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PORCINE IMMUNE RESPONSE AS MARKER TRAITS FOR SELECTIVE BREEDING

By

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Thesis submitted in partial fulfilment of the requirement for the degree of

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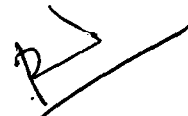

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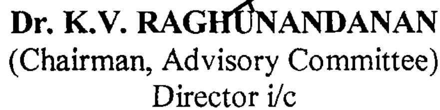

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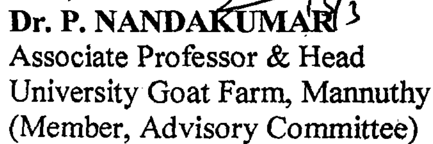

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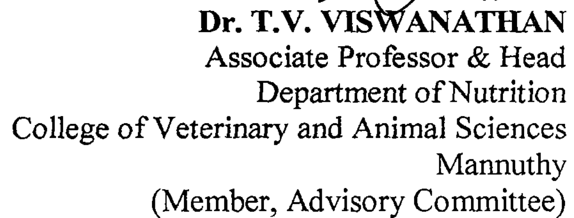

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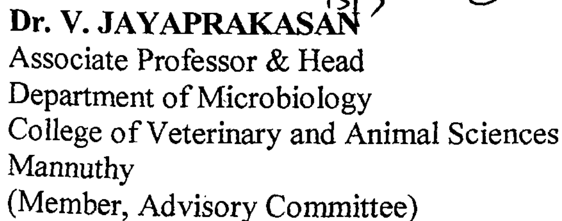

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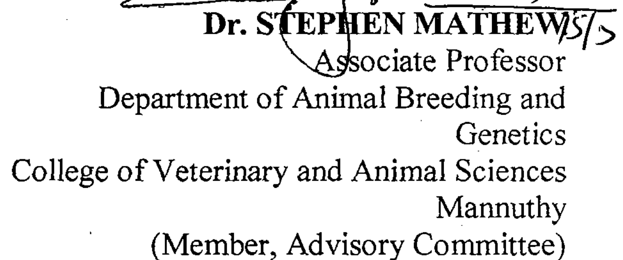

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INTRODUCTION

1. INTRODUCTION

The science and practice of pig production has changed dramatically over recent decades. The modern day pig industry envisages consideration of novel issues and opportunities to improve the quality of the end product and in the environmental impacts of pig production rather than considering it as a mere source of animal protein. This has been achieved through decades of intensive improvements and modifications in feeding, breeding, housing and management of pigs.

One of the greatest challenges that has arisen from these changes is the adaptation of temperate breeds to counteract the tropical stress and thereby improve the disease resistance capability of these breeds. The tradition in the practice of pig science has always focussed mainly on these two factors which appeared to have overwhelming influence on pig production. Genetic selection for improved pork production efficiency has resulted in substantially different pig populations. Initially, selection focussed on the more highly heritable post-weaning performance traits including growth rate, feed conversion and carcass lean percentage.

Several concepts are crucial to the discussion of genetic selection and in defining the genetics of a population. Genetic evaluation must be directed towards quantifying the underlying biological traits rather than conventionally measured performance traits. The selection criterion can be either a designed selection index,

a function of performance traits or BLUP genetic merit estimates or composite fitness traits like immune response traits.

Immune response, the strategic defense system in the fight against antigenic and microbial invasions is of primary importance to the survival and optimum performance of an animal. Immune response may be broadly defined as the cellular and humoral responses of the body to certain intrinsic and extrinsic factors. An animal's immune system basically consists of three major facets as phagocytosis, cell-mediated and humoral immunity which are preformed respectively by macrophages, T-lymphocytes and B-lymphocytes and each of these functions are specialised in the protection against some infections and scarcely effective or ineffective against others. In spite of the functional integration of these three cell types, basically involved in the three immune functions, they are distinct protagonists under separate polygenic control.

Selection for immune responsiveness and disease resistance has often been ignored by animal geneticists because of the difficulty in measuring these traits. Genetic considerations involved with selection and testing for disease resistance and immune responsiveness require the knowledge of associations between disease resistance, immune responsiveness and production traits. Serum gammaglobulin levels have often been associated with health, better performance and survival of all species of livestock. As early as 1960, Ross *et al.* demonstrated that the lines of Zebu cattle resistant to helminthiasis had higher serum gammaglobulin levels compared to susceptible lines. Animals with higher antibody response to an antigenic challenge often had higher serum gammaglobulin levels (Biozzi *et al.*,

1975; Halliday and Williams, 1980). Many diseases including neoplasms, unthriftiness and neonatal mortality in several species of livestock have been associated with hypogammaglobulinaemia. Humoral immune response to complex antigens like sheep red blood cells (SRBC) was complex, of polygenic inheritance and associated with several bacterial and viral diseases. Similarly cell-mediated immune response to mitogens like phytohaemagglutinin, or lymphocyte transformation test after inoculation of pigs with BCG vaccine were also considerably influenced by genetic group, sex and sire. The association between these cell-mediated immune responses and diseases like Marek's diseases in birds, brucellossis and protozoal infections in livestock were often significant. An understanding of the association of disease resistance, immune responses and production traits is very important in future breeding. Efforts are to be employed to improve disease resistance and production simultaneously. Resistance to diseases and specifically infectious diseases operates at several levels. Many researchers have examined the approaches to selection for disease resistance. Direct selection for disease resistance by challenge would be costly, hazardous, requires thorough standardization of challenge to a particular disease and the maintenance of isolation facilities for this type of selection. Indirect approaches to selection for genetic resistance to diseases have been proposed as the appropriate method and immune responsiveness has been suggested as a clear indicator of disease resistance.

Genetics of immune responses and the feasibility of utilizing immune response traits as markers in indirect selection for disease resistance have extensively been investigated in important species of livestock and poultry.

Though swines are endowed with tremendous potential as one of the major source of meat, swine production in humid tropics is hampered by high pre-weaning mortality and high incidence of diseases contributing to lowered reproductive and growth performance. Leitner *et al.* (1992) have pointed out that defective immune status predisposes animals to high morbidity and mortality. Any type of stress especially thermal stress is reported to have an immuno-suppressant effect. Adverse effect of tropical stresses of immune responsiveness might contribute to a lowered performance of temperate breeds of swine. It is interesting to note that genetic studies on immune responses and the feasibility of utilizing immune response traits for selection to diseases resistance and better performance of pigs are scanty, though these studies offer valuable possibilities. This background information necessitated a detailed comparative study on the genetics of immune responses in Large White Yorkshire and Desi pigs under the humid tropics and also an assessment of the relationship between immune responses, diseases, viability, growth and reproduction in this species. Immune responses to be assessed are humoral immune responses to sheep red blood cells (SRBC) and cell-mediated-immune response (CMI) to phytohaemagglutinin-M and Lymphocyte transformation response to BCG inoculation. These studies were apparently harmless to the health of breeding animals and hence would be of value as marker traits in selection.

The present investigation was undertaken in two breeds namely Desi and Large White Yorkshire maintained at the Centre for Pig Production & Research, College of Veterinary and Animal Sciences with the following objectives:

1. To assess the magnitude of humoral and cell-mediated-immune responses to specific antigens in pigs.

2. To compare the immune responsiveness in Desi and Large White Yorkshire pigs and to estimate the sire effect and heritability estimate of immune response traits.

3. To assess the relationship between immune response traits and economic traits.

REVIEW OF LITERATURE

2. REVIEW OF LITERATURE

The proven exotic breeds of pigs like Large White Yorkshire by virtue of their tremendous meat production potential offer unique possibilities for meat production in the tropics. But this breed fails to perform optimally under tropical climatic condition and is often subjected to infection. Lowered immune responsiveness under tropical stress appears to be a predisposing factor for this.

The immune system is a powerful tool in mammalian and avian homeostasis. It has a function in the adaptation of individual animal and population to the environment and is a major component in specific disease resistance. General resistance against diseases is the ability to resist any alteration of the state of the body by external causes (micro-organisms or stress) which interrupt or disturb proper performances. The main characteristic of immune system is the ability to detect and resist antigenic invasions by maintaining antigenic integrity against viruses, bacteria, parasites or transformed malignant cells.

There are two major categories of immune responses, those involving the antibody, which are humoral immune responses, and those that are independent of antibody called cell mediated immune responses. The pioneering work of Glick *et al.* (1956) and Claman *et al.* (1966) led to an understanding of the cellular basis of dichotomy of immune system. The production of antibody in humoral immune responses depends on the interaction of T-cells, B-cells and macrophages (Bach

et al., 1974; Unanue, 1984). Cellular immunity is a function of many types of leucocytes including T-cells (Nabholz and Mc Donald, 1983), Macrophages (Adams and Hamilton, 1984), NK cells (Herberman *et al.*, 1986) and LAK cells (Andriole *et al.*, 1985). In addition to cells of immune response, there are many soluble mediators of immune response. Primary molecules are antibody molecules, synthesized by activated B cells. Complement system (Reid and Porter, 1981) and other Lymphokines and monokines are soluble mediators of immune response. Hormones, prostaglandins and leukotrins also modify the immune responses. Cheng and Lamont (1988) classified that immune responses consist of three major facets, as phagocytosis, cell mediated immunity and humoral immunity which are performed respectively by macrophages, T-lymphocytes and B-lymphocytes. Both humoral and cell mediated immunities are under independent genetic control and there exists an inverse relationship between genetic regulations of antibody responsiveness and macrophage activity. The role of immune system in disease resistance is well established and the role of both non-specific and immunologic specific defense systems contributed to it substantially. Non-specific or innate immune mechanism operate through phagocytic leucocytes (neutrophils and macrophages), soluble mediators like lactoferrins, lactoperoxidases, thiocyanate system, hydrogen peroxidase system, lysozymes, cationic proteins and complement proteins. The specific or adaptive immune system consists of leukocytes and antibodies (Brian and Harp, 1989). Three sets of genes are reported to modulate the response of vertebrate hosts to infections, namely those controlling innate immunity, those determining the specificity of acquired immune responses

and those which affect the quality of acquired immunity (Doenhoff and Davis, 1991).

The overall complex and dynamic interactions between the hosts immune response and its pathogens are controlled by many genes. Wakelin (1989) had listed 16 distinct parameters of immune response to infection with *Trichinella spiralis* that are known to vary between strains of mice. The strains selected for particular response, either in respect of resistance to one pathogen species (Windon and Dineen, 1984) or for a more general enhanced immune response for high antibody production (Biozzi *et al.*, 1975) are not resistant against all pathogens. Oosterlee (1981) has summarised that selecting for high resistance against diseases by using immune response characteristics cannot be performed on a single item. He has concluded that a selection index in which macrophage activity and humoral and cell mediated immunity are included, might in future lead to an increase in disease resistance.

Immune response experiments in swine, its genetic association with disease and production have extensively been conducted by Buschmann *et al.* (1985), Edorf Lilja *et al.* (1985) and Meeker *et al.* (1987). Studies on the immune response in cattle and sheep by Muggli (1987), have also shown similar trend as in poultry. These immune response experiments have demonstrated considerable associations between immune responses, disease and several production traits.

The primary role of the immune system is to defend the host against foreign substances, commonly referred to as antigens. The immune system is classically divided into innate and acquired immunity. The defence against infectious organisms is maintained by both the innate and the acquired immunity and its total effectiveness is predetermined by genetic factors and further influenced by environmental factors.

Lymphoid organs

Although the anatomy and physiology of the porcine lymphoid organs are largely similar to that of other species, several aspects of the porcine lymphoid system are unique both regarding anatomy and function (Binns, 1982; Binns and Pabst, 1994). The most striking anatomic difference is the inverted structure of the porcine lymph nodes, with the medulla external, to the cortex. The physiology of the B and T cell compartments of the lymph nodes is broadly conventional but the cellular traffic within the porcine lymph nodes differs from that of other species. The T and B cells enter the node via paracortical postcappillary venula, but instead of leaving through the medulla into efferent lymphatic vessels, they emigrate directly back into blood through high endothelial venule (HEV).

Another tissue with special anatomical and functional features is the porcine gut associated lymphoid tissue (Binns and Pabst, 1994; Stokes *et al.*, 1994). In addition to the conventional jejunal Peyer's patches (PP), pigs also have a continuous ileal PP 1 to 3.5 m long, dependent on age. This ileal PP lack T cells, interfollicular areas and lymphocyte traffic in the young pig and involutes at an early age.

The porcine CD4-CD8+ expressing cells are a heterogenous cell population both concerning cell surface antigen expression and function (Sallmuller and Bryant, 1994). This cell population contains cells with NK (natural killer) activity as well as progenitors of MHC-restricted cytotoxic T lymphocytes.

Factors influencing immune functions

In most cases, susceptibility to infections is of polygenic origin. However, there are examples of single genetic loci that provide protection against certain infectious agents, e.g. the M x 1 gene and resistance to influenza virus in mice (Muller and Brem, 1991). In some cases it has been possible to associate resistance or susceptibility to infectious diseases of domestic animals with certain MHC haplotypes (Muller and Brem, 1991; Vanderzijpp and Egberts, 1989; Lunney and Grimm, 1994). For instance, resistance to Marek's disease in chicken is associated with the B^{21} allele (Bacon and Witter, 1992). In man, an association between resistance to malaria and certain HLA haplotypes has been reported (Hill *et al.*, 1991). In the pig, associations between MHC haplotypes and piglet mortality (Renard *et al.*, 1988), antibody response to vaccination (Rothschild *et al.*, 1984b; Vaiman, 1989), susceptibility to *Trichinella spiralis* infection (Lunney and Murrell, 1988) and malignant melanoma (Tissot *et al.*, 1987) have been observed. The knowledge about the role of the MHC molecules in the resistance/susceptibility to a certain disease is however still scarce (Kostyu, 1991).

In an experimental model, mice were identified as being "high" or "low" responders to a certain antigen. Selection for high antibody production in these

mice resulted in "high responders" that also had increased antibody responses to other antigens than that selected for (Biozzi *et al.*, 1984). However, "high" responding mice could also be "low" responders to other antigens. Further, the "high" responder mice had a poor lysosomal activity in their macrophages which resulted in a decreased resistance to intracellular microorganisms. Nevertheless, such phenotypical identification of responding individuals has been applied in breeding programmes to increase disease resistance in domestic animals. In sheep for instance, resistance to Trichostrongyle nematodes has been included in breeding programmes rather successfully (Gray, 1991; Windon, 1995).

Another approach to improve disease resistance is the use of immune response traits as selection markers (Mallard *et al.*, 1992). This strategy is based on the assumption of genetic variation in the traits used and the genetic correlation between the markers and disease resistance. In the pig, such variation has been found for several immune parameters, e.g. antibody responses to certain antigens (Rothschild *et al.*, 1984a; Mallard *et al.*, 1992; Joling *et al.*, 1993) proliferative responses of mononuclear cells (Mallard *et al.*, 1992; Joling *et al.*, 1993; Joling *et al.*, 1991) delayed-type hypersensitivity reactions (Joling *et al.*, 1993) and total and differential cell counts. Heritabilities have been estimated for most of these traits. Against this background a breeding programme for pigs based on combined breeding values for antibody responses, lymphocyte proliferation and monocyte phagocytosis and intracellular killing, has been established (Mallard *et al.*, 1992). Current results from this project (Appleyard *et al.*, 1992; Appleyard and Wilkie, 1992; Groves *et al.*, 1993; Ichim *et al.*, 1995; Moller *et al.*, 1995; Raymond *et*

al., 1995) indicate that selection for improved general immune capacity of pigs is possible.

A novel opportunity for dissection of the genetic influence on immune capacity is offered by genome mapping. In 1989 the PiGMaP consortium was established including 13 laboratories (Archibald *et al.*, 1991). From this international project detailed linkage maps have been established, now comprising over 200 markers, distributed on all chromosomes (Ellegren *et al.*, 1994; Archibald *et al.*, 1995; Marklund *et al.*, 1996). Quantitative trait loci (QTL) for production traits such as growth and fat deposition have been identified within this reference pedigree (Anderson, 1994). Identification of QTLs for immune capacity traits should thereby be feasible in the pig.

Environmental factors influencing the immune response

The immune and neuroendocrine systems are connected through an intimate network (Blalock, 1994). The classical example of this connection is the immunosuppressive properties of glucocorticoids (Quinn, 1990). Accumulating data testify to such an immuno neuroendocrine link also in the pig (Kelley, 1994). For instance, elevations in plasma cortisol levels lead to involution of neutrophilia (McGlone *et al.*, 1991; Wallgren *et al.*, 1994). In experimental stress models, administration of corticotropin releasing hormone (Johnson *et al.*, 1994) or ACTH to pigs (McGlone *et al.*, 1991; Wallgren *et al.*, 1994; Klemcke *et al.*, 1990) alter several immune function as assessed *in vitro*. Also other neurohormones exert

regulatory effects on the immune system of rodents (Blalock, 1994). Thus, factors that trigger the production of neurohormones are likely to influence the immune capacity of pigs.

In modern industrialized rearing systems, pigs are often exposed to several possible "stressors" presented by housing conditions and husbandry practices, such as weaning, regrouping and allocation. Accordingly, the impact of management routines on different porcine immune function have been the subject for many studies. For instance, weaning (Blecha *et al.*, 1983; Blecha *et al.*, 1985; Bailey *et al.*, 1992; Hessing *et al.*, 1995), transport (Dalin *et al.*, 1993), mixing and allocation (Artursson *et al.*, 1989; Hessing *et al.*, 1995; Wanglen *et al.*, 1998) has been found to alter several porcine immune parameters. In addition, other potential "stressors" such as restraint (Tokarsk *et al.*, 1992), social status (Mc Glone *et al.*, 1993; Morrow-Tesch *et al.*, 1994; Hessing *et al.*, 1994), heat (Morrow-Tesch *et al.*, 1994; Hessing *et al.*, 1994; Machado-Neto *et al.*, 1987), cold (Blecha and Kelley, 1981) and draught (Scheepens *et al.*, 1994) affects immune functions of pigs. Both enhancing and suppressive effects on immune functions have been observed, probably due to type of "stressor", duration of the stressful event and coping strategy of the animal (Mc Glone, 1990). However, the underlying mechanisms of the observed alteration in immune functions and their consequences for the defence against microorganisms, are yet far from elucidated in the pig.

Another environmental factor with a likely impact on the immune system is the pathogen load. As an example, the production of inflammatory cytokines during an infection may activate the neuroendocrine system through cytokine receptors on cells in the central nervous system and ultimately result in a glucocorticoid response (Blalock, 1994). Infectious diseases are a recognized problem in intensive rearing systems (Wanglen *et al.*, 1998; Falk and Lium, 1991), and may therefore add to the stress (Beisel, 1988; Elsasser *et al.*, 1995). However, the possible impact of infections, especially subclinical ones, on the immune functions of pigs is often neglected.

2.1 Humoral immune responses

The cellular basis of humoral immune response consists essentially of a phenomenon of multiplication of differentiation of T and B lymphocytes stimulated by the antigen processed by macrophages. The selection of specific clone of lymphocyte is made by stereospecific combination of antigenic determinants with pre-existing receptors on lymphocyte surface. In B lymphocytes these receptors are immunoglobulins. Intracellular and intercellular reactions are therefore integrated in the phenomenon of immune response. The efficiency and co-ordination of such a complex phenomenon are controlled and regulated at different levels by genetic controls. Biozzi *et al.* (1975) have attributed the general immune responsiveness as a polygenic trait determined by a group of about ten independent loci.

The immune response to a multideterminant immunogen has not been reported to be the mere effect of additive effect of Ir genes for each antigenic determinants. The rapid accumulation of experimental studies demonstrating the analogies between genetic control of specific and general antibody synthesis preludes the formulation in near future of a unified theory of genetic regulation of immune response (Biozzi *et al.*, 1985).

The initial production of antibodies after the first immunisation was markedly different from that of antibody production after the second immunisation. The former is called primary immune response and the latter is known as secondary or an anamnestic response. The primary response was sluggish, short-lived and comprised mainly of 19S IgM immunoglobulins. Secondary response was swift, prolonged and composed mainly of 7S immunoglobulins (Park and Good, 1974). The correlation between primary and secondary immune responses to complex antigen was highly significant (Burton *et al.*, 1989a).

Immune response to an antigen is influenced by several factors. Antigen presentation, site of entry, response of antigen binding cell and regulation of response, after the immune response in birds (Tizard, 1979), environmental stress and immunosuppressive in laboratory animals (Keller *et al.*, 1983). Protein malnutrition impairs humoral immune response (Mathew *et al.*, 1972). The decline in antibody production with advanced age has been studied in mice (Nordin and Makindoan, 1974; Folch *et al.*, 1982), rats (Kunz *et al.* 1983), man (Nagel *et al.*, 1985), pigs (Hyldgaard – Hensen, 1979), poultry (Mc Corkle and Glick, 1980; Munns and Lamont, 1991) and sheep (Watson and Gill, 1991).

Biozzi *et al.* (1975) concluded that the mammalian immune responses to complex immunogens such as sheep red blood cells (SRBC) are controlled by polygenic inheritance as evidenced by studies on mice using SRBC as test antigen. Hyldgaard – Jensen (1979) observed that the ability of swine to produce antibodies to standardised doses of human and bovine albumin were under genetic control. The studies of Almid *et al.* (1980) on the quantitative antibody response to diphtheria toxoid in goats and Buschman's (1980) study on antibody response in pigs demonstrated that the antibody response could significantly be modified by selective breeding over two to three successive generations. Aihara *et al.* (1983) observed a dominant type of inheritance in inbred mice strains selected for high antibody response to human thyroglobulin when measured by ELISA.

The feasibility of selective breeding depends in part upon the heritability of the trait or traits used to measure disease resistance as well as the amount of variation among animals (Stear *et al.*, 2001)

The genetics of a bird or flock has a profound impact on its ability to resist disease, because genetics defines the maximum achievable performance level. Careful attention should be paid to genetics as an important component of a comprehensive disease management program including high-level biosecurity, sanitation and appropriate vaccination programs (Lamont, 1998).

The past two decades have illustrated that single genes with a large impact on food animal health do exist and can be used to improve the health of domestic populations. It is also clear that genetic control over the immune system is not

limited to a few genes but is more likely influenced by many genes, each with small effects (Kelm *et al.*, 2001).

The existence and consequences of negative genetic correlations between production and fitness traits are dealt with, as is the procedure for multi-trait selection (Lindhe and Phillipsson, 1998).

There is a threshold for resistance below which animals will stop producing, and that there is also a threshold for resistance above which animals produce to production potential. In between both thresholds animals will show a decrease in production, the size of decrease depending on the severity of infection and the level of resistance (Vander waaij *et al.*, 2000). These can be classified under sustainability, feasibility and desirability (Stear *et al.*, 2001).

Future research directions will expand knowledge of the impact of genetics on disease resistance by identifying non-MHC genetic control of resistance and by further elucidating mechanisms regulating expression of genes related to immune response (Lamont, 1998).

A great deal of evidence points to substantial genetic control over at least some of the immune responses, although genetic parameters for clinical disease have been less favourable. Genetic control over the immune system is not limited to a few genes but is more likely influenced by many genes, each with small effects. The scarcity of information dealing with phenotypic and genetic relationships between measure of disease resistance and aspects of immune response complicates the situation even further (Kelm *et al.*, 2001).

