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Thirty-seven Yorkshire sows were bred to one of six boars; 34 of the sows were verified pregnant at 42 d of gestation. These sows were innoculated with ovine serum albumin (OSA) in Freunds' complete adjuvant at 65 d and later given a second injection in Freunds' incomplete adjuvant 93 d. Blood samples were collected from the sows 7 d post-second injection. Blood samples were also collected from their 386 piglets at 12 to 24 h after birth. At 10 wk, blood samples were collected from 175 of the piglets after which they were given the same series of injections at 10 and 14 wk as their dams at 65 and 93 d. At 15 wk, blood samples were collected from 169 of the pigs. Serum was harvested from all blood samples and was analyzed by kinetic enzyme-linked immunosorbant assay techniques to determine the effect of environmental and genetic sources of variation on titers of anti-OSA immunoglobulin G. Age, weight and litter size had no effect on

sow titer (P>.05) Sow age, sow weight, sow titer, litter size and birth weight had significant effects on piglet IgG titers at 12 to 24 h after birth. Significant two-factor interactions for piglet IgG titers at 12 to 24 h after birth were observed between birth weight x litter size, sex x sow age, sex x sow weight and sow titer x birth weight. At 10 wk, sources of variation affecting IgG titers included sow titer, litter size, titers at 12 to 24 h and weight at 10 wk (P<.01). At 15 wk, sow weight, sow age and titers at 12 to 24 h significantly affected immunoglobulin titers. Heritability estimates obtained for 12 to 24 h titers and 15 wk titers were 2.52 $\pm$ 2.37 and .37 $\pm$ .86, respectively.

SOURCES OF INFLUENCE ON PASSIVE AND ACTIVE IMMUNOGLOBULIN G RESPONSES IN SWINE

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Head of Department of Animal Science

Redacted for privacy

Dean of Graduate School

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DEDICATION

To my parents Bertha and Raymond Chitko
in gratitude for their love and support
through my worst as well as my best times.

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CHAPTER 1

INTRODUCTION

Humoral immunity refers to the circulating antibodies present in an individual as a result of exposure to antigens. Two types of humoral immunity are found in animals; active and passive. Active immunity refers to the production of antibodies due to the direct stimulation of the immune system by antigens. Passive immunity is immunity acquired from an individual that has been directly exposed to an antigen and subsequently transfers specific antibodies to a recipient (eg. via transplacental transfer or the colostrum).

Immunoglobulin G is the universal carrier of passive immunity from mother to young (Butler, 1973). Butler (1973) classified animals into three groups depending on how young obtain IgG from their dams; either transplacentally (group I), via colostrum (group III), or both (group II). Swine are placed in group III; because they possess an epitheliochorial placenta, no transplacental transfer of immunoglobulins normally occurs. It is essential to the survival of neonatal piglets that they acquire sufficient antibodies via colostrum from their dams. Transmission of passive immunity declines very rapidly after birth in suck-

ling piglets and effectively ceases at about 24 h (Brambell, 1970). As long as maternally derived, passively acquired antibodies persist in the piglets' circulation, the piglets are incapable of mounting a substantial immune response of their own (Uhr and Moller, 1968; Wilson and Svendsen, 1972).

Disease resistance is associated with the immune response to infectious agents and as such may be due to humoral and/or cellular responses. Individuals with a tendency for depressed antibody production when challenged by a pathogen are generally more susceptible to disease occurrence. Because of this relationship, the immune response may be used as an indicator of relative disease resistance (Crittenden, 1983). It has been shown that animals with a high antibody response to one antigen tend to exhibit high responses to other non-related antigens (Biozzi, 1970). It is therefore possible to estimate the relative ability of individuals to resist disease by exposing them to a non-pathogenic antigen and determining the levels of their corresponding immune responses.

Several researchers have demonstrated genetic variation in the immune response to pathogenic and non-pathogenic antigens. Edfors-Lilja et al. (1984) found pronounced variation within or between litters in antibody titers in piglets naturally reared on sows immunized to two Escherichia coli antigens. Genetic differences in serum-

neutralization titers of pigs after vaccination with pseudorabies modified live-virus vaccine were found by Rothschild et al. (1974). Huang (1977) found variation in the immune response of pigs injected with bovine serum albumin (BSA). Biozzi (1979) produced high and low responder lines of mice by selective breeding for maximal or minimal agglutinin response to sheep erythrocytes. After selective breeding of rabbits immunized with Group C-Carbohydrate, complete segregation was achieved between high response and low response populations (Eichmann et al., 1971). Development of general disease resistance through indirect selection primarily on immune response traits may be presently limited by insufficient understanding of resistance mechanisms (Gavora and Spencer, 1983).

CHAPTER 2

SOURCES OF INFLUENCE ON PASSIVE AND ACTIVE IMMUNOGLOBULIN G RESPONSES IN SWINE[1]

C. G. Chitko[2] and D. C. England[2]

Oregon State University
Corvallis 97331

[1]Tech. Paper No. _____, Oregon State Experiment Station.

[2]Dept. of Animal Science.

