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PESTE PORCINA AFRICANA
Y
PESTE PORCINA CLASICA

(parcialmente anotada)

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INSTITUTO INTERAMERICANO DE CIENCIAS AGRICOLAS

CENTRO INTERAMERICANO DE DOCUMENTACION, INFORMACION Y COMUNICACION AGRICOLA – CIDIA

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INTRODUCCION

Con el objeto de colaborar con los países de América Latina y el Caribe que afrontan en la actualidad, problemas en su economía pecuaria debido al brote de la Peste Porcina Africana, el CIDIA divulga en esta oportunidad la información sobre las investigaciones llevadas a cabo para controlar esta enfermedad infecciosa del cerdo.

A las 246 referencias identificadas sobre Peste Porcina Africana se agregaron 876 citas bibliográficas sobre Peste Porcina Clásica con la finalidad de permitir estudios comparativos entre las dos manifestaciones de los virus transmisores de estas enfermedades.

A las referencias bibliográficas se les agregan resúmenes cuando identificados en las respectivas unidades documentarias.

El Indice de Autores que se presenta al final de este documento permite al usuario de la información, identificar los autores personales e institucionales indizados en la lista bibliográfica.

Biblioteca y
Terminal de Servicios
Turrialba, Costa Rica
octubre de 1978

INTRODUCTION

With the purpose of collaborating with the countries of Latin America and the Caribbean that are at present facing problems in their economy due to the outbreak of African Swine Fever, CIDIA is publishing in this opportunity the information on research carried out to control this swine disease.

To the 246 references identified on African Swine Fever, 876 bibliographies on Classical Swine Fever were added with the purpose of permitting comparative studies between the two propagating virus' of these diseases.

Summaries are included when it appears in the original document.

The Author Index presented at the end of this document permits the user of the information to identify the personal and institutional authors indexed in the bibliographical list.

Library and Terminal Services
Turrialba, Costa Rica
October, 1978

PESTE PORCINA AFRICANA

PESTE PORCINA AFRICANA

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Co-autores: S. S. Stone, S. Bachrach y W. R. Hess.

ALMEIDA, J. D., WATERSON, A. P. and PLOWRIGHT, W. The morphological characteristics of African swine fever virus and its resemblance to *Tipula iridescent* virus. Archiv für die Gesamte Virusforschung 20:392-396. 1967. -0002-

BELLANI, L. and CAPORALE, G. Epizootiological mass investigations for the identification of hog cholera (H.CH.) and African swine fever (ASF) virus carrier pigs slaughtered in Italy and for the isolation of the respective viruses from stored pork of national and foreign origin. Bulletin de l'Office International des Epizooties 74:539-560. 1975. -0003-

BLACK, D. N. and BROWN, F. Purification and physicochemical characteristics of African swine fever virus. Journal of General Virology 32(3):509-518. 1976. -0004-

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_____, ORDAS ALVAREZ, A. and SANCHEZ BOTIJA, C. Fluorescent antibody test for African swine fever. Revista del Patronato de Biología Animal 14: 115-132. 1970. -0007-

BOULANGER, P. et al. Diagnosis of African swine fever by immunofluorescence and other serological methods. Bulletin de l'Office International des Epizooties 66:723-739. 1966. -0008-

Co-autores: G. L. Bannister, A. Greig, D. P. Gray y G. M. Ruckerbauer.

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Co-autores: P. Bannister, G. L. Gray, G. M. Ruckerbauer y N. G. Willis.

BOULANGER, P. et al. African swine fever. III. The use of the agar double-diffussion precipitation test for the detection of the virus in swine tissue. Canadian Journal of Comparative Medicine and Veterinary Science 31:12-15. 1967. -0010-

Co-autores: G. L. Bannister, D. P. Gray, G. M. Ruckerbauer y N. G. Willis.

_____ et al. African swine fever. IV. Demonstration of the viral antigen by means of immunofluorescence. Canadian Journal of Comparative Medicine and Veterinary Science 31:16-23. 1967. -0011-

Co-autores: G. L. Bannister, A. Greig, D. P. Gray y G. M. Ruckerbauer.

BRAÇO-FORTE JUNIOR, M. da C. African swine fever in Portugal. Anais da Escola Superior de Medicina Veterinaria 12:37-69. 1970. -0012-

BREESE, S. S. Jr. and DeBOER, C. J. Electron microscope observations of African swine fever virus in tissue culture cells. Virology 28:420-428. 1966. -0013-

_____ and DeBOER, C. J. Chemical structure of African swine fever virus investigated by electron microscopy. Journal of General Virology 1:251-252. 1967. -0014-

_____ et al. Electron microscopy of the interaction of African swine fever virus with ferritin-conjugated anti-body. Virology 31:508-513. 1967. -0015-

Co-autores: S. S. Stone, C. J. DeBoer y W. R. Hess.

BROTE DE fiebre porcina africana en Cuba. Avance Agrícola y Ganadero (México) 2(21):62-66, 68-72, 74. 1971. -0016-

BURRA, L. G. Differentiation of African and classical swine fever and pasteurellosis by the pathological features. Veterinariya no. 2:46-47. 1970. -0017-

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Co-autores: G. Gayot, C. Costes, G. Delclos y F. Plateau.

_____, COSTES, C. and PICARD, M. African swine fever: clinical and laboratory studies of the epidemic of January to March, 1974. Proceedings of the 20th World Veterinary Congress 3:2201-2203. 1976.
* Compendiado en. Veterinary Bulletin 47(9):681. 1977. -0019-

CARNERO, R., COSTES, C. and PICARD, M. Comparative experiences in the diagnosis of African swine fever. Presentado en Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. s.p. -0020-

COELHO, M. A. T. African swine fever in Angola. Revista Portuguesa de Ciencias Veterinarias 64:217-226. 1969. -0021-

* COGGINS, L. Growth and certain stability characteristics of African swine fever virus. American Journal of Veterinary Research 27(120):1351-1358. 1966. -0022-

Growths of African swine fever (ASF) virus in buffy coat and pig kidney (PK) cell cultures were similar except that the release of virus was delayed in the latter. In buffy coat culture, new-cell associated virus appeared between 6 and 8 hours and soon afterward in the culture fluid. Peak maximal virus titers of 10^6 hemadsorption (HAd_{50})/0.1 ml. were obtained in 36 to 72 hours. The virus was refractive to exposure to trypsin, ethylenediaminetetraacetate (EDTA), ultrasonic waves, and freezing and thawing. A residual amount of virus survived heating at 56 C, for 1 hour. Particle size of ASF virus ranged between 100 and 300 mμ. Virus titrations were generally accurate, especially when less sensitive batches of cultures were recognized and eliminated.

* _____ and MEUSCHELE, W. P. Use of agar diffusion precipitation test in the diagnosis of African swine fever. American Journal of Veterinary Research 27(117):485-488. 1966. -0023-

The agar diffusion precipitation test should be of value in the diagnosis of African swine fever (ASF). The test appears to be specific, is easier to perform than the hemadsorption diagnostic test, and would be especially useful in the field diagnosis of acute African swine fever. Lymph nodes, liver and kidneys were equally suitable test material. A specific ASF-antiserum of optimum titer, pre-ASF inoculation serum, or ASF control antigen were essential in the application of the agar diffusion precipitation test.

_____. A modified hemadsorption inhibition test for African swine fever virus. Bulletin of Epizootic Diseases of Africa 16:61-64. 1968. -0024-

* _____, MOULTON, J. and COLGROVE, G. Studies with Hinde attenuated African swine fever virus. Cornell Veterinarian 58(4):525-540. 1968. -0025-

African swine fever (ASF) virus was first attenuated in porcine kidney cell cultures by Malmquist and in porcine bone marrow cultures by Manso Ribeiro and colleagues, in Portugal. Before this time, most of the available ASF viruses were

extremely virulent and survivors of infection were rare. The development of attenuated ASF viruses has provided the opportunity to study the immunologic response of pigs to this virus. Some findings with the Hinde isolate of ASF virus, after attenuation in buffy-coat cultures, are reported here.

* COGGINS, L. Segregation of a nonhemadsorbing African swine fever virus in tissue culture. Cornell Veterinarian 58(4):12-20. 1968. -0026-

A nonhemadsorbing cytopathogenic virus was segregated from the hemadsorbing Uganda isolate of African swine fever virus by the terminal dilution technic in porcine buffy coat cultures. The cytopathic effect and growth characteristics of the virus in tissue culture were similar to the hemadsorbing virus, except hemadsorption was not observed. Specific immune serum neutralized the cytopathic effect. The virus was passaged 46 times by the terminal dilution technic and 65 times by the rapid passage method in porcine buffy coat cultures without the reappearance of hemadsorption. However, the nonhemadsorbing virus appeared to revert to hemadsorption when inoculated into swine. Hemadsorbing virus was recovered from 16 and nonhemadsorbing virus from 32 of the 68 swine inoculated with the nonhemadsorbing Uganda African swine fever virus. A second nonhemadsorbing African swine fever virus was segregated from the Hinde isolate of African swine fever virus, using the same terminal dilution technic. Both nonhemadsorbing viruses were capable of stimulating production of specific ASF precipitin antibody in swine, and all but three of the inoculated swine which were tested resisted challenge inoculation with virulent homologous African swine fever virus.

_____. African swine fever virus: pathogenesis. Progress in Medical Virology 18:48-63. 1974. -0027-

COLGROVE, G. S. Immunofluorescence and inclusion bodies in circulating leukocytes of pigs infected with African swine fever virus. Bulletin of Epizootic Diseases of Africa 16:341. 1968. -0028-

_____. Diagnosis of African swine fever by fluorescent antibody staining of blood films and buffy coat smears. Bulletin of Epizootic Diseases of Africa 17:39-44. 1969. -0029-

* _____, HAELTERMAN, E. O. and COGGINS, L. Pathogenesis of African swine fever in young pigs. American Journal of Veterinary Research 30(8): 1343-1359. 1969. -0030-

Viral assays and fluorescent antibody (FA) tests were conducted on tissues of newborn and older pigs killed sequentially between 8 and 152 hours after they were orally exposed

to African swine fever (ASF) virus. Quantitation and distribution of virus were correlated with histopathologic features. Infection usually started in tonsils and mandibular lymph nodes, although in a few newborn pigs there was evidence of primary infection in lungs or in mesenteric lymph nodes. Primary viremia was detected as early as 8th postinfection hour (PIH), and secondary viremia between 15th and 25th PIH. Spleen, liver, body lymph nodes, and lungs were the principal sites of secondary viral growth. At 30 hours, all tissues of newborn pigs contained some virus, and maximal titers were reached as early as the 72nd PIH. Generalization occurred later in older pigs. Specific immunofluorescence was not detected until relatively high titers of virus were found in tissues. In lymphatic tissue, viral antigen was initially in macrophages and reticular cells, and only in later stages of infection was it in lymphocytes. Hepatic cells and mononuclear cells in sinusoids were involved in the liver; septal cells and free macrophages in the lungs; megakaryocytes and blast cells in the bone marrow; and monocytes in the blood. In late stages of infection, viral antigen was found in the endothelium and tunica media of blood vessels in several tissues.

COMPAGNUCCI, M., MARTONE, F. and VACCARO, A. Behaviour of enzymes (GOT, GPT, AL, LDH, LAP, SDH, total acid phosphatase, alkaline phosphatase) in the serum of pigs with African swine fever. Veterinaria Italiana 20:385-394, 395-402. 1969. -0031-

_____, MARTONE, F. and VACCARO, A. Glycaemia and Taylor's test in African swine fever. Veterinaria Italiana 21:135-152, 153-166. 1970.
* Compendiado en Veterinary Bulletin 41(4):281. 1971. -0032-

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* CONCELLON MARTINEZ, A. La peste porcina africana y la clásica; métodos de diagnóstico y lucha contra las mismas. Barcelona, Aedos, 1965. 107 p. -0034-

* CONCLUSIONES Y recomendaciones: peste porcina africana. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal. Estudio no. 2. 1976. pp. 15-16. -0035-

* CONSULTA FAO/CEE DE EXPERTOS EN LA ERRADICACION DE LA PESTE PORCINA Y LA PESTE PORCINA AFRICANA, HANNOVER, 1976. Informe. FAO. Producción y Sanidad Animal. Estudio no. 1. 1976. 22 p. -0036-

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* Compendiado en Veterinary Bulletin 45(6):426. 1975. -0037-

COUBE BOGADO, S. Peste porcina africana. In Asociación de Veterinarios de Nueva Zelandia. Enfermedades de los animales domésticos: algunas enfermedades de los animales domésticos más comúnes de Sudamérica. Montevideo, Juan Angel Peri, 1969. pp. 648-650. -0038-

COWAN, K. M. Immunological studies on African swine fever. I. Elimination of the procomplementary activity of swine se[illegible] with formalin. Journal of Immunology 86:465-470. 1961. -0039-

* _____. Immunological studies on African swi[illegible]. II. Enhancing effect of normal bovine serum on the complement-fixation reaction. American Journal of Veterinary Research 24(101):756-761. 1963. -0040-

> The inclusion of dilute normal bovine serum (NBS) in complement-fixation (CF) reaction mixtures of the African swine fever virus (ASFV) system generally resulted in increased antibody and antigen titers. Two- to sixteenfold increases in antibody titer occurred and enabled detection of antibody in serums that otherwise was not possible. Antigen titers were increased approximately tenfold and provided a more sensitive assay procedure for following the effect of various treatments on antigen preparations. For diagnostic purposes, the increased sensitivity of the antigen detection test made it possible to demonstrate antigen in a limited number of tissues samples from swine infected with ASFV.

COX, B. F. and HESS, W. R. Note on an African swine fever investigation in Nyasaland. Bulletin of Epizootic Diseases of Africa 10:439-440. 1962. -0041-

CUBA. INSTITUTO NACIONAL MEDICINA VETERINARIA. Reporte preliminar del brote de fiebre porcino africana en Cuba. Habana, 1971. s.p. -0042-

DALSGAARD, K., OVERBY, E. and SANCHEZ BOTIJA, C. Crossed immunoelectrophoretic characterization of virus-specified antigens in cells infected with African swine fever (ASF) virus. Journal of General Virology 36(1): 203-206. 1977. -0043-

DARDIRI, A. H. and HESS, W. R. Pathogenesis of African swine fever in pigs. In U.S. Animal Health Association. Annual Meeting, 74th, 1970. Proceedings. s.l., 1971. pp. 213-224.
* Compendiado en Veterinary Bulletin 42(3):144. 1972. -0044-

_____, BACHRACH, H. L. and HELLER, E. Inhibition by rifampin of African swine fever virus replication in tissue culture. Infection and Immunity 4(1):34-36. 1971.
* Compendiado en Veterinary Bulletin 42(1):17. 1972. -0045-

DASKALOS, A. M. de O. y MENDES, A. M. Apontamentos sobre a leporização do virus da peste suina africana. Revista de Ciencias Veterinarias 59:35-36. 1962. -0046-

DAVYDOV, V. Ya. Histological changes in African swine fever. Trudy Vsesoyuznogo Instituta Eksperimentalnoi Veterinarii 29:209-216. 1962. -0047-

DeBOER, C. J. Studies to determine neutralizing antibody in Sera from animals recovered from African swine fever and laboratory animals inoculated with African swine fever virus with adjuvants. Archiv fur die Gesamte Virusforschung 20:164-179. 1967. -0048-

_____, HESS, W. R. and DARDIRI, A. H. Studies to determine the presence of neutralizing antibody in sera and kidneys from swine recovered from African swine fever. Archiv fur die Gesamte Virusforschung 27:44-54. 1969. -0049-

* _____, PAN, I. C. and HESS, W. R. Immunology of African swine fever. Journal of the American Veterinary Medical Association 160(4):528-532. 1972. -0050-

In areas where African swine fever (ASF) has become enzootic in domestic swine, subacute and chronic infections are no longer uncommon. Virus carriers may be increasingly involved in the maintenance and spread of the disease. A safe and satisfactory vaccine has not been developed. Subacute and particularly chronically infected swine develop high concentrations of complement-fixing and precipitating antibody, but their immunologic significance remains obscure. Advances made since 1960 and their contributions to the solution of problems posed by ASF have been enumerated and summarized. Studies to correlate the clinical manifestations of chronically infected swine with their immunologic response indicate development of hypergammaglobulinemia. Purification methods for the virus have been developed so that the role of the virus and its soluble antigens may be assessed separately.

DeKOCK, G., ROBINSON, E. M. and KEPPEL, J. J. G. Swine fever in South Africa (East African swine fever). Onderstepoort Journal of Veterinary Science and Animal Industry 14:31-93. 1940. -0051-

DELAY, P. D. and CARBREY, E. A. Experimentally induced hog cholera in pigs immunized with African swine fever. Proceedings of the U. S. Livestock Sanitary Association 67:170-176. 1964. -0052-

_____ and SHARMAN, E. C. The effect of stressor viruses on pigs inoculated with attenuated African swine fever virus. Bulletin de l'Office International des Epizooties 63:733. 1965. -0053-

* DeTRAY, D. E. and SCOTT, G. R. The effect of hyperimmune hog cholera serum on the virus of African swine fever. Journal of the American Veterinary Medical Association 126(937):313-314. 1955. -0054-

Hyperimmune hog cholera antiserum had neither an *in vivo* nor an *in vitro* effect on African swine fever (ASF).

_____. African swine fever: a review. Bulletin of Epizootic Diseases of Africa 5:475-478. 1957a. -0055-

* _____. African swine fever in wart hogs (*Phacochoerus aethiopicus*). Journal of the American Veterinary Medical Association 130(12):537-540. 1957. -0056-

African swine fever (ASF) was not readily transmitted to domestic swine by wart hogs. However, one apparently successful transmission, by this method occurred. African swine fever virus was recovered from a wart hog 54 days after it was exposed by inoculation. Previously, 17 days was the longest period confirmed for a wart hog to act as a carrier of ASF virus. Protection tests with wart hog serums in domestic pigs were inconclusive.

* _____ and SCOTT, G. R. Blood changes in swine with African swine fever. American Journal of Veterinary Research 18(68):484-490. 1957. -0057-

Pigs infected with African swine fever (ASF) developed a leukopenia beginning on the fourth day after exposure. The leukopenia was associated with a highly significant increase in the percentage of neutrophils, especially the juvenile forms, and a decrease in the percentage of lymphocytes. The leukopenia was indistinguishable from that associated with hog cholera infections. The erythrocyte counts, hemoglobin values, packed cell volumes and percentages of eosinophils, basophils and monocytes remained normal. Sedimentation rates were erratic.

* _____. Persistence of viremia and immunity in African swine fever. American Journal of Veterinary Research 18(69):811-816. 1957. -0058-

Eight domestic pigs, which survived for periods of 86 to 456 days after African swine fever (ASF) exposures, showed a persistence of viremia which was demonstrated by subinoculating susceptible pigs. Two of the 8 pigs showed a co-existence of viremia and circulating antibodies. Protective antibodies were demonstrated in the serums of these 2 pigs 27 and 33 days, respectively, after the initial exposure, but could not be demonstrated thereafter although the viremia persisted. Tw of these domestic pigs showed ASF viremia without showing clinical reaction to the original exposure. Domestic pigs, as well as wart hogs and bush

pigs, may become carriers and potential spreaders of ASF. Killed antigen vaccines failed to induce immunity to ASF. Resistance to reinfection with ASF virus may depend on a persisting infection rather than on the persistence of circulating antibodies.

DeTRAY, D. E. African swine fever - an interim report. Bulletin of Epizootic Diseases of Africa 8:217-223. 1960. -0059-

* _____, ZAPHIRO, D. and HAY, D. The incidence of African swine fever in wart hogs in Kenya - a preliminary report. Journal of the American Veterinary Medical Association 138(2):78-80. 1961. -0060-

African swine fever (ASF) virus was isolated from 5 of 9 wart hogs shot in one area of Kenya over a period of 10 days. Wart hogs may serve as inapparent carriers of ASF and constitute a potential threat to the swine industry.

* _____. African swine fever. Advances in Veterinary Science 8:299-333. 1963. -0061-

DORMAN, A. E. The control of African swine fever in Kenya. Bulletin de l'Office International des Epizooties 6:807-823. 1965. -0062-

* ELS, H. J. and PINI, A. Negative staining of a non-haemadsorbing strain of African swine fever virus. Onderstepoort Journal of Veterinary Research 44(1):39-45. 1977. -0063-

Since the application of negative staining, preceded by fixation, prevents the disruption and distortion of the capsid of the African swine fever virus, improved contrast and evaluation of the appearance and size of virus particles in the electron microscope is possible and, in addition, the icosahedral shape of the virus is demonstrable. The mature virus particle contains at least 2 capsid layers and an outer envelope.

ENJUANES, L., CARRASCOSA, A. L. and VIÑUELA, E. Isolation and properties of the DNA of African swine fever (ASF) virus. Journal of General Virology 32(3):479-492. 1976.

* Compendiado en Veterinary Bulletin 47(1):31. 1977. -0064-

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* Compendiado en Veterinary Bulletin 47(1):30-31. 1977. -0065-

Co-autores: A. L. Carrascosa, M. A. Moreno y E. Viñuela.

ENJUANES, L., CUBERO, I. and VIÑUELA, E. A sensitivity of macrophages from different species to African swine fever (ASF) virus. Journal of General Virology 34(3):455-463. 1977.
* Compendiado en Veterinary Bulletin 47(8):597. 1977. -0066-

* EVALUACION DEL estado sanitario del ganado porcino. Revista de la Asociación Argentina Criadores de Cerdos 52(612):9-27. 1973. -0067-

Incluye peste porcina y peste porcina africana.

FERNANDEZ E., J. L. Peste porcina africana; comentarios, síntesis y conclusiones. Revista Protinal (Venezuela) 18(1):17-25. 1971. -0068-

FIEBRE PORCINA africana en Cuba. Avance Agrícola y Ganadero (México) 2(22): 71-74. 1971. -0069-

GARCIA GANCEDO, A., RONDA-LAIN, E. and RUBIO-HUERTOS, M. Ultra-structure of the spleen and liver of pigs infected with African swine fever. Microbiología Española 27(3-4):177-189. 1974. -0070-

* GAYOT, G. et al. Peste porcina africana. Aislamiento e identificación en Francia. Hechos epidemiológicos, clínicos, anatomopatológicos y de laboratorio. Revista de Medicina Veterinaria (Argentina) 56(3):194. 1975. -0071-

Co-autores: R. Carnero, C. Costes, F. Paateau, G. Delclos y P. Cazaubon.

La peste porcina africana apareció en Francia y fue dominada en 1964, nuevamente en 1967 y en enero de 1974 en el sur, en el Depto. de Pireneos Atlánticos. Se considera que la aparición de la enfermedad en el Norte de España ha sido la causa. Las pruebas de laboratorio de hemadsorción, inhibición de la hemadsorción, la citolisis y la aparición de cuerpos de inclusión permitieron el diagnóstico en 48 horas en materiales originales y en 24 horas en tomas de sangre, en muestras de sangre de 48 horas post-inoculación. Dada la imposibilidad de diferenciar clínicamente las dos entidades se remarca la importancia de mantener una vigilancia permanente y un laboratorio de tal manera montado que pueda rápidamente efectuar el diagnóstico. (Dada la importancia económica de la industria porcina argentina es deseable que a nivel nacional exista permanentemente una capacidad de diagnóstico que pueda aminorar el desastre que sería la introducción de esta peste).

GIL-FERNANDEZ, C. et al. Non-specific and specific haemadsorption on normal pig kidney cells and cells infected with African swine fever virus. Microbiología Española 24(1):23-38. 1971.
* Compendiado en Veterinary Bulletin 42(8):524. 1972. -0072-

Co-autores: L. Alonso, A. García-Gancedo y E. Ronda.

_____, GARCIA-GANCEDO, A. and VILAS-MINONDO, P. Plaque formation by African swine fever virus in chick embryo fibroblasts in the absence of CO_2 atmosphere. Archives of Virology 52(3):207-216. 1976. -0073-

GONZALEZ J., G. Lapinization of African swine fever virus. Anales del Instituto de Investigaciones Veterinarias 18/19:197-202. 1969.
* Compendiado en Veterinary Bulletin 40(1):31-32. 1970. -0074-

GORET, P. La peste porcina africaine (maladie de Montgomery). Veterinarian 2(4):291-295. 1964. -0075-

También en: Bulletin Signaletique 14-26(9):222-247. 1965.

GREIG, A. The localization of African swine fever virus in the tick *Ornithodoros moubata porcinus*. Archiv für die Gesamte Virusforschung 39(1/3): 240-247. 1972.
* Compendiado en Veterinary Bulletin 43(3):129-130. 1973. -0076-

_____. Pathogenesis of African swine fever in pigs naturally exposed to the disease. Journal of Comparative Pathology 82(1):73-79. 1972.
* Compendiado en Veterinary Bulletin 42(6):381. 1972. -0077-

_____. The use of a microtitration technique for the routine assay of African swine fever virus. Archives of Virology 47:287-289. 1976.-0078-

GUDHAY G., B. Patogénesis de la peste porcina africana en lechones infectados por vía oral. Técnica en Agricultura y Ganadería (México) 3(39):48-50, 52, 54-56, 58. 1972. -0079-

GURZA O., J. Por qué la peste porcina africana sería una catástrofe en México? Porcirama (México) (5):22-24, 26. 1971. -0080-

HAAG, J. Evolution de la peste porcine africaine en Espagne. Encyclopédie Veterinaire Periodique 21:168-171. 1964. -0081-

_____, LARENAUDIE, B.et LUCAS, A. Le diagnostic experimental de la peste porcine africaine. Bulletin de l'Office International des Epizooties 63:143-162. 1965. -0082-

_____, LARENAUDIE, B. et RUIZ GONZALVO, F. Peste porcine africaine; action de la 5-iodo-2 desoxyuridine sur la culture du virus *in vitro*. Bulletin de l'Office International Epizooties 63(5-6):717-722. 1965. -0083-

HAAG, J. et LARENAUDIE, B. Peste porcine africaine: l'effect cytopathogene du virus en culture leucocytaire. Bulletin de l'Office International des Epizooties 63:191-198. 1965. -0084-

_____ et LARENAUDIE, B. Peste porcine africaine: réaction d'hémadsorption; standardisation de la technique de Tubiash pour les cultures leucocytaires. Bulletin de l'Office International des Epizooties 63:163-167. 1965. -0085-

_____ et al. Peste porcine africaine: recherches sur la taile et la morphologie du virus. Recueil de Médecine Vétérinaire 142(9):801-808. 1966. -0086-

Co-autores: A. Lucas, B. Larenaudie, F. Rufz Gonzalvo y R. Carnero.

* HAMMOND, R. A. and DeTRAY, D. E. A recent case of African swine fever in Kenya, East Africa. Journal of the American Veterinary Medical Association 126(938):389-390. 1955. -0087-

A recent outbreak of African swine fever in Kenya is described. The virus was isolated from domestic pigs and from 1 wart hog killed on the farm. Immediate quarantine, slaughter of all domestic swine, and burning the carcasses prevented this outbreak from spreading to adjoining farms.

HENRIQUES, R. P., PETISCA, J. L. N. and DURAO, J. C. Present situation in Portugal relating to swine fever and African swine fever. Bulletin de l'Office International des Epizooties 85(3/4):461-466. 1976. -0088-

HESS, W. R. and DeTRAY, D. E. The use of leukocyte cultures for diagnosing African swine fever (ASF). Bulletin of Epizootic Diseases of Africa 8:317-320. 1960. -0089-

* _____ et al. Propagation and modification of African swine fever virus in cell cultures. American Journal of Veterinary Research 26(110):141-146. 1965. -0090-

Several isolates of African swine fever virus (ASFV) were propagated in pig leukocyte and chicken embryo cell cultures. After a number of passages in either of these culture systems, the viruses were able to multiply and produce cytopathic changes in a strain of pig kidney cells (PK2a). Cytopathic changes and substancial virus yield were also produced in PK2a cells with ASFV isolates without previous adaptation when the infected cultures were subjected to frequent fluid change or frequent trypsinization. Attenuated strains of ASFV were obtained by serial passage in pig leukocytes and PK2a cells. One strain rapidly lost both virulence and immunogenicity, but with other strains, it was possible to obtain pigs that were immune to homologous virulent isolates. Their serums fixed complement and formed precipitates when reacted with ASFV antigen.