Studies at Rockhampton have demonstrated that completely resistant lines can be developed by genetic means within a commercially acceptable timeframe from even the most parasite-susceptible breeds (Frisch *et al.*, 2000).

Antibody response to vaccines was highest in High Immuno Responder (HIR) and non-responders were restricted to Low Immuno Responder (LIR) pigs. The HIR pigs had the best rate of weight gain. After infection with *Mycoplasma hyorhinis*, HIR developed more severe arthritis and less polyserositis. Differences were associated with variation in cytokine message in joint-related cells. Genetic selection for antibody and CMI may provide health and productivity advantages and complement traditional health-maintenance methods (Wilkie and Mallard, 1999).

Santanna *et al.* (1985) reported that lines of mice selected for high responsiveness of TNP-LPS had higher response TI-1 of mice confirming that genes accumulated through selective breeding could modify response to unrelated antigens. According to Ferreira *et al.* (1986) antibody response of mice to rabbit gammaglobulin and bovine serum albumin were controlled by additive effect of several independent loci. Gyles *et al.* (1986) concluded that there existed significant difference between breeding groups of chicken for antibody responses to different classes of antigens as Newcastle virus vaccine, infectious bronchitis vaccine and SRBC. Reynolds and Griffin (1986) observed that total antibody production was significantly impaired in ewes during gestation.

According to Burton *et al.* (1989a) humoral responses of calves to human red blood cells (HRBC) could be used as a marker trait of disease resistance in

diary calves. Gross and Seigel (1990) concluded that antibody responses of individual chicken to SRBC were influenced by their heterophile – lymphocyte ratios.

2.1.1 Humoral immune response status

Hyldgard – Jensen (1979) observed that peak antibody titers to bovine and human albumin were obtained two to three weeks after primary immunisation in pigs and the primary antibody responses were influenced by the adjuvant and dose of antigen. Seigel and Gross (1980) reported that additive genetic variation was noticed for high and low antibody titers at five days after SRBC inoculation. Vander Zijpp and Leenstra (1983) found that mean total antibody titer to SRBC was highest on seventh day after primary immunisation. According to Vander Zijpp *et al.* (1983) on day three and seven of post injection of chicken following primary immunisation, random size effect was not significantly different from day zero. Selection for primary antibody response could be based on total antibody titer at day five of post injection. Ubosi *et al.* (1985) reported that following fourth day of primary injection of chicken with SRBC, differences in response could be noticed. Peak value was reached in six days in all populations following primary immunisation with SRBC.

According to Burton *et al.* (1989a) peak primary antibody response was observed in calves by day seven to fourteen following Human Red Blood Cell (HRBC) inoculation. Miller *et al.* (1991) reported that peak antibody titer to SRBC occurred in chicken on day six or seven following primary immunisation.

Mounton *et al.* (1988) reported that mean antibody titer to SRBC was 6.6 ± 1.8 log_2 in mice resistant to Salmonella typhimurium while it was 8.1 ± 2.8 log_2 in susceptible ones. Burton *et al.* (1989a) found that peak primary titer to HRBC in calves was 4.465 log_2.

Pinard *et al.* (1992) reported a selection experiment which generated high and low response lines to SRBC in chicken. The mean post primary antibody titer was 4.73 log_2 in the base population. After nine generations of selection the antibody titer was 10.62 log_2 in high response group and 1.94 log_2 in low responder lines.

2.1.2 Effect of breed and sex

Vander Zijpp (1978) reported significant breed and strain differences in humoral immune response of poultry of SRBC. In a comparative study involving *Bos taurus* and *Bos indicus* cattle Banyard and Morris (1980) observed that *Bos indicus* cattle elicited a higher antibody response to Keyhole Limpet haemocyanin (KLH). Differences among genetic groups resulted in significant variation for total antibody titers to SRBC in poultry. Lamont and Smith (1984) reported significant breed difference in antibody producing capacity of poultry against SRBC in *Brucella abortus*.

Rothschild *et al.* (1984a) found significant breed differences in antibody responsiveness of pigs to inactivated *B. bronchiseptica*. The response was also influenced by dam. Okabayashi *et al.* (1987) pointed out that line differences in

antibody responses to SRBC were polygenic in poultry. There were considerable variation in antibody response between and within populations of poultry (Peleg *et al.*, 1985).

Ubosi *et al.* (1985) found significant population differences in response of chicken to SRBC. The peak response titer to SRBC occurred at the same time in all the lines and populations. Buschman (1986) found significant differences among different breeds of swine to humoral immune responses to different antigens. Muggli *et al.* (1987) reported no breed difference among Angus, Hereford and Red Poll calves to *Infectious Bovine Rhinotracheitis* virus (IBR), 60 days post vaccination.

White Leghorn chicken showed significant sub-line difference in their humoral antibody response to GAT (Glutamic acid^{60} – Alanine^{30} –Tyrosine^{30}) (Cheng and Lamont, 1988). Petrovsky *et al.* (1988) found significant breed differences among White Leghorn, Rhode Island Red and Rhode Island White breeds of poultry in their antibody response to *Brucella abortus* and SRBC. Buschman and Meyer (1990) reported that immune response of swine to tetanus toxoid was significantly influenced by breed.

Layer chicken lines differed in their humoral immune response to SRBCs (Genzel and Wigend, 1990). Benda *et al.* (1990) also observed significant breed difference among poultry for their humoral immune response to SRBC. Dunnington *et al.* (1992) evolved high and low antibody producing lines to SRBC in White Leghorns and White Plymouth Rock. Antibody response to Newcastle disease virus was consistently higher in high responding lines.

Nguyen (1983) investigated the effect of sex on the immune response to chicken red blood cells in sheep. He found that mean haemagglutinin titers of young female were higher than that of young males, though the differences between groups were not significant. No sex related differences were found in antibody response to sheep red blood cells, *Brucella abortus* or rabbit erythrocytes in chicken (McCorkle and Glick, 1980; Vander Zijpp *et al.*, 1986). Leitner *et al.* (1989) reported that response to SRBC in chicken was significantly influenced by sex with a female superiority contributing to the increased survival of female birds.

2.1.3 Sire effect and heritability

Compared with an estimate of 0.43 for primary immune response by Claringbold *et al.* (1957). Biozzi *et al.* (1970) estimated the heritability of antibody response to SRBC of 0.36 in mice.

In a study involving the humoral response of sheep to chicken RBC Nguyen (1983) found that the antibody titers of sires varied from four $\log_2$ and eight $\log_2$. The regression coefficient of sire was 0.41 indicating that effect of sire was significant on the antibody response. The heritability estimate approached 0.82. Lie *et al.* (1983) reported that in cattle sire families and not sires differed significantly in their antibody response to human serum albumin and synthetic peptides. Vander Zijpp (1983) reported that heritabilities of immune response to SRBC were 0.26 and 0.14 in White Leghorn and White Plymouth Rock breed at day seven post-immunisaiton. In beef cattle Muggli *et al.*, (1987) found a heritability of 0.21 ± 1.2 for antibody response to Infectious Bovine Rhinotrecheitis virus.

Burton *et al.* (1989a) estimated the heritability by paternal half sib correlation analysis and found the peak titers were more heritable for antibody response in calves to human red blood cell and ovalbumin. They reported a heritability estimate of 0.4 and 0.34 respectively for HRBC and ovalbumin. Leitner *et al.* (1992) found that heritability of immune response to *E.coli* vaccination in low response group was 0.35 and in high response group was 0.21.

Pinard *et al.* (1992) estimated the heritability of fifth day antibody titer to SRBC challenge in chicken as 0.31. Kean *et al.* (1994a) found that heritabilty of antibody response to *Mycoplasma gallisepticum* was 0.06 and 0.01 for high and low responder liens respectively. The heritability for antibody response to *Pasteurrela multocida* was 0.44.

2.1.4 Effect on growth and litter traits

It is generally believed that fast growing poultry strains are genetically less resistant to disease. Han and Smyth (1972) observed that selection for increased growth rate in broilers resulted in an increased susceptibility to Marek's disease. Seigel and Gross (1980) demonstrated that a line selected for low antibody response to SRBC had significantly higher four weeks body weight than either unselected control or high antibody response line. Vander Zijpp (1983) reported that the correlation between live weight and heamagglutinin antibody titers to SRBC indicated a negative genetic relationship. In beef cattle, Muggli *et al.* (1987) reported that there were no significant correlations between immune response traits and growth traits.

In pigs, Huang (1977) found that there was no association between immune response and early growth. Meeker *et al.* (1987) showed that there exists a negative association between humoral immune response traits and growth rate in pigs.

Leitner *et al.* (1992) observed no significant association between humoral immune response to *E.coli* at 10 days of age and growth traits. Kean *et al.* (1994b) reported that there were no significant association between immune response traits, juvenile and adult body weights, age at first egg, 32 weeks egg weight and rate of egg production in chicken.

2.1.5 Effect of diseases and mortality

In the analysis of humoral immune response to SRBC in mice, Biozzi *et al.* (1975) found that high responder mice were more susceptible to *Salmonella typhimurium* infections. Similar results were found in *Yersinia pestis* infection. There was a quicker destruction of T_4 bacteriophages and a slower rate of growth of *Listeria monocytogenes* in low responder groups. Gross *et al.* (1980) reported that chicken selected for ability to produce high antibody titres to SRBC showed stronger antibody response to Newcastle disease virus and were more resistant to *Mycoplasma gallisepticum, Eimeria necatrix, Splenomegalia* virus and feather mites. They were less resistant to *E.coli* and *Staphylococcus aureus*. The line of chicken selected for non persistence of antibody response to SRBC were more susceptible to all infectious agents.

Covelli *et al.* (1984) showed that mice selected for high antibody response to SRBC and Salmonella flagellar antigens had lower incidence of lymphomas. Dunnington *et al.* (1986) selected chicken for high and low antibody titere to SRBC. The low responder lines were more susceptible to Marek's disease. Mounton *et al.* (1988) described that innate resistance to intracellular pathogens were higher in mice selected for low immune response in terms of antibody production. This was due to faster antigen catabolism in macrophages of these lines. The mice selected for higher immune response, in lines of antibody production, had stronger innate or acquired resistance to all infections that could be cleared by means of antibody production.

Burton *et al.* (1989a) showed that diarrhoea prevalence was negatively correlated with high primary antibody response against HRBC. Chicken selected for four generations of early high antibody response to *Escherichia coli* showed greater resistance to challenge with *E. coli* (Pitcovski *et al.*, 1984). Larsgaard (1990) noticed improved health status in goats selected for high immune response. Lillehoj (1991) observed that inbred strains of chicken having higher antibody response and T-cell response had reduced susceptibility to *Eimeria tenella* infection. Pinard *et al.* (1992) could demonstrate that mortality rates on challenge with virulent Marek's disease virus was high in low immune response group to SRBC.

As early as 1983, Vander Zijpp (1983) showed that the lines of chicken with high antibody response to SRBC had a lower mortality rate. Covelli *et al.* (1989a) found the mice selected for high antibody response to SRBC had a higher

life span. Dunnington *et al.* (1986) observed higher cumulative mortality in mice selected for low antibody titer.

Lines of mice selected for high antibody responses to SRBC as measured by ELISA had a positive correlation with life span (Covelli *et al.*, 1984). Chicken selected for four generations for early high antibody response to *E. coli* and Newcastle disease virus vaccinations had a lower mortality rate (Pitcovsky *et al.*, 1989). According to Leitner *et al.* (1989) broiler chicken having higher antibody response to *E. coli* vaccination had lower mortality rates when challenged with pathogenic *E. coli*, Leitner *et al.* (1992) reported that birds with high antibody titre to *E.coli* vaccinations ten day post-vaccination had the lowest morbidity and mortality rate when challenged with pathogenic *E.coli*.

2.2 Cell mediated immune responses

A large number of genes of genetic factors influence the cell-mediated immune responses. They include (1) genes that impart on acquired immunity but not antigen specific such as cytokine genes and (2) genes that influenced primarily innate or non-specific immunity. Genes controlling the mitogenic responses come under first category. Knudtson *et al.* (1990) found that IL-2 production and mitogenic response to concanavalin A (Con A) were not always associated, suggesting that more than one gene or more than one mechanism were likely to influence the mitogenic response of T-lymphocytes. Numerous factors differentiate genetic difference in acquired or innate cell mediated immune responses. The responses for these differences are likely to relate to a variety of genes including those determining cytokines, cytokine receptors or the adhesion

proteins. The cellular T-lymphocyte dependant immune response has also been reported to be polygenically regulated. Stiffel *et al.* (1977) selected mice on the basis of lymphocyte stimulation by using phytohaemagglutinin (PHA) as mitogen. After six generations 3.8 times difference could be observed between high and low responder groups to PHA.

Several *in vivo* tests have been standardised to assess the cell mediated immune response and T-cell funcion by employing phytohaemagglutinin (PHA) and chemicals like2,4-dinitrochlorobenzene in animals (Hodgin *et al.*, 1978 and Rajan *et al.*, 1981). Wilkie *et al.* (1991) examined that cutaneous response content allergen dinitrochlorobenzene (DNCB) and mitogenes, phytohaemagglutinin (PHA) and concanavalin A (Con A) in a topic and normal dogs. The immune response to contact allergens such as DNCB results in a type IV or delayed type hypersensitivity (DTH) reaction following percutaneous absorption of the hapten in sensitised animals.

In vivo T-lymphocyte response has also been evaluated by intradermal injection of mitogens. This is believed to provoke a delayed type hypersensitivity reaction without the need for prior sensitization by polyclonal stimulation of lymphocytes (Wilkie *et al.*, 1991).

Tiwary and Goel (1985) confirmed the efficacy of DNCB skin test in assessing the cell mediated immune response of chicken, comparing with lymphocyte transformation tests and graft versus host reaction (GVH).

Repeatability of T-cell dependant cellular parameters in pigs were reported to be high (Buschman, 1986). According to Burton *et al.* (1989b) DTH reaction in calves to access the cell mediated immune response could be used as a marker trait in selection for disease resistance.

2.2.1 Cell mediated immune response status

Trindle *et al.* (1980) measured the CMI response in mule deer fawns (*Odocoileus hemionus*) at the age of one week using DNCB skin sensitisation test. Newly hatched turkey poults demonstrated DTH response to PHA and Freund's adjuvant containing Mycobactarium. Two week-old poults had a higher DTH than eight week-old poults (McCorkle *et al.*, 1984). The DTH reactions of fowls to human gammaglobulin were more intense at six to twelve weeks of age than at three weeks of age (Watable and Glick, 1983). Edelman *et al.* (1986) reported that the differences between T-cell reactivity in immunocompetent normal chicken and transplantable fibrosarcoma bearing chicken could be readily detected *in vivo* at an age of three to four weeks using PHA wattle test.

Warner (1987) studied the CMI response by DTH reaction to DNCB in dogs. He observed that younger dogs had a higher CMI response than aged dogs. Paulik and Urzula (1989) found a higher CMI response to dintrofluorobenzene (DNFB) in calves aged 11 months than in younger calves. Corrier and Deloach (1990a) reported that in chicken CMI response could be elicited in young birds of 10 to 14 days of age by sensitizing with *Mycobacterium tuberculosis* and challenging with tuberculin intradermally. By using PHA, CMI could be assessed as early as three to fourteen days of age (Corrier and Deloach, 1990b).

Rajan *et al.* (1982) found that increases in skin thickness following DNCB challenge in pigs were 6.07 ± 0.38, and 4.71 ± 0.56 mm at 24 and 48 hours post challenge respectively.

The skin thickness following DNCB challenge averaged 0.607 ± 0.036 mm at 24 hours in challenge (Tiwary and Goel, 1985). The cutaneous response to DNCB and PHA-M in dogs as indicated by increased skin thickness was 0.536 ± 0.262 and 0.777 ± 0.362 mm respectively (Wilkie *et al.*, 1991).

2.2.2 Effect of breed and sex

In broiler chicken CMI response to diphtheria toxoid varied between different genetic stocks (Klesius *et al.*, 1977). Significant breed difference were seen in CMI response of chicken to PHA test (Vander Zijpp, 1983 and Lamont and Smith, 1984). Breed differences were also reported for CMI responses in pigs (Buschman, 1986). Cheng and Lamont (1988) found significant sub line differences in birds for CMI response as assessed by PHA test. According to Benda *et al.* (1990), significant breed differences were noticed in cell mediated immune response of fowl when assessed by wattle injection with SRBC.

Cheng and Lamont (1988) reported that there existed significant differences between male and female chickens in the T-cell response to PHA-M, with females having a higher CMI over males. Leitner *et al.* (1989) reported that females showed an earlier and greater T-cell response to a purified protein derivative *Mycobacterium avium.*

2.2.3 Sire effect and heritability

Stiffel *et al.* (1977) found that effect of sire on the T-lymphocyte dependent immune response as significant. They selected mice on the basis of lymphocyte stimulation by phytohaemagglutinin as mitogen. After six generations a 3.8 times greater difference between high and low responder line could be observed. Heritability estimate of T-cell response to PHA was 0.28 ± 0.08. Lie *et al.* (1983) reported significant differences between sire families in cattle in their CMI responses. Cheng and Lamont (1988) found that haplotype differences was significant on phagocytic index and T-cell response to PHA and sire family differences were significant on T-cell response. Cheng *et al.* (1991) reported that heritability of T-cell response to PHA measured by wing web assay was only 0.06 to 0.07 in base population, while the combined data for first generation for this trait gave heritability estimate of 0.12 to 0.14. Kean *et al.* (1994b) reported that heritability of cell-mediated immune responses to PHA was 0.15 in chicken.

2.2.4 Effect on growth and litter traits

Reports on the association of cell-mediated immune responses and production traits and growth rate are scanty. The role of cell mediated immunity especially T-cell immunity have been found to be important in several diseases like coccidiosis which adversely affect growth rate in animals and birds (Rose *et al.*, 1990).

2.2.5 Effect on diseases and mortality

Brown *et al.* (1967) studied the efficacy of DNCB skin tests in untreated Hodgkin's disease patients and found the DNCB sensitisation could be used as one of the most reliable skin tests in evaluation CMI status of patients by measuring the double skin fold thickness. Chicken that were to develop clinical Marek's disease had significantly lower CMI response compared to resistant birds. It was also observed that the correlations between high CMI response and several other diseases were significant (Chauhan *et al.,* 1984). Palival *et al.* (1984) observed that CMI responses to DNCB were markedly lower to Johne's disease affected cattle. Tiwary and Goel (1985) produced CMI deficient chicken by thymectomy and inoculation of antithymocyte serum. These birds had a reduced CMI response in terms of DTH response to DNCB challenge. Edelman *et al.* (1986) found that the CMI response of chicken bearing transplantable fibrosarcoma were totally inadequate without any response in the form of inadequate wattle swelling when assessed by PHA test. Burton *et al.* (1989b) could not find any significant effect of CMI as assessed by cutaneous response to DNCB on the incidence of naturally occurring diarrhoea and pneumonia in calves. Rose *et al.* (1990) reported that lymphocyte responses contributed considerably to coccidiosis in chicken. Deshmukh *et al.* (1990) reported significant increase in CMI responses of kids naturally infected with goat pox, when assessed by DNCB test. Dogs with atopic dermatitis had lower CMI responses to DNCB contact sensitivity and PHA skin test (Wilkie *et al.,* 1991).

2.3 Associations between immune response traits

Biozzi *et al.* (1975) showed that there was no association between immune response to SRBC and T-cell response to PHA. However, total Ig level was found to be associated with immune response to SRBC. Muggli *et al.* (1987) showed that IgG_1 levels in calves were correlated with pre-and post-vaccination titre to IBRV vaccination. Cheng and Lamont (1988) found that there existed a significant negative correlation between phagocytic index and T-cell response to PHA especially among female chicken. Mounton *et al.* (1988) reported that vaccination response of high responder lines to SRBC may be as high as 200 times compared to low responders. They also reported that innate resistance to inter cellular pathogens were higher in low antibody response lines owing to the differences in macrophage activity in low lines, antigen catabolism being faster in low response lines. Cheng *et al.* (1991) found that the genetic correlation between immune response to *Pasteurella multocida, Mycoplasma gallisepticum* and the T-cell response as measured by PHA test were negative. Similarly, the association between phagocytic activity and T-cell response was also negative. Pollock *et al.* (1991) recorded significant positive correlation between IgG_1, IgG_2, IgA and the cutaneous response to keyhole limpet haemocyanin (KLH). Parmentier *et al.* (1994) observed that the dissimilarity of immune response to BSA in high and low responder lines to SRBC in chicken suggest that selection for enhanced response to one antigen may not influence the improvement in immunity to another antigen.

MATERIALS AND METHODS

3. MATERIALS AND METHODS

One hundred and fifty piglets of both the sexes belonging to Large White Yorkshire and Desi breed within the age group of 2-3 months maintained at the Centre for Pig Production and Research, College of Veterinary and Animal Sciences, Mannuthy, Kerala Agricultural University formed the experimental animals. Seventy five piglets of either breed were sired by eight boars. They were maintained under optimum identical conditions of feeding and management. The immune response traits were assessed during 1998-2000. The herd of experimental animals were monitored for pre weaning mortality and incidence of any neonatal diseases. Only apparently healthy weaned piglets below 75 days of age were chosen for the study. The litter traits studied were litter size at birth, litter weight at birth, birth weight, weaning litter size, weaning litter weight and body weight at weaning.

Body weights of all the experimental animals were recorded at the beginning of the experiment. Ten millilitres of blood were collected from the cranial vena cava using sterile glass syringe from each animal. Immediately, three ml of blood was transferred to a tightly capped storage vial containing 150 IU heparin for assessing the pre-immunisation lymphocyte transformation. The remaining seven ml of blood was allowed to clot and left standing upright at 4^0C for 24 hours before being centrifuged at 1500 rpm for fifteen minutes. The serum samples were stored in tightly capped storage vials at -20^0C for assessing the pre-immunisation titres to sheep red blood cells (SRBC).

3.1 Humoral immune responses

3.1.1 Test antigen preparation

Sheep red blood cells (SRBC) were chosen as the test antigen, since they were complex and apparently harmless antigens. One hundred millilitres of blood was collected from a single sheep in Alsever's solution and was washed thrice in sterile phosphate buffered saline (PBS 0.01M, pH 7.2) by repetitive centrifugation (1500 rpm of 10 minutes). The red blood cells were resuspended in fresh sterile PBS to get a final concentration of 20 per cent (V/V) and stored at 4^0C in sterile glass containers until used for immunisation/ antibody titration.

3.1.2 Administration of antigen

For immunization test antigen was injected intravenously at the rate of 0.5 ml per kilogram body weight through marginal ear vein.

3.1.3 Collection of blood for serum

From blood samples collected at days zero, seven, fifteen and twenty one days post immunisation, sera were separated and stored in tightly capped storage vials at -20^0C until used.

3.1.4 Swine anti SRBC titre assay

Serum antibody titre to SRBC was titrated by microhaemolytic test as described by Hines (1985). The test was carried out in 96 welled microtitre plates

(Laxbro, Pune). To each well 0.05 ml of dilute serum was added followed by 0.025 ml of two per cent suspension of SRBC in PBS and 0.025 ml of fresh guinea pig serum was added as source of complement. The microtitre wells were covered and the plates were shaken well. Readings of the test were taken after four hours of incubation at 30^0C. The extent of haemolysis was read as follows:

0: All wells intact and settled at bottom. Supernatant was clear

1: Nearly twenty per cent of cells were lysed. Supernatant was reddish coloured.

