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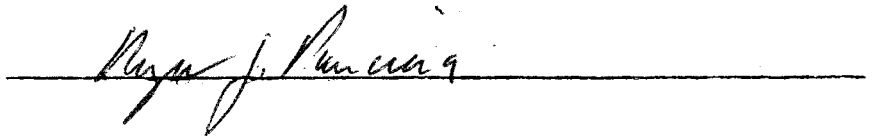
and Method of Study: The purpose of this report is to review current concepts regarding the history and geographic distribution, etiology, mode of transmission, pathogenesis, clinical signs, macrc scopic and microscopic pathology, diagnosis, prophylaxis, control, and immunology of transmissible gastroenteritis (TGE). The report includes a description of the clinical and pathologic features of TGE, and the experimental inoculation of a litter of pigs with TGE virus.

ngs and Conclusions: Transmissible gastroenteritis is caused by a coronavirus whose primary target tissue is the absorptive epithelia cells of the small intestine. The infection is transmitted natural ly by oral and nasal routes. The incubation period is very short (18-24 hours) followed by vomiting, diarrhea, dehydration, and high mortality in suckling pigs. Histopathologic examination of infecte pigs demonstrates villous atrophy. Presumptive diagnosis is based on the clinical signs and histologic demonstration of villous atrophy. Diagnostic confirmation is obtained by demonstration of TGE antigen by means of immunofluorescence, seroneutralization tests, and/or virus isolation. Severity of the disease is related to the age of the animal and immunologic protection. The protective mechanism against TGE is directly related to the presence of IgA in colostrum and milk. The immunology of the disease has been extensively studied. However, the development of a good protective vaccine requires further research. The experimental exposure of pigs susceptible to TGE virus resulted in the production of a disease with similar clinical signs and pathologic changes to those described for the natural disease.

ER'S APPROVAL 

TRANSMISSIBLE GASTROENTERITIS OF SWINE:

A REVIEW OF CURRENT CONCEPTS

By

RIXA R. RINCON DE LIENDO

Medico Veterinario

Universidad del Zulia

Maracaibo, Venezuela

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TRANSMISSIBLE GASTROENTERITIS OF SWINE:

A REVIEW OF CURRENT CONCEPTS

Report Approved:

Wayne J. Pancoast
Report Adviser

S. T. Irving

Ernest L. Stair, Jr.

Norman N. Durham
Dean of the Graduate College

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CHAPTER I

INTRODUCTION

ansmissible gastroenteritis (TGE) is a widespread, highly trans-
e disease of pigs caused by a corona virus whose principal target
differentiated absorptive epithelial cells of the small intestine.
ease was first described in the United States by Doyle and Hutch-
: Purdue University in 1946.[15] Subsequently, it was reported in
iropean countries, Japan, Taiwan and Canada.[16] TGE is a major
of death in newborn pigs and economically is one of the most im-
: diseases in the swine industry in the United States and the
[17,55] It is especially prevalent in those countries where there
intensive system of swine production and whose principal stock has
nported from the United States or Europe.[71]

ne widespread and devastating impact of TGE in the swine popula-
as led to extensive research into the nature, epidemiology, and
logy of the disease. An important goal in the swine industry is
vide suckling pigs immunity against TGE virus.[74] Numerous vac-
have been developed without satisfactory results. There is no
ic antiviral treatment for the disease; control must be based on
tion.[22,61] Control, therefore, is based on avoidance of trans-
n and immunization. The identification and elimination of TGE
omatic carriers in a swine herd must be of utmost importance be-
they disseminate the virus to susceptible pigs.[30]

The objective of the present study is to review current knowledge E, emphasizing histologic changes and pathogenesis of the disease fected pigs. The sequential pathological changes that occur in the intestine of pigs infected experimentally with TGE virus will be described.

CHAPTER II

REVIEW OF STUDY

History and Geographic Distribution

Transmissible gastroenteritis was first reported in the United
es by Doyle and Hutchings at Purdue University in 1946 when they des-
ed sporadic outbreaks of the disease in swine herds, and successfully
oduced the disease in experimentally infected pigs.[15,16] There was
oubt that the disease had existed before this time and outbreaks of
sease with similar clinical signs to TGE were described by various
ican authors in 1933, 1935 and 1937.[16] In succeeding years TGE was
rted in Japan (1956), England (1957), and yet later in many countries
ughout Europe, Taiwan (1958), and Canada (1960).[16,71] Although
smissible gastroenteritis is not a new disease, it has recently be-
more important due to intensification of husbandry.[71]

Etiology

The causative agent of TGE of swine is a coronavirus.[28,48,49]
ough strains of variable virulence have been described, there appear
e only one serologic type.[2,3,61] Morphologically TGE virus is char-
rized by pleomorphic enveloped particles of virus with an average
eter ranging from 75 to 160 nm.[28,35,61] The surface is surrounded
lub-shaped projections 12 to 24 nm long.[3,28,49]
The virus contains ribonucleic acid (RNA),[56] is ether and chloro-

labile,[3,49] and is easily destroyed by high temperature and drying.[2]
moderately resistant to bile and trypsin and relatively stable at
[3,49] These characteristics contribute to the survival of the virus
s passage through the alimentary tract.[3] It is rapidly inactivated
posure to bright sunlight, hence the virus survives longer in the
intense sunlight of winter. That, cooler temperatures, and more
ate contact of swine in crowded houses facilitate survival and
mission of the virus during winter months.[2,32,61]

Viral particles replicate in the cytoplasm of differentiated epithe-
cells of the small intestine, primarily the jejunum, of infected
[41] Villous epithelial cells of the ileum and duodenum are affected
lesser degree.[41] The gastric and colonic epithelial lining cells
r to be refractory to infection by TGE virus.[21,26] In this regard,
irus is in contrast to the coronavirus of calf diarrhea agent that
infects the colonic epithelium.[37] Viral particles may occur singly
e epithelial cytoplasm or form clusters that include as many as 5
ns.[65] The virus has been reported to be not associated with mem-
s or cellular organelles.[65] Replication of TGE virus occurs rapidly
an be demonstrated within 6 hours after exposure of susceptible pigs
fection.[26]