HESS, W. R. African swine fever virus. Virology Monographs 9:1-33. 1971.
* Compendiado en Veterinary Bulletin 42(11):708. 1972. -0091-

_____ and PAN, I. C. Laboratory diagnosis of African swine fever. In American Association of Veterinary Laboratory Diagnosticians. Annual Meeting, 17th, Roanoke, Va., 1975. Proceedings. pp. 33-42. -0092-

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HEUSCHELE, W. P. and COGGINS, L. Isolution of African swine fever virus from a giant forest hog. Bulletin of Epizootic Diseases of Africa 13(3):255-262. 1965. -0094-

_____, STONE, S. S. and COGGINS, L. Observations on the epizootiology of African swine fever. Bulletin of Epizootic Diseases of Africa 13:157-160. 1965. -0095-

_____ and COGGINS, L. Studies on the transmission of African swine fever virus by arthropods. Proceedings of the U.S. Livestock Sanitary Association 69:94-100. 1965. -0096-

* _____, COGGINS, L. and STONE, S. S. Fluorescent antibody studies on African swine fever virus. American Journal of Veterinary Research 27(117):477-484. 1966. -0097-

Preliminary investigations with fluorescein-labeled antibody on African swine fever virus (ASFV) antigen in porcine kidney cell cultures (PK-2a) and in infected swine tissues were described. Specific staining of ASFV antigen was proved by (1) failure to demonstrate fluorescent inclusions in uninfected preparations and (2) the blocking of fluorescent staining of antigen by prior treatment of infected preparations with unlabeled specific antibody. Specific fluorescent antigen first appeared in the cytoplasm of infected PK-2a cells at 10 hours postinoculation as finely granular or globular inclusions. Spread of infection from primary sites appeared to be by direct extension to adjacent cells. Cytopathic effect developed at 84 hours postinoculation, acellular areas being surrounded by cells containing considerable fluorescent material. Hog cholera-immune serum and rinderpest-immune serum did not prevent specific fluorescent staining of ASFV antigen. Leukocytes seen in preparations of buffy coat, spleen, gastric lymph node, and liver from both normal and ASF-infected pigs had considerable nonspecific fluorescence by the methods used in this study. Specific intracytoplasmic fluorescent inclusion globules of diagnostic value were consistently found in cells believed to be macrophages in tissue impressions from spleen, gastric lymph node, and liver of 5 pigs that died following an

acute infection with 3 isolates of ASFV collected from widely separated geographic locations. Liver tissue had the greatest number of such cells and appeared to be the tissue of choice for diagnosis by fluorescent antibody technique. Three pigs, which did not die in the acute course of the disease and were killed, did not have demonstrable specific fluorescent antigen in any tissue impressions, presumably due to the antibody in the tissues that blocked reaction of antigen and labeled antibody.

HEUSCHELE, W. P. Studies on the pathogenesis of African swine fever. I. Quantitative studies on the sequential development of virus in big tissues. Archiv für die Gesamte Virusforschung 21:349-555. 1967. -0098-

_____ and COGGINS, L. Epizootiology of African swine fever virus in warthogs. Bulletin of Epizootic Diseases of Africa 17:179-183. 1969. -0099-

_____ and HESS, W. R. Diagnosis of African swine fever by immunofluorescence. Tropical Animal Health and Production 5(3):181-186. 1973.
* Compendiado en Veterinary Bulletin 44(1):26. 1974. -0100-

* KONNO, S., TAYLOR, W. D. and DARDIRI, A. H. Acute African swine fever: proliferative phase in lymphoreticular tissue and the reticuloendothelial system. Cornell Veterinarian 61:71-84. 1971. -0101-

Proliferative cellular change was recognized as the predominant lesion in 13 pigs dying of acute disease after experimental infection with two African swine fever virus isolates. Proliferating cells were observed around the central and sheathed arteries of the spleen, in lymphoid tissues of the lymph nodes, in Kupffer's cells in hepatic sinusoids, and in vascular mesenchyma of liver, kidney, lung, and heart. Two morphologic cell types, basophil round cells and reticuloendothelial (RE) cells, are described. Simultaneous regressive changes included atrophy or focal degeneration in lymphatic tissues of the spleen and lymph nodes, cellular foci and central lobular necrosis in the liver, and hyaline droplet degeneration in epithelial cells in renal urinary tubules.

* _____ et al. Liver pathology in African swine fever. Cornell Veterinarian 61(1):125-150. 1971. -0102-

Co-autores: W. D. Taylor, W. R. Hess y W. P. Heuschele.

Lesions in livers from 65 experimentally infected pigs confirm the occurrence of subacute and chronic phases in African swine fever. Additionally, the description supplements

preceding reports which dealt almost entirely with the disease's acute phase. Principal lesions in livers of pigs dying in the acute phase were liver cell degeneration, accumulation of cellular debris and serous fluid in vascular connective tissues, and Kupffer's cell degeneration. Proliferation and, in a lesser degree, degeneration were the liver lesions considered characteristic of the subacute disease. Proliferating cell types included reticuloendothelial (RE) cells (Kupffer's cells and the RE cells in vascular connective tissue), lymphoid cells, and basophil round cells. Concurrent liver cell degeneration took the form of focal necrosis. Chronically infected livers characteristically included foci of lymphoid cells in the lobules and in vascular connective tissues and myeloid foci containing erythropoietic and granulopoietic cells. Distinct lesion patterns of the three phases appear progressive and probably are influenced by the virus's virulence as well as the sequence of tissue response.

* KONNO, S. et al. Spleen pathology in African swine fever. Cornell Veterinarian 62:486-506. 1972. -0103-

Co-autores: W. D. Taylor, W. R. Hess y W. P. Heuschele.

Lesions in spleen from 38 pigs experimentally infected with African swine fever virus are described. Spleen pathology characteristic of the disease's subacute and chronic phases is reported for the first time. Additional information supplements other workers' observations about the spleens of pigs dying of acute ASF. Comparative discussion contributes to understanding the pathogenesis of ASF infection. Pathologic changes characteristic of the acute phase spleen were diffuse degeneration of lymphatic tissue and severe hyperemia of hemorrhage in the pulp which usually accompanied vascular wall degeneration. Splenomegaly was usual in these pigs. Principal spleen lesions in the subacute phase were atrophy of lymphoid tissues concurrent with formation of multiple foci of myeloid cells in the pulp. Of the chronic phase lesions, the diffuse formation of myeloid cells in the pulp was outstanding. Regressive changes in lymphatic tissue- focal degeneration and atrophy- were less intense than those of the disease's subacute form. A less pronounced change, the proliferation of RE cells and basophil round cells, was observed in each of the disease's three phases.

KOVALENKO, Y. R., SIDOROV, M. A. y BUBRA, L. G. African swine fever. I. Routes of infection. II. Diagnostic significance of amylase activity in the pancreas. Trudy Vsesoyuznogo Instituta Eksperimental'noi Veterinarii 31: 336-342, 343-346. -0104-

KOVALENKO, Y. R. et al. Experimental infection of pigs with the virus of African swine fever. Trudy Vsesoyuznogo Instituta Eksperimental'noi Veterinarii 24:53-61. 1961. -0105-

Co-autores: B. G. Ivanov, A. G. Bakhtin y E. P. Isaenko.

_____. Les données scientifiques actuelles sur la peste porcine Africaine. Trudy Vsesoyuznogo Instituta Eksperimental'noi Veterinarii 29:177-200. 1962. -0106-

_____. Peste porcine africaine. Veterinariya 39(11):75-79. 1962. -0107-

_____, SIDOROV, M. A. y BUBRA, L. G. Biological properties of African swine fever virus. Doklady Akademii nauk imeni Lenina (1):35. 1964. -0108-

_____, BUBRA, L. G. and SIDOROV, M. A. Pathological and anatomical changes observed in African swine fever. Veterinariya 41(6):35-40. 1964. -0109-

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_____, SIDOROV, M. A. y BUBRA, L. G. Use of leucocyte culture for the differentiation of viruses of African swine fever and classical swine fever. Doklady Akademii nauk imeni Lenina no. 2. 1964. -0111-

_____, SIDOROV, M. A. and BUBRA, L. G. Experimental investigations on African swine fever. Bulletin de l'Office International des Epizooties 63:169-189. 1965. -0112-

KRIUKOV, N. N. et al. Diagnostics of African swine fever by the hemadsorption reaction in cultures of leukocytes. Veterinariya 10:19-22. 1965. -0113-

Co-autores: V. N. Suirin, N. R. Zorina, Z. L. Sorvacheva y B. I. Surin.

KRUPAL'NIK, V. L. Combined immunization of piglets with live vaccines in breeding herds. Sbornik Nauchnykh Trudov Moskovskaya Veterinarnaya Akademiya 73(1):154-157. 1974. -0114-

LARENAUDIE, B., HAAG, J. et LACAZE, B. Identification en France métropolitaine de la peste porcine africaine ou maladie de Montgomery. Bulletin de l'Academie Veterinaire de France 37(6):257-259. 1964. -0115-

_____, HAAG, J. et LACAZE, B. La peste porcine africaine. Encyclopedie Veterinaire Periodique 21:172-177. 1964. -0116-

_____, HAAG, J. et CARNERO, R. La purification du virus de la peste porcine africaine par le fluorocarbone. Bulletin de l'Office International de Epizooties 63(5-6):711-716. 1965. -0117-

LARENAUDIE, B. et al. Etude des propriétés biologiques et chimiques du virus de la peste porcine africaine en culture leucocytaire. Recueil de Médecine Vétérinaire 142:903-919. 1966. -0118-

Co-autores: J. Haag, R. Carnero y F. Ruíz Gonzalvo

LECLERC, J. J. Pig leucocyte culture: application to the diagnosis of African swine fever. Thesis. Alfort, Paris, Ecole National de Vétérinaire, 1970. 87 p. -0119-

LEITE VELHO, E. Observations sur la peste porcine en Angola. Bulletin de l'Office International des Epizooties 46:335-340. 1956. -0120-

_____. La peste porcine africaine. Bulletin de l'Office International des Epizooties 48:395-402. 1957. -0121-

LOMBARDO, A. African swine fever, its spread and harmfulness, and prophylactic measures taken in Naples Province. Acta Medica Veterinaria (Italia) 15:3-15. 1969. -0122-

LUCAS, A. et al. Essai de classification du virus de la peste porcine africaine. Recueil de Médecine Vétérinaire 142:1065-1068. 1966. -0123-

Co-autores: B. Larenaudie, J. Haag, R. Carnero y F. Ruíz Gonzalvo.

* _____, HAAG, J. y LARENAUDIE, B. Peste porcina africana (enfermedad de Montgomery). Trad. del francés por C. Sánchez Acedo. Zaragoza, Acribia, 1971. 123 p. -0124-

LUPINI, P. M. et al. Outbreaks of African swine fever in 1968 and related research. Atti della Societa Italiana delle Scienza Veterinaire 22:864-869. 1969. -0125-

Co-autores: A. L. Stammati, A. Ioppolo y Z. Orfei.

MacINTOSH, B. M. The propagation of African swine fever virus in the embryonated hens' egg. Journal of the South African Veterinary Medical Association 23:217-220. 1952. -0126-

* MALMQUIST, W. A. and HAY, D. Hemadsorption and cytopathic effect produced by African swine fever virus in swine bone marrow and buffy coat cultures. American Journal of Veterinary Research 21(80):104-108. 1960. -0127-

Swine bone marrow and buffy coat cells were cultured on a glass substrate, using 80 per cent mixture 199 and 20 per cent normal pig serum as nutrient medium. African swine fever (ASF) virus infected certain large granular cells (macrophages) and produced two distinct reactions-hemadsorption followed by

cytolysis. Fibroblast-like cells were not involved in the reaction and continued to proliferate in infected cultures. It is believed that the reactions observed are specific for differentiation of ASF and hog cholera viruses. Several different sources of ASF virus were used as inoculum; adaptation was not necessary. All of the strains studied thus far produced identical reactions. The time required for the appearance of a hemadsorption reaction depended upon the titer of the virus. As little as 0.05 ml. of infectious blood collected during the acute phase gave positive results in 24 hours or less. The results of titration of material containing ASF virus were comparable in bone marrow and buffy coat cell cultures; however, the latter were preferred as they were easier to prepare. Serum from survivor-carriers inhibited the hemadsorption reaction but did not neutralize the cytopathic effect to a significant degree. It is believed that the antigen responsible for hemadsorption is distinct from the infectious particle.

* MALMQUIST, W. A. Propagation, modification and haemadsorption of African swine fever virus in cell culture. American Journal of Veterinary Research 23(93):241-247. 1962. -0128-

Eight strains of African swine fever (ASF) virus were tested for their ability to produce a cytopathic effect in a strain of swine kidney cell cultures. One strain, the Hinde, became adapted to the cell cultures and was carried through 119 serial passages. In earlier passages, a cytopathic effect was produced only after prolonged incubation. Later, however, cytolysis occurred in 72 hours. Virus removed at the 8th, 48th and 75th passages was successfully propagated in cell strains derived from ovine and bovine sources. These cells were susceptible only after they had undergone alteration. The ASF virus that adapted to an established cell line of swine kidney cultures also produced a cytopathic effect in cultures of primary swine kidney cells. After a single back passage in pigs, however, the reisolated virus did not have the same cytopathogenic properties. The Hinde strain of ASF virus became modified between the 63rd and 75th serial passage in swine kidney cell cultures. As the virus became modified, it also produced less reaction in buffy coat cultures. Some modification may have occurred during prolonged incubation of infected cultures of ovine kidney cells; however, it was more pronounced when the virus was passed serially in "altered" ovine kidney cells. Erythrocytes from only three of ten animal species were adsorbed on ASF virus-infected cultures; however, erythrocytes from only two species failed to react when Newcastle disease (ND) virus was used as inoculum. Swine erythrocytes previously incubated with ND virus would still adsorb to ASF virus-infected cultures but not to those infected with ND virus.

* MALMQUIST, W. A. Serologic and immunologic studies with African swine fever virus. American Journal of Veterinary Research 24(100):450-459. 1963. -0129-

Swine surviving infection with African swine fever virus (ASFV) or infected with a modified form of virus developed hemadsorption-inhibiting and precipitating antibodies detectable in agar gel. The hemadsorption-inhibiting antibodies, which reached a maximum titer in 35 to 42 days, appeared to be specific for the virus involved. Precipitating antibodies, however, appeared to be nonspecific, and cross reactions occurred with a number of antigens closely associated with infected cell debris. With both types of antibodies, however, coexisting viremia was possible. Hog cholera (European swine fever) hyperimmune serums contained neither hemadsorption-inhibiting nor precipitating antibodies. Buffy coat cell cultures, prepared from a survivor-carrier with donor serum in the medium, were protected against cytolysis, and there was no hemadsorption with a super-imposed infection by homologous virus. However, no protection was evident against most of the heterologous virus isolates. If the same buffy coat cells were washed and implanted with normal serum in the medium, they were completely susceptible to all isolates. The capability of inducing resistance in swine was related to the degree of virulence associated with the virus. If the virus still possessed enough virulent properties to produce a severe reaction in the infected pigs, it conferred protection against the homologous virulent isolate but not against heterologous isolates. However, if the virus became avirulent or nearly so through a high number of serial passages in cultures of porcine kidney cells, it did not confer protection against the homologous or heterologous isolates.

MANSVELT, P. R. The incidence and control of African swine fever in the Republic of South Africa. Bulletin de l'Office International des Epizooties 60:889-894. 1963. -0130-

MARTELL D., M. y CERON M., F. Peste porcina africana o fiebre porcina africana. México Ganadero 14(162):22-24. 1971. -0131-

MATSON, B. A. An outbreak of African swine fever in Nyasaland. Bulletin of Epizootic Diseases of Africa 8(4):305-308. 1960. -0132-

* MAURER, F. D., GRIESEMER, R. A. and JONES, T. C. The pathology of African swine fever - a comparison with hog cholera. American Journal of Veterinary Research 19(72):517-539. 1958. -0133-

The lesions of African swine fever have been described and compared with those of hog cholera. The primary lesions in

both diseases are found in lymphatic tissues and in the walls of arterioles and capillaries. The vascular lesions cause edema, hemorrhage and infection, the principal gross lesions. The most significant difference between the lesions of the two diseases is the severe karyorrhexis of lymphocytes that occurs only in African swine fever.

MAURER, F. D., GRIESEMER, R. A. and JONES, T. C. African swine fever (East African swine fever. Wart hog disease). In Dunne, H. W., ed. Diseases of swine. 2nd. ed. Ames, Iowa State University Press, 1964. pp. 187-202. -0134-

_____. African swine fever. In Dunne, H. W. and Leman, A. D., eds. Diseases of swine. 4th. ed. Ames, Iowa State University Press, 1975. pp. 256-272. -0135-

MAZZARACCHIO, V. The occurrence of African swine fever in Italy. Annali dell Instituto Superiore di Sanitá 4:650-673. 1968. -0136-

MENDES, A. M. and DASKALOS, A. M. de O. Algunas tentativas para a liponizaçao de virus de peste suina en Angola. Revista de Ciencias Veterinarias 50: 253-264. 1955. -0137-

_____. Existiria em Angola a doença de Montgomery? Revista de Ciencias Veterinarias 50:306-340. 1955. -0138-

_____ and DASKALOS, A. M. de O. Studies on the lapinisation of swine fever virus in Angola. Bulletin of Epizootic Diseases of Africa 3:9-14. 1955. -0139-

_____. Considérations sur le diagnostic et la prophylaxie de la peste porcine africaine. Bulletin de l'Office International des Epizooties 56: 408-417. 1961. -0140-

_____. The lapinisation of the virus of African swine fever. Bulletin de l'Office International des Epizooties 58:707-727. 1962. -0141-

_____. Virus da peste suina, tipo africano, em carnes fumadas. Revista de Ciencias Veterinarias 59:307-311. 1964. -0142-

MEXICO. DIRECCION GENERAL DE SANIDAD ANIMAL. Peste porcina africana. Boletín del Colegio Nacional de Médicos Veterinarios Zootecnistas (México) 3(2): 31-33. s.f. -0143-

* MEYER, N. Status of African swine fever. Journal of the American Veterinary Medical Association 161(11):1531-1532. 1972. -0144-

* MILLIAN, S. J. and ENGLEHARD, W. E. Application of the conglutination complement absorption test to detect hog cholera antibodies. I. The technique. American Journal of Veterinary Research 22(88):396-400. 1961. -0145-

The conglutinin complement absorption test (CCAT) may be used to detect hog cholera antibodies. The results of parallel CCAT titrations using heat-inactivated periodate-and trypsin-periodate-treated antiserums are presented. The poorest results in terms of number of serums reacting and serum sensitivity (expressed as titers) were observed in the heat-inactivated group of serums. Data indicate that pretreatment of serums with either periodate or trypsin-periodate facilitates antibody detection. The sensitivities of these treated groups were comparable (titers as high as 1:256 were observed).

MONTEIRO, C. Peste suina en Angola. Pecuaria 1:217-245. 1947. -0146-

MORNET, P. Peste porcine africaine: apparition et évolution au Sénégal. In Sénégal. Laboratoire National de l'Elevage. Rapport sur le fonctionement 1959-1960. pp. 118-125. -0147-

* MOULTON, J. and COGGINS, L. Comparison of lesions in acute and chronic African swine fever. Cornell Veterinarian 58(3):364-388. 1968. -0148-

También en El Veterinario (México) 5(3):5-10, 27-29. 1968.

A comparison was made of the lesions of acute and chronic African swine fever (ASF) in 116 domestic pigs. Of these pigs, 42 inoculated with virulent Hinde strain of ASF virus, died about 8 days postinoculation and had lymphadenitis, splenitis, tonsillitis, nephritis, dematitis, ophthalmitis, hepatitis, gastroenteritis, meningoencephalitis, and pulmonary edema with beginning interstitial pneumonia. A group of 74 pigs were injected with attenuated Hinde strain of virus. Of these, 40 died or were killed *in extremis* about 19 days postchallenge and had lesions similar to those in the "acute" pigs, plus pericarditis, viral type interstitial pneumonia, lymphadenitis, and lesions in the inoculation site. The 34 pigs remaining in this group survived the challenge virus and were killed about 38 days afterwards. These pigs had pericarditis, interstitial pneumonia, hyperplasia of lymph nodes, hepatitis and meningoencephalitis.

* _____ and COGGINS, L. Synthesis and cytopathogenesis of African swine fever virus in porcine cell cultures. American Journal of Vererinary Research 29(2):219-232. 1968. -0149-

Porcine buffy coat (BC) cells infected with African swine fever (ASF) virus had chromosomal, nucleolar, and cytoplasmic degeneration and developed intracytoplasmic inclusion bodies beginning 12 hours after infection. Cytolysis began 20 hours

after infection and was widespread 40 hours after infection. The inclusions stained specifically for deoxyribonucleic acid (DNA) with the Feulgen and acridine orange techniques, resisted deoxyribonuclease digestion, localized fluorescent antibody, and incorporated ^{3}H-labeled thymidine. Similar cytologic and cytochemical changes were observed in porcine kidney (PK) 2a cells infected with cell culture-attenuated strains of the virus, but the time sequence of changes was extended. Viral synthesis in BC cells was partially inhibited with 5-iododeoxyuridine (IUDR), 5-fluorodeoxyuridine (FUDR), 5-bromodeoxyuridine (BUDR), and hydroxyurea. The addition of BUDR to BC cultures at intervals between 2 and 20 hours and titration after 24 hours of infection indicated that viral DNA was synthesized between 6 and 7 hours, and infectious virus, between 10 and 11 hours.

* MOULTON, J. E. et al. Pathologic features of chronic pneumonia in pigs with experimentally induced African swine fever. American Journal of Veterinary Research 36(1):27-32. 1975. -0150-

Co-autores: I. C. Pan, W. R. Hess, C. J. DeBoer y J. Tessler.

Chronic pneumonia developed in 14 pigs inoculated with an attenuated strain of African swine fever (ASF) virus. The pathogenesis of the pneumonia was as follows: (1) Interalveolar septums became thickened by accumulation of lympocytes and monocytes; (2) lung developed focal areas of lymphocytes and macrophages; (3) necrosis began abruptly in these foci, beginning with the cells in the alveolar lumens, developing in centrifugal direction, and eventually affecting all structures in its path; (4) necrotic tissue became calcified; and (5) a mantle of mononuclear cells (including plasma cells) and fibrous tissue formed around the necrotic area. Viremia occurred in the 14 pigs at postinoculation day (PID) 14, and precipitating antibody was increased significantly at PID 58.

MOURA NUNES, J. F., VIGARIO, J. D. and TERRINHA, A. M. Ultrastructural study of African swine fever virus replication in culture of swine bone marrow cells. Archives of Virology 49(1):59-66. 1975. -0151-

* NEITZ, W. O. Peste porcina africana. In Enfermedades de importancia naciente de los animales. FAO. Estudios Agropecuarios no. 61. 1964. pp. 3-74. -0152-

NUNES PETISCA, J. L. Etudes anatomopathologiques et histopathologiques sur la peste porcine africaine (Virose L.) au Portugal. In Etudes et recherches sur la peste porcine africaine. Paris, Office International des Epizooties, 1965. pp. 103-142. -0153-

NUNES PETISCA, J. L. Quelques aspect morphologiques des suites de la vaccination contre le peste porcine africaine (virose L) au Portugal. Bulletin de l'Office International des Epizooties 63:199-237. 1965. -0154-

_____ and LIMPO SERRA J. J. B. Pathology of some diseases of domesticated animals. III. African swine fever. Vet. Moçambique 3:1-6. 1970. -0155-

A detailed description of the macroscopic changes in all affected organs and tissues. Colour plates show eight characteristic lesions. The relative frequency of the different lesions is listed.

ORDAS ALVAREZ, A. y SANCHEZ BOTIJA, C. Cambios en las propiedades del virus de la peste porcina africana y su repercusión en la epizootiología y el diagnóstico. Proceedings of the 20th World Veterinary Congress 3:2195-2200. 1976.
* Compendiado en Veterinary Bulletin 47(8):597. 1977. -0156-

ORTIN, J. and VINUELA, E. Requirement of cell nucleus for African swine fever virus replication in Vero cells. Journal of Virology 21(3):902-905. 1977.
* Compendiado en Veterinary Bulletin 47(9):681. 1977. -0157-

PAN, I. C., DeBOER, C. J. and HEUSCHELE, W. P. Hypergammaglobulinemia in swine infected with African swine fever virus. Proceedings of the Society for Experimental Biology and Medicine 134:367-371. 1970. -0158-

* _____, DeBOER, C. J. and HEUSCHELE, W. P. Immune response of swine to African swine fever infection. Journal of the American Veterinary Medical Association 156(9):1238-1239. 1970. -0159-

In the field, African swine fever (ASF) virus is maintained in wild pigs which are subclinical virus carriers. In domestic swine, ASF virus produces a highly infectious, generally fatal disease. Occasional survival can not be attributed with certainty to protective antibody. Two groups of 15 and 4 swine were inoculated with the 89th and 83rd swine bone marrow culture passage virus, respectively. The 83rd passage virus was attenuated to a much greater degree than the 89th passage, based on clinocopathologic manifestations. The group of 15 swine became carriers and experienced recurrent fever. All the swine in this group had absolute hypergammaglobulinemia with high complement-fixing (CF) and precipitating antibody titers detectable 21 days postinoculation (DPI) and thereafter. The electrophoretic patterns of increased *gamma* globulin were mostly polyclonal; however, monoclonal and diclonal patterns were also seen in several serum samples

collected in an early phase of the disease. Virus was recovered from most of the swine. The 4-swine group, given well-attenuated virus, had fever for 1 to 2 days within 7 DPI and had no recurrence. They resisted challenge inoculation with virulent virus. The *gamma* globulin levels of all swine in this group remained within normal limits. However, these swine had low CF and precipitating antibody titers but were apparently free from virus. Hypergammaglobulinemia may reflect continuous antigenic stimulation by persistent viral infection.

PAN, I. C. et al. Purification of African swine fever virus. Federation Proceedings 55:913. 1971. -0160-

Co-autores: C. J. DeBoer, W. R. Hess y S. S. Breese Jr.

_____, DeBOER, C. J. and HESS, W. R. African swine fever: application of immunoelectroosmophoresis for the detection of antibody. Canadian Journal of Comparative Medicine and Veterinary Science 36(3):309-316. 1972. -0161-

* _____ et al. African swine fever: comparison of four serotests on porcine serums in Spain. American Journal of Veterinary Research 35(6):787-790. 1974. -0162-

Co-autores: R. Trautman, W. R. Hess, C. J. DeBoer, J. Tessler, A. Ordas, C. Sánchez Botija, J. Ovejero y M. C. Sánchez.

Four serodiagnostic tests for African swine fever (ASF) were compared: reverse radial immunodiffusion (RRID), immunoelectroosmophoresis (IEOP), indirect immunofluorescence (IFI), and agar gel diffusion precipitation (AGDP). Test serums were obtained from swine in Spain where ASF has been enzootic. Since some serums gave positive results by one of the tests and negative results by the others, the minimal detectable antibody frequency was computed by combining the results of all tests on each serum. Three of the tests (RRID, IEOP, and IFI) each detected more than 80% of the antibody-containing serums. Combining results of 2 tests (RRID and IEOP, RRID and IFI, or IEOP and IFI) increased the efficacy of antibody detection to at least 95%. Only the RRID test could be used under field conditions, but its efficacy was reduced to 48% if the test result was taken in 2 instead of 18 hours.

* _____ et al. African swine fever: detection of antibody by reverse single radial immunodiffusion. American Journal of Veterinary Research 35(3): 351-354. 1974. -0163-

Co-autores: R. Trautman, W. R. Hess, C. J. DeBoer y J. Tessler.

A reverse single radial immunodiffusion (RRID) test was developed for detection of antibody in swine infected with

African swine fever (ASF) virus. A round plastic petri dich (60 by 15 mm.), containing agarose gel approximately 1.2 mm. thick and impregnated with an appropriate concentration of ASF virus-soluble antigen, could be prepared a week in advance of use. Fifty wells of 2.4 mm. size (diameter) were cut in each plate and filled (5µ 1./well) with test serums, using capillary tubes. Within 2 hours after the serum was applied, 71% of 453 serums known to contain antibody developed distinct rings; the efficacy increased to 87% by overnight holding (18 hours). Comparison of the results of RRI test with those of agar gel diffusion-precipitin (AGDP) and immunoelectroosmophoresis (IEOP) tests on the same serums indicated that the efficacy of either RRID or IEOP tests was much greater than that of the AGDP test. The RRID test was better than the IEOP test for serums from swine with acute ASF but slightly poorer for serums from vaccinated swine and from swine with chronic ASF. The application of RRID for detecting antibody seems feasible for both field and laboratory use.

* PAN, I. C. et al. African swine fever: hypergammaglobulinemia and the iodine agglutination test. American Journal of Veterinary Research 35(5):629-631. 1974. -0164-

Co-autores: R. Trautman, C. J. DeBoer y W. R. Hess.

Hypergammaglobulinemia in 17 domesticated swine with chronic African swine fever (ASF) induced by an attenuated viral isolate was accompanied by hypoalbuminemia without statistically significant changes occurring in the α1-, α2-, or β-globulin electrophoretic fractions. Results of the iodine agglutination test (IAT) on mixtures of test-positive porcine serum and normal bovine serum albumin depended directly on the γ-globulin content and inversely on the albumin content. All normal porcine serums gave a negative IAT result, but the serum protein changes in swine with chronic ASF were sufficient to give a positive test result in many, but not all, instances.

* _____, MOULTON, J. E. and HESS, W. R. Immunofluorescent studies on chronic pneumonia in swine with experimentally induced African swine fever. American Journal of Veterinary Research 36(4):379-386. 1975. -0165-

Chronic pneumonia experimentally produced in 14 pigs with African swine fever (ASF) virus was studied by immunofluorescene (IF) and histopathologic techniques. Frozen sections prepared from pulmonary tissues of the infected pigs were stained with fluorescein-conjugated antiserums against ASF viral antigen, porcine immunoglobulin G (IgG), porcine complement (C), and porcine fibrinogen. The viral antigen(s) was mainly seen in macrophages and cell debris in alveolar

walls and lumens. This finding indicates that the virus replicated in the cytoplasm of alveolar macrophages that subsequently degenerated and released the viral antigen. Diffuse immunoglobulin (Ig) deposition was found in necrotic cells and debris. Immunoglobulin also was seen bound to intracytoplasmic inclusion bodies in some degenerating alveolar macrophages. This finding indicates that antibody against ASF viral antigen(s) exuded from blood circulation or produced by local immunocytes (or both) reacted with viral antigen at intramacrophage and extra-macrophage levels and resulted in the formation of insoluble antigen-antibody (Ag-Ab) complexes. The participation of C in the immune complex was evident in the early stage of the pneumonia, but was less evident in the subsequent extensive, progressive necrotic processes. Fibrin deposits were visible only in the early necrotic area of alveolar walls and lumens. Possible mechanisms inducing extensive necrosis are discussed.