2: Nearly 50 per cent of the cell were lysed. The intact cells formed a small button or ring at bottom. Supernatant was red.

3: Nearly 90 per cent of the cells were lysed. Supernatant was bright red which on shaking became cloudy.

4: All cells were lysed. Whole liquid was bright red and retained the brightness even after shaking.

The antibody titre was recorded as the highest dilution of serum giving reaction to the extent of two.

3.2 Cell mediated immune response

The cell mediated immune responses were assessed by cutaneous responses to the intradermal injection of phytomitogen, phytohaemaggutinin-M (PHA-M) and lymphocyte transformation response to BCG inoculation.

3.2.1 PHA skin test

In vivo T-lymphocyte response to PHA-M was assessed as described by Wilkie *et al.* (1991) with suitable modifications. The skin on the base of left ear was clipped and cleaned with 70 per cent alcohol. The skin thickness of double skin fold was measured using a Harpenden skin fold caliper. Phytohaemagglutinin – M (PHA – M) (Sigma Chemicals, St Louis, USA) was dissolved in sterile saline and diluted to contain 50 μg in 0.1 ml. To ensure uniformity of reagent, the prepared solution was kept frozen at -30^0C until half an hour before use. A 0.1 ml quantity of this solution was injected intradermally using a 25 gauge needle. As a control 0.1 ml of sterile saline was injected at the base of right ear. The skin thickness was recorded at 24,48 and 72 hours after injection. The increase in skin thickness was expressed in mm and tabulated.

3.3 Lymphocyte transformation test

3.3.1 Inoculation

The piglets were vaccinated with BCG (BCG Vaccine Laboratory Guindy, Chennai). Vials of 20 doses were diluted with 2 ml normal Saline, 0.1 ml of the vaccine was given to each piglet as intradermal injection using sterile tuberculin syringe and blood was collected on zero day and 15, 30 and 45th day post inoculation.

3.3.2 Cell collection

Two millilitres of heparinised (50 iu/ml) blood were diluted with equal volume of phosphate buffer saline (PBS) and layered directly on two milliliter Ficoll paque (Amersham Pharmacia Biotech, Asia and Pacific Ltd., Chennai) in sterile centrifuge tubes, after centrifugation at 550 xg in a swing out head for 30 minutes at room temperature of 20^0C. The mononuclear leukocytes and thrombocytes were layered in the interphase between Ficoll paque and plasma. The cell disc were collected with a sterile Pasteur pipette and resuspended in PBS for washing. The cells were washed twice to remove the anticoagulant, plasma and Ficoll paque.

3.3.3 Cell counting

Twenty blood samples were subjected to cell counting using Trypan blue stain to assess the number of mononuclear leukocytes or lymphocytes per ml of blood. It was found that the whole blood on an average contained 8.5 x 10^6 cell/ml. After Ficoll paque fractions 6 x 10^6 cell were obtained and after fractionation the number of dead cells were almost negligible.

3.3.4 Culture and culture medium

After the final washing the cells were suspended in two ml of freshly prepared Rosewell Park Memorial Institute medium 1640 (RPMI 1640) with L-glutamine (Himedia Private Ltd., Mumbai) supplemented with penicillin

(200 iu/ml) streptomycin (100 μg/ ml) and 20 per cent foetal calf serum (FCS) 0.4 ml each from each suspension was drawn to four microfuge tubes and to this 50 μl of PPD M.Bovis (BCG Vaccine Laboratory, Guindy, Chennai)was added in two tubes labeled with PPD and the final volume was adjusted to 0.5 ml by adding 50 μl of RPMI 1640. To the tube without PPD, 0.1 ml of RPMI 1640 was added to make the final volume to 0.5 ml. The 0.5 ml culture contained approximately 2.4 x 10^6 cell. The cultures were incubated at 37^0C for 48 hours.

3.3.5 Pulse labeling

A 24 hours pulse labeling with 3_H-Thymidine (Bhaba Atomic Research Centre, Mumbai specific activity 5 ci/mM) was used as described by Kristenin *et al.* (1982). After 24 hours of incubation 10 μl containing 1μc 3_H Thymidine was added to each culture and mixed thoroughly and incubated for further 24 hours.

3.3.6 Harvesting

The microfuge tubes were microfused (0.5 ml microfuge Eppendorf, Germany) at 1500 xg for 15 minutes and the supernatent was removed. The sedimented cells were resuspended in 0.5 ml of PBS and mixed thoroughly in a cyclomixer and microfuged again at 1500 xg for 15 minutes for removed of the thymidine from the cell surface. After two such washings, the cells were resuspended in 0.5 ml of PBS, thoroughly mixed in cyclomixer and the entire cells were transferred to the Teflon vials (Supplied from the Radio Tracer Laboratory, Kerala Agricultural University).

3.3.7 Counting of CPM

Radiation emitted by the samples were counted in a Wallac 1480 Wizard 3" automatic gamma counter at the Radiotracer Laboratory, College of Horticulture, Vellanikkara. The Wizard was used as a standlone CPM counter, though the instrument has other computational facilities. The instrument was pre-normalised for background radiation as well as for the isotope ^{3}H-Thymidine so as to ensure that the "gain" of the detector is optimum for the isotope and that the effect of background was removed from measured counts.

Sample tubes of 10 ml size were used for counting, with 0.5 ml of serum sample, mixed with E x 2 Scintran (E-Merck Germany) cocktail of chemicals. Ten sample tubes each were positioned on each sample rack and the sample racks were loaded to the conveyor of the counter. A counting protocol was set with 60 seconds as counting time. Results of counting were printed out in CPM units, using an external printer.

3.3.8 Presentation of results

For every animal two stimulated and two unstimulated samples of cell culture were set for incubation and the CPM was calculated as an average of these two samples for background radiation as well as for CPM for a stimulated sample for a particular day. The stimulation index was computed by dividing the CPM of stimulated sample with the CPM of unstimulated sample. These indexes were used for the analysis for partitioning the variation and measure the magnitude of response.

3.4 Assessment of body weights, litter traits and disease incidence

3.4.1 Body weight

Weaning body weight in kilograms was recorded for each of the 150 experimental animals at the beginning of the experiment.

3.4.2 Litter traits

The litter sizes at birth and at weaning were noted. The pre-weaning mortality percentage for each farrowing was worked out from the litter size at birth and at weaning. The litter weight at birth and at weaning were recorded.

3.5 Statistical analysis

Antibody titres to SRBC were transformed to $\log_e$ of antibody titre plus one so that antibody responses measured were normally distributed (Burton *et al.*, 1989a). The preweaning mortality percentage were subjected to arc sine transformation for making the distribution normal. Breed-wise mean and standard error for each of the traits under study were worked out.

Animals were grouped into five classes based on the weaning body weight. The following were the groups based on body weight.

Table 3.1 Classification of weaned piglets based on body weight

Class	Frequency
4.0-6.5	33
6.5-9.0	44
9.0-11.5	46
11.5-14.0	23
14.0-16.5	4

Least squares analyses (Harvey, 1975) was performed on 1 + $\log_e$ transformed antibody titre to SRBC in three separate steps in an attempt to distinguish the effect of breed, sex, sire and weaning body weight classes (Model 1). Model two was designed to test the effect on litter traits, adjusted for Model one effects, Model three was designed to test the effects of diarrhoea, pneumonia and pre-weaning mortality adjusted for Model one effects.

Model 1

Y_{ijkl} = $\mu + Bi + Sx_j + SR_k : B_i + wt_l + e_{ijkl}$

Where

Y_{ijkl} = Antibody titre of Y_{ijkl}^{th} piglet

μ = The overall population mean

Bi = Effect of ith breed (i= 1,2)

Sx_j = Effect of sex of piglets(j = 1,2)

SR_k:B_j = Effect of kth sire in i^{th} breed (k = 1,2,...8)

wt_l = Effect of 1th body weight class (1 = 1,2,...5)

e_{ijkl} = error

Model 2

Model 2 was used to test the effect of litter traits on antibody titre to SRBC

$Y_{iklpq} = \mu + B_i + SR_k : B_i + wt_l + SB_p \cdot SW_q + e_{ijkl}$

Where, all terms are as defined in Model 1 except

SB_p	=	Litter size at birth (p =3..............13)
SW_q	=	Liter size at weaning (q =2.....12,)

Model 3

This model was used to test the effect of occurrence of diseases and preweaning mortality on antibody response to SRBC

$Y_{ijklmno} = \mu+Bi+Sx_j+SR_k:B_i+wt_l+Dg_m+Pc_n+Mt_o+e_{ijklmno}$

Where, all terms are as defined in Model 1 expect

Dg_m	=	Incidence of diarrrhoea(0,1)
Pc_n	=	Incidence of pneumonia(0,1)
Mt_o	=	Incidence of mortality (0,1)

Least squares analysis (Harvey, 1975) was performed using similar three models on cutaneous response to phytomitogen, PHA-M and lymphocyte transformation index to BCG. Each of the immune response traits studied (j = 1,2) 1 + $\log_e$ transformed antibody responses to SRBC at 1st, 2nd, and 3rd week. Cutaneous responses to PHA-M at 0, 24, 48 and 72 hours and lymphocyte transformation index to BCG challenge at 0, 15 ,30 and 45 days post injection were tested using the above three models to assess the effect of breed, sex, sire and body

weight on the traits and the effect of these immune response traits on the incidence of disease and litter traits.

Sire and error components of variance were used to estimate heritabilities by paternal half sib correlation for each of the immune response traits studied. Approximate standard errors were computed from variance – covariance matrix of sire and error variance components. The phenotypic correlations between different immune response traits were worked out. The correlations between immune response traits and weaning body weight were estimated. The correlations between immune response traits and litter traits including litter weight at birth, at weaning and pre-weaning mortality was estimated.

RESULTS

4. RESULTS

Table 4.1 presents the breed-wise means and standard errors of weaning body weight, litter size at birth, litter size at weaning, pre weaning mortality, litter weight at birth and litter weight at weaning. Weaning bodyweight, averaged 9.501 ± 0.192 kg in Large White Yorkshire and 6.063 ± 0.062 kg in Desi pigs. The litter size at birth, litter size at weaning, pre weaning mortality percentage, litter weight at birth in kilograms and litter weight at weaning in kilograms were 8.453 ± 0.240, 6.107 ± 0.229, 27.38 ± 2.163, 15.554 ± 0.508 and 58.019 ± 2.299 for Large White Yorkshire pigs and 8.907 ± 0.219, 7.520 ± 0.697, 16.663 ± 1.974, 6.710 ± 0.163 and 45.627 ± 1.450 for Desi pigs respectively (Fig.4.1).

4.1 Humoral immune responses

4.1.1 Antibody response to sheep red blood cells (SRBC)

Table 4.2 documents the antibody titre ($1+\log_e$) to sheep RBC. Neither of the pigs belonging to Large White Yorkshire or Desi showed naturally occuring antibodies to sheep RBC as evidenced by the pre immunisation titre values. On seventh day after primary immunisation the overall antibody titre in Large White Yorkshire pigs rose to a mean value of 4.445 ± 0.163. The females had a titre of 4.864±0.198 and males had a titre of 4.448±0.147 . In the case of Desi pigs the overall antibody titre was 5.216±0.160. The mean antibody titres in the case of Desi female and male pigs were 5.419±0.200 and

Table 4. 1. Means and standard errors of litter traits in Large White Yorkshire and Desi pigs

BREED						
Parameter	**n**	**Large White Yorkshire**		**n**	**Desi Pigs**	
		Mean	**SE**		**Mean**	**SE**
Litter size at birth	75	8.453	0.240	75	8.907	0.219
Litter weight at birth (kg)	75	15.554	0.508	75	6.710	0.163
Litter size at weaning	75	6.107	0.229	75	7.520	0.697
Weaning body weight (kg)	458*	9.501	0.192	664*	6.063	0.062
Litter weight at weaning (kg)	75	58.019	2.299	75	45.627	1.450
** Pre-weaning mortality (%)	75	27.38	2.163	75	16.633	1.974

* Includes all the litter mates of the experimental animals

** Pre-weaning mortality of litter mates of experimental animals

Fig .4.1. Comparison of litter traits in Large White Yorkshire and Desi pigs

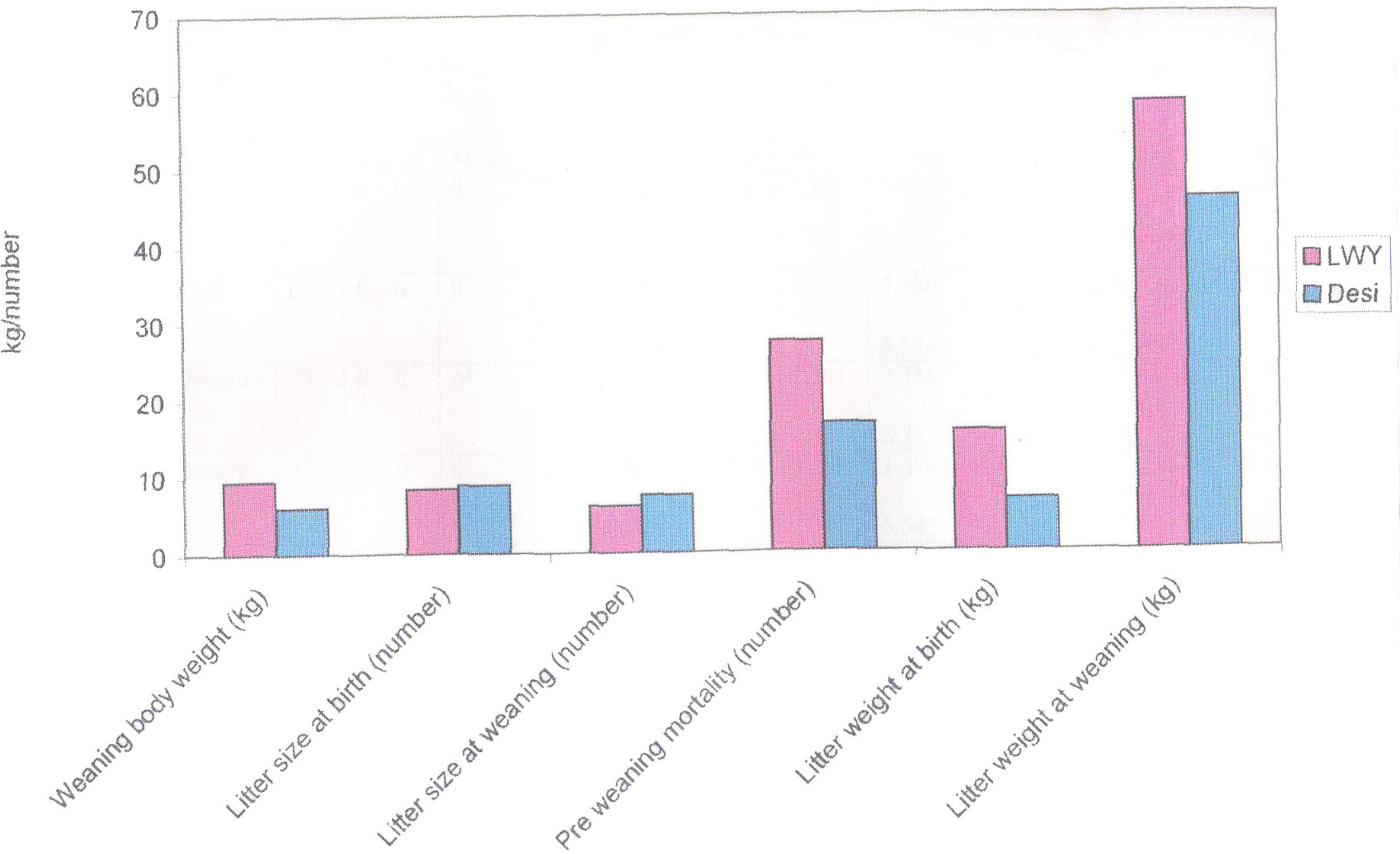


Table 4. 2. Antibody response to sheep RBC in Large White Yorkshire and Desi Pigs

Breed	Sex	n	Pre-immnunisation titre			$1+\log_e$ of titre 7th day			$1+\log_e$ of titre 15th day			$1+\log_e$ of titre 21st day		
			Range	Mean	SE	Range	Mean	SE	Range	Mean	SE	Range	Mean	SE
Large White Yorkshire	Female	40	0 – 0	0	0	3.079 – 6.545	4.864	0.198	3.079 – 6.545	4.433	0.222	2.386 – 6.545	3.971	0.204
	Male	35	0 – 0	0	0	2.386 – 6.545	4.448	0.147	2.386 – 6.545	4.256	0.149	2.386 – 6.545	3.861	0.150
	Overall	75	0 – 0	0	0	2.386 – 6.545	4.445	0.163	2.386 – 6.545	4.398	0.172	2.386 – 6.545	3.919	0.178
Desi Pigs	Female	41	0 – 0	0	0	3.079 – 6.545	5.419	0.200	3.079 – 6.545	4.688	0.203	3.079 – 6.545	4.410	0.223
	Male	34	0 – 0	0	0	2.386 – 6.545	4.971	0.136	2.386 – 6.545	4.565	0.141	2.386 – 6.545	4.284	0.151
	Overall	75	0 – 0	0	0	2.386 - 6.545	5.216	0.160	2.386 - 6.545	4.632	0.164	2.386 – 6.545	4.352	0.178
Overall		150	0 - 0	0	0	2.386 – 6.545	4.830	0.083	2.386 – 6.545	4.515	0.086	2.386 – 6.545	4.135	0.089

4.971±0.136 respectively. The overall seventh day antibody response in the 150 animals had a mean of 4.830 ± 0.083.

The fifteenth day overall post immunisation antibody response in Large White Yorkshire had a mean of 4.398±0.172. The mean antibody response in females and males were 4.433±0.222 and 4.256±0.149 respectively. In the case of Desi pigs the female, male and overall antibody response had a mean of 4.688±0.203, 4.565±0.141 and 4.632±0.164 respectively. The cumulative overall antibody response to Sheep RBC in both the breeds had a mean titre of 4.515±0.086.

The twenty first day overall antibody response in Large White Yorkshire pigs had a mean titre of 3.919±0.178. The antibody titre in females and males had a mean titre of 3.917±0.204 and 3.861±0.150 respectively. Desi pigs had an overall mean titre of 4.352±0.178 where as females and males had 4.410±0.223 and 4.284±0.151 respectively. On twenty-first day Desi females had range of 3.079 to 6.545 and both sexes in Large White Yorkshire and Desi males had a range of 2.386 to 6.545. The cumulative overall antibody response to Sheep RBC in both the breeds ranged from 2.386 to 6.545 with a mean titre of 4.135±0.089.

4.1.2 Effect of breed, sex and body weight classes

The least squares analysis of variance on the effect of breed, sex and weaning body weight on the antibody response to sheep RBC is given in Table 4.3.

The effect of breed and sex was not found to be significant (P>0.05) on the antibody response to SRBC on seventh, fifteenth and twenty first day of immunisation.

Table 4. 3. Least squares analysis of variance of the effect of breed, sex, weaning body weight classes and sires within breed on the antibody response to sheep red blood cells

Source	df	7th day		15th day		21st day	
		MSS	Probability	MSS	Probability	MSS	Probability
Breed	1	1.1896 NS	0.0689	1.0098 NS	0.1083	1.0854 NS	0.0852
Sex	1	0.8317 NS	0.4103	0.6241 NS	0.4105	0.1387 NS	0.8437
Weaning body weight	4	1.8532 NS	0.0893	1.8037 NS	0.0784	0.9384 NS	0.2783
Sires within Large White Yorkshire	7	2.476 **	0.0028	2.318 **	0.0031	2.542**	0.0038
Sires within Desi pigs	7	2.666**	0.0033	2.562**	0.0043	2.854**	0.0041
Error	129	0.6968		0.6231		0.8112	

NS – Not Significant ** - Significant at 1% level

The body weight at weaning had no significant effect on antibody response to SRBC on the seventh, fifteenth and twenty first day post immunisation. The Large White Yorkshire and Desi pigs had a least squares mean of 4.4058 ± 0.1627 and 5.0567±0.1842 on the seventh day (P=0.0689), 4.3817±0.1759 and 4.6164±0.0987 (P=0.1083) on the fifteenth day and 3.9485±0.1243 and 4.4539±0.1749 (P=0.0852) on the twenty first day respectively.

The least squares mean for the effect of sex on immune response to SRBC on seventh, fifteenth and twenty first day are presented in Table 4.4. The overall least squares mean values for females and males were 4.7385±0.2183 and 4.5893±0.1873 (P=0.4103) for seventh day, 4.5784±0.1956 and 4.4456±0.1671 (P=0.4135) for the fifteenth day and 4.2964±0.2072 and 4.1789±0.1687 (P=0.8437) for the twenty first day of post immunisation. The effect of weaning body weight classes on immune response to SRBC on seventh, fifteenth and twenty-first day of post immunisation was non significant and are presented in Table 4.4.

4.1.3 Effect of sire within breed and heritability

Least squares analyses of variances for the effect of sires within breeds are presented in Table 4.3. The effect of sires within Large White Yorkshire and Desi breed was found to be highly significant ($P<0.01$) on the antibody response to sheep RBC on the seventh day, fifteenth day and also on the twenty-first day. The effect of sires within breed both in Large White Yorkshire and Desi was highly significant. The least squares mean for each sire within Large White Yorkshire and Desi breeds on the seventh,

Table 4.4. Least squares mean of antibody response to Sheep RBC between breeds, sires within breed, sex and weaning body weight classes

Classes		Antibody response to Sheep Red Blood Cell (1+ $\log_e$)					
		7th day		15th day		21st day	
Breed	n	Mean	SE	Mean	SE	Mean	SE
		(P = 0.0689) NS		(P = 0.1083) NS		(P = 0.0852) NS	
Large White Yorkshire	75	4.4058	0.1627	4.3817	0.1759	3.9485	0.1243
Desi Pigs	75	5.0567	0.1842	4.6164	0.0987	4.4539	0.1749
Sires within LWY		**(P= 0.0028)****		**(P =0.0031)****		**(P =0.0038)****	
1	7	5.8729	0.4160	5.7922	0.4103	5.4995	0.4205
2	11	3.2487	0.3761	3.5849	0.3841	2.9843	0.3777
3	8	4.4916	0.3845	4.2177	0.3709	3.5751	0.3982
4	11	4.9708	0.3701	4.8501	0.3641	4.3912	0.3673
5	11	3.9552	0.3674	4.4902	0.3592	3.7855	0.3694
6	9	3.8741	0.5255	3.7814	0.5125	3.5817	0.5194
7	9	3.6942	0.3754	3.4698	0.3654	3.1735	0.3908
8	9	5.1390	0.3817	4.8673	0.3581	4.5973	0.3694
Sires within Desi		**(P = 0.0033)****		**(P = 0.0043)****		**(P = 0.0041)****	
1	9	4.1793	0.3693	3.5055	0.3718	3.4817	0.3491
2	9	5.3873	0.3874	4.9871	0.3541	4.2893	0.3723
3	13	5.9878	0.3491	5.8299	0.3398	5.4937	0.3194
4	10	4.8963	0.3494	4.2173	0.3551	4.3178	0.3339

Table 4.4. contd..................

