CHARACTERIZATION OF CREEP FEEDING AND ITS SUBSEQUENT EFFECTS ON IMMUNE RESPONSE, SCOURING INDEX, AND PERFORMANCE OF WEANLING PIGS

by

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(ABSTRACT)

Four trials were conducted to examine the pattern of creep feed consumption of nursing pigs and the effect of creep feeding - from 10 d to weaning at 28 d - a 20% crude protein corn-soybean meal and dried whole whey diet (REG, 9 litters) containing 1.0% chromic oxide for detection of creep feed consumption, or the same diet with 2.7% ovalbumin (OVA) added as a dietary antigen (14 litters), or no creep feed (CON, 11 litters) on the immune response, scouring index, and subsequent performance of weanling pigs. All nursing pigs were denied access to sow feeders but had water ad libitum. At weaning, pigs were fed either a 20% corn-soybean meal diet, with or without 2.7% OVA. Creep-fed litters began eating at 11 d of age and disappearance of creep feed increased linearly until weaning ($P < .01$). However, the presence of chromic oxide in the feces was not consistently observed in the same pigs thus suggesting that creep consumption by individual pigs was quite variable. Average feed consumption based on

total creep disappearance per litter was 13 to 194 g/pig preweaning. Daily gain and body weight of nursing pigs was similar during the first 4 wk. Pigs from larger litters had lower birth weights ($P < .05$), lower 4 wk body weights ($P < .09$) and daily gains ($P < .09$), as well as less feed disappearance per pig ($P < .02$). Pigs fed the OVA diet had higher ($P > .001$) antibody titers to OVA than did pigs receiving the REG or CON treatments at 14, 21, or 28 d of age. At 56 d and 63 d, all pigs given an OVA injection (at 49 d 1 ml of 3 mg/ml OVA) responded, ($P < .001$) with the lowest titers for REG pigs, intermediate for CON pigs, and the highest for OVA pigs during the nursery phase. Scouring began 4 to 5 d postweaning with creep-fed pigs scouring slightly more. Feed consumption, although not statistically different, was higher ($P = .18$) during the first week postweaning for CON pigs and was consistent throughout the study, but daily gains and feed efficiency were similar. In summary, creep feed disappearance begins increases linearly until weaning with no effect on cumulative daily gain or body weight. While larger litters consume more total feed, smaller litters consume more feed per pig. Creep feeding had some effect on antibody response and scouring our results suggest little apparent effect on postweaning performance.

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CHAPTER I

INTRODUCTION

For todays swine producers to continue to be competitive, they must increase yearly sow productivity, which can be improved by increasing litter size and by producing more litters per sow per year. In order to produce more litters per sow per year, producers have changed from conventional weaning at 6 to 8 wk of age, to early weaning at 3 to 4 wk of age. Early weaning, however, has lead to new problems; primarily a severe postweaning performance check that is associated with severe postweaning scours. Although the severity of the postweaning performance check may be reduced by providing a more controlled environment, this problem has not been prevented through improved nutrition (Bark et al. 1986, Etheridge et al., 1984), environmental conditions (Crenshaw et al., 1986; Gore et al., 1986), or through the use of feed additives (Holden, 1975).

With the conventional weaning, nursing pigs are given feed (creep feed) to provide additional energy and to improve the transition from milk to a solid feed postweaning. However, with early weaning, the benefits of creep feeding have been questioned.

Newby et al. (1985), hypothesized that short exposure to certain feed ingredients (mainly antigenic proteins) in

the creep feed can lead to the development of a transient dietary hypersensitivity upon re-exposure to the same ingredients (antigens) after weaning. In particular, it has been surmised that a pig consuming less than 100 g of creep feed preweaning, may not become tolerant to this feed antigen before weaning. Thus, secondary exposure of the pig to the dietary antigens at weaning may result in an immune response (hypersensitivity) damaging the lining of the intestinal tract (Newby et al., 1985). This hypersensitivity is said to be transient due to the apparent ability of the pig to recover from this phenomenon in less than 2 wk postweaning (Miller et al., 1983). The resulting scours can cause malabsorption and loss of electrolytes which, in conjunction with depressed appetite, result in poor postweaning performance (Etheridge, 1984).

The hypothesized state of hypersensitivity occurs at the time when the immune system of the weanling pig is changing from passive (antibodies provided in the milk) to active immunity (self-made antibodies). Thus, the pig is more susceptible to bacteria and disease; and in conjunction with the added stress of weaning, the sensitized immune system may possibly allow the onset of a postweaning performance check. Thus, the veracity of the hypersensitivity theory must be determined.

CHAPTER II

REVIEW OF LITERATURE

Creep feeding

Creep feeding is a management practice which consists of presenting freshly prepared diet (creep feed) containing high quality ingredients to nursing pigs between 7 to 14 d of age. The creep feed is placed in an area in which the pigs can move about freely, but where the sow does not have access. Ideally, creep feed should be offered frequently and in small amounts to promote curiosity for increased feed intake (Barczewski and Hartsock, 1987), and to prevent mold formation which could cause intestinal sickness leading to feed aversion and decreased performance (Etheridge, 1984). Water should also be provided ad libitum. In a conventional weaning system where pigs are weaned at 6 to 8 wk of age, creep feed is provided to supplement energy as well as to improve the transition at weaning from milk to dry feed.

Weaning at the conventional 6 to 8 wk of age produces a larger pig which is better able to adapt to the added stresses in the postweaning environment, resulting in less severe weight depression. Studies by Leibbrandt et al. (1975) demonstrate that pigs weaned at or before 21 d of age gain slower than pigs of the same age left to nurse

the sow. But, by 6 wk of age, all pigs increased to equal weight. Thus, sow productivity may increase with early weaning without sacrificing final weight gain outcome.

Traditionally, in a conventional 6 to 8 wk weaning system, one of the foremost reasons for creep feeding is that at 4 wk of age the baby pigs energy requirement had increased dramatically; whereas, the quantity of the sows milk had begun to decline steadily (peaks between 3 to 4 wk after parturition) (Pork Industry Handbook, 1987). Thus, the sow milk production can no longer meet the energy requirements of the fast growing pig. Therefore, some form of supplemental energy is required for the pig to continue to grow at a maximum level.

Another reason for feeding creep diets is to ensure that pigs nursing poor milking sows have supplemental energy as well as other nutrients. Pigs that show signs of hunger may require supplemental energy for survival (Newport, 1977). The common practice of placing creep feed in all sow farrowing crates may also help prevent overlooked malnourished pigs resulting from nursing problems in the sow, and may be beneficial for pigs within very large litters (Pork Industry Handbook, 1986).

Additionally, there is also some evidence to show that creep feeding may help prevent a postweaning performance check, because the creep feed presented

postweaning would not be viewed as a "foreign" substance upon weaning. Pigs, having been creep fed, would recognize the diet as feed, and thus increased feed intake postweaning would be promoted (Okai et al., 1976).

Poor performance of pigs weaned at less than 3 wk of age has been shown to be related to inadequate digestive development which could result in poor nutrient utilization (Leibbrandt et al., 1975). At birth, lactase activity is very high, but as the pig matures sucrase activity becomes much higher and lactase activity decreases dramatically (Kenworthy and Crabb, 1963). With creep feeding, even if the pig consumes only a small amount of feed, enzymes - such as proteases, and pancreatic lipases - can develop to a greater extent preweaning. Thus, the digestion of the corn-soybean meal diets can be improved, thereby, increasing the rate of gain postweaning (Leibbrandt et al., 1975). Likewise, a gut flora modification can occur that prepares the young pigs for weaning by providing a more gastrointestinal tract that is better suited for digestion and absorption of the corn-soybean meal diet (Okai et al., 1976).

Pigs begin eating creep feed at 7 to 12 d of age, with consumption increasing sharply at 14 to 21 d of age, while coincidentally, nursing frequency decreases at 21 d of age. Preweaning, average creep feed consumption was

determined by Barczewski and Hartsock (1986) to be approximately 107 g per pig. However, in the study of Barczewski and Hartsock (1986) sow feeders were available to the baby pigs. Thus, creep feed intake estimates may actually underestimate the amount of feed that might have been consumed. The creep feed consumption of 107 g coincided with a 5 kg increase in weight, showing that the gain did not necessarily come from the creep feed consumption; however, the small amount of feed consumed may have promoted the development of important enzyme (Barczewski and Hartsock, 1986; Okai et al., 1976). In these studies, pigs which were not creep fed preweaning (but were exposed to the sow feeder) gained as well as those creep fed.

Types of Creep Feed

Several studies have compared feeding simple and complex diets to determine if complex diets could reduce the postweaning performance check. Although most studies showed low creep feed intake for both complex and simple diets, complex diets generally improved feed intake and weight gain of pigs weaned at 5 wk of age (Okai et al., 1976). However, Bayley and Carlson (1970) showed that the postweaning check occurred using either simple or complex diets when pigs were weaned at 3 wk of age. Complex diets

showed a slight improvement over simple diets two weeks postweaning, but were not economically efficient. In addition, adding glucose to the diet did not reduce the postweaning performance check, but, in effect, aggravated it. Glucose is not completely absorbed and may serve as a readily available energy source for the microflora of the gastrointestinal tract (Bayley and Carlson 1970).

Lecce et al. (1979) showed that weaning at an early age onto a liquid diet rather than the conventional dry corn-soybean meal whey diet could improve the postweaning death loss (2% vs 6%). This phenomenon may actually be a result of reducing the irritation to the gut lining which may result when changing from a liquid to a solid feed (Tzipori et al., 1984).

Campbell et al. (1976) demonstrated that feed consumption of weanling pigs was increased when provided a starter diet containing the same flavor as that included in the sow diet. Speculation was that the weaned pig associates the creep feed flavor with the flavor of the milk. Thus, the flavor association may promote increased postweaning feed intake which could improve postweaning performance. To the contrary, Ogunbameru et al. (1979) demonstrated that regardless of whether or not the feed flavor was fed previously in the lactation diet, daily gain, feed intake and feed per gain was similar for pigs

fed flavored or unflavored diets postweaning. Furthermore, when choice is not available, there is no difference in diet consumption or performance of pigs fed sweetened (flavored) or unsweetened (unflavored) diets (Kornegay et al., 1979).

Immunity

The depressed immunological response of the pig between the ages of 1 and 3 wk is of concern when pigs are weaned less than 4 wk of age (Inque et al., 1978). Newborn pigs are basically devoid of antibodies due to very limited transplacental transfer as the epitheliochorial mode of placentation is totally impermeable to the sows serum proteins (Milon et al., 1983). The baby pig must absorb the antibodies through the mother's first milk designated "colostrum" (Allan, 1980; Bourne, 1976; Halsey and Benjamin, 1976.) The pig absorbs two thirds of these antibodies through an immature gut mucosa without degradation (passive immunity) during the first 12 hr after birth up to 48 hr (Hanson et al. 1979a; Bourne, 1976). Macromolecules of antibodies are absorbed via pinocytosis into the intestinal epithelium (Bush and Staley, 1980; Staley and Bush, 1985). Closure of macromolecular uptake (gut closure) is thought to be not only time dependent, but also dependent upon the

amount of colostrum consumed which leads to the exhaustion of the pinocytotic membrane (during starvation the intestine remains open) (Bainter, 1986). Gut closure progresses from duodenum to jejunum and lastly to ileum (Bainter, 1986).

Continuous presence of orally acquired colostral and milk antibody in the alimentary tract is of prime importance for protection of neonatal pigs against infective diseases until gut associated lymphoid tissue and other parts of the immune system become functional to provide local cell mediated and humoral immunity (Stone et al., 1979). Thus, the amount of colostrum and the time of consumption is of great importance. The antibodies are secreted into and are produced in the mammary glands of the sows and mirror the antibody profile of the adults intestinal fluid (Bourne, 1976). They can be found in the mammary tissue and milk secretory cells 3 wk prior to parturition (Bourne et al., 1978).

Humoral immunity is mediated by serum antibodies which are a heterogeneous mixture of serum globulins containing antibody activity (Barrett, 1983). Each antibody is made up of two identical light chains and two identical heavy chains (Eisen, 1980; figure 1). The three major types of antibodies contained in colostrum are IgG, IgM, and IgA. Eighty percent of the antibodies in

colostrum are in the form of IgG, the smallest antibody normally found crossing the placenta (Benjamini and Leskowitz, 1988). IgM is found to a much less degree (Inque et al., 1978). Composition of specific kinds of antibodies can be altered using an intramammary vaccination rather than the intramuscular vaccination producing local and systemic responses specific to these antigens which may be passed to the young through colostrum (Bourne, 1976).

As lactation progresses over the first week (from colostrum to milk), there is more than a 30-fold decrease in the production of IgG (Bourne, 1976). During this first week IgA, which is a minor component of serum and colostrum, becomes the major milk protein. IgA shows a greater resistance to enzymatic proteolysis than IgG antibody (Stone et al., 1979). IgA is most effective in intestinal defense of the alimentary tract because of continuous presence. It lasts for more than 90 minutes, much greater than normal suckling interval, allowing for continuous antibody on the intestinal epithelium of the suckling pig (Porter, 1973). IgA, also, provides a coating that limits the uptake of macromolecular and other nonreplicating antigens from the intestinal lumen. Additionally, antibody-antigen complexes formed in the lumen may enhance the degradation of the antigen in

glycocalyx and may stimulate an increased production of mucin from the goblet cells of the intestine (Ogra, 1976). Furthermore, intact antibody and fragments (Fab_1 and Fab_2) may be present in the gastrointestinal tract to neutralize viruses or other pathogens (Stone et al., 1979). In effect, the IgA secreted in milk functions much like secretory IgA of the adult intestine which the gut begins producing at 7 d of age. Initially, IgA production in the young pig is stimulated in Peyers Patches from where the stem cells migrate, while in the lymphoblast stage. Here, the cells mature, begin synthesis, and secretion of IgA (Bourne et al., 1978). A complete mature profile of antibody producing cells will be acquired by 28 d of age (Bourne, 1976). Thus, while colostrum provides the circulating antibodies, milk provides the intestinal antibody protection (Inque et al., 1978).

In conjunction with depressed immunological response at 3 to 4 wk of age, the pig after weaning is no longer receiving IgA antibody for coating of the intestine. Also, weaning is itself an acute stressor; although not detrimental, it may set up situations for immunological reactions to occur. In particular, regrouping the pigs at weaning according to size, weight, optimal growth performance, and space in nursery, may actually cause alterations in immune function and subsequent increase in

susceptibility to disease (Blecha et al., 1985). Pigs weaned at 2 to 3 wk of age have lower lymphocyte blastogenic responses than do nonweaned littermates. Also, Blecha et al. (1985) showed plasma cortisol concentrations to be higher in non-littermate pigs than littermates housed together at weaning. Additionally, intradermal responses to PHA were decreased and primary antibody responses to SRBC were deprived for non-littermate pigs. Although these immunologic depressions occur postweaning, there appears to be little effect on long term performance. But, abscesses and bacterial infections may be noted on pigs due to fighting, which may result in lacerations opening the skin and allowing penetration of bacteria (Blecha et al., 1985). Consequently, many producers keep the litter together at weaning or use various odor masking agents or even tranquilizers to decrease this problem of fighting postweaning.

Hypersensitivity and the immune system

The transient immune response that is theorized as hypersensitivity may be contributing to postweaning problems in this vulnerable stage of changing immunity. Pigs confronted with large amounts of antigenic food are susceptible to dietary immune response when exposed to the

feed for the first time (Miller et al., 1983). Huntley (1979) demonstrated that only small amounts of the dietary antigen are required to induce a hypersensitivity response amounting to less than .01% of the daily protein intake of the animal. Thus, the pigs, having been given creep feed and consumed small amounts, may produce circulating antibodies to that food protein (Newby et al., 1985). This increase in antibody production, whether transient or permanent, is associated with subjects deficient in IgA. IgA is considered deficient in nonruminant species (Newby et al., 1980), which may contribute in the development of hypersensitivity. IgA deficiency in man is associated with the presence of high titers of antibodies to milk and intestinal atrophy in human infants. With IgA no longer forming complexes with antigens for retaining the antigen in the lumen for digestion, there is no longer an immune exclusion, thus, this defectiveness may elicit an IgE, or a cell mediated immune response (Stokes et al., 1983a). The small intestine mucosa contains both thymus dependent and thymus independent lymphoid cells and thus has the capacity to act via humoral and cellular mechanisms as local immunity hypersensitive reactions (MacDonald and Ferguson, 1976). Huntley et al. (1979), however, could not stimulate cell mediated immune response through oral immunization in view of high levels of normal stimulation

of the entire immune system. A local cell mediated immune response may cause villous atrophy, crypt hyperplasia and malabsorption including coeliac disease, food allergies and intestinal infections (MacDonald and Ferguson, 1976). The physical integrity of the mucous membranes and mucous layers as well as other nonspecific mechanisms may be altered and the entry of an antigen into the systemic circulation through the mucous membranes of the gut mucosa is no longer prevented (Stokes et al., 1982).

