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# PERSISTENT INFECTIONS OF HOG CHOLERA: A REVIEW

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ABSTRACT

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Persistent infections are the most important mechanisms by which hog cholera (HC) virus is perpetuated in the domestic pig population. As result of mainly transplacental transmission of HC virus and to a lesser extent postnatal contact, persistent infections may develop with or without chronic illness, retardation in growth or runting. In the early weeks of life persistently viraemic piglets do not show any differences in appearance when compared with their non-viraemic littermates but they transmit HC virus to their uninfected littermates. Piglets with persistent viraemia living up 153 days post partum and longer have been reported. There is a good chance that persistently infected weaners go to the market or directly into fattening herds. Stamping out of such fattening herds, which is where this type of infection usually occurs, reduces the number of infectious foci but does not necessarily achieve eradication of the virus in a region or country as long as the so-called chronic-type of infection exists in breeding herds. In order to break the cycle, development of persistent infections must be prevented by appropriate measures. Persistent infections acquired in utero or postnatally do reflect an impaired ontogeny of the immune response specific for HC virus and therefore can not be influenced a posteriori. This creates an epidemiological factor which can not be ignored in discussions on proper control of hog cholera.

Persistent infections are known for many viruses. They have been described in three main types characterizing the interaction between virus and host (Fenner et al., 1974):

1. Latent virus infections
2. Chronic virus infections
3. Slow virus infection

It is the chronic type of infection (2) which, according to Matumoto (1969), by persistent mechanism of virus perpetuation in hosts is reponsible for the infection of new hosts continuously or intermittently for a long period of time. This applies to many viruses and particularly to non-arbo togaviruses with members of the genus pestivirus: hog cholera (HC) virus and bovine viral diarrhea (BVD) virus (species); in addition border disease (BD) virus has been

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listed by Fenner (1976) although it might be identical with BVD virus.

Chronic hog cholera has been defined as a lethal form with a duration of 30 or more days with the pigs having a persistent viraemia and an impaired antibody response to this virus (Mengeling and Cheville, 1968). Persistent infections, however, have been observed in only a few instances after experimental infections of weaner pigs resulting in death 6 to 8 weeks later (Liess et al., 1976). They cannot be totally ignored although they are certainly of minor importance compared with the variety of effects which might result from infection with HC virus acquired in utero (Sautter et al., 1953; Huck and Aston, 1964; Cowart and Morehouse, 1967). This is probably directly related to the ontogeny of the immune response mainly due to transplacental transmission of HC virus and its effects on fetal development initially described by Young et al. (1955).

From a theoretical point of view as well as for practical reasons prenatal infections of the fetus following transplacental transmission of HC virus gained more interest during the recent years. Sequential studies on sows infected at various stages of pregnancy tried to elucidate the relationship between persistent viraemia and the immunological response of the fetus (Figure 1). According to Meyer (1978) the number of piglets born alive with persistent viraemia was highest in groups of sows inoculated between 70 and 90 days of gestation with a low virulent strain of HC virus.

The occurrence in the field of HC virus strains with low virulence and their relation to the "pregnant carrier sow syndrome" has been intensively described by Aynaud et al. (1977) and Carbrey et al. (1977). Although the term virulence has not been sufficiently defined, yet, it seems reasonable to require that the strain of HC virus concerned must have the property of infecting pregnant sows without production of gross lesions (subclinical infection) as well as transplacental transmissibility. The HC virus strain 'GLENTORF' fulfilled these requirements (Liess et al., 1976; Frey et al., 1980) and so did the strain 'BERGEN' used by Van Oirschot and Terpstra (1977). It is still an open question whether HC virus strain '331' reported by Mengeling and Cheville (1968) to induce chronic hog cholera in pigs between 2 and 8 months of age, is transmitted transplacentally to the fetuses or not. More recent studie showed that this strain of HC virus seems to be unique in its failure of horizontal transmission from intranasally inoculated to in-contact weaner pigs (Liess and Prager, unpublished). So it appears that this strain is not only of low virulence but capable of infecting pigs without showing excretion of the virus. This is similar to African Swine Fever virus infection in warthogs. Thus strain 331 seems to possess a higher degree of modification than both the other strains mentioned before. The existence of such strains in nature must

be strongly considered especially in view of their role in establishing persistent infections.

Congenital infections acquired in utero mainly by transplacental transmission of HC virus can result in lifelong or at least long-lasting persistent infections. For practical reasons it is important to know how long pigs born with persistent viraemia live and whether they transmit HC virus to their littermates not infected prenatally or any other pigs on the same premises or after introduction into other herds (Liess, 1981).