5	7	5.1948	0.4157	4.8430	0.4081	4.1873	0.3974
6	9	4.7851	0.3712	4.4932	0.3697	4.5617	0.3801
7	10	4.5979	0.3598	3.8137	0.3658	3.9124	0.3488
8	8	5.4185	0.4135	5.2413	0.3917	5.3871	0.4012
Sex		**(P = 0.4103)NS**		**(P = 0.4105)NS**		**(P = 0.8437)NS**	
Female	81	4.7385	0.2183	4.5784	0.1956	4.2964	0.2072
Male	69	4.5893	0.1873	4.4456	0.1671	4.1789	0.1687
Body weight class		**(P = 0.0893) NS**		**(P = 0.0784) NS**		**(P = 0.2783) NS**	
1	33	4.9717	0.2813	4.6318	0.2723	4.4639	0.2693
2	44	4.8592	0.2398	4.5938	0.2482	4.5916	0.2284
3	46	4.7208	0.2489	4.5891	0.2398	3.9847	0.2394
4	23	4.5983	0.2718	4.3986	0.2837	4.1837	0.3178
5	4	4.6918	0.9864	4.2917	0.5987	4.3984	1.0891

NS – Not Significant * - Significant at 5% level ** - Significant at 1% level

fifteenth and twenty-first day are presented in Table 4.4 and diagrammatically presented in Fig.4.2 and 4.3 respectively.

Heritability estimates of the antibody response to SRBC are presented in Table 4.17. The heritability estimates were very high with values of 0.8969±0.4238, 0.9187±0.4893 and 0.8174±0.4387 respectively for the seventh, fifteenth and twentyfirst day post immunisation.

4.1.4 Effect of litter traits

The least squares mean for the effect of litter size at birth and litter size at weaning on the antibody response to SRBC are presented in Table 4.5. The least squares mean for different litter size at birth had a probability of P=0.4978, P=0.5178 and P=0.6438 for seventh, fifteenth and twenty-first day respectively. and litter size at weaning were non significant. The least squares mean for different litter size at weaning had probability values of P=0.3987, P=0.5138 and P=0.5891 for seventh, fifteenth and twenty first day of post immunisation respectively.

4.1.5 Effect of diseases and pre weaning mortality

Least squares means for the effect of antibody response to SRBC on the occurrence of diarrhoea, pneumonia and pre weaning mortality among piglets is given in Table 4.6. The effect of antibody response to SRBC on seventh, fifteenth and twenty first day after primary immunisation was not found to be significant for the occurrence of diarrhoea, pneumonia and pre-weaning mortality among piglets.

Table 4.5. Least squares means for the effect of litter traits among Large White Yorkshire and Desi Pigs on antibody response to Sheep RBC

Independent Variables	n	Antibody response to Sheep RBC (1 + $\log_e$ of titre)					
		7th day		15th day		21st day	
		Mean	SE	Mean	SE	Mean	SE
Litter size at birth		(P=0.4978)NS		(P=0.5178)NS		(P=0.6438)NS	
3	2	3.6893	1.3381	3.7896	1.2893	3.2831	1.0981
4	3	4.3894	0.8179	4.2893	0.8274	4.0283	0.7983
5	9	4.3979	0.6017	4.2118	0.5998	3.9849	0.5987
6	7	4.6381	0.6260	4.5837	0.6128	4.0813	0.6139
7	14	4.3187	0.5950	4.1711	0.5632	3.9460	0.6014
8	19	5.2839	0.5843	5.1283	0.5717	4.3896	0.5789
9	44	4.8431	0.4718	4.6183	0.4597	4.4389	0.4817
10	22	4.4583	0.4917	4.3987	0.4893	3.9846	0.5081
11	15	5.1739	0.5173	4.6893	0.5917	4.8961	0.6978
12	12	4.2781	0.5896	4.1789	0.6817	4.1913	0.6172
13	3	5.2891	1.2963	4.9637	1.0983	4.8713	1.1738
Litter size at weaning		(P=0.3987)NS		(P=0.5138)NS		(P=0.5138)NS	
2	2	3.7817	0.6931	3.8974	0.5060	3.4987	0.7183
3	5	5.4986	0.9137	5.2789	0.8713	5.1853	0.9186
4	11	4.4981	0.4867	4.1376	0.5187	4.2895	0.5098

Table 4.5. contd................

5	15	4.7963	0.5168	4.5861	0.4986	4.3178	0.4876
6	13	4.8737	0.6172	4.3857	0.5060	4.4387	0.5987
7	22	4.5819	0.4308	4.2987	0.4187	4.1087	0.3978
8	27	5.1087	0.5176	4.4987	0.3918	4.3897	0.4832
9	32	4.8751	0.3998	4.5524	0.3817	4.3896	0.3184
10	10	5.1738	0.6130	4.8731	0.5450	4.7893	0.5968
11	8	4.5893	0.6287	3.9876	0.6317	3.8176	0.6483
12	5	5.0813	0.8961	4.7183	0.7918	4.5818	0.8093

NS – Not Significant

Fig . 4.2. Antibody response to sheep RBC sires within Large White Yorkshire

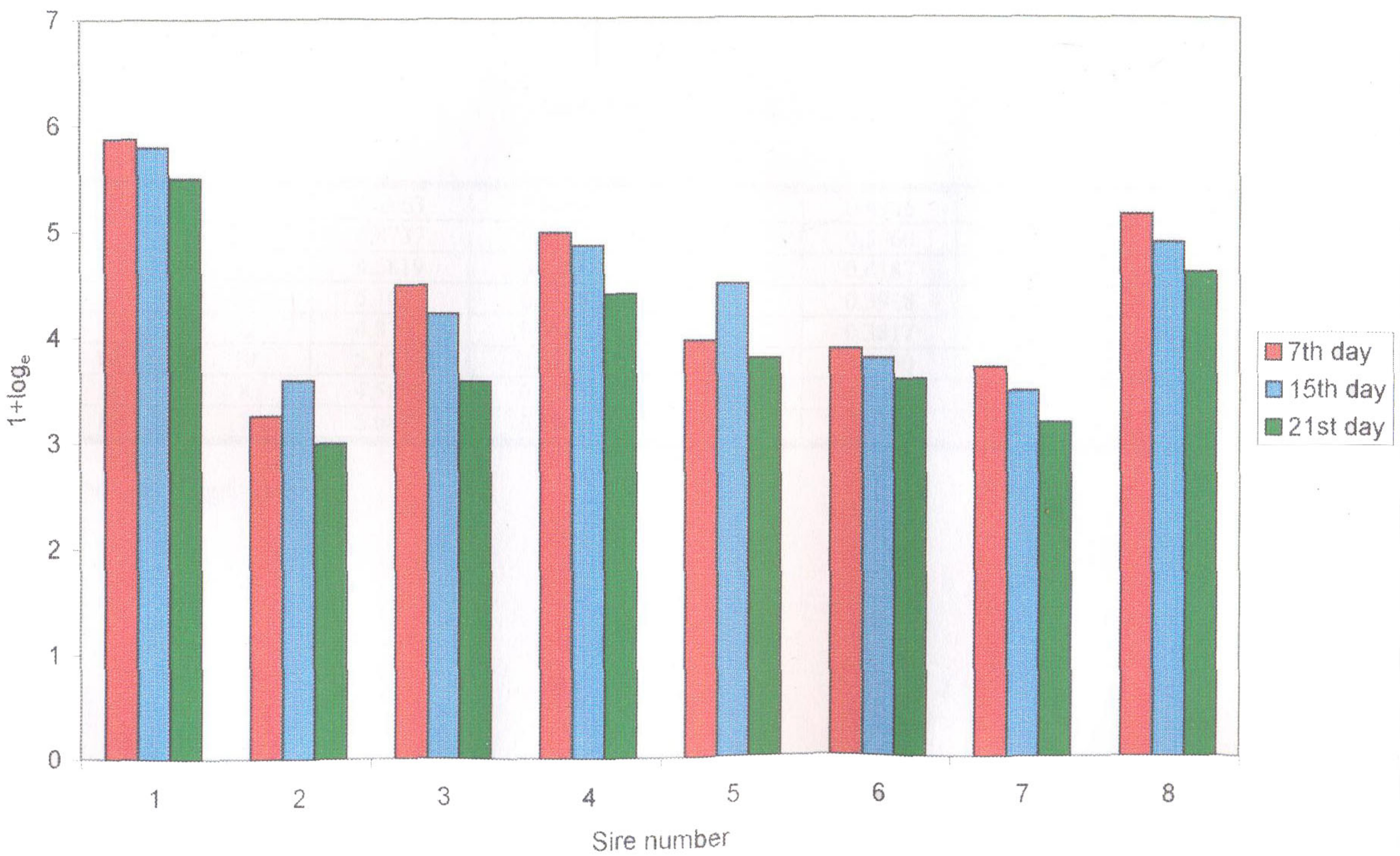
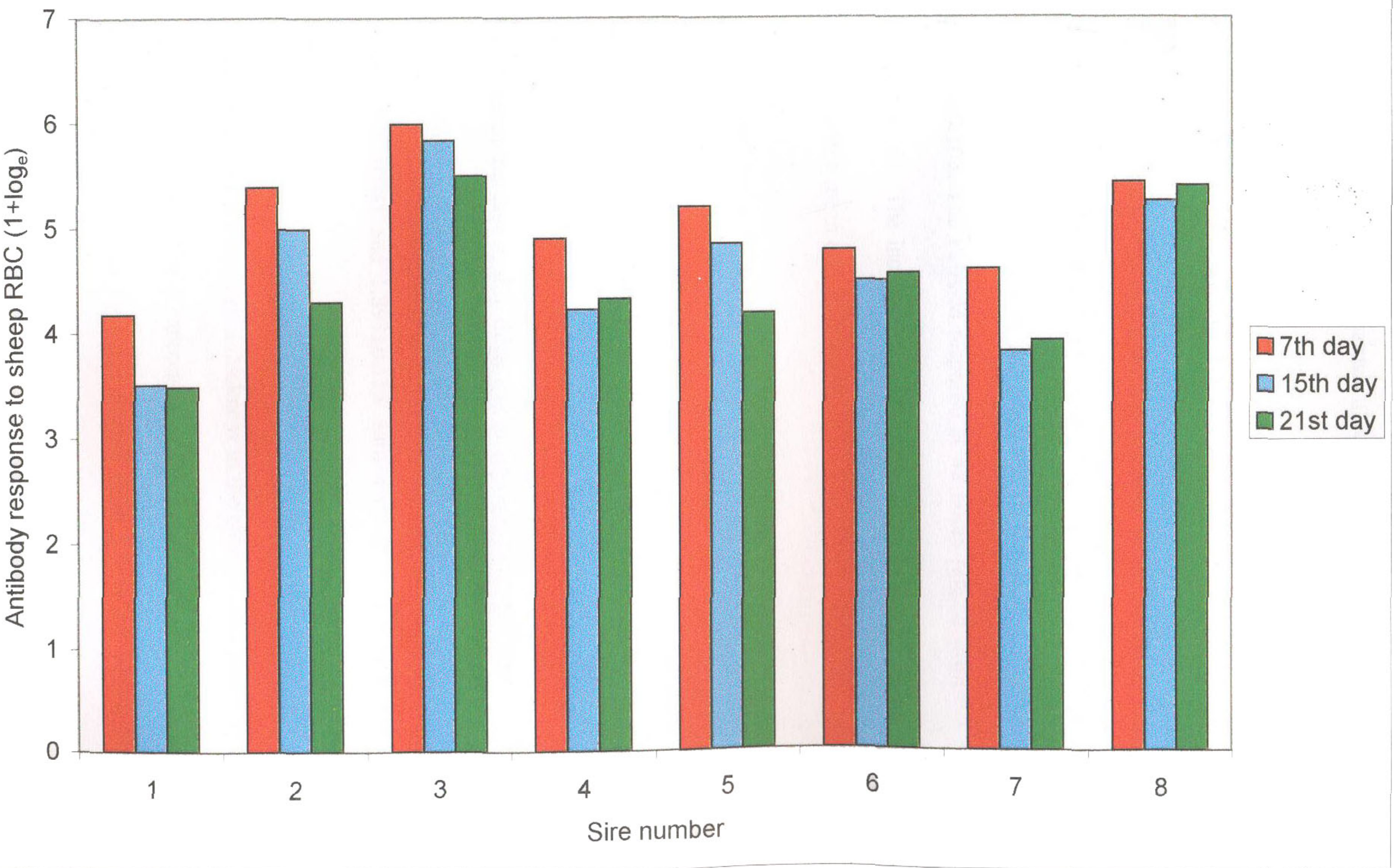


Table 4.6. Least squares means for the effect of occurrence of diarrhoea, pneumonia and pre-weaning mortality among piglets on antibody response to Sheep RBC

Independent variables		n	7th day		15th day		21st day	
			Mean	SE	Mean	SE	Mean	SE
Diarrhoea			(P=0.3174)NS		(P=0.2753)NS		(P=0.2693)NS	
	Yes	69	4.5987	0.3478	4.3891	0.3387	4.2178	0.3481
	No	81	4.8731	0.3218	4.5387	0.3159	4.5937	0.3108
Pneumonia			(P=0.1892)NS		(P=0.16180)NS		(P=0.0963)NS	
	Yes	11	4.8173	0.5891	4.4893	0.6831	4.0891	0.6183
	No	139	4.7891	0.1981	4.4317	0.1281	4.1998	0.1173
Pre-weaning mortality			(P=0.2974)NS		(P=0.4672)NS		(P=0.3182)NS	
	Yes	94	4.7861	0.3108	4.5116	0.2983	3.9874	0.2873
	No	56	4.7139	0.3786	4.3871	0.3876	4.2987	0.3582

NS – Not Significant

Fig . 4.3. Antibody response to sheep RBC sires within Desi



4.2 Cell mediated immune responses

4.2.1 Cutaneous response to intradermal injection to phytohaemagglutinin-M

The cutaneous response to intradermal injection with phytohaemagluttinin-M (PHA M) is given in Table 4.7 and Fig. 4.4.

The overall pre injection skin thickness in Large White Yorkshire ranged from 2.2 to 5.0 with a mean of 3.508±0.045. The cutaneous response to PHA-M 24 hours post-injection as evidenced by increase in skin thickness ranged from 1.0 to 4.8 with a mean value of 3.253±0.103. The PHA-M response at 48 hours ranged from 0.5 to3.9 with a mean of 2.623±0.101. The increase in skin thickness at 72 hours post injection ranged from 0.0 to 3.1 with a mean of 1.927±0.010.

The females had a mean pre injection skin thickness of 3.487±0.052 with a range of 2.2 to 4.8 and males had a pre injection skin thickness of 3.523±0.078 with a range of 2.5 to 5.0. The increase in skin thickness 24 hours post injection in case of female and male are 3.365±0.148 with range of 1.0 to 4.8 and 3.125±0.159 with range of 1.0 to 4.5. The 48 hour mean increase in skin thickness in females and males were 2.672±0.118 and 2.568±0.148 with a range of 0.5 to 3.5 and 0.5 to 3.9. The 72 hour skin thickness in both sexes were 1.958±0.081 and 1.893±0.113 with a range of 0.5 to 2.9 and 0.5 to 3.1 respectively.

The overall pre injection skin thickness in the case of Desi pigs was 3.012±0.044 with a range of 1.9 to 4.5. The cutaneous response to PHA-M 24 hours post injection as evidenced by increase in skin thickness ranged from 1.2 to 4.8 with a mean value of

Table 4.7. Cutaneous response to intradermal injection of PHA – M in Large White Yorkshire and Desi Piglets

Breed	Sex	n	Pre-injection skin thickness (mm)			Post-injection increase in skin thickness (mm)								
						24 hours			48 hours			72hours		
			Range	Mean	SE	Range	Mean	SE	Range	Mean	SE	Range	Mean	SE
Large White Yorkshire	Female	40	2.2 – 4.8	3.487	0.052	1.0 – 4.8	3.365	0.148	0.5 – 3.5	2.672	0.118	0.5 – 2.9	1.958	0.081
	Male	35	2.5 – 5.0	3.532	0.078	1.0 – 4.5	3.125	0.159	0.5 – 3.9	2.568	0.148	0.5 – 3.1	1.893	0.113
	Overall	75	2.2 – 5.0	3.508	0.045	1.0 – 4.8	3.253	0.103	0.5 – 3.9	2.623	0.101	0.0 – 3.1	1.927	0.10
Desi Pigs	Female	41	1.9 – 4.2	2.879	0.049	1.2 – 4.8	3.575	0.149	0.5 – 3.8	2.816	0.139	0.5 – 3.0	1.937	0.120
	Male	34	2.0 – 4.5	3.173	0.076	1.5 – 4.8	3.483	0.162	1.0 – 3.7	2.698	0.158	0.5 – 3.0	1.789	0.137
	Overall	75	1.9 – 4.5	3.012	0.044	1.2 – 4.8	3.533	0.108	0.5 – 3.8	2.762	0.107	0.5 – 3.0	1.870	0.098
Overall		150	1.9 – 5.0	3.26	0.031	1.0 – 4.8	3.393	0.053	0.5 – 3.9	2.693	0.051	0.0 – 3.1	1.899	0.050

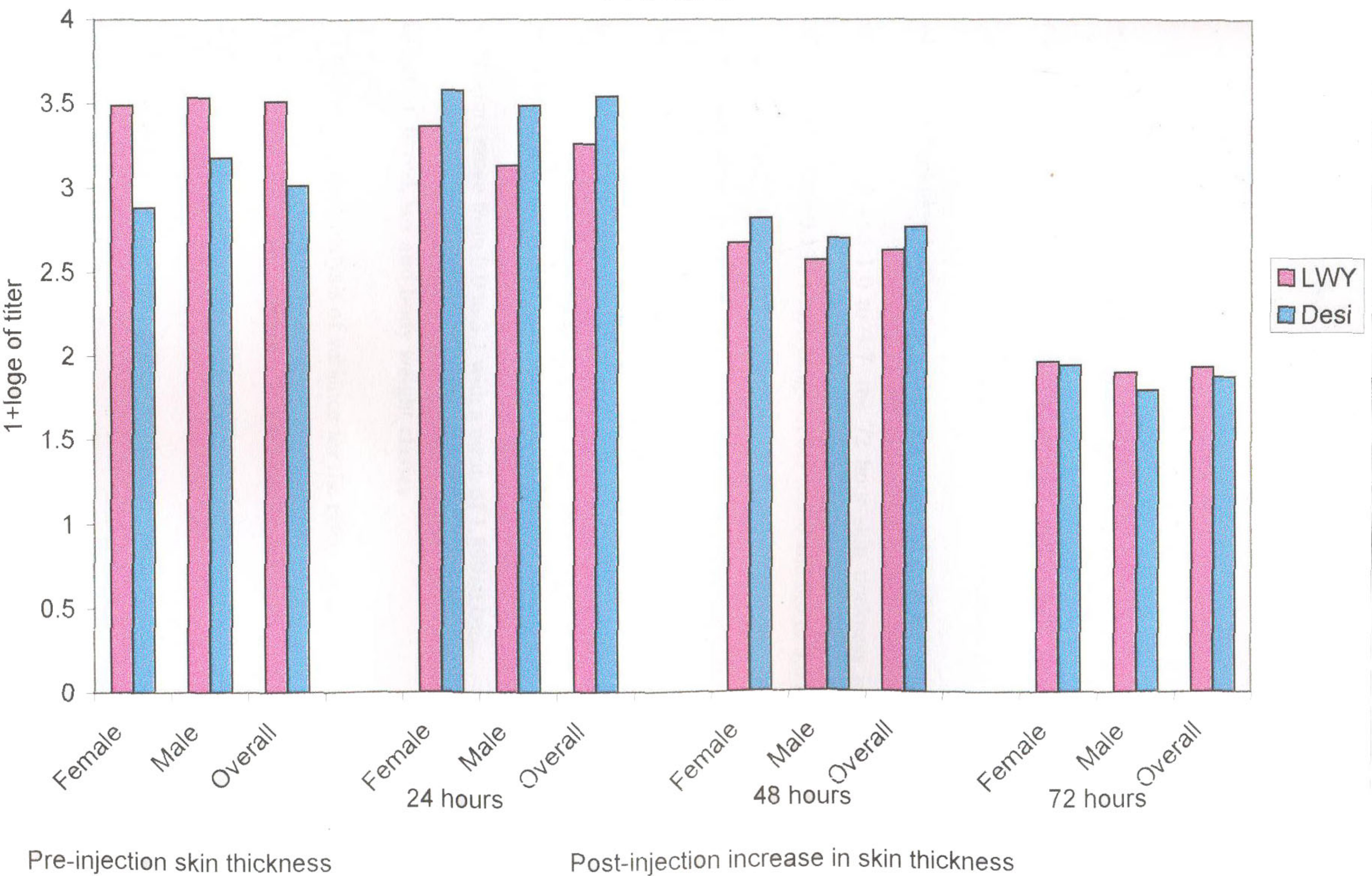


Fig. 4.4 Cutaneous response to intra dermal injection of PHA - M in Large White Yorkshire and Desi Piglets

3.533±0.108. The PHA-M response at 48 hours ranged from 0.5 to3.8 with a mean of 2.762±0.107. The increase in skin thickness at 72 hours post-injection ranged from 0.5 to 3.0 with a mean of 1.870±0.098.

The mean pre-injection skin thickness in the case of Desi female and male pigs were 2.879±0.049 and 3.173±0.076 with a range of 1.9 to 4.2 and 2.0 to 4.5 respectively. The mean increase in skin thickness for females and males at 24 hours was 3.575±0.149 and 3.483±0.162 with a range of 1.2 to 4.8 and 1.5 to 4.8 respectively. The 48 hour mean increase in skin thickness in females and males were 2.816±0.139 and 2.698±0.158 with a range of 0.5 to 3.8 and 1.0 to 3.7. the 72 hour skin thickness in both sexes were 1.937±0.120 and 1.789±0.137 with a range of 0.5 to 3.0 and 0.5 to 3.0 respectively.

The cumulative overall pre injection skin thickness of the 150 animals tested from both the breeds ranged from 1.9 to 5.0 with a mean of 3.26±0.031.The increase in skin thickness 24 hours post injection range from 1.0 to 4.8 with a mean of 3.393±0.053. The increase in skin thickness at 48 hours range from 0.5 to 3.9 with a mean of 2.693±0.051 and for 72 hours range from 0.0 to 3.1 with a mean of 1.899±0.050.

4.2.2 Effect of breed, sex and body weight classes

The least squares analysis of variance for the effect of breed, sex and body weight at weaning on the cutaneous response to intradermal injection of PHA-M is given in Table 4.8.

The effect of breed on pre-immunisation skin thickness was significant (P=0.0498) where as the effect of sex on pre-immunisation skin thickness within the

breed was not significant (P=0.3685). The effect of breed on 24, 48 and 72 hours post injection increase in skin thickness was not significant. Similarly the effect of sex within breed for 24, 48 and 72 hours post injection increase in skin thickness were also not significant.

The effect of weaning bodyweight classes for pre immunisation skin thickness was significant (P=0.0501). But the post injection increase in skin thickness after 24, 48 and 72 hours were non significant (P=0.5271,0.6758 and 0.8538) respectively.