Transmissible gastroenteritis virus (TGEV) infects and produces dis-
only in swine.[2,24] However, the virus can infect the small intes-
of dogs and foxes and is shed in the feces for 7 and 15 days re-
ively.[71] The virus has been demonstrated to replicate in the lungs
idneys of swine,[22,26] primarily in feeder pigs, thus constituting
rce of infection for susceptible pigs. The pathogenic significance
E virus in tissues other than small intestine remains unknown.[13]

Isolation of the virus is difficult because the cytopathic effect produced by field strains is very slight or negligible in the first passages.[16,61] However, depending on the amount of virus present in noculum and the susceptibility of the cell culture used, CPE is ac- ıated in later passages.[16,61]

Transmission

Natural infection of pigs is believed to occur most commonly and ef- ıntly by the oral route.[22,26] Greater amounts of virus are required oduce clinical signs in pigs when parenterally inoculated than when istered orally.[22,26] On the other hand, infection by inhalation of olized particles of fecal material has been described.[61,65] The respiratory tract has been reported as the probable portal of entry fection in adult swine.[31] Numerous reports in the literature agree the oral and nasal routes are the most effective routes of inocula- 2,31,61,72

Transmissible gastroenteritis virus is present in large amounts in eces of diseased pigs and is excreted for periods up to 10 weeks.[61] ted sows may excrete the virus through milk, nasal secretions, and .[31] Feeder pigs are considered a major reservoir of the virus be- pigs that have recovered from the disease have been shown to be ers of the virus in the small intestine for periods up to 6 weeks ıfection.[39] Pigs that harbor virus in their lungs may remain car- during interepizootic periods; they are a source of infection for herds or for reinfection in a continuous farrowing system.[30,65] ally, the introduction of new animals into a herd precedes a TGE otic.[2,24]

Starlings may passively transmit the virus for about 32 hours.[71] ;ted dogs and foxes become active shedders of TGE virus for up to 2 post exposure.[2,24,71] The infected or contaminated farm dog, which ently is not sick and has access to swine facilities, may be an im- nt source of TGEV for susceptible swine populations.[35] Contaminated ing, transport vehicles, and the use of frozen infective intestinal ial for immunization procedures are considered important factors in ransmission and propagation of the infection.[2,61,65]

Pathogenesis

Infection is acquired either by ingestion or inhalation of TGEV. rimary target tissue is mature, absorptive, epithelial cells of the intestine.[16,21,38] Viral replication occurs within 4 to 6 hours in ytoplasm of differentiated epithelial cells with highest titers be- resent in the jejunum.[47] Most cells in the upper duodenum and those e villi covering lymphoid tissue (Peyer's patches) in the ileum are nfected.[21,22,47] As a result of the rapid viral replication, the ted epithelial cells degenerate.[21,38]

Epithelial cell degeneration is characterized by the formation of lasmic vacuoles of variable size, irregularity of the brush border, trophy of the nuclei.[71] Although many epithelial cells are destroy- d sloughed, there is no obvious inflammatory response, and the sur- of the villi remains covered by flattened or cuboidal immature elial cells that migrate to the villous surfaces from the crypts of rkuhn.[16,21,25] As a result of epithelial cell loss, atrophy of occurs. Crypt epithelial cells and the lamina propria are not af- d by the virus. However, there is an increased rate of cell produc-

and hyperplasia of crypt epithelium that is inadequate to compensat
he loss of cells on the villous surfaces.[33,44,63] In cases of sever
us atrophy with extensive epithelial cell loss the villus-height:
-depth ratio decreases from the normal of about 7:1 to 1:1.[22,47]
Villous atrophy is reversible and regeneration of villi occurs in re
ed pigs either by elongation of affected villi, the formation of new
or both.[41] Villous elongation occurs when new, immature epithelial
differentiate into columnar epithelial cells (between 96 and 168
postinfection); regrown villi may be fused at their tips or at thei
halves.[47] Return of normal function coincides with regrowth of
[40]

Large concentrations of many enzymes such as disaccharidases, alka
phosphatase, aminopeptidase, lactase, etc., are found in the absorp-
epithelial cells of the small intestine.[62,64] Therefore, atrophy of
illi with the consequent loss of the surface area markedly reduces
igestive and absorptive functional capacity of the small intestine
esults in acute malabsorption.[17,22,33,38,41]
The combination of malabsorption due to reduced surface area, lack
urity of intestinal epithelium, and the passage of undigested
al (lactose primarily) to the colon results in a highly osmolar
ic content that exceeds the absorptive capacity of the colon and
ates water movement. As a consequence, there is diarrhea, dehy-
n, electrolyte imbalance and finally death.[1,2,29,38,55,71] Al-
malabsorption appears to be the major diarrheagenic mechanism in
aldigestion, hypersecretion or continued secretory activity of in-
al glands (crypts of Lieberkuhn), and alterations of fermentation
bute to complicate the process.[38] By supplying only water and

olding milk (food deprivation) may result in stopping the diar-
33,71

Stools of infected pigs contain large amounts of sodium, potassium,
hloride.[6,29] The high concentrations of sodium are suggestive of
r a deficit in intestinal absorption of sodium or excessive secre-
of this ion into the intestinal lumen.[2,8,29] The migration and
nce of undifferentiated, functionally immature cells onto the
al villi contribute to the defective sodium transport in TGE.[34]
otion of fat, glucose, and other nutrients is also diminished in
infected with TGE.[33,38]

Clinical Signs

The disease (TGE) is characterized by an incubation period of 18-24
, and rapid spread among susceptible animals.[2,9] The first clinical
is usually vomiting followed by yellowish watery diarrhea, dehydra-
and high mortality in suckling pigs.[22] In piglets, the diarrhea is
it and profuse, and the feces usually contain small curds of undi-
l milk.[16] In affected animals, there is marked depression, dehydra-
weakness and emaciation that progress to death in 2 to 5 days.[2,16]
n piglets infected under 10 days of age, the mortality rate can be
gh as 100%.[2,29] Pigs older than 3 weeks at infection have a 50%
e of survival.[2,22] It is believed that younger pigs suffer more
ely because intestinal epithelial cells of newborn pigs are replaced
ess rapidly than are those of older pigs[41] and because the epithe-
cells of newborn pigs are considerably older and more mature than
able cells in 21-day-old pigs. Older pigs have greater capacity
duce antibody, and interferon production increases with age.[45]