* PAN, I. C., SHIMIZU, M. and HESS, W. R. African swine fever: microplaque assay by an immunoperoxidase method. American Journal of Veterinary Research 39(3):491-497. 1978. -0166-

A microplaque assay for Vero cell-adapted Lisbon '60 strain of African swine fever virus (L'60-uncloned) and a large plaque-forming strain cloned from the L'60-uncloned strain was developed by an immunoperoxidase method. The immunoperoxidase method can be used to stain microplaque 3 days after inoculation, whereas the conventional plaque assay requires 5 to 7 days to develop visible plaques. A linear relationship between viral concentration in the inoculum and plaque numbers was observed. Viral titers obtained by both microplaque assay and conventional plaque assay were comparable, and both methods were reproducible and reliable. The viral titer obtained by either one of the plaque assay methods was approximately 0.9 $\log_{10}$ lower than that obtained by the hemadsorption test.

* PARKER, J. and PLOWRIGHT, W. Plaque formation by African swine fever virus. Nature 219(5153):524-525. 1968. -0167-

* PESTE AFRICANA - nuevo némesis del porcicultor. Agricultura de las Américas 21(5):32-33. 1972. -0168-

PESTE PORCINA africana. México Ganadero 14(165):34-35. 1971; 15(167):36-38. 1972. -0169-

* PESTE PORCINA africana. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal, no. 2. 1976. pp. 11, 14. -0170-

* PIEGAS, N. S. Peste suina africana. Biológico (Brasil) 40(8):223-227. 1974. -0171-

PINI, A. and WAGENAAR, G. Isolation of a non-haemadsorbing strain of African swine fever (ASF) virus from a natural outbreak of the disease. Veterinary Record 94(1/2). 1974.
* Compendiado en Veterinary Bulletin 44(5):299. 1974. -0172-

_____ and HURTER, L. R. African swine fever: an epizootiological review with special reference to the South African situation. Journal of the South African Veterinary Association 46(3):227, 229-232. 1975.
* Compendiado en Veterinary Bulletin 46(5):352. 1976. -0173-

_____. Isolation and segregation of non-haemadsorbing strain of African swine fever virus. Veterinary Record 99(24):479-480. 1976.
* Compendiado en Veterinary Bulletin 47(5):360. 1977. -0174-

_____ and HURTER, L. R. Situazione epizoologica della peste suina africana in Sud Africa. Selezione Veterinaria 18(4):357. 1976. -0175-

PLOWRIGHT, W. and FERRIS, R. D. African swine fever. In East African Veterinary Research Organization. Annual report 1956-1957. no. 20. -0176-

_____, BROWN, F. and PARKER, J. Evidence for the type of nucleic acid in African swine fever virus. Archiv fur die Gesamte Virusforschung 19: 290-304. 1966. -0177-

_____, PARKER, J. and STAPLE, R. F. The growth of a virulent strain of African swine fever virus in domestic pigs. Journal of Hygiene 66:117-134. 1968. -0178-

* _____, PARKER, J. y PEIRCE, M. A. African swine fever virus in ticks (*Ornithodoros moubata*, Murray) collected from animal burrows in Tanzania. Nature 221(5185):1071-1073. 1969. -0179-

_____, PARKER, J. and PEIRCE, M. A. The epizootiology of African swine fever in Africa. Veterinary Record 85(24):668-674. 1969. -0180-

* _____, PERRY, C. T. and PEIRCE, M. A. Transovarial infection with African swine fever virus in the argasid tick, *Ornithodoros moubata porcinus*, Walton. Research in Veterinary Science 11(6):582-584. 1970. -0181-

Adult ticks (*Ornithodoros moubata porcinus*) collected from warthog burrows were tested for infection by examining their excretions for African swine fever virus (ASFV). All of 3 infected female ticks from one burrow transmitted virus to their eggs and nymphal offspring and filial infection rates were high (55-81%). Nymphae infected transovarially transmitted virus regularly by feeding on pigs. Four pools of unfed first-stage nymphal ticks, collected from warthog burrows, contained ASFV.

Transovarial infection of the tick is one of the natural maintenance mechanisms for ASFV. The soft tick, *Ornithodoros moubata porcinus*, Walton (*sensu* van der Merwe, 1968), occurs in considerable numbers in large animal burrows in East Africa and is sometimes infected with African swine fever virus (ASFV). The main vertebrate reservoir of ASFV is the warthog (*Phacochoerus aethiopicus* Pallas) and we believe that infection in many localities occurs through the bite of infected ticks (Plowright et al., 1969a, 1969b). However, the level of viraemia in all infected warthogs examined to date would be inadequate to produce persistent infection in the arthropod. Transovarial infection of the tick has, therefore, been investigated as a possible mechanism for natural maintenance of the virus.

* PLOWRIGHT, W., PERRY, C. T. and GREIG, A. Sexual transmission of African swine fever virus in the tick, *Ornithodoros moubata porcinus*, Walton. Research in Veterinary Science 17(1):106-113. 1974. -0182-

Adult male ticks of the species *Ornithodoros moubata porcinus* were infected with 2 strains of African swine fever (ASF) virus by feeding on capillaries of infected pig blood. Virtually all of them became persistently infected and were placed in separate tubes with single clean females. No transfer of virus to the female was detectable at 8 days following copulation, as evidenced by the presence of an exospermatophore at the genital orifice; after the 48th day, however, 120 of 137 females (87.6%) were infected. Virus was probably transferred in the seminal fluid secreted by the accessory glands. Virus infection in sexually-infected females was systemic, as proved by the excretion of virus in the coxal fluid and by the ability to transmit ASF virus to pigs during "natural" feeding. The titer of virus in these ticks corresponded closely with that following an infective blood meal. Transovarial infection was not demonstrable in II batches of their eggs or unfed, first-stage nymphal offspring. Transfer of virus from infected female to clean male ticks occurred in only one of 35 instances where copulation had definitely occurred. It was concluded that male-to-female transmission of ASF virus occurs frequently in *O. moubata* and is probably an important factor in maintenance of the virus in tick populations. The phenomenon may well account for the 6-fold increase in tick infection rates, which was recorded between the late nymphal and adult stages in "wild" ticks collected from warthog burrows in N. Tanzania. The behaviour of ASF virus in *O. moubata* is closely analogous to that reported for *Rickettsia rickettsi* in *Dermacentor andersoni* and, less frequently, for *Borrelia duttoni* in *O. moubata*.

* PLOWRIGHT, W. Peste porcina africana. In Consulta FAO/CEE de Expertos en Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal, no. 2. 1976. pp. 5-7. -0183-

_____. A summary of research work on African swine fever reported to the EEC seminar. Presentado en Consulta de FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. s.p. -0184-

_____. Vector transmission of African swine fever virus. Presentado en Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. s.p. -0185-

* POLATNICK, J. and HESS, W. Altered thymidine kinase activity in culture cells inoculated with African swine fever virus. American Journal of Veterinary Research 31(9):1609-1613. 1970. -0186-

Baby hamster kidney (BHK) cells in the stationary phase of growth had increased thymidine (TdR) kinase activity when inoculated with African swine fever virus (ASFV). Enzyme activity of extracts from inoculated cells started increasing at 3 hours and reached a peak at the 26th hour postinoculation, when it was sevenfold higher than in normal cells. Thymidine phosphorylation was linear for the first 20 minutes at 37 C. The subsequent decrease in activity was apparently due to enzime thermal instability rather than substrate depletion. Enzyme extracts from normal and infected cells were similar in their kinetics and thermal stability, but differed significantly in their response to substrate concentration and in their pH-activity curves. Puromycin interfered with the increase in enzymatic activity in infected cells. African swine fever virus infection of BHK cells may induce the formation of a different TdR kinase than that in normal cells.

_____, PAN, I. C. and GRAVELL, M. Protein kinase activity in African swine fever virus; brief report. Archiv fur die Gesamte Virusforschung 44(2): 156-159. 1974. -0187-

POLO JOVER, F. y SANCHEZ BOTIJA, C. La peste porcina africana en España. Bulletin de l'Office International des Epizooties 55:107-175. 1961.
* Compendiado en Veterinary Bulletin 31(11):645. 1961. -0188-

_____ y SANCHEZ BOTIJA, C. 2° Informe sobre la peste africana en España. Bulletin de l'Office International des Epizooties 56:388-398. 1961. -0189-

_____ y SANCHEZ BOTIJA, C. 2° Rapport sur l'apparition de la peste porcine africaine en Espagne. Bulletin de l'Office International des Epizooties 56:399-407. 1961. -0190-

POLO JOVER, F. Rapport sur la peste porcine africaine en Espagne. Bulletin de l'Office International des Epizooties 63:57-70. 1965. -0191-

PRELIMINARY REPORT on the African swine fever epizootic in Cuba; methods of diagnosis and control. Bulletin de l'Office International des Epizooties 75(7/8):415-437. 1971. -0192-

QUESADA, A. African swine fever; diagnosis and action taken in the jurisdiction of the Mezzogiorno Research Station at Portici. Acta Medica Veterinaria (Italia) 15:17-27. 1969.
* Compendiado en Veterinary Bulletin 40(2):109. 1970. -0193-

REPORTE PRELIMINAR del brote de fiebre porcina africana en Cuba; métodos de diagnóstico y lucha. México Ganadero 14(162):25-29. 1971. -0194-

REUNION DE Consultation et d'Information de l'OIE sur la peste porcine africaine. Bulletin de l'Office International des Epizooties 67:999-1035. 1967. -0195-

RIBEIRO, A. M. R., VIGARIO, J. D. and LAGE, M. D. African swine fever in Portugal. Proceedings of the 20th World Veterinary Congress 3:2190-2194. 1976.
* Compendiado en Veterinary Bulletin 47(9):681. 1977. -0196-

RIBEIRO, J. M. and AZEVEDO, J. R. Réapparition de la peste porcine africaine (p.p.a.) au Portugal. Bulletin de l'Office International des Epizooties 55:88-106. 1961. -0197-

_____. Déclaration sur la vaccination contre la peste porcine africaine a la XXX Session Générale de l'Office International des Epizooties. Bulletin de l'Office International des Epizooties 58:1031-1040. 1962. -0198-

_____. Etude de la valeur spécifique de l'épreuve d'hémadsorption dans le diagnostic de la peste porcine africaine. In Symposium International de Virologie Vétérinaire, Lyon, 1962. Compt rendu. pp. 39-42. -0199-

_____ et al. Vaccination contre la peste porcine africaine. Bulletin de l'Office International des Epizooties 60:921-937. 1963. -0200-

Co-autores: J. L. N. Petisca, F. L. Frazao, y M. Sobral.

* RIBEIRO, L. O. C., SUGAY, W. e MUELLER, S. B. K. Comportamiento de linhagem celular IB-RS-2 contra a peste suína. Arquivos do Instituto Biológico de Sao Paulo 49(4):339-342. 1973. -0201-

The authors report two experiments done with the cell clones C-12, C-13 and C-19 of the IB-RS-2 swine kidney cell line,

in order to examine their ability to protect pigs against hog cholera. All of them protected the animals inoculated with 5mk of the cell lysates. Serial dilutions of the C-12 lysate inoculated into pigs showed that 5 to 10 cells contain enough antigen to induce protection. Besides, an inhibition of the protective factor was achieved by the incubation of a 10^{-3} lysate dilution of anti-hog cholera serum.

RODRIGUEZ, O. N. Alterations in the blood picture in the course of African swine fever. Revista Cubana de Ciencias Veterinarias 3(1):19-26. 1972. -0202-

_____ et al. Clinical and post-mortem observations on the outbreak of African swine fever in Cuba. Revista Cubana de Ciencias Veterinarias 3(1): 1-18. 1972.
* Compendiado en Veterinary Bulletin 44(7):400. 1974. -0203-

Co-autores: A. Fernández, E. Chamizo y E. del Pozo.

RUIZ GONZALVO, F. et al. Peste porcine africaine; adaptation d'une souche de virus aux cultures de rein de porc. Recueil de Médecine Vétérinaire p. 142. 1966. -0204-

Co-autores: J. Haag, R. Carnero y B. Larenaudie.

SANCHEZ BOTIJA, C. Diagnóstico diferencial entre la peste porcina africana. In Symposium International de Virologie Vétérinaire, Lyon, 1962. Compte rendue. pp. 43-50. -0205-

_____. Estudios sobre la peste porcina africana en España. Bulletin de l'Office International des Epizooties 58:707-727. 1962. -0206-

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_____. La peste suina africana. Zooprofilassi 18:587-607. 1963. -0208-

_____. Reservorios del virus de la peste porcina africana; investigación del virus de la P. P. A. en los artrópodos mediante la prueba de la hemoadsorción. Bulletin de l'Office International des Epizooties 60:895-899. 1963. -0209-

_____ y SANCHEZ BOTIJA, R. El diagnóstico de la peste porcina africana por la prueba de la hemoadsorción (Test de Malmquist et Hay). Bulletin de l'Office International des Epizooties 63:239-250. 1965. -0210-

SANCHEZ BOTIJA, C. and ORDAS ALVAREZ, A. Routine differential diagnosis of swine fever and African swine fever by a combination of immunofluorescence and haemadsorption tests. Bulletin de l'Office International des Epizooties 72(1):763-784. 1969. -0211-

También en: Revista del Patronato de Biología Animal 14:139-158. 1970.

_____, ORDAS ALVAREZ, A. and GARCIA GONZALEZ, J. African swine fever. I. Complement-fixing antibodies in outbreaks accompanied by low mortality. II. Indirect fluorescent antibody technique. Revista del Patronato de Biología Animal 14:133-138, 159-180. 1970.
* Compendiado en Veterinary Bulletin 41(12):1004-1005. 1971. -0212-

_____. Diagnosis of African swine fever by immunofluorescence. Bulletin de l'Office International des Epizooties 73:1025-1044. 1970.
* Compendiado en Veterinary Bulletin 41(12):1004. 1971. -0213-

_____ et al. Procedures in use for the diagnosis of African swine fever. Presentado en Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. s.p. -0214-

Co-autores: A. Ordas Alvarez, F. Ruíz Gonzalvo y A. Solana.

* SCOTT, G. R. Notes on animal diseases. XI. Virus diseases of pigs. East African Agricultural Journal 22(4):168-174. 1957. -0215-

Twelve virus diseases are known to affect domestic pigs and five of them are present in Kenya. The latter are discussed fully in this paper. Brief notes on the other infections are included because of the increased facility for the introduction of exotic diseases occasioned by modern forms of transport. In addition, atrophic rhinitis is discussed although the aetiology of the disease is still unknown.

_____. African swine fever. Veterinary Record 77:1421-1427. 1965. -0216-

_____. Peste porcine africaine: prevention, lutte contre la maladie, éradication. Bulletin de l'Office International des Epizooties 63(5-6): 765-777. 1965. -0217-

_____. Prévention et éradication de la peste porcine africaine, et moyens de lutte contre cette maladie. In Reunión Internationale F.A.O.-O.I.E. sur la Peste Porcine Classique et la Peste Porcine Africaine, Rome, 1965. -0218-

_____. The virus of African swine fever and its transmission. Bulletin de l'Office International des Epizooties 63:645-677. 1965. -0219-

SCOTT, G. R. and HILL, D. H. Failure to detect African swine fever virus in Nigerian dwarf pigs. Bulletin of Epizootic Diseases of Africa 14:55-57. 1966. -0220-

* _____. Comments on African swine fever. Journal of the American Veterinary Medical Association 160(4):532-533. 1972. -0221-

SHARMAN, E. C. African swine fever. In U.S. Animal Health Association. Annual Meeting, 75th, 1971. Proceedings. pp. 176-179. -0222-

* SHIMIZU, M., PAN, I. C. and HESS, W. R. Cellular immunity demonstrated in pigs infected with African swine fever virus. American Journal of Veterinary Research 38(1):27-31. 1977. -0223-

> Twenty-two pigs infected with African swine fever virus (ASFV) were used to demonstrate delayed hypersensitivity (DH) *in vitro* by the leukocyte migration-inhibition test. The results indicated that ASFV-infected pigs developed DH against ASFV antigen (ASF antigen) as early as 20 days after inoculation, and the presence of viremia did not interfere with the leukocyte migration-inhibition test. Three ASFV-infected pigs that were also sensitized to mycobaterium developed DH against both ASF antigen and mycobacterium. The conclusion was that the cellular immune systems is not impaired by ASFV infection in pigs.

SILVA, J. M. C. DA. African swine fever in Portuguese wild boar. Magyar Allatorvosok Lapja 32(11):733-734. 1977. -0224-

STEYN, D. G. Preliminary report on a South African virus disease amongst pigs. In South Africa. Veterinary Education and Research, 13th and 14th report of the Director, 1928. pp. 415-428. -0225-

_____. East Africa virus disease in pigs. In South Africa. Veterinary Service. Report of the Director 18:99-109. 1932. -0226-

STONE, S. S. and HESS, W. R. The effect of some chemical inactivants on the immunogenicity of African swine fever. In Réunion Internationale F.A.O.-O.I.E. sur la Peste Porcine Classique et la Peste Porcine Africaine, Rome, 1965. -0227-

_____ y HEUSCHELE, W. P. Le role de l'hippopotame dans l'épizootiologie de la peste porcine africaine. Bulletin of Epizootic Diseases of Africa 13(1):23-28. 1965. -0228-

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PESTE PORCINA CLASICA

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A rapid, simple fluorescent-antibody (FA) technique performed with tissue impressions was found useful for diagnosing hog cholera (HC). The technique employs a minimum of equipment and specialized procedures and can be conducted in less than 1 hour. Specificity and accuracy of results obtained with this technique compared favorably with specificity and accuracy obtained with fluorescent-antibody techniques performed with frozen tissue sections. Positive test results were obtained with tissues from 22 pigs experimentally inoculated 5 to 16 days previously and with tissues from 10 pigs with naturally occurring hog cholera.

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virus and 2 strains of virulent HC virus and cross-tested against each of the 8 strains using the Nebraska fluorescent-antibody (FA) technique. Heterologous strains could not be distinguished from homologous strains using this FA system. Distribution of positive cells in tonsils from pigs infected with virulent HC virus was distinctly different from that found in tonsils of pigs infected with attenuated HC virus.

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A mixed live virus was composed of the "Thiverval" strain of hog cholera virus and of the "Alfort-26" strain of Aujeszky's disease virus. These two attenuated strains

were obtained in tissue culture at low temperature by means of serial passages at limited dilution. Injected by intramuscular route, the mixed vaccine induced an immune response studied by means of the serum neutralizing antibodies kinetics and by means of the resistance against virulent challenge. Concerning humoral immunity, there was no significant difference in the production of serum neutralizing antibodies specific of both viruses between piglets vaccinated with the mixed vaccine and those inoculated with the "Thiverval" strain of hog cholera virus alone or with the "Alfort-26" strain of Aujeszky's disease virus alone. In other words antibodies specific of hog cholera and Aujeszky's disease viruses were produced in piglets vaccinated with a mixture of the "Thiverval" and "Alfort-26" strains. The mixed vaccine protected piglets against hog cholera virulent challenge, but at the present time these preliminary results could not conclude for its activity against Aujeszky's virulent challenge.

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Hog cholera virus adapted to monolayer cultures of porcine embryo kidney cells exerted a cytopathic effect. Attenuation was attained without loss of antigenic properties by serial passages on the kidney cells. Vaccine prepared from the adapted virus had a high titer and was stable. It did not regain virulence when serially passed 8 times through pigs, and was safely used without serum to immunize susceptible pigs. The immunity titer of the vaccine was higher when pigs were vaccinated without serum. The vaccine afforded partial protection on the 1st day and complete protection beginning the 3rd day after vaccination. Also it overcame serum block and immunized pigs vaccinated 3, 6 and 10 days after they had been given 15 cc. of serum.

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Co-autores: E. H. Bloch, L. Warner, F. Brooks, J. A. Anliker y M. H. Knisely.

The circulating blood and blood vessels of the bulbar conjunctiva of live pigs were studied with a high power dissecting microscope before and after inoculation with hog-cholera virus (*Tortor suis*). Prior to inoculation, the blood vessel walls appeared normal. Seven days after inoculation with hog-cholera virus, all, or almost all, the red cells were agglutinated into small soft masses which did not bulge the terminal arterioles. In some cases, these basic masses came together, forming larger, cohering aggregates, which broke up again quite easily. Over large parts of the conjunctiva of each pig, most vessels had contracted, obliterating the lumen. A special type of pigmentation of the bulbar conjunctiva, which is described in detail, made this contraction clearly visible.

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BRAN, L. et al. Immunization of young pigs against swine fever under form conditions, using strain C (Chinese) lapinized virus. Lucrarile Institutului de Cercetari Vet. Bioprep. Pasteur 6:35-45, 1969. -0332-

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Co-autores: S. Mihaita, M. Popa y N. Totorcea.

BROWN, L. N. et al. Hog cholera in Texas: an inflammatory comment. In International Pig Veterinary Society Congress, 4th, Ames, Iowa, 1976. Proceedings. pp. H6. -0334-

Co-autores: A. B. Anguio, W. L. Schwartz y J. W. Dunn.

BRUCKNER, A. H., SCOTT, R. M. and SWART, R. W. Two year stability of modified hog cholera vaccines, Swivax and Swivine. Allied Veterinarian 33:100-102. 1962. -0335-

BRUS, J. H. The value of field-vaccinations against swine fever using C-vaccine. In International Pig Veterinary Society Congress, 4th, Ames, Iowa, 1976. Proceedings. p. H15. -0336-

BRUYCKERT, M. DE. La peste porcine en 1974. Revue de l'Agriculture 28(4): 777-794. 1975. -0337-

BUCKSCH, U. Detection of neutralizing antibodies against the virus of diarrhoea-mucosal disease (BVD-MD) and precipitating antibodies against purified swine fever virus antigen in the serum of pigs using the micro-precipitation technique. Inaugural Dissertation. Hannover, Tierarztliche Hochschule, 1974. 53 p. -0338-

* BUENO, P. Alteraçoes cardiacas encontradas em casos de peste suina. Arquivos do Instituto Biológico (Brasil) 11:59-68. 1940. -0339-

Histological examinations of the heart have been made in 68 animals which died of spontaneous swine fever. Eighteen of these animals presented clearly developed degenerative and proliferative changes of the heart muscle. In 3 cases a diffuse interstitial myocarditis of a non suppurative character was seen. The microscopic picture was that of a globular degeneration of the muscle fibers with a proliferation of histiocytarian cells and infiltration by eosinophiles. Six other animals presented signs of an interstitial myocarditis developed only to a rather low degree. Multiple microscopic granulomas distinctly defined and formed by histiocytarian cells in a state of intense proliferation, were found in three other cases. Heavy degenerations of the muscle fibers, sometimes reaching a state of necrobiosis of large areas, were encountered three times; twice in association with inflamatory lessions. Multiple metastatic abcesses with preferential localization on papillary muscle of the left ventricle were verified in three other animals. The 50 other cases under examination were not always completely free from alterations of the heart muscle, but these in the majority of the animals observed were only slight. Besides these cases 28 animals with experimentally induced swine fever were examined showing only weakly developed histological changes of the heart muscle. Inflamatory symptoms of the endocard were observed in 6 cases, as well spontaneous as experimentally. An ulceration of the wall of a vene was seen in a single experimental case, this being the unique vascular change in our whole material. The described changes of the myocard are similar to those observed in some cases of sudden death in swine. The possibility is therefore suggested of the swine fever virus to be responsable for a certain number of sudden deaths, apparently without a sufficient explanatory cause.

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* BUTHALA, D. A. and MATHEWS, J. Electrophoretic evaluation of the commercial processing of hog cholera antiserum. Cornell Veterinarian 45:514-519. 1955. -0341-

Data have been presented which show a progressive alteration of serum proteins during commercial processing of hog-cholera antiserum. These changes were relatively small in all steps prior to pasteurization and phenolization. Pasteurization and phenolization resulted in diminishing the percentage of albumin and gamma globulin, with a compensatory rise in a component migrating in the alpha-beta-globulin zone. There was an 18.4 percent decrease in gamma globulin.

BUT'YANOV, D. D. and KOCKO, F. A. Effectiveness of ASV live, attenuated vaccine against swine fever. Veterinariya no. 5:71-72. 1970. -0342-

_____. Simultaneous immunization of pigs against swine fever and erysipelas. Veterinariya no. 2:39-41. 1970. -0343-

BYCHKOV, I. S. and ZABLOTSKII, T. M. Immunofluorescent method for detecting swine fever virus in pathological specimens. Trudy Moskovskoi Veterinarnoi Akademii 61:132-133. 1972. -0344-

CAGGIANO, A. F. Inmunización contra la peste porcina. Revista de la Asociación Argentina de Criadores de Cerdos 53(619):15, 17, 19. 1975. -0345-

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CAMPBELL, A. D. The swine fever programme in Great Britain. Proceedings of the U. S. Livestock Sanitary Association 1966:390-409. -0347-

CAMPBELL, C. L. The field use of modified virus vaccine. In U. S. Livestock Sanitary Association. Annual Meeting, 61th., St. Louis, Mo., 1957. Proceedings. St. Louis, Mo., 1958. pp. 276-281.
* Compendiado en Veterinary Bulletin 29(4):187. 1959. -0348-

CAPORALE, G. et al. Epidemiology, diagnosis and criteria for the prophylaxis of swine fever in Italy. Bulletin de l'Office international des Epizooties 72(1):849-883. 1969. -0349-

Co-autores: B. Castagnoli, G. L. Gualandi, L. Nardelli y V. Torlone.

CAPURSO, A. Experimental study of the residual pathogenicity of the lapinized Chinese strain of swine fever virus. Acta Medica Veterinaria 17(6):533-538. 1971.
* Compendiado en Veterinary Bulletin 42(9):593. 1972. -0350-

CAPURSO, A. Further studies of the residual virulence of lapinized C strain swine fever virus. Atti della Società Italiana delle Scienze Veterinarie 26:606-609. 1972.
* Compendiado en Veterinary Bulletin 43(11):609. 1973. -0351-

* CARACTERISTICAS DEL virus de la PP aislado recientemente en los EE.UU. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal no. 2. 1976. p. 8. -0352-

* CARBREY, E. A. The role of immune tolerance in transmission of hog cholera. Journal of the American Veterinary Medical Association 146(3):233-237. 1965. -0353-

The study of field cases of hog cholera has caused veterinarians to suspect the immune sow exposed to hog cholera virus during pregnancy of shedding virus at parturition through the bodies of her pigs. Although immune to the disease, the sow develops a viremia upon exposure to the virus, and the pigs are infected *in utero*. The unborn pigs are in the biologic state of immune tolerance, and the virus is able to produce a persistent infection which lasts until birth. Other reports indicate that this persistent infection with hog cholera virus has occurred naturally and can be produced experimentally. Other viruses(swine influenza, rubella, and poliomyelitis) have been found capable of infecting the developing embryo. Lymphocytic choriomeningitis of mice provides the most complete picture of a virus capable of producing a lasting infection with continual shedding of the virus after exposure *in utero* or shortly after birth. Immune tolerance may be responsible for the creation of persistently infected carrier animals which remain virus shedders for long periods of time. Such animals would harbor the virus during winter months when the insect vectors are not active and eliminate the need for a reservoir host to explain virus transmission. In the case of hog cholera, such persistently infected animals would serve to carry the virus to other farms or maintain the virus in the herd until a new group of susceptible pigs had been raised or purchased. Epizootiologic investigation of virus infections in the field should include attempts to isolate virus from pigs born after and epizootic, since these pigs could have been infected *in utero* while they were in a state of immune tolerance and, by this means, developed into persistent carriers of the virus.

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Co-autores: W. C. Stewart, J. I. Kresse y L. R. Lee.

* CARBREY, E. A., STEWART, W. C. and YOUNG, S. H. The changing picture of hog cholera: case studies. Journal of the American Veterinary Medical Association 149(12):1720-1724. 1966. -0355-

The concept of hog cholera (HC) as an acute, fatal disease of swine should be expanded to include a disease characterized by chronic signs of illness, recovery following supportive treatment in older pigs, mortality only in young pigs, and baby pig losses with congenital defects. Transmission of hog cholera virus by the pregnant sow at farrowing through the birth of baby pigs infected *in utero* with hog cholera virus; infection of susceptible pigs by contact exposure to pigs recently vaccinated with modified live-virus vaccines; and illness and losses in young feeders and baby pigs associated with hog cholera infections caused by virus strains of reduced virulence were observed.

* _____ et al. Transmission of hog cholera by pregnant sows. Journal of the American Veterinary Medical Association 149(1):23-30. 1966. -0356-

Co-autores: W. C. Stewart, S. H. Young y G. C. Richardson.

Hog cholera virus was detected in the tissues of baby pigs farrowed by sows which were exposed to infection during pregnancy. The fluorescent-antibody, tissue-culture technique (FATCT) was used to isolate and identify the virus, and pigs inoculation was employed for additional evidence in 5 of the 6 cases. The atypical nature of the hog cholera infections in these herds delayed recognition of the disease and emphasized the value of laboratory assistance in reaching a diagnosis. The ability of the pregnant sow to harbor and transmit hog cholera virus was confirmed.