Sources of Influence on Passive and Active Immunoglobulin G Responses in Swine

C. G. Chitko and D. C. England

ABSTRACT

Thirty-seven Yorkshire sows were bred to one of six boars; 34 of the sows were verified pregnant at 42 d of gestation. These sows were innoculated with ovine serum albumin (OSA) in Freunds' complete adjuvant at 65 d and later given a second injection in Freunds' incomplete adjuvant at 93 d. Blood samples were collected from the sows 7 d post-second injection. Blood samples were also collected from their 386 piglets at 12 to 24 h after birth. At 10 wk, blood samples were collected from 175 of the piglets after which they were given the same series of injections at 10 and 14 wk as their dams at 65 and 93 d. At 15 wk, blood samples were collected from 169 of the pigs. Serum was harvested from all blood samples and was analyzed by kinetic enzyme-linked immunosorbant assay techniques to determine the effect of environmental and genetic sources of variation on titers of anti-OSA immunoglobulin G. Age, weight and litter size had no effect on sow titer ($P>.05$). Sow age, sow weight, sow titer, litter size and birth weight had significant effects on piglet IgG titers at 12 to 24 h after birth. Significant two-factor interactions for piglet IgG titers at 12 to 24 h after birth were observed between birth weight x litter size, sex

x sow age, sex x sow weight and sow titer x birth weight. At 10 wk, sources of variation affecting IgG titers included sow titer, litter size, titers at 12 to 24 h and weight at 10 wk (P<.01). At 15 wk, sow weight, sow age and titers at 12 to 24 h significantly affected immunoglobulin titers. Heritability estimates obtained for 12 to 24 h titers and 15 wk titers were 2.52 $\pm$ 2.37 and .37 $\pm$ .86, respectively.

(Key Words: Swine, Immune Response, Immunoglobulin G, Kinetic Enzyme-Linked Immunosorbant Assay, Heritability.)

INTRODUCTION

Disease resistance is associated with the immune response to infectious agents and as such may be due to humoral and/or cellular responses. Individuals with a tendency for depressed antibody production when challenged by a pathogen are generally more susceptible to disease occurrence. Because of this relationship, the immune response may be used as an indicator of relative disease resistance (Crittenden, 1983). It has been shown that animals with a high antibody response to one antigen tend to exhibit high responses to other non-related antigens (Biozzi et al., 1970). It is therefore possible to estimate the relative ability of individuals to resist disease by exposing them to a non-pathogenic antigen and determining the levels of their corresponding immune responses.

Several researchers have demonstrated genetic variation in the immune response to pathogenic and non-pathogenic antigens. Edfors-Lilja et al. (1984) found pronounced variation within or between litters in antibody titers in piglets naturally reared on sows immunized to two Escherichia coli antigens. Genetic differences in serum-neutralization titers of pigs after vaccination with pseudorabies modified live-virus vaccine were found by Rothschild et al. (1974). Huang (1977) found variation in the immune response of pigs injected with bovine serum albumin (BSA). Biozzi et al. (1979) produced high and low responder lines of mice by selective breeding for maximal or minimal agglutinin response to sheep erythrocytes. After selective breeding of rabbits immunized with Group C-Carbohydrate, complete segregation was achieved between high response and low response populations (Eichmann et al., 1971). Development of general disease resistance through indirect selection primarily on immune response traits may be presently limited by insufficient understanding of resistance mechanisms (Gavora and Spencer, 1983).

The objectives of this study were to examine the effects of sources of environmental variation including age of dam, dam's weight, litter size, dam's active immune response, sex of pig, birth weight and piglet weight at direct exposure of antigen on passively acquired immunity

and in active immune responses of piglets to ovine serum albumin. Genetic variation was also evaluated for effects on the immune response. Correlations between passive and active immune traits and between immune status and independent variables were calculated.

MATERIALS AND METHODS

Population. Swine at the Oregon State University Swine Center, Corvallis, Oregon were utilized in this study. The herd consisted of three inbred lines of purebred Yorkshires from a population closed in 1961 except for the occasional addition of replacement boars. Swine were maintained as a specific pathogen-free herd in total confinement. Females were bred according to a three-way rotational within-breed line cross and were hand mated.

From April to August 1984, a total of 37 sows were bred to one of six boars. Thirty-four sows were pregnant when tested by ultrasound (Ilis Preg Test Model 757, International Livestock Improvement Services Corp., Ames, Iowa) at 42 d (d 0= day of hand mating). At 65 d sows were subcutaneously injected at three sites (on both sides of the neck and in one ham) with a total of 2 ml of a solution containing 50 mg ovine serum albumin (OSA; Sigma, St. Louis, MO.) in 20 ml of a 1:1 emulsion of physiological saline and Freund's complete adjuvant. At 93 d, sows were injected with 2 ml of 25 mg OSA in a 1:1 emulsion of

physiological saline and Freund's incomplete adjuvant. Two control sows were given a similar series of injections except that OSA was omitted from the preparations. At 100 d, 10 ml of blood were collected from each sow via jugular venipuncture.

Between 12 and 24 h postpartum, 10 ml of blood were collected via anterior vena cava puncture from all piglets farrowed by sensitized and control sows. Piglets were weaned between 4 and 8 wk of age. At this time, if possible, three piglets of each sex from each litter were selected randomly for determination of active immunity. Pigs were given the same series of injections at 10 and 14 wk of age as were their dams at 65 d and 93 d of gestation. As controls, six pigs from each of three litters of nonsensitized sows were given the same series of injections without OSA. Ten ml of blood were collected from each pig at 10 and 15 wk of age as pre- and post-injection samples, respectively.

Blood samples were allowed to clot at room temperature for 12 h, then stored at 4° C for 12 h followed by centrifugation at 5000 x g for 15 min. Serum was separated and stored at -20° C. All serum samples were assayed for swine anti-OSA immunoglobulin G (swine anti-OSA IgG) by a kinetic enzyme immunosorbant assay (K-EIA) as described by Tsang et al. (1980) with modifications.

Antigen Preparation. Lyophilized OSA was diluted to 10

ug/ml in .05M carbonate-bicarbonate buffer (pH 4.0). One hundred ul of diluted antigen were dispensed into each well of 96 well 1/2 area EIA plates (Costar, Cambridge, MA). Plates were wrapped in parafilm (VWR Scientific, San Francisco, CA), placed in humid containers and stored at 4° C for 1 to 14 d.