The least squares mean for the effect of sex on cutaneous skin response to intradermal injection of PHA-M along with pre injection skin thickness are presented in Table 4.9. The overall least squares mean value for female and male were 3.2165±0.6513 and 3.3188±0.7299 (P=0.3685) as pre injection skin thickness, 3.3828±0.7138 and 3.2516±0.5893 (P=0.1218) as 24 hour post injection increase in skin thickness, 2.6017±0.5178 and 2.5797±0.7081 (P=0.2075) as 48 hour increase in skin thickness and 1.8695±0.6397 and 1.8287±0.4875 (p=0.3093) as 72 hour post injection increase in skin thickness. The weaning body weight class differed significantly for pre injection skin thickness (P=0.0501) but the post injection increase in skin thickness were not significantly affected by the weaning body weight classes at 24, 48 and 72 hours post injection (P=0.5271,0.6758 and 0.8538).The least squares mean for different body weight classes are presented in Table 4.9.

4.2.3 Effect of sires within breed and heritability

Least squares analyses of variances for the effect of sires within breeds are presented in Table 4.8.

Table 4.8. Least squares analysis of variance on the effect of breed, sex, weaning body weight classes and sire within breed on the cutaneous response to intradermal injection of PHA-M

Source of Variation	df	Zero Hour		24 hours post-injection increase		48 hours post-injection increase		72 hours post-injection increase	
		MSS	Probability	MSS	Probability	MSS	Probability	MSS	Probability
Breed	1	1.9646*	0.0498	1.4589 NS	0.0567	1.2936 NS	0.0759	0.9958 NS	0.1093
Sex	1	0.3974 NS	0.3685	0.8973 NS	0.1218	0.6185 NS	0.2075	0.4173 NS	0.3093
Weaning body weight	4	0.8973*	0.0501	0.4994 NS	0.5271	0.2678 NS	0.6758	0.1045 NS	0.8538
Sires within Large White Yorkshire	7	0.1892 NS	0.5676	1.9538**	0.0070	1.3945*	0.0173	0.8311 *	0.0483
Sires within Desi pigs	7	0.2169 NS	0.3217	2.0975**	0.0035	1.1873*	0.0321	1.2891 *	0.0381
Error	129	0.1998		0.5473		0.4108		0.3095	

NS – Not Significant   * - Significant at 5% level   ** - Significant at 1% level

The effect of sires on pre-injection skin thickness of Large White Yorkshire and Desi were not significant. But for 24 hours post injection increase in skin thickness was highly significant for both the sires within Large White Yorkshire and Desi pigs (P=0.0070 and 0.0035). The post-injection increase in skin thickness for 48 and 72 hours were significant between sires within Large White Yorkshire (P=0.0173 and 0.0321) and for Desi sires (P=0.0483 and 0.0380). The least squares mean for pre-injection skin thickness and post-injection increase in skin thickness for 24,48 and 72 hours for each sire within Large White Yorkshire and Desi breeds are given in Table 4.9 and diagrammatically shown in Fig.4.5and 4.6 respectively.

The least squares mean to the cutaneous response to PHA-M injection among breed and the sires within breed is presented in Table 4.9. The Large White Yorkshire and Desi pigs had a least squares mean of 3.4375±0.7595 and 3.0895±0.6386 as preinjection skin thickness and was significantly different (P=0.0498). The increase in skin thickness at 24, 48 and 72 hours for Large White Yorkshire and Desi pigs were not significant (P=0.0567,0.0759 and 0.1093). The post injection increase in skin thickness on 24, 48 and 72 hours for Large White Yorkshire had a least squares mean of 3.0058±0.6628, 2.3273±0.5689 and 1.5730±0.6015 whereas the Desi pigs had least squares means of 3.4178±0.7024, 2.6575±0.6178 and 1.8109±0.5875 respectively.

The heritability estimates for skin thickness and cutaneous response to PHA-M at twenty four, forty eight and seventy two hours post injection are presented in Table 4.17. High heritability of 0.5173±0.4179, 0.8136±0.5843, 0.6816±0.5187 and 0.7134±0.5283

Table 4.9 Least squares mean for the effect of breed, sires within breed, sex and weaning body weight class on the pre-injection skin thickness and PHA-M responses to 24, 48 and 72 hours post-injection

Class	n	Pre-injection skin thickness		Increase in skin thickness as PHA –M Response					
				24 Hours		48 hours		72 hours	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
Breed	1	(P=0.0498)*		(P=0.0567)NS		(P=0.0759)NS		(P=0.1093)NS	
Large White Yorkshire	75	3.4375	0.0759	3.0058	0.0662	2.3273	0.0568	1.5730	0.0601
Desi	75	3.0895	0.0638	3.4178	0.0702	2.6575	0.0617	1.8109	0.0587
Sires within Large White Yorkshire		(P=0.5676)NS		(P=0.0070)**		(P=0.0173)*		(P=0.0123)*	
1	7	3.3258	0.3650	4.0138	0.4137	3.1085	0.3783	2.1968	0.3578
2	11	3.1986	0.3126	1.9831	0.3685	1.5983	0.3135	1.2780	0.3010
3	8	3.5173	0.2981	3.7489	0.4316	2.8713	0.3987	1.9873	0.3294
4	11	3.4870	0.2875	2.8985	0.4108	1.7035	0.3389	1.3098	0.3172
5	11	3.2988	0.2918	3.1782	0.3978	2.1938	0.3289	1.0738	0.2978
6	9	3.5740	0.3086	2.9843	0.4031	2.3895	0.3875	1.9729	0.3584
7	9	3.7105	0.3185	3.2815	0.4085	2.3915	0.3511	1.7985	0.3289
8	9	3.4450	0.3108	2.8953	0.3481	2.1895	0.3418	1.0835	0.3092

Table 4.9 contd..........

Sires within Desi		(P=0.3217)NS		(0.0035)**		(P=0.0321)*		(P=0.0380)*	
1	9	3.2987	0.3045	2.8793	0.2987	2.4895	0.2491	1.6985	0.2371
2	9	3.0910	0.2995	3.0955	0.2847	2.5361	0.2511	1.8173	0.2291
3	13	3.1081	0.2410	3.8598	0.2510	3.1785	0.2173	2.2078	0.1739
4	10	2.9387	0.2695	2.6135	0.2718	1.8085	0.2095	1.2871	0.1875
5	7	2.8395	0.3385	4.2873	0.3131	3.6138	0.2931	2.7893	0.2594
6	9	3.1985	0.3185	3.8375	0.3098	2.6123	0.2617	1.7778	0.2413
7	10	3.0125	0.2981	3.2185	0.2873	2.4817	0.2498	1.3198	0.2598
8	8	3.1255	0.3181	3.6881	0.3291	2.7137	0.3018	1.8611	0.3108
Sex		**(P=0.3685)NS**		**(P=0.2318)NS**		**(P=0.3575)NS**		**(P=0.8930)NS**	
Female	81	3.2165	0.0651	3.3826	0.0589	2.6017	0.0517	1.8695	0.0487
Male	69	3.3188	0.0729	3.2516	0.0713	2.5797	0.0708	1.8287	0.0639
Weaning body weight class		**(P=0.0501)***		**(P=0.5271)NS**		**(P=0.6758)NS**		**(P=0.8538)NS**	
1	33	2.7781	0.4131	3.4013	0.3879	2.7316	0.3689	1.9958	0.3516
2	44	3.1756	0.3981	3.3817	0.3573	2.5289	0.3178	1.8573	0.3218
3	46	3.4895	0.3098	3.2173	0.3173	2.5956	0.3081	1.8607	0.3011
4	23	3.7015	0.5289	3.2993	0.4931	2.5408	0.4172	1.6046	0.3817
5	4	3.7489	0.9137	3.2516	0.8139	2.2573	0.9137	1.7533	0.9283

NS – Not Significant   * - Significant at 5% level   ** - Significant at 1% level

Fig . 4.5 Pre injection skin thickness and PHA-M responses at 24, 48 and 72 hours post injection in Large White Yorkshire

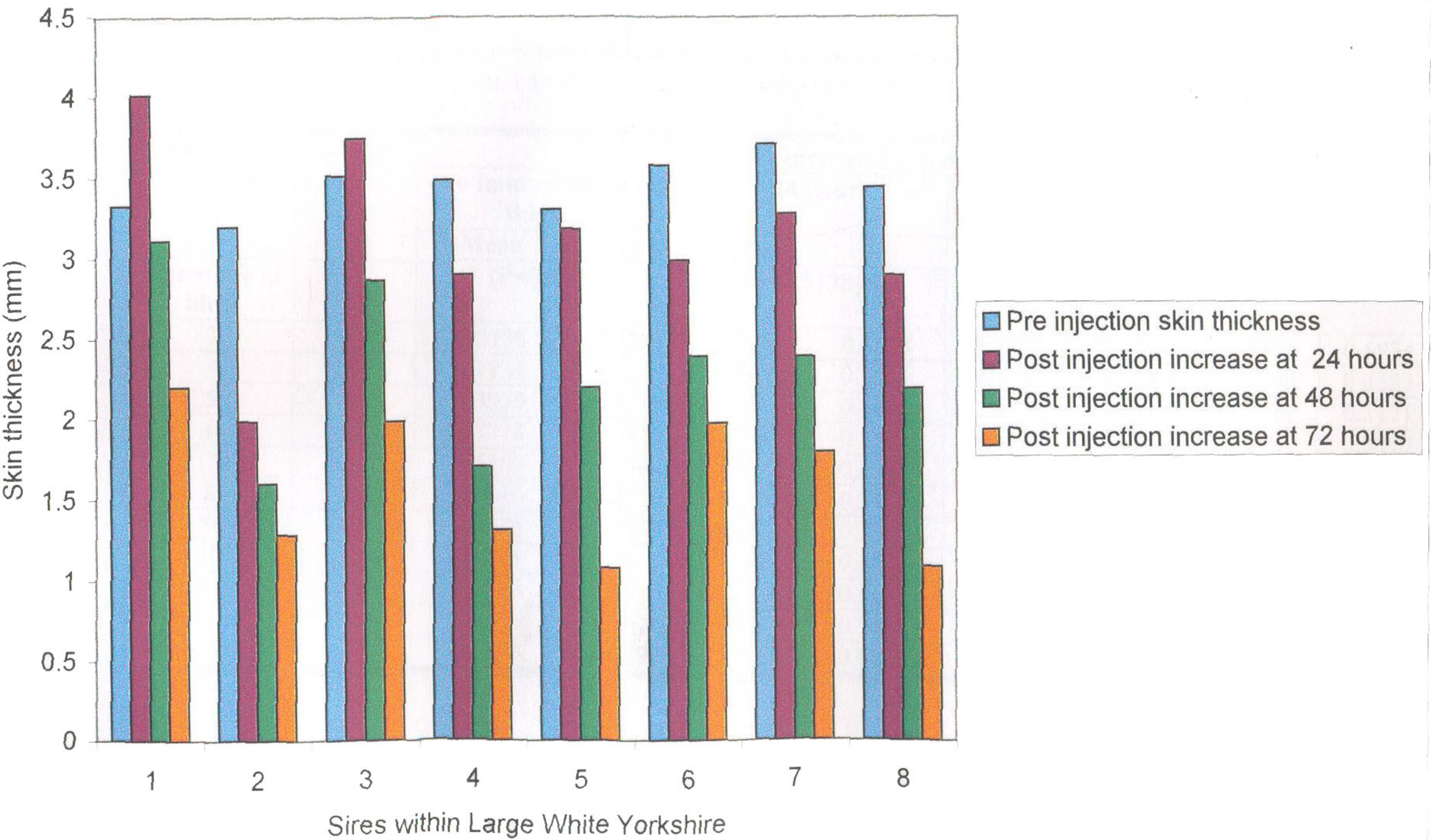


Table 4.10 Least squares mean for the cutaneous response to injection of PHA-M based on litter traits of Large White Yorkshire and Desi piglings

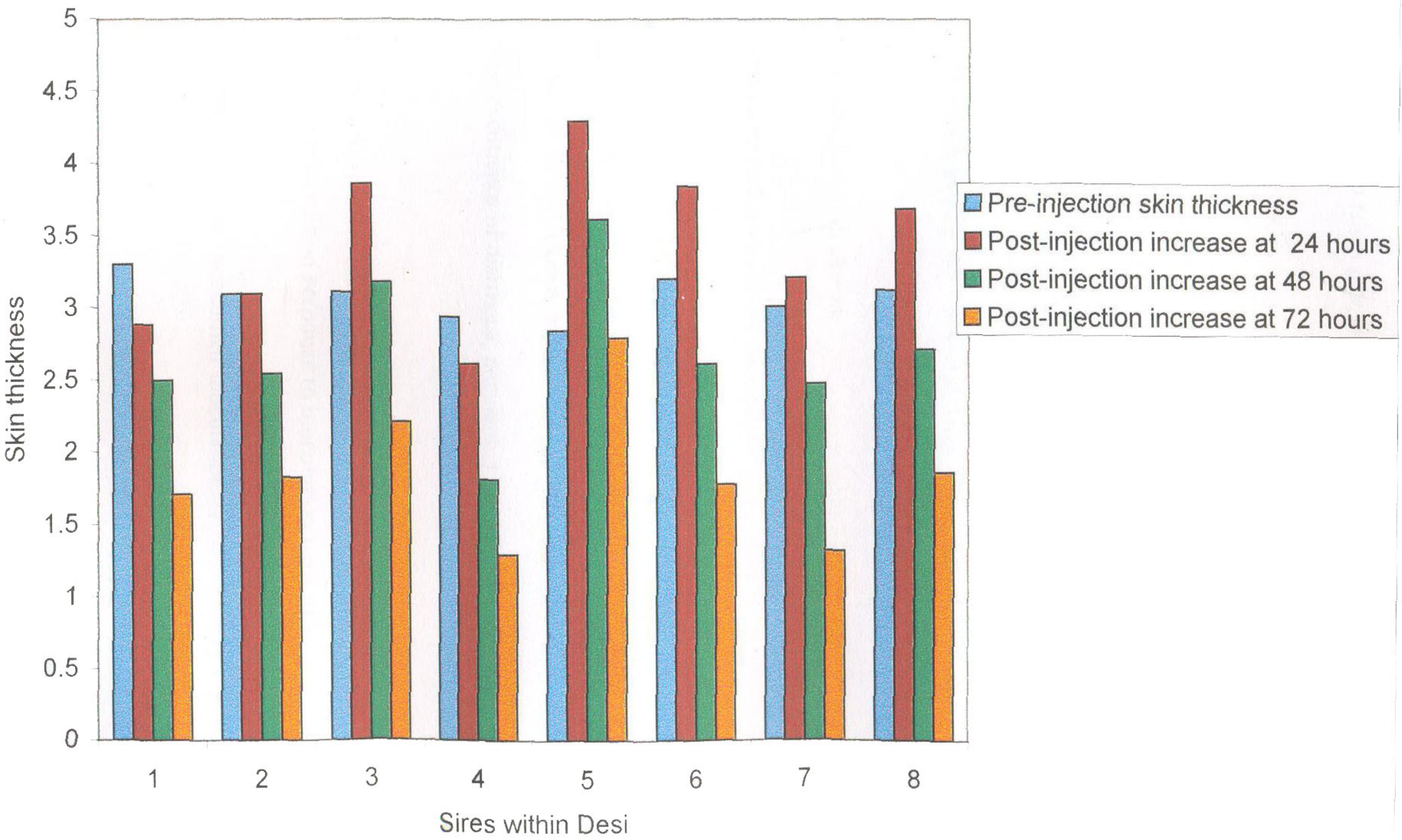
Independent variable	n	Increase in skin thickness							
		Pre-immunization skin thickness		24 hours		48 hours		72 hours	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
Litter size at birth		(P=0.0417)*		(P=0.5178)NS		(P=0.6773)NS		(P=0.7938)NS	
3	2	4.6138	0.7381	2.7185	0.6198	2.5289	0.7102	2.3411	0.6934
4	3	4.1735	0.6837	2.3411	0.3911	2.2193	0.3715	1.5890	0.3583
5	9	4.3919	0.4217	3.3185	0.3291	2.8734	0.307	1.7713	0.3171
6	7	3.3718	0.4873	2.9481	0.3572	2.6315	0.3671	1.6341	0.3417
7	14	3.5103	0.3581	3.4575	0.3598	2.5185	0.3313	1.8938	0.3174
8	19	3.3116	0.3156	3.7163	0.3171	2.2835	0.2921	1.7019	0.2781
9	44	3.2875	0.2153	3.4170	0.2208	2.5791	0.1928	1.8891	0.1829
10	22	3.0841	0.3011	3.3481	0.4598	2.8710	0.3998	2.0178	0.3701
11	15	2.8173	0.3628	3.1874	0.5118	2.4806	0.4819	1.7935	0.4917
12	12	2.5011	0.3814	3.8731	0.7017	3.4731	0.6811	2.8175	0.6979
13	3	2.7333	0.6390	4.0855	0.8108	3.7168	0.7981	3.2178	0.8070

Table 4.10 contd..........

Litter size at weaning	n	(P=0.1873)NS		(P=0.3804)NS		(P=0.4173)NS		(P=0.6175)NS	
2	2	4.0875	0.7183	3.2010	0.5585	2.6595	0.5618	2.2955	0.5318
3	5	4.2893	0.6573	3.0987	0.4671	2.2175	0.4713	1.9939	0.4598
4	11	3.9847	0.3974	3.5614	0.4116	2.3875	0.3814	1.4879	0.3718
5	15	3.3161	0.3398	3.2986	0.3871	3.3178	0.3784	2.2315	0.3217
6	13	3.5243	0.4093	3.3489	0.2593	2.1482	0.2410	1.8596	0.2017
7	22	3.2561	0.3175	3.7162	0.4083	2.8917	0.3847	1.8739	0.3491
8	27	3.1758	0.2985	3.1985	0.2897	2.5131	0.2673	1.7895	0.2281
9	32	3.0173	0.2431	2.8938	0.3218	2.6011	0.3318	1.5873	0.3010
10	10	3.1691	0.4019	3.6175	0.3281	2.9175	0.3091	1.8371	0.2678
11	8	2.7768	0.4591	3.8167	0.6971	2.9873	0.5991	1.9378	0.5178
12	5	2.5169	0.5931	3.4895	0.6018	3.0748	0.6073	2.1787	0.5537

NS – Not Significant   * - Significant at 5% level   ** - Significant at 1% level

Fig . 4.6 Pre injection skin thickness and PHA-M responses at 24, 48 and 72 hours post injection in Desi



were estimated for initial skin thickness, cutaneous response to PHA-M at twenty four, forty eight and seventy two hours post injection respectively.

4.2.4 Effect of litter traits

The least squares mean for the effect of litter size at birth and litter size at weaning on the cutaneous response to injection of PHA-M are presented in Table 4.10. The effect of litter size at birth was significant (P=0.0417) on pre-immunisation skin thickness whereas the effect of litter size at birth was not significant for the increase in skin thickness at 24, 48 and 72 hour post injection. The effect of litter size at weaning was not significant for pre immunisation skin thickness as well as the 24, 48 and 72 hour post injection increase in skin thickness.

4.2.5 Effect of diseases and preweaning mortality

Least squares mean for the effect of occurrence of diarrhoea, pneumonia and pre weaning mortality among piglets on intradermal injection of PHA-M is given in Table 4.10. The increase in skin thickness at 24, 48 and 72 hour post injection was not significant for the occurrence of diarrhoea, pneumonia and pre weaning mortality among piglets.

4.3 Lymphocyte transformation test

4.3.1 Lymphocyte transformation response to inoculation with BCG

The stimulation index to intradermal inoculation of BCG is given in Table 4.12 and Fig.4.7.

The overall pre inoculation stimulation index in Large White Yorkshire ranged from 1.000 to 1.703 with a mean of 1.076±0.012. The stimulation index 15 days post inoculation ranged from 3.821 to 8.507 with a mean value of 6.0161±0.1131, for 30 days post inoculation range from 3.259 to 8.631 with a mean of 6.1070±0.0910 and for 45 days post inoculation ranged from 3.152 to 8.018 with a mean of 6.0020±0.1020.

The females had a mean pre inoculation stimulation index of 1.100±0.019 with a range of 1.000 to 1.583 and males had a pre inoculation stimulation index of 1.049±0.021 with a range of 1.00 to 1.783. The stimulation index 15 day post inoculation in case of female and male were 5.984±0.2101 with range of 3.821 to 8.278 and 6.0535±0.2381 with range of 3.987 to 8.507. The 30 day post inoculation stimulation index in females and males were 6.086±0.1240 and 6.1301±0.1380 with a range of 3.501 to 8.319 and 3.259 to 8.631. The 45 day post inoculation stimulation index in both sexes were 5.9755±0.130 and 6.0335±0.127 with a range of 3.281 to 7.912 and 3.157 to 8.018 respectively.

The overall pre inoculation stimulation index in the case of Desi pigs was 1.094±0.013 with a range of 0.908 to 1.598. The 15 day post inoculation stimulation index ranged from 3.875 to 8.531 with a mean value of 6.3340±0.1541, for 30 day post inoculation ranged from 3.795 to 8.491 with a mean of 6.5920±0.1340 and at 45 day post inoculation ranged from 3.945 to 8.445 with a mean of 5.9890±0.109.

