al.* (1980) and LEONG *et al.* (1982 a), as in all the mammalian species studied so far. The latter authors have also localized the gene for pig GLA (α-galactosidase) on the X chromosome.

In addition to the above mentioned enzyme loci, the other cases of sex-linkage so far reported in pigs concern the splayleg condition (LAX, 1971) and the recessive gene responsible for the congenital tremor A III (HARDING *et al.*, 1973).

The genes for pyruvate kinase (PKM2), mannose phosphate isomerase (MPI) and purine nucleoside phosphorylase (NP) have been shown to be syntenic by GELLIN *et al.* (1981).

The gene for superoxide dismutase (SOD1) has been assigned to the chromosome 9 of the pig karyotype by LEONG *et al.* (1982 b).

The study of linkage between natural or induced chromosomal rearrangements and blood group or biochemical markers has led FRIES *et al.* (1982) to provisionally assign the G blood group locus to the pig chromosome 15. On the other hand, a loose linkage between the G and Hal loci had formerly been suggested by JØRGENSEN (1979) on the basis of associations between halothane sensitivity and G blood group types. From this result and some of their own findings, FRIES *et al.* (1982) have suggested that, besides the G locus, the Phi-Hal-S-H-Pgd linkage group might also be located on the chromosome 15.

XV. Population studies

A large number of studies have been devoted to estimating allelic frequencies in a variety of pig populations for the well-known polymorphic loci, particularly those controlling erythrocyte antigens, serum proteins and red blood cell enzymes.

In most studies, the populations involved are usual breeds or strains of *Sus scrofa domestica* : see, among others, GAVALIER *et al.* (1966), BAKER (1968), SMITH *et al.* (1968), DINKLAGE & GRUHN (1969), MAJOR *et al.* (1970), WILLER & NEUFFER (1970), MEYER (1973), VERHORST *et al.* (1974), WIDAR *et al.* (1975), AGERGAARD *et al.* (1976, 1977), VAN ASTEN & BUIS (1977), and OISHI *et al.* (1978, 1979 b). Available information on gene frequencies at seven polymorphic loci (E and H blood groups, Ada, Pgd, Phi, Hpx and Tf) has been summarized by FRANCESCHI & OLLIVIER (1981). Incidence of halothane sensitivity has also been extensively studied and the frequency of *Hal*^{s} gene varies to a large extent in domestic pig breeds, as reviewed by FRANCESCHI & OLLIVIER (1981) and WEBB *et al.* (1982). Particular attention has been given to breed differences in gene and haplotype frequencies for the linkage group containing the Hal, Phi, H and Pgd loci : e.g. GUÉRIN *et al.* (1978, 1980), ANDRESEN (1979 c), JØRGENSEN & HYLDGAARD-JENSEN (1981), CEPICA *et al.* (1981, 1982), ANDRESEN *et al.* (1981 b), and VÖGELI & SCHWÖRER (1982). As regards the pig major histocompatibility complex, present evidence on SLA haplotype frequencies in various breeds has been reviewed by RENARD *et al.* (1982).

Some studies have dealt with gene frequencies (especially for blood group systems) in pig populations such as native breeds or strains of miniature pigs (e.g. GRUHN & DINKLAGE, 1971 ; TIKHONOV & RATIANY, 1973 ; OISHI & TOMITA, 1976 ; and OISHI *et al.*, 1980 a) and populations of wild pigs (subspecies of *Sus scrofa*) distributed throughout the Eurasian continent (e.g. BUSCHMANN, 1965 b ; WIATROSZAK, 1970 ; TIKHONOV *et al.*, 1972, 1974 ; and KUROSAWA *et al.*, 1979) or the North-American continent (e.g. SMITH *et al.*, 1980).