)lder infected pigs usually have a mild diarrhea, and depression
› longer than 2 weeks.[6] Elevated temperatures, anorexia, and aga-
ɜ have been described in sows affected shortly after parturition.[32]
ɜre often observed to vomit in field outbreaks of TGE; but respira-
;igns have not been observed.[32] Complete recovery usually occurs
ı 7 to 10 days.[32]

Transmissible gastroenteritis tends to appear as violent, dramatic
ɜaks that usually resolve in a period of 3-5 weeks or less. Gener-
because the farrowing schedule has been completed or, where con-
ıs farrowing is practiced, due to maternal resistance transmitted
;kling pigs by sows infected early in the outbreak.[22]

Macroscopic Findings

'iglets dead of TGE are usually severely dehydrated but in good
:ional condition. Postmortem lesions are confined to the gastro-
:inal tract where there is congestion of the mesenteric vessels and
ıtion of the entire gastrointestinal tract with foamy yellow fluid
ıs. As a result, the small intestine appears thin-walled and
:.[12,14,27,61,62] The stomach may be distended with curdled, undi-
l milk, and may be inflamed.[27,61] In severely dehydrated
s, there is fundic and pyloric congestion, and focal hemorrhage in
ıbmucosa of the greater curvature.[16,25,27] Yellowish streaks (ac-
ıtion of urates) in the renal medulla have been described in some
of TGE.[16,25,61] The absence of obvious fat and chyle in the in-
ıal and mesenteric lymphatics have been described at 24 hours or
ıfter infection with TGE virus.[11,12,14,27]

The gastric content in pigs with marked gross lesions are slightly

cidic while contents of the small and large intestine are slightly cidic than normal.[11]

he most important subgross and sometimes gross lesion is a marked ning of the villi in the small intestine, primarily jejunum and [16,27] Under the dissecting microscope (6 magnifications), the ed villi appear as small mounds that produce a pattern resembling lestone street.[11] Macroscopic lesions in experimentally infected re the same as those in naturally infected pigs.[14]

Microscopic Findings

he mucosal surface of the normal intestine is composed of columnar (also called absorptive or main cells) and mucus-producing goblet Goblet cells are most numerous in the colon.[66] Paneth and chromaffin cells are located deep in the ileal crypts.[66] Columnar lial cells originate from undifferentiated crypt epithelial cells proliferate and differentiate into mature villous absorptive cells y migrate to the villous tips from the crypts.[36,64] The normal of the small intestine presents a maximal absorptive surface to traluminal contents of the intestine.[64] This absorptive surface amplified 14-39 times by the presence of the microvilli.[64]

ong, tongue-shaped villi (in normal piglets) are found primarily in oximal part of the small intestine.[42] In the duodenum the villi mostly short to long, thick and finger-shaped with rounded tips.[42] lli in the jejunum are commonly long, slender, and finger-shaped rounded tip; whereas in the ileum the villi are mainly short, r, and finger-shaped with a pointed tip.[42]

he primary microscopic change in TGE is destruction (by viral re-

tion) and eventual loss of absorptive, villous epithelial cells,[33] ting in contraction of the lamina propria, broadening and fusion of , and cuboidal to squamous metaplasia of remaining villous epithe-

These changes have been referred as villous atrophy.[25]

At about 12 hours after infection, columnar epithelial cells are ened and lose microvilli.[21] Between 12 and 18 hours postinfection, is cellular desquamation accompanied by shortening of the villi.[21] affected villi are covered with immature, flat or cuboidal, cells have basophilic cytoplasm and lack striated borders.[21,62] These un-entiated cells migrate from the crypts of Lieberkuhn that appear hyperplastic and elongated with an increased rate of cellular pro-ation.[17,21,33,36] Mitotic activity in crypt cells is increased in ed animals leading to a decreased villous epithelium-crypt epithe-atio.[62] The villus-height/crypt-depth ratio is reduced from 7:1 mal pigs to less than 1:1 in the jejunum of severely diseased [6,27,46] The normal length of jejunal villi in healthy suckling s 795µ, and the depth of the crypts is approximately 110µ.[16,25] hours postexposure to TGE virus, the length of the villi is near nd the depth of the crypt is 157µ.[16,25]

pithelial cells of infected pigs are flat to cuboidal and poorly entiated.[27] They have vacuolated cytoplasm with indistinct cyto-c borders, and short poorly defined microvilli.[25,27,46,62] In cases of TGE, villous atrophy may be so extensive that the villi ortened to such an extent that only small protrusions of a rela-flat mucosal surface are observed.[27]

ild congestion and infiltration of the lamina propria of the small ine with inflammatory cells have been described.[62] Nevertheless,

l or no inflammatory response is the most frequent histologic
g.[16]

icroscopic findings in tissues other than the small intestine are
on and minimal, but when present consist of vascular congestion in
rge intestine along with mild round-cell infiltration, and degen-
e changes in the convoluted tubular epithelium of the kidney.[16]
totic activity of the stomach tends to decrease in TGE-infected
7 Inclusion bodies have not been reported in any tissue.[71]
n conclusion, villous atrophy is the salient and most significant
copic finding in TGE-infected pigs.[62]

Diagnosis

iagnosis of TGE is usually based on the epizootiology of the out-
clinical signs, and histopathological findings.[61] Clinical signs
s acute onset of vomiting, diarrhea throughout the herd, and high
ity and mortality in suckling pigs are significant factors leading
diagnosis of TGE.[12,61] Histologically, marked villous atrophy in
all intestine is an important and useful tool in the diagnosis of
sease.[11,12,46] Although villous atrophy is extensive in TGE-
ed pigs, it is not unique to TGE.
ransmissible gastroenteritis is likely to be confused with coli-
osis, caused by enteropathogenic strains of E. coli,[16] and there-
TGE must be differentiated from it and other enteric diseases of
Clinical signs may be present in older feeding or breeding
s affected with TGE virus; whereas animals of this age are commonly
fected in colibacillosis.[16] The marked villous atrophy that is
lly extensive and constant in TGE cases is limited or absent in

cillosis.[11,16] The colonic contents of pigs with colibacillosis kaline and intestinal lactase is abundant while in TGE, the colonic ts are acid and lactase activity is absent.[11,12] Clinical signs sions similar to TGE have been described in porcine rotavirus in- ns.[2,23,54] However, diarrhea due to porcine rotavirus infection between the ages of 10 to 28 days and younger pigs are supposedly uently infected.[57] On the other hand, TGE has been described as evere, clinically and pathologically, than porcine rotavirus in- n.[54,57] A definitive diagnosis must be based on fluorescent anti- nd virologic procedures. Currently, coccidiosis in piglets, as in ave been reported to produce marked villous atrophy of the small ine. Diagnosis is based on finding of coccidial forms in the af- intestinal mucosa at histopathologic examination.