Cell morphology of the intestine

Villous lesions, similar to those seen in humans suffering from cow's milk allergy, reduced levels of certain enzymes and reduced absorptive capacity were the results of a transient hypersensitivity to antigens in the diet of the mouse (Porter et al., 1973) and the pig (Stokes et al., 1982). The immune system of early weaned pigs may not be able to adapt to the small amounts of creep feed consumed before weaning. Villous changes begin when larger amounts of feed are consumed (Bourne, 1976). Poor postweaning performance may be due to reduction in villi heights and increased depths of crypts in the small intestine along with severe reductions in brush border enzyme activities (Hampson, 1986a). Normally, pigs 3 to 4 wk of age have enterocytes migrating from the bottom to

the top of villi in 4 d (mature). The bottom cells of the villi are secretory while the top cells are used for absorption. At weaning, immature cells at the top lead to postweaning performance checks and scours, allowing for bacteria such as Escherichia coli (E. coli) to enter and takeover the intestine (Bourne, 1986). However, villous atrophy has not been consistent in all hypersensitive responses (Miller et al., 1984c). Structural changes may be caused by factors other than hypersensitivity including physical damage to the gastrointestinal lining resulting from the change of liquid to solid diets, irregular supply of nutrients postweaning, action of rotaviruses and interactions of bacteria metabolites with the diet consumed (Hampson, 1986b).

Microflora colonize the cecum, large intestines, and rectum of healthy pigs. However, there is evidence of noninfective scouring, which shows no abnormalities of the alimentary tract, or disturbance of bacterial flora, and negative results of transmission of scouring to pigs within a pen. It is theorized that the biggest pigs are the ones most likely to show signs of nontransmissable scouring due to the larger pigs receiving the most milk preweaning and thus consuming the least amount of dietary antigens (creep feed). This phenomenon occurs much more commonly than E. coli and generally causes poor weight

gains; thus, making these economical losses possibly more important than those of E. coli (Smith, 1963). Thus, at 2 to 3 wk of age, an underdeveloped intestinal immune system with no outside IgA present in the milk, may indicate that colonization of undesirable microorganisms may no longer be inhibited (Newby et al., 1985).

Bacterial diseases

Enteropathogenic serotypes of E. coli are present in the gastrointestinal tract but they may predominate in the coliform flora of healthy pigs (Hampson et al., 1985). Thus, healthy animals may have large numbers of E. coli, but challenging them with E. coli does not consistently produce disease. In order to develop scouring, there must be a host predisposition which is influenced by weaning and diet (Miller et al., 1984a). Perhaps change in diet or withdrawal of milk production may promote scouring (Miller et al., 1984b). Other theories for increased scouring may be common progenitors of nutritional and bacterial scours due (in part) to increased metabolic activity of "normal" microflora (Kenworthy, 1976). That is, the E. coli present in healthy animals may become activated causing increased scouring.

Weaned pigs are more sensitive than unweaned pigs to diarrhea (Hamilton and Roe, 1977; Stevens et al., 1972).

Susceptibility of the pig decreases with age; however, it is not known whether the increase in resistance is due to action against microbacteria or enterotoxins or both (Stevens, 1963b).

Postweaning scours

Pigs weaned before 3 wk of age that are creep fed tend to consume very little feed and are susceptible to many postweaning digestive disturbances (Bayley and Carlson, 1970). Many stresses are known agents for scouring such as weaning, rotavirus, and *E. coli* (Miller et al., 1984b). Nutritional causes - such as moldy feed or feed without fiber - may predispose pigs to widespread enteritis, but it is possible to house healthy pigs with the affected pigs without spreading disease (Goodwin, 1957).

Consequently, shortened villi in the small intestine and changes in bacterial flora may result in decreased digestion and absorption of feed nutrients (Stevens, 1963a). Usually movement of water and electrolytes go out through the secretory cells and back via absorptive cells of the intestine which involves up to 30% of the body fluid per day (Hamilton and Roe, 1977). Imbalances occur with an increase in secretory cell capacity and decrease absorptive capacity resulting in a net significant fluid

loss (Newby et al., 1983; Porter et al., 1974). Scouring tends to peak 4 to 5 d postweaning with partial recovery occurring 10 to 14 d postweaning (Bourne, 1986).

With exposure to small amounts of feed preweaning, duration and severity is great. Pigs fed less antigenic diets scour less postweaning (Miller et al., 1983).

Immunogenicity of feedstuffs

Antigenicity, or in this case, immunogenicity (the ability of a substance to induce humoral or cell-mediated immunity) is determined by the number of antigenic determinants of a protein. The immune system does not recognize the macromolecular sites of a protein. Rather, it recognizes only the distinctive sites of immunological positions of the antigen molecule. Therefore, only the antigenic determinants on the surface of the macromolecule come into direct contact with the combining antibodies or Thymus (T) lymphocytes (Allan, 1988; Elgert, personal communication).

Sow milk contains approximately a 50:50 ratio of casein to whey protein (Newport and Henschel, 1985). Casein is known to be less immunogenic than many of the creep starter proteins that producers feed their weaned pigs. Casein, in particular, hydrolyzed casein, can be cleaved into small peptides. The smaller amino acid

chains may potentially carry less antigenic determinants and are more readily digestible (Hampson, 1986c). However, feeding casein reduces height of the villi (which is normal postweaning), but does not increase depth of crypt as much as that observed for pigs from other weaned groups not fed casein. A stimulation of increased crypt cell production may occur causing associated reductions in enterocyte maturity at weaning and promoting scouring. Newport and Henschel (1985) showed that a ratio of 60:40 casein to whey protein provided maximum growth rate, feed efficiency, and nitrogen retention. After feeding large proportions of whey (20:80 or 0:100), the pH of the gastrointestinal tract rose tremendously interfering with the degradation of nutrients and subsequently decreasing performance.

Providing a liquid milk replacer postweaning for primary nutrition of weanlings causes the gut mucosa to mimic that of the mucosa of preweaned pigs, where villi are less shortened and fewer reductions in brush border enzymes (Seegraber and Morrill, 1986; Wahlstrom et al., 1974). Milk replacers tend to lower the increases in crypt depth in the distal portion of the small intestine. Seegraber and Morrill (1986) produced an actual reversal of intestinal mucosa atrophy when milk was fed again within a 2 wk period postweaning.

Soybean meal, which is commonly placed as a cheap source of protein is considered highly antigenic. Pre-ruminant calves fed soybean protein with an increased proportion of protein from heated soybean flour, displayed diarrhea, weight loss, and poor growth (Kilshaw and Sissons, 1979a). Titer responses to soybean constituents were not detected during the first exposure, but gradually increased with successive feedings. Increased titer responses were associated with abnormal digesta movements through the small intestine as well as poor nitrogen absorption (Kilshaw and Sissons, 1979a). Barrett (1983), determined the antibody titers to be predominantly IgG. These antibodies interfere with digestive function by formation of complexes with soybean antigens and the fixation of complement (non-immunoglobulin serum proteins that are sequentially activated by antigen-antibody complexes or other substances). Biopsies of the ileal mucosa showed villous atrophy and edema, which is distinctly similar to soybean protein intolerance in the human infant. Antibody titers to soybean meal in diets fed to preruminant calves, rose and were maintained 7 to 8 wk postweaning (Kilshaw and Sissons 1979b). IgM was thought to have contributed very little to these sustained titers, but IgA or suppressor T lymphocyte cells may have caused a local immune response, limiting absorption. Some

type I hypersensitivity reactions (producing anaphylactic shock) were noted where calves were sensitized 30 minutes after feeding, suggesting that increased amounts of vascular permeability in the gut mucosa allow for exudation of considerable quantities of plasma proteins.

Different protein levels and methods of processing soybean meal vary in their ability to cause digestive disturbances and to the extent that they induce antibody production in calves. Extractions of soybean meal (hot aqueous ethanol at 65 to 80 C) caused no ill effects and were not immunogenic (Kilshaw and Sissons, 1979b). Steam heated and acid precipitated soybean meal, however, caused antibody production and digestive disturbances. IgG antibodies - specific for storage globulins (glycinin and B-conglycinin) and trypsin inhibitor - were not detected when using the extraction process. Due to the heat resistance to denaturation of both substances, antibodies were produced with steamed and acid precipitating processes, causing a gastrointestinal allergy. Antibody production could, therefore, be a way of predicting suitability of soybean meal products for nutritional purposes (Kilshaw and Sissons, 1979b).

Some areas of research have concentrated on reducing antigenicity of soybean meal by adding milk to buffer the diet in preruminant calves. Decreased weight gain, feed

efficiency, increased amounts of diarrhea, and villous atrophy, are associated with feeding 66% soybean meal and 34% milk. Trypsin inhibitor within the soybean meal reduced the amount of coagulated milk protein. Less protease action - pancreatic proteases and decreased hydrolytic activity of pepsin - was observed (Silva et al., 1986). However, adding 17.5% whole dried whey, has been shown to improve corn-soybean meal diets in newly weaned pigs. Gains and feed efficiency both were greatly improved. Decreasing the protein level itself to 14% for 10 to 14 d postweaning did not significantly affect performance or scouring (Kornegay et al., 1974).

Susceptibility of an animal to an antigen may be partially determined due to genetic differences. Certain strains of mice have been determined to be genetically deficient in synthesizing antibodies to antigens. These strains are inefficient in antigen elimination and have poor macrophage function. Differences may not only affect the immune system through antigen entry causing a subsequent immune response disorder; but, may reflect genetic differences in capacity for certain microflora in the gut associated with housing in colonies, since both the germ free and conventional mice are affected equally (Stokes et al., 1983a). Orally-induced and immune exclusion are inherited independently due to mucosal

permeability and nonimmune elimination influencing inductions (assuming that factors regulating them are in part not antigen-specific). The diversity in immune response capabilities could contribute to different vulnerability to food allergies in such physiological circumstances as artificial infant feeding (Stokes et al., 1983b).

Tolerance

Continued exposure to an antigen present in the lumenal tract may lead to a state of tolerance (Huntley et al., 1979), which is a failure of the immune system to respond to an antigen. This suppression of immune response (which may be temporary) usually results from a large inoculum of an antigen - now referred to as toleragen (Barrett, 1983). Pigs consuming small amounts of antigen (creep feed) prior to weaning are prime candidates for not achieving tolerance (Bourne et al., 1978).

A continuous challenge seems necessary to maintain the secretory antibody system and dietary inclusion offers a means of ensuring this challenge (Hanson et al., 1979b). Therefore, the pig immunizes itself every time it eats, thereby, saving the labor of individual dosing (Porter et al., 1973; Swarbrick et al., 1979). It has been shown

that feeding of certain proteins to mice may result in a form of tolerance, thus, it can be transferred via serum rather than lymphocytes (Kagnoff, 1978). Feeding of ovalbumin or other soluble proteins to adult C57B1/6 X DBA/2 (BDF_1) mice results in substantial dose-dependent tolerance expressed in IgE and other antibody responses. BDF_1 mice fed large amounts of ovalbumin showed a decrease in antibody response 21 d post-sensitization. Given 1 mg of ovalbumin per day (after receiving 5 mg) 84.0% of the mice became tolerant. After 20 mg, 92.6% were totally unresponsive (Miller and Hanson, 1979). Feeding of ovalbumin in subsequent trials for tolerance is characterized by loss of protein-specific T lymphocyte helper cells and associated with T lymphocyte suppressor cells. B lymphocytes specific for ovalbumin retain capacity to respond, although feeding of other proteins may induce tolerance in specific B lymphocytes of mice (Hanson, 1981). Antigen induced unresponsiveness (immunotolerance) is much easier accomplished in T lymphocyte cells and lasts for a long time even when induced by minute quantities of antigen considered "low-dose tolerance" (Barrett, 1983).

Other studies have investigated the possibility of the mechanism of suppressor mediation of the immune response. Kagnoff (1978) confirmed that serum-borne

factors induced by feeding sheep red blood cells (SRBC) to mice, could reduce anti-SRBC plaque forming cells; IgG, IgA and IgM were suppressed after 8 wk of feeding. The size of the suppressor factor and the ability to remove the suppressor activity from the serum by anti-mouse antibodies suggest that the suppression is mediated by antibodies. Thus, antigen feeding may provide a means for producing transferable factors that suppress humoral antibody responses.

Weaning represents the most profound period of diet change (Stokes et al., 1983a). However, more importantly when creep feeding, weaning interferes with the normal process of tolerance (Hanson, 1981). The consumption of small amounts of creep feed before weaning (smaller amounts consumed at younger ages) may be more of a problem than not creep feeding at all (Bourne, 1986). Weaning of pigs less than 3 wk old may reduce postweaning scours. Herd mortality due to severe scouring may actually be reduced from 4 to 9% to a loss of 1 to 3% (Miller et al., 1984b). At slaughter, there is no significant difference in weights between creep fed and noncreep-fed pigs. Therefore, an option in a production system where creep feeding is not working (such as in some early weaning systems) may be abrupt weaning (Miller et al., 1984a). The problem, however, of hypersensitivity or the promotion

of tolerance (depending upon the amount of feed consumed) may still exist if the pigs are allowed to consume remaining sow feed.

Summary

A combinations of factors may be involved with a postweaning performance check resulting from poor intakes and scours. Many management practices have been implemented for decreasing this problem including disinfecting the farrowing house facilities, immunizing the sow herd against various infections, assuring passive immunity transfer, and preparing the pig for its postweaning environment by providing the young pigs solid food in an appropriate manner prior to weaning. But, there is evidence to show that consumption of small amounts of feed preweaning may be more damaging to the gastrointestinal lining of the pig than not feeding anything at all when weaning before 3 wk of age. Changes in the gastrointestinal mucosa and bacteria flora associated with the transient immune response may lead to postweaning scouring, which provides a possible route of decreasing optimal performance as well as inviting disease from the added stress at weaning. While there are still some contradictions in the research pertaining to hypersensitivity, especially its direct mechanism, there appears to be evidence to corroborate this theory.

Further investigations should be made to determine the actual mode of this transient immune response and to what extent it is harmful or helpful in the consequences of the young weaned pig.

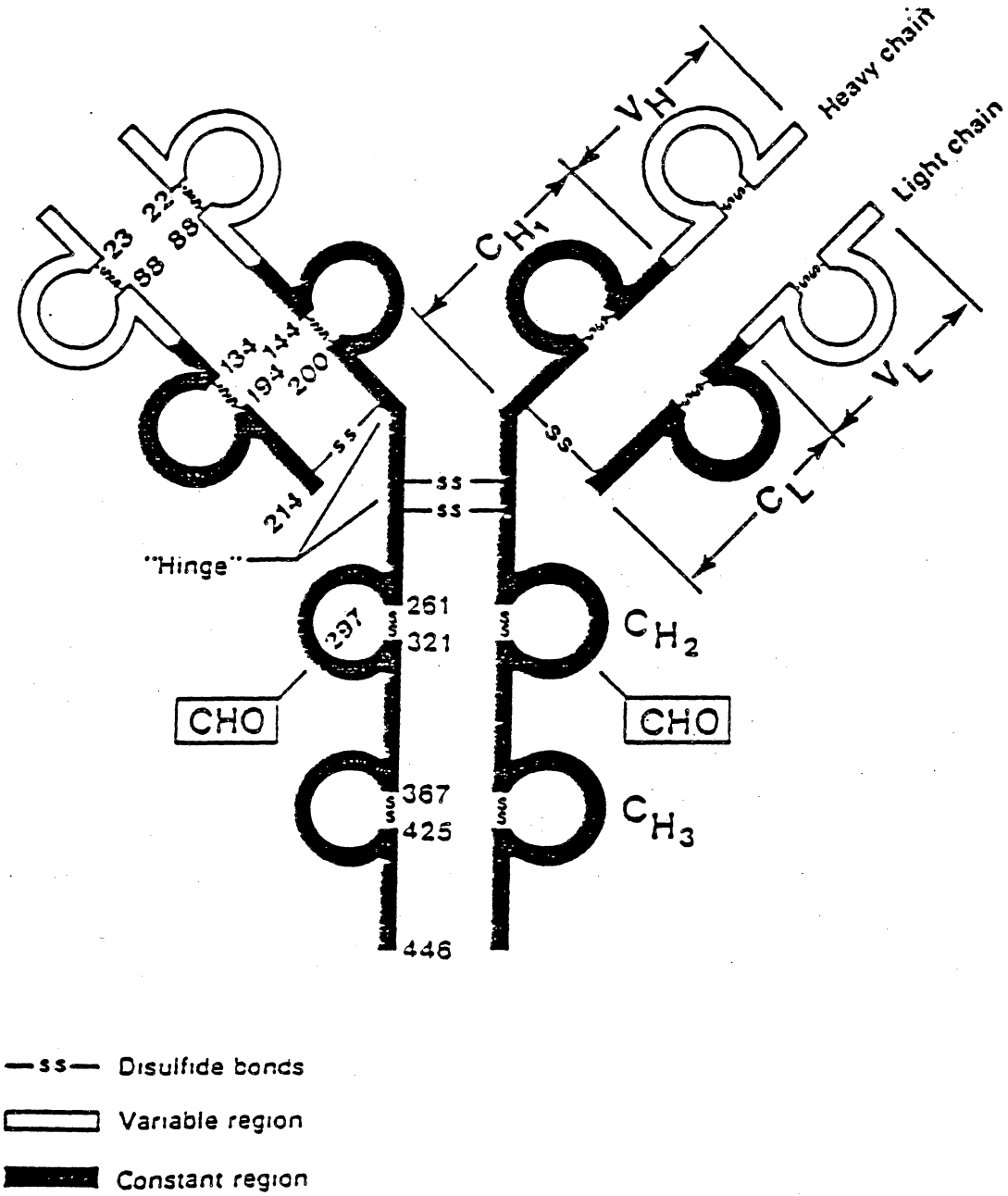

Figure 1. IgG molecule containing 6 domains with two light (L) chains and four heavy (H) chains and with intra-chain and interchain disulfide bonds.