Enzyme-Antibody Conjugate. The immunoglobulin G (IgG) fraction of rabbit antisera against swine IgG (light and heavy chains) conjugated to horseradish peroxidase (Miles Labs, Naperville, IL) was divided into 100 ul aliquots and stored at -20° C. Aliquots in use were stored at 4° C. The working dilution of enzyme-antibody preparation, as determined by preassay trials at varying concentrations, was 1:500 in a buffer (pH 7.4) containing 1% gelatine (Knox Gelatine, Inc., Englewood Cliffs, NJ), 8.7 g NaCl, .372 g EDTA, and 6.05 g Tris base/liter with .5 ml Tween 20.

Substrate Solution. Thirty percent H_2O_2 was diluted 1:10 in 1.05% citrate buffer (pH 4.0). Seventy-five ul of 40 mM 2,2'azino-di-(3-ethylbenz-thiazaline-6-sulfonic acid) (ABTS) stock was diluted in 10 ml of citrate buffer. Twenty-five ul of diluted H_2O_2 were added to diluted ABTS immediately before each assay.

Kinetic Enzyme-Linked Immunosorbant Assay. Sensitized EIA plates were washed four times with phosphate-buffered saline (PBS, pH 7.2) containing 8.5 g NaCl, .22 g NaH_2PO_4 and 1.19 g Na_2HPO_4/liter. Each serum sample, diluted in

the Tris buffer previously described, was assayed in quadruplicate on the same plate as was a serum control. One hundred ul of serum at optimum dilution were dispensed into each of the four wells. Plates were placed in a humid container and incubated at room temperature for 1 h. Plates were then washed four times with the PBS. One hundred ul of diluted conjugate were dispensed into each well. Plates were placed in humid containers and incubated for 30 min at room temperature. Plates were again washed four times in PBS and 100 ul of substrate solution dispensed into each well after which plates were read at 0, 2 and 4 min on a Biotech EL-310 EIA reader (Biotech, Burtlington, VT) set at 405 nm.

High, medium and low responder serum pools were developed to serve as standards from each of the following: sow, newborn and 15 wk groups. Serum pools for each group were titrated in Tris buffer and were assayed on the same plate in duplicate. Slopes for each dilution of each pool were developed by plotting optical density (OD) vs time. Titration curves were plotted as each pools' slope vs increasing dilution. The dilution corresponding to 50% of the maximum slope (Y value) was set to equal one EIA Unit (EU). The dilution, when expressed as a decimal, provided the volume of sera required to achieve that dilution. Values were then converted to EU/ml. Titers of the high, medium and low pools were expressed in this manner. The

optimum dilution for each group of samples was that value which was on the linear portion of all three titration curves. This value was 1:3,000 for sows, 1:1,000 for piglets and 1:4,000 for 15 wk old pigs. The three pools were assayed on each plate with sera from animals of their respective groups. Slopes of the standard pools were plotted against their corresponding EUs to develop a standard curve and each serum sample slope was regressed onto this curve. If a sample fell above or below the values of its standard curve a new dilution of that sample was made and reassayed. Ten wk serum samples expressed passively acquired immunity as did the piglet samples and were run at a 1:10 dilution utilizing the piglet standard curve.

An intraassay coefficient of variation (CV) was determined for each assay as the average of the CV's among sample quadruplicates. Interassay CV's were determined from the serum standards included on each plate to evaluate the assay's repeatability.

Statistical Analysis. Swine anti-OSA IgG levels measured in EUs in the sows (SOWEU), piglets at 12 to 24 h after birth (0EU), at 10 wk of age (10EU) and at 15 wk of age (15EU) were analyzed separately by least-squares analysis of variance with unequal subclass numbers (SAS, 1985) to determine which variables significantly affected the immune response. The amount of time between sow samp-

ling and farrowing (DAYSBLD) was included in all analyses as a covariate. Dependent variables (SOWEU, 0EU, 10EU and 15EU) and the covariate were the only two continuous variables in the analysis, all other factors were considered discrete; levels of the independent factors are listed in table 1. In the preliminary analysis, all main effects and two-factor interactions were included in the model; non-significant interactions were removed from the model in a stepwise manner and were not included in the final model.

Main effects included in the model for each dependent variable are listed below:

SOWEU: litter size, age and weight

EU0: litter size, age of dam, weight of dam, birth weight, sex, dam's EU

10EU: litter size, age of dam, weight of dam, birth weight, sex, dams EU, weight at 10 wk

15EU: litter size, age of dam, weight of dam, birth weight, sex, dams EU, EU10, weight at 15 wk.

Phenotypic correlations among immune traits and independent variables were calculated. For 0EU and 15EU, independent variables and two-factor interactions which attained statistical significance (P = .05) were included as fixed effects in a variance component analysis (VARCOMP, SAS, 1985). Sire and dam nested within sire were random effects. Maximum likelihood estimates were used to

estimate heritability in the following paternal full sib analysis:

$$h^2 = \frac{4\sigma_s^2}{\sigma_s^2 + \sigma_d^2 + \sigma_e^2} = \frac{4\sigma_s^2}{\sigma_p^2}$$ (Turner and Young, 1969)

where σ_s^2 = sire variance

σ_d^2 = dam within sire variance

σ_e^2 = error

Standard errors for the heritability estimates were calculated using the formula:

$$\hat{\sigma}(h^2) \cong \frac{4A}{\sigma_p^2}$$

where $$A = \sqrt{\frac{2}{k_2^2}\left[\frac{MS_s^2}{n_s} + \frac{MS_d^2}{n_d}\right]}$$