The mean pre inoculation stimulation index in the case of Desi female and male pigs were 1.097±0.018 and 1.090±0.022 with a range of 1.000 to 1.489 and 0.908 to

Table 4.11 Least squares mean for cutaneous response to PHA-M for the incidence of diarrhoea, pneumonia and pre-weaning mortality

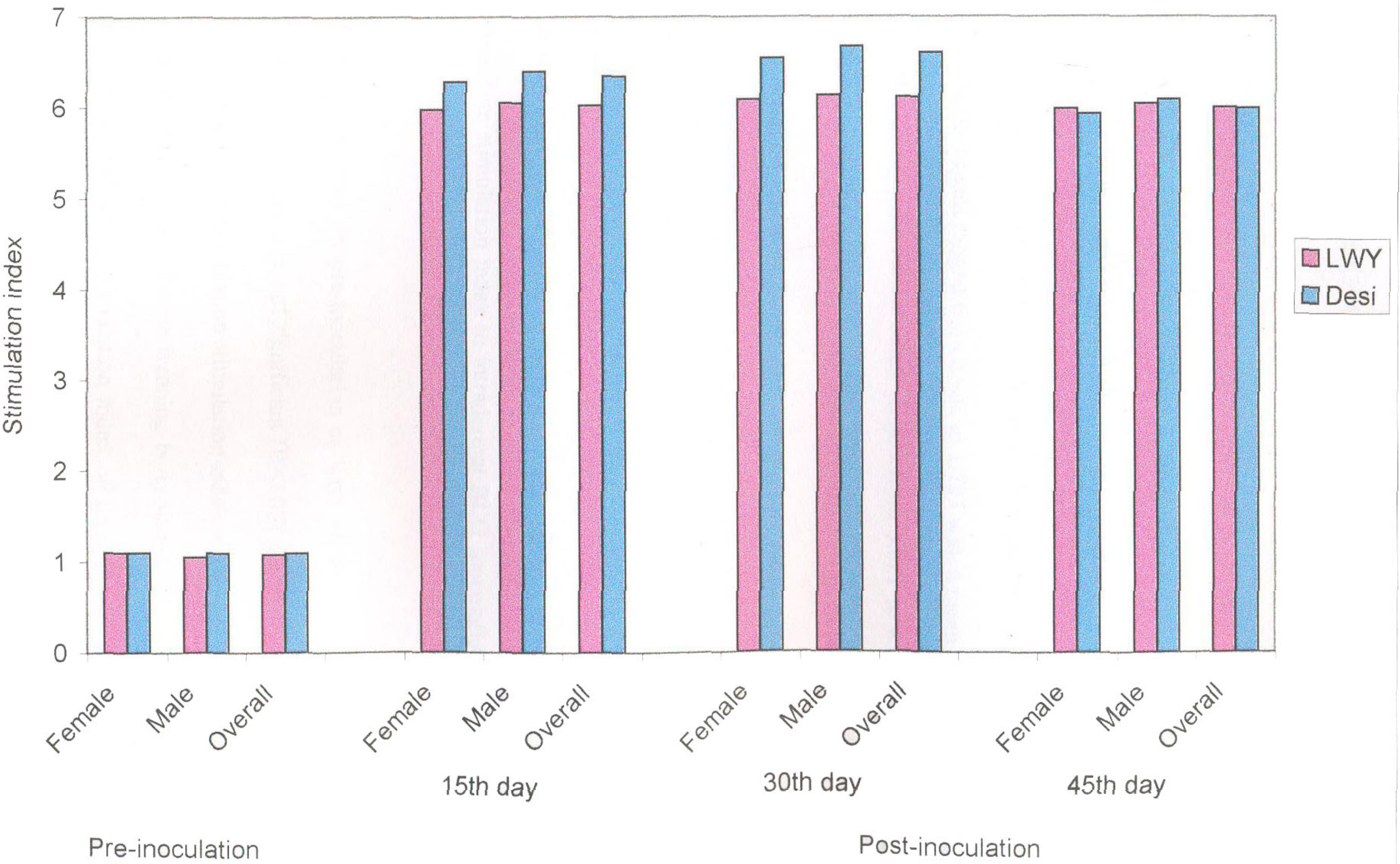
Independent variable		n	24 hours		48 hours		72 hours	
			Mean	SE	Mean	SE	Mean	SE
Diarrhea			(P=0.2531)NS		(P=0.5781)NS		(P=0.4173)NS	
	Yes	69	3.2925	0.3175	2.5178	0.3075	1.7995	0.3173
	No	81	3.3275	0.2895	2.6555	0.2731	1.8891	0.2593
Pneumonia			(P=0.5127)NS		(P=0.6134)NS		(P=0.5341)NS	
	Yes	11	3.1839	0.7103	2.4985	0.6931	1.8537	0.6872
	No	139	3.3219	0.2173	2.5994	0.2218	1.8483	0.2093
Pre-weaning mortality			(P=0.4876)NS		(P=0.5631)NS		(P=0.6147)NS	
	Yes	94	3.2875	0.2615	2.5598	0.2585	1.8518	0.2471
	No	56	3.3513	0.4183	2.6460	0.4238	1.8434	0.4178

NS – Not Significant

Table 4.12 Lymphocyte transformation response to BCG inoculation in Large White Yorkshire and Desi pigs

Breed	Sex	n	Stimulation index											
			Pre-inoculation			15 days post-inoculation			30 days post-inoculation			45 days post-inoculation		
			Range	Mean	SE	Range	Mean	SE	Range	Mean	SE	Range	Mean	SE
LWY	Female	40	1.000 to 1.583	1.100	0.019	3.821 to 8.278	5.984	0.2101	3.501 to 8.319	6.086	0.1240	3.281 to 7.912	5.9755	0.130
	Male	35	1.000 to 1.783	1.049	0.021	3.987 to 8.507	6.0535	0.2381	3.259 to 8.631	6.1301	0.1380	3.157 to 8.018	6.0335	0.127
	Overall	75	1.000 to 1.703	1.076	0.012	3.821 to 8.507	6.0161	0.1131	3.259 to 8.631	6.1070	0.0910	3.152 to 8.018	6.0020	0.102
Desi	Female	41	1.000 to 1.489	1.097	0.018	4.021 to 8.437	6.2853	0.1892	3.981 to 8.276	6.5360	0.1890	3.945 to 8.371	5.9165	0.152
	Male	34	0.908 to 1.5981	1.090	0.022	3.875 to 8.531	6.3945	0.2017	3.795 to 8.914	6.6605	0.2011	4.083 to 8.445	6.0785	0.189
	Overall	75	0.908 to 1.598	1.094	0.013	3.875 to 8.531	6.3340	0.1541	3.795 to 8.491	6.5920	0.1340	3.945 to 8.445	5.9890	0.109
Overall		150	0.908 to 1.783	1.085	0.010	3.821 to 8.531	6.1751	0.0982	3.259 to 8.631	6.3040	0.0870	3.157 to 8.445	5.9950	0.093

Fig. 4.7 Lymphocyte transformation response to BCG inoculation in Large White Yorkshire and Desi piglets



1.5981 respectively. The mean 15 day post inoculation stimulation index for females and males was 6.2853±01892 and 6.3945±0.2017 with a range of 4.021 to 8.437 and 3.875 to 8.531 respectively. The mean 30 day post inoculation stimulation index in females and males were 6.5360±0.1890 and 6.605±0.2011 with a range of 3.981 to 8.276 and 3.795 to 8.914 and for 45 day ranged from 3.945 to 8.371 and 4.083 to 8.45 with a mean of 5.9165±0.152 and 6.0785±0.189 respectively.

The cumulative overall pre inoculation stimulation index of the 150 animals tested from both the breeds ranged from 0.908 to 1.783 with a mean of 1.085±0.010. The 15 day post inoculation stimulation index ranged from 3.821 to 8.531 with a mean of 6.1751±0.0982 and the 30 day post inoculation stimulation index ranged from 3.259 to 8.631 with a mean of 6.3040±0.0870. The 45 day post inoculation stimulation index ranged from 3.157 to 8.445 with a mean of 5.9950±0.093.

4.3.2 Effect of breed, sex and body weight classes

The least squares analysis of variance for breed, sex and body weight at weaning on the lymphocyte stimulation index to intradermal BCG inoculation is given in Table 4.13

The effect of breed on pre-inoculation as well as the 15, 30 and 45 day post inoculation stimulation index was not significant ($P>0.05$). Similarly the effect of sex on the pre-inoculation and post-inoculation stimulation index on day 15. 30 and 45 were also not significant ($P>0.05$). The different weaning body weight did not influence the pre inoculation and post inoculation stimulation index on days 15, 30 and 45 significantly

(P>0.05). The least squares mean for the effect of breed and sires within breed on the lymphocyte stimulation index to intradermal inoculation of BCG are presented in Table 4.14.

The Large White Yorkshire and Desi pigs had a least squares mean of 1.0531±0.0893 and 1.0683±0.0783 as preinoculation stimulation index and was not significantly different (P>0.05). The 15, 30 and 45 days post-inoculation stimulation index for Large White Yorkshire and Desi pigs were not significant (P=0.0861,0.1028 and 0.0983). The Large White Yorkshire had a least squares mean of 5.9381±0.1133, 6.0231±0.1896 and 6.1078±0.2335 for 15,30 and 45 day post immunisation stimulation index where as the Desi pigs had a least squares mean of 6.3735±0.1593, 6.4381±0.1785 and 6.0021±0.1853 respectively.

The least squares mean on the effect of sex and body weight class on the lymphocyte stimulation index to intra dermal BCG inoculation is given in Table 4.14

The overall least squares mean values for female and male were 1.0896±0.1785 and 1.0785±0.1995 (P=0.5928) as pre-inoculation stimulation index, 6.0897±0.2187 and 6.1255±0.2475 (P=0.8948) as 15 day post inoculation stimulation index, 6.3018±0.0919 and 6.3585±0.1248 (P>0.05) as 30 day post-inoculation stimulation index and 5.9188±0.0781 and 6.1255±0.1197 (p=0.1135) as 45 day post-inoculation stimulation index. The weaning body weight classes did not influence significantly the pre and post inoculation stimulation index. The least squares mean for different body weight classes are presented in Table 4.14.

4.3.3 Effect of sire within breed and heritability

Least squares analyses of variances for the effect of sires within breeds on the lymphocyte stimulation index to intradermal inoculation of BCG are presented in Table 4.13.

The effect of sire within breed on pre-inoculation stimulation index was not significant for sires within Large White Yorkshire and Desi pigs (P>0.05). The 15, 30 and 45 day post-inoculation stimulation index for sire within large white Yorkshire were highly significant (P=0.0007, 0.0003 and 0.0.0000) whereas the 15 day post inoculation stimulation index within sires of desi pigs was significant (P=0.0037) and the 30 and 45 day post-inoculation stimulation index were highly significant (P= 0.0085 and 0.0000). The effect of sire on pre-inoculation stimulation index of Large White Yorkshire and Desi was not significant (P>0.05), but was highly significant (P=0.0007) for 15 days post-inoculation stimulation index for sires within Large White Yorkshire and was significant (P=0.0337) for sires within Desi pigs. The post inoculation stimulation index for 30 and 45 day was highly significant for sires within Large White Yorkshire (P=0.0003 and 0.0000) and for Desi sires (P=0.0085 and 0.0000). The least squares mean for pre and post-inoculation stimulation index for 15,30 and 45 days for each sire within Large White Yorkshire and Desi breeds are presented in Table 4.14 and Fig.4.8 and 4.9 respectively.

The estimate of heritability for lymphocyte transformation response are presented in Table 4.17. High heritability of 0.5171±0.2893, 0.6289±0.3817 and 0.4983±0.2583

Table 4.13 Least squares analysis of variance for the effect of breed, sex, weaning body weight and sire within breed on the LT test to BCG inoculation

Source of variable	Df	Pre-inoculation		15th days post-inoculation		30th day post-inoculation		45th day post-inoculation	
		MSS	Probability	MSS	Probability	MSS	Probability	MSS	Probability
Breed	1	0.0826 NS	0.9573	1.9201NS	0.0861	1.0833 NS	0.1028	1.7389 NS	0.0983
Sex	1	0.1782 NS	0.5928	1.8734 NS	0.0893	1.8311NS	0.1675	1.9385 NS	0.0768
Weaning body weight	4	0.0962 NS	0.8913	0.9885 NS	0.7875	1.1257 NS	0.6834	1.0834 NS	0.8365
Sires within Large White Yorkshire	7	0.0100 NS	0.9332	2.8434 **	0.0007	2.1256 **	0.0013	4.1235 **	0.0000
Sires within Desi	7	0.0050 NS	0.9738	3.5568**	0.0037	2.0855 **	0.0085	4.2435 **	0.0000
Error	129	1.2934		0.6755		0.4568		0.4719	

NS – Not Significant

** - Significant at 1% level

Table 4.14 Least squares mean for the effect of breed, sires within breed, sex and weaning body weight class on LT response to BCG inoculation

Breed	n	Pre-inoculation		15th da post-inoculation		30th day post-inoculation		45th day inoculation	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
		(P=0.9573)NS		(P=0.0861)NS		(P=0.1028)NS		(P=0.0983)NS	
Large White Yorkshire	75	1.0531	0.0893	5.9381	0.1133	6.0231	0.1896	6.1078	0.2335
Desi	75	1.0683	0.0783	6.3735	0.1593	6.4381	0.1785	6.0021	0.1853
Sires within Large white Yorkshire		(P=0.9332)NS		(P=0.0007)**		(P=0.0013)**		(P=0.0000)**	
1	7	1.0391	0.0598	7.2458	0.3194	6.6158	0.2789	6.8045	0.2698
2	11	1.1358	0.0494	6.0000	0.2593	6.3059	0.2075	5.7208	0.2218
3	8	1.0908	0.0585	5.9987	0.2978	6.5818	0.2588	6.5328	0.2518
4	11	1.0648	0.0498	6.2855	0.2489	6.3818	0.2195	6.8509	0.2253
5	11	1.0639	0.0458	5.7755	0.2693	5.8911	0.2295	5.5403	0.2185
6	9	1.0508	0.0597	5.3198	0.2831	5.2733	0.2256	4.9813	0.2319
7	9	1.0998	0.0585	5.4659	0.2885	5.5583	0.2310	5.6218	0.2408
8	9	1.0578	0.0691	6.3098	0.2983	6.3529	0.2418	6.1829	0.2491

Table 4.14 contd............

Sires within Desi		(P=0.9738)NS		(P=0.0037)*		(P=0.0085)**		(P=0.0000)**	
1	**9**	1.1295	0.0318	6.9815	0.4183	7.5728	0.3598	6.9985	0.2248
2	**9**	1.1038	0.0328	6.8355	0.4175	6.9618	0.3598	5.4961	0.2257
3	**13**	1.0738	0.0308	5.9779	0.3481	6.2988	0.2975	6.4385	0.1879
4	**10**	1.0985	0.0315	5.1938	0.3958	6.7138	0.3385	6.4568	0.2189
5	**7**	1.0791	0.0451	6.6618	0.4748	6.6458	0.4534	5.3475	0.2589
6	**9**	1.0638	0.0395	6.7483	0.4184	6.8385	0.3548	4.9815	0.2398
7	**10**	1.1158	0.0328	6.6128	0.3943	6.8195	0.3355	5.6518	0.2198
8	**18**	1.0971	0.0445	5.9555	0.4458	7.0460	0.3798	6.2159	0.2355
Sex		**(P=0.5928)NS**		**(P=0.8948)NS**		**(P=0.9568)NS**		**(P=0.1135)NS**	
Female	**81**	1.0896	0.1785	6.2155	0.2187	6.3585	0.0919	6.1255	0.0781
Male	**69**	1.0785	0.1995	6.0897	0.2475	6.3018	0.1248	5.9188	0.1197
Weaning body weight class		**(P=0.4469)NS**		**(P=0.5412)NS**		**(P=0.7138)NS**		**(P=0.3548)NS**	
1	**33**	1.1083	0.1985	6.5783	0.2585	6.3595	0.2355	6.1598	0.2385
2	**44**	1.0952	0.1755	6.0835	0.1995	6.4708	0.2025	5.9837	0.2055
3	**46**	1.0758	0.11885	6.2055	0.1938	6.3125	0.2005	6.0175	0.1995
4	**23**	1.0298	0.2085	5.8915	0.2745	6.3895	0.2697	5.8148	0.2375
5	**4**	1.0699	0.3899	6.0395	0.5983	6.2995	0.5785	5.9375	0.4988

NS – Not Significant ** - Significant at 1% level

were estimated for lymphocyte transformation at fifteenth, thirtieth and forty fifth day post injection

4.3.4 Effect of litter traits

The least squares means for the effect of litter size at birth and litter size at weaning on the lymphocyte stimulation index to intra dermal BCG inoculation are given in Table 4.15. The litter size at birth as well as the litter size at weaning did not significantly influence the pre and post-inoculation stimulation index.

4.3.5 Effect of diseases and preweaning mortality

Least squares mean for the effect of lymphocyte stimulation index to intradermal inoculation of BCG on the occurrence of diarrhoea, pneumonia and pre weaning mortality among piglets is given in Table 4.16. The diseases and pre weaning mortality did not significantly influence the post inoculation stimulation index.

4.4 Association between immune response traits

Table 4.18 details the association between immune response traits analysed. Correlations between antibody responses at seven and fifteen days (0.897) and at seven and twenty-first day (0.7193) were highly significant($P < 0.01$). Correlation between antibody responses at fifteen and twenty-first day (0.875) were highly significant ($P < 0.01$). Association between PHA-M response at twenty-four hours and responses at forty-eight and seventy-two hours were highly significant ($P < 0.01$). PHA-M response at twenty-four hours had a highly significant ($P< 0.01$) correlation with lymphocyte transformation responses at fifteenth day and a significant ($P< 0.05$) association at

Table 4.15 Least squares mean for the effect of LT response to BCG inoculation on the litter size at birth and at weaning of Large White Yorkshire and Desi pigs

Breed	n	Pre-inoculation		15th day post-inoculation		30th day post-inoculation		45th day inoculation	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE
Litter size at birth		(P=0.8728)NS		(P=0.6175)NS		(P=0.5985)NS		(P=0.4995)NS	
3	2	1.0659	0.0417	6.3955	0.8995	6.6175	0.9175	5.9835	0.8593
4	3	1.1305	0.0408	6.1385	0.6875	6.3895	0.6579	6.2855	0.6595
5	9	1.0585	0.0328	6.2855	0.4185	6.5133	0.4275	6.1173	0.4098
6	7	1.0713	0.0455	5.9785	0.4538	6.3781	0.4615	6.0835	0.4575
7	14	1.0608	0.0315	6.2125	0.3315	6.1985	0.3185	5.8975	0.3095
8	19	1.0801	0.0385	6.0875	0.3095	6.2105	0.3118	6.1738	0.2978
9	44	1.0615	0.0185	6.1525	0.1975	6.2285	0.2045	6.0569	0.2013
10	22	1.0585	0.0285	6.1730	0.2875	6.1035	0.2797	5.9985	0.2578
11	15	1.0938	0.0305	6.2015	0.3285	6.2885	0.3198	6.1025	03045
12	12	1.0785	0.0315	6.1895	0.3815	6.3175	0.3985	6.0135	0.3675
13	3	1.0895	0.0415	5.9875	0.5998	5.8793	0.5589	6.1537	0.5895

Table 4.15 contd………

Litter size at weaning		(P=0.7975)NS		(P=0.3175)NS		(P=0.2573)NS		(P=0.2815)NS	
2	**2**	1.0598	0.0408	5.8975	0.8135	6.0598	0.8350	5.8391	0.7938
3	**5**	1.0895	0.0395	6.1585	0.4695	6.2283	0.4837	6.0831	0.4598
4	**11**	1.0781	0.0325	6.1085	0.3785	6.1985	0.3685	6.0591	0.3598
5	**15**	1.0975	0.0299	6.0985	0.3025	6.1839	0.3163	5.9893	0.3095
6	**13**	1.0688	0.0308	6.2017	0.3215	6.3035	0.3328	6.1783	0.3175
7	**22**	1.0915	0.2788	6.1585	0.2389	6.2473	0.2418	6.0875	0.2357
8	**27**	1.0598	0.0217	6.1875	0.2153	6.2275	0.2235	6.0550	0.2075
9	**32**	1.0598	0.0195	6.1289	0.1938	6.2195	0.2043	6.1275	0.1986
10	**10**	1.0683	0.0335	6.2108	0.3875	6.3118	0.3988	6.1785	0.3597
11	**8**	1.0675	0.0347	6.1995	0.4065	6.1983	0.0413	6.0874	0.4095
12	**5**	1.0795	0.0418	6.2573	0.4593	6.2598	0.4585	6.1597	0.4497

NS – Not Significant

Table 4.16 Least squares mean for the LT response (stimulation index) to BCG immunisation on the incidence of diarrahoea, pneumonia and pre-weaning mortality

Index variable	n	15th day		30th day		45th day	
		Mean	SE	Mean	SE	Mean	SE
Diarrahoea		(P=0.1926)NS		(P=0.1031)NS		(P=0.1875)NS	
Yes	69	6.0538	0.0459	6.1375	0.0628	5.9738	0.0395
No	81	6.2428	0.0296	6.3099	0.0378	6.1240	0.0217
Pneumonia		(P=0.5285)NS		(P=0.6075)NS		(P=0.7378)NS	
Yes	11	6.1259	0.3739	6.2103	0.3138	5.9875	0.2873
No	139	6.1581	0.0068	6.2322	0.0035	6.0673	0.0087
Pre-weaning mortality		(P=0.2078)NS		(P=0.1897)NS		(P=0.2175)NS	
Yes	94	6.0985	0.0995	6.1835	0.0828	6.0073	0.0679
No	56	6.2519	0.1181	6.3096	0.1378	6.1349	0.1178