CHAPTER III

OBJECTIVES

This study was conducted to examine the effects of creep feeding on postweaning performance of pigs.

Specific objectives were:

1) To characterize the pattern of creep feed consumption of nursing pigs.
2) To evaluate the immune response of pigs re-exposed during weaning to the same feed antigen (ovalbumin) introduced in the creep feed.
3) To determine the effect of creep feed consumption on postweaning scouring: incidence, severity, and duration.
4) To determine preweaning and postweaning performance of creep-fed versus noncreep-fed pigs.

CHAPTER IV

CHARACTERIZATION OF CREEP FEEDING AND ITS SUBSEQUENT EFFECTS ON IMMUNE RESPONSE, SCOURING INDEX, AND PERFORMANCE OF WEANLING PIGS

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ABSTRACT

Four trials were conducted to examine the pattern of creep feed consumption of nursing pigs and the effects of creep feeding - from 10 d to weaning at 28 d - a 20% crude protein corn-soybean meal (CS) whey diet and 1.0% chromic oxide (REG, 9 litters), or the same diet with 2.7% ovalbumin added as a dietary antigen (OVA, 14 litters), or no creep feed (CON, 11 litters) on the immune response scouring index, and subsequent performance of weanling pigs. At weaning, pigs were fed either a 20% CS diet, with or without 2.7% OVA. creep-fed litters began eating at 11 d of age and disappearance of creep feed increased linearly until weaning ($P < .01$). However, evidence suggested that creep feed consumption by individual pigs was quite variable (13 to 194 g). No differences were observed between creep-fed and CON litters in daily gain for the first 4 wk (.17 vs .19 kg) or in 4 wk body weights (6.7 vs 7.2 kg). Larger litters had lower 4 wk body weights ($P < .09$) and daily gains ($P < .09$), as well as

less feed disappearance per pig (P < .02). Weekly blood samples showed that pigs fed the OVA diet had a higher (P < .001) passive hemagglutination titer to OVA than did REG or CON at 14 d, 21 d, 28 d of age. At 56 d and 63 d, all pigs given an OVA injection (at 49 d 1 ml of 3 mg/ml OVA) responded (P < .001), with the lowest titers for REG pigs during nursery, intermediate titers for CON pigs during nursing, and the highest titers for OVA pigs. No scouring was observed in Trial 2, but in Trials 1 and 3, pigs began scouring 4 d postweaning (pw) with the highest frequency 5 to 8 d pw. Feed consumption although not statistically different was slightly higher (P = .18) during the first week pw for CON pigs and was observed throughout the study, but daily gain and feed efficiency was similar. Overall, there was no difference in daily gain, and FE.

Key words: Creep feed, immune response, scouring, performance.

INTRODUCTION

Baby pigs are given feed while still nursing (creep feeding) to provide additional energy and to improve the transition from milk to a solid feed at weaning. However, poor postweaning performance continues to be a major problem which has not been prevented by improved environmental conditions (Crenshaw et al., 1986; Gore et al., 1986), nutrition (Bark et al. 1986, Etheridge et al.,

1984) or through the use of feed additives (Holden, 1975).

It has been hypothesized that short exposure to certain feed ingredients (mainly antigenic proteins) in the creep feed can lead to the development of a transient dietary hypersensitivity. Exposure to the antigenic proteins preweaning may induce tolerance to these antigens. If the duration of exposure to creep feed is too short, or if the pig consumes less than 100 g of creep feed preweaning, the pig may not become tolerant to the antigen and may actually become sensitized. Thus, secondary exposure of the sensitized pigs to the dietary antigens at weaning may result in an immune response which damages the lining of the intestinal tract (Newby et al., 1985). This hypersensitivity is thought to be transient due to the apparent ability of the pig to recover from this phenomenon in less than 2 wk postweaning (Miller et al., 1983). The resulting postweaning scours can cause malabsorption and loss of electrolytes which, in conjunction with depressed appetite, result in very poor postweaning performance (Miller et al., 1984a; Etheridge et al., 1984).

The objectives of this study were to characterize the pattern of creep feed consumption of nursing pigs and to examine the effects of creep feeding on the immune response, scouring index, and postweaning performance of pigs weaned at an average of 28 d of age.

EXPERIMENTAL PROCEDURES

Four trials were conducted with nursing and weanling pigs to examine the effects of creep feeding a 20% crude protein corn-soybean meal whey diet (REG) or the same diet with 2.7% ovalbumin (OVA), or no creep feed (CON) on the immune response, scouring, and subsequent performance of weanling pigs (Table 1). Ovalbumin was used as a measurable dietary antigen assessing for the immune response. Hanson (1981) showed that ovalbumin was a viable dietary antigen in mice, thus it was used here because soybean meal contains many antigenic protein determinants that cannot be easily tested.

Prior to breeding, gilts and sows were vaccinated (4 wk and 2 wk for gilts and 2 wk for sows) using a 3-way vaccine[1] for *Bordetella bronchiseptica*, *Pasteurella multocida*, and Erysipelas, as well as vaccines for *Escherichia coli*[2] and parvovirus[3]. Also, sows and gilts were dewormed with dichlorvos[4] (mixed in the feed) 2 wk prior to the start of breeding.

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[1]Rhinovac 3 contains *Bordetella bronchiseptica*, *Erysipelothrix rhusiopathiae*, *Pasteurella multocida* bacterin - Norden Laboratories 85-4822-01, Lincoln, NE.

[2]Litter Guard contains *Escherichia coli* bacterin - Norden Laboratories 85-4400, Lincoln, NE.

[3]Leptopar - Parvovirus Vaccine contains a killed virus of Leptospira conicola, Grippotyphosa hardjo, *Icterohaemorrhagiae pomona* bacterin - Norden Laboratories - 7125-26, Lincoln, NE.

[4]Atgard C contains 9.6.5 dichlorvos fed as .4% of the diet - Fermenta, Animal Health Specialties - DSL 454-8408, Harrisonburg, VA.

Gestating gilts and sows were housed in individual stalls in a ventilation controlled, partially-slotted floor building. Gilts and sows were offered 2 to 3 kg of a 14% crude protein corn-soybean meal diet (Appendix Table 1) daily, until 1 wk prior to parturition. At this time, gilts and sows were washed thoroughly, brought into the farrowing house, and placed in individual farrowing crates where they were fed 2 to 3 kg twice daily until parturition.

After farrowing gilts and sows were fed as much as they would consume in 1 hr for two periods daily (consuming at their peak intake approximately 4.5 to 9 kg per d). Nursing pigs were denied access to sow feeders while sows were eating by placing the pigs in removable wire cages located on each side of the farrowing crate. Any excess feed remaining in the sow feeder after 1 hr was removed. Sows and nursing pigs were provided with water at all times from nipple or paddle type waterers. Pens containing nipple waters for baby pigs were also provided bowls of water to ensure adequate amounts of available water. Farrowing house temperature was maintained at 24 to 27 C with supplemental heat provided to the young pigs using heat lamps (infrared, 250 W, 125 V) placed approximately 55.8 cm above the floor on each side of the farrowing crate.

Day old pigs received .5 ml of penicillin[5] and 2 ml of iron dextran[6] at 3 d of age. At 7 d of age, pigs were castrated and received a 2 ml injection of atrophic rhinitis vaccine[7] which was repeated at 21 d. Pigs were dewormed at 14 d of age with 1 ml of fenbendazole[8] dosed orally, and sows were also dewormed at this time using top dressed fenbendazole at a dosage of 10 ml per 45 kg body weight.

Over the course of the entire study there were 16, 10 and 11 litters, respectively, for OVA, REG and CON used to examine hypersensitivity. However, in the individual trials, the number of litters varied for the three treatments. Trial 1 contained 5, 3 and 2 litters, Trial 2 contained 5, 1 and 3 litters, Trial 3 contained 2, 2 and 2, litters, while Trial 4 contained 4, 4 and 4 litters respectively, for OVA, REG, and CON diets. In Trial 2, by chance only one full litter was available from REG diets for the weanling phase because of poor mothering (pigs mashed) and poor milking ability of the sows randomly assigned as REG litters.

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[5]Benza-pen contains 150,000 units of Penicillin G benzathine and Penicillin G procaine in 1 ml of solution - Beecham Laboratories NDC 0029-6050-21, Bristol, TN.

[6]Iron Dehydrogenated Dextran supplies 100 mg/ml injection - Vedco, Inc. 862152, Overland Park, KS.

[7]Atrobac vaccine contains Bordetella bronchiseptica, Pasteurella multocida bacterin - Beecham Laboratories 7046-21, Bristol, TN.

[8]Panacur contains fenbendazole in a 10% solution supplying 100 mg/ml - Hoest-Roussel Co. NDC 12799-097-70, Somerville, NJ.

Pigs were offered the REG or CON creep diet containing 1.0% chromic oxide[9] as a consumption marker from 10 d until weaning at 28 d. During this time fecal samples were obtained twice daily using fecal loops for observation of green fecal material until weaning. No evidence of scouring was noted while the pigs were nursing. The age of the pig and the number of times that chromic oxide was first and subsequently observed, were recorded. Creep feed that was not consumed was weighed back, and consumption (disappearance) was calculated every 3 d.

At weaning, eight pigs from each litter were randomly chosen, with regard to sex and weight, and divided into two groups of four. Each group (pen) was then fed a 20% crude protein corn-soybean meal diet with or without 2.7% ovalbumin for the next two wk (Table 1). The CON diet with no ovalbumin was fed for the last two wk. Whey was omitted from the REG and CON starter diet as an added dietary challenge to the lining of the intestinal tract in addition to the physical challenge of changing from a liquid feed to a dry feed.

Weaned pigs were housed in .9 X 1.2 m cages with plastic coated welded wire flooring. Underneath the pens, a gravity drain type, flush system was used for waste removal. A stainless steel baffle separated the upper

[9]Chromium oxide, sesqui-, powder, Fisher Scientific Co. no. C334-500, Fairlawn, NJ.

pens from the lower pens to help prevent the passage of waste feed and water as well as feces and urine from the upper to the lower pens. Temperature was maintained at 32 C (87 F) for the first two wk and lowered 2 C (3 F) weekly afterwards. At 21 d postweaning (49 d of age) one pig in each nursery pen of 4 pigs was challenged with 1 ml of ovalbumin[10] solution (3 mg/ml) injected intraperitoneally.

Blood samples were obtained weekly from 7 to 63 d of age (during nursing and weanling phases) for determination of serum antibody titers to ovalbumin using a passive hemagglutination assay (Schurig et al, 1978). The blood was centrifuged (2000 X g) for 10 min. Serum stoppers were used for easy separation of serum from blood cells. The serum was stored at -20 C for later determination of antibody titers. In the antibody assay, ovalbumin was attached to sheep red blood cells (SRBC) via chromic chloric coating. Serial dilutions of serum were made to determine the antibody titers. The titer is indicated as the last well containing the antibody and is noted by a diffuse spread of SRBC along the bottom of the well (appendix table 2).

Postweaning scour scores were taken daily for the first 15 d. After this period, any scouring was assumed to be caused from something other than hypersensitivity. The following scour scoring system was used: 1 = hard

[10]Ovalbumin (GIII) - Sigma no. A5378, St. Louis, MO.

feces (seen only in farrowing barn), 2 = normal consistency of feces (firm but not hard - no scours), 3 = slightly loose (mild scours), 4 = loose and watery feces (severe scours), and 5 = watery feces (very severe scours). Pigs were weighed weekly and feed consumption was recorded for 35 d postweaning for determination of daily feed, daily gain, and feed to gain ratios to evaluate weanling pig performance.

Data was analyzed using the Statistical Analysis System (SAS, 1986). A general linear models procedure using repeated measures over time was used to analyze the individual trials as well as combined trials of titer data and scour scoring data. Because the repeated measures statement requires that only observations with complete values can be tested, titer data from Trial 2 was deleted due to the large number of missing values over the trial. Also, Trial 3 contained a very unusual situation in which none of the pigs scoured over any period of the 10 wk on test. Thus, since all scour scores were graded as 2, that trial was deleted from analysis. Performance data were analyzed using a General Linear Models F test, where all creep feed (OVA, REG and CON) treatments were tested with the error term containing litter due to known genetic differences among litters. Also, in this study the litters themselves were nested within the particular treatment because each creep diet could only be applied to

an entire litter rather than an individual pig, and each litter could only have received that diet within a particular trial. Therefore, error was tested with litter nested within treatment and trial.

RESULTS AND DISCUSSION

Preweaning

Creep feed consumption. Creep-fed litters began eating at 11 d of age and creep feed consumption increased linearly ($P < .01$) until weaning for all trials (Figure 1). Total litter creep feed consumption ranged from 107 to 1550 g and individual pig creep feed consumption from 13 to 194 g per pig for the 28-d period. Newby et al. (1985) reported an average creep feed consumption per pig of 107 g by 21 d of age (a much larger amount considering our weaning age of 28 d). However, in the last week of our experiment (21 d to 28 d of age) feed consumption increased dramatically possibly demonstrating that weaning later than 21 d of age allows for a longer exposure to creep feed and thus increases the probably of creep feed consumption; therefore, weaning at a young age, i.e. 21 d, may pose a bigger problem in the amount of creep feed consumed than weaning at 28 d of age. There were no differences ($P > .10$) in creep feed consumption of the two diets (OVA and REG) on a total feed per litter basis (670 vs 790 g $\pm$ 100) or individual feed per pig basis (82.7 vs 71.7 g $\pm$ 12.4).

Litter size was thought to have a direct influence on the amount of creep feed consumed. While total creep feed

consumption (424 vs 627 g) increased with larger litter size (6 to 13 pigs per litters), feed consumption per individual pig (80 vs 63 g) was less ($P < .01$) in the larger litters (Table 2). Thus, larger litters do not consume enough additional creep feed to compensate for the larger number of pigs in the litter. Trial differences existed in the amount of total feed consumed ($P < .05$); pigs in Trial 1 consumed ($P < .05$) less creep feed (310 vs 880 g) than did pigs in the other trials (Figure 1 and Appendix Figure 1). In Trials 2, 3, and 4 (Appendix Figures 2, 3 and 4 - respectively) pigs began eating at 11 d of age and creep feed consumption increased linearly until weaning; whereas in Trial 1, there was a lag time, in which feed consumption did not increase linearly until 20 d of age. Creep feed consumption in Trial 1, however, reached the same linear increase as the other trials but at a later date. The average number of pigs per litter in these trials also differed (Trial 1, n=6.8; Trial 2, n=9.7; Trial 3, n=8.3; and Trial 4, n=11.5); however, these differences in litter size did not compensate for the small amount consumed in Trial 1. Trial 1 was the only summer trial performed, results may reflect some seasonal effects.

Newby et al. (1985) did not report whether or not he accounted for pigs within a litter that may not have

consumed any creep feed preweaning. Feed intake, however, must be determined on a pen (litter) basis, and our results show that there are pigs within a litter which may not have consumed any creep feed. The presence and absence of chromic oxide in the feces indicates that pigs that had eaten may not continue to eat throughout the course of the trial, or that some pigs may have consumed very small amounts of feed in combination with an overabundance of milk which resulted in masking of the green color indicative of chromic oxide. In fact, because of the wide range of the number of pigs seen within a litter having green color in their feces, the data was subsequently analyzed for observation of chromic oxide on the following basis: 1) pigs observed having chromic oxide in their feces more than twice, 2) pigs observed with chromic oxide in the feces only one time, and 3) pigs in which chromic oxide was never observed in the feces. However, the analysis was not useful because of large variation of observation of chromic oxide and small numbers of litter involved.