dentification of animals exposed to TGE virus has been based on isolation in cell culture, presence of virus-neutralizing anti- in the serum of recovered animals (serum neutralization tests), the use of fluorescent antibody techniques (FAT) to demonstrate antigen.[61,73] Immunofluorescence has been widely utilized in the sis of TGE and a positive diagnosis is based on finding fluores- in the cytoplasm of epithelial cells at the tips of the villi.[61] rks efficiently at either early or late stages of the disease (de- tive and regenerative stage).[39] In cases of severe and extensive s atrophy, however, demonstration of TGE viral antigen may be ult.[39]

t is well known that the small intestine is the primary target of rus; therefore, the tissue of choice for demonstrating immuno- scence of TGE antigen is intestinal villous.[46] Laboratory samples

croscopic subgross and histopathological examination should include affected piglets.[61] Other laboratory tests are also best done with s from live piglets.

Other techniques reported for the diagnosis of TGE include Leukocyte ation assay (LA) and Immune Electron Microscopy (IEM).[54,73] The ay has been described as slightly more sensitive than the viral lization test in the early diagnosis of TGE infected pigs.[73] On her hand, using the IEM technique, it is possible to demonstrate esence of viral particles in a sample within 24 hours or less after ion.[54]

iagnosis can be confirmed by feeding homogenized, filtrated intes- tissue or fecal samples from suspected cases to susceptible baby 3

Prophylaxis and Control

t is of primary importance to protect baby pigs against TGE, since sease may cause massive and spectacular losses of newborn pigs. nnot be effectively treated, control must be based on prevention ns of avoidance of transmission and immunization.[18,61]

rotection is dependent upon passive or active immunity. Passive ty in newborn pigs is directly associated with the continuous sup- specific antibodies of TGE in the intestinal tract to neutralize rus and prevent atrophy of the villi.[58,60,72] Passive immunity is ed in baby pigs by ingestion of antibodies contained in the nurs- w's colostrum and milk.[18] The term "lactogenic immunity" has been o describe this important protective mechanism.[19,25,57]

Active immunity, on the other hand, involves the active production

antibodies as a result of exposure to TGE virus or antigen. Cur-
in the United States, there is only 1 (one) vaccine for TGE that
ensed by the Veterinary Biologics Division of the United States
ment of Agriculture.[60] It consists of a modified live-virus vac-
hat has been attenuated to avoid causing sickness or death when ad-
ered orally to baby pigs.[60] Other vaccines such as: 1) an inacti-
virus vaccine intramuscularly (I.M.) administered, 2) a modified
irus vaccine I.M. administered, and 3) an inactivated virus vaccine
ammary administered, have been evaluated by the Veterinary Bio-
Division.[60] Although all 3 vaccines have been found to be safe,
re not recommended because not one has the desired efficacy.[60]
he deliberate infection of sows, about 4-6 weeks before farrowing,
infected intestinal contents, has been described as an effective
to develop active immunity in the sows so that colostral antibody
duced to protect the new litters.[61] It has the disadvantage of
, that it is a source for infection of susceptible pigs.
Finally, admission of unnecessary visitors or contaminated vehicles
d be avoided to protect a susceptible herd.[61]

Immunology

The alimentary tract contains lymphoid tissue capable of producing
munologic response that will protect the epithelial barrier against
ration by viral antigens.[68] Protection against infection by organ-
that penetrate the body through the intestinal tract generally
ds on local immune mechanisms.[70] The local immune response is in-
dent of systemic immune reactions; IgA is an important factor in
mmunologic defense of epithelial surfaces.[68] Intestinal IgA anti-

represent the most important factor of host defense at the
lial surface.[69] They neutralize viruses, restrain bacterial pro-
tion, and prevent penetration of enterotoxins and intestinal anti-
6,69

ecretory IgA is the prevalent immunoglobulin in intestinal secre-
51,70 It is composed of two 7S monomers of IgA united by a J
ng)-chain, and contains an unique nonimmunoglobulin protein known
retory component (S.C.).[70] This component is responsible for the
nic properties of the molecule and its greater resistance to the
lytic action of the alimentary enzymes.[50,67,75] IgA is more re-
t to the action of the digestive enzymes than IgG and it has been
bed as being readily distributed throughout the epithelial mucosa
alimentary tract.[4,75] These features constitute an advantage for
evention of infection within the alimentary tract.[75]
he production of IgA is mediated by plasma cells situated in the
propria near the epithelial surface.[69] Plasma cells are stimu-
by contact with intestinal antigens to proliferate and differenti-
to antibody-secreting cells.[69] They are more numerous in the
duodenal and jejunal mucosa than plasma cells containing IgG im-
obulin.[10] Peyer's patch germinal centers are the precursors of
testinal IgA plasma cells.[20]
bsorption of immunoglobulins of colostral origin is for a rela-
limited period of time in the alimentary tract of newborn pigs.[25]
36 hours after the first meal of colostrum the intestine becomes
eable to intact proteins and no further antibody is absorbed.[22,
However, immunoglobulins of colostral origin function in the gut
by controlling bacterial and viral multiplication during pre-

g.[76]

eonatal pigs acquire passive immunity by the postnatal system;
ore, maternal immunity plays an important role in transferring
e resistance to the newborn pig.[7,50] Sows recovered from TGE may
it passive immunity to their offspring via colostrum.[22] Passive
ty against TGE is predicated on neutralization of TGE virus within
strointestinal lumen of suckling pigs by the continual ingestion
ibodies in milk or colostrum.[25,34] Thus, pigs suckling immune
re capable of resisting infection as long as they continue to
immune sows and as long as specific antibody persists in milk.
ecome susceptible to TGE virus within a few hours after withdrawal
cific antibody from the diet.[25,60]