* _____ et al. Confirmation of hog cholera diagnosis by rapid serum-neutralization technique. Journal of the American Veterinary Medical Association 155(12):2201-2210. 1969. -0357-

Co-autores: W. C. Stewart, J. I. Kresse y L. R. Lee.

A fluorescent antibody, serum-neutralization (FASN) test was developed and standardized for routine diagnosis use in the detection of serum antibody against hog cholera (HC) virus. Serum-neutralization titers were obtained by determining the highest serum dilution which caused a 90% reduction in the number of virus infectious units (VIU) on a coverslip cell culture. A test dose of HC virus of approximately 1,000 VIU was inoculated with each serum dilution on confluent PK-15 pig kidney cell cultures maintained in Leighton tubes. Employing a fourfold dilution scheme, $\log_{10}$, serum titers of 1.8 to 3.0 (1:64 to 1:1,024) were detected at least 48 days following parenteral exposure of 8 pigs to a field strain of

HC virus of low virulence. The mean $\log_{10}$ titer was 2.3, which corresponded to an actual serum dilution of 1:200. About 4 months after HC vaccination of 12 pigs as weanlings, $\log_{10}$ antibody titers of 1.2 to 2.4 (1:16 to 1:256) were obtained. After inoculation with virulent HC virus, the titer range of these pigs was 2.4 to 3.0. The mean post-vaccination titer was 1.8, which increased to 2.6 following exposure of the pigs to virulent virus. Serum titers were reported in pigs following ingestion of colostrum, exposure to bovine viral diarrhea virus, vaccination with inactivated virus, inoculation with antiserum, and short and long periods after exposure to HC virus.

CARBREY, E. A. et al. The incidence and characteristics of strains of hog cholera virus causing fetal abnormalities, death and abortion in swine. In Symposium on Factors Producing Embryonic and Fetal Abnormalities Death and Abortion in Swine, Chicago, Ill., 1967. Washington, D. C., Agricultural Research Service, 1969. pp. 111-116. -0358-

Co-autores: W. C. Stewart, J. I. Kresse y L. R. Lee.

_____ et al. Comparison of frozen section and cell culture immunofluorescent techniques for the detection of hog cholera infection in experimentally infected pigs. Proceedings of the Animal Health Association 74:502-514. 1970. -0359-

Co-autores: H. A. Mc Daniel, W. C. Stewart, E. J. Henry y J. I. Kresse.

_____ et al. Comparison of tissue section and cell culture immunofluorescent techniques for the detection of hog cholera infection in experimentally infected pigs. In U. S. Animal Health Association. Annual Meeting, 74th, Maryland, 1970. Proceedings. Maryland, 1971. pp. 502-514.
* Compendiado en Veterinary Bulletin 42(3):143. 1972. -0360-

Co-autores: H. A. Mc Daniel, W. C. Stewart, E. J. Henry y J. I. Kresse.

_____ et al. Bovine viral diarrhea infection in pigs and its differentiation from infection with hog cholera strains of low virulence. In Diagnosis and Epizootiology of Classical Swine Fever. Luxembourg, Commission of the European Communities, 1976. pp. 126-158. -0361-

Co-autores: W. C. Stewart, J. I. Kresse y M. L. Snyder.

* CARBREY, E. A. et al. Natural infection of pigs with bovine viral diarrhea virus and its differential diagnosis from hog cholera. Journal of the American Veterinary Medical Association 169(11):1217-1219. 1976. -0362-

Co-autores: W. C. Stewart, J. I. Kresse y M. L. Snyder.

Natural infection of pigs with bovine viral diarrhea virus (BVDV) through contact with infected cattle has caused problems in diagnosing hog cholera (HC). Low cross-reacting serum antibody titers against HC caused by BVDV infection were found in clinically normal pigs as well as those suspected of having HC. Bovine viral diarrhea virus was isolated from specimen tissues and initially identified as HC virus (HCV), using the fluorescent antibody cell culture technique. Additional cell cultures, as well as pig calf trials, were necessary to identify it as BVDV. The isolate caused clinical signs of illness in the calves, whereas the pigs remained healthy. Bovine viral diarrhea virus may be detected in tissue sections or isolated in cell cultures and confirmed as HCV, using the HC fluorescent antibody conjugate. Laboratories performing the neutralization test for HC should use descretion when interpreting HC titers unless BVD titers are determined on the same serums.

CASAS, J. L., MUDESPACHER, M. E. and MARTELL, M. The determination of efficacy of an heterotypical vaccine against hog cholera. In International Pig Veterinary Society Congress, 4th, Ames, Iowa, 1976. Proceedings. p. H3. -0363-

CASSELBERRY, N. W. and WALKER, W. W. Tissue culture modified live virus hog cholera vaccine. Proceedings of the U. S. Livestock Sanitary Association 55:230-233. 1951. -0364-

CASTAÑON A., J. Erradicación del cólera porcino. Porcicultura Mexicana 1(1):30-31. 1969. -0365-

* CASTRO, A. R., CALETTI, E. and CHANG SAY, F. Comparación antigénica de una cepa atenuada y una cepa virulenta del virus del cólera porcino. Revista de Investigaciones Pecuarias (Argentina) 1(1):85-90. 1972. -0366-

Por el método de la inmunodifusión se ha identificado un antígeno intracelular soluble, producido en el quinto pasaje de una cepa atenuada (lapinizada) propagada en cultivo de células renales de cerdo. Empleando la inmunoelectroforesis, se ha determinado que este antígeno posee carga eléctrica negativa. Al realizar estudios comparativos mediante inmunodifusión e inmunoelectroforesis, se identificó un antígeno precipitante en órganos infectados con cólera porcino, el que posee también carga eléctrica negativa.

Al parecer tal antígeno no está relacionado antigénicamente con el antígeno intracelular de la cepa atenuada. La producción de un suero hiperinmune preparado con conejos ofrece la posibilidad de realizar el diagnóstico del cólera porcino mediante la identificación del antígeno precipitante. La ausencia de reacción cruzada entre el antígeno precipitante de los órganos infectados y el antígeno intracelular de la cepa atenuada, permitiría eventualmente diferenciar ambas cepas, lo cual tendría gran valor en determinar brotes post-vacunales.

CASTRO, M. P. DE. An infectious agent causing "spontaneous" degeneration of swine *in vitro*. In Vitro no. 1:8-16. 1973. -0367-

CASTRUCCI, G. et al. Relationship between bovine viral diarrhea virus and hog cholera virus; the possible influence of the antigenic variability of BVD virus in the immunologic comparison between the two viruses. Bollettino dell'Istituto Sieroterapico Milanese 49(6):461-476.
* Compendiado en Veterinary Bulletin 42(4):204. 1972. -0368-

Co-autores: V. Torlone, V. Cilli y F. Titoli.

_____ et al. Capacity of strain TVM2 of bovine viral diarrhea (BVD) virus, at different passage level in cell culture, to protect pigs against hog cholera virus. Bollettino dell'Istituto Sieroterapico Milanese 52(2):85-88. 1973.
* Compendiado en Veterinary Bulletin 44(5):298. 1974. -0369-

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_____ et al. Immunological relationship between bovine diarrhea virus and swine fever virus; influence of the strain of swine fever virus on the result of cross-immunity experiments in pigs. Bollettino dell'Istituto Sieroterapico Milanese 52(4):309-314. 1973.
* Compendiado en Veterinary Bulletin 44(5):299. 1974. -0370-

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CESSI, D. Diagnosis of swine fever in Italy. In Seminar on Diagnosis and Epizootiology of Classical Swine Fever, Amsterdam, 1974. Luxembourg, 1976. pp. 17-18. -0372-

CILLI, V. Analogies and relationships between the virus of bovine virus diarrhoea and the virus of swine fever. Kteniatrika Nea 4(2-3):35-41. 1972. -0373-

CISAR, Z. Swine fever in wild boar. Veterinarstvi 26(8):359-360. 1976. -0374-

COGGINS, L. and SHEFFY, B. E. A serological (neutralization) test for hog cholera. Proceedings of the U. S. Livestock Sanitary Association 65: 333-337. 1961. -0375-

_____, SHEFFY, B. E. and BAKER, J. A. Response of swine to hog cholera vaccines. Proceedings of the U. S. Livestock Sanitary Association 66: 316-323. 1962. -0376-

_____ and SEO, S. Serological comparison with rabbit antisera of hog cholera virus and bovine virus diarrhea virus. Proceedings of the Society for Experimental Biology and Medicine 114:778. 1963. -0377-

* _____ and BAKER, J. A. Standardization of hog cholera neutralization test. American Journal of Veterinary Research 25(105):408-412. 1964. -0378-

A neutralization test for hog cholera (HC) has been developed and standardized. Characteristics studied and controlled were: dose-response curves, variation within and between tests, and the neutralization slope or relationship of amount of virus to serum titers. This test has been shown to be accurate to within a threefold dilution under ordinary circumstances.

* _____. Study of hog cholera colostral antibody and its effect on active hog cholera immunization. American Journal of Veterinary Research 25(105):613-617. 1964. -0379-

Using a standardized neutralization test to measure hog cholera (HC) antibody, the transfer of maternal antibody to newborn pigs and its subsequent decline were studied. Before nursing, pigs had no detectable serum antibody, but within 24 hours after ingestion of colostrum, pigs had acquired antibody equal to that found in the sow's serum. This acquired antibody declined at the fairly constant rate; *i.e.*, by one-half each 13 days. When present at a high level transferred maternal HC depressed the immunologic response of pigs to hog cholera vaccine. Also, approximately 100 times as much vaccine virus was required to immunize pigs protected by such maternal HC antibody than was needed to immunize pigs that had suckled nonimmune sows.

COLE, C. G., HENLEY, R. R. and HUBBARD, E. D. Concentration of hog cholera virus in the blood of artificially infected swine at different stages of the disease. Journal of the American Veterinary Medical Association 108:143-147. 1946. -0380-

_____ and HENLEY, R. R. Crystal-violet hog cholera vaccine. Proceedings of the U. S. Livestock Sanitary Association 1946:102-108. -0381.

* _____ and HENLEY, R. R. Experiments on the combined use of crystal-violet vaccine and anti-hog-cholera serum in the prevention of hog cholera. U.S. Department of Agriculture, Circular no. 807. 1949. 12 p. -0382-

Seven experiments were carried out at the Ames, Iowa, Field Station of the Bureau for the purpose of determining the effectiveness, in the prevention of hog cholera, of treatment with crystal-violet vaccine and anti-hog-cholera serum combined. In the experiments, 247 pigs were used. The principal methods of treatment were simultaneous use of serum and vaccine, use of serum first and then vaccine, vaccine first and then serum, serum alone and vaccine alone. Doses of vaccine used ranged from 2 1/2 to 30 cc. and the doses of serum from 5 to 45 cc. All the treated pigs were exposed to hog-cholera virus. In most of the experiments, all the pigs were exposed at one time - about 90 days after treatment. In a few experiments, half of the pigs that received a particular treatment were exposed 7 days after treatment; the other half about 90 days after treatment. In one experiment, all pigs were exposed 7 days after treatment. No evidence was found that vaccine interfered with the action of serum; that is, pigs treated with an adequate dose of serum and vaccine were found to be as well protected when exposed on the seventh day as were pigs that had been treated with similar doses of serum alone. The results consistently indicated that serum interfered with the action of vaccine. The interference varied from very slight when small (10 cc.) doses of serum and large (20 to 30 cc.) doses of vaccine were used to 100 percent when normally adequate or standard (5 cc.) doses of vaccine were used along with standard (15 to 35 cc.) doses of serum. The use of small doses of serum along with large doses of vaccine would not be recommended in general practice not only because of the cost involved, but also because there is no assurance that a particular proportion of serum and vaccine would be generally effective. No justification was therefore found for the combined use of serum and vaccine.

COLE, C. G., TORREY, J. P. and ZINOBER, M. R. Twelve years' successful vaccination of farm herds with crystal violet-glycerol hog-cholera vaccine. In U. S. Livestock Sanitary Association. Annual Meeting, 60th, Chicago, 1956. Proceedings. Chicago, 1957. pp. 263-269. -0383-

_____ et al. History of hog cholera research in the U.S. Department of Agriculture 1884-1960. U.S. Department of Agriculture. Agricultural Information Bulletin no. 241. 1962. pp. 44-45. -0384-

Co-autores: R. R. Henley, C. N. Dale, L. O. Mott, J. P. Torrey y M. R. Zinober.

* EL COLERA es difícil de descubrir entre los lechones recién nacidos. El Campo (México) 48(961):13. 1972. -0385-

* EL COLERA porcino. Nuestra Tierra Paz y Progreso (Nicaragua) 9(1-2):36-37. 1965. -0386-

EL COLERA porcino. Avance Agrícola y Ganadero 2(24):12, 14. 1971. -0387-

* EL COLERA porcino. Agropecuaria Moderna (Venezuela) 2(17):28-29. 1974. -0388-

EL COLERA porcino en México. Porcicultura Mexicana 1(9-10):17-19. 1970. -0389-

COLERA PORCINO; signos clínicos y hematología. México Ganadero 11(119): 22-23, 59. 1968. -0390-

* COLLINS, K. E. Effects of ultraviolet radiation on hog cholera virus. American Journal of Veterinary Research 19(72):540-544. 1958. -0391-

Hog cholera virus (HCV) in swine blood serum preparations was irradiated with 2,537 Å radiation. Approximately 10^5 infective doses (i.d.) of HCV, when ultraviolet-inactivated, were found to protect swine, 2 to 4 months old, to a 5.000 i.d. challenge. Reactivation of the infectivity of two of three HCV preparations was found to occur when ultraviolet doses appreciably in excess of the initially inactivating dose were used.

* _____. Enhancement of infectivity of hog cholera virus. American Journal of Veterinary Research 21(82):472-474. 1960. -0392-

Hog cholera virus (HCV) in blood serum, both diluted and undiluted, was subjected to different conditions of pH, temperature, and trypsin content. The treated preparations were then titrated in susceptible swine. No significant increases in infectivity titer were found for the diluted preparations. Large (ten thousand-fold)

increases in the measured $i.d._{50}$ per milliliter were found for undiluted preparations treated at pH 8.5 and 40 C. in the presence of 0.5 percent trypsin or at pH 5.5 and 25 C. in the absence of trypsin. In the latter case, the infectivity titer was found to be reversibly dependent on pH.

* COLLINS, K. E. The passage of hog cholera virus through cellulose dialysis membranes. American Journal of Veterinary Research 21(82):475-477. 1960. -0393-

Hog cholera virus in blood serum was dialyzed against normal swine serum and against phosphate buffer solutions, using cellulose dialysis membranes. Hog cholera virus was found to pass through the membranes into normal swine serum at pH 8.5 and into phosphate buffer solution at pH 5.5.

* COMO ATACAR la peste porcina. Boletín Agrícola (Colombia) no. 618: 12700-12701. 1972. -0394-

* COMPORTAMIENTO DEL virus de la DVB en los porcinos. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal no. 2. 1976. p. 8. -0395-

* CONCLUSIONES Y RECOMENDACIONES; peste porcina. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal no. 2. 1976. p. 15. -0396-

CONN, H. Why hog cholera serum sometimes fails. The Swine World 7:13. 1920. -0397-

* CONSULTA FAO/CEE DE EXPERTOS EN LA ERRADICACION DE LA PESTE PORCINA Y LA PESTE PORCINA AFRICANA, HANNOVER, 1976. Informe. FAO. Producción y Sanidad Animal no. 2. 1976. 22 p. -0398-

* CORREA GIRON, P. et al. Una nueva posibilidad para el control del cólera porcino. Porcirama (México) 57:41-45. s.f. -0399-

Co-autores: J. A. Baker, B. E. Sheffy, M. del C. Ochoa y N. Mancisidor Ahuja.

Las pruebas realizadas en México con la vacuna elaborada con virus de diarrea viral bovina (BVD) Cepa Andy 1 y con la cepa vacunal del cólera del cerdo (CC) PAV-250, confirmaron los estudios realizados en los E.U.A. y demostraron, además que la vacunación con la combinación de las cepas BVD-Andy-1 y CC-PAV-250 confirió excelente protección (100%) contra el CC. La vacuna BVD-Andy-1, sola protegió (94%) en contra de la cepa virulenta Cornell A de CC,

confirió protección (83%) en contra de la cepa Ames. Una vacuna recomendable para el control del CC podría ser la combinación de las cepas BVD-Andy-1 y CC-PAV-250. Estas vacunas no se difunden de cerdos vacunados a los susceptibles, ni de los cerdos vacunados a las ternèras. Son enteramente inocuas y combinadas estimulan una protección completa (100%), contra la cepa de exposición Ames.

* CORREA GIRON, P. y UGARTE ROMANO, C. Potencia de sueros y vacunas comerciales contra el cólera porcino. Técnica Pecuaria en México no. 21:47. 1972. -0400-

_____ et al. Inocuidad y potencia de vacunas contra el cólera porcino. Resúmenes de la 11a. Reunión Anual del Instituto Nacional de Investigaciones Pecuarias, SAG, San Jerónimo Lídice, México, D. F., 1974. pp. 10-11. -0401-

Co-autores: N. Mancisidor Ahuja, M. del C. Ochoa, J. Aguirre y F. Larios.

* _____ et al. Una nueva vacuna para el mejor control del cólera porcino. In Reunión Anual del Instituto Nacional de Investigaciones Pecuarias, 12., México, D. F., 1975. Resúmenes. México, D. F., 1975. p. 8. -0402-

Co-autores: J. A. Baker, M. del C. Ochoa y N. Mancisidor A.

Se realizaron pruebas en cerdos con objeto de evaluar dos nuevas vacunas contra el cólera del cerdo y la combinación de ambas. Una vacuna fue elaborada con el virus del diarrea viral bovina (BVD) Cepa Andy 1; otra con la cepa vacunal de cólera del cerdo (CC) PAV-250; y la otra con la combinación de ambas. En un primer experimento se vacunaron, por vía intramuscular, 16 cerdos de 6-8 semanas de edad, procedentes de madres vacunadas. A cada cerdo se le aplicaron 2 ml de la vacuna BVD-Andy 1, conteniendo 30,000 dosis infectantes para cultivos celulares 50% (DICC50). Cuando los cerdos tenían entre 5 1/2 y 6 meses de edad se les expuso en la Unidad Central de Palo Alto, D. F. a la cepa virulenta Cornell A de CC, y sobrevivieron 15 a los 16 cerdos vacunados (94%). Los 8 cerdos controles murieron con signos y lesiones de CC, no obstante que siempre permanecieron en la misma granja de tipo comercial con los vacunados. En un segundo experimento se vacunó un lote de 6 cerdos con la vacuna BVD Andy 1 (30.000 $DICC_{50}$ X dosis) y otro lote de 8 cerdos con la combinación de las vacunas BVD Andy 1 a la misma dosis y CC PAV-250 (3.000 DICC50 X dosis). Los cerdos, al ser vacunados en esta prueba, tenían 12 semanas de edad y procedían de madres no vacunadas. Dos meses después , todos estos cerdos fueron expuestos al virus virulento Ames de CC. Cinco de los 6 vacunados con BVD Andy 1 (83%) sobrevivieron a la exposición, así como los ocho

cerdos vacunados con la combinación BVD Andy 1 + CC PAV-250 (100%). Los 4 controles utilizados murieron presentando lesiones de CC, no obstante que, desde el día de la vacunación, estuvieron en contacto con los vacunados. Los resultados obtenidos, aunados a los estudios realizados en los EUA permiten concluir que la vacuna elaborada con el virus BVD Andy 1 protege (83-94%) contra el cólera del cerdo. La combinación de las vacunas BVD Andy 1 y CC PAV-250 confirió 100% de protección, y por lo tanto es la vacuna recomendable. En estos experimentos los virus vacunales fueron completamente inocuos y no se difundieron de los cerdos vacunados a los susceptibles. De acuerdo a los estudios realizados en la Universidad de Cornell, no se difunden al poner en contacto cerdos libres de patógenos específicos (SPF) vacunados, con cerdos SPF susceptibles. Y tampoco de cerdos SPF vacunados, a terneras susceptibles puestas en contacto.

CORTHIER, G. et al. Comparison of the antigenic activities of two togaviruses: swine fever virus and mucosal disease virus. Annales de Recherches Vétérinaires 5(3):373-393. 1974. -0403-

Co-autores: J. M. Aynaud, C. Galicher y J. Gelfi.

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_____. Influence of passive immunity on the response of piglets to viral infections; application to classical swine fever. Annales de Zootechnie 24(4):815-816. 1975. -0405-

_____, GALICHER, C. and GELFI, J. Swine fever: comparative study of the immunogenicity of live virus vaccines and inactivated virus, by the kinetics of neutralizing antibodies in the serum. Annales de Recherches Vétérinaires 6(1):93-101. 1975. -0406-

_____. Antigenic comparison of swine fever virus (SFV) and mucosal disease virus (MDV). In Studies on Virus Replication. Proceedings of Seminar, Brussels, 1974. Luxembourg, Directorate-General Scientific and Technical Information and Information Management, 1976. pp. 372-381. -0407-

_____. Comparison of two *in vitro* methods for titration of swine fever virus neutralizing antibodies. Annales de Recherches Vétérinaires 7(4):349-360. 1976. -0408-

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* Compendiado en Veterinary Bulletin 48(1):26. 1978. -0409-

CORTHIER, G. and AYNAUD, J. M. Comparison of the immune response in serum and bucco-pharyngeal secretions following immunization by different routes with a live hog cholera virus vaccine (Thiverval strain). Annales de Recherches Vétérinaires 8(2):159-165. 1977. -0410-

* COSTA, A. M. DA. Quelques essais d'immunisation contre la peste porcine. Veterinary Bulletin 5:212. 1935. -0411-

The experiments described consisted in inoculating swine with the chemically attenuated virus of swine fever and ascertaining the degree of immunity by introducing into the pen a pig inoculated with virus and a normal pig. The addition of 1 per cent of chloroform to defibrinated virulent blood and of 1.5 per cent to ether-treated suspensions of spleen, liver and lymphatic glands did not render these tissues innocuous, even after an interval of 68 days at room temperature. The addition of 0.4 per cent of formalin to liver tissue did not induce complete attenuation, but of four pigs inoculated with the suspension three did not show symptoms and the fourth recovered after a mild attack when their immunity was tested three weeks later. The immunizing properties of formalinized virus is further demonstrated by another experiment, but in this, of six pigs vaccinated, two that had received double quantities died before being infected. Swine fever could not be demonstrated in these pigs. The remaining four withstood infection when tested.

* COWART, W. O. and MOREHOUSE, L. G. Effects of attenuated hog cholera virus in pregnant swine at various stages of gestation. Journal of the American Veterinary Medical Association 151(12):1788-1794. 1967. -0412-

Exposure of 16 sows to attenuated hog cholera virus at intervals ranging from gestation days 30 to 109 resulted in a high incidence of stillborn pigs and mummified fetuses in litters exposed prior to gestation day 100. Ten sows in this category farrowed a total of 37 live pigs, 10 mummies, and 26 stillborn pigs. Six sows exposed between gestation days 100 and 109 farrowed 57 live pigs, 3 mummies, and 1 stillborn pig. Five control sows farrowed 53 live pigs and 2 mummies. The principal gross pathologic change in stillborn pigs was subcutaneous edema; virus was demonstrated by fluorescent antibody (FA) technique in spleen and tonsillar tissue of fetuses and in stillborn and live pigs farrowed by sows exposed between 70 and 109 days in their gestation. Splenic suspensions from these pigs inoculated into susceptible pigs rendered the latter immune to challenge with virulent hog cholera virus 20 days after exposure. There was no clinical evidence of spread of the virus from exposed sows to contact susceptible pregnant gilts or their pigs.

* COWART, W. O., MOREHOUSE, L. G. and MOODY, R. Laboratory diagnosis of hog cholera in Missouri. Journal of the American Veterinary Medical Association 154(12):1581-1582. 1969. -0413-

_____ and MOREHOUSE, L. G. The role of hog cholera virus in producing fetal and neonatal deaths in swine. U. S. Department of Agriculture. Agricultural Research Service. Animal Health Division 73:88-95. 1969. -0414-

* CRAWFORD, J. G. and DAYHUFF, T. R. Hog cholera: preparation of hog cholera immunogen from photodynamically inactivated virus. American Journal of Veterinary Research 29(9):1741-1747. 1968. -0415-

Inactivation of hog cholera (HC) virus by a photodynamic process using toluidine blue and light was rapid and irreversible. In contrast crystal violet (CV) did not have an inactivating effect. It was found that HC virus could be adsorbed to hydrated aluminum oxide under defined conditions.

* _____, DAYHUFF, T. R. and GALLIAN, M. J. Hog cholera: replication of hog cholera virus in tissue culture with cytopathic effect. American Journal of Veterinary Research 29(9):1733-1739. 1968. -0416-

A method for propagating virulent hog cholera (HC) virus in tissue culture was devised whereby cytopathic effects (CPE) were routinely observed. The CPE developed only in cultures of porcine testicular cells or porcine ovarian cells and only in the presence of an arginine-buffered medium.

* _____, WHITE, E. A. and DAYHUFF, T. R. Hog cholera: response of pigs vaccinated under field conditions with photodynamically inactivated hog cholera vaccine of tissue culture origin. American Journal of Veterinary Research 29(9):1761-1767. 1968. -0417-

Either 1 or 2 doses of photodynamically inactivated hog cholera (HC) vaccine was administered to 1,195 of 8,628 pigs from 17 herds. Pigs from both immune and nonimmune sows were vaccinated. Adverse effects were never seen in either vaccinated pigs or contact pigs. Considering ages, breeds, weaning and vaccination history of the sows, 80% of the pigs given 1 dose of vaccine were protected and 90% of those given 2 doses were protected, as measured by their resistance to an intramuscular injection of virulent virus. When vaccinated at 6 to 8 weeks of age, pigs from modified live-virus (MLV) vaccinated sows resisted the effects of virulent virus in the same measure as vaccinated pigs from susceptible sows.

* CRAWFORD, J. G., DAYHUFF, T. R. and WHITE, E. A. Hog cholera: safety and protection studies with photodynamically inactivated hog cholera virus. American Journal of Veterinary Research 29(9):1749-1759. 1968. -0418-

Swine inoculated with vaccine prepared with photodynamically inactivated hog cholera (HC) virus were protected against the disease as measured by resistance against an injection of virulent virus or exposure to infected pigs. Swine tested 21 days after vaccination with as little as 1.5 ml of some vaccines were immune to virulent HC virus. Swine with protective levels of passive antibody at the time of vaccination were resistant to challenge inoculation after the passive antibody became extinct. A single dose of vaccine protected swine through market age, but there was evidence of a decreased of immunity thereafter. Swine given 2 vaccinal doses, 3-week interval between doses, maintained resistance to challenge inoculation beyond 6 months of age.

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* CUDHAY G., B. Constantes hemáticas en cerdos infectados de cólera porcino. Porcirama (México) 5(54):8-13. s.f. -0420-

CUNLIFFE, H. R. and REBERS, P. A. The purification and concentration of hog cholera virus with electron micrographs. Canadian Journal of Comparative Medicine and Veterinary Science 32:409-411. 1968. -0421-

CUPERA, Z. et al. Effect of humidity on the efficacy of the TVM-1 vaccine against swine fever in the expiration period. Veterinaratvi 24(10): 451-452. 1974. -0422-

Co-autores: R. Polacek, B. Macura, L. Dedek y V. X. Jordán.

CURY, R. Observaçoes e pesquisas em torno da rotina de preparo da vacina de cristal violeta contra a peste suina. Atualidades Veterinarias 1(1): 18-19. 1972. -0423-

CHISIU, N. S. and POPA, M. N. Homologous molecular translocation and a critique of immunofluorescence methods applied to swine fever virus. Archiva Veterinară 7(1/2):21-33. 1970. -0424-

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* DALE, C. N. and ZINOBER, M. R. Variations (variants) of hog cholera virus. II. Perpetuation and attempts at enhancement of variant characteristics of hog cholera virus by means of serial passage with antiserum and without antiserum. Journal of the American Veterinary Medical Association 125(929):137-143. 1954. -0426-

A commercial hog cholera virus responsible for postvaccination losses in 1949 and shown to have variant characteristics was passed serially nine times simultaneously with serum and followed by seven serial passages without serum. The ninth passage with serum retained its variant characteristics as also did the seventh passage without serum. When similarly tested, the fourteenth passage did not retain like variant characteristics. Passing the virus seven more times simultaneously with serum failed to cause the reappearance of variant characteristics. Thus, variant hog cholera virus may be prepared by either of the first two procedures, but not by either of the last two procedures. Variant characteristics of a variant hog cholera virus were maintained after storage at -70 C. for 1,197 days and also of a variant virus stored for 439 days at -70 C. and additional 740 days at -40 C.

_____, ZINOBER, M. R. and TORREY, J. P. Variations (variants) of hog cholera virus. III. Further attempts to enhance its variant characteristics by simultaneous passage with varied amounts of different serum. In American Veterinary Medical Association. Annual Meeting, 91st., Seatle, Was., 1954. Proceedings. pp. 124-131. Discussion pp. 131-132. -0427-

* _____ et al. Variations (variants) of hog cholera virus. Journal of the American Veterinary Medical Association 118(890):279-285. 1951. -0428-

Co-autores: H. W. Schoening, C. G. Cole, R. R. Henley y M. R. Zinober.

