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Effect of Ambient Temperatures on Multiplication of Attenuated Transmissible Gastroenteritis Virus in the Bodies of Newborn Piglets

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Newborn piglets were found to be more resistant to infection with attenuated transmissible gastroenteritis virus when maintained at higher temperatures. This was attributed to a decreased rate of virus propagation and spreading in the bodies of the infected animals. The highest virus levels were detected in the tissues of piglets maintained at 8 to 12 C. In contrast, no virus was recovered from piglets maintained at 35 to 37.5 C. The virus was found only in the lymph nodes and respiratory organs in the piglets maintained at 20 to 23 C.

Because the greatest losses from transmissible gastroenteritis (TGE) occur in suckling piglets, considerable effort has been devoted to deriving methods of immunizing young pigs. One method is the immunization of pregnant sows prior to farrowing. The newborn piglets may obtain antibodies from the colostrum and milk of the sows and may thus be passively immunized (2). Another method of protecting young pigs from TGE is active immunization with attenuated virus. This method succeeded in establishing protection in newborn piglets beginning on day 3 after vaccination (5). During studies on the multiplication of attenuated virus in the bodies of newborn piglets, considerable differences in the extent of virus distribution were noted among piglets raised at different temperatures.

The purpose of the present study was to investigate the influence of ambient temperature on the multiplication of attenuated TGE virus in the bodies of newborn piglets.

Seven littermates from a susceptible dam were obtained immediately after birth and divided into three groups, which were kept at 8 to 12, 20 to 23, and 35 to 37.5 C, respectively. The piglets were reared on sterile food (Spf LAC, Borden Inc., Norfolk, Va.) and did not receive colostrum. Rectal temperatures were recorded twice daily until examined at necropsy. Five milliliters (about 10^7 50% tissue culture infective doses) of the TO strain of attenuated TGE virus was given orally to each animal. This attenuated TGE virus strain had been serially passaged 160 times in swine kidney cell cultures and is referred to as the TO-160 strain. The properties of this strain in vivo or in vitro have been described in detail previously (6, 7). The methods used for the collection and processing samples from piglets have also been described (7). Briefly, each tissue sample was homogenized to make a 10% suspension. Virus levels were determined by inoculating tube cultures of primary swine kidney cells with 0.1 ml of serial 10-fold dilutions of the tissue suspensions using eight cultures per dilution. Titers were expressed as a 50% tissue culture infective doses.

Two of the three inoculated piglets held at 8 to 12 C died on days 1 and 2 after inoculation. Rectal temperatures declined below 35 C 10 h before death. In contrast, four piglets maintained at higher temperatures remained in good health on day 3 after inoculation. The surviving piglets were killed, and specimens were taken 3 days after inoculation. Of these the two piglets held at normal temperature (20 to 23 C) had rectal temperatures ranging from 36 to 38.5 C, and the two piglets maintained at the elevated temperature (35 to 37.5 C) had rectal temperatures ranging from 38 to 41 C.

Recovery of virus from the various tissues of the piglets is summarized in Table 1. The frequency of viral recovery and the viral titers of tissues from piglets kept at low temperatures (8 to 12 C) were markedly higher than those kept at higher temperatures. Virus was recovered from the serum, lymph nodes, and tissue of respiratory and digestive tracts of all of the piglets of this group. With the exception of the low titers in spleen, virus was not recovered from parenchymal tissues. In the two piglets kept at 20 to 23 C, the virus was detected in the respiratory and lymphatic tissues of one and only in the lymph nodes of the other. A very low titer of virus was also detected in the esophagus

TABLE 1. *Distribution and concentration of attenuated TGE virus after oral administration in newborn piglets maintained at different temperatures*