ıe origin of IgA TGE antibodies in milk is not clear but it is
that their presence in milk is related to infection of the intes-
tract.[5,53] This observation led to the suggestion that antigenic
ation of the small intestine results in stimulation of IgA-
competent cells which migrate to and colonize in the mammary glands
they contribute to the local synthesis of antibodies of the IgA
,52] On the other hand, it has been observed that most of the
globulin, of the IgA class, in porcine colostrum is derived from
ood serum antibody pool by a transudation mechanism during the
0 days of gestation.[7]

lk of sows infected orally contains IgA TGE antibodies whereas
k of sows vaccinated intramuscularly or intramammarily does not;
lies stimulated by intramuscular or intramammary injections of TGE
ıs are primarily, if not entirely, associated with the IgG
,7,53

irculating antibodies are reported to provide little if any protec-
gainst TGE intestinal infection.[5,17,25] Consequently, to protect
nd their litters against TGE, exposure to the antigen should be by
testinal route rather than systemically.[70] Thus, extensive re-
has been oriented to the production of vaccines that increase the
of IgA antibody in the mammary secretions.[19] An ideal TGE vaccine
ɔe one that was sufficiently virulent to infect the intestinal
leading to the production of IgA TGE antibodies in the milk of
ɟ sows, but sufficiently attenuated so as not to produce sickness
ıatal pigs.[5] Currently in the United States, there is a licensed
ːcine; it is a modified live-virus vaccine, but its effectiveness
ɨtionable.

ıe mechanism of active immunity to TGE is unknown.[3,16] Active im-
is apparently based on the resistance or the protection of a
ent number of surface epithelial cells of the intestinal tract
: they may be infected but not suffer impaired function.[3] The
sm for the resistance or the protection of these cells is unknown,
ıechanism related to the presence of antibodies, either freely or
ely associated with the epithelial cells of the alimentary tract,
n suggested.[3] The continual elaboration of antibodies to TGE by
cells in the lamina propria of the small intestine and their
on through or around the epithelial cells will specifically pro-
otection to such cells.[3,16] Furthermore, the presence of anti-
in saliva and gastrointestinal secretions tends to neutralize TGE
efore its absorption to intestinal epithelial cells.[3] Another
sm may be related to the replication of epithelial cells that, as
t of the effect of the virus on the progenitor cells, are resis-

o TGE virus.[3]

urrent information indicates that a significant degree of active
ty only occurs as a result of infection of the intestinal tract
GE virus.[16] Swine that have recovered from TGE are immune when
ted to challenge, but the duration of this immunity is unknown.[3,16]
ield observations suggest that when feeder or older animals are in-
, they may be clinically protected for 9-12 months; the duration
unity is shorter in younger pigs.[3]

CHAPTER III

EXPERIMENTAL DISEASE

Materials and Methods

perimental Animals

Experimental animals used in this experiment consisted of a litte 8 purebred Yorkshire piglets obtained from Oklahoma State Universit condary specific pathogen free (S.P.F.) herd. All were farrowed nat ly and left with their dam until they reached 2 days of age and ther re placed in isolation at the College of Veterinary Medicine. Four oups of 2 pigs each were kept in individual stainless steel cages. oup was left in a separate isolator (separate room) as uninoculated ntrols. Pigs were fed substitute milk (Similac*) three times daily e rate of 2 oz. per feeding. To assure clearance of possible lacto- nic immunity, pigs were not exposed to TGE virus until approximately hours after separation from the sow.

rus

The Purdue strain of TGE virus was used for infecting susceptible glets. The viral pool contained 10^6 pig infectious doses (PID) per lliliter. The virus preparation was thawed and diluted at the rate 10 with transport media.

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*Similac. Ross Laboratories, Columbus, Ohio 43216.

·iment in Pigs

Each piglet (except the controls) was administered 2 ml of diluted preparation orally by syringe at 5 days of age. Infected pigs examined three times daily. Pigs were euthanized at 24 hours, urs, 72 hours, and 96 hours postinoculation. Control pigs were nized at 24 hours and 96 hours postinoculation. They were anesthe- with barbiturates (Pentobarbital Sodium Solution*) and annular pen sections of small intestine were collected at about 30 cm. vals through the length of the small intestine from the duodenum e ileocecal valve. The intestinal mucosal surface was carefully d with saline solution.

The pH of the gut content was examined at 3 different levels: num, jejunum-ileum, and colon. After completion of the intestinal ction, the animals were exsanguinated and sections of kidney, brain , spleen, mesenteric lymph node and lung were obtained. The in- nal segments remaining between the sites of tissue collection were to be used for virus isolation. Tissues for histopathologic exami n were fixed by immersion in neutral buffered 10% formalin and 's solution. Additional sections of small intestine were preserved utaraldehyde for possible electron microscopic examination. Frozen ons of mesenteric lymph node, liver, and small intestine were pro- d for immunofluorescence. Histologic sections for light microscopy embedded in paraffin, cut 5 um thick, and stained with hematoxylin osin.

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*Pentobarbital Sodium Solution. Fort Dodge Laboratories, Inc., Dodge, Iowa 50501

A susceptible 4-day-old pig was inoculated with a filtrate of inal contents, filtered through a disposable filter assembly of * obtained from the infected pigs.

Results

The results obtained in this exercise correspond to those described e literature.[16,21,24,26,27] The incubation period of the disease pproximately 18-24 hours. The first clinical signs, vomiting and e, watery, yellow diarrhea were evident in all 6 infected pigs urs post inoculation. At this time pig #1 was found severely deted, weak, and unable to rise. Other affected pigs continued to nd drink.

Primary gross findings were the presence of a moderate amount of curd that filled the stomach, and the distention of the gastrointes tract with yellowish, watery fluid which in some cases contained sh flecks of milk curd. The intestinal wall was thinner than that e noninfected control pigs, and the mesenteric blood vessels were sted. The carcasses were, in general, very thin and dehydrated. gross lesions not related to the disease were found in control Control pig #1 had a severe colitis and control pig #2 developed teral aspiration pneumonia. Among infected pigs, the lesions were icted to the gastrointestinal tract.

The pH values were variable among the infected pigs, ranging from 7 in the small intestine and 7 to 8 in the colonic contents. Reobtained from control pigs were similar to those of infected pigs.

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*Gelman. Ann Arbor, Michigan 48106.

biologic examinations were reported as a growth of E. coli from the intestine and colon.