NS – Not Significant

Fig . 4.8 LT response to BCG inoculation - sires within Large White Yorkshire

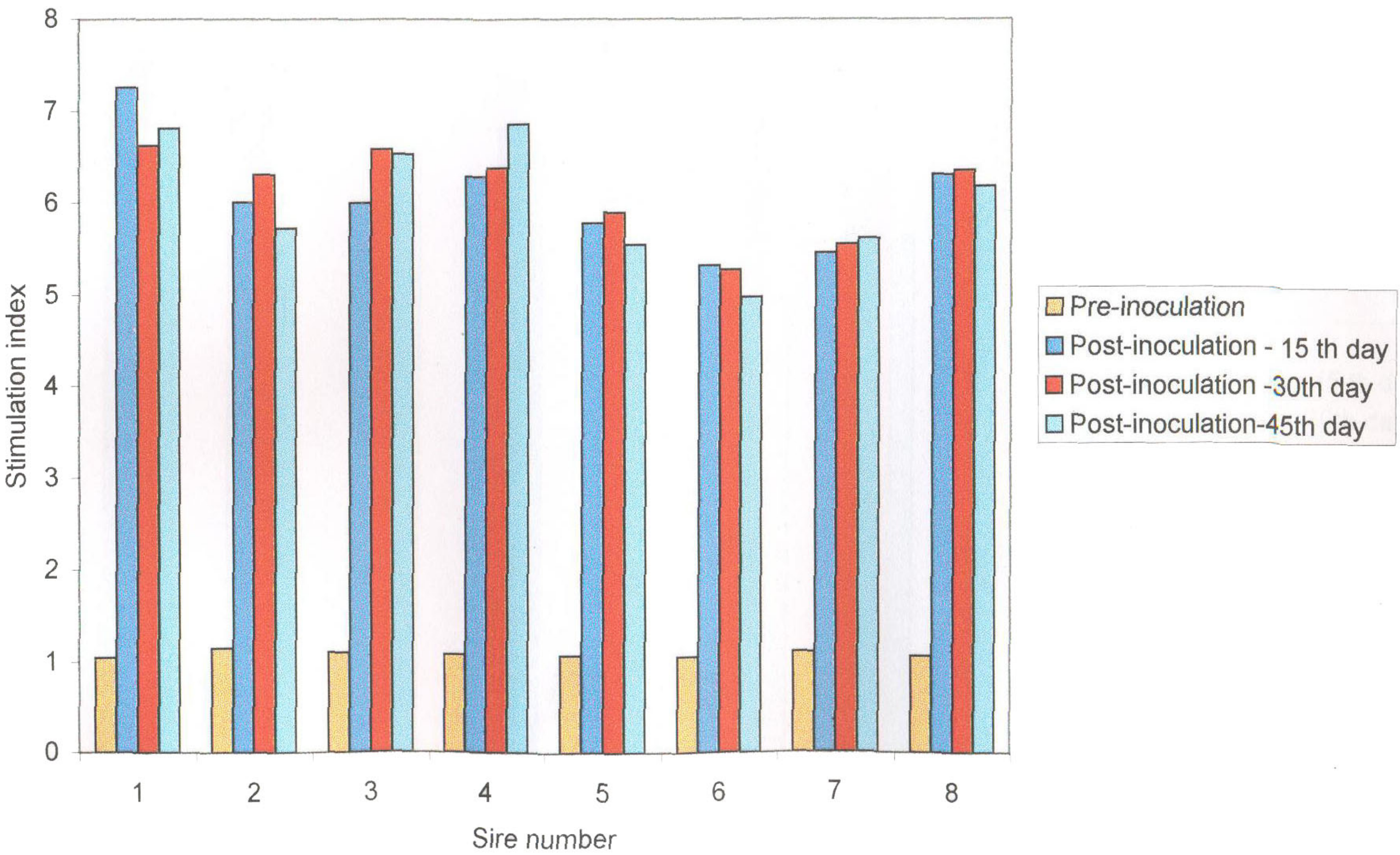


Fig . 4.9 LT response to BCG inoculation - sires within Desi

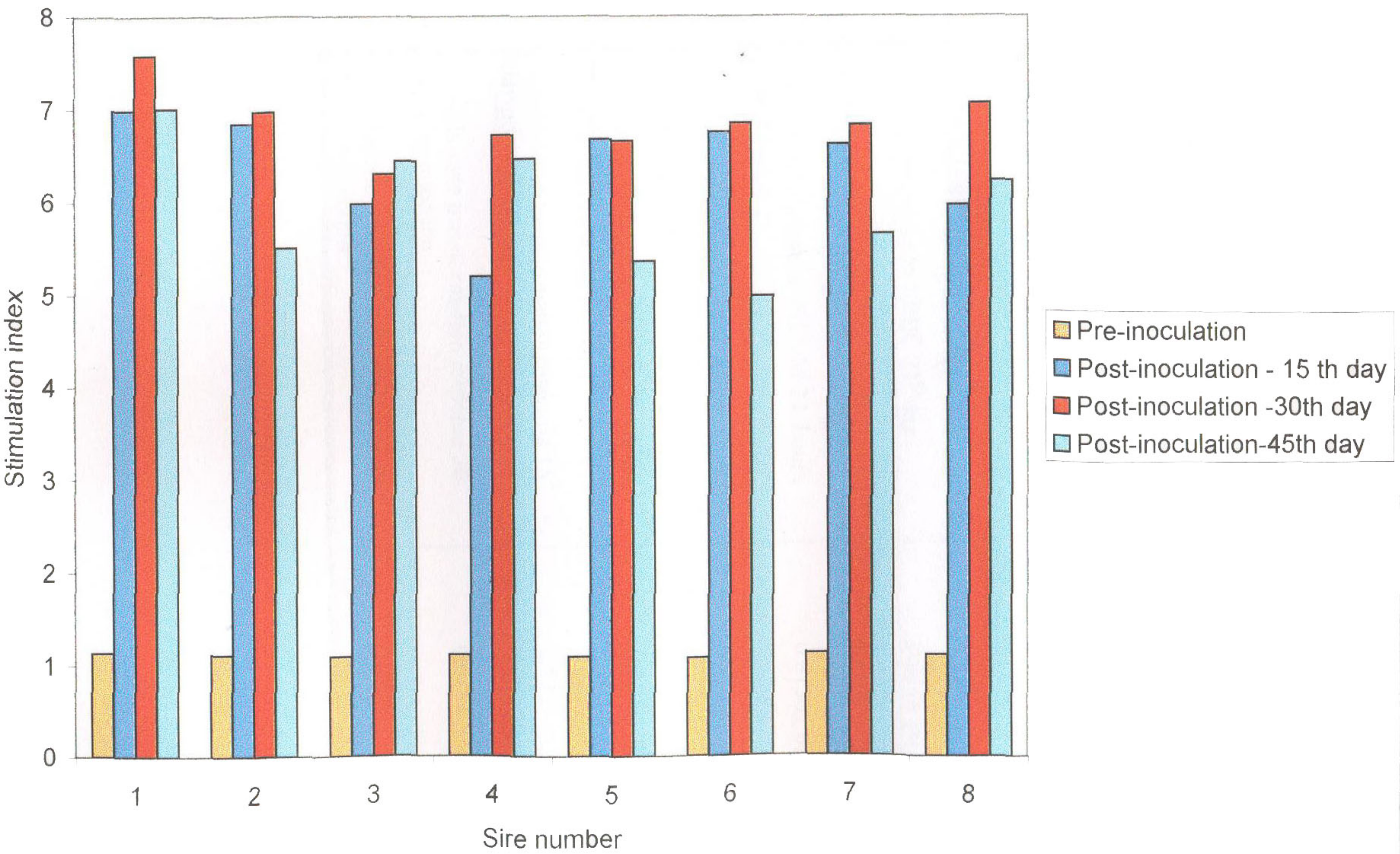


Table 4.17 Heritability estimates of immune response traits

Trait	Heritability
Antibody response to SRBC 7th day	0.8969 ± 0.4238
Antibody response to SRBC 15th day	0.9187 ±0.4893
Antibody response to SRBC 21th day	0.8174 ±0.4387
Skin thickness	0.5173 ± 0.4179
Cutaneous response to PHA-M 24 hours post-injection	0.8136± 0.5843
Cutaneous response to PHA-M 48 hours post-injection	0.6816 ±0.5187
Cutaneous response to PHA-M 72 hours post-injection	0.7134± 0.5283
Lymphocyte transformation response 15th day post-injection	0.5171 ±0.2893
Lymphocyte transformation response 30th day post-injection	0.6289 ± 0.3817
Lymphocyte transformation response 45th day post-injection	0.4983 ± 0.2583

Table 4.19 Correlations between immune responsive traits with weaning body weight, litter weight at birth, litter weight at weaning and pre weaning mortality

	Weaning body weight	Litter weight at birth	Litter weight at weaning	Pre-weaning mortality
Ab.T. on 7th day	-0.307*(P=0.028)	0.091	-0.0113	-0.028
Ab.T. on 15th day	-0.291*(P=0.028)	0.081	0.098	-0.021
Ab.T. on 21th day	-0.301*(P=0.038)	0.069	0.063	-0.081
Cutaneous response to PHA M at 24 hours	0.081	0.059	-0.121	0.048
Cutaneous response to PHA M at 48 hours	0.059	-0.131	-0.112	0.098
Cutaneous response to PHA M at 72 hours	0.078	-0.128	-0.131	0.128
Lymphocyte transformation response at 15th day	0.039	-0.178	-0.081	-0.237*(P=0.035)
Lymphocyte transformation response at 30th day	0.051	0.094	0.128	-0.181
Lymphocyte transformation response at 45th day	0.063	0.15	0.110	-0.113

Ab. T - Antibody titre (1+ log_e)

thirtieth day. Lymphocyte transformation at fifteenth day had a highly significant association ($P < 0.01$) with thirtieth and forty-fifth day transformation responses. Similarly thirtieth and forty-fifth day transformation responses had a highly significant ($P < 0.01$) positive correlations.

4.5 Association between immune responses, litter traits and pre-weaning mortality

Associations between immune response traits, litter traits and pre weaning mortality are presented in Table 4.19. Antibody responses at seven, fifteen and twenty-first day had a significant high negative influence on body weight at weaning ($P<0.05$). The correlations between antibody titres and weaning body weight were always negative and significant. Lymphocyte transformation responses at fifteenth day decreased the pre-weaning mortality significantly ($P < 0.05$).

DISCUSSION

5. DISCUSSION

Genetic variation in immune responsiveness has been documented both in laboratory and domestic animals. Resistance to infectious diseases might thus be improved by taking immune responsiveness as a marker trait in animal breeding. Studies have revealed that immune response traits in pigs reflecting antibody and cell mediated immunity (Buschman *et al.*, 1985; Edfors-Lilja *et al.*, 1985) and mononuclear proliferative response induced by mitogens (Kristenin *et al.*, 1982) are potential tools in the evolution of disease resistant pigs. Vastly different performance testing and selection procedures have resulted in substantially different genetic populations of pigs. To understand the biological changes made as a result of selection, genetic evaluation must be directed towards quantifying the underlying biological traits. By this procedure the proportion of the total variation in a population that can be attributed to the variation in genetic factors can be critically assessed and it would be possible to predict the usefulness of certain traits as a genetic marker. In an effort to evaluate and quantify the biological traits, the genetics of three immune response traits along with their association with litter traits and fitness traits is critically discussed.

Weaning bodyweight, averaged 9.501 kg in large white Yorkshire and 6.063 kg in Desi pigs. The litter size at birth, litter size at weaning, pre weaning mortality percentage litter weight at birth in kilograms and litter weight at weaning in kilograms were 8.453, 6.107, 27.38, 15.554 and 58.019 respectively for Large White Yorkshire and 8.907, 7.520, 16.663, 6.710 and 45.627 respectively for Desi pigs.

The Large White Yorkshire had higher birth weight, litter weight at birth and increased body weight and litter weight at weaning whereas Desi pigs are in an

advantageous position with respect to litter size at birth and weaning with comparatively lesser pre weaning mortality. But the above body weights and other litter traits with respect to Large White Yorkshire was below the performance reported under temperate condition. Keller *et al.*, 1983; Leitner *et al.*, 1992 reported that any type of stress, especially thermal stress leads to immunosuppression, high endemicity of diseases and sub optimal performance.

5.1. Humoral immune response

5.1.1 Antibody response to sheep red blood cell (SRBC)

On seventh day after primary immunisation the overall antibody titre (1+ $\log_e$) in Large White Yorkshire pigs increased to a mean value of 4.445. The females had a titre of 4.864 and males had a titre of 4.448. In the case of Desi pigs the overall antibody titre was 5.216. The mean antibody titre in the case of Desi female and male pigs were 5.419 and 4.971 respectively. The overall seventh day antibody response in the 150 animals tested had a mean of 4.830.

The fifteenth day overall post immunisation antibody response in Large White Yorkshire had a mean of 4.398. The mean antibody response in females and males were 4.433 and 4.256 respectively. In the case of Desi pigs, the female, male and the mean overall antibody response were 4.688, 4.565 and 4.596 respectively. The cumulative overall antibody response to SRBC in both the breeds had a mean titre of 4.482.

The mean twenty first day overall antibody response in Large White Yorkshire pigs was 3.919. The antibody titres in females and males were 3.971 and 3.861 respectively. Desi pigs had an overall mean titre of 4.410 where as females and males had 4.284 and 4.284 respectively. The cumulative overall antibody response to SRBC in both the breeds had a mean titre of 4.127.

Naturally occuring antibodies to SRBC could not be detected in both the breeds as evidenced by the zero titre in preimmunized sera. The high antibody titre ($1+\log_e$) was observed at seventh day post immunisation. The titre declined gradually during fifteenth and twenty first day.

The results of this study are in close conformity with the research works conducted in different parts of the world. Hyldgard – Jensen (1979) observed that peak antibody titres to bovine and human albumin were obtained two to three weeks after primary immunisation in pigs and the primary antibody response was influenced by the adjuvant and dose of antigen. Vander Zijpp and Leenstra (1983) found that mean total antibody titer to SRBC was highest on seventh day after primary immunisation. Ubosi *et al.* (1985) reported that following fourth day of primary injection of chicken with SRBC, differences in response could be noticed. Peak value was reached at six days in all populations following primary immunisation with SRBC. The magnitude of antibody titre ($1+\log_e$) closely agrees with that obtained in calves against human red blood cell (Burton *et al.*, 1989a). Pinard *et al.* (1992) observed a peak antibody response ($1+\log_e$) of 4.73 in chicken against SRBC which is in close agreement with the result obtained.

Buschman (1986) found significant differences among different breeds of swine to humoral immune responses to different antigens.

According to Burton *et al.* (1989a), peak primary antibody responses were observed in calves by day seven to fourteen following HRBC inoculation. Miller *et al.* (1991) reported that peak antibody titer to SRBC occurred in chicken on day six or seven following primary immunisation.

Mounton *et al.* (1988) reported that mean antibody titer to SRBC was 6.6 $\log_2$ in mice resistant to *Salmonella typhimurium* while it was 8.1 $\log_2$ in susceptible ones.

Dunnington *et al.* (1992) evolved high and low antibody producing lines to SRBC in White Leghorns and White Plymouth Rock. Antibody response to Newcastle disease virus was consistently higher in high responding lines.

Pinard *et al.* (1992) reported a selection experiment which generated high and low response lines to SRBC in chicken. The mean post primary antibody titre was 4.73 $\log_2$ in the base population. After nine generations of selection the antibody titer was 10.62 $\log_2$ in high response group and 1.94 $\log_2$ in low responder lines.

The peak antibody response observed at the seventh day post immunisation concurring with the research results in avian and bovine (Vander Zijpp and Leenstra, 1983; Burton *et al.,* 1989b). It would be pertinent to note that all the experimental animals had no naturally occuring antibodies to SRBC at zero day of immunisation, evidenced by zero preimmunisation titre. This is in contrast to lapine immune response models wherein Frossman antibody to SRBC has been well documented (Aitken, 1964; Nandakumar 1995). The antibody titre rose to the peak at seventh day of immunisation and later on showed a decreasing trend from second week onwards. The trend in antibody response in pigs is in full agreement with the observations made by Burton *et al.* (1989a)

5.1.2 Effect of breed, sex and body weight class

The effect of breed and sex within breed was not found to be significant on the antibody response to SRBC on seventh, fifteenth and twenty-first day of immunisation.

The overall least squares mean value for female and male were 4.7385 and 4.5893 for seventh day, 4.5784 and 4.4456 for the fifteenth day and 4.2964 and 4.1789 for the twenty-first day of post-immunisation.

Nguyen (1983) investigated the effect of sex on the immune response to chicken red blood cells in sheep. He found that mean haemagglutinin titers of young females were higher than that of young males, though the differences between groups were not significant. No sex related differences were found in antibody response to sheep red blood cells, *Brucella abortus* or rabbit erythrocytes in chicken (McCorkle and Glick, 1980 and Vander Zijpp *et al.*, 1986). Leitner *et al.* (1989) reported that response to SRBC in chicken is significantly influenced by sex with a female superiority contributing to the increased survival of female birds.

Ubosi *et al.* (1985) found significant population differences in response of chicken to SRBC. The peak response titer to SRBC occurred at the same time in all the lines and populations. Buschman (1986) found significant differences among different breeds of swine to humoral immune responses to different antigens. Muggli *et al.* (1987) reported no breed difference among Angus, Hereford and Red Poll calves to *Infections Bovine Rhinotracheitis* virus (IBR), 60 days post vaccination.

Buschman and Meyer (1990) reported that immune response of swine to tetanus toxoid was significantly influenced by breed.

Rothschild *et al.* (1984a) found significant breed differences in antibody responsiveness of pigs to inactivated *B. bronchiseptica.* The response was also influenced by dam.

Okabayashi *et al.* (1987) pointed out that line differences in antibody responses to SRBC were polygenic in poultry. There were considerable variation between and within populations of poultry (Peleg *et al.*, 1985). Dunnington *et al.* (1992) evolved high and low antibody producing lines to SRBC in White Leghorns and White Plymouth Rock. Antibody response to Newcastle disease virus was consistently higher in high responding lines.

The present study did not reveal any significant difference between breeds in antibody response to SRBC and the finding fully concur with the observations of Muggli *et al.*(1987). The absence of significant breed difference in antibody response can be considered as a species specific phenomena or a result specific to this study. It can be elucidated that the humid tropical stress might have contributed to an immunosuppressant effect so that final resolutions to the immune response capacity characteristic to this native as well as exotic breeds could not be fully expressed.

The body weight at weaning had no significant effect on antibody response to SRBC on the seventh, fifteenth and twenty-first day post immunisation. The Large White Yorkshire and Desi pigs had a least squares mean of 4.4058 and 5.0567 on the seventh day, 4.3817 and 4.6164 on the fifteenth day and 3.9485 and 4.4539 on the twenty-first day respectively.

In beef cattle, Muggli *et al.* (1987) reported that there were no significant correlations between immune response traits and growth traits.

In pigs earlier studies of Huang (1977) found that there was no association between immune response and early growth.

Leitner *et al.* (1992) observed no significant association between humoral immune response to *E.coli* at 10 days of age and growth traits. Kean *et al.* (1994a) reported that there were no significant association between immune response traits, juvenile and adult body weights, age at first egg, 32 weeks egg weight and rate of egg production in chicken.

It is generally believed that fast growing poultry strains are genetically less resistant to disease. Han and Smyth (1972) observed that selection for increased growth rate in broilers resulted in an increased susceptibility to Marek's disease.

Seigel and Gross (1980) demonstrated that a line selected for low antibody response to SRBC had significantly higher four weeks body weight than either unselected control or high antibody response line. Vander Zijpp (1983) reported that the correlation between live weight and heamagglutinin antibody titres to SRBC indicated a negative genetic relationship.

In the present study no significant effect of weaning body weight classes, sex and breed of pigs to immune response to SRBC could be observed. The effect of breed, though not significant, approached near significant levels on the antibody responses. This result is in contradiction to the results of Han and Smith (1972) and Seigel and Gross (1980) who reported that rapidly growing strains in poultry are less resistant to disease and also selection for increased growth rate resulted in increased disease susceptibility. The results of the present study agree with the findings of Muggli *et al.* (1987) who found no correlation between antibody response and growth traits in beef cattle. This could be explained partly due to the species variation and also taking into account the fact that the strain of pigs on which the study was conducted was unselected for enhanced growth. Tropical stresses might have an adverse effect on growth rate and also on the antibody responses interfering with the results of the study.

5.1.3 Effect of sires and heritability

The effect of sires within Large White Yorkshire and Desi breeds was found to be highly significant ($P<0.01$) on the antibody response to SRBC on the seventh day, fifteenth day and also on the twenty-first day. The effect of sires within breed both in Large White Yorkshire and Desi, was highly significant. The heritability estimates were very high with values of 0.8969, 0.9187 and 0.8174 respectively for the seventh, fifteenth and twenty-first day post-immunisation.

In a study involving the humoral response of sheep to chicken RBC, Nguyen (1983) found that antibody titer of sires varied from four log_2 and eight log_2. The regression coefficient of sire was 0.41 indicating that effect of sire was significant on the antibody response. The heritability estimate approached 0.82. Lie *et al.* (1983) reported that in cattle sire families and not sires differed significantly in their antibody response to human serum albumin and synthetic peptides. Vander Zijpp (1983) reported that heritability of immune response to SRBC were 0.26 and 0.14 in White Leghorn and White Plymouth Rock breeds at day seven post-immunisaiton.

Burton *et al.* (1989a) estimated the heritability by paternal half sib correlation analysis and found that the peak titers are more heritable for antibody response in calves to human red blood cell and ovalbumin. They could get a heritability estimate of 0.4 and 0.34 respectively for HRBC and ovalbumin. Leitner *et al.* (1992) found that heritability of immune response to *E.coli* vaccination in low response group was 0.35 and for high response group was 0.21.

Pinard *et al.* (1992) estimated the heritability of fifth day antibody titer to SRBC challenge in chicken as 0.31. The high heritability for antibody responses obtained in this study clearly brings out the underlying genetic variation which can be successfully exploited in the evolution of disease resistant porcine strains. This possibility has also been suggested by Stear *et al.* (2001). This emerging trend in breeding for immune responsiveness and the associated disease resistance is likely to open up potentially rewarding frontiers of porcine genetics aimed at the development of sustainable pig production in tropics.

5.1.4. Effect of litter traits

The least squares means for different litter sizes at birth had no significant on seventh, fifteenth and twenty-first day post immunisation response and the effect of

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litter size at weaning were also non-significant. The least squares mean for different litter size at weaning had probability values of P=0.3987, P=0.5138 and P=0.5891 for seventh, fifteenth and twenty-first day of post-immunisation respectively. Meeker *et al.* (1987) showed that there exists a negative association between humoral immune response traits and litter traits in pigs. Though such a trend could not be observed in this study, possibility of a negative association cannot be ruled out and requires further detailed investigation.

5.1.5 Effect of disease and pre-weaning mortality

The effect of antibody response to SRBC on seventh, fifteenth and twenty-first day after primary immunisation was not found to be significant for the occurrence of diarrhoea, pneumonia and pre-weaning mortality among piglets.

Burton *et al.* (1989a) showed that prevalence diarrhoea was negatively correlated with high primary antibody response against HRBC. Chicken selected for four generations of early high antibody response to *Eschearichia coli* showed greater resistance to challenge with *E. coli* (Piticovski *et al.*, 1989). Larsgaard (1990) noticed improved health status in rats selected for high immune response. Lillehoj (1991) observed that inbred strain of chicken having higher antibody response and T-cell response had reduced susceptibility to *Eimeria tenella* infection. Pinard *et al.* (1992) could demonstrate that mortality rates on challenge with virulent Marek's disease virus was high in low immune response group to SRBC.

Leitner *et al.* (1992) reported that birds with high antibody titer to *E.coli* vaccinations ten day post vaccination had the lowest morbidity and mortality rates when challenged with pathogenic *E.coli.*

The overall incidence of diseases was substantially low possibly due to the emergence of resistant animals under natural selection of humid tropical stress reducing the variability to the occurrence of diseases.

5.2 Cell mediated immune response

5.2.1 Cutaneous response to intradermal injection to phytohaemagglutinin M

The overall pre injection skin thickness in Large White Yorkshire had a mean of 3.508. The cutaneous response to PHA-M 24 hours post injection as evidenced by increase in skin thickness ranged from 1.0 to 4.8 with a mean value of 3.253. The PHA-M response at 48 hours had a mean of 2.623. The increase in skin thickness at 72 hours post injection had a mean of 1.927.

The overall pre injection skin thickness in the case of Desi pigs was 3.012. The cutaneous response to PHA-M 24 hours post injection as evidenced by increase in skin thickness had a mean value of 3.533. The PHA-M response at 48 hours had a mean of 2.762. The increase in skin thickness at 72 hours post injection had a mean of 1.870.

The cumulative overall pre injection skin thickness of the 150 animals tested from both the breeds had a mean of 3.260. The increase in skin thickness 24 hours post-injection had a mean of 3.393. The increase in skin thickness at 48 hours had a mean of 2.693 and at 72 hours had a mean of 1.899.

Kundtson *et al.* (1990) found that IL-2 production and mitogenic response to concanavalin A (Con A) were not always associated, suggesting that more than one gene or more than one mechanism were likely to influence the mitogenic response of T-lymphocytes.

Numerous factors differentiate genetic difference in acquired or innate cell mediated immune responses. The responses for these differences are likely to relate to a variety of genes including those determining cytokines, cytokine receptors or the adhesion proteins. The cellular T-lymphocyte dependant immune response has also been reported to be polygenically regulated.

Stiffel *et al.* (1977) selected mice on the basis of lymphocyte stimulation by using phytohaemagglutinin (PHA) as mitogen. After six generations 3.8 times difference could be observed between high and low responder groups to PHA.

PHA responses being an index of generalised CMI status, duration and intensity of this trait has tremendous potential of being used as a tool in the development of disease resistant pig strains. A strong underlying genetic regulation of PHA responses might be exploited to the evolution of porcine strains with high and low CMI status.

5.2.2 Effect of breed, sex and body weight classes

The effect of breed on pre immunisation skin thickness was significant while the effect of sex on pre immunisation skin thickness within the breed was not significant (P>0.05). The effect of breed on 24, 48 and 72 hours post injection increase in skin thickness was not significant. Similarly the effects of sex within breed for 24,48 and 72 hours post injection increase in skin thickness were also not significant.

The Large White Yorkshire and Desi pigs had a significantly different preinjection skin thickness. The increase in skin thickness at 24, 48 and 72 hours for Large White Yorkshire and Desi pigs were not significant.

The females had a mean pre injection skin thickness of 3.487 and males had a pre injection skin thickness of 3.523. The increase in skin thickness 24 hours post injection in case of female and male are 3.365 and 3.125. The 48 hour mean increase in skin thickness in females and males were 2.672 and 2.568. The 72 hour skin thickness in both sexes were 1.958 and 1.893 respectively.

The mean pre-injection skin thickness in the case of Desi female and male pigs were 2.879 and 3.173 respectively. The mean increase in skin thickness for females and males at 24 hours was 3.575 and 3.483 respectively. The 48 hour mean increase in skin thickness in females and males were 2.816 and 2.698 and the 72 hour skin thickness in both sexes were 1.937 and 1.789 respectively.