XVI. Performance traits

Pig is primarily, if not exclusively, used as a source of animal proteins in human food and has a great economic importance in agriculture. As a consequence much attention has been devoted to the genetic control of the components of the overall efficiency of pigmeat production and, since LUSH (1937), to the application of genetics to the improvement of traits such as prolificacy, viability, growth rate or lean meat content of the carcass (e.g. LEGAULT & OLLIVIER, 1974 ; JONSSON, 1975 ; CUNNINGHAM, 1976 ; FOWLER *et al.*, 1976 ; FREDEEN, 1980). Most of these traits show a continuous variation and the genetically controlled part of this variation depends on gene differences at many loci, the effects of which are not individually distinguishable. The methods of quantitative genetics (FALCONER, 1981 ; OLLIVIER, 1981) are relevant to the study of the polygenic inheritance of performance traits and have been extensively used. In the present review, no attempt will be made to give an exhaustive survey of this vast field of research. Our aim is only to summarize the current views on the nature of the genetic variation of quantitative traits and to guide the reader towards pertinent literature through a list of papers and reviews. Particular attention will be drawn to the respective importance of additive gene effects, as statistically assessed by heri-

tability (h^2), and of non-additive gene effects (mainly dominance), as assessed by inbreeding depression and heterosis effect which respectively correspond to decreased and increased heterozygosity. Special emphasis will be put on the known influences of marker genes on performance traits (SPOONER, 1974 ; GAHNE, 1979 ; OISHI, 1979).

A. *Reproductive traits*

Prolificacy is a major component of reproductive performance of the pig. Additive genetic variance of number of youngs per litter, at farrowing or at weaning, is of low magnitude and heritability estimates are generally comprised between 5 and 15 p. 100 for litter size : e.g. URBAN *et al.* (1966), LEGAULT (1970), STRANG & KING (1970), REVELLE & ROBISON (1973), STRANG & SMITH (1979), ALSING *et al.* (1980), JOHANSSON (1981), JOHANSSON & KENNEDY (1982), and review by BOLET & LEGAULT (1982). In contrast, litter size is one of the traits which are the most affected by inbreeding depression (e.g. BERESKIN *et al.*, 1968, 1973) and by heterosis in breed crosses, as reviewed by SELLIER (1970, 1976) and JOHNSON (1981), these effects arising from the modified heterozygosity of the genotypes of both mother and youngs of the litter. Non-additive gene effects appear to be mainly responsible for the genetic variation of litter size or at least of prenatal and postnatal survival rate. Indeed *ovulation rate*, which is an entirely maternal component of litter size, has a moderately high heritability (30-40 p. 100 : e.g. YOUNG *et al.*, 1978 ; LEGAULT & GRUAND, 1981) and has been successfully selected for (CUNNINGHAM *et al.*, 1979 ; PUMFREY *et al.*, 1980). On the other hand, ovulation rate does not exhibit heterosis to a significant extent (e.g. JOHNSON *et al.*, 1978 ; LEGAULT & GRUAND, 1981). Several specific genetic causes of *prenatal mortality* have been recognized. According to MC FEELY (1967), chromosomal abnormalities may explain about one third of the early embryonic mortality which itself affects one third of the fertilized eggs. Various immunogenetic incompatibilities and lethal factors also play a role in determining litter size through their incidence on fertilization rate and embryonic or neonatal loss (HANLY, 1961 ; ANDRESEN & BAKER, 1963 ; BISHOP, 1964 ; MATOUŠEK, 1970 ; IMLAH, 1970 a ; DUNNE, 1975 ; GAHNE, 1979). Lowered fertility and sterility of adult breeding animals, in some cases, result from aberrant chromosome complements : see FECHHEIMER (1981) for review.

The relationships between *marker loci* and *reproductive traits* have often been investigated. At least two studies (JENSEN *et al.*, 1968 ; RASMUSEN & HAGEN, 1973) support the hypothesis that genes at the H blood group locus significantly influence litter size, with a detrimental effect associated with the H^a allele. The A blood group system, possibly because of the interrelationships between the A and H systems (see above), seems to be also involved, as well as other blood group factors to a lesser extent : e.g. DINKLAGE & HOHENBRIK (1970), IMLAH (1972 b), KENNEDY *et al.* (1973), HAGEN & RASMUSEN (1974), and RASMUSEN (1975 b). Some authors have found associations between litter size or fertility of females and genes at the Tf locus for serum transferrin : e.g. KRISTJANSSON (1964), KUZMENKO (1968 a, 1968 b), IMLAH (1970 a), TRIPATHI & HOWELL (1974), and RADOVIC (1974) ; however, such associations have not been detected in other studies : e.g. JENSEN *et al.* (1968), FÉSÜS & RASMUSEN (1971), KAWECKI *et al.* (1974), and HUANG & RASMUSEN (1982). Associations have also been reported between reproductive traits and the SLA complex (KRISTENSEN *et al.*, 1980 ; VAIMAN & RENARD, 1980 ; RENARD *et al.*, 1982) or the monogenic halothane sensitivity (see WEBB *et al.*, 1982).