Performance data. Body weight at 4 wk of age (Table 2, 6.7 vs 7.2 kg, $P < .15$) and cumulative average daily gain (.17 vs .19, $P < .30$) during the first 4 wk were similar for creep-fed and noncreep-fed pigs, and pig weight and weight gain did not affect the amount of creep

feed consumed. Generally, body weights and daily gain were not different (P > .10) between pigs fed OVA and REG creep diets.

Litter size affected preweaning performance (Table 2). Based on a linear regression, larger litters had lower birth weights (P < .05), lower 4 wk body weights (P < .09) and daily gains (P < .09), in addition to less feed disappearance per individual pig (P < .02).

Preweaning performance was similar in cumulative daily gain and 4 wk body weight of pigs classified as eaters and non-eaters (P < .20); but, 4 wk body weights favored the non-eaters (6.9 vs 7.3 kg). Some trial differences occurred, but no pattern was observed in these differences.

Immune response. Antibody titers to ovalbumin at 7 d of age (first blood sampling and before ovalbumin was fed) were high for all creep treatments (Table 3), suggesting nonspecific binding of serum antibodies to ovalbumin at one week of age. This binding may be have occurred partially due the IgG absorbed in colostrum at one day of age which may still be active at one week of age, and due to nonspecific antibodies, such as IgA, remaining in the milk at one week of age (Porter et al., 1973). Porter et al. (1973) also showed high antibody titers to different *E. coli* strains at 1 to 7 d of age, before pigs were

exposed to be the creep feed containing a specific E. coli strain. The high antibody titers were attributed to an increased passive immunity during this time period.

Antibody titers remained elevated over time ($P < .01$) for OVA fed pigs, but returned to a low base level for REG and CON pigs (Table 3) suggesting that an immune response (hypersensitivity) to ovalbumin. There were trial by treatment differences ($P < .001$); in Trial 4 (Appendix Table 3) OVA antibody titers remained elevated for OVA over time (3.97 vs 1.38 and 1.03). Some differences in the magnitude of the response for treatments may be partially attributed to differences in times that assays were performed (using different chemicals, solutions, and even the inactivation of complement can interfere with the antibody titers observed). In agreement, Kilshaw and Sissons (1979b) reported that antibody titers to soybean meal rose and were maintained 7 to 8 wk postweaning. Sustained titers were thought to have developed through IgA or suppressor T-cells which may cause a local immune response limiting absorption.

Postweaning

Immune response. After weaning, antibody titers increased linearly ($P < .04$) through d 49 with OVA creep-fed litters having the highest titers (Table 3). There

was also a trend for litters ($P < .07$) fed OVA after weaning to have higher titers up to 49 d of age.

Antibody titers behaved similarly in Trials 2 and 3 for REG and CON decreasing to almost 0 from 21 to 49 d of age, but in Trial 4 antibody titers were elevated during the same period; however, OVA remained as the highest titer (Appendix Table 3).

Pigs injected with ovalbumin intraperitoneally at d 49 had higher antibody titers than those not injected demonstrating an immune response at d 56 (3.34 vs 1.26) and d 63 (2.88 vs 1.11) (Appendix Table 4). Hanson (1981) also found subsequent titer responses in mice after priming mice orally and injecting with ovalbumin in a Dinitrophenyl (DNP) mixture. No response was noted, however, to DNP, thus showing antibodies were ovalbumin specific. In our study, all noninjected pigs had titers of less than 1.86 and remained low until 63 d of age (termination of the trial). There were some trial differences in antibody titers of injected pigs (Appendix Table 5). The lower immune response of pigs in Trial 2, may have been due to the fact that only ovalbumin in saline was injected; whereas, in the other trials Freund's incomplete adjuvant was added to the injection solution.

In Trial 4, antibody titers were higher for both injected and noninjected pigs, with injected pigs having

the highest levels ($P < .04$). There were litter differences in all cases ($P < .01$).

There were no interactions between creep and starter treatments. However, upon subsequent re-exposed to ovalbumin by injection, creep-fed pigs were able to mount a greater immune response (3.9 vs 2.8 and 3.4) than pigs not creep-fed ovalbumin ($P < .05$), demonstrating that pigs creep fed ovalbumin previously developed an immune response when young.

There were significant creep treatment by injection interactions over time; pigs fed ovalbumin previously had the highest antibody titers to injected ovalbumin, followed by CON pigs with REG pigs having the lowest titers (Appendix Table 6). To the contrary, Miller and Hanson (1979) found that stimulating the gut can lead to suppression of humoral and cell mediated responses upon subsequent parenteral antigen challenge. In studies where soybean meal was fed to preruminant calves, Silva et al. (1986) determined that some calves were more susceptible to a soybean meal immune response than others and that older animals may not react as severely as younger ones.

When pigs were first exposed to ovalbumin after weaning, the response to injected ovalbumin was not as great as that of pigs exposed at an earlier age (during nursing). No signs of anaphylactic shock were observed

after injection of ovalbumin suggesting that no IgE formation or type 1 hypersensitivity reactions occurred, contrary to reports by Kilshaw and Sissons (1979b) whose preruminant calves were sensitized 30 minutes after feeding.

Interactions of creep treatment, starter and injection were not significant ($P < .20$); however, pigs fed the OVA in creep and starter diets had the highest overall titers (4.3) compared to all other treatment combinations (3.5). Injected OVA creep pigs not given OVA in the nursery had titers similar to CON and REG pigs that had not received ovalbumin in the diet postweaning. There were trial differences; titers were highest in Trials 3 and 4 for OVA (creep) pigs injected with ovalbumin (6.5), with intermediate titers for REG (creep and starter treatments) and OVA (starter diet) pigs injected with ovalbumin (3.8). No trial by treatment interactions were noted.

Interaction of creep consumption and immune response. When titer data were analyzed by those pigs designated as EAT, NONE or CON, higher titers at 14 d of age were obtained (3.8) for pigs which were not observed eating (NONE) and lowest titers for noncreep-fed pigs (CON); pigs considered eaters (EAT) had intermediate titers (Table 4). CON remained low until 49 d of age. EAT titers decreased

steadily to 35 d of age where partial tolerance may be indicated. NONE pigs remained high up to 49 d and increased. Therefore, pigs considered non-eaters are the most sensitized pigs to ovalbumin as demonstrated by their high antibody titers, with control pigs showing not quite as strong of an immune response; and, pigs that are considered eaters may actually be showing some signs of immunological tolerance to ovalbumin.

Effects of pigs observed eating on subsequent nursery treatments showed interactions ($P < .04$) where NONE pigs given OVA had the highest titers (Table 5). CON pigs given REG in the nursery showed the lowest titers, and all other treatment combinations were intermediate.

When challenged with the OVA injection, pigs never seen to eat had the highest response, suggesting that NONE pigs are the pigs most likely to become hypersensitive to feed when challenged (Table 6), CON pigs had the second highest titers possibly showing a primary immune response since they were not creep-fed ovalbumin. EAT pigs had the lowest titers (3.09) demonstrating some possible tolerance. The antibody titers of all injected pigs decreased the second week post injection. Perhaps, when pigs start to eat solid food, gut damage occurs thus allowing some ovalbumin (antigen) to cross the gut and stimulate antibodies to form to the OVA. As the pig

adjusts to the feed, the gut recovers from the antigen and no longer responds to the antigen by d 28.

Scouring. The majority of pigs began scouring 4 to 5 d postweaning regardless of treatment (Figure 2). This lag period corresponds to the period when the absorption of whole molecule of ovalbumin absorption increases and xylose absorption decreases indicating damage to the gut lining as reported by Risley et al. (1987) 4 d postweaning. Scouring increased ($P < .01$), peaking at 10 d postweaning, and then decreasing at 15 d postweaning in the form of a quadratic curve ($P < .01$). After 15 d, pig scour scores returned to normal. Hampson et al. (1986a) observed that suckling pigs provided a creep diet containing *E. coli* (weaned at 21 d) scoured 5 to 15 d postweaning, whether the pigs consumed creep feed or did not consume creep feed preweaning. Perhaps, this phenomenon is a type 4 hypersensitivity bringing about increased mitogenic rate in enterocytes causing mild scours through decreased water absorption in the small intestine which can take place without the aid of bacteria (Newby et al., 1983).

Litter differences over time were evident ($P < .01$). As shown by Stokes et al. (1983b) different strains are genetically deficient in their ability to synthesize high affinity antibodies associated with susceptible antigens.

Some mice become tolerant after 30 mg of antigen ingested while others are tolerant after 2 and 20 mg. Other research groups have shown that flooding the system with an antigen (500 ug rather than .01 to 5 ug) of polysaccharide, may not develop immunity and quickly succumb to bacterial infection when challenged (Barrett, 1983). This phenomenon is called immunoparalysis and occurs when excess antigen is "sponged up" by circulating antibody as it is formed. Thus, there are differences in levels of antigens which may cause tolerance even among animals of the same species.

Time by creep treatment interactions were apparent ($P < .01$) for combined trials; creep-fed litters tended to scour more over time than non creep-fed litters. When divided into three 5-d periods (Appendix Table 7), means of starter treatments (OVA and REG) revealed that in period 2, OVA litters tended to scour more than REG litters but probably was not biologically significant due to the small differences in magnitude of the scour scores. There were trial differences ($P < .01$); in Trials 1 and 4 creep-fed litters tended to have higher scour scores than control litters (Appendix Figure 5 and 7). In Trial 3, no clear pattern was observed (Appendix Figure 6). When the data were analyzed according to the chromic oxide viewed

in the feces no significant differences were found in EAT, NONE and ONCE ($P < .73$).

Performance. Postweaning performance was not influenced ($P > .10$) by preweaning treatments (Table 7, Appendix Tables 8, 9, 10 and 11). Although daily feed intake for noncreep-fed pigs (CON) was highest during the first week postweaning (Table 7), it was not significantly different ($P = .18$). Daily gain and feed per gain ratios were not different. To the contrary, Porter et al. (1974) showed a dramatic difference in daily gain (.93 and .07 kg) and feed efficiency (1.09 vs. 10.69) one week postweaning (between antigen and control fed groups). Antigen fed pigs continued to gain faster than control pigs up to two week postweaning, but after three week, no differences in performance were noted. While Porter et al. (1974) did not perform slaughter checks, Bourne (1976) reported no differences in carcass quality of noncreep-fed vs. creep-fed animal. Kenworthy (1976) showed no feed intake one day postweaning and a decreased daily gain up to 8 d postweaning, leading to nutritional stress possibly allowing for reduced resistance and interference with antibody formation.

Litter differences occurred in all trials but diet by litter interactions were not observed. No trial by treatment interactions were noted. Performance in the

nursery was similar for pigs fed OVA or REG starter diets for combined trial data (Table 8); or for individual trial data (Appendix Tables 12, 13, 14 and 15 - respectively for Trials 1, 2, 3 and 4). Differences in performance of pigs considered eaters (EAT) or non-eaters (NONE) were not observed in Trials 1, 2 and 3 (Appendix Tables 16, 17 and 18 - respectively), but differences were noted in Trial 4 (Appendix Table 19); EAT pigs had a higher cumulative daily gain than NONE pigs. This difference is carried over in the combined trial data showing up for EAT pigs to have a slightly higher ($P < .17$) cumulative daily gain than NONE pigs (Appendix Table 20) but not statistical different.

CONCLUSIONS

In summary, creep feed disappearance began at 11 d of age and increased linearly until weaning with no effect on cumulative daily gain or body weight. While larger litters consume more total feed, smaller litters consumed more feed per pig. Our results suggest that creep feeding may affect the immune response and scouring indexes creep-fed pigs tend to have higher immune responses and more severe scouring may have been the result of intestinal gut damage postweaning. But there was little effect of creep feeding on preweaning or postweaning performance. The poor performing pigs, associated with creep feeding of pigs less than 28 d of age, may actually be the pigs within a litter which consume only very small amounts of feed prior to weaning; however, there is no evidence of differences in postweaning performance (daily gain or final weight) or in postweaning scouring data.

Other possibilities for further research not examined by this study would be to change diets so as to reduce antigen levels in the diets to change brush border less dramatically (Hampson, 1986a). Also, offering solid food in an appropriate manner postweaning so as feed may be kept clean and fresh can reduce the severity of diarrhea or prevent its onset altogether (Newby et al., 1983). Antibiotics can be put in the feed to control E. coli but

they do not reduce the primary gut damage and its effect on performance (Bourne et al., 1978). The management practice of removing the sow from the litter (rather than moving the litter) may decrease postweaning stress as well as other management practices of keeping pigs on the same food postweaning as preweaning and keeping litters weaned as a group (Stevens, 1963b). Abrupt weaning causes increased inflammatory response and reduction in plasma cell populations by 7 to 8 d postweaning with no severe scours and abnormal proliferation by intestinal coliform bacteria (Kenworthy, 1976).

TABLE 1. PERCENTAGE COMPOSITION OF CREEP AND STARTER DIETS[a]

Ingredients	Creep	Starter
Ground corn	55.61	66.90
Soybean meal (48% CP)	27.04	29.50
Dried whole whey	15.00	
Defluorinated phosphate	.93	
Dicalcium phosphate		1.40
Limestone	.77	1.00
Salt	.30	.40
Vitamin premix[b]	.25	.50
Trace mineral premix[c]	.10	.05
ASP-250d		.20
Selenium premix[e]		.05
Chromic oxide[f]	1.00	
Ovalbumin (GII)[g]	2.70	2.70

[a]Calculated to contain 20% crude protein, 0.8% Ca and 0.6% P.

[b]Supplied per kg of vitamin premix: 1,760,000 ICU vitamin A, 176,000 IC vitamin D, 4,400 IU vitamin E, 1760 mg riboflavin, 8,800 mg d-pantothenic acid, 8,800 mg niacin, 195,800 mg choline, 8.8 mg vitamin B_{12}, 199.76 mg menadione, 176 mg d-biotin, 40 mg selenium.

[c]Contains per kg of premix: 15.0% Zn, 6.0% Mn, 17.5% Fe, 1.75% Cu, 0.2% I.

[d]Contains per kg: 44 g chlortetracycline, 22 g procaine penicillin, 4.4% sulfamethazine.

[e]Calculated to contain 0.3 mg/kg Se (Na_2SeO_3 contains 46% active Se).

[f]Chromic oxide (Fisher no. C334-500) was used as a creep feed consumption indicator.

[g]Ovalbumin (Grade II Sigma no. A5376) was added at a level of 2.7% to the basal mix of OVA diets and was omitted from REG diets. The protein level was not adjusted.

TABLE 2. EFFECT OF LITTER SIZE ON PREWEANING PERFORMANCE AND CREEP CONSUMPTION

Litter size	Birth wt[b]	Daily gain[c]	Body wt[c]	Feed Consumed per litter[b]	Feed Consumed per pig[b]
	kg			g	
6 (3)[a]	1.78	.20	6.4	424	80
7 (2)	1.76	.19	7.0	493	70
8 (3)	1.98	.20	7.6	786	98
9 (3)	1.81	.20	7.7	617	69
10 (4)	1.56	.18	6.2	841	84
11 (3)	1.50	.18	6.1	827	83
12 (3)	1.35	.18	6.5	725	73
13 (2)	1.63	.19	6.7	627	63

[a]Number of litters in parenthesis.
[b]Linear regression of litter size effect ($P < .05$).
[c]Linear regression of litter size effect at 4 wk of age, ($P < .09$).

TABLE 3. EFFECT OF PREVIOUS CREEP TREATMENT (OVA, REG AND CON) AND STARTER TREATMENTS (OVA AND REG) ON POSTWEANING ANTIBODY TITER RESPONSE[a] TO OVALBUMIN (ALL TRIALS)

	Creep Treatment[b]			Starter Treatment[c]	
Age[de]	OVA	REG	CON	OVA	REG
Day 7	3.48	1.72	2.38	---	---
14	2.54	.84	.82	---	---
21	2.28	.73	.52	---	---
28	1.98	.52	.37	---	---
35	1.46	.61	.70	1.03	.82
42	1.67	.92	.99	1.39	.99
49	1.86	1.29	.94	1.58	1.15
56	2.89	1.92	2.09	2.46	2.15
63	2.57	1.50	1.90	2.09	1.89

[a]Converted to log base 2.
[b]Creep treatment effect over d ($P < .04$).
[c]Starter treatment effect through d 49 ($P < .07$); d 56 and 63 ($P < .01$).
[d]Weaned at d 28.
[e]Injected d 49 with 1 ml ovalbumin solution (3 mg/ml).