MS_s^2 = mean square^2 of sire

MS_d^2 = mean square^2 of dam within sire

n_s = number of sires

n_d = number of dams (Turner and Young, 1969)

$$K_2 = \frac{N - \sum N_i^2/N}{s-1}$$

where N = total number of offspring

N_i = number of offspring of the ith sire

s = number of sires

RESULTS

Intra- and interassay coefficients of variation for the sow, 0, 10 and 15 wk assays were 12% and 26%, 9% and 14%, 10% and 16% and 14% and 20%, respectively. High intra-assay coefficients of variation were obtained due to the rapid rate of the enzyme-substrate reaction in our assay system; this could be slowed by diluting the substrate further. The interassay coefficients of variation might be lowered by performing the assays in a temperature controlled room. The average correlation coefficients (r) for standard curves of each assay were .97, .97, .98 and .95, respectively.

Sow Elisa Units. The overall mean for anti-OSA IgG as measured by SOWEU was 4263 $\pm$ 41. Sow Elisa Units were neither significantly affected by nor correlated with litter size or weight (Tables 2 and 3). Sow Elisa Units did not differ significantly among age groups, but age and SOWEU were significantly correlated (r= 0.29920; P=.0852).

Elisa Units at 12-24 hours. Elisa Units at 12 to 24 h represent the level of anti-OSA IgG present in the piglets' bloodstream after a 12-24 h suckling period. Because the piglets were not directly exposed to OSA, these IgG levels could only have been obtained via their dam's colostrum; the overall mean for 0EU was 1746 $\pm$ 22. Sex was not a significant factor in antibody levels at birth nor were sex and antibody levels at birth correlated (Tables 2 and 3).

Age of the dam was significant and was positively correlated to 0EU. Weight of dam contributed to the value of 0EU, with sows in the middle weight classes producing piglets with higher immunoglobulin titers. Size of litter was highly significant in that as litter size increased, 0EU decreased. Birth weight was also highly significant and positively correlated to 0EU as was SOWEU (Tables 2 and 3). Changes in the variables affecting the immune response resulted in the significant interactions between BW X Littersize, BW X SOWWT, BW X SOWEU, Sex X SOWAGE and Sex X SOWWT. Tables 6, 7, and 8 list means and standard errors for significant interactions. Elisa Units at 10 weeks. Elisa Units at 10 wk measured the residual anti-OSA IgG obtained after birth from the colostrum. The overall mean was 12 $\pm$ .3. Age of sow, SOWWT, BW and sex were neither correlated with nor did they significantly affect 10EU (Tables 2 and 3). Littersize and SOWEU significantly affected this trait, but no trends were evident. Elisa Units at 12 to 24 h and WT10 both had a highly significant effect upon and high correlations with 10EU.

Elisa Units at 15 weeks. Actively produced IgG against anti-OSA was measured as 15EU. Only four of the 10 variables measured had a significant effect on this trait (Tables 2 and 3). The overall mean value for 15EU = 4332 $\pm$ 163. Variation in age of sow was a significant source of influence on the immune response, but trends were not

sufficient to result in a significant correlation. As SOWWT increased, so did 15EU; as 0EU increased, 15EU decreased indicating possible inhibition of the immune response due to residual passive immunity, but the correlation between these two traits was non-significant.

Heritability. Heritability estimates for passive immunity measured as 0EU and active immunity measured as 15EU, presented in Table 11, were determined by paternal full-sib analyses.

DISCUSSION

The IgG concentration in sow serum sharply decreases during 14 to 17 wk of gestation (Klobasa et al., 1985). Because this was the period during which sow serum samples were taken and due to it being a variable for which we were directly responsible and not a characteristic of the sows, DAYSBLD was entered in all analyses as a covariate.

Means of significant factors and their standard errors are listed in tables 4, 5, 9 and 10.

In a study to determine factors influencing the IgG concentration in swine colostrum, Inoue et al. (1980) found a strong correlation between the number of parturitions and IgG level, yet age and IgG level were poorly correlated. Klobasa et al. (1985b) compared serum IgG levels between sows in their first gestation to sows in later parities. First gestation sows had the lowest total serum IgG levels

with the levels increasing up to the sixth or seventh gestation; as age was related to parity, the oldest sows were approximately 4 1/2 yr of age. The largest increase in IgG was between the first and second gestations. Results of a later study (Klobasa et al., 1986) confirmed these data, with first litter sows having lower serum IgG levels than older sows. The relationship between age of sow and piglet serum concentration is different for each class of immunoglobulins (Klobasa et al., 1986), but is consistent with the sow data for IgG. Our results indicate that as age of sow increased, SOWEU increased as did 0EU. SOWAGE was not not a significant influence on either SOWEU or 15EU due to the large variation within age groups. Although results indicate an increase in immunity with age, it may be that sows with a greater immune response than their counterparts produce healthier litters and in turn are kept longer in the herd, as well as having a greater opportunity to become exposed to more antigens. Because the sows in our study had no earlier opportunity to become exposed to OSA, the former explanation would seem more likely although exposure to other antigens with similar determinants could cause increased production of the anti-OSA antibodies.

Sow weight significantly affected IgG levels at 0EU and 15EU. Titers of IgG after birth peaked in SOWWT class 2 then decreased across classes 3, 4 and 5. Sow weight was

a highly significant factor in determining 15EU; as SOWWT increased to class 4, so did the active immunity of the offspring at 15 weeks. In SOWWT classes greater than 4, the levels dropped to those of SOWWT class 2. These results may indicate that underweight and obese sows do not produce offspring which are as immunocompetent as offspring from more fit sows of middle weight classes. Other studies dealing with the associations between sow weight and immunity need to be conducted. Because sow weights were taken prior to farrowing, larger weights were correlated to larger litters; this in turn corresponds to the values obtained for Litter size.