The genetic control of other characters conditioning the *reproductive ability* of breedings animals has also been investigated. Several aspects, such as intersexuality and other defects of the reproductive tract, the occurrence of abnormal teats and leg weakness, have been dealt with in previous sections. Age at puberty in gilts has a moderate heritability (around 30 p. 100) and generally exhibits heterosis in breed crosses : see REUTZEL & SUMPTION (1968), YOUNG *et al.* (1978), LEGAULT & GRUAND (1981), HUTCHENS *et al.* (1981), and the reviews of heterosis estimates by SELLIER (1976) and JOHNSON (1981). Measurements of fertility of the breeding sow, such as conception rate or weaning to fertile mating interval, appear to be much more affected by non-additive than by additive genetic effects : e.g. LEGAULT *et al.* (1975), AUMAITRE *et al.* (1976), JOHNSON *et al.* (1978), and FAHMY *et al.* (1979). Estimates of heritability for teat number range from 10 to 40 p. 100 and average approximately 30 p. 100, while heterosis effect is unimportant for this trait : e.g. ENFIELD & REMPEL (1961), SKJERVOLD (1963), HANSET & CAMERLYNCK (1974), PUMFREY *et al.* (1980), and CLAYTON *et al.* (1981). Gestation length is moderately to highly heritable : e.g. COX (1964), FAHMY & BERNARD (1972), and GARNETT & RAHNEFELD (1979). Quantitative and qualitative aspects of semen production, as well as fertility, sexual precocity and libido of the boar, are affected, to some extent, both by additive and non-additive genetic effects : e.g. CLEM *et al.* (1967), HUHN (1970), DU MESNIL DU BUISSON *et al.* (1974), FAHMY & HOLTMANN (1977), COUROT & LEGAULT (1977), WILSON *et al.* (1977), CONLON & KENNEDY (1978), LEGAULT *et al.* (1979), and NEELY *et al.* (1980). Genetic aspects of specific causes of postnatal mortality, namely diseases and stress-induced sudden death (malignant hyperthermia syndrome), have been reviewed above.

B. *Growth and body composition traits*

Since the pioneer work of LUSH (1936) on the *Danish Landrace* breed, additive genetic variation of *growth traits* (average daily gain, efficiency of food utilization, age at slaughter) and *body composition traits* has been studied in a large variety of breeds, especially by analysing data of central testing stations : e.g. JOHANSSON & KORKMAN (1950), OSTERHOFF (1956), SMITH *et al.* (1962), SMITH & ROSS (1965), LANGHOLZ (1966), JONSSON (1963, 1965, 1974), FLOCK (1970), OLLIVIER (1970), SIERS & THOMSON (1972), HANSET & VAN SNICK (1972, 1973), PFLEIDERER (1973), PUFF (1975), SWIGER *et al.* (1979), KINTABA *et al.* (1981), OLLIVIER *et al.* (1981), OLLIVIER & DERRIEN (1981), and KENNEDY *et al.* (1982). The heritability of postweaning growth rate and food conversion ratio is around 30 p. 100. Daily food consumption (appetite) under *ad libitum* feeding is moderately to highly heritable : e.g. MC PHEE *et al.* (1979) and WYLLIE *et al.* (1979). Estimates of heritability for carcass composition traits such as lean and fat percentages, lean to fat ratio, backfat thickness and loin eye area are generally in the neighbourhood of 50 p. 100. Heritability of carcass length is somewhat higher (about 60 p. 100), as well as that of number of vertebrae (e.g. BERGE, 1948 ; FREDEEN & NEWMAN, 1962). Additive gene effects are therefore a major source of variation of growth and carcass traits and large genetic gains for these two groups of traits have been achieved in several one-trait or multiple-trait selection experiments, as reviewed by GLODEK (1982). However, while carcass composition traits are not significantly influenced by inbreeding and heterosis, growth rate and feed efficiency are detrimentally affected by inbreeding and generally exhibit heterosis in breed crosses, indicating that non-additive gene effects are involved in the genetic control of the latter traits : see, for instance, KING & ROBERTS (1959), BERESKIN *et al.* (1968), MIKAMI *et al.* (1977) and LEYMASTER & SWIGER (1981) for inbreeding effects and