TABLE 4. ANTIBODY RESPONSE[a] OF PIGS DESIGNATED AS EATERS (EAT), NON-EATERS (NONE) OR UNEXPOSED TO CREEP FEED (CON) BEFORE WEANING (ALL TRIALS)

	Observation of Chromic Oxide[b]		
Age[cd]	EAT (n=86)	NONE (n=47)	CON (n=72)
Day 7	2.64	3.11	2.38
14	1.80	2.09	.82
21	1.82	1.76	.52
28	1.23	1.66	.37
35	.97	1.41	.70
42	1.40	1.63	.94
49	1.65	1.80	.94
56	2.27	2.88	2.10
63	2.02	2.41	1.90

[a]Converted to log base 2.
[b]Chromic oxide appearance effect ($P < .04$).
[c]Weaned at d 28.
[d]Injected d 49 with 1 ml ovalbumin solution (3 mg/ml).

TABLE 5. ANTIBODY RESPONSE OF PIGS DESIGNATED AS EATERS (EAT), NON-EATERS (NONE) OR UNEXPOSED TO CREEP FEED (CON) BEFORE WEANING WHEN PROVIDED STARTER DIETS WITH (OVA) AND WITHOUT OVALBUMIN (REG) (ALL TRIALS)[a]

	Observation of Chromic Oxide[b]					
	EAT		NONE		CON	
Age[de]	OVA[c] (n=50)	REG[c] (n=36)	OVA (n=19)	REG (n=28)	OVA (n=36)	REG (n=36)
Day 35	.87	1.07	1.88	.96	.86	.53
42	1.38	1.43	2.13	1.13	1.09	.88
49	1.69	1.61	2.19	1.40	1.11	.76
56	2.39	2.15	3.28	2.48	2.21	1.98
63	2.01	2.01	2.77	2.05	1.95	1.85

[a]Converted to log base 2.
[b]Chromic oxide appearance in the feces effect ($P < .04$).
[c]Starter treatments in the nursery.
[d]Weaned at d 28.
[e]Injected d 49 with 1 ml ovalbumin solution (3 mg/ml).

TABLE 6. ANTIBODY RESPONSE[a] OF PIGS DESIGNATED AS EATERS (EAT), NON-EATERS (NONE) OR UNEXPOSED TO CREEP FEED (CON) BEFORE WEANING AFTER OVALBUMIN INJECTION AT DAY 49 (ALL TRIALS)

	Observation of Chromic Oxide[b]					
	EAT		NONE		CON	
Age[d]	NIJ[c] (n=50)	INJ[c] (n=36)	NIJ (n=19)	INJ (n=28)	NIJ (n=36)	INJ (n=36)
Day 56	1.45	3.09	1.50	4.24	.84	3.34
63	1.35	2.67	1.19	3.62	.77	3.02

[a]Converted to log base 2.
[b]Chromic oxide appearance in feces effect ($P < .04$).
[c]Injection treatments: NIJ=not injected, INJ=injected with 1 ml ovalbumin solution (3 mg/ml).
[d]Weaned at d 28.

TABLE 7. POSTWEANING PERFORMANCE OF SUCKLING PIGS PROVIDED CREEP FEED WITH OVALBUMIN (OVA), WITHOUT OVALBUMIN (REG), OR UNEXPOSED TO CREEP FEED (CON) (ALL TRIALS)

	Creep Treatment				
	OVA	REG	CON	±SE	P > F
Avg weight (kg)					
Week 1	8.2	7.6	8.5	.24	.20
2	9.4	8.8	10.0	.31	.20
3	11.9	11.0	12.6	.43	.22
4	15.4	14.6	16.1	.44	.30
5	19.3	18.7	20.4	.54	.35
Avg daily feed intake (kg)					
Week 1	.16	.16	.19	.03	.18
2	.29	.29	.31	.03	.92
3	.60	.55	.62	.05	.29
4	.74	.72	.74	.08	.82
5	1.07	1.04	1.09	.08	.74
1-2	.24	.23	.25	.02	.56
1-3	.36	.34	.38	.05	.37
1-4	.46	.44	.48	.03	.48
1-5	.58	.56	.60	.03	.48
Avg daily gain (kg)					
Week 1	.20	.18	.16	.03	.64
2	.22	.20	.21	.04	.59
3	.36	.32	.37	.05	.57
4	.48	.49	.46	.06	.96
5	.59	.62	.64	.05	.52
1-2	.18	.19	.19	.01	.56
1-3	.25	.23	.25	.02	.75
1-4	.30	.29	.31	.02	.84
1-5	.36	.36	.37	.01	.72
Feed/gain					
Week 1	1.11	1.45	1.67	.95	.59
2	1.48	1.52	1.78	.20	.54
3	1.78	1.76	1.68	.15	.84
4	1.61	1.49	1.57	.11	.66
5	2.07	1.74	1.83	.11	.23
1-2	1.54	1.31	1.43	.13	.52
1-3	1.54	1.47	1.55	.06	.66
1-4	1.53	1.46	1.53	.05	.24
1-5	1.63	1.54	1.61	.04	.49

TABLE 8. POSTWEANING PERFORMANCE OF PIGS PROVIDED STARTER FEED WITH OVALBUMIN (OVA), OR WITHOUT OVALBUMIN (REG)(ALL TRIALS)

	Starter Treatment			
	OVA	REG	±SE	P > F
Avg weight (kg)				
Week 1	17.7	17.9	.20	.46
2	20.6	20.8	.26	.77
3	25.8	26.5	.36	.22
4	33.5	34.0	.40	.30
5	43.0	42.9	.46	.77
Avg daily feed intake (kg)				
Week 1	.18	.17	.02	.59
2	.29	.31	.03	.37
3	.59	.59	.04	.89
4	.75	.72	.07	.45
5	1.06	1.07	.07	.75
1-2	.23	.25	.02	.24
1-3	.36	.36	.02	.58
1-4	.46	.45	.02	.72
1-5	.58	.57	.07	.86
Avg daily gain (kg)				
Week 1	.18	.18	.03	.73
2	.20	.19	.03	.73
3	.33	.37	.04	.12
4	.49	.48	.07	.69
5	.64	.59	.05	.09
1-2	.19	.18	.01	.35
1-3	.24	.25	.02	.41
1-4	.30	.31	.01	.69
1-5	.37	.36	.01	.33
Feed/gain				
Week 1	1.16	.98	.80	.88
2	1.40	1.79	.15	.11
3	1.83	1.67	.12	.89
4	1.55	1.56	.09	.92
5	1.77	1.99	.09	.14
1-2	1.38	1.47	.11	.63
1-3	1.52	1.53	.05	.89
1-4	1.53	1.49	.04	.53
1-5	1.58	1.60	.03	.68

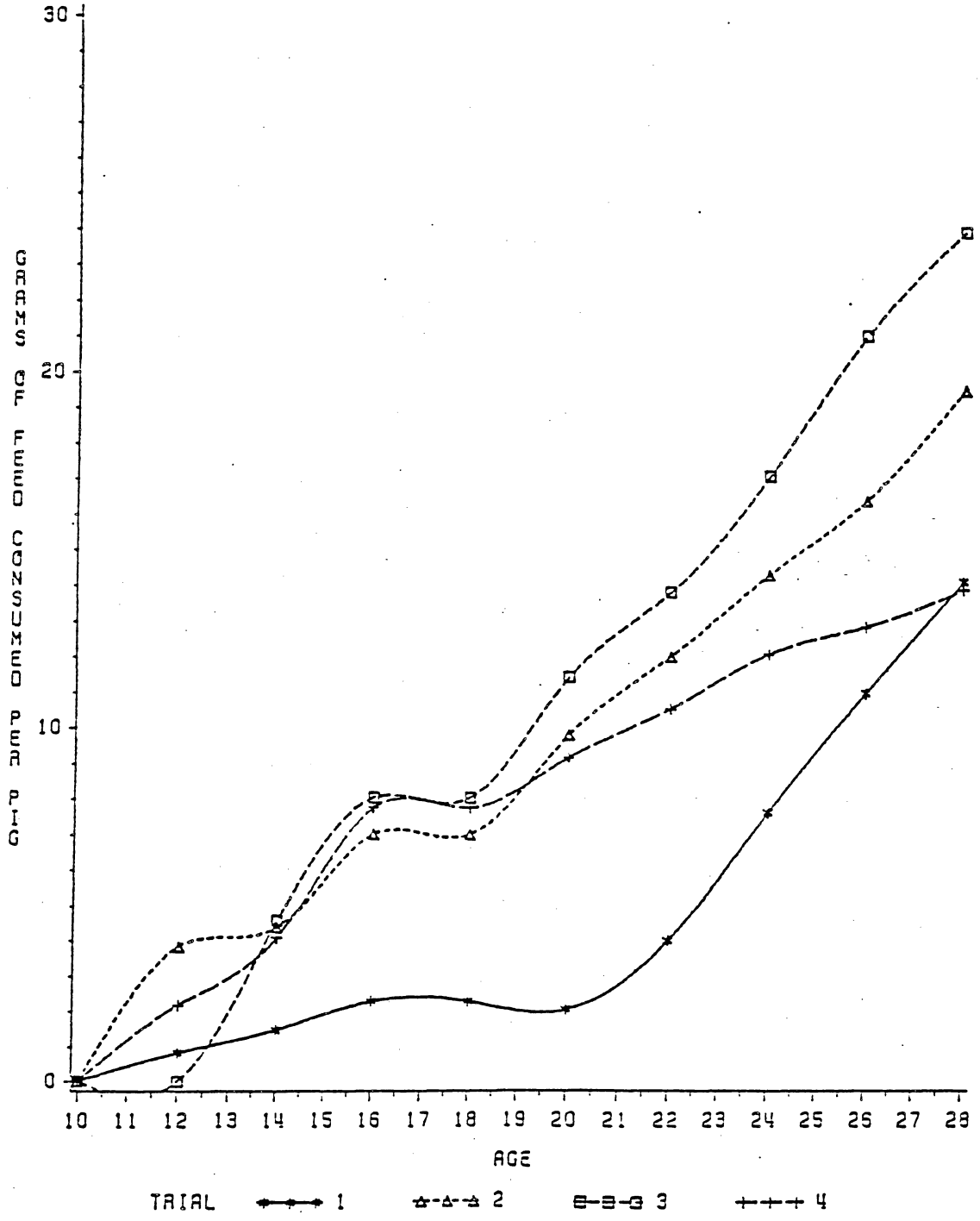

Figure 1. Daily feed consumption (OVA and REG creep diets combined) per pig increasing linearly ($P < .01$). All Trials combined.

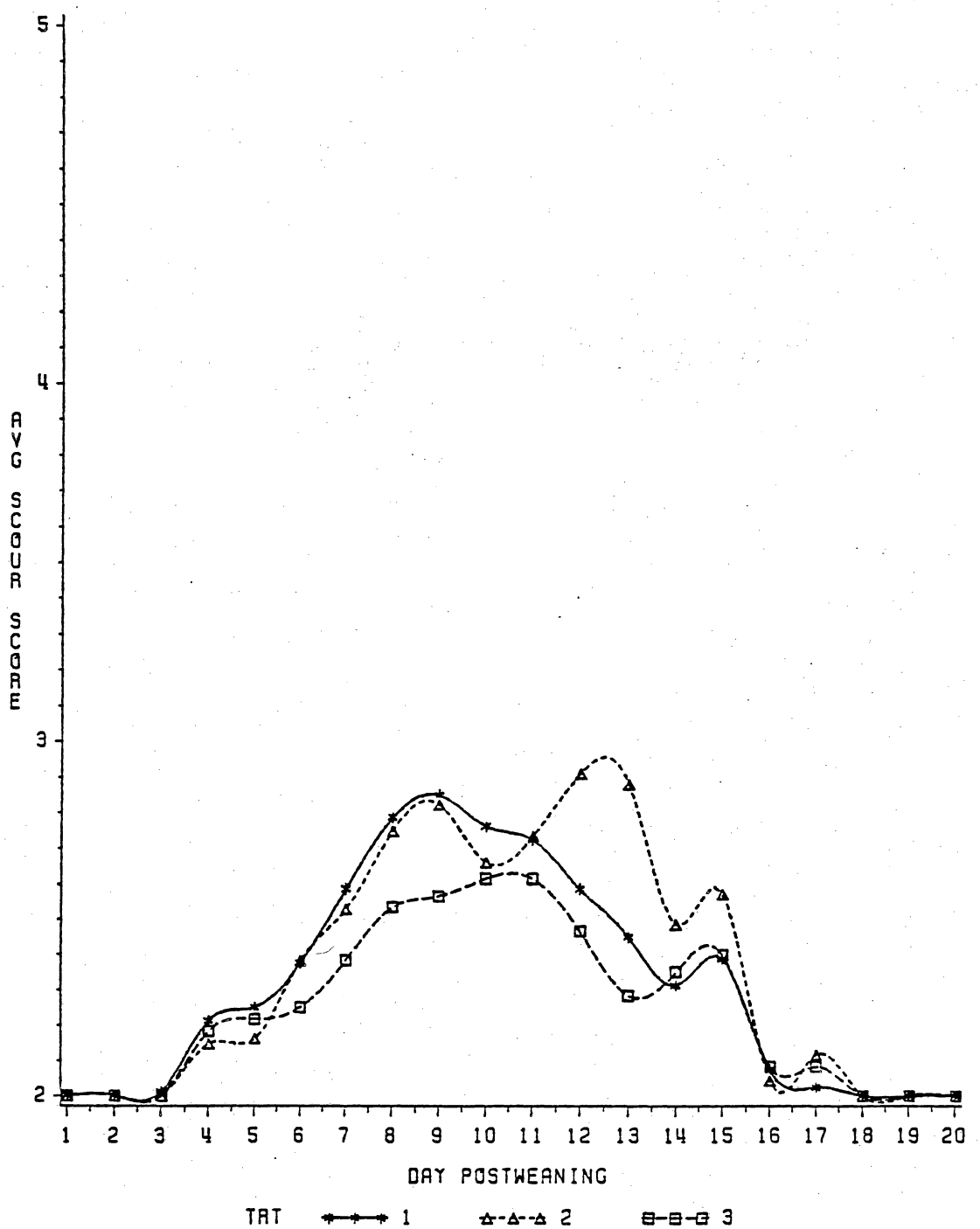

Figure 2. Postweaning scour scores (2=normal, 5=very severe scours) as influenced by previous creep treatment (1=OVA creep diet, 2=REG creep diet, 3=CON noncreep-fed). All Trials combined.

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APPENDIX TABLE 1. COMPOSITION OF SOW RATION[a]

Ingredients	%
Ground corn	81.64
Soybean meal (44% CP)	15.60
Defluorinated phosphate	1.57
Limestone	.64
Salt	.25
Vitamin-selenium premix[b]	.25
Trace mineral premix[c]	.05

[a]Calculated to contain 14.0% crude protein, 0.8% Ca and 0.6% P.
[b]Supplied per kg of premix: 1,763,698 IU vitamin A, 176,370 IU vitamin D3, 4,409 IU vitamin E, 441 mg vitamin K, 1,764 mg riboflavin, 8,818 pantothenic acid, 8,818 mg niacin, 8,818 ug vitamin B12, 176 g choline, 176 mg biotin, and 40.1 mg selenium.
[c]Contained 20% Zn, 10% Fe, 5.5% Mn, 1.1% Cu and 0.15% I.

APPENDIX TABLE 2. PASSIVE HEMAGGLUTINATION ASSAY FOR DETECTION OF ANTIBODIES TO OVALBUMIN (Schurig et al. 1978)

Reagents

1. Physiological saline - 0.9% NaCl solution in autoclaved deionized water.

2. Chromium (III) Chloride hexahydrate (CrCl) - Sigma no. C-1896.

3. Packed Sheep Red Blood Cells (SRBC) - sheep red blood cells were collected in 10 ml heparinized vacutainer tubes and centrifuged for 10 minutes at 2000 X g. The clear serum layer was syphoned off top, leaving the "packed" red blood cells.

4. Microtiter plates containing 96 well "U" bottom - Dynatech lab

5. Ovalbumin (grade III) Sigma no. A-5378.

Procedure

A. Chromic Chloride coating of SRBC

   1. Prepare a 0.1% CrCl solution in physiological saline.

   2. Wash 5 ml of packed SRBC by mixing with 5 ml of physiological saline. Centrifuge for 10 minutes at 2000 X g. Syphon off the clear liquid layer (saline). Repeat procedure three times.