Losses in swine during the summers of 1949 and 1950 following vaccination with hog cholera virus and serum, were investigated both in the field and in the laboratory. Laboratory investigations were conducted at the Pathological Division's Animal Disease Station at Beltsville, Md., and at its Hog Cholera Research Station at Ames, Iowa, and more than 2,000 experimental swine were used. The investigations revealed that some of the viruses used in the field possessed unusual characteristics; these viruses have been designated variants. The pigs already immunized against hog cholera by serum and virus were resistant to inoculation with the variant virus. However, a number of cholera-susceptible swine, when injected with variant virus and serum, either became sick and recovered or died, whereas similar pigs treated with regular virus and the same serum remained well. Weaned pigs when vaccinated were

more susceptible to the effects of variant virus than suckling pigs. By increasing the dose of serum, the effect of the variant virus could be reduced or eliminated. It is recommended that in hog cholera vaccination careful attention should be given to the condition of the herd to be vaccinated, and that the dose of the serum should be increased 50 per cent over minimum dose.

* DALE, C. N. and SONGER, J. R. *In vitro* propagation of hog cholera virus. I. Method of cultivation and observations on color changes in the medium. American Journal of Veterinary Research 18(67):362-368. 1957.
-0429-

In vitro propagation of hog cholera virus was readily established in a fluid medium (modified Tyrode's) containing minced tissue of swine spleen. By serial passages of three or four days' incubation, the fifteenth passage gave a 10^{-5} infectivity titer when tested in swine. Dilutions of 10^{-4}, 10^{-5}, 10^{-6}, and 10^{-7} were injected into pigs, using 2 pigs for each dilution. Those pigs receiving 10^{-5} dilutions or lower died from hog cholera, while those receiving higher dilutions remained normal. Crystal-violet vaccines separately prepared with virus from the tenth, eleventh and twelfth passages failed to produce any detectable degree of immunity in swine given subcutaneous injections of 10 cc. An agent which produced discoloration of the medium was propagated concomitantly with the hog cholera virus. By *in vitro* titration, it was shown to have a titer varying from $10^{-7.6}$ to $10^{-8.3}$. A correlation between the titers of the two propagated agents was not demonstrated.

* _____, ZINOBER, M. R. and TORREY, J. P. Variations (variants) of hog cholera virus. IV. Comparative potencies of Crystal-violet glycerol vaccines prepared from regular hog cholera virus and variant hog cholera virus. American Journal of Veterinary Research 18(66):112-118. 1957.
-0430-

Three hog cholera crystal-violet glycerol (CVG) vaccines were each tested for immunogenicity in one experiment against three different viruses. In another experiment, one vaccine from the previous experiment was retested and another vaccine was tested for potency against two viruses. The viruses were (1) regular BAI virus 286; (2) variant virus 8014; the ninth serial, simultaneous passage of variant virus and serum; (3) "variant" virus 8125, the third serial passage of variant virus without serum; and (4) variant virus 9096, the seventh serial passage without serum of a variant virus following nine passages simultaneously with serum. The vaccines were (1) CVG vaccine 96, a vaccine prepared from a regular BAI virus; (2) CVG

vaccine 8014 (variant), a vaccine prepared from the virus representing the ninth serial, simultaneous passage of variant virus and serum; (3) CVG vaccine 8125 ("variant"), a vaccine prepared from the third serial passage of variant virus without serum and (4) CVG vaccine 9096 (variant), a vaccine prepared from the virus representing the seventh serial passage without serum of a variant virus following nine serial passages simultaneously with serum. The pigs were challenged three weeks after vaccination. Crystal-violet glycerol vaccine 96 in 5 cc. doses adequately protected all of 16 pigs (100%) when challenged by regular BAI virus 286 variant virus 8014, or variant virus 8125 and adequately protected 7 of 10 pigs (70.0%) when challenged by variant virus 9096. In 2 cc. doses, CVG vaccine 96 adequately protected all of 7 pigs (100%), 1 of 7 pigs (14.0%) and 5 of 7 pigs (71.0%) when challenged by regular BAI virus 286, variant virus 8014, and variant virus 8125 respectively. Crystal-violet glycerol vaccine 8014 (variant) gave no appreciable protection in either 5 cc. or 2 cc. doses against regular BAI virus 286 variant virus 8014, or variant virus 8125. Crystal-violet glycerol vaccine 8125 (variant) in 5 cc. doses adequately protected 2 pigs (62.5%) when challenged by regular BAI virus 286. It gave no appreciable protection in 5 cc. doses against variant virus 8014 or variant virus 8125. In 2 cc. doses, it failed to protect any of the animals against variant virus 8014 but adequately protected 2 of 7 pigs (28.6%) against variant virus 8125. Crystal-violet glycerol vaccine 9096 (variant) in 5 cc. doses adequately protected 1 of 10 pigs (10.0%) when challenged by regular BAI virus 286 and 3 of 10 pigs (30.0%) when challenged by variant virus 9096.

* DALE, C. N. and SONGER, J. R. *In vitro* propagation of hog cholera virus. II. Some biological and immunological characteristics of hog cholera virus grown in tissue culture. American Journal of Veterinary Research 20(75):304-310. 1959. -0431-

Additional studies of *in vitro* propagation of hog cholera virus in a Maitland system of tissue culture are reported. No difficulty was encountered in establishing a new line of serial passage. The eleventh and fifteenth passages of a new line each had infectivity titers of 10-5. The thirtieth passage was highly virulent, and produced typical hog cholera and death of affected animals usually within 15 days. In infectivity titration tests of the virus, a decreased pathogenicity was obtained in some cases with the highest dilutions showing infectivity. The course of cholera in some of these pigs was more prolonged and less acute than in pigs given the higher concentration. By the tests used, with virus propagated *in vitro* was somewhat

less potent than commercially produced anti-hog cholera serum. Several possible causes for this are suggested, one being the low-infectivity titer of the virus. No qualitative or quantitative differences in the virus were detected when horse serum was substituted for swine serum in the tissue culture medium. Swine spleen, after refrigeration at 4 C. for three and four days, supported virus propagation, but not after refrigeration for seven days. Pigs treated simultaneously with virus propagated *in vitro* and anti-hog cholera serum prepared with virus propagated *in vitro* were solidly immune when challenged nine months later. Vaccines were prepared by incubation of the *in vitro*-propagated virus at 37 C. for two weeks. Of 12 vaccines prepared from 12 different serial passages, three showed immunogenic value, but in each case only 1 of 2 pigs vaccinated survived challenge.

* DALE, C. N. and SONGER, J. R. *In vitro* propagation of hog cholera virus. III. Cultivation of an immunological variant with retention of its identifying characteristics. American Journal of Veterinary Research 20(75):311-318. 1959. -0432-

Whether procedures could be developed whereby the immunological variant of hog cholera virus responsible for the losses of swine in the Midwest in 1949 could be isolated was investigated. The methods used proceeded from the assumption that the simultaneous virus used in vaccination was a mixture of regular and variant viruses. Since current studies had shown that regular virus could be propagated in tissue culture medium, it seemed possible that this method, with modifications, might also be used to separate the variant virus in a stable form. Previous investigations and shown that it was necessary to make serial passages of the variant virus in swine simultaneously with a subprotective dose of serum in order to maintain a virus capable of showing immunological variation. Earlier work had shown that the mixture of regular virus and variant virus retained variant characteristics after 16 passages in swine; the first nine passages simultaneously with subprotective doses of serum and the last seven without serum. However, the variant characteristics were lost after seven more serial passages without serum. Consequently, starting with the sixteenth passage, 12 additional passages were made. The first seven passages were made with serum and the last five without serum, making a total of 28 passages. When tests in swine showed that the twenty sixth passage had characteristics of variant virus, the twenty eighth passage was used as inoculum for the first passage in tissue culture. The tenth serial passage in tissue culture when

tested in swine still retained its variant characteristics. If the virus continues to maintain its stability under these procedures, it will provide new approaches to viral studies of hog cholera.

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* _____ and SONGER, J. R. Evaluation of crystal-violet glycerol hog cholera vaccine; comments on a proposed reproducible test. American Journal of Veterinary Research 27(121):1657-1662. 1966. -0434-

From the fall of 1955 through the spring of 1959, 6 tests of the potency of a single crystal-violet glycerol (CVG) hog cholera vaccine were made in swine, and highly reproducible results were obtained in each test. To obtain these reproducible results, the following factors were known: (1) The breeding history of each pig in 5 of the 6 tests was available; in the 6th test, all the pigs were known to be crossbred. (2) All the test pigs came from herds in which there were no carriers of *Salmonella choleraesuis*. (3) Portions of the same challenge virus were used. (4) The ages of the pigs ranged from 4 to 8 months.

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DALSGAARD, K. and OVERBY, E. Vaccination of pigs against hog cholera (classical swine fever) with a detergent split vaccine. Acta Veterinaria Scandinavica 17(4):465-474. 1976. -0436-

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Co-autores: B. Macura, Z. Cupera, V. Dubansky y J. Jerabek.

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* Compendiado en Veterinary Bulletin 30(8):446. 1960. -0443-

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DIMITROV, K. and GAVRILOV, N. Antibody production in serum-producing pigs immunized with a lapinized C vaccine and swine fever serum. Veterinarnomeditsinski Nauki 12(9):41-45. 1975. -0447-

_____. Determination of the immunizing potency of a single dose of dried, lapinized swine fever vaccine (Chinese strain). Veterinarnomeditsinski Nauki 13(1):59-62. 1976. -0448-

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Co-autores: W. Becker, U. Forster, M. Marks, B. Tiefenbach y G. Wachendorfer, D. Manz.

_____ et al. Investigations on the use of swine fever vaccine "Suiferin C" in breeding animals. I. Effects on reproduction. Deutsche Tierarztliche Wochenschrift 83(4):129-133. 1976. -0450-

Co-autores: W. Becker, D. Manz, B. Tiefenbach y G. Wachendorfer.

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Co-autores: C. N. McBryde, W. B. Nile e I. H. Rietz.

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_____, CARBREY, E. A. and STEWART, W. C. Preliminary findings on a thermal inactivation curve for hog cholera virus. Presentado en Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. s.p. -0458-

* DOYLE, T. M. and SPEARS, H. N. Injection of crystal violet swine fever vaccine in the ear. Veterinary Record 67(22):408-409. 1955. -0459-

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Co-autores: J. Jerabek, R. Hsabak y V. Dubansky.

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Co-autores: L. Dedek, J. Jerabek, Z. Novak y B. Macura.

_____ and JERABEK, J. Colostral immunity after vaccination of sows with the TVM-1 swine fever vaccine. Veterinarni Medicina 22(1):9-17. 1977. -0463-

* DUNNE, H. W. et al. A study of an encephalitic strain of hog cholera virus. American Journal of Veterinary Research 13(48):277-289. 1972. -0464-

Co-autores: E. M. Smith, R. A. Runnells, H. J. Stafseth y F. Thorp.

An encephalitic strain of hog cholera virus was isolated and maintained through 16 intracranial passages in pigs without

the loss of ability to produce encephalitic symptoms or other characteristics. Encephalitic symptoms occurred just as frequently in subcutaneously inoculated swine as in intracranially inoculated ones. This virus, designated as virus "A", produced a short incubation period, as indicated by the increased body temperature of inoculated pigs to 105.0 F. or higher twenty-four to seventy-two hours following inoculation of the virus. A short incubation period was further indicated by the rapid drop of total white cell counts twenty-four to seventy-two hours following inoculation. The course of disease caused by virus "A" was short, with deaths occurring as early as three to five days following inoculations. In 100 animals inoculated, there were no recoveries. All animals died or were moribund when destroyed. Incomplete protection against virus "A" was provided by Bureau of Animal Industry Experimental Serum no. 1. Commercial serum did not give complete protection in regulation serum test doses. Thalamic lesions were evident in 100 per cent of all cases studied. Severity of lesions appeared greatest in animals dying between the tenth and the fourteenth days. A commercial virus tested in pigs produced almost no lesions of the brain. Hydropic degeneration and proliferation of the vascular endothelium appear to be the primary pathological changes, with hemorrhages and other lesions apparently secondary or the result of these changes. No pathogenic bacteria other than *Pseudomonas aeruginosa* were isolated from the brain, blood, or intestine. This organism was isolated from 5 cases.

DUNNE, H. W. et al. Variations in the virus of hog cholera. Proceedings Book of the American Veterinary Medical Association 1955:148-153. -0465-

Co-autores: C. V. Reich, J. F. Hokanson y E. S. Lindstrom.

_____ et al. Variations in the virus of hog cholera; a study of chronic cases. In American Veterinary Medical Association. Annual Meeting, 92nd, 1955. Proceedings. 1956. pp. 148-153. -0466-

Co-autores: C. V. Reich, J. F. Hokanson y E. S. Lindstrom.

_____ et al. Bone structure changes in pigs infected with hog cholera. Journal of the American Veterinary Medical Association 130(6):260-265. -0467-

Co-autores: S. C. Benbrook, E. M. Smith y R. A. Runnells.

Rib lesions observed in pigs acutely infected with hog cholera occurred at the costochondral junction and were characterized by a mildly irregular epiphyseal line with an adjacent narrow transverse band of hemorrhage. In pigs with subacute infections of hog cholera, there was a marked

widening of the epiphyseal line by an increased number of cartilage cells with enlarged lacunae. Pigs with chronic cases of hog cholera commonly had dense osseous transverse lines proximal to the epiphyseal disc. The metabolic disturbance was characterized by an early increase in phos phorus and a decrease in calcium. A comparison is drawn between the occurrence of rib lesions, fetal malformation, soft-shelled eggs, and encephalitic symptoms as the result of virus infection.

* DUNNE, H. W. et al. The *in vitro* growth of hog cholera virus in cells of peripheral blood. American Journal of Veterinary Research 18(68): 502-507. 1957. -0468-

Co-autores: A. J. Luedke, C. V. Reich y J. F. Hokanson.

The hog cholera virus was shown to propagated in culture using cells from peripheral blood. Leukocytes but not erythrocytes appeared capable of supporting virus growth. The alteration of a strain of hog cholera virus resulted from passage in blood cell culture but the resulting virus proved to be antigenetically impotent. Two virus strains reacted somewhat differently in serial passage. The "Sprankle" strain appeared to be more readily adaptable to *in vitro* culture and showed less antigenic variation than the Michigan A virus. A method was shown for the demonstration of subprotective immunity.

* _____, LUEDKE, A. J. and HOKANSON, J. F. The growth of animal leukocytes and their use in the cultivation of animal viruse. American Journal of Veterinary Research 19(72):706-711. 1958. -0469-

Leukocytes from cattle, swine, and chickens were grown on glass surfaces in serum medium. The growing cell appeared to be the mononuclear leukocyte, which developed into a macrophage and then into multinucleated giant cell, subsequently spreading into a large number of individual cells. Avian leukocytes developed extensive vacuolation in the growth process. Viruses were shown to multiply in leukocytes through as many as 61 serial passages. Hog cholera virus multiplied in swine leukocytes over a period of more than ten weeks with 20 changes of medium before the cells deteriorated. Cytopathogenicity associated with some viruses was shown by the loosening of cells from the glass tube walls, by hemolysis of erythrocytes in the medium, or by degeneration of leukocytes. These reactions to viruses were lost in serial passage even though the viruses were shown by other tests to be present. The system of leukocite propagation provides many possibilities for the study of viruses, leukocytes, and the pathogenesis of virus diseases.

* DUNNE, H. W., HOKANSON, J. F. and LUEDKE, A. J. The pathogenesis of hog cholera. I. Route of entrance of the virus into the animal body. American Journal of Veterinary Research 20(77):615-618. 1959. -0470-

The virus of hog cholera, carefully enclosed in double gelatin capsules to prevent contact of the virus and the pharyngeal tissues, proved incapable of producing hog cholera when liberated in the stomachs of susceptible swine. Animals exposed by the gastric route with the more concentrated samples of virus appeared to react more quickly and to develop more marked lesions on subsequent challenge than those exposed in the same manner to less concentrated virus, indicating a sensitizing effect of an apparently nonimmunizing antigen. Successful infection through the tonsillar area was accomplished in pigs in which the trachea was severed to prevent possible respiratory infection. The virus was detected in the blood stream 24 hours post-inoculation in pigs exposed by the tonsillar route. Respiratory infection with hog cholera virus was successfully accomplished in pigs with tracheas that had been severed to prevent simultaneous exposure of the tonsillar area. The virus was detectable in the blood of pigs surviving for 24 hours or longer following exposure, but not in pigs which survived for 21 hours or less.

* _____ and LUEDKE, A. J. The pathogenesis of hog cholera. II. The virus eclipse phase and sensitization of the host. American Journal of Veterinary Research 20(77):619-624. 1959. -0471-

Virulent hog cholera blood virus injected intravenously into susceptible swine was present in the blood stream at 15 minutes postinfection but not at 30 minutes. It was detected again at 12 hours when the inoculating did not exceed 10^5 l.d. When the dose was 10^6 l.d., the virus was detected as early as five hours postinoculation, and there may not have been a period in which virus was completely absent from the blood stream. Blood cells and lymphatic tissue cells refrigerated or frozen at two hours, five hours, and eight hours postinfection proved noninfectious in swine. Animals injected with five-hour unfrozen, eight-hour unfrozen, and eight-hour frozen blood and tissue cells showed somewhat greater reactions than those injected with five-hour frozen and two-hour frozen and unfrozen blood and tissue. Those which showed the more pronounced reactions subsequently appeared "sensitized" to challenge with virulent virus. Freezing of hypotonic suspensions of cells appeared to stop viral multiplication. There appeared to be a little evidence of virus multiplication at five hours. Refrigeration

appeared to retard but not completely stop viral multiplication. The phenomenon of "sensitization" was discussed and theories for its occurrence were presented.

DUNNE, H. W. and ALIBASOGLU, M. A preliminary report on attempts to vaccinate pigs following anti-hog cholera serum alone treatment. Proceedings of the U. S. Livestock Sanitary Association 64:309-313. 1960. -0472-

* _____. "Breaks" following vaccination with attenuated hog cholera vaccines. Journal of the American Veterinary Medical Association 138(6): 311-316. 1961. -0473-

_____. Field and laboratory diagnosis of hog cholera. Veterinary Medicine 58:222-239. 1963. -0474-

* _____ and CLARK, C. D. Embryonic death, fetal mummification, stillbirth, and neonatal death in pigs of gilts vaccinated with attenuated live-virus hog cholera vaccine. American Journal of Veterinary Research 29(4): 787-796. 1968. -0475-

Vaccination of hog cholera-susceptible gilts with attenuated live-virus vaccines and anti-hog cholera serum at 24 to 60 days of gestation resulted in fewer pigs being born, more mummified fetuses, more stillbirths, and fewer pigs surviving to 5 days after birth than were produced by nonimmune and immune control gilts. Also, 4 of 6 gilts pregnant for 24 days at the time of vaccination were barren at the time of hysterectomy. Increased embryonic death in the group of gilts vaccinated at 24 days of gestation was evidenced by the increased number of corpora lutea in excess of the combined number of live pigs and dead fetuses at hysterectomy, as well as the increased number of barren gilts. Virus was detected by fluorescent antibody (FA) cell culture tests and by FA technique in frozen sections of pigs dying at birth or within 5 days after birth. Virus from an infected pig dying at birth was lethal when injected into colostrum-deprived 4-week-old pigs. The evidence confirms earlier theories and evidence that hog cholera virus can transverse the placental barrier to infect fetuses *in utero*. At birth, the newborn pigs which were infected *in utero* may retain the virus and become foci of infection in a susceptible herd.

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Co-autores: J. T. Wang, C. D. Clark, J. F. Hokanson, T. Morimoto y O. R. Dubash.

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* _____. Aspectos epidemiológicos y vacunales de la peste porcina clásica. Gaceta Veterinaria (Argentina) 38(316):543-544. 1976. -0482-

En la transmisión del contagio de la peste porcina y en su expansión, tienen importancia el movimiento de los cerdos infectados y la suministración a los suinos de residuos y desechos varios no sometidos a esterilización preventiva. Modesto es en cambio el significado de otras vías de contaminación, como por ejemplo, el transporte pasivo del virus por obra de aves, dípteros y otros animales o a causa de envases de alimento y utensilios varios contaminados. El virus patógeno ha sido evidenciado fácilmente en el agua servida de los mataderos de cerdos, en embutidos y similares. También se ha hallado la posibilidad de encontrar virus patógeno en cerdos "recuperados" durante el sacrificio en establecimientos infectados durante la muerte y destrucción sólo de los sujetos con manifiestaciones clínicas visibles. El virus puede ser desvitalizado con diversos desinfectantes pero la soda cáustica parece el desinfectante más aconsejable. Debe ser usada en soluciones al 2% en superficies limpias y al 3% en presencia de materias fecales. El tiempo de contacto no debe ser menor de 30 minutos. Los excelentes resultados obtenidos en EE.UU. en la campaña de erradicación sistemática, parecen debidos a los siguientes elementos: 1) Información intensa y propaganda sistemática en los sectores interesados. 2) Imposición de la esterilización de los residuos y desechos destinados a la alimentación de los cerdos. 3) Bloqueo de todo movimiento de cerdos en una área extensa de un foco de peste porcina. 4) Sacrificio de todos los cerdos presentes en el foco infectado, sin tentativa de

recuperación de los animales aparentemente normales. 5) Pago rápido de adecuada indemnización a los propietarios de los animales sacrificados. 6) Amplia aplicación de métodos de diagnóstico de laboratorio. 7) Bloqueo de las importaciones de cerdos y de carne suina de países no indemnes de peste porcina. En EE.UU. se suspendió el recurso de la vacunación, habiéndose antepuesto el criterio de la erradicación. De cualquier manera se había puesto ya de manifiesto la eficiencia de la protección inmunizante obtenida con cepas vacunales "vivas", de variada atenuación y particularmente la llamada cepa China, cuyo empleo ofrece numerosas ventajas. La cepa China se ha mostrado además privada de acción patógena aún para los cerdos muy jóvenes y para el feto en el caso de la vacunación de cerdas en gestación. El desarrollo de la protección es rápido y parece ligado primero con una producción interferónica al 3º - 4º día de la vacunación. Aún la aparición de anticuerpos es precoz e intensa. Se trata de una cepa viral, la China, de muy baja infectividad y por lo tanto, aunque los cerdos vacunados difundan virus en el ambiente no es fácil el desarrollo de esta cepa en cerdos con contacto con vacunados. Un amplio experimento de vacunación con la cepa China, efectuado en condiciones controladas sobre cerca de 143.000 cerdos, no ha dado lugar a ningún inconveniente. Sin embargo, es verdad que en los sujetos vacunados el virus atenuado puede permanecer en el organismo por un cierto tiempo (por lo menos 12 días), pero inyectando suspensiones de órganos de estos sujetos a cerdos no vacunados, se obtiene en ellos un verdadero efecto vacunante. Este fenómeno no tiene desde el punto de vista práctico ningún significado.

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Co-autores: B. Mihajlovic, T. Dujin y M. Marjanov.

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ELLIS, P. R. The economics of swine fever control and eradication. In Seminar on Diagnosis and Epizootiology of Classical Swine Fever, Amsterdam, 1974. Luxembourg, Directorate-General Scientific and Technical Information and Information Management, 1976. pp. 236-248. -0486-

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* EMERSON, J. L. and DELEZ, A. L. Cerebellar hypoplasia, hypomyelinogenesis, and congenital tremors of pigs, associated with prenatal hog cholera vaccination of sows. Journal of American Veterinary Medical Association 147(1):47-54. 1965. -0488-

Cerebellar hypoplasia, hypomyelinogenesis, and congenital tremors in newborn pigs were attributed to vaccination of sows with a tissue-culture-modified hog cholera virus. The disease occurred in 35 of 63 litters and in addition, multiple fetal malformations and stillbirths were observed. Susceptible pigs inoculated with infected fetal tissues resisted a challenge dose of virulent hog cholera virus. It is theorized that the cerebellar hypoplasia resulted from hypoxia due to vasculitis, and that the virus could have injured the oligodendroglia, resulting in hypomyelinogenesis, and this in turn resulted in the clinical syndrome known as congenital tremors.

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A refinement of the application of the conglutination complement-adsorption test (CCAT) for detection of hog cholera antibodies was devised. To determine the minimum amount of reagent required for the conglutination test, the bovine serum (conglutinin) and horse serum titration series were studied over a narrow range, namely, 0.01 to 0.25 ml. Three different amounts of horse serum were used for the conglutinin titration and 4 different amounts of conglutining for horse complement titration. To evaluate the anticomplementary effect, the latter test was also carried out in the presence of negative serum and in the presence of antigen. Using the CCAT and a modified live-virus vaccine (rabbit origin) as the antigen immunologic responses were observed in (1) immune pigs hyperimmunized with an attenuated hog cholera vaccine of porcine origin; (2) nonimmunized pigs penned with porcine vaccine immunized pigs; and (3) pigs immunized with bovine virus diarrhea, Oregon strain. The prozone phenomenon for all serums tested was negligible.

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_____ et al. Immunohistological studies on swine fever. II. Place of the contrast-immunofluorescence technique in routine laboratory diagnosis. Archiv fur Experimentelle Veterinarmedizin 26(4):661-682. 1972. -0492-

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Co-autores: Y. Tateyama, M. Tunoda, T. Uno, S. Watanabe y T. Ichihara.

EVALUACION DE pérdidas económicas producidas por un brote de cólera porcino; un caso. Porcirama (México) 24:30-33. 1973. -0498-

FERNELIUS, A. L. et al. Bovine viral diarrhea virus in swine: characteristics of virus recovered from naturally an experimentally infected swine. Canadian Journal of Comparative Medicine 37(2):2-13-20. 1973. -0499-

Co-autores: W. C. Antower, G. Lambert y A. W. McClurkin.

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FLORENT, A., THOMAS, J. and LEUNEN, J. Inspection of live vaccine against swine fever; use of immunodepression to detect residual virulence. Bulletin de l'Office International des Epizooties 72(1):665-669. 1969. -0501-

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* FLORES MENENDEZ, J. A. y AGRAZ GARCIA, A. A. Ganado porcino; cría, explotación e industrialización. México, D. F., Trucco, 1965. pp. 502-509. -0503-

* FLYNN, D. M. and JONES, T. E. The position regarding swine fever in Victoria. Australian Veterinary Journal 40:131-137. 1964. -0504-

The general position regarding disease in Victorian pigs prior to and after the recognition of swine fever in NSW is described. No increase in the incidence of disease, which would have been expected had low-grade swine fever been present in the pig population was found. Transmission experiments failed to provide any evidence of the presence of swine fever in Victoria. Gel diffusion precip itin tests on pig sera showed a 6.8% reactor rate in the pig population with a higher incidence of reactors in adult pigs, 7.9% in one survey and 30.9% in another. Challenge experiments with virulent swine fever virus revealed the existence of resistant pigs and there appeared to be good correlation between the GDPT, tissue culture neutralization test, and resistance to challenge. The significance of reactions to the GDPT and resistance to challenge is discussed. A reactor rate of 46.1% to the GDPT in Victorian cattle is reported and the significance of this discussed.

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Immunity against swine erysipelas was less solid in areas of Yugoslavia where pigs were vaccinated simultaneously against erysipelas and cholera when anti-hog cholera serum was used. Tests proved that ten of 11 commercial lots (91%) of anti-hog cholera examined contained antibodies against swine erysipelas. One lot (9%) had a titer of 4.65 immunity units and showed ability to lower the immunizing capacity of commercial adsorbed bacterin by 47 per cent. Since anti-hog cholera serum, being homologous, is eliminated slowly, vaccination for erysipelas should either be postponed more than two weeks after its use, or a slowly adsorbed erysipelas vaccine should be used.

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* Compendiado en Veterinary Bulletin 30(3):123. 1960. -0511-

* FUENTES ZENON, G. Se investiga nueva vacuna contra el cólera porcino. Tierra (México) 31(11-12):497-498. 1976. -0512-

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* Compendiado en Veterinary Bulletin 46(8):593. 1976. -0513-

Co-autores: N. Ogawa, H. Yamamoto, Y. Iwata, M. Sawada y H. Sazawa.

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Co-autores: J. H. Fernández de Linger, E. J. Galofre, N. S. Audisio y E. Imaz.

Se presenta un tratamiento que se estima positivo en la enfermedad de las mucosas. Consiste en la aplicación de Ficocoloides Quelatados (R. CAR.) y vacuna modificada contra peste porcina. Se produce regresión de las lesiones ulcerosas, desaparición de las diarreas, retorno del apetito en un plazo inferior a los siete días. La experiencia se realizó con 930 animales enfermos, en cuatro focos distintos aparecidos durante los años 1971 y 1972, en época de verano.

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GREEN MACIAS, J. Calendario de vacunación contra el cólera porcino. Porcicultura Mexicana 1(10):8-9. 1970. -0542-

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Comparisons have been made between tissue cultures of ten tissues involving 600 explants prepared from a pig six days following inoculation with hog cholera virus and of similar cultures from a susceptible litter mate. The following observations were made: significant changes were observed in endothelial cells of spleen cultures from inoculated pigs. There was increased granularity and vacuolization of the cytoplasm. Time-lapse phase cinematographic records

showed markedly fever lymphocytes to be present in the cultures of spleen from the inoculated pig. In epithelial cells of lung cultures from the inoculated pig there was some death of cells, reduced cytoplasm with increased vacuolization, and reduced nuclear size; however, cinematographic records of comparable cultures did not bear out these observations. Hydrogen ion concentrations as estimated by indicator comparisons revealed little difference between cultures from the inoculated pig and the uninoculated pig up to 21 days. The virus presumed to be present in the tissues at the time of culture apparently had only a relatively mild inhibiting effect on the gross growth characteristics of the cultures from the inoculated pig. Fixation and staining techniques suggested by Jacobson caused such alterations of the cytoplasmic elements and nucleoli that these preparations were useless for further study of cytopathogenic effects of the virus of hog cholera.