the surveys of heterosis estimates given by SELLIER (1970, 1976) and JOHNSON (1981). Overall feed efficiency of the growing pig depends on several components (digestive utilization of nutrients, maintenance requirements, growth requirements) : available evidence on genetic influences on each of these components has been reviewed by OLLIVIER & HENRY (1978). Additive genetic variation appears to be of low to moderate magnitude for type, size and number of muscle fibres : e.g. STAUN (1972) and MILLER *et al.* (1975). Intramuscular fat content is highly heritable (MALMFORS & NILSSON, 1979), as well as the lipid content of the muscle (DUNIEC *et al.*, 1961). There is limited evidence that fatty acid composition and content of adipose tissue are influenced by additive genetic factors (KELLOGG *et al.*, 1977).

The phenomenon of *muscular hypertrophy*, which first appeared in the Belgian *Piétrain* breed, is a major feature of the genetic variation of body tissue composition in the pig and, as stated earlier, its hereditary determination is likely to be monogenic. A strong genetic association exists between *halothane sensitivity* and most carcass characters, especially those related to muscular development and muscle to bone ratio. At a given slaughter liveweight, halothane reactors give a significantly heavier, shorter and leaner carcass than non-reactors : e.g. EIKELENBOOM & MINKEMA (1974), MONIN *et al.* (1976, 1981), EIKELENBOOM *et al.* (1978), OLLIVIER *et al.* (1978), WEBB & JORDAN (1978), CARLSON *et al.* (1980), LAMPO (1981 b), and other reports reviewed by WEBB *et al.* (1982). In addition, there are indications that the heterozygote at the Hal locus may be intermediate between the two homozygotes with respect to killing out percentage and carcass lean content : see EIKELENBOOM *et al.* (1980), JENSEN & ANDRESEN (1980), SCHNEIDER *et al.* (1980), JENSEN (1981), and WEBB (1981). The halothane gene, which is recessive for halothane sensitivity, would therefore be approximately additive for its effect on muscular development and body composition traits. Relationships between the latter traits and genotypes at the H and Phi loci, closely linked to Hal, have also been reported (e.g. ANSAY & OLLIVIER, 1978 ; ANDRESEN & JENSEN, 1979 ; GUÉRIN *et al.*, 1979) : they may be explained by strong linkage disequilibria between the H or Phi locus and the Hal locus in the breeds involved.

As to *other marker loci*, no association with growth and body composition traits has been repeatedly found. Slight influences of various genetic systems have occasionally been reported but other authors have failed to detect any significant relationship : see BALTZER (1964), JENSEN *et al.* (1968), TRIPATHI & HOWELL (1969), KRAELING *et al.* (1971), GAVALIER (1972), KENNEDY *et al.* (1973), EBERMANN *et al.* (1973), WIATROSZAK (1974), LENGERKEN & PFEIFFER (1974), BERNDT *et al.* (1978), and CAPY *et al.* (1981).