Meyer (1978) found that viraemic piglets (in the early weeks of life) do not show any differences in appearance when compared with their non-viraemic littermates but that they transmit the virus to the uninfected littermates. The fact that fetuses present in the same uterus can be selectively infected or are born uninfected is one of the peculiarities of the HC virus infection. As soon as they leave the uterus and are in direct conctact with each other, the uninfected piglets receive the virus from their virus excreting littermates and react in the normal way: no signs of illness but development of neutralizing antibody against HC virus.

Meyer et al. (1981) recognized three typical groups of piglets belonging to infected litters with the first group consisting of stillborn or aborted fetuses with typical lesions and/or demonstration of HC virus.

The second group consisted of piglets with congenital viraemia lasting from birth to death. These persistent infections occurred exclusively when the sows were inoculated between 70 and 90 days of gestation. Of a total of 82 piglets farrowed by sows in which transplacental transmission was demonstrated, 30 (36%) were born viraemic of which 21 (25%) died within two weeks postpartum and the remaining 9 piglets (11%) within three to eight weeks. In spite of high titer viraemia and virus excretion up to two weeks, there were no signs of illness. It was not possible to identify such animals among their non-viraemic littermates. Only later, a chronic form of the disease developed together with runting and animals died within three to eight weeks af age.

The third group of piglets comprised those not infected at birth since they neither showed detectable viraemia nor measurable amounts of neutralizing antibody prior to ingestion of colostrum. They remained healthy and after decline of maternally derived antibody to HC virus the rise of neutralizing antibody commenced at about six weeks of age as result of contact with their viraemic and virus excreting littermates.

Similar results have been reported by Van Oirschot (1979) with observation of piglets with persistent viraemia living up to 153 days postpartum. The longest survival periods of piglets in the group of sows inoculated on the sixty-fifth day of gestation comes close to the results reported by Meyer (1978).

In conclusion it is obvious that in countries with enzootic hog cholera there is a good chance for persistently infected and possibly HC virus excreting weaners to go to the markets or directly into fattening herds where they can cause the short-cycle type of infection characterized by an acute fatal disease with high mortality. Therefore eradication of HC virus in a region or country will not be successful as long as control measures are only based on stamping out of such fattening herds rather than to detect the so-called chronic-type of infection in breeding herds where virus persistence guarantees perpetuation of HC virus. This creates an epidemiological situation which must not be ignored and, yet, finds so little respect in formulating control programs.

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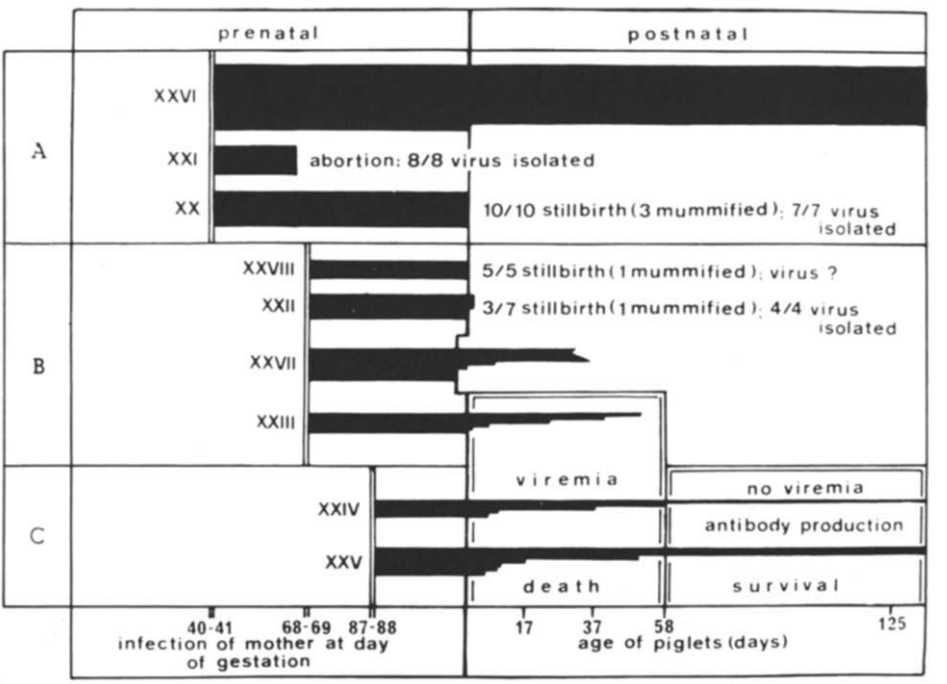


Fig. 1. Pre- and postnatal fate of piglets in litters of sows exposed to HC virus strain 'GLENTORF' by intranasal inoculation at various stages of gestation (groups A - C).