   3. Prepare a 1.0% ovalbumin solution (10 mg ovalbumin per ml of physiological saline).

   4. Mix at the same time:
      a. 10 ml of packed SRBC
      b. 10 ml of 0.1% CrCl solution
      c. 10 ml of ovalbumin (antigen) dissolved in physiological saline

   3. Set for 4 minutes at room temperature.

4. Then wash cells with 40 to 50 volumes of physiological saline.

5. Store in refrigerator for up to one week.

B. Hemagglutination titers

1. Add 100 ul of heat inactivated serum (56 C for 30 minutes) in first well of each column on the 96 "U" bottom well microtiter plate. Each serum sample is placed in a different well.

2. Add 50 ul of physiological saline to each of the remaining 84 wells.

3. Serial dilutions were made along one axis of the plate using a Costar octapette making dilutions of 1:1 to 1:4,096.

4. Make all serial dilutions, then add 50 ul of treated SRBC (linked to ovalbumin) to each well.

5. Allow plates to sit for 2 hr at room temperature.

6. Then observe plates to note a positive reaction which is indicated by a diffuse spreading of the SRBC on the bottom of the well (shield). A negative result is indicated by the formation of a tight button of red blood cells on the bottom of the well. The reciprocal of the last dilution at which the "shield" is seen is defined as the agglutination titer of the test.

7. Make further dilutions for testing if no "buttons" are observed at the end of a serial dilution.

APPENDIX TABLE 3. EFFECT OF PREVIOUS CREEP TREATMENT (OVA, REG and CON) and STARTER TREATMENTS (OVA and REG) ON POSTWEANING ANTIBODY TITER RESPONSE[ab] TO OVALBUMIN (BY INDIVIDUAL TRIALS)

	Creep Treatment			Starter Treatment	
Age[cd]	OVA	REG	CON	OVA	REG
Trial 2					
Day 7	3.86	1.58	3.00	---	---
14	2.55	.33	.79	---	---
21	2.06	.50	.33	---	---
28	1.41	.00	.08	---	---
35	.28	.00	.38	.20	.24
42	.44	.00	.33	.26	.26
49	.80	.56	.38	.42	.74
56	1.25	1.17	.86	.91	1.27
63	1.42	.54	1.08	.95	1.08
Trial 3					
Day 7	4.38	2.44	2.31	---	---
14	3.44	1.38	.94	---	---
21	2.19	.56	.13	---	---
28	.56	.19	.00	---	---
35	.13	.00	.00	.08	.00
42	.13	.00	.00	.00	.08
49	.00	.25	.00	.17	.00
56	2.79	1.50	2.67	2.50	2.14
63	2.29	1.13	1.92	1.92	1.64
Trial 4					
Day 7	2.22	1.13	1.81	---	---
14	1.63	.81	.72	---	---
21	2.59	1.13	1.09	---	---
28	3.97	1.38	1.03	---	---
35	3.97	1.84	1.72	2.81	2.21
42	4.44	2.75	2.63	3.92	2.63
49	4.78	3.06	2.44	4.15	2.71
56	4.56	3.21	2.83	4.06	3.07
63	4.04	2.83	2.71	3.47	2.92

[a]Converted to log base 2; effect of creep and starter treatments on immune response over time ($P < .05$).
[b]Adjusted for differences in litters using least square means.
[c]Weaned at d 28.
[d]Injected d 49 with 1 ml ovalbumin solution (3 mg/ml).

APPENDIX TABLE 4. EFFECT OF INJECTION TREATMENT ON ANTIBODY TITER RESPONSE[ab] OF WEANLING PIGS (TRIALS 2, 3 and 4)

Age[c]	Not Injected	Injected
Day 56	1.26	3.34
Day 63	1.11	2.88

[a]Converted to log base 2; effect of injection treatment on antibody titer response over time ($P < .001$).
[b]Adjusted for differences in litters using least square means.
[c]Injected d 49 with 1 ml ovalbumin solution (3 mg/ml).

APPENDIX TABLE 5. EFFECT OF INJECTION TREATMENT ON ANTIBODY TITER RESPONSE[ab] OF WEANLING PIGS (ALL TRIALS COMBINED)

	Not Injected	Injected[c]
Trial 2[d]		
Day 56	.33	1.85
Day 63	.53	1.49
Trial 3[e]		
Day 56	.38	4.25
Day 63	.22	3.33
Trial 4[e]		
Day 56	3.00	4.13
Day 63	2.56	3.88

[a]Converted to log base 2; effect of injection treatment over time ($P < .001$).
[b]Adjusted for differences in litters using least square means.
[c]Injected d 49 with 1 ml ovalbumin solution (3 mg/ml).
[d]Carrier for ovalbumin was physiological saline.
[e]Carrier for ovalbumin was Freund's incomplete adjuvant.

APPENDIX TABLE 6. EFFECT OF PREVIOUS CREEP TREATMENT ON ANTIBODY TITER RESPONSE[ab] AFTER INJECTION TREATMENT (ALL TRIALS COMBINED)

	Creep treatments					
	OVA		REG		CON	
Age[c]	NIJ[d]	INJ[d]	NIJ	INJ	NIJ	INJ
Day 56	1.86	3.91	1.08	2.77	.86	3.35
63	1.62	3.52	.91	2.09	.78	3.00

[a]Converted to log base 2; interactions of creep and injection treatments on antibody titer response ($P < .001$).
[b]Adjusted for differences in litters using least square means.
[c]Weaned at d 28.
[d]Injection treatments: NIJ=not injected, INJ=injected at d 49 with 1 ml ovalbumin solution (3 mg/ml).

APPENDIX TABLE 7. EFFECT OF STARTER TREATMENTS OF WEANLING PIGS ON SCOURING AT 5-DAY INTERVALS POSTWEANING (ALL TRIALS COMBINED)

	Periods[a]			
Starter Treatment	1[b]	2[c]	3[d]	4
OVA	2.10	2.65	2.57	2.00
REG	2.08	2.55	2.53	2.00

[a]5-d intervals postweaning.
[b]Effect of starter treatment on scouring index 1 to 5 d postweaning ($P < .20$).
[c]Effect of starter treatment on scouring index 6 to 10 d postweaning ($P < .10$).
[d]Effect of starter treatment on scouring index 11 to 15 d postweaning ($P < .53$).

APPENDIX TABLE 8. POSTWEANING PERFORMANCE[a] OF SUCKLING PIGS PROVIDED CREEP FEED WITH OVALBUMIN (OVA), WITHOUT OVALBUMIN (REG), OR UNEXPOSED TO CREEP FEED (CON) (TRIAL 1)

	Creep Treatment		
	OVA	REG	CON
Avg weight (kg)			
Week 1	8.6	8.3	8.6
2	9.8	9.9	10.1
3	12.2	12.3	13.2
4	15.8	16.0	15.9
5	20.8	21.0	21.6
Avg daily feed intake (kg)			
Week 1	.19	.21	.20
2	.32	.37	.36
3	.63	.60	.64
4	.79	.78	.67
5	.99	.96	1.01
1-2	.25	.29	.28
1-3	.38	.39	.40
1-4	.48	.49	.47
1-5	.58	.59	.58
Avg daily gain (kg)			
Week 1	.20	.16	.15
2	.17	.24	.20
3	.34	.34	.45
4	.52	.52	.38
5	.71	.71	.83
1-2	.19	.20	.18
1-3	.24	.24	.27
1-4	.31	.31	.29
1-5	.39	.39	.40
Feed/gain ratio			
Week 1	1.23	1.40	1.35
2	.98	1.57	1.86
3	1.91	1.92	1.57
4	1.56	1.51	2.00
5	1.45	1.34	1.28
1-2	1.34	1.49	1.63
1-3	1.59	1.65	1.56
1-4	1.56	1.57	1.58
1-5	1.51	1.48	1.45

[a]Means of pen performance data.

APPENDIX TABLE 9. POSTWEANING PERFORMANCE[a] OF SUCKLING PIGS PROVIDED CREEP FEED WITH OVALBUMIN (OVA), WITHOUT OVALBUMIN (REG), OR UNEXPOSED TO CREEP FEED (CON) (TRIAL 2)

	Creep Treatment		
	OVA	REG	CON
Avg weight (kg)			
Week 1	9.3	7.6	8.4
2	11.0	9.3	10.1
3	13.7	11.7	13.4
4	18.5	16.4	17.9
5	23.1	21.1	22.1
Avg daily feed intake (kg)			
Week 1	.16	.17	.16
2	.40	.41	.31
3	.72	.67	.73
4	1.01	.97	.97
5	1.32	1.31	1.22
1-2	.28	.29	.24
1-3	.43	.42	.40
1-4	.57	.56	.54
1-5	.72	.71	.68
Avg daily gain (kg)			
Week 1	.25	.24	.17
2	.24	.24	.24
3	.40	.34	.47
4	.68	.68	.64
5	.65	.67	.61
1-2	.25	.24	.20
1-3	.30	.28	.29
1-4	.39	.38	.38
1-5	.44	.44	.43
Feed/gain			
Week 1	.70	.72	1.17
2	1.91	1.71	1.41
3	2.12	2.01	1.56
4	1.63	1.53	1.54
5	2.03	1.96	2.02
1-2	1.13	1.18	1.20
1-3	1.46	1.52	1.38
1-4	1.47	1.49	1.43
1-5	1.63	1.63	1.60

[a]Means of pen performance data.

APPENDIX TABLE 10. POSTWEANING PERFORMANCE[a] OF SUCKLING PIGS PROVIDED CREEP FEED WITH OVALBUMIN (OVA), WITHOUT OVALBUMIN (REG), OR UNEXPOSED TO CREEP FEED (CON) (TRIAL 3).

	Creep Treatment		
	OVA	REG	CON
Avg weight (kg)			
Week 1	7.5	6.2	9.5
2	8.4	6.9	10.8
3	10.8	8.5	13.2
4	13.3	10.8	15.7
5	15.9	13.6	18.6
Avg daily feed intake (kg)			
Week 1	.13	.10	.22
2	.21	.16	.35
3	.45	.35	.57
4	.54	.39	.62
5	.92	.75	.96
1-2	.17	.14	.30
1-3	.29	.50	.41
1-4	.36	.28	.47
1-5	.49	.38	.58
Avg daily gain (kg)			
Week 1	.25	.27	.26
2	.15	.11	.20
3	.34	.24	.35
4	.36	.32	.36
5	.37	.41	.42
1-2	.19	.20	.18
1-3	.24	.24	.27
1-4	.31	.31	.29
1-5	.39	.39	.40
Feed/gain			
Week 1	.53	.38	.86
2	1.40	1.46	2.71
3	1.36	1.50	1.61
4	1.50	1.25	1.74
5	3.11	1.96	2.26
1-2	.92	.78	1.32
1-3	1.15	1.12	1.47
1-4	1.28	1.16	1.53
1-5	1.63	1.41	1.74

[a]Means of pen performance data.

APPENDIX TABLE 11. POSTWEANING PERFORMANCE[a] OF SUCKLING PIGS PROVIDED CREEP FEED WITH OVALBUMIN (OVA), WITHOUT OVALBUMIN (REG), OR UNEXPOSED TO CREEP FEED (CON) (TRIAL 4)

	Creep Treatment		
	OVA	REG	CON
Avg weight (kg)			
Week 1	7.2	8.0	7.8
2	8.4	9.4	9.3
3	10.9	12.2	11.9
4	13.8	15.8	15.7
5	17.7	20.0	19.7
Avg daily feed intake (kg)			
Week 1	.19	.16	.20
2	.27	.26	.26
3	.59	.59	.64
4	.64	.76	.75
5	1.05	1.17	1.15
1-2	.23	.21	.23
1-3	.35	.34	.37
1-4	.43	.46	.47
1-5	.54	.59	.59
Avg daily gain (kg)			
Week 1	.05	.06	.07
2	.17	.43	.20
3	.35	.41	.38
4	.37	.45	.48
5	.64	.70	.65
1-2	.11	.13	.14
1-3	.19	.22	.22
1-4	.23	.29	.29
1-5	.31	.36	.35
Feed/gain			
Week 1	2.03	-.78	3.32
2	1.74	1.40	1.29
3	1.76	1.46	1.79
4	1.74	1.74	1.59
5	2.30	1.66	2.53
1-2	2.75	.79	1.66
1-3	1.97	1.54	1.71
1-4	1.82	1.60	1.63
1-5	1.76	1.63	1.68

[a]Means of pen performance data.

APPENDIX TABLE 12. POSTWEANING PERFORMANCE OF PIGS PROVIDED STARTER FEED WITH OVALBUMIN (OVA), OR WITHOUT OVALBUMIN (REG) (TRIAL 1)

	Starter Treatment[a]	
	OVA	REG
Avg weight (kg)		
Week 1	8.4	8.7
2	10.2	9.7
3	12.4	11.9
4	16.1	15.8
5	21.6	18.9
Avg daily feed intake (kg)		
Week 1	.20	.20
2	.35	.34
3	.65	.60
4	.78	.76
5	1.02	.96
1-2	.28	.26
1-3	.35	.37
1-4	.49	.46
1-5	.60	.57
Avg daily gain (kg)		
Week 1	.15	.20
2	.25	.15
3	.39	.32
4	.52	.47
5	.80	.68
1-2	.20	.18
1-3	.24	.25
1-4	.31	.30
1-5	.41	.38
Feed/gain		
Week 1	1.30	1.31
2	1.39	1.29
3	2.13	1.64
4	1.52	1.71
5	1.30	1.45
1-2	1.34	1.51
1-3	1.66	1.56
1-4	1.58	1.55
1-5	1.46	1.51

[a]Means of pen performance data.

APPENDIX TABLE 13. POSTWEANING PERFORMANCE OF PIGS PROVIDED STARTER FEED WITH OVALBUMIN (OVA), OR WITHOUT OVALBUMIN (REG) (TRIAL 2)

	Starter Treatment[a]	
	OVA	REG
Avg weight (kg)		
Week 1	8.6	19.8
2	10.4	10.6
3	13.1	13.7
4	17.8	18.2
5	22.3	22.8
Avg daily feed intake (kg)		
Week 1	.21	.15
2	.25	.38
3	.72	.72
4	.97	1.01
5	.71	.70
1-2	.27	.27
1-3	.42	.42
1-4	.56	.57
1-5	.71	.70
Avg daily gain (kg)		
Week 1	.21	.23
2	.25	.24
3	.39	.45
4	.68	.65
5	.64	.64
1-2	.23	.24
1-3	.28	.30
1-4	.38	.39
1-5	.43	.45
Feed/gain		
Week 1	.94	.79
2	1.55	1.90
3	2.21	1.64
4	1.56	1.61
5	2.06	1.97
1-2	1.19	1.13
1-3	1.52	1.36
1-4	1.48	1.44
1-5	1.95	1.97

[a]Means of pen performance data.

APPENDIX TABLE 14. POSTWEANING PERFORMANCE OF PIGS PROVIDED STARTER FEED WITH OVALBUMIN (OVA), OR WITHOUT OVALBUMIN (REG) (TRIAL 3)

	Starter Treatment[a]	
	OVA	REG
Avg weight (kg)		
Week 1	7.6	7.8
2	8.6	8.8
3	11.9	10.8
4	13.4	13.2
5	16.3	15.9
Avg daily feed intake (kg)		
Week 1	.14	.15
2	.24	.24
3	.46	.45
4	.59	.45
5	.87	.88
1-2	.20	.20
1-3	.31	.31
1-4	.31	.35
1-5	.49	.47
Avg daily gain (kg)		
Week 1	.26	.26
2	.16	.15
3	.33	.29
4	.35	.34
5	.41	.39
1-2	.20	.20
1-3	.30	.24
1-4	.28	.26
1-5	.41	.39
Feed/gain		
Week 1	.57	.61
2	1.51	2.21
3	1.40	1.58
4	1.64	1.35
5	2.16	2.73
1-2	.96	1.05
1-3	1.20	1.30
1-4	1.36	1.29
1-5	1.59	1.60

[a]Means of pen performance data.

APPENDIX TABLE 15. POSTWEANING PERFORMANCE OF PIGS PROVIDED STARTER FEED WITH OVALBUMIN (OVA), OR WITHOUT OVALBUMIN (REG) (TRIAL 4)

	Starter Treatment[a]	
	OVA	REG
Avg weight		
Week 1	7.8	7.6
2	9.1	8.9
3	11.9	11.5
4	15.2	15.0
5	19.3	18.9
Avg daily feed intake (kg)		
Week 1	.19	.40
2	.22	.31
3	.60	.61
4	.75	.69
5	1.12	1.12
1-2	.20	.24
1-3	.34	.37
1-4	.45	.46
1-5	.57	.57
Avg daily gain (kg)		
Week 1	.07	.06
2	.19	.19
3	.40	.37
4	.42	.44
5	.68	.64
1-2	.13	.12
1-3	.22	.20
1-4	.28	.27
1-5	.34	.34
Feed/gain		
Week 1	1.93	1.11
2	1.24	1.72
3	1.53	1.81
4	1.80	1.58
5	1.68	1.75
1-2	1.65	1.65
1-3	1.60	1.70
1-4	1.66	1.71
1-5	1.66	1.72

[a]Means of pen performance data.