C. *Meat quality traits*

Since 20 years, an increasing attention has been paid to a problem of great concern to the pork industry, namely the occurrence of *abnormal meat quality*, either PSE (pale, soft and exudative) meat (BRISKEY, 1964 ; BENDALL & LAWRIE, 1964) or, to a lesser extent, DFD (dark, firm and dry) meat. As stated by CHRISTIAN (1972), CASSENS *et al.* (1975) and SYBESMA & EIKELENBOOM (1978), the changes which occur in the *post mortem* conversion of muscle to meat reflect the *ante mortem* condition of the musculature and are partly genetically determined. Since the first studies of JONSSON (1963) and OLLIVIER & MESLE (1963), additive genetic variances of such traits as pH, colour and water binding capacity of meat have been estimated by several authors : e.g. PEASE & SMITH (1965), LANGHOLZ (1966), JENSEN *et al.* (1967), FLOCK (1968), WENIGER *et al.* (1970), WALSTRA *et al.* (1972), JONSSON *et al.* (1972), STAUN & JENSEN (1974),

Mc Gloughlin & Mc Loughlin (1975), Lundström (1975), Scheper (1979), Malmfors & Nilsson (1979), Schwörer *et al.* (1980), Lundeheim *et al.* (1980), and Ollivier *et al.* (1981). Heritability estimates for meat quality criteria are around 20-30 p. 100. Effects of inbreeding and heterosis on the same traits are in general unimportant : e.g. Molenaar (1976), Young *et al.* (1976), Schneider *et al.* (1982), and earlier reports reviewed by Sellier (1974).

There is no doubt that *PSE meat condition* and *porcine stress syndrome* (PSS) are associated phenomena, even though the relationship is not as simple as formerly anticipated (Cassens *et al.*, 1975). There is strong evidence that pigs which are susceptible to malignant hyperthermia syndrome, as detected by halothane sensitivity, are more likely to develop PSE meat condition : e.g. Eikelenboom & Minkema (1974), Eikelenboom *et al.* (1978), Webb & Jordan (1978), Mc Phee *et al.* (1979), Carlson *et al.* (1980), Mc Gloughlin *et al.* (1980), Jensen & Andresen (1980), Monin *et al.* (1981), and other reports reviewed by Webb *et al.* (1982). Available comparisons between the three genotypes at the Hal locus with respect to meat colour are rather controversial as far as the exact position of the heterozygote is concerned : e.g. Andresen & Jensen (1978 b), Eikelenboom *et al.* (1980), Jensen & Andresen (1980), Schneider *et al.* (1980), Andresen *et al.* (1981 a), and Webb (1981). On the other hand, higher incidences of the PSE condition of meat in animals possessing the H^a allele at the H blood group locus and (or) the Phi^B allele at the locus for PHI enzyme have been found in several studies : Barton *et al.* (1977), Andresen (1979 b), Andresen & Jensen (1979), Imlah & Thomson (1979), Watanabe *et al.* (1979), Vögeli *et al.* (1980), Jørgensen (1981), Frøystein *et al.* (1981), and Oishi *et al.* (1981). These findings are to be related to the associations observed between either H or PHI systems and halothane sensitivity (e.g. Rasmusen & Christian, 1976 ; Jørgensen *et al.*, 1976 ; Jørgensen, 1978, 1979 ; Guérin *et al.*, 1978 ; Hojny *et al.*, 1979 c ; Andresen, 1979 c, 1980 ; Vögeli & Schwörer, 1982) and can be explained by linkage disequilibria between loci of this chromosomal region in the breeds where the halothane sensitivity gene (Hal^s) is preferentially associated with the Phi^B and H^a genes.

With respect to the organoleptic quality of pigmeat, Jonsson & Wismer-Pedersen (1974), Jonsson & Andresen (1979) and Willeke *et al.* (1980) have presented evidence for appreciable additive gene effects on the intensity of « sex odour » of meat from entire males, a defect (the so-called « *boar taint* ») which is mainly due to a high concentration of a testicular steroid (androstenone) in the fatty tissue.

D. *Metabolic traits*

In recent years, a number of studies have dealt with the mode of inheritance of specific metabolic measurements, in an attempt to find new selection criteria which, in terms of potential genetic improvement, would be more efficient than the traditional « gross » measurements of performance. Examples of such genetic studies are given below.