Litter size significantly affected 0EU and 10EU, but by 15 wk this association was no longer evident. Yaguchi et al. (1980) measured serum gamma globulin levels in pigs 12 to 18 h after birth by ammonium sulphate reaction. Our negative correlation between Litter size and 0EU is in agreement with their results which indicated the number of animals with low gamma globulin levels increased as litter size increased. This observation may be due to several factors. First, Hendrix et al. (1978) reported that pigs born earlier in a litter suckle when up to 50% more immunoglobulins are present in the colostrum. If immunoglobulins in colostrum are limited, then piglets born later in large litters will obtain fewer immunoglobulins when first suckling than their older littermates. Secondly, there is gen-

erally a higher ratio of smaller piglets in larger litters. As these pigs are normally less viable than their heavier littermates, they often do not suckle as long and/or as often as their littermates nor are they able to suckle from the more productive udder sections due to competition. As serum immunoglobulin concentration in newborn piglets is dependent on intake up to a maximum of 60 ml of colostrum (Brambell, 1970), these piglets therefore obtain less colostrum and a lower quantity of immunoglobulins than their littermates.

In our study, SOWEU significantly affected 0EU and 10EU but had no influence on 15EU. Because newborn piglets could only have obtained anti-OSA IgG from their dams it was surprising to find these traits so lowly correlated, perhaps indicating that SOWEU is only one of several significant factors affecting 0EU. Brambell (1970) lists levels of immunoglobulins in piglet sera up to four times that of the sows serum, depending on the specificity of the antibodies. Our low correlation may be due to the wide variation of 0EU within litters due to litter size and birth weight. The significant effect of SOWEU on 10EU may indicate that piglets suckling sows with medium titers of IgG may maintain elevated IgG titers at 10 wk as compared to piglets suckling sows with high titers. This may be due to a more rapid growth rate and in turn a greater dilution factor of remaining immunoglobulins in piglets suckling

sows with high IgG titers (Schultze and Heremans, 1966).

Sex was non-significant in all our analyses. Because swine in this population do not normally reach sexual maturity until approximately 7 mo, it was doubtful that sex would play any role in the immune response of the pigs sampled. A comparative study of immune response and production traits and/or carcass characteristics as animals approach maturity might prove useful and informative.

Birth weight as expected, played a significant role in the acquisition of passive immunity measured in the newborn pig. In our study it was the trait most significantly and highly correlated with OEU. Our results are in agreement with those of Hendrix et al. (1978) and Yaguchi et al. (1980) who found levels of gamma globulins at 12 h and 12 to 18 h, respectively, to be greatly influenced by birth weight. As previously stated, it is generally the larger pigs that are more viable and competitive, thus they suckle more than their smaller littermates and from the more productive mammary glands. Studies also indicate that piglets which obtained greater quantities of passively acquired immunoglobulins are more likely to survive than their littermates with inadequate quantities (Hendrix et al., 1978; Yaguchi et al, 1980; and Klobasa and Werhahn, 1981). Although time and labor consuming, piglet survival may be enhanced by removing the larger pigs from the sow after farrowing thus allowing the smaller pigs an oppor-

tunity to suckle with less competition. When all small piglets have nursed sufficiently, the larger pigs can once again be reunited with the sow.

Passive immunity to OSA as measured by 0EU had significant effects both on the residual passive immunity measured at 10 wk and on subsequent active immunity at 15 wk. The correlation between 0EU and 10EU was the highest obtained in this study. Although other factors affect the quantity of passively acquired antibodies in pig serum, it is obvious that the initial quantity present is of extreme importance. Although 0EU was significant in the analyses of IgG at 15 wk, no differences between means could be detected; there was no correlation between these traits.

Curtis and Bourne (1971) suggest that the rapid decrease in piglet immunoglobulin level after 24 h is due to the equilibration of intra- and extra-vascular body fluids. After 2 d the decrease results from catabolism and the dilution of immunoglobulins due to the rapid increase in body size and hence blood volume (Schultze and Heremans, 1966). On the basis of this report, it is anticipated that a negative correlation would exist between 10 wk weight and residual passive immunity (10EU). On the contrary, a highly significant positive correlation was found between the two traits. An explanation may be that pigs with high initial passive levels, thus high residual levels, were more viable and as such, reached a greater 10 wk weight.

When active immunity was measured in pigs at 15 wk, weight was not an important factor affecting immune response.

Passively acquired IgG levels measured in piglet serum reach a minimum between 4 and 8 wk of age (Brambell, 1970; Klobasa and Werhahn, 1981; and Klobasa et al., 1986). Various studies have demonstrated that although passively acquired antibodies are necessary for the survival of neonatal pigs, if they exist in too great a quantity when a pig is actively exposed to a specific antigen, they have an inhibitory effect. Wilson and Svendsen (1972) compared piglets suckling sows vaccinated against E. coli with piglets suckling non-vaccinated sows. Pigs suckling vaccinated sows were unable to actively produce antibodies against E. coli up to 5 wk of age. Similarly, piglets with less immunoglobulins after suckling had higher immunoglobulin levels at 3 wk than their littermates, that is, those obtaining fewer immunoglobulins from colostrum synthesized more later on and vice-versa (Klobasa et al., 1986).