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In the search for a useful cytopathogenic effect of hog cholera virus (HCV), studies were conducted utilizing about 4,400 primary explant tissue cultures representing ten tissues from swine embryos. The following observations were made: 1) Lymph node cultures exposed prior to being embedded in plasma clots developed cytoplasmic changes in cells of the outgrowth. The mitochondria of the juxtanuclear zone were changed to spheroidal shape with clear light halos, cytoplasmic volume was reduced with attendant bubbling of the cellular membranes and subsequent filamentous strands of attachment to the glass at previous sites of attachment; and there was increased granularity of the cytoplasm. Lymphocytes were observed to die following exposure to virulent HCV. 2) Choroid plexus cultures were retarded in outgrowth grossly and showed inconsistent microscopic changes. 3) Cerebellum, kidney, testes, duodenum, spleen, heart, liver and lung tissue cultures were not affected measurably by the presence of HCV under the conditions of these studies.

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> The prevalence of salmonellae in swine tissues was determined by bacteriologic examination of 2,774 specimens from swine where hog cholera was suspected. A total of 831 isolations of salmonella were made, which included 42 serotypes. *Salmonella choleraesuis* var. *kunzendorf*, the most common serotype, accounted for 62.5% of all isolates.

* _____ and ELLIS, E. M. Erysipelothrix infection in swine suspected of having hog cholera. American Journal of Veterinary Research 33(4): 853-854. 1972. -0556-

> The occurrence of *Erysipelothrix insidiosa* in tissues of swine was determined by bacteriologic examination of 1,081 specimens where hog cholera was suspected. A total of 313 (28.9%) were test positive for *E. insidiosa*.

* HARVEY, M. J. and COOPER, F. Effect of exposure to hog cholera virus before and after vaccination with modified live virus vaccine. Journal of the American Veterinary Medical Association 124(923):141-142. 1954. -0557-

From the results of these trials, it would appear that, in healthy pigs, significant protection may be afforded by a single injection of modified live virus vaccine even when it is used one day after exposure to hog cholera virus by contact with pigs dying of the disease. However, the degree of protection is not such as to obviate the need for hog cholera antiserum where danger of exposure exists.

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Co-autores: A. Kawakubo, H. Matsuzawa, K. Tomizawa y J. Nakamura.

Guided by the results of pilot experiments with Newcastle disease virus, various culture conditions were used in the propagation of hog cholera virus in cultures of adult pig spleen tissue fragments. The most simple method of culture, in which the tissue fragments were suspended in Tyrode's solution plus 10 per cent normal pig serum, gave a final virus titer of 10^{-5}. Main improvements in culture, conditions which were, effective in raising the final virus titer of the culture were: 1) tissue fragments were fixed and cultivated on a rolling drum; 2) a relatively high concentration of tissue fragments was cultivated under adequate ventilation and frequent changes of culture medium. For this purpose, the tissue concentration was made to 10 per cent, air was allowed to pass freely through a cotton plug placed at the neck of the culture vessel, and the culture medium was renewed twice daily for the first two days of the three days' cultivation. The virus titer of a whole culture which had been prepared under the improved culture conditions was roughly 10^{-8}. This titer was comparable to that obtained from the solid spleen tissue of artificially infected pigs.

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* HELMBOLDT, C. F. and JUNGHERR, E. L. The neuropathologic diagnosis of hog cholera. American Journal of Veterinary Research 11(38):41-49. 1950.
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The value of neuropathologic examination in the differential diagnosis of porcine diseases is discussed. Gross, microscopic, and bacteriologic, and, occasionally, animal inoculation tests on 124 diseased and normal specimens submitted during the past eleven years were supplemented by histologic examination of the brain at five cross-sectional levels. Of 35 normal swine, 6-to 360-days old, 24 showed under, or near, the ependyma of the lateral ventricle both loose and compact cell aggregates which were found to be primarily composed of oligodendroglia and thus of ectodermal origin . These foci seemed to decrease in number and intensity with advancing age and to have no pathologic significance. Of 31 uncomplicated hog-cholera specimens, the gray and white matter of the brain showed, in 28 instances, mesodermal reactions consisting of vascular and perivascular infiltrates, microgliosis, capillary hemorrhages, and leptomeningeal infiltrates, indicative of nonsuppurative encephalitis. Of 13 hog-cholera cases complicated by samonellosis, pasteurellosis, etc., on the basis of bacteriologic findings,12 showed similar encephalitic lesions. Of 23 cases presenting noninfectious disorders and 22 affected with various infectious diseases, only 4 cases showed isolated, barely recognizable, vascular infiltrates of no diagnostic importance. Recognized infectious entities, such as erysipelas, pasteurellosis, salmonellosis, and hemorrhagic dysentery, which must be considered in the differential diagnosis of hog cholera, and successful hog-cholera vaccination, failed to induce the characteristic neuropathologic lesions. Encephalitic changes have been described in Aujeszky's disease, Teschen disease, rabies, and listeriosis, none of which have been recognized in Connecticut. These diseases are believed to have been ruled out in this study on the basis of anamnesis, bacteriology, and animal inoculation tests. Thus, the encephalitic changes were interpreted as indicating hog cholera, which diagnosis was supported by virus isolation in one subtypical case, and by field observations on the effectiveness of simultaneous hog-cholera revaccination. Neuropathologic examination is considered to be a valuable aid in the differential diagnosis of porcine diseases.

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When varying concentrations of hog cholera virus (HCV) were given to pigs by the oral route, the incidence of infection was relatively low; only two of ten trials were successful. However, when similar amounts of HCV were mixed with an innocuous amount of *Salmonella choleraesuis* and given by the oral route, hog cholera ensued in all ten trials. Hog cholera virus plus *S. choleraesuis*, when placed on the unbroken skin of pigs, caused cholera in all 3 pigs exposed, while only 1 of 3 pigs developed hog cholera when exposed to HCV without *S. choleraesuis*. All pigs exposed to HCV by

way of the conjunctival sac or the abraded skin contrated the disease with and without the presence of *S. choleraesuis*. Transmission was successful in six of nine aerosol transmission trials in which susceptible pigs were exposed to the exhaust air from isolation cans containing HCV-infected pigs. English sparrows, when allowed to fly from hog cholera-affected pigs to susceptible pigs, transmitted the disease in 2 of 3 trials. Pigs housed only 3 ft. away from the affected pigs, but which had no contact with the sparrows, failed to contract cholera. Attempts to transmit cholera using earthworms, stableflies, and houseflies were unsuccessful.

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To study persistent hog cholera (HC) viral infection *in vitro*, monolayer cell cultures were prepared from the kidneys of pigs experimentally infected with HC. The cultures were maintained for at least 600 days by changing the medium periodically and by serial passages of cells 80 times. Evidence indicated that the cells maintained by both methods were carriers of HC virus. The virus harvested from the cells at the 63rd to 66th passage was no longer lethal for susceptible pigs, whereas virus was not attenuated in the nonpassaged cells. The HC viral carrier state observed was characterized as follows: 1) Persisted virus was not eliminated with anti-HC viral serum; 2) free virus seemed unnecessary to maintain cell-to-cell transmission of virus in the carrier state; 3) nearly all of the cultural cell population seemed to carry HC viral antigen.

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Co-autores: K. Matsumoto, M. Sagawa, H. Iwabuchi y M. Soekawa.

Viral samples were serially harvested 3 times at approximately 150-day intervals from a hog cholera (HC) virus-carrier cell line, between cultivation days 689 and 1,102, in order to compare the reaction of pigs inoculated with the viral samples. According to the progress of serial passage of the carrier cell line, the clinical manifestations, leukopenia, viremia, virus-neutralizing antibody formation, and contact infection in pigs were gradually lessened. The HC virus that did not cause leukopenia and viremia, which

was detectable with the method used, was stable upon serial back-passages. Protection from a virulent HC virus, without the development of circulating virus-neutralizing antibody, was observed in pigs 42 to 72 hours after they were inoculated with the attenuated virus. Viral recoveries from superficial inguinal lymph nodes of pigs inoculated with the viral samples were negative as determined by the cell culture technique (END test), whereas viral recoveries were positive as determined by inoculating susceptible pigs. Formation of virus-neutralizing substance was demonstrated in these lymph nodes.

IZAWA, H., SOEKAWA, M. and NAGABAYASHI, T. A mutant of hog cholera virus derived from pig kidney cell culture persistently infected with the virus. In International Pig Veterinary Society Congress, 4th., Ames, Iowa, 1976. Proceedings. p. H.12. -0587-

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Co-autores: L. Dedek, J. Drazan, Z. Novak y B. Macura.

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* _____. The transenteral entrance of the virus in hog cholera. Journal of the American Veterinary Medical Association 136(4):149-155. 1960. -0614-

Experiments to produce hog cholera by depositing 2 or 3 ml. of virus in the stomach or in the small intestine of cholera-susceptible pigs failed on every attempt. The disease-producing and antigenic properties of the virus appear to have been destroyed in 15 of 23 pigs exposed in

this manner. They succumbed to cholera when virus was injected intramuscularly at a later date. Only the disease-producing component of the virus was destroyed in 8 pigs. They were immune when challenged and, it appears, the immunity was provoked by the virus used at the time of exposure. Results were quite different where the virus was applied by spreading on the membranes of the mouth. Of 30 pigs exposed in this manner, 21 developed hog cholera and died; 9 survived the exposure and were immune when challenged. The immunity, it appears, was stimulated by the virus swabbed on the oral membranes. Preliminary attempts to localize "sites" in the oropharynx where the transenteral entrance of the virus occurs, although far from conclusive, failed to show by the methods employed that virus was present in the tonsils, buccal mucous membranes, or retropharyngeal lymph nodes, 1, 4, or 12 hours after it was swabbed on the surface of the oral membranes. The "reaction" which occurred in some pigs a few days following exposure represented a time when the forces of infection and the forces of defense both were developing, with the latter eventually becoming more powerful. The forces of defense in this case were the production of antibodies or immune substance. Insofar as the mouth is a part of the digestive tract, the results support the concept held for many years that the portal of entry of the virus in hog cholera is the digestive tract.

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Co-autores: W. C. Stewart, E. A. Carbrey y M. L. Snyder.

A 2-step technique for the isolation of hog cholera (HC) virus consisting of an initial culture on buffy coat (BC) cultures and subinoculation to a pig kidney cell line (PK-15) was described. By this technique, HC virus was confirmed in specimens from 65 herds in which the conventional cell culture isolation technique failed. The herds were located in 20 states and Puerto Rico. Specimens from 29 of the 65 herds were inoculated into specific-pathogen-free (SPF) pigs. Hog cholera virus was recovered from 27 of the test pigs. The 2 pigs from which virus was not recovered had signs of acute infection and, on necropsy, had gross lesions of HC infection.

* KRESSE, J. I. et al. Sensitivity of swine buffy coat culture to infection with hog cholera virus. American Journal of Veterinary Research 37(11): 1315-1318. 1976. -0638-

Co-autores: W. C. Stewart, E. A. Carbrey y M. L. Snyder.

The susceptibility of swine buffy coat (BC) cultures inoculated with hog cholera (HC) virus in the presence of homologous antiserum was greater than that of a pig kidney (PK-15) cell line similarly inoculated. The virus was isolated from BC cultures grown in the presence of 0.1% hyperimmune serum, whereas it could not be consistently recovered from the PK-15 cell line in which hyperimmune serum concentrations exceeded 0.025%. Maximal viral titers in BC culture were reached between postinoculation days 4 and 8. Peak titers were not influenced by the age of cells at infection or dose of virus. Data were present in support of the hypothesis that the decrease of HC viral isolations in BC cultures from HC epizooties was related to lessened use of attenuated vaccines.

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Co-autores: T. Morimoto, T. Shimizu, S. Sasahara y M. Watanabe.

KUMAGAI, T. et al. A new *in vitro* method (END) for detection and measurement of hog cholera virus and its antibody by means of effect of HC virus on newcastle disease virus in swine tissue culture. IV. Repraisal of effect of serum in culture medium and time of challenge with ND virus. National Institute of Animal Health Quarterly 4:135-144. 1964. -0647-

Co-autores: T. Shimizu, S. Ikeda y M. Matumoto.

_____ et al. A new *in vitro* method (END) for detection and measurement of hog cholera virus and its antibody by means of swine tissue cultures. I. Establishment of standard procedure. Journal of Immunology 87:245-256. 1969. -0648-

Co-autores: T. Shimizu, S. Ikeda y M. Matumoto.

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Co-autores: A. Mestyan, Z. Prettenhoffer, J. Szabó y L. Varju.

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Co-autores: J. M. Aynaud, G. Corthier y H. Laude.

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Co-autores: J. M. Aynaud, G. Corthier y H. Laude.

* _____ and AYNAUD, J. M. Swine fever vaccination of piglets in natural conditions with the "Thiverval"strain in presence of colostrum derived passive immunity. Annales de Zootechnie 24(4):816-817. 1975. -0660-

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The "Thiverval" strain is a cold mutant of swine fever isolated in tissue culture at 29-30º C. Because of its immuniz ing properties and its innocuity, this strain is used as live vaccine. 142 piglets born from sows immunized or not against swine fever several months ago, were vaccinated with Thiverval strain in natural conditions between the 10th and 90th day of life using per nasal, per os or intramuscular route. The colostrum derived passive immunity and the active immunity induced by vaccination were determined by means of challenge with virulent Alfort strain and by means of titration in tissue culture of specific neutralizing antibodies of serum. Seroneutralizations were made in cell cultures by means of immunofluorescent techniques. In absence of passive immunity, vaccination performed in 18 days old piglets was efficient. In piglets from immune sows, vaccination was unefficient on days 10-13 and 28 of life, because of the too high level of passive immunity. But on day 46, active immunization became possible. Piglets were protected against virulent infection (challenge) on days 54-55 of life. Results from these experiments suggest that a particular period exists around the 30th and 60th day of life of piglets having passive immunity. During this period, maternal passive immunity protects piglets against virulent infections, but also allows induction of active immunity following vaccination with live vaccine. New investigations are needed to determine limits and characteristics of this period.

* LAUNAIS, M. and AYNAUD, J. M. Classical swine fever: duration of the immunity obtained in sows by vaccination with the Thiverval strain. Annales de Zootechnie 25(3):451. 1976. -0661-

Study of the kinetics of serum antibodies neutralizing the swine fever virus enabled us to determine the characteristics of the immunity obtained after one only injection of a live vaccine (Thiverval strain) to gilts. The rapidly obtained immunity reached a high level (index < 5) and remained for at least 39 months at that level, thus providing and efficient protection to the gilts during their whole reproductive life.

* LEANIZ, R. et al. Peste porcina. I. Diagnóstico por inmunofluorescencia usando cortes de tejidos y frotis de sangre. Gaceta Veterinaria (Argentina) 38(308):62-69. 1976. -0662-

Co-autores: R. Bermúdez, A. M. Siri, J. Pereira, R. Lagos y D. Carrera.

Se empleó el test directo de inmunofluorescencia, demostrando la presencia de antígeno viral en los leucocitos circulantes y en cortes de bazo, amígdala y ganglio. La variación introducida por los autores (frotis de sangre como antígeno) permitió hacer el diagnóstico en las primeras etapas de la viremia. Se confirmó además la alta sensibilidad y especificidad de la técnica.

* _____ et al. Peste porcina. II. Presencia de antígeno viral en las plaquetas. Gaceta Veterinaria (Argentina) 38(309):100-104. 1976. -0663-

Co-autores: A. Morelli, A. M. Siri, R. Bermúdez y F. Pérez A.

Los autores emplearon el método de inmunofluorescencia directa en frotis de sangre de cerdos inoculados con virus de Peste Porcina Clásica, encontrando la presencia de antígeno viral en las plaquetas. Este hallazgo da un nuevo enfoque a la patogenia de la PPC; explicaría el origen del púrpura y podría servir de modelo experimental para otros púrpuras trombocitopénicos de origen viral.

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Co-autores: C. S. Chen, T. C. Lin, P. P. Liou y B. E. Sheffy.

LEE, R. C. T. and LIN, T. C. A potent and safe strain of lapinized hog cholera virus used for hog cholera control in Taiwan. In International Pig Veterinary Society Congress, 4th., Ames, 1976. Proceedings. p. H4. -0665-

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LEUTENEGGER, R. Development of an icosaedral virus in hemocytes of Galleria Melonella. Virology 42:200-204. 1964. -0670-

LEWIS, P. A. and SHOPE, R. E. The blood in hog cholera. Journal of Experimental Medicine 50:719-736. 1929. -0671-

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LIESS, B. and WITTE, K. Use of fluorescent antibody technique in diagnosing swine fever in endemic conditions. Bulletin de l'Office International des Epizooties 72(1):543-557. 1969. -0673-

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_____ et al. Studies on swine fever. V. Detection of inapparent infection on pig breeding farms in three communities of North-West Germany. Berliner und Munchener Tierarztliche Wochenschrift 88(20;21):397-399, 405-409. 1975.
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Co-autores: B. Roder, K. Eife, R. Hirchert, J. Berger y C. Bachmann.

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Co-autores: H. R. Frey, D. Prager, S. M. Hafez y B. Roeder.

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Co-autores: H. Frey, U. Bucksch, B. Roeder y J. Frost.

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Co-autores: L. A. Mel'nikova, O. I. Sharabrin, N. A. Kal'chenko y N. K. Mishchenko.

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Co-autores: N. P. Nikolaeva, N. K. Mishchenko y E. I. Skalinskii.

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Co-autores: T. J. Yang, M. S. Chow, J. S. Lin y S. S. Chen.

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Co-autores: Y. Shimizu, T. Kumagai y J. Sasahara.

LIN, T. C., SHIEH, C. M. and SU, J. F. Virus multiplication in pigs inoculated with live lapinized swine fever vaccine. Chinese Journal of Microbiology 7(1):13-19. 1974. -0694-

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Co-autores: C. Y. Chen, C. M. Wang, C. C. Lin, S. S. Chen y S. C. Yang.

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* LOAN, R. W. and GUSTAFSON, D. P. Cultivation of hog cholera virus in subculturable swine buffy coat cells. American Journal of Veterinary Research 22(89):741-745. 1961. -0697-

Studies were made of the general problem of the detection of the presence of hog cholera virus (HCV) through the use of tissue culture techniques. Two subculturable swine buffy coat cell lines were developed. The propagation of HCV in one of these cell lines was demonstrated by the reaction of susceptible pigs to the inoculation of harvested materials. A 10^{-4} dilution of supernatant fluid from a 12th tissue culture passage of virulent HCV was infective for pigs. This represented a dilution of the original stock virus of more than 10-21. In 2 studies of the propagation of HCV in tissue culture, the virus was demonstrated in concentrations of 10^2 and approximately 10^5 infective doses per milliliter on the 4th day following inoculation of the cultures. Hog cholera virus was demonstrated in a persistently infected swine buffy coat cell culture after 204 days, at which time the virus was present in supernatant fluids in a concentration of approximately 10^5 infective doses per milliliter. No cytopathic effect of HCV was observed in subculturable swine buffy coat cultures.

_____ and McCAIN, C. S. Goat sera as media supplement in the exaltation of newcastle disease virus (END) test for hog cholera virus. Proceedings of the U. S. Livestock Sanitary Association 1963:348-357. -0698-

* _____ and GUSTAFSON, D. P. Persistent infections of subculturable swine buffy coat cells with hog cholera virus. American Journal of Veterinary Research 25(107):1120-1123. 1964. -0699-

Studies were made of the prolonged persistent infection of cultures of subculturable swine buffy coat cells with hog cholera virus (HCV). After 702 to 737 days of persistent

infection, virulent HCV was attenuated so that undiluted tissue culture fluid was not lethal for pigs. The liter of this tissue culture fluid was 100,000 pig infective doses per milliliter. Inoculation of pigs with this material resulted in a high degree of immunity as measured by the resistance of these pigs to challenge of their immunity, with virulent HCV. A 2nd isolate of HCV, after 462 to 471 days of persistent infection in tissue culture, was lethal for 5 of 10 inoculated pigs. Attenuated HCV did not interfere with the propagation of virulent HCV in tissue culture, and in one instance both attenuated and virulent HCV were propagated in a dually infected culture. Anti-hog cholera serum in the culture medium did not eliminate the persistent HCV infection.

LOAN, R. W. Studies of the nucleic acid type and essential lipid content of hog cholera virus. American Journal of Veterinary Research 25(108): 1366-1370. 1964. -0700-

Concentrations of 1.0 µg of 5-bromo-2'-deoxyuride (BDU) and 10 µg of 5-iodo-2'-deoxyuridine (IDU) per milliliter of culture medium completely suppressed the cytopathic effects (CPE) of vaccinia virus in primary cultures of swine testicular cells. Suppression of the CPE of vaccinia virus by BDU and IDU was eliminated by the addition of 100 µg of thymidine per milliliter of culture medium. Concentrations of 100 µg of BDU or IDU per milliliter of culture medium did not suppress the synergistic CPE of dual infections with hog cholera virus (HCV) and Newcastle disease virus (NDV) in the exaltation of Newcastle disease virus (END) test for HCV. It was concluded that HCV contains ribonucleic acid rather than deoxyribonucleic acid. Incubation of HCV with 20% diethyl ether at 4 C for 18 hours reduced the titer by at least $10^{3.3}$ 50% tissue culture infective doses ($TCID_{50}$) in 1 trial and at least $10^{1.7}$ $TCID_{50}$ in a 2nd trial. It was concluded that HCV contains essential lipid. Incubation of HCV at pH 2.9 to 3.0 for 5 hours at room temperature reduced the titer by at least $10^{3.3}$ and $10^{3.5}$ $TCID_{50}$/ml in 2 trials.

_____. Increased sensitivity of the END (Exaltation of Newcastle Disease Virus) test for hog cholera virus. American Journal of Veterinary Research 26(114):1110-1113. 1965. -0701-

The exaltation of Newcastle disease virus (END) test for hog cholera virus (HCV) in cell culture was found to be more sensitive if the introduction of Newcastle disease virus (NDV) was delayed until 7 days following the inoculation of HCV. Primary cultures of swine testicular cells 1 to 4 days old were found to be satisfactory for inocula-

tion with HCV in the END test. Virulent HCV consistently gave positive results with the END test. Some attenuated HCV and immunizing field strains of HCV gave positive END tests, but other strains did not.

* LOAN, R. W. and RODABAUGH, D. E. Serologic studies of hog cholera immunization. American Journal of Veterinary Research 27(120):1333-1338. 1966. -0702-

The antibody titers of pigs were determined by the enhancement of Newcastle disease virus (END) method in pigs following different immunization procedures. There appeared to be a positive correlation between virus host adaptation (virulence) and antibody titer, that is, the more virulent the virus the higher the immune response. Antibody titers following vaccination with selected hog cholera vaccines as measured by serum neutralization tests carried out by the END method were as follows: Rabbit origin attenuated hog cholera vaccine, 19.0; tissue culture origin hog cholera vaccine, 41.0; crystal violet vaccine (2 times), 11.0; and virulent hog cholera virus, 533.0. Pigs born to sows previously vaccinated with attenuated hog cholera vaccine and later exposed to virulent hog cholera virus (HCV), were found to have retained an average passively acquired antibody titer of 72.5 at 14 weeks of age. These pigs could not be successfully vaccinated with crystal violet vaccine at that age. Pigs born to sows vaccinated twice with attenuated vaccine were successfully vaccinated 8 weeks of age when their antibody titer had dropped to 6.0 or less. Passively acquired antibody titers of 50.0 or higher protected pigs from clinical infection with HCV. The antibody "half life" was found to be approximately 14 days.

* _____ and STORM, M. M. Propagation and transmission of hog cholera virus in nonporcine hosts. American Journal of Veterinary Research 29(4):807-811. 1968. -0703-

Propagation and transmission of hog cholera virus (HCV) were studied in 11 species of wild and domesticated animals following inoculation and cohabitation with non-inoculated animals. Viral propagation was determined by development of significant antibody levels in animals inoculated with small amounts of HCV. Antibody titers were determined by the enhancement of Newcastle disease virus (END) method. Antibody production was not detected in wild mice, cottontail rabbits, sparrows, wild rats, raccoons, or pigeons after inoculation with HCV. Significant antibody production was detected in peccaries, calves, goats, sheep, and deer inoculated with HCV; this indicated

propagation of HCV in these species. The inoculated animals did not transmit HCV naturally to penmates. This was determined by the failure of the penmates to develop antibody titers against HCV. Hog cholera virus was not transmitted by inoculated calves to susceptible cohabiting pigs.

* LOPEZ PALAZON, J. Ganado porcino; cría, recría y ceba de cerdos. 3a. ed. Madrid, Ministerio de Agricultura, 1968. pp. 430-434. -0704-

LORA, C. et al. Introduction in Perú of lapinized C strain swine fever virus vaccine. Revista del Instituto de Zoonosis e Investigación Pecuaria (Perú) 2(1/2):72-77. 1973. -0705-

Co-autores: H. Acuña, H. Sánchez y C. McGregor.

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* Compendiado en Veterinary Bulletin 30(4):183. 1960. -0706-

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Co-autores: G. Bouley, A. Paraf y C. Quinchon.

LUNGER, P. D. The isolation and morphology of the lucke-frog kidney tumor virus. Virology 24:138-145. 1964. -0709-

LYSENKO, I. P. et al. Simultaneous immunization of pigs against swine fever and erysipelas with an inactivated vaccine. Veterinariya no. 36:3-6. 1973. -0710-

Co-autores: A. A. Tsymbal, M. Z. Kul'bachnaya y M. I. Galishchev.

_____ et al. Simultaneous immunization of pigs with crystal violet swine fever vaccine and the attenuated VR2 strains of *Erusipelothrix rhusiopathiae*. Veterinariya no. 37:23-28. 1974. -0711-

Co-autores: O. M. Tsymbal, M. Z. Kul'bachna, M. G. Galishev.

* McBRYDE, C. N. and COLE, C. G. Crystal-violet vaccine for the prevention of hog cholera; progress report. Journal of the American Veterinary Medical Association 42(6):652-663. 1936. -0712-

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* Compendiado en Veterinary Bulletin 42(3):143. 1972. -0715-

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* McKISSICK, G. E. and GUSTAFSON, D. P. *In vivo* demonstration of lability of hog cholera virus to lipolytic agents. American Journal of Veterinary Research 28(125):909-914. 1967. -0717-

The immunogenicity of an *in vitro* attenuated strain of hog cholera virus (HCV) was destroyed by 30 minutes of agitated exposure to 20% ether or 10 minutes of exposure to 10% chloroform. Pathogenicity and immunogenicity of a partially attenuated form of the same virus was reduced more than 10,000-fold by 20 minutes of agitated exposure to 10% chloroform. Control portions of sham-exposed virus were effective at dilutions greater than 10^{-4} in all *in vivo* trials. Ether and chloroform inactivated the virus, and the virion of CJ-HCV by inference contained essential lipids in its coat.

* MACHADO, L. C. P. Los cerdos. Trad. por C. M. Vieites. México, D. F., Centro Regional de Ayuda Técnica, 1973. pp. 467-469. -0718-

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Co-autores: V. A. Sergeev, E. V. Rudobel'skii y V. I. Popov. -0721-

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* MALMQUIST, W. A., FERNELIUS, A. L. and GUTEKUNST, D. E. Interference of bovine viral diarrhea virus by hog cholera virus in swine kidney cell cultures. American Journal of Veterinary Research 26(115):1316-1327. 1965. -0723-

A strain of bovine diarrhea virus, NADL-MD, was adapted to primary and a subculturable cell line of swine kidney cell cultures. The adaptation was followed by comparative titrations in embryonic bovine kidney cell cultures and swine kidney cell cultures and by immunofluorescence. A modified strain of hog cholera virus used to infect swine kidney cell cultures interfered with the cytopathic effect and virus yield of cell-adapted NADL-MD virus. Interference by modified hog cholera virus was dosage dependent and required infection of cells before interference was expressed. Interferon prepared by a conventional method played no role in interference. A swine-passaged strain of hog cholera virus did not produce interference as completely as the strain passaged in swine kidney cell cultures. Early infection of the swine kidney cell line with modified hog cholera virus abolished the cytopathic effect and suppressed the yield of adapted NADL-MD virus. The interference occurred before the formation of soluble antigen, since this was decreased two- to fourfold, and virus yield was decreased by 90%. Application of the interference test made it possible to study the development of neutralizing antibody against hog cholera virus.

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Washed red blood cells from hog-cholera-susceptible swine were tested against hyperimmune hog cholera antiserum by the plate agglutination technic. Eleven animals showing strong agglutination were vaccinated by the simultaneous method, using one of the agglutinating sera. Nine of these animals showed temperature elevation within 1 week after vaccination. Six animals showing no agglutination were similarly vaccinated. Four of these showed no temperature elevation within 1 week postvaccination. The possible role that isoantibodies may play in field vaccination breaks is stated.

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Co-autores: D. E. Gutekunst, A. L. Fernelius y E. C. Pirtle.