Genetic differences have been reported for total plasma cholesterol concentration, which presents a moderate heritability : Heidenreich *et al.* (1964), Reetz *et al.* (1975), Rothschild & Chapman (1976), Wegger (1978). Soluble protein content of fat tissue is moderately to highly heritable according to Rogdakis & Strutz (1978). A lipid mobilizing factor might be genetically controlled, as suggested by Standal *et al.* (1973). Heritability estimates for hemoglobin level have been provided by Gabris (1974),

REETZ *et al.* (1975) and FAHMY & BERNARD (1978). Additive genetic effects have been reported for ceruloplasmin, copper and iron contents in blood plasma (REETZ & FEDER, 1974) and for various blood cell counts in the young piglet (GABRIS, 1974). Blood glutathione content appears to be genetically controlled according to HYLDGAARD-JENSEN (1980). There is some evidence of genetic differences in the capacity for ascorbic acid synthesis according to LUND *et al.* (1980) and PALLUDAN *et al.* (1982).

The quantitative activity of several enzymes has been studied from a genetic point of view. The arylesterase activity in blood serum shows a discontinuous variation which has been shown to be under the control of a set of multiple alleles by AUGUSTINSSON & OLSSON (1961), GAHNE (1970) and GAHNE *et al.* (1972). Activities of lipogenic enzymes (NADPH-generating dehydrogenases) in porcine adipose tissue are affected to a significant extent by additive gene effects (STRUTZ & ROGDAKIS, 1979). Evidence of genetic differences in glutathione peroxidase (GSH-Px) activity has been presented by JØRGENSEN *et al.* (1977). In the case of the adenosine deaminase (ADA) enzyme, which exhibits electrophoretic variants controlled by the Ada locus, as indicated earlier, it is worth mentioning that its erythrocytic quantitative activity seems to depend on the Ada genotype (WIDAR *et al.*, 1974 ; HYLDGAARD-JENSEN & WEGGER, 1977) and also to be associated with susceptibility to lung infections (HYLDGAARD-JENSEN, 1978). Activity of serum alkaline phosphatase is influenced by genetic effects (IMLAH, 1970 b ; LOCNISKAR *et al.*, 1975), and PRZYTULSKI *et al.* (1982) have recently shown that the activity of this enzyme is related to the genotype at the Akp locus. In the same way, it has been suggested by BATRA *et al.* (1981) that serum amylase activity level could differ according to the Am phenotype based on electrophoresis. Several studies have shown that the activity level of creatine phosphokinase (CPK) enzyme in blood serum is influenced by additive gene effects, with heritability estimates generally falling in the range 20-50 p. 100 : e.g. RICHTER *et al.* (1973), BICKHARDT *et al.* (1977), LAMPO (1980), SCHWÖRER *et al.* (1980), SCHMITTEN *et al.* (1981), and review by BICKHARDT (1981). The halothane locus plays a major role in these genetic influences as halothane reactors present elevated serum CPK activities, as compared to non-reactors : see BICKHARDT (1981) and WEBB (1981) for review. Genetic influences, in particular those involving the Hal locus, have also been reported for blood serum activity levels of enzymes such as lactate dehydrogenase and aldolase, which like CPK mainly originate from skeletal muscle (e.g. LAMPO, 1981 a ; SCHMITTEN *et al.*, 1981), and for other quantitative blood parameters such as lactic acid content (e.g. MONIN *et al.*, 1979).

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Résumé

La génétique du porc : mise au point

Une mise au point générale sur la génétique du porc est faite. Les principaux domaines couverts sont : caryotype et anomalies chromosomiques, couleur de la robe, anomalies héréditaires, résistance aux maladies, immunogénétique, polymorphismes biochimiques, « linkages » et synténies, caractères quantitatifs d'importance économique. Sur ce dernier point, une attention particulière est accordée aux influences connues de gènes marqueurs sur les caractères zootechniques. Des listes de gènes gouvernant la couleur de la robe, les groupes sanguins (antigènes érythrocytaires) et les variants électrophorétiques de protéines sériques et d'enzymes des cellules sanguines sont présentées dans des tableaux. Une liste de 29 anomalies génétiques qui peuvent être considérées comme probablement monogéniques chez le porc est également donnée. Le nombre total de locus actuellement identifiés avoisine 100.

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