Hoerlein (1957) concluded that vaccination prior to farrowing would protect pigs shortly after birth, but may interfere with active immunization later; in this case between 3 to 6 wk of age. Considering the above, since residual passive immunity at 10 wk had no significant effect on active immunity at 15 wk, it can safely be assumed that the levels of anti-OSA antibodies remaining at 10 wk were insufficient to inhibit the active synthesis of

anti-OSA IgG.

The heritability estimate obtained for 0EU (2.52 ± 2.37) exceeds the theoretical maximum value possible. This overestimation is due in part to a zero sow within sire variance component. Although sows were assigned randomly to be bred to one of six boars, it may be possible that sow immune responses within the three lines are similar. It is doubtful that they would be so similar that zero variation would result, therefore, our estimate must be confounded by other undetermined factors. The heritability estimate of 15EU is more probable. Huang's (1977) estimate of the heritability of peak immune response to BSA in swine when measured at 10 wk was .40 ± .04; very close to our estimate of .37. Unfortunately, neither of our estimates are reliable due to large standard errors. This may be due in part to the small number of sires utilized and the limited numbers of offspring sampled, particularly at 15 wk.

CONCLUSIONS

It is generally accepted that immunologically competent piglets are better able to survive than are their littermates with low immunoglobulin levels. Because the immunity status of piglets is dependent upon colostral immunglobulins for several weeks after birth, it is important that sows possess high serum immunoglobulin levels and therefore also high colostral levels. These passively

acquired antibodies may delay development of active immune responses by piglets due to interference. To ensure that piglets are adequately protected against pathogens, sows should only be innoculated against those agents that will threaten the piglets survival prior to the time they are capable of developing their own immune responses; at that time, piglets can be vaccinated against potential pathogens. By following a vaccination regimen that takes these factors into consideration, producers can more efficiently protect their herds from disease.

Residual passive immunity at 10 wk was greatly affected by 12 to 24 h titers of immunoglobulin and also the weight at 10 wk. This may indicate that those animals with high passive titers at birth grew more rapidly than their littermates which obtained low titers after birth. Studies of the immune response and its association with growth rate might provide useful information for enhanced production of swine.

Active immunity at 15 wk was affected by several assessed factors, but was significantly correlated only with SOWWT. Immunoglobulin G titers at 10 wk had no influence on the development of active immunity measured at 15 wk indicating that residual titers of anti-OSA IgG did not interfere with the active production of these antibodies. Further studies need to be conducted to determine optimum levels of factors for obtaining increased immune

response. In addition, optimum immunoglobulin titers for specific disease situations need to be determined. It is not always the animals with the high immunoglobulin titers that survive, in certain instances the outcome is just the opposite.

The heritability estimates obtained in this study suggest that both passive and active immunity are heritable; further studies utilizing greater numbers of sires, dams and offspring need to be conducted to obtain more definitive estimates of these genetic influences.

In addition to humoral immunity, cellular immunity also plays a large role in disease resistance. An evaluation of the effect of factors examined in this study on their effect on cellular immunity, and also the relationship between cellular and humoral immunity, would provide relevant further information.

TABLE 1. CLASSIFICATION OF INDEPENDENT VARIABLES.

INDEPENDENT VARIABLE	RANGE	CLASS
SOWAGE	≤ 1 year	= 1
	> 1 year < 2 years	= 2
	> 2 years < 3 years	= 3
	≥ 3 years	= 4
SOWWT	< 136 Kg	= 1
	> 136 Kg < 182 Kg	= 2
	≥ 182 Kg < 227 Kg	= 3
	≥ 227 Kg < 272 Kg	= 4
	≥ 272 Kg	= 5
SOWEU	< 1000 EU	= 1
	≥ 1000 EU < 2000 EU	= 2
	≥ 2000 EU < 3000 EU	= 3
	≥ 3000 EU < 4000 EU	= 4
	≥ 4000 EU < 5000 EU	= 5
	≥ 5000 EU < 6000 EU	= 6
	≥ 6000 EU < 7000 EU	= 7
	≥ 7000 EU < 8000 EU	= 8
	≥ 8000 EU < 9000 EU	= 9
	≥ 9000 EU < 10000 EU	= 10
	≥ 10000 EU < 11000 EU	= 11
	≥ 11000 EU	= 12
LITTER SIZE	= 6,7,8,9,10,11,12,13,14,15	
SEX	MALE = 1 FEMALE = 2	
BW	< .90 Kg	= 1
	> .90 Kg < 1.36 Kg	= 2
	≥ 1.36 Kg < 1.82 Kg	= 3
	≥ 1.82	= 4
WT10	< 13.6 Kg	= 1
	> 13.6 Kg < 18.2 Kg	= 2
	≥ 18.2 Kg < 22.7 Kg	= 3
	≥ 22.7 Kg < 27.2 Kg	= 4
	≥ 27.2 Kg < 31.8 Kg	= 5
	≥ 31.8 Kg	= 6
WT15	< 18.2 Kg	= 1
	> 18.2 Kg < 22.7 Kg	= 2
	≥ 22.7 Kg < 27.2 Kg	= 3
	≥ 27.2 Kg < 31.8 Kg	= 4
	≥ 31.8 Kg < 36.3 Kg	= 5
	≥ 36.3 Kg < 40.9 Kg	= 6
	≥ 40.9 Kg < 45.4 Kg	= 7
	≥ 45.5 Kg < 49.9 Kg	= 8
	≥ 49.9 Kg < 54.5 Kg	= 9
	≥ 54.5 Kg	= 10
0EU	< 1000 EU	= 1
	> 1000 EU < 2000 EU	= 2
	≥ 2000 EU < 3000 EU	= 3
	≥ 3000 EU	= 4
10EU	< 1 EU	= 1
	> 1 EU < 10 EU	= 2
	≥ 10 EU < 20 EU	= 3
	≥ 20 EU	= 4

TABLE 2. SUMMARY OF ANOVAS FOR ANTI-OSA IgG TITRES (EUS).