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Evaluation of the fluorescent antibody-cell culture test (FACCT) was made to determine its reliability as a routine procedure for diagnosing hog cholera (HC). Eight hundred and fifty samples of blood and splenic tissue from 322 suspected herd epizootics of HC were examined by FACCT. Results of FACCT were compared with results obtained by inoculation of a portion of the same samples in HC-susceptible swine. In samples from 225 herds, hog cholera virus (HCV) was identified by results of both FACCT and the swine inoculation test (SIT). In samples from 13 herds, HCV was identified by results of SIT but not by results of FACCT. In samples from the remaining 84 herds, HCV was not identified by results of either test. Splenic tissue was better than blood as a diagnostic test sample. Hog cholera virus was identified by FACCT results in 324 (89%) of 363 spleens from herds proved by SIT results to be infected. In contrast, HCV was identified in only 178 (60%) of 296 samples of blood from most of the same herds.

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Several aspects of the pathogenesis of chronic hog cholera were investigated. Based on the severity of clinical signs, illness was divided into 3 phases: (1) early acute reaction, (2) period of partial recovery, and (3) relapse and death. Coincident with general improvement in clinical signs (2nd phase), there were a transient disappearance of hog cholera virus (HCV) from serum and an increased level of serum *gamma* globulin. Either antibodies or heat-stable inhibitors of viral infectivity, or both, were identified in samples of serum collected during this interval. As the disease progressed, virus reappeared and thereafter persisted in the serum of affected pigs until death. Virus was identified from most of the tissues collected from pigs killed during the course of chronic illness. Early sites of viral replication were tonsils and ileum.

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Replication of the Ames strain of hog cholera virus (HCV) in cells of an established porcine kidney (PK-15) cell line was determined by fluorescent antibody staining and by titrating the progeny virus. Viral antigen was first detected at the 4th hour postinoculation and progeny virus at the 6th hour. The percentage of cells that contained viral antigen increased from less than 1% at the 4th hour postexposure to more than 99% at the 12th hour postexposure. The titer of progeny virus increased during the interval from the 6th hour to the end of the experiment at the 24th hour postexposure, and mature virus was rapidly released from the cell. Identification of antigen only in the cytoplasm of infected cells suggested that replication of HCV was entirely extranuclear. Serial passage of HCV in PK-15 cells resulted in adaptation of the virus to this mode of replication. Adapted virus was characterized by a decrease in the interval between infection of cells and appearance of progeny virus (latent period) from 6 to 5 hours and by its ability to replicate to a higher titer in cell culture. The influence of the host cell on the replication of HCV was emphasized by the differences between replication in PK-15 cells and primary cells of porcine kidney and testicle origin. The latent period was shorter in PK-15 cells (6 hours) than in primary cells (7 hours), and growth curves were characteristic of the type of host cell.

* MENGELING, W. L. Endogenous neutralization of virus during fatal hog cholera illness. American Journal of Veterinary Research 31(1):91-95. 1970. -0757-

Production of antibody against hog cholera virus (HCV) and against killed *Brucella abortus* by pigs during fatal hog cholera (HC) illness was investigated. Hog cholera virus was recovered in cell culture from 10 of 10 serums collected from infected pigs on the 7th postinoculation day (PID) but from only 1 of 10 serums collected on the 14th PID. Neutralizing antibody was identified in all 10 of the serums collected on the 14th PID. Both HCV and homologous neutralizing antibody were detected in 3 of 5 serums collected on the 21st PID, whereas only neutralizing antibody was identified in the remaining 2 serums. Except for 1 pig which developed the chronic form of HC, all virus-infected pigs either died or were killed in the terminal stage of illness by the 23rd PID. Comparison of production of B. *abortus* agglutinins by virus-infected pigs with that by noninfected pigs indicated that antibody production was not significantly decreased during clinically recognized illness until perhaps later in the course of the disease.

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Persistent infection was effected in a porcine kidney cell line (PK-15) with each of 5 strains of hog cholera virus (HCV). For 63 weeks, infected cultures were examined and compared with the noninfected, parallel control culture. Neither virus-induced morphologic nor cytogenetic changes were observed, but viral persistence was repeatedly confirmed by isolation of virus and by immunofluorescence. Infection was apparently maintained by distribution of virus from mother to daughter cells during mitosis. Despite the low yield of extracellular virus, most and perhaps all cells contained the complete viral genome. Differences associated with strains of virus were observed among infected cultures in that some consistently produced more extracellular virus and contained more viral antigen than others. During subculture of infected host cells, all 5 strains of virus were attenuated in virulence, and their administration to pigs resulted in protection against subsequent challenge exposure with highly virulent HCV.

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Six cultures of an established porcine kidney (PK-15) cell line were prepared from a common source. Each of 5 of the cultures was exposed to a different strain of hog cholera virus and the

remaining culture was kept as a noninfected control. Thereafter all cultures were maintained by parallel treatment for an interval of more than 1 year during which they were subcultured twice weekly. Following exposure, cultures became permanent carriers of hog cholera virus. However, other than for continued presence of virus and associated antigens such cultures could not be differentiated from the noninfected control and it was believed at the termination of the study that infected as well as noninfected cultures could have been propagated indefinitely. The carrier state was characterized by: 1) The lack of either virus-induced cytopathology or morphologic changes in infected cells; 2) the persistence of infection in most and probably all the cells in a culture; 3) a growth rate of infected cells similar to that observed with noninfected cells; 4) some resistance to superinfection with the same virus but little or no resistance to superinfection with vesicular stomatitis, Newcastle, parainfluenza 3 or pseudorabies viruses; 5) attenuation of hog cholera virus during subculture or persistently infected cells; and 6) an apparent relationship between growth curves of virus and host cell. Strains of hog cholera virus differed in both their ability to replicate in persistently infected cells and the time required for their attenuation in virulence for pigs. A direct relationship between these two properties was suggested by the fact that the lower yielding strains were also attenuated in less time. Certain pigs that were persistently infected with a moderately virulent strain of hog cholera virus had some resistance to superinfection with a highly virulent strain. Nonetheless, death of such pigs was hastened by the administration of the more virulent strain.

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En el Departamento de Bacteriología del Instituto Nacional de Investigaciones Pecuarias, se investigó la posibilidad de que las vacunas vivas contra el cólera porcino predispongan a los cerdos lo suficiente para permitir la invasión del tracto respiratorio posterior por *Pasteurella multocida*. Para esto, se vacunaron 3 lotes de 5 cerdos cada uno de la raza Yorkshire de 4 meses de edad, con una vacuna comercial de virus vivo modificado contra el cólera porcino y se expusieron por vía intratraqueal a los 2, 7, 17 días postvacunación con un cultivo vivo, encapsulado, de *Pasteurella multocida* (cepa de campo) en una concentración de 1,560 x 10 por ml, inoculándose 3 ml. Se dejaron tres lotes controles: a) con vacuna 3 cerdos; b) con *Pasteurella*, 3 cerdos, y c) sin inocular 4 cerdos. Todos los animales fueron sacrificados 15 días más tarde, dándose un valor numérico a la lesión neumónica, de 1 a 15 puntos, dependiendo de su extensión. Se encontró que todos los animales de los lotes con virus y *Pasteurella* presentaban lesiones neumónicas que promediaban 7.8, 7.8 y 6.6 respectivamente. En los lotes de *Pasteurella* sola y sin inóculo, la mitad de los animales presentaba ligeras lesiones neumónicas que promediaron 1.7 en ambos casos, mientras que en los lotes con virus solo, no se observaron lesiones. Las diferencias entre los lotes tratados y los controles son estadísticamente significativas a $P<0.05$.

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Co-autores: H. Nakagawa, M. Sawada, T. Hanaki. y H. Sazawa.

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Co-autores: H. Yamamoto, A. Fukusho, M. Sawada y H. Sazawa.

OIRSCHOT, J. T. VAN. Persistent congenital swine fever infections. Presentado en Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. s.p. -0795-

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OKANIWA, A. et al. Studies on the lapinized hog cholera virus; pathological observation on the hogs inoculated with lapinized virus and ROVAC. Abstracts of Proceedings Suijokai, Tokyo, 1956. pp. 20-21.
* Compendiado en Veterinary Bulletin 27(7):353. 1957. -0797-

Co-autores: J. Sasahara, R. Ishitani y K. Sugimura.

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* Compendiado en Veterinary Bulletin 30(2):73. 1960. -0798-

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A widespread epizootic of hog cholera in the Dismal Swamp vicinity of North Carolina and Virginia has resulted in a different approach to hog cholera eradication--a "task force" of specialists under focal point control and grouped into functional, as well as geographical, teams (similar to the Emergency Disease Eradication Organization that would apply to foot-and-mouth or other emergency diseases).

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* Compendiado en Veterinary Bulletin 40(10):775-776. 1970. -0801-

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* OSBORNE, J. C. The successful control of hog cholera virus leukopenia with *Hemophiluspertusis* bacterin. American Journal of Veterinary Research 19(73):902-906. 1958. -0804-

* OSBURN, B. I. et al. Border disease-like syndrome in lambs: antibodies to hog cholera and bovine viral diarrhea viruses. Journal of the American Veterinary Medical Association 163(10):1165-1167. 1973. -0805-

Co-autores: G. L. Clarke, W. C. Stewart y M. Sawyer.

Serum samples were collected from fetal and newborn lambs with a naturally occurring border disease-like syndrome and from lambs inoculated with brain-spleen suspensions obtained from affected lambs. Serum neutralization (SN) tests for antibodies to bovine viral diarrhea (BVD) and hog cholera viruses and the single radial immunodiffusion assay for quantitating immunoglobulin G (IgG) were performed. In precolostral samples, serum IgG content was less than 0.10 mg./ml. in control lambs and ranged from 0,10 to 1.1 mg./ml. in affected lambs; postcolostral values exceeded 7.5 mg./ml. Serum-neutralizing antibodies to hog cholera were detected in all of 13 samples examined, the titers ranging from 1:4 to 1:1,024. Serum-neutralizing antibodies to BVD were in 8 of 13 samples that had titers to hog cholera.

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PANITKOV, M. A. The use of the fluorescent antibody technique for studying the interaction between swine fever virus and the SPEV cell culture. Doklady Vsesoyuznoi Akademii Sel'skokhozyaistvennykh Nauk no. 11:40. 1974. -0810-

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* PATOGENIA DE la PP en relación con su difusión. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal, no. 2. 1976. p. 9. -0813-

PAVLOVIC, A. M. Contribution to the knowledge of the role of Chinese strain of lapinated virus in the epizootiology of swine fever. Acta Veterinaria 21(2):121-128. 1971. -0814-

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Hog cholera was diagnosed in 177 herds by examination of specimens from 363 pigs. Specimens from 3,207 pigs in 1,988 herds were examined by fluorescent antibody (FA) procedures on tonsil and spleen, and by histopathologic examinations of brain. Both FA frozen tissue section (FATS) and FA tissue culture (FATC) techniques were utilized. The FA tests were positive in 285 (79%), suspect in 41 (11%), and negative in 37 (10%) pigs from infected herds. Encephalitis was detected in 249 (82%) of 304 brains submitted from these herds. There was agreement between tonsil and spleen FATS examinations for 361 pigs from 199 herds which had either positive or suspect specimens.

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* PESTE PORCINA. In Consulta FAO/CEE de Expertos en la Erradicación de la Peste Porcina y la Peste Porcina Africana, Hannover, 1976. Informe. FAO. Producción y Sanidad Animal, no. 2. 1976. pp. 12-13. -0820-

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LA PESTE porcine classique en France en 1974. Recueil de Médecine Vétérinaire 151(6):377. 1975. -0822-

* PETER, C. P., TYLER, D. E. and RAMSEY, F. K. Characteristics of a condition following vaccination with bovine virus diarrhea vaccine. Journal of the American Veterinary Medical Association 150(1):46-52. 1967. -0823-

> Within 10 to 20 days after vaccination with bovine virus diarrhea (BVD) vaccine, a mucosal disease (MD)-like syndrome occurred in a small number of cattle in a few herds. Clinically, pathologically, and serologically, the condition appeared similar to the field form of MD. The pathogenesis of the condition was not understood, and although the vaccine was etiologically involved, it was believed that the condition was primarily caused by failure of the immune mechanism in a few animals.

PETROV, P. G. and PHYLIPOV, G. G. Change in the activity of some serum enzymes in swine effected by acute hog cholera. In World Veterinary Congress, 20., Thessaloniki, Greece, 1975. Summaries. v.2, pp. 992-993. -0824-

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PETROV, V. F. and MAKSIMOVICH, V. V. Experiments with combined vaccine against swine fever, erysipelas and anthrax in laboratory animals. Uchenye Zapiski Vitebskogo Veterinargogo Instituta 25:15-19. 1972. -0826-

_____ and BUTYANOV, D. D. A single vaccination to protect pigs against several infections. Veterinariya no. 11:66-68. 1974. -0827-

PHILLIPS, C. E. Safety testing hog cholera live virus modified vaccines. Proceedings of the U. S. Livestock Sanitary Association 70:302-309. 1966. -0828-

* PHILLIPS, C. E. *In vitro* potency tests for anti-hog cholera antibodies: a test for anti-hog cholera serums and a test for herd exposure. American Journal of Veterinary Research 29(5):1097-1102. 1968. -0829-

A new test using the serum neutralization-fluorescent antibody (SN-FA) technique for the evaluation of anti-hog cholera serum was devised. The antibody content of 127 commercially produced antiserums was evaluated. Validity was established by titrating serials representative of a range of antibody contents in susceptible test pigs. Large numbers of coverslip monolayer cell cultures were processed rapidly with apparatus of special design. A modification of the serum-neutralization test was used to determine the serologic status of pigs.

* PILCHARD, E. I. Hog cholera lesions in swine given modified vaccine. Journal of the American Veterinary Medical Association 148(1):48-51. 1966. -0830-

Gross and microscopic lesions of hog cholera were experimentally produced in pigs which had been given modified hog cholera vaccine. Of 16 pigs given modified vaccine, 4 developed hemorrhages of the lymph nodes and 7 developed petechial hemorrhages of the mucosa of the urinary bladder. Mild nonsuppurative meningoencephalitis and infiltration of the choroid plexus by lymphocytes and macrophages were observed microscopically. Fluorescent antibody (FA) test reactions were obtained with specimens from 3 of 16 vaccinated pigs. All the pigs given modified hog cholera vaccine remained clinically normal. None developed leukopenia, and body temperatures remained normal.

* _____. Experimental inactivated-virus hog cholera vaccines: induction period of immunity. American Journal of Veterinary Research 28(125): 915-923. 1967. -0831-

New experimental inactivated-virus hog cholera vaccines were used to shorten the period of induction of immunity and to maximize the relative number of swine which develop immunity among those vaccinated. Weaned pigs given *beta* propiolactone (BPL)-inactivated hog cholera virus, which was chemically linked to inactivated *Bordetella pertussis* organisms, mixed with bacterial endotoxin, or mixed with bacterial endotoxin and suspended as a water-in-oil emulsion, were immune to hog cholera virus a week later. Of 10 experimental vaccines studied, bordetella-azo-BPL-inactivated-virus hog cholera vaccine required the shortest period for induction of immunity and produced immunity in a greater proportion of the total number of vaccinated swine than was produced by other experimental inactivated-virus hog cholera vaccines. Three of 4 pigs given bordetella-azo-BPL-inactivated-virus vaccine were immune to hog cholera virus a week later.

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* _____. Chromosomal variation in a pig kidney cell line persistently infected with hog cholera virus. American Journal of Veterinary Research 27(118): 737-745. 1966. -0837-

A pig kidney cell line with a modal chromosome number of 38 was infected with virulent hog cholera virus and carried as a persistently infected culture for 84 passages. Continued presence of the virus was confirmed by staining infected cells with fluorescent antibody. Cytogenetic studies were performed on chromosome preparations made at the 82nd passage, and results were compared with noninfected cells of the same line. The modal chromosome number of the infected cells was 37, and several karyotype variations were observed in metaphase figures containing this chromosome number. The most obvious findings were deletion of telocentric chromosomes and concomitant appearance of unmatched submetacentric chromosomes. Most of the infected cells also had an increased number of sutelocentric chromosomes. It thus appeared that most of the alterations resulted from centromeric fusions. A large metacentric marker chromosome was in both the noninfected cell line and the persistently infected cells.

* _____ and WOODS, L. K. Cytogenetic alterations in swine kidney cells persistently infected with hog cholera virus and propagated with and without antiserum in the medium. American Journal of Veterinary Research 29(1): 153-164. 1968. -0838-

An established swine kidney cell line was infected with the Ames strain of virulent hog cholera (HC) virus and carried

as a persistently HC virus-infected culture through a series of 41 passages. Noninfected cells of identical passage levels were studied in parallel. The first 17 subcultures were propagated in medium containing no anti-HC viral serum. The infected cells underwent a burst of endoreduplication (chromosomal doubling during interphase) for 4 passages shortly after they were initially infected. The mode of the near-diploid cell populations remained relatively unaffected, but there was a decrease in their numbers in the total cell populations. There was an increase in the near-tetraploid cell population of the noninfected culture, but this was significantly less in the infected culture. There was a relatively high incidence of chromosomal pulverization in the infected culture. In addition to endoreduplication, the occurrence of anomalous mitoses also contributed to the evolvement of the near-tetraploid cell populations of both noninfected and infected cultures. The next 16 subcultures of the infected cells were propagated in medium containing anti-HC viral serum. At the end of these 16 passages, approximately 98% of both the infected and the noninfected cell populations were at the near-tetraploid level. During these 16 passages, endoreduplication was observed rarely, and chromosomal pulverization in the infected culture decreased steadily. The HC virus was not detected in the supernatant culture medium of the infected culture, but there was HC viral antigen in the cells. The final 8 subcultures of noninfected and infected cultures were propagated in the same medium as the first 17 (i.e., without added anti-HC viral serum). Near-tetraploid cell populations of both cultures remained above the 95% level. Endoreduplication was practically nonexistent. Chromosomal pulverization increased markedly in the infected culture during the final 8 passages.

* _____ and KNIAZEFF, A. J. Susceptibility of cultured mammalian cells to infection with virulent and modified hog cholera viruses. American Journal of Veterinary Research 29(5):1033-1040. 1968. -0839-

Twenty-four primary cell cultures, 14-low-passage cell line cultures, and 13 high-passage established cell line cultures derived from 7 orders and 29 species of mammals were exposed to virulent and modified hog cholera (HC) viruses. The presence of HC viral antigen in exposed cultures was determined with homologous fluorescent antibody technique (FAT). Twelve of 24 primary cell cultures were susceptible to both virulent and modified HC viruses. Ten of the 14 low-passage cell line cultures were susceptible to virulent HC virus, and 13 of the 14 cultures were susceptible to modified HC virus. Seven of the 13 high-passage established cell line cultures were susceptible to virulent HC virus, and 8 of the 13 cultures were susceptible to modified HC virus. Implications of these *in vitro* results were discussed in relation to possible natural reservoirs of HC virus.

* PIRTLE, E. C. *In vitro* spread of hog cholera viral infection from cell to cell: demonstration of viral antigen in cytoplasmic bridges. American Journal of Veterinary Research 30(11):1913-1919. 1969. -0840-

Porcine kidney /PK-15 (NADL)/ cells were incubated in Sykes-Moore chambers and were observed and photographed, using phase-contrast optics. Cytoplasmic bridges were formed between cells: a) by the extension and fusion of cytoplasmic projections from one cell to another cell; b) by the initial contact of cells and subsequent stretching and narrowing of the areas of cytoplasm involved; and c) by the fusion of areas of cytoplasm of closely adjacent cells which remained adjacent during the bridging process. Incomplete monolayer cultures on coverslips of unfixed PK-15 (NADL) and embryonic swine kidney (ESK) cells exposed with the Ames virulent strain (AHCV) and a tissue culture-adapted vaccinal strain (TCV) hog cholera (HC) virus were examined by phase-contrast microscopy. Noninfected control cultures were similarly examined. All cultures examined had a relatively large number of cytoplasmic bridges which varied in length, diameter and number between cells. Apparent differences in the occurrence of cytoplasmic bridges were not observed in control and HC virus-infected cultures or in infected cultures which were nourished with anti-HC serum after they were exposed to HC virus. Similar incomplete monolayer cultures also were examined by the fluorescent antibody technique (FAT). Cytoplasmic bridges containing HC viral antigen were observed in all cultures exposed to HC virus. Relatively short bridges connecting 2 cells, relatively long bridges connecting cells across acellular areas, and bridges connecting cells in sequence were observed.

* _____. *In vitro* spread of hog cholera viral infection from cell to cell: demonstration of viral antigen in dividing cells. American Journal of Veterinary Research 30(11):1909-1912. 1969. -0841-

Incomplete monolayer cultures of porcine kidney /PK-15 (NADL)/ cells and primary embryonic swine kidney (ESK) cells were exposed to the Ames virulent strain (AHCV) and to a tissue culture-adapted vaccine strain (TCV) of hog cholera (HC) virus. Some of the cultures were nourished with medium containing anti-HC viral swine serum. A direct fluorescent antibody technique (FAT) was used to detect HC viral antigen in cells of infected cultures. Both types of cell cultures had infected mitotic cells in all stages of cell division, regardless of the strain of HC virus to which they were exposed or of the nutrient medium which was used.

* PIRTLE, E. C. and MENGELING, W. L. Deoxyribonucleic acid synthesis in cultured porcine kidney cells infected with hog cholera virus. American Journal of Veterinary Research 31(10):1795-1799. 1970. -0842-

The time intervals of the phases of the cell cycle were estimated for the PK-15 (NADL) cell strain by pulse-labeling replicate cultures with tritiated thymidine (H^3TdR) and determining the percentage of labeled metaphases at 3-hour intervals after labeling. The mean generation time of the cell culture was 15.2 hours, the period of deoxyribonucleic acid (DNA) synthesis (S phase) was 6.9 hours, the post-S phase (G_2) plus mitosis was 3.8 hours and the postmitotic phase (G_1) was 4.5 hours. Acute infections and persistent infections of PK-15 (NADL) cells with hog cholera virus (HCV) extended the period of DNA synthesis 0.9 and 0.7 hours, respectively.

* _____ and MENGELING, W. L. Antigenic differences in two hog cholera virus strains. American Journal of Veterinary Research 32(10):1473-1477. 1971. -0843-

Using strain-specific antiserums, strains Ames and 331 of hog cholera virus (HCV) were examined for differences in antigenic properties. Strains Ames and 331 of HCV were neutralized to a greater degree by homologous than by heterologous antiserums. Fluorescent antibody conjugates prepared from anti-Ames serums stained cells infected with Ames HCV more intensely than cells infected with 331 HCV. Conjugates prepared from anti-331 serums stained cells infected with Ames HCV or 331 HCV with equal intensity. Ames antiserums present in overlay medium inhibited the spread of Ames HCV in cell culture to a significantly greater degree than 331 HCV, and 331 antiserums inhibited the spread of 331 HCV to a greater degree than Ames HCV.

* _____. Hog cholera virus yields in swine kidney cells fuses with Beta-propiolactone-inactivated Sendai virus. American Journal of Veterinary Research 33(1):121-125. 1972. -0844-

PK-15 (NADL) swine kidney cell cultures were treated with beta-propiolactone (BPL)-inactivated Sendai virus (parainfluenza-1 virus), and cells in these cultures developed numerous syncytia. Sendai virus-treated and nontreated control cell cultures were exposed to tissue culture-adapted stock (2×10^6 plaque-forming units (PFU)/ml.) of hog cholera (HC) virus, 5×10^4 PFU/ml. of HC virus, or to HC virus harvested from Sendai-treated and nontreated cell cultures. In all instances, the greatest yields of HC virus were obtained from cell cultures that were treated with Sendai virus before exposure to HC virus.

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Co-autores: I. R. Littlejohus, A. C. Gardiner, J. T. Vantsis y R. A. Huck.

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PLEVA, J. and CABADAJ, R. Gel diffusion test in forensic veterinary medicine (for evidence of immunity to swine fever). Veterinársky 16(5/6):175-177. 1974. -0850-

POLAK, L. and SIMA, O. Swine fever, its development and eradication in Czechoslovakia. Bulletin de l'Office International des Epizooties 72(1):653-664. 1969. -0851-

POPA, M. et al. Serum protein response in the pig, correlated with the pathogenicity of swine fever virus strains. Archiva Veterinaria 8(2):45-52. 1972. -0852-

Co-autores: S. N. Chisiu, S. Mihaita y S. Tebres.

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Co-autores: N. Manolescu, D. Paltineanu y R. Movanu.

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Co-autores: N. S. Chisiu, T. Albu, S. Tibrea, G. Barnaure y N. Totorcea.

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Co-autores: S. Tibrea, G. Baurnaure, N. Totorcea y T. Albu.

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POPOVIC, M. et al. Safety trials of lapinized swine fever C strain vaccine for immunoprophylaxis in pregnant gilts. Praxis Veterinaria 22(4):175-178, 180-184. 1974. -0860-

Co-autores: S. Murgaski, I. Ninkov, B. Marjanski y K. Salahovic.

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Co-autores: R. Milojević, A. Ovdijenko y B. Stajner.

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* PRATT, D. W. A study of postvaccinal (hog cholera) losses. American Journal of Veterinary Research 13(49):526-530. 1952. -0863-

Typically affected pigs from 16 Illinois herds that suffered heavy postvaccinal losses revealed gross lesions of septicemia at autopsy. Blood and other tissues collected at the autopsy of typically affected pigs from these herds were injected into: 1) cholera-susceptible pigs; 2) cholera-susceptible pigs that simultaneously received protective doses of hog cholera antiserum; 3) pigs previously given hog cholera antiserum and hog cholera virus. The results of these inoculations revealed reasonable evidence of the presence of hog cholera virus in each inoculum. This means that: (a) where a single herd was represented in an inoculum, hog cholera virus was present in each herd, and (b) where specimens from several herds were combined for inoculation, hog cholera virus was present in one or more of the herds represented in the composite inoculum Two brands of commercial hog cholera antiserum and hog cholera virus were

used in cross-immunization pig inoculation tests. Field use of one of the brands had been followed by considerable post-vaccinal loss. The viruses produced fatal illness in all but 1 nonprotected pig but failed to produce noticeable effect when injected into pigs that had been previously vaccinated. Moreover, all simultaneously treated pigs remained healthy, indicating that each hog cholera antiserum provided protection against each of the viruses.

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* Compendiado en Veterinary Bulletin 43(10):554. 1973. -0865-

_____ and PERRENOT, F. Titration of anti-swine fever neutralising antibodies by immunofluorescence. Recueil de Médecine Vétérinaire 149(12): 1567-1576. 1973.
* Compendiado en Veterinary Bulletin 44(5):298. 1974. -0866-

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RIBEIRO, A. M. R., RIBEIRO, J. M. and AZEVEDO, J. R. Eradication of swine fever in Portugal. In World Veterinary Congress, 19th, Mexico, D. F., 1971. Proceedings. México, D. F., 1971. pp. 520-524. -0897-

* RIBEIRO, L. O. C., SUGAY, W. and PASQUALIN, O. L. Proteção contra peste suína por células da linhagem IB-RS-2. Arquivos do Instituto Biológico São Paulo (Brasil) 38(4):279-281. 1971. -0898-

Personal communication from German researchers (A. Mayr and K. Danner) that the cells of the IB-RS-2 line had a hog cholera antigen, revealed through fluorescent antibody technique, led us to the proposition of verifying whether such cells contained some protection factor against hog cholera that would be active *in vivo*. Susceptible pigs, weighing about 40 kg were inoculated by the intramuscular route with either whole cells or lysed cells or with supernatant fluid of cell cultures. After 4, 8 and 18 days of the cell inoculation all the pigs, along with a control group, were submitted to a challenge of lethal doses of virulent hog cholera virus. The results obtained were the following: 1) the inoculation of either whole cells or lysed cells or supernatant culture medium did not cause clinical signs of hog cholera till the 18th day postinoculation; 2) the animals inoculated with cellular material or with supernatant culture medium were protected against hog cholera. This fact was stated by death of the animals of the control group, which received the same challenge; 3) the protection conferred to the animals was already present at the 4th day postinoculation. Lysed cell inocula, as small as 0.5 ml, were enough to protect animals against the challenge. These results are coincident with those obtained by French workers, as stated by P. Prunet (personal communication).

* _____ et al. Proteção de porcos contra o vírus da peste suína (VPS) pelo clone C-13 da linhagem celular suína IB-RS-2. Arquivos do Instituto Biológico de São Paulo (Brasil) 43(1/2):49-52. 1976. -0899-

Co-autores: I. Koseki, T. G. Abuhad y W. Sugay.

Experiments were done with cells C-13 of the IB-RS-2 swine cell line in order to examine their ability to protect pigs against hog cholera. So far the results have showed that 6 x 10^6 cells and the nutrient medium of one cell culture flask could induce protection of a great number of pigs.

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Co-autores: J. A. Rosa Azevedo, M. J. O. Teixeira, M. C. Braço Forte, A. M. R. Ribeiro, E. N. E. Oliveira, C. Grave Pereira y J. D. Vigario.

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ROBERTSON, A. et al. Hog cholera. IV. Detection of the virus in tissue culture preparations by the fluorescent antibody technique. Canadian Journal of Comparative Medicine and Veterinary Science 29:234-241. 1965. -0904-

Co-autores: A. S. Greig, M. Appel, A. Girard, G. L. Bannister y P. Boulanger.

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Co-autores: G. L. Bannister, P. Boulanger, N. Apple y D. Gray.