Rajan *et al.* (1982) found that increases in skin thickness following DNCB challenge in pigs were 6.07 and 4.71 mm at 24 and 48 hours post-challenge respectively.

The overall least squares mean value for female and male were 3.2165 and 3.3188 as pre injection skin thickness, 3.3828 and 3.2516 as 24 hour post injection increase in skin thickness, 2.6017 and 2.5797 as 48 hour increase in skin thickness and 1.8695 and 1.8287 as 72 hour post-injection increase in skin thickness.

In broiler chicken CMI response to diphtheria toxoid varied between different genetic stocks (Klesius *et al.*, 1977). Significant breed difference were seen in CMI response of chicken to PHA test (Vander Zijpp, 1983 and Lamont and Smith, 1984). Breed differences were also reported for CMI responses in pigs (Buschman, 1986). Cheng and Lamont (1988) found significant sub line differences in birds for CMI response as assessed by PHA test. According to Benda *et al.* (1990), significant breed differences were noticed in cell mediated immune response of fowl when assessed by wattle injection with SRBC.

Cheng and Lamont (1988) reported that there existed significant differences between male and female chickens in the T-cell response to PHA-M, with females having a higher CMI over males. Leitner *et al.* (1989) reported that females showed an earlier and greater T-cell response to a purified protein derivative *Mycobacterium avium*.

PHA response was not found to be modulated by the breed and sex of pigs in this study. Generally effect of breed was reported to be significant on the delayed type hypersensitivity responses among cattle and poultry. Though not significant, the effect of breed approached near significant level on the porcine PHA responses.

The effect of weaning bodyweight classes for pre-immunisation skin thickness was significant. But the post injection increase in skin thickness after 24, 48 and 72 hours were non-significant.

Reports on the association of cell mediated immune responses and production traits and growth rate are scanty. The role of cell mediated immunity especially T-cell immunity have been found to be important in several diseases like coccidiosis which adversely affect growth rate in animals and birds (Rose *et al.*, 1990).

The effect of weaning body weight classes on the PHA responses were not significant possibly due to the overriding and multifactorial influences of climate, diseases, maternal effects, management and plane of nutrition on the weaning body weight.

5.2.3. Effect of sires and heritability

The effect of sire on pre injection skin thickness of Large White Yorkshire and Desi was not significant. But it was highly significant for 24 hours post-injection increase in skin thickness for both the sires within Large White Yorkshire and Desi

pigs. The post-injection increase in skin thickness for 48 and 72 hours were significant between sires within Large White Yorkshire and for Desi sires.

High estimates of heritability of 0.5173, 0.8136, 0.6816 and 0.7134 were observed for initial skin thickness, cutaneous response to PHA-M at twenty four, forty eight and seventy two hours of post injection respectively.

Con A induced proliferation and IL-2 production were used as *in vitro* functional tests for PBMC in I and medium high heritabilities were estimated for both traits (h^2 = 0.38 $\pm$ 0.21 and 0.44 $\pm$ 0.23, respectively). These results agree with the heritabilities earlier estimated for nitrogen induced proliferation in the pig (Romagnani, 1992; Joling *et al.*, 1991 and Joling *et al.,* 1993). Regarding IL-2 production, differences between inbred lines of rats (Lukic, 1987) and chickens (Khudtson and Lamont, 1989 and Khudtson *et al.*, 1990) and between paternal halfsib pigs (Edforts *et al.,* 1991) have been reported.

Lie *et al.* (1983) reported significant differences between sire families in cattle in their CMI responses. Stiffel *et al.* (1977) found that effect of sire on the T-lymphocyte-dependent immune response as significant. They selected mice on the basis of lymphocyte stimulation by phytohaemagglutinin as mitogen. After six generation a 3.8 times greater difference between high and low responder line could be observed. Heritability estimate of T-cell response to PHA was 0.28 ± 0.08. Cheng and Lamont (1988) found that haplotype differences were significant on phagocytic index and T-cell response to PHA and sire family differences were significant on T-cell response. Cheng *et al.* (1991) reported that heritability of T-cell response to PHA measured by wing web assay was only 0.06 to 0.07 in base population, while the combined data for first generation for this trait gave heritability of 0.12 to 0.14. Kean

et al. (1994b) reported that heritability of cell mediated immune responses to PHA was 0.15 in chicken.

The significant effect of sire on PHA responses and the high heritability estimates demonstrate the strong genetic influence on these traits which can successfully be used to develop lines/ strains for high DTH responses. These strains can be used as animal models to study the CMI responses, disease resistance and in understanding the molecular mechanisms underlying the disease susceptibility and resistance.

5.2.4 Effect on litter traits

The effect of litter size at birth was significant for pre immunisation skin thickness whereas it was not significant for the increase in skin thickness at 24, 48 and 72 hour post injection. The effect of litter size at weaning was not significant for pre immunisation skin thickness as well as the 24, 48 and 72 hour post injection increase in skin thickness.

Litter traits, immune responses, disease resistance and viability constituting the fitness profile of an organism are very important components in pig production. The results of the present study on the effect of litter traits on CMI status, though not significant offer promising prospects in resolving the components associated with fitness profile.

5.2.5 Effect on diseases and pre-weaning mortality

The increase in skin thickness at 24, 48 and 72 hour post-injection was not found to be significant for the occurrence of diarrhoea, pneumonia and pre-weaning mortality among piglets.

Burton *et al.* (1989b) could not find any significant effect of CMI as assessed by cutaneous response to DNCB on the incidence of naturally occurring diarrhoea and pneumonia in calves. Rose *et al.* (1990) reported that lymphocyte responses contributed considerably to coccidiosis in chicken.

The results of the present study are in agreement with the reports of Burton *et al.* (1989b). Vander Waaij *et al.* (2000) found the loss of production potential among animals below the threshold for disease resistance while those above the threshold would perform normally. Thus animal production, immune responses and disease resistance can be classified together under sustainability, feasibility and desirability (Stear *et al.*, 2001). Analysis of effect of CMI responses on the disease resistance, viability and pre-weaning mortality assumes significance in the above context.

5.3 Lymphocyte transformation test

5.3.1 Lymphocyte transformation response to BCG inoculation

The overall pre-inoculation stimulation index in Large White Yorkshire had a mean of 1.076. The stimulation index, 15 days post-inoculation had a mean value of 6.0161, 30 days post inoculation had a mean of 6.1070 and 45 days post inoculation had a mean of 6.0020.

The overall pre-inoculation stimulation index in the case of Desi pigs was 1.094. The 15 day post-inoculation stimulation index had a mean value of 6.3340, 30 day post-inoculation had a mean of 6.5920 and 45 day post-inoculation had a mean of 5.9890.

There was an increasing trend in stimulation index from day 15 and peak stimulation index was obtained in day 30 and began to decline thereafter. This agrees with the observations of Muscuplat *et al.* (1975). The high variability of lymphocyte

transformation obtained endorsed the reports of Jensen and Christensen (1981). Increased stimulation of lymphocytes could be demonstrated from 5-7 weeks period after infection with *Mycobacterium avium* in pigs (Pirchard *et al.* 1977; Bergman 1980). The degree of lymphocyte transformation was reported to vary from week to week and the index ranged between 0.7 and 16.6 at 55 days to 0.9 and 8.0 on day 71.

5.3.2 Effect of breed, sex and body weight classes

The females had a mean pre inoculation stimulation index of 1.100 and males had a pre inoculation stimulation index of 1.049. The stimulation index 15 day post inoculation in case of females and males were 5.984 and 6.0535. The 30 day post inoculation stimulation index in females and males were 6.086 and 6.1301. The 45 day post inoculation stimulation index in both sexes were 5.9755 and 6.0335 respectively.

The mean pre-inoculation stimulation indexes in the case of Desi female and male pigs were 1.097 and 1.090 respectively. The mean 15 day post inoculation stimulation index for females and males was 6.2853 and 6.3945 respectively. The mean 30 day post inoculation stimulation index in females and males were 6.5360 and 6.605 and 45 day post inoculation had a mean of 5.9165 and 6.0785 respectively.

The Large White Yorkshire had a least squares means of 5.9381, 6.0231 and 6.1078 for 15,30 and 45 day post immunisation stimulation index where as the Desi pigs had a least squares means of 6.3735, 6.4381 and 6.0021 respectively.

The overall least squares mean values for female and male were 1.0896 and 1.0785 as pre-inoculation stimulation index, 6.0897 and 6.1255 as 15 day post-inoculation stimulation index, 6.3018 and 6.3585 as 30 day post inoculation stimulation index and 5.9188 and 6.1255 as 45 day post inoculation stimulation

index. The weaning body weight classes did not influence significantly the pre and post-inoculation stimulation index.

The effect of breed on pre-inoculation as well as the 15, 30 and 45 day post inoculation stimulation index was not significant. Similarly the effect of sex on the pre inoculation and post-inoculation stimulation index on day 15. 30 and 45 was also not significant. The different weaning body weight did not influence the pre inoculation and post inoculation stimulation index on days 15, 30 and 45.

The effect of breed, sex and body weight class were found to exert no significant effect on the stimulation index. Stimulation index being a measure of CMI responses should have a comparable response to DTH response to PHA M. Breed difference were also reported for CMI responses in pigs (Buschman, 1986). The wide variation in the transformation index among different pigs which can vary substantially under a variety of influences, individual influences and unknown technical problems (Dickson and Alan, 1978) can blur the resolution.

5.3.3 Effect of sires and heritability

The 15, 30 and 45 day post inoculation stimulation indexes for sires within Large White Yorkshire were highly significant whereas the 15 day post inoculation stimulation index within sires of Desi pigs was significant and the 30 and 45 day post inoculation stimulation indexes were highly significant.

High estimate of heritability of 0.5171, 0.6289 and 0.4983 for lymphocyte transformation at fifteenth, thirtieth and forty fifth day post injection were observed.

Stiffel *et al.* (1977) found that effect of sire on the T-lymphocyte dependent immune response as significant. They selected mice on the basis of lymphocyte stimulation by phytohaemagglutinin as mitogen. After six generation a 3.8 times

greater difference between high and low responder line could be observed. Heritability estimate of T-cell response to PHA was 0.28 ± 0.08.

Stimulation index which measures the lymphocyte transformation basically follows the pattern of CMI responses which have been reported to be influenced by sire effects. Con A induced proliferation was reported to have a high heritabilty (Edfors-Lilja, 1991). Con A induced proliferation and IL-2 production were used as *in vitro* functional tests for PBMC in I and medium high heritabilities were estimated for both traits (h^2 = 0.38 ± 0.21 and 0.44 ± 0.23, respectively). These results agree with the heritabilities earlier estimated for nitrogen induced proliferation in the pig (Joling *et al.*, 1991;Romagnani, 1992 and Joling *et al.,* 1993). The results of the present study elucidating a high significant sire effect clearly establishes that genetic influences contribute substantially to the lymphocyte stimulation index.

5.3.4 Effect on litter traits

The litter size at birth as well as the litter size at weaning did not significantly influence the pre and post inoculation stimulation index.

Kean *et al.* (1994b) reported that there were no significant association between immune response traits, juvenile and adult body weights, age at first egg, 32 weeks egg weight and rate of egg production in chicken.

Though the litter traits, comprised of litter size at birth and weaning are important economic traits, no significant association was observed between these traits and cell mediated immunity. The results of present study are in close agreement with the previous reports on the absence of association between CMI status and litter traits (Buschman *et al.*, 1985; Buschman, 1986).

5.3.5 Effect on diseases and pre-weaning mortality

The diseases and pre weaning mortality did not significantly influence the post inoculation stimulation index. Burton *et al.* (1989b) could not find any significant effect of CMI as assessed by cutaneous response to DNCB on the incidence of naturally occurring diarrhoea and pneumonia in calves.

Non-significant effect of lymphocyte stimulation index on the occurrence of diseases and mortality is in the present study agreement with the findings of Burton *et al.*(1989a).

5.4. Association between immune response traits

Correlation between antibody responses at seven and fifteen days (0.897) and at seven and twenty-first day (0.7193) were highly significant. Antibody responses at fifteen and twenty-first day (0.875) were highly significant. Association between PHA-M response at twenty four hours and responses at forty eight and seventy two hours were highly significant. PHA-M response at twenty four hours had a highly significant correlation with lymphocyte transformation responses at fifteenth day and a significant association at thirtieth day. Lymphocyte transformation at fifteenth day had a highly significant association with thirtieth and forty-fifth day transformation responses. Similarly thirtieth and forty-fifth day transformation responses had a highly significant positive correlation.

Biozzi *et al.* (1975) showed that there was no association between immune response to SRBC and T-cell response to PHA in mice. Cheng and Lamont (1988) found that there existed a significant negative correlation between phagocytic index and T-cell response to PHA especially among female chicken. Mounton *et al.* (1988)

reported that vaccination response of high responder lines to SRBC may be as high as 200 times compared to low responders.

Cheng *et al.* (1991) found that the genetic correlation between immune response to *Pasteurella multocida, Mycoplasma gallisepticum* and the T-cell response as measured by PHA test were negative. Similarly the association between phagocytic activity and T-cell response was also negative.

The correlations observed in this study are in agreement with the results of the previous studies. In general there was strong positive correlations between antibody responses while association between antibody and cell mediated immune responses were non significant. It was evident that CMI responses were always positively correlated and highly significant.

5.5 Association between immune response, litter traits and pre weaning mortality

Antibody responses at seven, fifteen and twenty first day had significant high influence on body weight at weaning. The correlations between antibody titres and weaning body weight were always negative and significant.

The correlation between preweaning mortality and lymphocyte transformation responses were always negative and significant. Lindhe and Philipson (1998) have discussed on the existence and consequence of negative correlation between production and fitness traits. Negative correlations between antibody responses and growth rate in poultry have already been documented by Vander Zjipp (1983). The negative association between antibody response and growth rate obtained in the present study closely agrees with the above findings.

The absence of Frossman antibody response to SRBC among swine and the peak response to SRBC at day 7 post-immunisation has been found to be a characteristic of porcine immune response. The highly significant effect of sire on antibody response establishes the strong genetic modulation of antibody response and its feasibility in developing high and low responder lines. The negative association between the weaning body weight and antibody response offers the potential scope of using this traits in developing piglines endowed with fast growth rate. The peak and persistency of CMI responses in swine followed the traditional trend here also. A very strong genetic modulation with a highly significant sire effect could be established. The significant negative association between pre weaning mortality and lymphocyte transformation response at 15th day indicate possibility of this trait being used as a marker to reduce the pre weaning mortality. Generally immune responses had a high heritability indicating the underlying additive variance and the quantitative nature of these traits.

SUMMARY

6. SUMMARY

Antibody response to SRBC, DTH response to intradermal injection of PHA and LT response to BCG were evaluated, in order to analyse the genetics of immune response among Large White Yorkshire and Desi pigs. The association between these immune response traits and production performance of piglets were also analysed.

Naturally occurring antibodies to SRBC were not detected in both the breeds. Peak antibody response to SRBC was obtained at day 7 post immunisation. The effects of breed and sex were not significant on antibody response at 7th day, 15th day and 21st day post immunisation. However, antibody response to SRBC at day 7 approach near significant levels. Body weight classes were not found to be significantly associated with antibody response to SRBC. Sire effect within Large White Yorkshire and Desi breeds were found to influence significantly the antibody response to SRBC on 7th, 15th and 21st day post immunisation. Heritability estimates of antibody response to SRBC were 0.8969 ± 0.4235, 0.9187 ± 0.4893 and 0.8174 ± 0.4893 respectively at 7th day, 15th day and 21st day of immunisation. Litter size at birth and weaning had no significant influence on antibody response. Similarly, antibody response to SRBC among piglets was not influenced by the incidence of diarrhoea, pneumonia and pre weaning mortality to a significant level.

DTH responses to intradermal injection of PHA peaked at 24 hours post injection. The mean pre injection skin thickness was 3.508 mm and 3.012 mm

among Large White Yorkshire and Desi pigs respectively. This difference was found to be significant ($P \leq 0.05$) and was due to the significant breed difference confounding on body weight classes. The effect of breed on PHA responses at 24, 48 and 72 hours was not significant. Sex of the pig also did not influence the PHA responses significantly. The body weight classes did not influence the DTH response to PHA significantly. Sire effect was not significant on the pre injection skin thickness. But the DTH response at 24 hours was influenced by the sires in both Large White Yorkshire and Desi pigs to a highly significant level ($P \leq 0.01$). At 48 and 72 hours post injection also DTH responses were influenced by sires to a significant level ($P<0.05$). The heritability estimates for pre injection skin thickness and DTH responses at 24, 48 and 72 hours were 0.5173 ± 0.4179, 0.8136 ± 0.5643, 0.6816 ± 0.5187 and 0.7134 ± 0.5283 respectively. The litter size at weaning was not influenced by the initial skin thickness. DTH responses to PHA at 24, 48 and 72 hours had no significant influence on the litter size at birth and weaning. PHA responses at 24, 48 and 72 hours were not influenced significantly by the incidence of diarrhoea, pneumonia and pre weaning mortality.

The lymphocyte transformation and stimulation index to BCG on zero day was around one indicating that there was no marked increase in the lymphocyte multiplication in PPD stimulated samples. The stimulation index on 15th day was 6.0161 in Large White Yorkshire and 6.3340 in Desi. This index further increased to 6.1070 and 6.5920 on 30th day and began to decline from 45th day with a mean value 6.0020 in Large White Yorkshire and 5.9890 in Desi pigs. The effects of breed, sex and body weight class of piglets were not found to

influence the stimulation index significantly. Sire effect was not significant on the pre inoculation index while it was highly significant on 15th, 30th and 45th day post inoculation in Large White Yorkshire and Desi. The estimates of heritability on 15th, 30th and 45th day stimulation index were 0.5171 $\pm$ 0.2893, 0.6289 $\pm$ 0.3817 and 0.4983 $\pm$ 0.2583 respectively on 15th, 30th and 45th day post injection. Litter size at birth and weaning was not found to have any significant influence on the LT response to BCG. LT response to BCG not influenced significantly by the incidence of diarrhoea, pneumonia and preweaning mortality.

Antibody response to SRBC at 7, 14 and 21st day had highly significant positive correlations. Similarly the correlation between PHA responses at 24, 48 and 72 hours were also highly significant and positive. PHA responses at 24 hours and LT responses at 15th day was also positive and significant. LT response at 15th and 30th day were also significant and positive. The association between LT responses during different time intervals were always positive and significant. Correlation between initial skin thickness and PHA responses were negative and significant.

Antibody response at 7, 15 and 21st day had a significantly high negative influence on the body weight at weaning. There was a significant decrease in pre-weaning mortality associate with LT response at 15th day.

The results of the present study clearly reveal the underlying genetic influence and associated variation in immune responses. The high heritablility for antibody response, CMI responses namely PHA responses and LT responses point out the possibility of developing porcine lines endowed with high and low

immune responses. This would facilitate analysis of molecualr mechanisms underlying immune responses and its association with economic traits and disease resistance in swine.

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PORCINE IMMUNE RESPONSE AS MARKER TRAITS FOR SELECTIVE BREEDING

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ABSTRACT OF THE THESIS

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ABSTRACT

Survivability and better performance of pigs under tropical stress have been reported to be significantly influenced by immune responses. Immune response traits under genetic control offer potential possibilities for exploited in commercial pig production. The present research project on the utilisation of porcine immune responses by estimating the magnitude of humoral and cell mediated immune responses in Desi and Large White Yorkshire attempted to evaluate the genetics of immune responses and to identify the association between the immune response traits and economic traits. The immune response traits were studied in 150 piglets aged between two to three months, 75 each belonging to Desi and Large White Yorkshire of both sexes and sired by eight sires each. The immune response traits studied were antibody response to sheep red blood cells (SRBC), delayed type hypersensitivity (DTH) to intradermal injection of PHA-M and lymphocyte transformation response to BCG. The economic traits recorded were litter size at birth, litter weight at birth, litter size at weaning, litter weight at weaning, weaning mortality and the occurrence of diarrhoea and pneumonia.

Naturally occurring antibodies to SRBC could not be detected in both the breeds. Peak antibody response to SRBC was obtained at day 7 post immunisation with a mean titre of 4.830. Heritability estimates of antibody response to SRBC were 0.8969 ± 0.4235, 0.9187 ± 0.4893 and 0.8174 ± 0.4893 respectively at 7th day, 15th day and 21st day post-immunisation. Litter size at birth and weaning had no significant influence on antibody response. Similarly, antibody response to SRBC among piglets was not influenced by the incidence of diarrhoea, pneumonia and pre-weaning mortality to a significant level.

DTH responses to intradermal injection of PHA peaked at 24 hours post injection with a mean value of 3.39 mm. The mean pre injection skin thickness was 3.508 mm and 3.012 mm among Large White Yorkshire and Desi pigs respectively. This difference was found to be significant ($P<0.05$) and this difference was due to the significant breed difference confounding with body weight classes. The effect of breed on PHA responses at 24, 48 and 72 hours were not significant. Sex of the pig also did not influence the PHA responses significantly. The body weight classes did not influence the DTH response to PHA significantly. Sire effect was not significant on the pre injection skin thickness. But the DTH response at 24 hours was influenced by the sires in both Large White Yorkshire and Desi pigs to a highly significant level ($P<0.01$). At 48 and 72 hours post injection also DTH responses were influenced by sires to a significant level ($P<0.05$). The heritability estimates for pre injection skin thickness and DTH responses at 24, 48 and 72 hours were 0.5173 ± 0.4179, 0.8136 ± 0.5643, 0.6816 ± 0.5187 and 0.7134 ± 0.5283 respectively. The litter size at weaning was not influenced by the initial skin thickness. DTH responses to PHA at 24, 48 and 72 hours had no significant influence on the litter size at birth and weaning. PHA responses at 24, 48 and 72 hours were not influenced significantly by the incidence of diarrhoea, pneumonia and pre-weaning mortality.

The analysis of lymphocyte transformation and stimulation index to BCG on zero day was around one indicating that there was not marked increase in the lymphocyte multiplication in PPD stimulated samples. The stimulation index on 15th day was 6.0161 in Large White Yorkshire and 6.3340 in Desi. This index further increased to 6.1070 and 6.5920 on 30th day and began to decline from 45th day with a mean value of 6.0020 in

Large White Yorkshire and 5.9890 in Desi pigs. The effect of breed, sex and body weight class of piglets was not found to influence the stimulation index significantly. Sire effect was not significant on the pre inoculation index while it was highly significant on 15th, 30th and 45th day in Large White Yorkshire and Desi. The estimates of heritability on 15th, 30th and 45th day stimulation index were 0.5171 $\pm$ 0.2893, 0.6289 $\pm$ 0.3817 and 0.4983 $\pm$ 0.2583 respectively. Litter size at birth and weaning was not found to have any significant influence on the LT response to BCG.

Correlation analysis among different immune response traits revealed that antibody response to SRBC at 7, 14 and 21st day had highly significant positive correlation. Similarly, the correlation between PHA responses at 24, 48 and 72 hours were also highly significant and positive. PHA responses at 24 hours and LT responses at 15th day was also positive and significant. LT responses at 15th and 30th day were also significant and positive. The association between LT responses during different time intervals were always positive and significant. PHA responses were always negatively correlated with initial skin thicknesss to a significant level.

Antibody response at 7, 15 and 21st day had a significantly high negative influence on the body weight at weaning. There was a significant decrease in pre-weaning mortality associated with LT response at 15th day.