Organ	Virus titer (log $TCID_{50}$/g of tissue)[a]						
	8-12 C			20-23 C		35-37.5 C	
	1(1D)[b]	2(2D)	3(3E)	4(3E)	5(3E)	6(3E)	7(3E)
Nasal mucosa	1.0	3.5	0.63	2.5	0	0	0
Trachea	0.75	3.0	1.75	0.75	0	0	0
Lung	1.5	6.25	0	3.25	0	0	0
Tonsil	2.75	3.0	2.0	1.0	0.88	0	0
Mandibular lymph nodes	2.75	4.25	3.0	2.75	2.25	0	0
Pulmonary lymph nodes	1.75	4.5	3.5	1.5	0	0	0
Mesenteric lymph nodes	5.25	4.25	4.0	0	1.75	0	0
Brain	0.75	0	0	0	0	0	0
Liver	1.5	0	0	0	0	0	0
Spleen	2.75	1.13	2.5	0	0	0	0
Pancreas	0	0	0	0	0	0	0
Kidney	0	0	0	0	0	0	0
Thymus	0.75	0	0	0	0	0	0
Urinary bladder	0.63	0	0	0	0	0	0
Esophagus	2.5	1.13	0	0.63	0	0	0
Stomach	1.5	0.75	0	0	0	0	0
Duodenum	1.13	0	0.63	0	0	0	0
Jejunum	4.5	3.5	1.0	0	0	0	0
Ileum	4.5	0	0	0	0	0	0
Colon	0	0	1.13	0	0	0	0
Serum	3.75	0.63	0.75	0	0	0	0

[a] $TCID_{50}$, 50% tissue culture infective dose.

[b] Piglet number. Number in parentheses indicates days after inoculation; letter in parentheses indicates the status of piglets when they were autopsied (D, dead; E, euthanatized).

of one of the two piglets. In contrast to these findings, virus could not be isolated from any of the tissues of the inoculated piglets kept at the highest temperatures (35 to 37.5 C).

Adverse ambient temperatures are known to influence the pathogenesis of several viral diseases (1, 3, 8). A rise in temperature above the level of the normal temperature often results in an increase of resistance to viral infection. The mechanism by which high body temperature exerts its beneficial effects is obscure. The present study indicates that ambient temperature is an important factor in determining whether growth of TGE virus in the newborn piglet will be restricted or become disseminated throughout the body. Viral growth was greatly diminished when animals were kept at high temperatures. Since the optimal temperature for growth of TGE virus in cell culture ranges between 30 to 40 C, it seems unlikely that the body temperatures (37.5 to 41 C) induced by the high ambient temperatures directly inhibit virus growth in the body. The influence of ambient temperature on viral growth might be due to a variety of physiological mechanisms. Perhaps changes in temperature influence the development of immunity and host resistance to infection (9). A precise explanation of the mechanisms involved in the immunological response must await further study.

The majority of herd outbreaks of TGE take place during the colder months, from December to April. The annual peaks of epizootics were described by several investigators as occurring in March, coinciding with the long spring farrowing period (4). Decreased resistance of the newborn piglets to TGE virus at low ambient temperatures should be considered as a possible factor affecting the seasonal pattern of the outbreaks.

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LITERATURE CITED

1. **Bell, J. F., and G. J. Moore.** 1974. Effects of high ambient temperature on various stages of rabies infection in mice. Infect. Immun. 10:510-515.
2. **Bohl, E. H., and L. J. Saif.** 1975. Passive immunity in transmissible gastroenteritis of swine: immunoglobulin characteristics of antibodies in milk after inoculating virus by different routes. Infect. Immun. 11:23-32.
3. **Carmichael, L. E., F. D. Barnes, and D. H. Percy.** 1969. Temperature as a factor in resistance of young puppies. J. Infect. Dis. 120:669-678.
4. **Ferris, D. H.** 1973. Epizootiology of porcine transmissible gastroenteritis. Adv. Vet. Sci. Comp. Med. 17:57-87.

5. **Furuuchi, S., Y. Hashiguchi, and T. Kumagai.** 1974. Immunization of newborn piglets with an attenuated strain of transmissible gastroenteritis virus. Proc. Jpn. Soc. Vet. Sci. 77:43.
6. **Furuuchi, S., Y. Shimizu, and T. Kumagai.** 1975. Properties of an attenuated strain of transmissible gastroenteritis virus. Proc. Jpn. Soc. Vet. Sci. 9:47.
7. **Harada, K., S. Furuuchi, T. Kumagai, and J. Sasahara.** 1969. Pathogenicity, immunogenicity and distribution of transmissible gastroenteritis virus in pigs. Natl. Inst. Anim. Health Q. 9:185–192.
8. **Lycke, E., S. Hermodsson, K. Kristensson, and B. E. Ross.** 1971. The herpes simplex virus encephalitis in mice at different environmental temperatures. Acta Pathol. Microbiol. Scand. Sect. B 79:502–510.
9. **Subba Rao, D. S. V., and B. Glick.** 1970. Immunosuppressive action of heat in chickens. Proc. Soc. Exp. Biol. Med. 133:445–448.