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Abstract

Germfree piglets rapidly develop pneumonia after Haemophilus pleuropneumoniae is inoculated into the lung, providing a basis of comparison for future studies of pneumonia in SPF and conventionally reared piglets.; Swine Day, Manhattan, KS, November 19, 1987

Keywords

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HAEMOPHILUS (ACTINOBACILLUS) PLEUROPNEUMONIAE INFECTION IN GERMFREE PIGLETS[1]

N. V. Anderson[2] and C. A. King[3]

Summary

Germfree piglets rapidly develop pneumonia after Haemophilus pleuropneumoniae is inoculated into the lung, providing a basis of comparison for future studies of pneumonia in SPF and conventionally reared piglets.

Introduction

Pleuropneumonia of swine, caused by H. pleuropneumoniae, accounts for considerable loss to some swine producers in Kansas. Treatment with antibacterial drugs and control by vaccination have been partly effective. Other infectious agents, such as Pasteurella, Bordetella and Mycoplasma, are often present in these herds and their presence makes it difficult to determine the effect from H. pleuropneumoniae alone. Therefore, we studied experimental pleuropneumonia in germfree piglets, in order to exclude the effects of other infectious agents.

Procedures

This study was conducted at the Research Laboratory, Department of Veterinary Science, University of Nebraska-Lincoln. Germfree piglets were obtained by caesarian section and maintained in germfree isolators for 35 days. Piglets were allotted to one of 2 treatments: a) 10^6 H. pleuropneumoniae bacteria inoculated into trachea (infected, n = 8); and b) uninoculated controls (not infected, n = 5).

All uninoculated controls were necropsied at 0 hr. Four of the infected piglets were necropsied at 1 hr, and 4 at 4 hr. Cells were obtained from blood and lung at necropsy.

Leukocytes (white blood cells) from blood and lung were assayed by differential counts (Tables 1 and 2). Histopathologic study of lung tissue confirmed the presence of leukocytes in vessels and alveoli of lung. Nonspecific esterase, EA rosette, and phagocytic activity of leukocytes were also assayed (data not reported). Data were analyzed by analysis of variance and covariance (SAS).

[1]We gratefully acknowledge the cooperation of Dr. E. D. Erickson and Dr. A. R. Doster, our colleagues at the Department of Veterinary Science, University of Nebraska-Lincoln, Lincoln, NE 68583.

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Results and Discussion

Blood leukocytes promptly entered the site of infection in the lung, reconfirming that germfree piglets are capable of developing a typical inflammatory response on first exposure to bacteria. Pneumonia was similar to that occurring in field cases of H. pleuropneumoniae. This indicates that H. pleuropneumoniae can cause pneumonia in susceptible germfree pigs in the absence of other bacteria, such as Pasteurella multocida, Bordetella bronchiseptica, and Mycoplasma spp. The comparisons of cell numbers in blood and lung (Table 1) are interpreted to mean that the pneumonia rapidly became more severe during the interval from 1 hr to 4 hr after infection. Having documented the response of germfree piglest to H. pleuropneumoniae, we can do further experiments to compare pleuropneumonia in germfree, SPF, and conventionally reared piglets.

Table 1. Blood Leukoctye Counts in Germfree Piglets Infected with H. pleuropneumoniae (cells/mm^3)

Piglets	Total WBC	Neutrophils	Immature	Lymphocytes	Monocytes
Controls (n=5)	5560	1967	78	3447	122
Infected, 1 hr (n=4)	3512*	1352	46	2020	81
Infected, 4 hr (n=4)	3637*	1691	211*	1582	145

*Different ($P<.05$) from other groups.

Table 2. Lung Leukocytes (%) in Germfree Piglets Infected with H. pleuropneumoniae

Piglets	Leukocytes in Lung		
	Neutrophils	Lymphocytes	Macrophages
Controls (n=5)	1%	1%	98%
Infected, 1 hr (n=4)	1%	1%	98%
Infected, 4 hr (n=4)	27%*	1%	72%*

*Different ($P<.05$) from other groups.