APPENDIX TABLE 16. EFFECT OF PIGS OBSERVED WITH CHROMIC OXIDE IN FECES PREVIOUSLY ON POSTWEANING PERFORMANCE (TRIAL 1).

	Observation of Chromic Oxide		
	EAT[a] (18)	NONE[b] (22)	ONCE[c] (7)
Avg weight (kg)			
Week 1	8.6	8.1	7.7
2	10.2	9.8	9.1
3	12.4	12.2	11.4
4	16.2	15.9	14.7
5	20.9	21.1	20.3
Avg daily gain (kg)			
Week 1	.19	.11	.15
2	.23	.25	.21
3	.32	.34	.32
4	.54	.52	.48
5	.68	.75	.75
1-2	.21	.18	.18
1-3	.25	.24	.23
1-4	.32	.31	.29
1-5	.39	.39	.38

[a]EAT=chromic oxide observed in feces more than once.
[b]NONE=chromic oxide was never seen in the feces.
[c]ONCE=chromic oxide observed in feces only once.

APPENDIX TABLE 17. EFFECT OF PIGS OBSERVED WITH CHROMIC OXIDE IN FECES PREVIOUSLY ON POSTWEANING PERFORMANCE (TRIAL 2)

	Observation of Chromic Oxide		
	EAT[a] (40)	NONE[b] (6)	ONCE[c] (2)
Avg weight (kg)			
Week 1	9.0	9.0	9.3
2	10.6	10.9	11.5
3	13.3	13.7	13.6
4	18.1	18.1	17.8
5	22.7	22.6	22.9
Avg daily gain (kg)			
Week 1	.26	.23	.16
2	.23	.27	.32
3	.39	.40	.30
4	.69	.64	.61
5	.65	.64	.71
1-2	.24	.25	.24
1-3	.29	.30	.26
1-4	.39	.39	.35
1-5	.44	.44	.42

[a]EAT=chromic oxide observed in feces more than once.
[b]NONE=chromic oxide was never seen in the feces.
[c]ONCE=chromic oxide observed in feces only once.

APPENDIX TABLE 18. EFFECT OF PIGS OBSERVED WITH CHROMIC OXIDE IN FECES PREVIOUSLY ON POSTWEANING PERFORMANCE (TRIAL 3)

	Observation of Chromic Oxide		
	EAT[a] (11)	NONE[b] (21)	ONCE[c] (0)
Avg weight (kg)			
Week 1	6.7	6.9	
2	7.4	7.8	
3	9.4	9.8	
4	11.7	12.2	
5	14.7	14.8	
Avg daily gain (kg)			
Week 1	.23	.27	
2	.12	.14	
3	.29	.29	
4	.33	.35	
5	.44	.37	
1-2	.16	.19	
1-3	.21	.23	
1-4	.24	.27	
1-5	.29	.29	

[a]EAT=chromic oxide observed in feces more than once.
[b]NONE=chromic oxide was never seen in the feces.
[c]ONCE=chromic oxide observed in feces only once (not observed in this trial).

APPENDIX TABLE 19. EFFECT OF PIGS OBSERVED WITH CHROMIC OXIDE IN FECES PREVIOUSLY ON POSTWEANING PERFORMANCE (TRIAL 4)

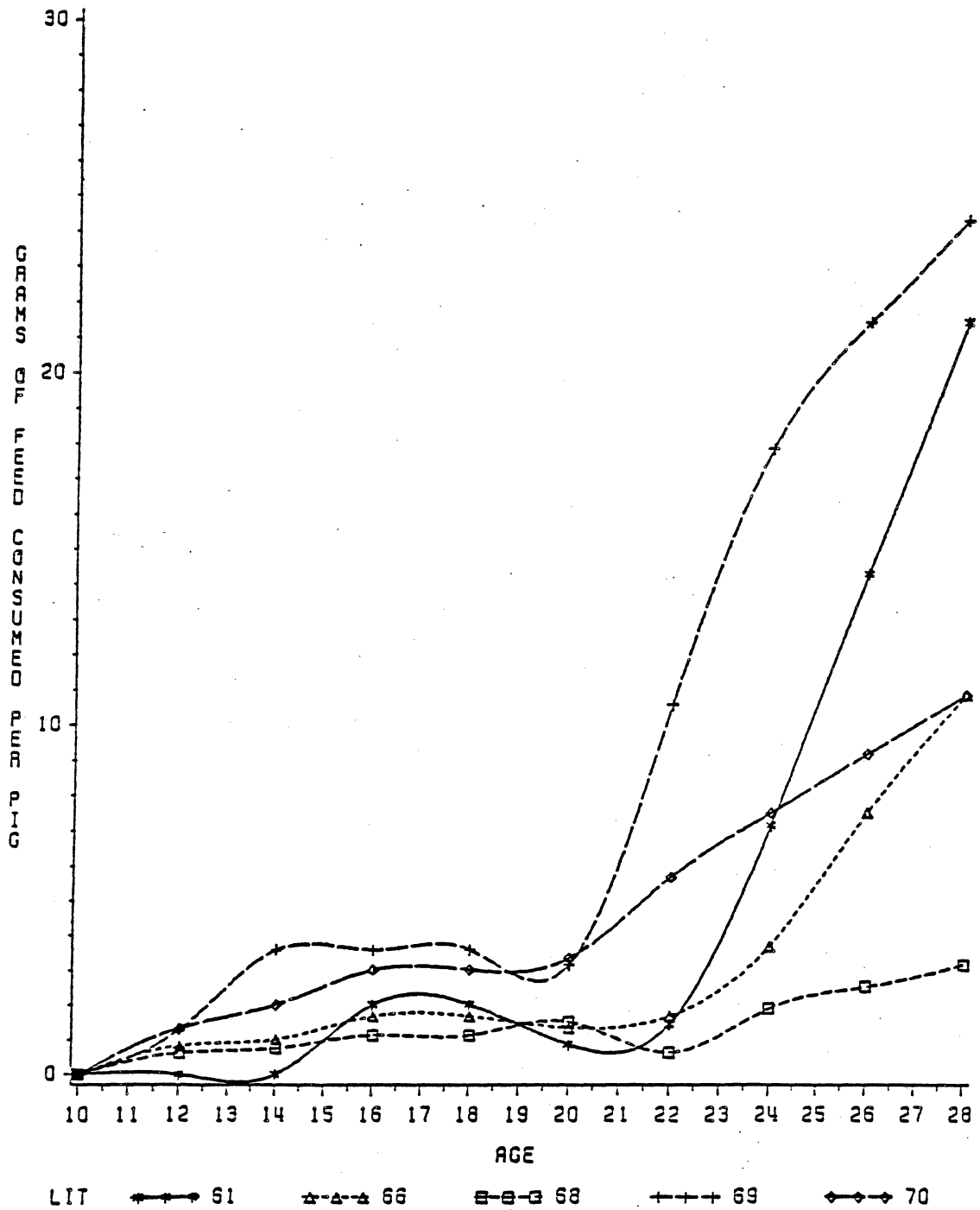
	Observation of Chromic Oxide		
	EAT[a] (31)	NONE[b] (21)	ONCE[c] (12)
Avg weight (kg)			
Week 1	8.0	7.0	7.7
2	9.4	8.0	9.1
3	12.2	10.5	11.8
4	15.4	13.6	15.5
5	19.9	17.2	19.0
Avg daily gain (kg)			
Week 1	.09	.01	.05
2	.20	.15	.19
3	.40	.36	.39
4	.40	.39	.46
5	.75	.59	.59
1-2	.14	.08	.12
1-3	.22	.17	.21
1-4	.28	.23	.28
1-5	.35	.29	.33

[a]EAT=chromic oxide observed in feces more than once.
[b]NONE=chromic oxide was never seen in the feces.
[c]ONCE=chromic oxide observed in feces only once.

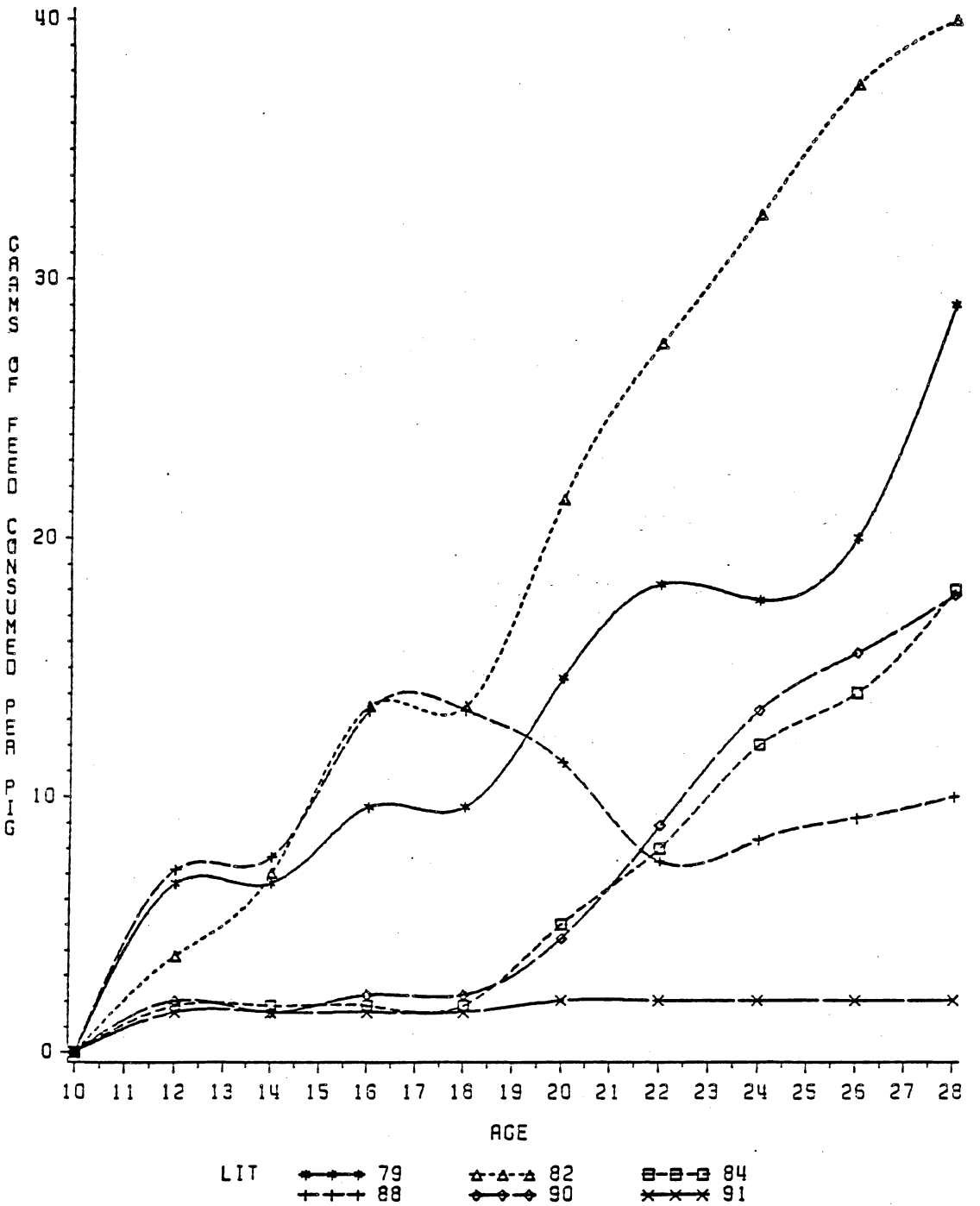
APPENDIX TABLE 20. EFFECT OF PIGS OBSERVED WITH CHROMIC OXIDE IN FECES PREVIOUSLY ON POSTWEANING PERFORMANCE (ALL TRIALS COMBINED)

	Observation of Chromic Oxide				
	EAT[a] (n=100)	NONE[b] (n=70)	ONCE[c] (n=21)	±SE	P > F
Avg weight (kg)					
Week 1	8.1	7.7	7.9	.43	.43
2	9.4	9.1	9.3	.50	.59
3	11.9	11.6	11.8	.62	.71
4	15.4	14.9	15.5	.74	.55
5	19.7	18.9	19.7	.84	.31
Avg daily gain (kg)					
Week 1	.13	.16	.13	.02	.12
2	.20	.20	.21	.03	.77
3	.35	.35	.36	.04	.76
4	.49	.46	.48	.04	.70
5	.64	.59	.65	.05	.25
1-2	.16	.18	.15	.02	.44
1-3	.22	.23	.22	.03	.61
1-4	.28	.23	.29	.02	.40
1-5	.37	.35	.36	.02	.17

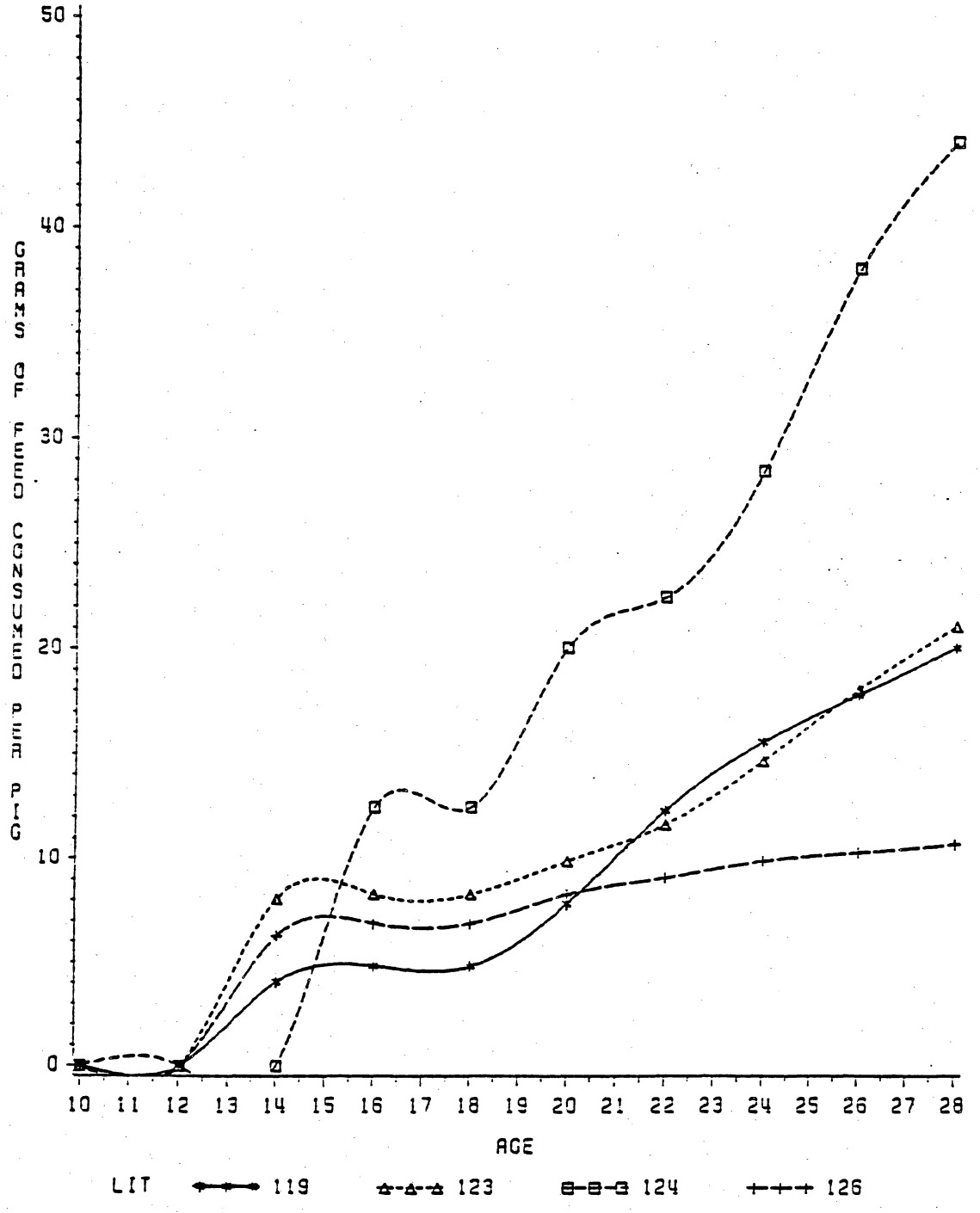
[a]EAT=chromic oxide observed in feces more than once.
bNONE=chromic oxide was never seen in the feces.
cONCE=chromic oxide observed in feces only once.