DEPENDENT VARIABLE	N	DF	SOURCE OF VARIATION	F VALUE	P VALUE
SOWEU	34	1	DAYSBLD	.61	.4447
		9	LITTERSIZE	1.00	.4759
		3	SOWAGE	.12	.9491
		4	SOWWT	.69	.6097
0EU	386	1	DAYSBLD	.02	.8918
		9	LITTERSIZE	7.30	.0001
		3	SOWAGE	6.56	.0003
		4	SOWWT	7.68	.0001
		3	BW	10.67	.0001
		1	SEX	.84	.3600
		10	SOWEU	3.78	.0001
		17	BW X LITTERSIZE	2.62	.0010
		3	SEX X SOWAGE	5.26	.0015
		6	BW X SOWWT	2.82	.0110
		4	SEX X SOWWT	2.45	.0462
		18	SOWEU X BW	2.12	.0055
10EU	175	1	DAYSBLD	.13	.7190
		9	LITTERSIZE	3.48	.0007
		3	SOWAGE	1.30	.2786
		4	SOWWT	.69	.5974
		3	BW	1.58	.1981
		1	SEX	.41	.5243
		10	SOWEU	3.00	.0019
		3	0EU	11.51	.0001
		5	WT10	4.51	.0008
15EU	169	1	DAYSBLD	1.90	.1711
		9	LITTERSIZE	1.64	.1109
		3	SOWAGE	2.32	.0789
		4	SOWWT	4.06	.0040
		3	BW	1.65	.1001
		1	SEX	1.44	.2357
		10	SOWEU	1.37	.2438
		3	0EU	2.73	.0470
		2	10EU	.05	.9531
		9	WT15	.72	.6901

TABLE 3. CORRELATION COEFFICIENTS BETWEEN ANTI-OSA IgG TITERS AND INDEPENDENT VARIABLES.

	SOWEU N = 34	0EU N = 386	10EU N = 175	15EU N = 169
SOWAGE	.29920	.09678	.02102	.09851
P	.0856	.0575	.7825	.2026
SOWWT	.14383	.04471	.09138	.23676
P	.4171	.3810	.2291	.0019
LITTERSIZE	-.26672	-.15871	.11982	-.00768
P	.1273	.0018	.1142	.9211
SOWEU	----	.14396	-.03699	.12761
P		.0046	.6270	.0983
SEX		-.07767	-.04388	-.06434
P		.1277	.5642	.4059
BW		.36264	.08153	.09794
P		.0001	.2834	.2052
0EU		----	.47839	-.03359
P			.0001	.6646
WT10			.39220	.09385
P			.0001	.2249
10EU			----	-.00921
P				.9054
WT15				.01776
P				.8188

TABLE 4. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 12 TO 24 H (SOW TRAITS).

SOURCE	N	MEAN	SE	P
SOWAGE				<.05
1	28	1987[a]	97	
2	231	1654[b]	47	
3	60	1739[a,b]	80	
4	67	1967[a]	90	
SOWWT				<.01
1	24	1286[a]	129	
2	18	2150[b]	87	
3	201	1760[c,d]	50	
4	129	1796[b,c]	63	
5	14	1350[a,d]	149	
SOWEU				<.01
1	11	2072[a,b]	164	
2	51	1764[a,c]	51	
3	94	1610[a]	58	
4	45	1708[a,c]	86	
5	79	1432[a]	92	
6	34	2262[b]	114	
7	27	2092[b,c,d]	85	
8	13	1378[a,d]	189	
9	9	2439[b,c]	93	
10	--	----	--	
11	10	2039[a,b]	243	
12	13	2002[a,b]	224	

[a,b,c,d]Means within sources with different superscripts differ (P<.05).

TABLE 5. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 12 TO 24 H (LITTER TRAITS).

SOURCE	N	MEAN	SE	P
LITTERSIZE				<.01
6	6	2375[a]	92	
7	7	2122[a,b]	570	
8	8	2057[a,b]	204	
9	18	1879[a,b]	193	
10	49	1619[a,b]	143	
11	86	1813[a]	64	
12	24	1992[a]	90	
13	77	1677[a,b]	83	
14	81	1772[a]	64	
15	30	1290[b]	115	
BW				<.01
1	59	1052[a]	103	
2	217	1829[b]	41	
3	106	1942[b]	58	
4	4	2241[b]	334	

[a,b]Means within source with different superscripts differ (P<.05).

TABLE 6. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 12 TO 24 H (SIGNIFICANT INTERACTIONS).

SOURCE	N	MEAN	SE	P
BW X LITTER SIZE				<.01
1-6	1	2697	0	
1-7	-	----	-	
1-8	-	----	-	
1-9	0	0	2	
1-10	4	468	231	
1-11	14	1282	194	
1-12	3	1631	519	
1-13	16	854	223	
1-14	15	1223	127	
1-15	4	658	309	
2-6	3	2268	70	
2-7	7	2122	216	
2-8	3	1704	515	
2-9	11	2307	101	
2-10	25	1395	189	
2-11	48	1893	74	
2-12	20	2065	76	
2-13	37	1766	79	
2-14	47	1880	79	
2-15	16	1505	147	
3-6	2	2375	205	
3-7	--	----	---	
3-8	5	2269	88	
3-9	5	1689	227	
3-10	18	2066	210	
3-11	24	1965	99	
3-12	1	1607	0	
3-13	23	2080	94	
3-14	19	1940	106	
3-15	9	1191	177	
4-1	--	----	---	
.	.	.	.	
.	.	.	.	
.	.	.	.	
4-10	2	2696	86	
4-11	--	----	---	
4-12	--	----	---	
4-13	1	2285	0	
4-14	--	----	---	
4-15	1	1286	0	

TABLE 7. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 12 TO 24 H (SIGNIFICANT INTERACTIONS).