ROBIJNS, K. G. Swine fever and swine fever eradication in the Netherlands. Bulletin de l'Office International des Epizooties 72(1):633-651. 1969. -0906-

ROBSON, D. S. et al. The serum neutralization test as an indicator of immunity against hog cholera. Proceedings of the U. S. Livestock Sanitary Association 65:338-342. 1961. -0907-

Co-autores: L. Coggins, B. E. Sheffy y J. A. Baker.

* RODABAUGH, D. E. y ELDER, C. The effect of a low protein ration in hog cholera immunization. Journal of the American Veterinary Medical Association 126(938):418-421. 1955. -0908-

* _____, WRIGHT, H. B. and ELDER, C. Factors influencing immunity in hog cholera vaccination. Journal of the American Veterinary Medical Association 136(12):617-621. 1960. -0909-

Two series of tests were conducted on swine that had been vaccinated with a hog cholera vaccine either of swine origin or of rabbit origin. Pigs that were maintained on a protein-deficient ration at the time of vaccination showed a severe reaction, with several death losses, when challenged with live hog cholera virus at 75 and 145 days after vaccination. Swine fed a balanced ration at time of vaccination survived challenge with very little reaction and no death losses. Reactions and

death losses were greater in groups that had been vaccinated with a commercial vaccine of swine origin, but these results were not regarded as an indication of an inferior product. *Salmonella choleraesuis* and hog cholera virus, given simultaneously, caused the death of 3 out of 4 pigs which had previously been vaccinated with a vaccine of swine origin while being fed a balanced ration. Filtered blood from the pigs that died caused typical clinical signs and lesions of hog cholera when injected into a susceptible pig.

* RODABAUGH, D. E., WRIGHT, H. B. and ELDER, C. The effect of *Salmonella choleraesuis* infection on immunity to hog cholera. Journal of the American Veterinary Medical Association 145(3):252-257. 1964. -0910-

According to evidence presented herein, pigs vaccinated against hog cholera with vaccine that would protect against a standard challenge dose of virulent hog cholera virus would not lose that protection when simultaneously exposed to *Salmonella choleraesuis* organisms and the challenge virus. *Salmonella choleraesuis* infection induced at the time of vaccination with a hog cholera vaccine of rabbit origin did not significantly interfere with establishment of immunity to hog cholera. Slight differences in morbidity and mortality at the time of challenge with hog cholera virus were considered to be a result of stunting of some pigs from previous exposure to *Salmonella* infection.

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ROSAS CORTES, M. L. Campaña nacional de erradicación de cólera porcino. Técnica en Agricultura y Ganadería (México) 2(17):43-44, 46-48. 1970. -0912-

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* ROUZE, P., HOUDAYER, M. and METZGER, J. J. Immune response in piglets; the effect of specific passive immunity on immune response against an inert antigen, hen egg-white lysozyme. Annales de Zootechnie 24(4):817. 1975. -0915-

In previous reports, we already showed that piglets have been actively immunized with lysozyme in the presence of specific antibodies acquired by the colostral way or by intraperitoneal

injection. The purpose of this new experiment, realized without any adjuvant was to study the role of this parameter. In these conditions the injection of increasing doses of lysozyme, even in the absence of any specific antibody, stimulated a less efficient immunization. This active immunity was inhibited by rather low passive antibody amounts. This result led us to define the passive antibody threshold beyond which lysozyme antigenic stimulation was ineffective and to show that its level was lowered in the absence of adjuvant. In this paper, the practical implication of this threshold concept is discussed. In the same experiment, we compared the effect of the stimulation with a living and an inert antigen, namely hog-cholera virus vaccine and lysozyme, in passively immune animals. We observed a narrow parallelism between these two systems. It appeared, at last, that the challenge performed with the fully virulent Alfort strain of swine fever virus led to an immunodepression which tends to be suppressed by the vaccination.

RUBAJ, B. Morphology of adrenals in swine fever. Annales Universitatis Mariae Curie-Sklodowska. Section DD 1957. 12:1-27. 1959. (Origjnal en polaco. Sumarios en inglés y ruso).
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Co-autores: M. Apple, G. L. Vannister, K. Mori, D. Cochrane y P. Bolulanger.

RUTILI, D., TITOLI, F. and MOROZZI, A. Ultrastructural studies of some organs from pigs with swine fever. Folia Veterinaria Latina 7(2):165-173. 1977.
* Compendiado en Veterinary Bulletin 48(4):314. 1978. -0918-

RYU, E. et al. Efficacy of combined vaccination with Japanese encephalitis attenuated vaccine and lapinized swine fever vaccine. Taiwan Journal of Veterinary Medicine and Animal Husbandry no. 27:1-5. 1975. -0919-

Co-autores: C. Y. Chen, C. M. Wang, C. C. Lin, S. S. Chen y S. C. Yang.

* SAM T., R. y CALETTI P., E. Estudio de antígenos intracelulares relacionados con la replicación del virus del cólera porcino. Revista de Investigaciones Pecuarias (Perú) 2(2):173-181. 1973. -0920-

Se ha realizado el estudio de algunos antígenos relacionados con el virus del cólera porcino, empleándose como fuente de antígeno, páncreas de cerdos infectados, provenientes de los

alrededores de Lima y de animales experimentalmente infectados con cepas virulentas y cepas atenuadas de cólera porcino, produciéndose los respectivos antisueros homólogos en conejos. Mediante el empleo de la inmunodifusión e inmunoelectroforesis, se observó una línea de precipitación específica para el material con virus virulento y otra común con los antígenos de la cepa de virus atenuado, lo que permite afirmar que existe una reacción cruzada entre ambas cepas. La identificación de una línea específica para el virus virulento, mediante la inmunodifusión permite que esta prueba pueda emplearse como un método rápido, económico y eficaz para el diagnóstico de la enfermedad. En un intento de aislar el antígeno específico con los métodos de precipitación con sales de sulfato de amonio y los procedimientos cromatográficos sólo se ha podido obtener una parcial purificación.

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Co-autores: V. Imamura, H. Kuwabara, M. Kamizono, S. Kuramasu, A. Kawakubo y N. Hayashi.

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Co-autores: A. Kawakubo, H. Kuwahara, I. Kurata y N. Hayashi.

SANCHEZ BENITEZ, G. Contribución al estudio de la sanidad animal de climas tropicales, valoración de los diferentes tipos de vacuna para cólera porcino en Iguala, Guerrero. México, D. F., Universidad Nacional Autónoma, 1964. 32 p. -0923-

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* Compendiado en Veterinary Bulletin 29(7):374. 1959. -0925-

SANDERS, E. F. et al. Serum interference with modified live hog cholera vaccines. Veterinary Medicine 56:261-266. 1961. -0926-

Co-autores: A. H. Brueckner, J. W. Newberne y F. E. Mitchell.

SASAHARA, J. et al. Supplementary studies on lapinized hog cholera virus. Bulletin of the National Institute of Animal Health 37:33-45. 1959. (Original en japonés).
* Compendiado en Veterinary Bulletin 30(2):73. 1960. -0927-

Co-autores: S. Hayashi, T. Kumagai, K. Sugimura, M. Chikatsune, K. Munekata, T. Nagai, K. Hirasawa y K. Kato.

_____ et al. Field experiments of hog cholera live vaccine prepared in guinea-pig kidney cell culture. National Institute of Animal Health. Quarterly 9:83-91. 1969. -0928-

Co-autores: T. Kumagai, Y. Shimizu y S. Furuuchi.

_____. Hog cholera: diagnosis and prophylaxis. National Institute of Animal Health. Quarterly 10:57-81. 1970. -0929-

_____. New hog cholera live virus vaccine. In International Pig Veterinary Society Congress, 3rd., Lyon, 1974. Proceedings. pp. HC14(4 p.). -0930-

_____. Hog cholera live vaccine. Bulletin of the National Institute of Animal Health 70:53-55. 1975. -0931-

_____. Diagnosis of swine fever in Japan. In Diagnosis and Epizootiology of Classical Swine Fever. Luxembourg, Commission of the European Communities, 1976. pp. 51-52. -0932-

_____. Further experiments on the GP vaccine of swine fever. In Diagnosis and Epizootiology of Classical Swine Fever. Luxembourg, Commission of the European Communities, 1976. pp. 67-82. -0933-

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Co-autores: Y. Nichimura, T. Hanaki y K. Nobuto.

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Co-autores: H. Tamura, F. Obata y C. Hiraga.

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* _____. Final phase of hog cholera eradication. Journal of the American Veterinary Medical Association 164(3):304-306. 1974. -0940-

* SAUNDERS, G. C. Development and evaluation of an enzyme-labeled antibody test for the rapid detection of hog cholera antibodies. American Journal of Veterinary Research 38(1):21-25. 1977. -0941-

A rapid enzyme-labeled antibody (ELA) microtechnique for the screening of swine for hog cholera antibodies was developed and evaluated with a blind study, using a 640-sample hog cholera serum bank. The total time to run a group of 22 samples was approximately 1 hour. The ELA test results correlated greater than 99% with hog cholera serum-neutralization test results on the same serums. Test results also indicated that the ELA test shares with the hog cholera serum-neutralization test the problem of cross reactions between the antibodies of hog cholera and bovine viral diarrhea.

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Co-autores: G. A. Young, A. J. Luedke y R. L. Kitchell.

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SCHULZ, N. E. Field trials using succinylcholine chloride for euthanasia of hog cholera infected swine herds. Proceedings of the U. S. Livestock Sanitary Association 72:280-282. 1969. -0950-

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Formalinized rabbit, swine, or sheep erythrocytes were coated with hog cholera antibodies through diazo linkages with tetrazotized benzidine. Suspensions of such antibody-coated erythrocytes were agglutinated by blood and splenic extracts containing hog cholera virus, but not by blood and splenic extracts free of virus.

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SERRES, H. and RAMISSE, J. Introduction of swine fever into Madagascar. Revue d'Elevage et de Médecine Vétérinaire des Pays Tropicaux 22:315-318. 1969. -0963-

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SHIMIZU, K. et al. Antibody survey in pigs inoculated with a live swine fever virus vaccine. Journal of the Japan Veterinary Medical Association 25(10): 604-610. 1972. -0967-

Co-autores: Y. Hanamoto, Y. Sakano, K. Sugawara, A. Tadokoro, E. Fujimura, H. Oyamada, H. Mizushima, R. Tanaka y K. Aikawa.

SHIMIZU, M. Experimental infection of goats with swine fever virus. Bulletin of the National Institute of Animal Health 72:11-16. 1976. -0968-

_____ and FURUUCHI, S. Neutralizing antibodies in goat serum against the viruses of swine fever and bovine viral diarrhea. Bulletin of the National Institute of Animal Health 72:7-10. 1976. -0969-

* SHIMIZU, Y. et al. A mutant of hog cholera virus inducing interference in swine testicle cell cultures. American Journal of Veterinary Research 31(10):1787-1794. 1970. -0970-

Co-autores: S. Furuuchi, T. Kumagai y J. Sasahara.

Two clonal preparations designated E^+ and E^- hog cholera (HC) viruses, which differed in interactions with Newcastle disease (ND) viruses in swine testicle (ST) cell cultures, were obtained during adaptation of HC virus to guinea pig kidney cells. The E^+ virus exerted an enhancing effect on the cytopathic effect (CPE) of ND virus in ST cells, whereas the E^- virus inhibited multiplication of ND virus in ST cells. After 4 passages at limiting dilution, E^- virus retained its distinctive character against ND virus. A quantitative assay procedure was developed for E^- virus, using its interfering action. Although attenuated and wild strains of viruses were antigenically similar, attenuated virus was more sensitive to specific antibody than was the wild viral strain.

_____, KUMAGAI, T. and SASAHARA, J. GP vaccine controls hog cholera in Japan. Norden News 51(2):11. 1976. -0971-

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Paper presented at the Autumn Meeting of the National Academy of Sciences, Rockefeller Institute and New York Botanical Garden, New York, 1957.

Consideration of the epidemiology of hog cholera suggests that its causative virus must be perpetuated in nature in a nonporcine reservoir intermediate host. Experiments conducted with the swine lungworm indicate that this nematode can serve as a reservoir host for the hog cholera virus but also that it harvors the virus in a masked or occult noninfective form. Swine fed lungworm larvae containing the masked hog cholera virus ordinarily do not come down with hog cholera. However, the appearance of good health shown by such animals is misleading because all that is required to bring them down with a fatal attack of hog cholera is the application of some relatively innocuous provocative stress. In the experiments reported, migrating *Ascaris* larvae supplied the stimulus that provoked masked hog cholera virus to infectivity.

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SIMONE, F. DE and CIVARDI, A. Practical results of the immuno-fluorescence technique for the diagnosis of hog cholera, Aujeszky's disease, equine rhinopneumonitis. Veterinaria Italiana 24(5/6):223-231. 1973.
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SIMONYI, E. and REGŐS, G. Investigation on the pathological lesions in pigs vaccinated with crystal-violet swine fever vaccine. Acta Veterinaria Academiae Scientiarum Hungaricae 6:49-54. 1956.
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SINGH, V. P. et al. Diagnosis of swine fever by indirect haemagglutination test. Indian Journal of Animal Health 15(1):45-48. 1976.
* Compendiado en Veterinary Bulletin 47(4):269. 1977. -0979-

Co-autores: R. P. Misra, N. C. Srivastava, K. C. Kinha y T. S. Gulrajani.

SIRBU, Z., IEREMIA, D. and BONA, C. Immunofluorescent microscopy in the diagnosis of swine fever. British Veterinary Journal 120:587-591. 1964. -0980-

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Co-autores: R. Bermúdez, F. Pérez-Abella, R. Vignale y M. Ferrari.

SKOKNIC K., A. Prevenir la peste porcina es tarea esencial en los criaderos. Agroinformativo (Chile) no. 83:1-4. 1973. -0982-

SKUPYI, M. F. and TIKHONOV, M. F. Role of wild swine in the distribution of swine fever. Veterinariya no. 11:59-60. 1973. -0983-

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_____. Nationwide eradication of hog cholera committee. Proceedings of the U. S. Animal Health Association 79:285-286. 1975. -0985-

* SMITH, H. R., FERGUSON, L. C. and SANGER, V. L. The effect of high levels of Aureomycin (Chlortetracycline) in the ration on development of immunity following vaccination against hog cholera. Journal of the American Veterinary Medical Association 129(4):162-166. 1956. -0986-

Chlortetracycline was fed to swine at levels of 100, 200, and 400 Gm. per ton of feed 18 days prior to and 26 days after vaccination against hog cholera with a modified level virus. The production of immunity was not affected by the chlortetracycline. The pigs given chlortetracycline gained more rapidly than the controls, the rate increasing with the level of antibiotic fed. Vaccinated animals which did not receive the antibiotic gained an average of 13 lbs. less than controls which received neither antibiotic nor vaccine. Animals fed the highest level of chlortetracycline displayed less change in temperature or appetite when vaccinated than those receiving less of the antibiotic, the reaction being greatest in the animals that received no antibiotic. All pigs which were given the vaccine survived a challenge with virulent hog cholera virus, while 9 of 10 unvaccinated pigs died of the disease.

* _____ and KING, N. B. Passive immunity to hog cholera in nursing pigs. Journal of the American Veterinary Medical Association 132(3):107-109. 1958. -0987-

A preliminary study of the duration of passive immunity to hog cholera in pigs whose dams were immunized with antiserum and virulent virus, or with one or more doses of a modified live virus vaccine (Rovac), indicated the following: 1) a single dose of the vaccine at 6 weeks of age gave only slight evidence of protection in the offspring and they all succumbed when challenged; 2) doses of the vaccine at 6 weeks of age and when 90 days pregnant resulted in a resistance to challenge at 4 and 6 weeks of age, and in 1 of 2 pigs at 8 weeks of age; and 3) vaccination with antiserum and virulent virus at 6 weeks of age produced sufficient passive antibodies to enable the offspring to withstand the four-and six-week challenge. The pigs of the gilt which was given the vaccine when 6 weeks of age, 90 days pregnant, and two weeks postpartum were the result of superfetation, obtained no colostrum, lacked *gamma* globulin, and consequently all failed to survive. All pigs from the unvaccinated control gilt died when challenged.

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The present knowledge of the serological relationship between bovine mucosal disease (MD) virus and the swine fever (SF) virus is reviewed. Contrary to reported findings, the results of serum neutralisation tests on a number of Australian pig serums indicates that the MD virus may be infecting pigs in Australia. Pigs were exposed to four strains of MD virus, two of which stimulated both MD and SF neutralising antibodies and the pigs were resistant to challenge with SF virus. A third strain of MD virus produced moderate titres of MD antibody, no SF antibody, and protected pigs against challenge with SF virus; a fourth strain produced only very low titres of MD antibody, no SF antibody and the pigs died after challenge with SF virus. It is concluded that the presence of SF neutralising antibody in Australian pig serums can be explained in most cases on the basis of a previous MD virus infection.

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Co-autores: J. E. Thigpen, D. M. Bedell y W. L. Schwartz.

Specimens from swine representing 462 cases of suspected hog cholera, submitted to a clinical laboratory for diagnosis, were examined by fluorescent antibody (FA) test, using a pig kidney (PK-15) cell line. Of these, 146 (32%) were positive on the FA tests, and 169 (37%) were positive when brain lesions were used as the diagnostic criterion. The results of the 2 methods agreed in 81.6% of the cases examined and disagreed in 18.4%. There were 53 cases (11.0%) diagnosed as hog cholera on the basis of brain lesions that were not confirmed by the FA test. There were 32 cases (7.0%) positive by the FA test that were negative for brain lesions. The FA test was found to be a specific, accurate, and rapid method for the routine diagnosis of hog cholera. According to our experience, this method was superior to conventional methods for diagnosis of hog cholera.

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Swine from scattered origins in Georgia were tested at slaughter for the presence of hog cholera virus in their spleens by means of the fluorescent antibody tissue culture test (FATCT). Virus was not found in 572 healthy hogs from 13 abattoirs. Virus was not found in 454 sows and rough hogs. In 254 swine sent to slaughter because of confirmed cases of hog cholera in the herds of origin, 7 pigs had hog cholera virus.

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STEWART, W. C. A preliminary report on *in utero* transmission of hog cholera virus in pregnant sows. In Symposium on Factors Producing Embryonic and Fetal Abnormalities, Death and Abortion in Swine, Chicago, Ill., 1967. Proceedings. Washington, D. C., Agricultural Research Service, 1969. pp. 117-121. -1006-

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Co-autores: E. A. Carbrey, E. W. Jenney, C. L. Brown y J. I. Kresse.

Sixty-six pigs were exposed experimentally to bovine viral diarrhea (BVD) virus. Several strains of the virus and routes of administration were utilized. The immunologic, pathologic, serologic, and virologic responses of pigs in

selected groups were determined. In a group of pigs exposed twice to a field strain of virus, the serologic responses consisted of moderate to high BVD antibody titers and low cross-neutralizing titers against hog cholera (HC) virus. When given virulent HC virus, severe clinical reactions occurred in the BVD-exposed pigs, but 8 of 9 pigs survived. Further evidence of a viremia was established by the isolation of BVD virus from 7 pigs. Isolates were cultured from the lungs of 1 pig and the blood of 5 pigs exposed intranasally (I.N.). In 2 pigs, the blood isolates were detected 14 days postexposure (DPE). Isolates were recovered from the ileum, mesenteric lymph nodes, and spleen of 1 pig exposed intramuscularly (I.M.). In 2 swine herds investigated for HC, serologic tests for BVD were done after low to moderate HC antibody titers were detected. In each case, higher BVD antibody titers were detected, suggesting that the former were cross-neutralization titers resulting from BVD infection. One herd had been in contact with BVD-vaccinated cattle and the other herd had been fed bovine offal.

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Pregnant sows, vaccinated with attenuated or inactivated vaccines, were inoculated with a field strain of low-virulent hog cholera (HC) virus to determine if transplacental infection occurred. Serologic responses were detected with the fluorescent antibody serum-neutralization technique and transplacental infection was confirmed by isolating the virus from the offspring, using the fluorescent antibody cell culture technique (FACCT) and pig inoculation. Transplacental infection did not occur in 9 sows that had been vaccinated with attenuated virus and had an antibody titer of $\log_{10}$ 1.2 or greater when given injections of the field virus. One sow which was supposed to have been vaccinated, but lacked measurable antibody titer and died after infection, was shown to have transmitted the virus to its young *in utero*. In contrast, antibody titers were low or nondetectable in 12 sows that were vaccinated with the inactivated vaccine. When inoculated with the field virus, these sows had variable clinical responses, and in 3 of the 6 sows that produced litters, transplacental infection occurred. Data obtained from the experiments provided information on the dangers associated with inoculation of virus in vaccinated immune and vaccinated nonimmune pregnant females.

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Susceptible sows in various stages of pregnancy were vaccinated simultaneously with attenuated or virulent (Ames) hog cholera (HG) virus and anti-HC serum. Tissue specimens from offspring of the sows were examined for HC virus by the fluorescent antibody cell culture technique (FACCT). Isolates from the newborn pigs and composite suspensions of tissues that gave negative results by the FACCT were inoculated in susceptible pigs for confirmation. Transplacental infection occurred in 4 sows vaccinated with Ames virus and either 35 or 75 ml. of anti-HC serum. The dose of antiserum and stages of gestation in which the vaccination was performed were factors that were considered to affect the results of the experiment. These 4 sows were in the first month of pregnancy and 3 of the 4 sows had been given the smaller dose (35 ml.) of antiserum. Transplacental infection was not confirmed in 12 sows vaccinated with attenuated virus and 20 ml. of anti-HC serum nor in 8 sows vaccinated with Ames virus and either 35 or 75 ml. on antiserum.

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Mosquitoes trapped during an epizootic of hog cholera (HC) in Maryland in 1969 were prepared into 40 pools which were inoculated in pigs. Hog cholera virus was confirmed in pigs inoculated with 8 of 40 pools of mosquitoes. Generally, the pigs contracting HC developed chronic infections with persistent viremia that lasted 30 or more days. Two pigs seemed healthy when euthanatized 62 and 80 days after inoculation, yet viremia of high titer was detected in each. Experimental studies were performed with 2 laboratory strains of mosquitoes, *Aedes aegypti* and *Culex tarsalis*, to determine if biological and mechanical transmission occur. Biological transmission was not confirmed, but HC virus was retained in A. *aegypti* for 3 days. Mechanical transmission was confirmed with A. *aegypti* in 2 of 9 experiments.

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The Nebraska fluorescent-antibody (FA) technique was used successfully to detect and differentiate virulent, attenuated, and inactivated hog cholera (HC) viruses in the tonsils of suceptible 10-week-old pigs. Fluorescence became diffusely distributed throughout the epithelial and lymphoid tissues in tonsils of pigs infected with virulent HC virus. Positive cells were characterized by a bright homogenous fluorescence in the cell cytoplasm. Following infection in the pigs with attenuated HC vaccines, fluorescence was observed primarily in plaquelike areas in tonsillar crypt epithelium. These areas fluoresced intensely and increased in size and number for 4 to 5 days, then regressed and disappeared. Individual reticuloendothelial cells in the diffuse lymphoid tissue and small plaques in the germinal centers were seen infrequently. Significant differences were not observed in pigs given attenuated virus and immune serum and those inoculated with attenuated virus alone. A transient fluorescence was observed inconsistently in tonsil sections from pigs inoculated with either of 2 inactivated HC vaccines. Fluorescence was granular and limited to tonsillar germinal centers.

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Co-autores: W. D. Dean, M. A. Tidwell, G. P. Combs, D. W. Anderson, W. O. Cowart, y R. C. Axtell.

Epidemiologic evidence indicates that insects, specifically flies (Diptera) may disseminate hog cholera virus (HCV). Two species of horseflies (Tabanidae), *Tabanus lineola* Fabricius,

and *Tabanus quinquevittatus* Wiedemann, experimentally transmitted HCV to susceptible swine within 2 hours after biting a virus-source pig. Three other *Tabanus* species were incriminated. An apparent 24-hour delayed transmission of the virus by horseflies occurred. Transmission attempts using 6 species of mosquitoes were unsuccessful. Laboratory diagnostic tests used for the detection of HCV were the fluorescent-antibody tissue section technique (FATST) for tonsillar tissue and the fluorescent-antibody cell culture technique (FACCT) for splenic tissue. The fluorescent-antibody serum-neutralization test (FASNT) was used for the detection of serum antibody against hog cholera (HC).

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The hematology of normal young pigs has been reported and compared with previously published results. Total leukocyte and erythrocyte counts increased from birth to approximately 7 weeks of age. The ratio of neutrophils increased at the expense of lymphocytes, contrary to previous reports. Pigs experimentally infected with ascarid ova developed a mild anemia that was noticeable 32 days after infection. Circulating eosinophils increased four days postinfection, reached a peak by the sixteenth day, and returned to normal levels within 28 days. Pigs immunized with rabbit-modified hog cholera vaccine used without serums developed a milk leukopenia following both vaccination and challenge. The leukopenia following vaccination lasted for approximately eight days, whereas that following challenge existed for at least 16 days. A leukocytosis developed following the vaccination leukopenia in these pigs. The leukopenia existed for a longer time in ascarid-infected animals, and the count was slower in returning to normal than it was in the control pigs. These observations may be one of the explanations for postvaccination difficulties encountered by veterinarians in the field with some lots of pigs.

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Thirteen pigs nursing gilts which were not vaccinated against hog cholera died five to 15 days after their immunity was challenged with virulent virus, regardless of age at challenge. Fifteen pigs nursing gilts which had been vaccinated at 8 weeks of age with virulent virus and anti-hog cholera serum were immune to challenge with virulent virus at 4, 6, 8, and 10 weeks of age. No temperature elevations occurred and only transitory leukopenia existed. Thirty of 50 pigs (60%) that nursed gilts which had been given a single dose of modified live-virus vaccine survived the challenge dose of virus. Eleven of 22 pigs (50%) that nursed gilts which had been vaccinated with modified live-virus vaccine at 8 weeks of age and then given 2 additional doses during the latter part of pregnancy survived the challenge dose of virus. Pigs which

died in both these groups had severe leukopenia; percentage of decline in leukocytes was greatest in pigs whose immunity was challenged at older ages. These data indicated that pigs nursing gilts which had been vaccinated with virulent virus and anti-hog cholera serum were the best protected. Pigs nursing gilts vaccinated with modified live-virus vaccine were protected at 4 weeks of age but were increasingly susceptible at 6, 8, and 10 weeks of age. Even though pigs were resistant at 4 weeks, leukopenia was severe enough to theoretically render the pigs susceptible to secondary organisms. Booster injections of modified live-virus vaccine to gilts at 90 and 110 days pregnancy did not alter the degree of immunity in nursing pigs. Differences in total circulating leukocyte levels indicated variations in the effect of hog cholera virus on vaccinated and nonvaccinated pigs. Leukocyte levels served as an indication of the degree of resistance present.

* WEIDE, K. D. and KING, N. B. Electrophoretic studies of passive immunity against hog cholera in young pigs. American Journal of Veterinary Research 23(95):744-747. 1962. -1094-

Serum electrophoretic patterns were summarized for 101 pigs nursing dams which had been vaccinated against hog cholera by various methods. Significant variations of patterns among groups were not observed in pigs up to 70 days of age. The inoculation of virulent hog cholera virus into 14 susceptible pigs resulted in a decreased albumin fraction and increased *alpha* globulin levels. *Beta* and *gamma* globulin changes were not observed in the sample periods. The inoculation of virulent hog cholera virus into 87 pigs, totally and partially resistant to hog cholera, resulted in a decreased albumin fraction (-25%) and increased *alpha* globulin levels (+20%). *Gamma* globulin levels were increased at 14 days (or later) after virus inoculation. Older pigs nursing immune dams were more susceptible to challenge with virulent virus than were younger pigs and had greater increases in *gamma* globulin levels. *Beta* globulin levels were not affected by the virulent hog cholera virus.

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Germfree techniques were employed to study the clinical signs and lesions caused by the hog cholera virus. Ten germfree and 11 monocontaminated pigs were inoculated at 14 days of age with hog cholera virus. Six uninoculated pigs (4 germfree

and 2 monocontaminated) were raised under the same conditions and served as controls. Rectal temperatures were diphasic following virus administration, reaching two mean peaks of 106.0 and 106.3 F. Total circulating leukocytes decreased to nearly one third their preinoculation levels within 24 hours. There was a reduction in hemoglobin level and packed cell volume, followed by a rise in numbers of circulating nucleated erythrocytes. Death occurred in all 21 inoculated pigs; the time of death ranged from 4 to 15 days after inoculation. At necropsy, hemorrhagic lymph nodes and petechial and ecchymotic hemorrhages in the kidneys were found consistently. These signs and lesions were attributed to the effects of the virus itself.

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Edema of newborn pigs and a variety of abnormalities have been experimentally produced by injection of pregnant sows or gilts with rabbit-modified hog cholera viruses, with or without immune serum. These injections were made ten to sixteen days after breeding. Abnormalities were produced in 38 per cent of the fetuses of 24 litters from the experimentally injected dams as compared with 3 per cent abnormalities among 16 control litters. Some of the difficulties encountered are illustrated. Serological tests eliminated leptospirosis as a primary or associated agent in the experimentally produced abnormalities. Hog cholera virus originating from rabbit-modified hog cholera virus of porcine origin and virulent enough to cause death in 12-day-old susceptible test pigs was isolated from the spleens of 6 of 10 fetuses from four litters. Virus was not demonstrable in the spleens of the dams.

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INDICE DE AUTORES

INDICE DE AUTORES

EDITORIAL IICA