Microscopically, the characteristic villous atrophy was present in nfected pigs. The jejunum was the most severely affected section e small intestine. Duodenum and ileum were affected less severely onstantly. The lesions were characterized by extensive areas of action of the lamina propria with marked shortening of the villi re 1). In contrast, the controls had long villi (Figure 2A,B). illous surface was covered by undifferentiated, flat to cuboidal elial cells. Cytoplasmic vacuolation was observed in some epithe- cells at the tips of villi. The lamina propria had an increased larity, however, inflammatory cells were absent (Figure 3). The s of Lieberkuhn were hyperplastic and tall, and numerous mitotic es were seen in pigs killed during the regenerative stage of the se, 72 hours and 96 hours postinfection (Figure 4). The villous- h/crypt-depth ratio was obviously decreased compared to those of oninfected controls which had the normal ratio of approximately in infected pigs the ratio ranged between 2:1 and 1:1 or less.

A positive confirmation of TGE virus was made by demonstrating cytoplasmic fluorescent material at the tips of the villi. Immuno- escence was positive in samples from all 6 infected pigs examined lahoma Animal Disease Diagnostic Laboratory (OADDL) (Figure 5). ttempts were made to isolate the virus and both were negative. -day-old pig inoculated with a filtrate of intestinal contents ob- d from the infected pigs developed signs and lesions of TGE. By AT this pig had demonstratable antigen in its intestinal epithelium

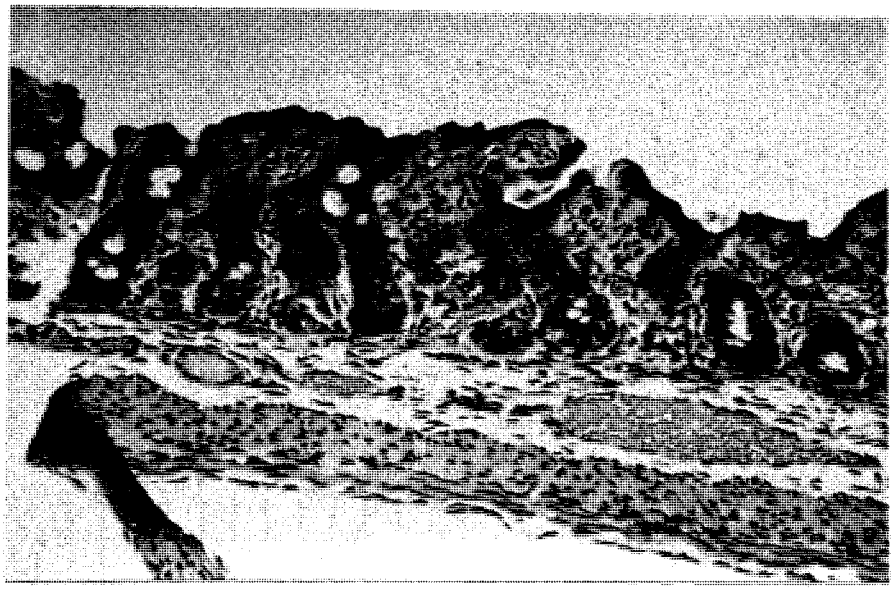

Figure 1. Jejunum, 24 hours postexposure. Observe marked shortening of villi, contraction of the lamina propria, and fusion of villi. Villous surface is covered with immature, cuboidal epithelial cells.