SOURCE	N	MEAN	SE	P
SEX X SOWAGE				<.01
1-1	14	2118	106	
1-2	119	1692	66	
1-3	32	1722	132	
1-4	39	2066	119	
2-1	14	1855	160	
2-2	112	1615	67	
2-3	28	1759	83	
2-4	28	1829	137	
BW X SOWWT				<.01
1-1	4	314	213	
1-2	--	----	---	
1-3	30	1194	154	
1-4	20	1025	164	
1-5	5	896	284	
2-1	5	1318	128	
2-2	16	2139	98	
2-3	128	1865	55	
2-4	52	1804	87	
2-5	9	1603	108	
3-1	8	1722	149	
3-2	2	2234	114	
3-3	43	1843	100	
3-4	53	2046	79	
3-5	---	----	---	
4-1	---	----	---	
4-2	---	----	---	
4-3	---	----	---	
4-4	4	2241	334	
4-5	---	----	---	
SEX X SOWWT				<.01
1-1	11	1466	166	
1-2	10	2269	72	
1-3	104	1805	70	
1-4	73	1808	92	
1-5	6	1361	293	
2-1	13	1132	189	
2-2	8	2001	166	
2-3	97	1711	70	
2-4	56	1781	81	
2-5	8	1343	162	

TABLE 8. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 12 TO 24 H (SIGNIFICANT INTERACTIONS).

SOURCE	N	MEAN	SE	P
SOWEU X BW				<.01
1-1	---	----	---	
1-2	7	2124	123	
1-3	4	1982	435	
1-4	--	----	---	
2-1	8	1398	229	
2-2	31	1920	96	
2-3	11	1635	268	
2-4	1	1287	0	
3-1	15	1093	146	
3-2	50	1657	79	
3-3	29	1797	72	
3-4	--	----	---	
4-1	5	995	281	
4-2	25	1686	112	
4-3	15	1982	93	
4-4	--	----	---	
5-1	13	640	226	
5-2	45	1563	113	
5-3	20	1612	148	
5-4	1	2285	0	
6-1	3	520	473	
6-2	18	2314	74	
6-3	13	2592	81	
6-4	--	----	---	
7-1	7	1841	189	
7-2	16	2135	101	
7-3	4	2361	169	
7-4	--	----	---	
8-1	4	842	536	
8-2	8	1573	76	
8-3	1	1961	0	
8-4	--	----	---	
9-1	--	----	---	
9-2	8	2428	105	
9-3	1	2522	0	
9-4	--	----	---	
10-1	--	----	---	
10-2	--	----	---	
10-3	--	----	---	
10-4	--	----	---	
11-1	1	153	0	
11-2	3	2047	223	
11-3	4	2172	204	
11-4	2	2696	86	
12-1	3	1066	756	
12-2	6	2210	141	
12-3	4	2394	155	
12-4	--	----	---	

TABLE 9. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 10 WK.

SOURCE	N	MEAN	SE	P
LITTERSIZE				<.01
6	3	19.67[a,b]	4.91	
7	4	5.25[a,c]	1.11	
8	4	17.75[a,b]	1.31	
9	9	12.89[a,b]	1.32	
10	27	10.59[a,b]	1.48	
11	44	9.59[a]	1.00	
12	7	10.00[a,b]	1.23	
13	33	15.42[b]	.96	
14	32	15.03[b,c]	1.34	
15	12	9.75[a,b]	1.40	
SOWEU				<.01
1	6	14.83[a,b]	2.20	
2	22	16.18[b,c]	1.68	
3	42	12.21[a,b]	.93	
4	18	10.56[a,c]	1.40	
5	37	7.76[a]	1.08	
6	18	17.28[b]	1.52	
7	13	11.54[a,b]	1.79	
8	5	14.80[a,b]	1.66	
9	5	12.00[a,b]	1.82	
10	--	----	----	
11	3	16.33[a,b]	1.86	
12	6	12.17[a,b]	2.55	
EU0				<.01
1	14	3.64[a]	1.24	
2	86	11.09[b]	.66	
3	73	15.25[c]	.75	
4	2	17.00[b,c]	3.00	
WT10				<.01
1	46	9.02[a]	.85	
2	22	8.55[a]	1.06	
3	58	13.91[b]	.94	
4	32	13.97[b]	1.14	
5	16	17.00[b]	1.68	
6	1	23.00[a,b]	0	

[a,b,c]Means within sources with different superscripts differ (P<.05).

TABLE 10. MEANS AND STANDARD ERRORS FOR ANTI-OSA IgG TITERS AT 15 WK.

SOURCE	N	MEAN	SE	P
SOWWT				<.01
1	12	3070[a]	493	
2	9	3933[a,b]	620	
3	91	4058[a,b]	230	
4	53	5186[b]	321	
5	4	3949[a,b]	760	
EU0				<.05
1	14	5606	805	
2	82	4008	256	
3	71	4444	230	
4	2	4378	911	

[a,b]Means within source with different superscripts differ (P<.05).

TABLE 11. HERITABILITY ESTIMATES OF 12 TO 24 H AND 15 WK ANTI-OSA IgG TITERS.

TRAIT	h^2	SE
0EU	2.52	2.37
15EU	.37	.86

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