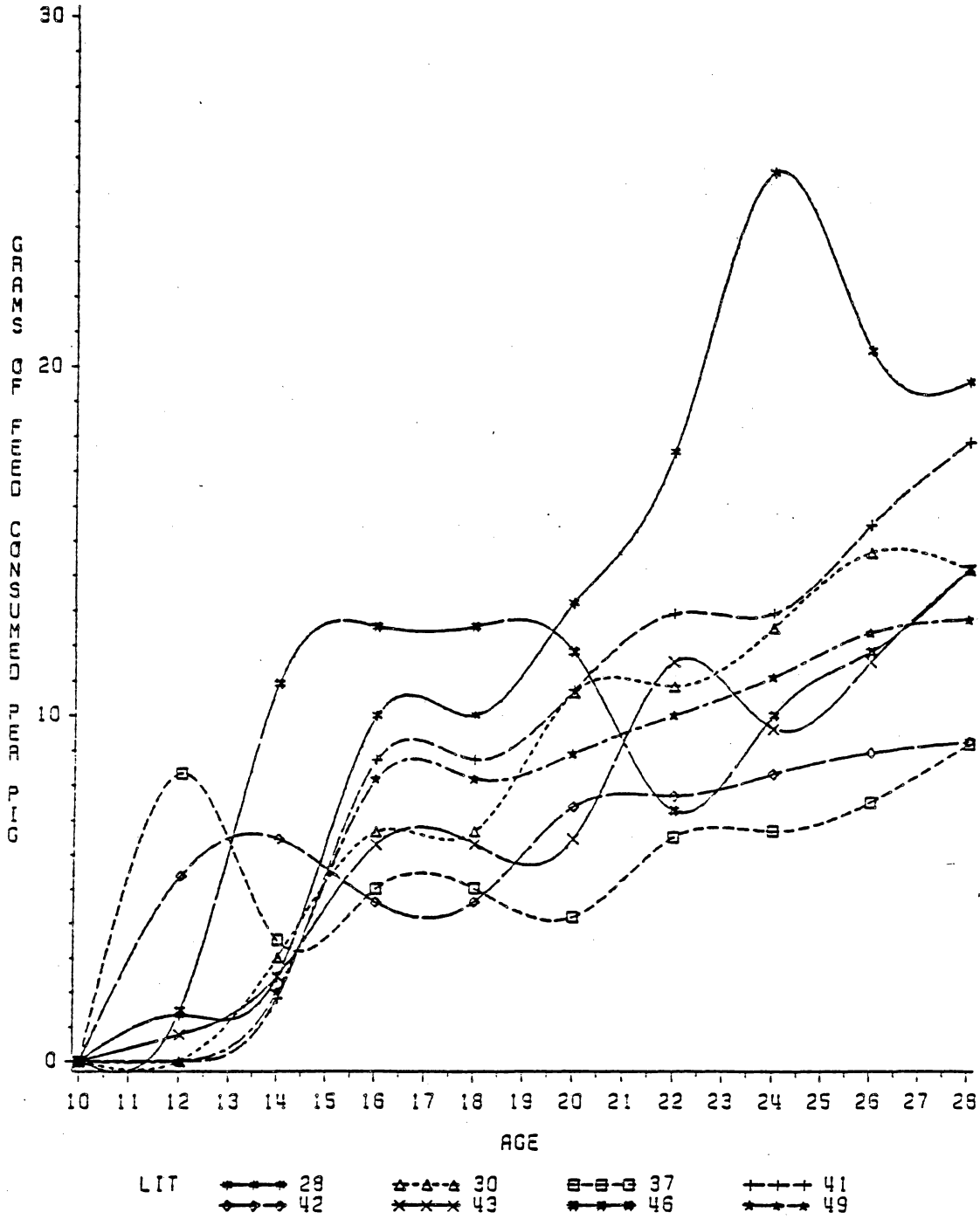
Appendix Figure 1. Daily feed consumption per pig per litter (lit) for Trial 1 (OVA creep diet=lit 61, 66 and 68; REG creep diet= lit 69 and 70).



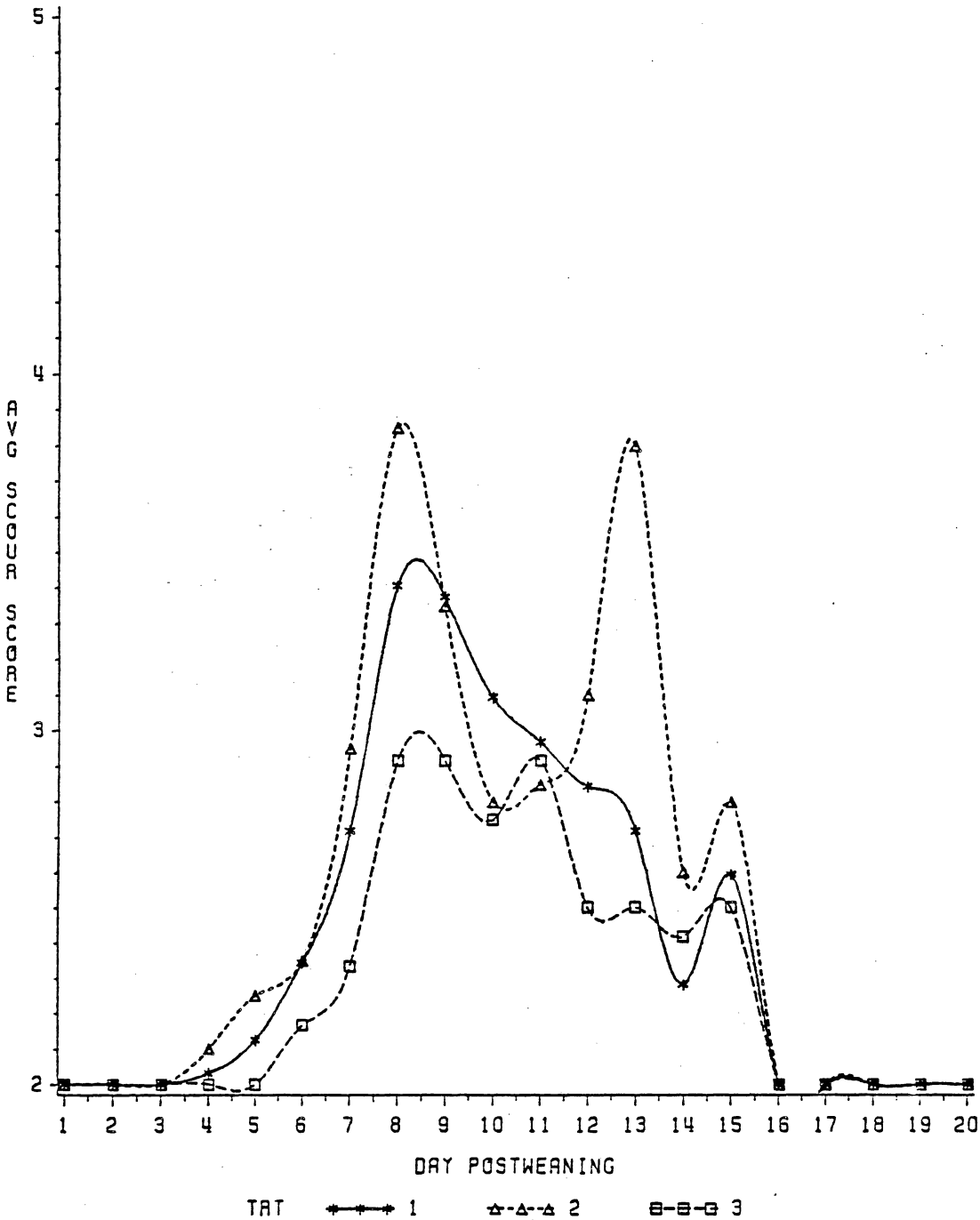
Appendix Figure 2. Daily feed consumption per pig per litter (lit) for Trial 2 (OVA creep diet=lit 79, 82, 84, 90 and 91; REG= lit 88).



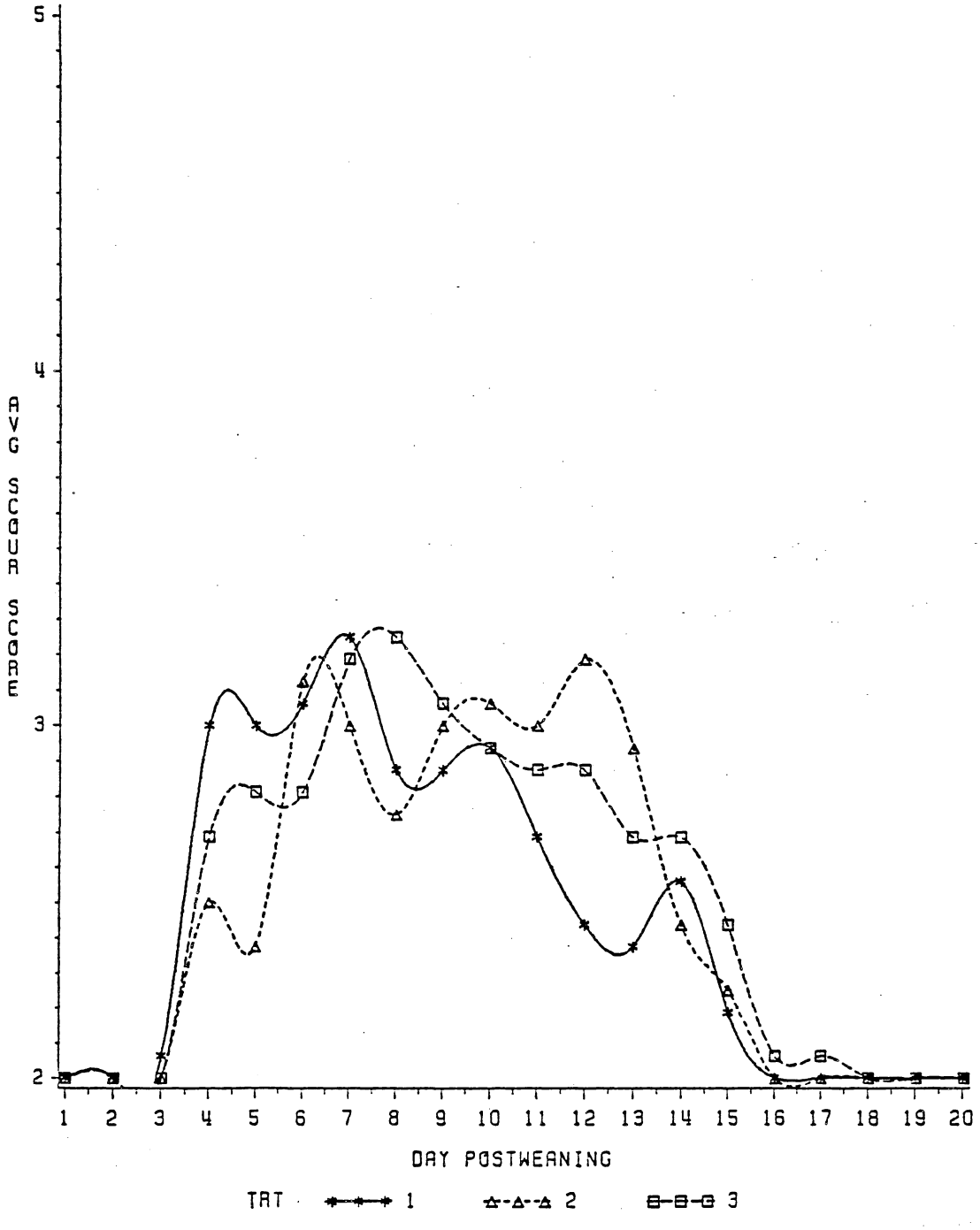
Appendix Figure 3. Daily feed consumption per pig per litter (lit) for Trial 3 (OVA creep diet=lit 119 and 124; REG creep diet= lit 123 and 126).



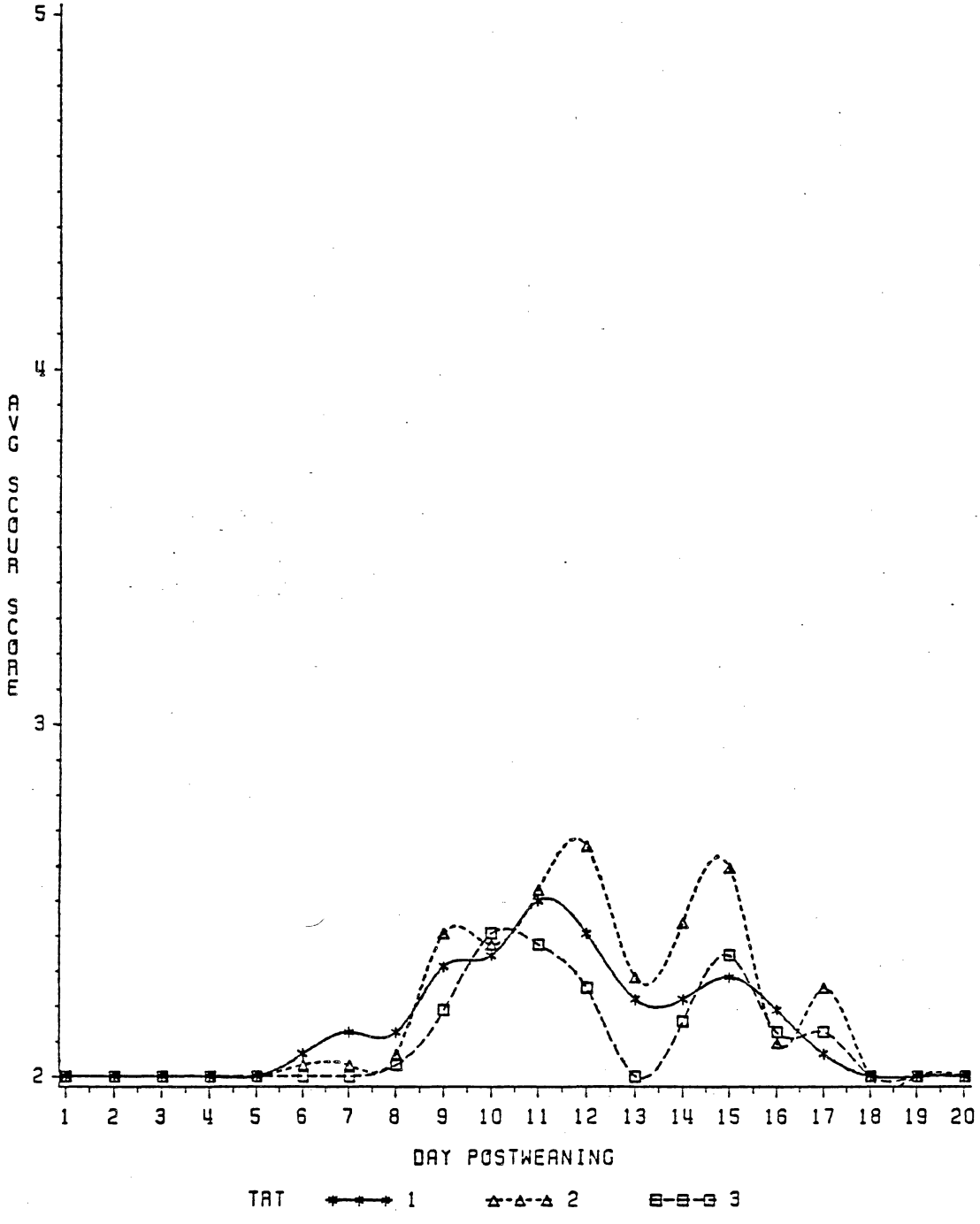
Appendix Figure 4. Daily feed consumption per pig per litter (lit) for Trial 4 (OVA creep diet=lit 119 and 124; REG creep diet= lit 123 and 126).



Appendix Figure 5. Postweaning scour scores (2=normal, 5=very severe scours) as influenced by previous creep treatment (1=OVA creep diet, 2=REG creep diet, 3=CON noncreep-fed). Trial 1.



Appendix Figure 6. Postweaning scour scores (2=normal, 5=very severe scours) as influenced by previous creep treatment (1=OVA creep diet, 2=REG creep diet, 3=CON noncreep-fed). Trial 3.



Appendix Figure 7. Postweaning scour scores (2=normal, 5=very severe scours) as influenced by previous creep treatment (1=OVA creep diet, 2=REG creep diet, 3=CON noncreep-fed). Trial 4.

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