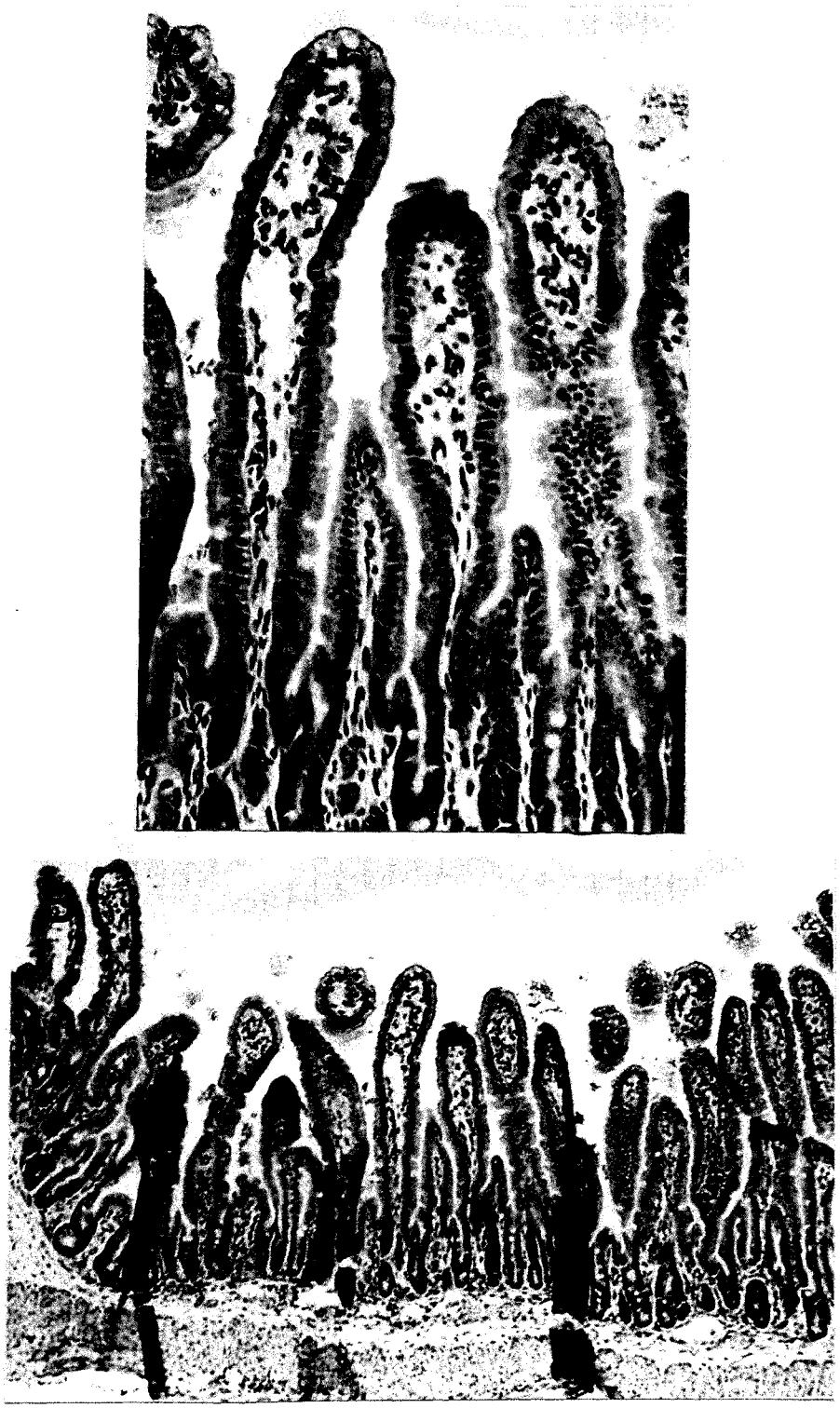

Figure 2. Small intestine of uninfected control pig. A. Villous surface is covered by mature, tall, columnar epithelial cells. B. Note length of the villi and depth of crypts.

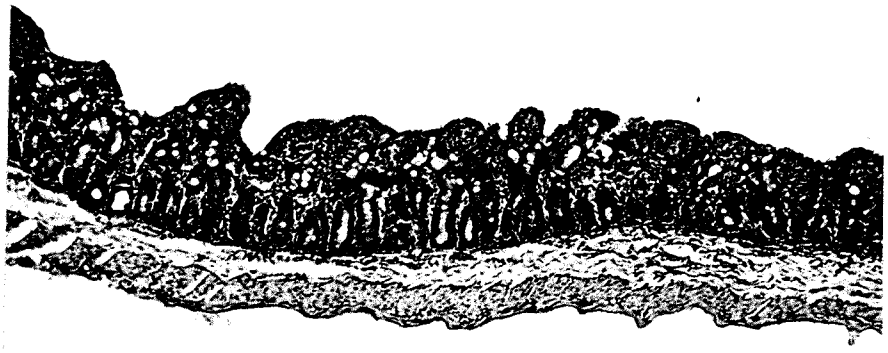

Figure 3. Jejunum, 72 hours postexposure. Observe marked villous atrophy with increased cellularity of the lamina propria.

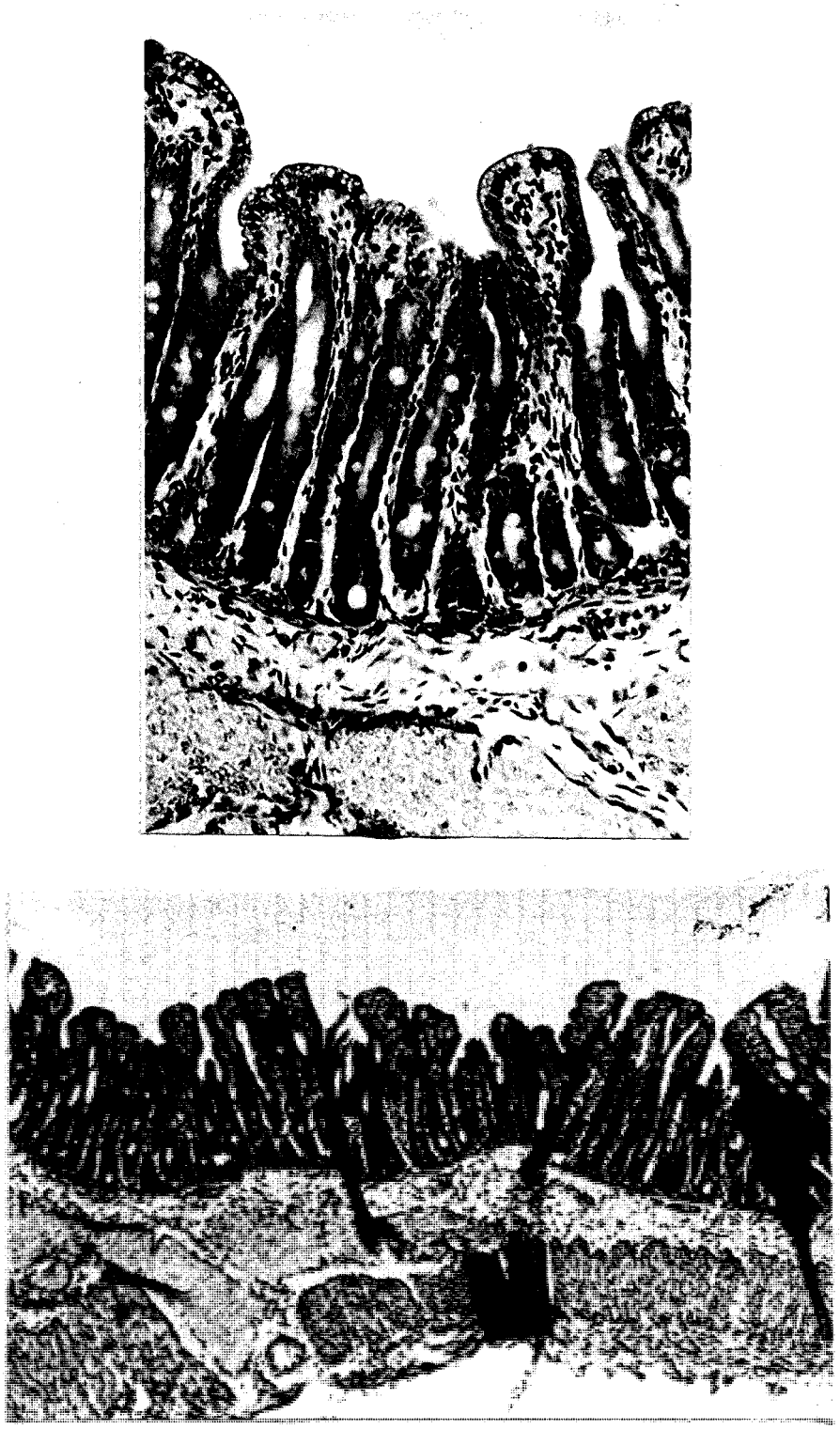

Figure 4. Jejunum, 96 hours postexposure. Depth of crypts of Lieberkuhn is increased due to hyperplasia. Length of villi is returning to normal in this regenerative stage of disease.

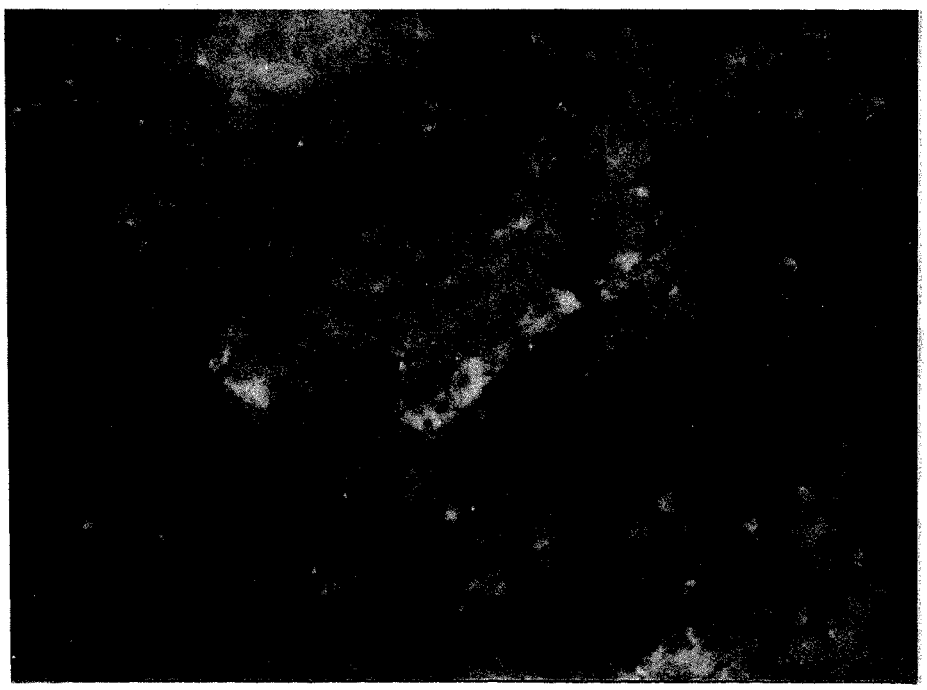

Figure 5. Fluorescent-antibody treated section of small intestine from an experimentally infected pig. Epithelial cells contain yellow-green fluorescing viral antigen.

Discussion

The experimental disease had the clinical and pathologic changes acteristic of TGE. The alkaline pH values obtained in the colonic ents did not correspond to those described in the literature.[11,72] might be explained by the growth of contaminant E. coli in both l and large intestine. Failure to isolate the virus is also relate ighly contaminated samples. E. coli has been reported to be a ndarily invasive agent of the intestinal mucosa in TGE infected .[21] It increases the severity of clinical signs and the mortality , and prolongs regeneration of the intestinal mucosa.[21]

CHAPTER IV

SUMMARY AND CONCLUSIONS

The nature, epidemiology, immunology, and the gross and microscopic
ns of TGE have been reviewed.

Transmissible gastroenteritis is a specific infectious disease of
caused by a coronavirus and characterized by a short incubation
d, vomiting, diarrhea, dehydration, and high mortality among suckl-
igs. Older susceptible pigs may be infected, but the severity of
linical disease is greater in newborn infected pigs. Histologi-
, TGE is characterized by degeneration of intestinal villous epi-
al cells, and by villous atrophy in the small intestine. Lesions
ore severe in the jejunum than in the duodenum and ileum. These
ns constitute the basis for the severe diarrhea and dehydration and
esponsible for death. Rapid replacement of damaged epithelial cell
esult in recovery from the disease. Because epithelial replacement
s much more rapidly (2 to 4 days) in older pigs than in baby pigs
10 days), the mortality rate in older swine is considerably lower
in neonates.

The lamina propria and crypts are not directly affected by the
. However, crypt hyperplasia occurs in response to the loss of
on the villous surface. As is well known, epithelial cell renewal
e small intestine is normally confined to crypts of Lieberkuhn
immature crypt cells proliferate and then migrate onto the villi.

, they differentiate into mature columnar epithelial cells. In pig survive infection, absorptive epithelial cells destroyed by TGE s are rapidly replaced by immature epithelial cells which are comtively resistant to virus replication.

The passive immunologic mechanism of TGE is directly associated the production of secretory IgA antibody in the colostrum of sows have been orally exposed to TGE virus. Secretory IgA is not broke by digestive enzymes and is not absorbed into circulation but is rbed onto the epithelial cell surface. Circulating IgG immunoglobu have little, if any, immunologic importance on the protective anism of TGE.

The mechanism of active immunity is unknown; it occurs as a result rior infection of the intestinal tract with TGE virus. In the Unit es, there is 1 (one) modified live-virus vaccine licensed by the rinary Biologics Division of the United States Department of Agricu . Swine recovered from TGE are immune when subjected to challenge.

Presumptive diagnosis of TGE is based on rapid spread, age incie, clinical signs and the presence of severe villous atrophy in the l intestine. Several serologic tests, virus isolation, and immunorescence techniques may be used for confirmation of TGE in infected .

In conclusion, although TGE is an infectious disease that lately been minimized (epidemiologically and clinically), it still is and be a health hazard within swine herds until an efficaceous vaccine eveloped. Therefore, extensive research should be continued on bot ive and active immunity of TGE.

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VITA

Rixa R. Rincon de Liendo

Candidate for the Degree of

Master of Science

Report: TRANSMISSIBLE GASTROENTERITIS OF SWINE: A REVIEW OF CURREN CONCEPTS

Major Field: Veterinary Pathology

Biographical:

Personal Data: Born in Maracaibo, Venezuela, May 1, 1952, the daughter of Mr. and Mrs. Roberto R. Rincon; married to Guillermo Liendo in December, 1977 and have one daughter, Andreina.

Education: Graduated from Rafael Maria Baralt High School, Maracaibo, Venezuela, in July, 1969; received Medico Veterinario degree from Universidad del Zulia, Maracaibo, Venezuela in September, 1975; completed requirements for Master of Science degree at Oklahoma State University in December, 1981.

Professional Experience: Worked as veterinarian at a swine far in Maracaibo, Venezuela for one year in 1976. Worked as Veterinarian I at the Public Health Department and Zoonosi in San Fernando de Apure, Venezuela, 1977-1978.