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Principal Features of the Epidemiology of Aujeszky's Disease – *Suid herpesvirus 1* Infection – in Swine and Cattle.

Genital/venereal infection has been common in small swine herds in some European countries, and there is evidence that infection of cattle resulting in pruritus on the hindquarters was regularly sexually transmitted from swine by man.

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Abstract

Aujeszky's disease – infection with *Suid herpesvirus 1* (SuHV1) – is an infection of pigs, which under certain conditions can be spread to several other animal species, usually with a fatal outcome. It has been found world-wide, but has now been eradicated in a number of countries. Denmark is the country, where Aujeszky's disease has been studied most intensively, and the results from investigations of cattle have been of greatest importance for the understanding of the epidemiology of the infection in swine. Denmark was the first country to initiate eradication and was also the first to complete eradication of the indigenous infection. In this review, important features are

recapitulated at the end of each section or subsection.

In cattle, the infection will most often give rise to an intense pruritus of a skin area. In iatrogenic cases the site of virus introduction will determine the site of pruritus, but in natural cases pruritus will appear (1) in the head region or on the chest, which is associated with respiratory infection, or (2) on the hindquarters of females, which indicates vaginal infection. In cases of respiratory infection, virus can be found in the mucous membranes of the nasal or pharyngeal cavity of animals with head pruritus, while in cases with chest pruritus virus can be found in lung tissue. In cases with pruritus on the hindquarters, virus can most often be found in the vagina. Pruritus must be considered a phantom sensation due to stimulation or damage of the central nervous system including sensory ganglia. Practically speaking, respiratory infection of cattle is a dead end infection, as spread among cattle has never been demonstrated. Relatively high virus titres found in mucous membranes in a few cases, however, may be taken to indicate that transmission can not be totally excluded.

The source of respiratory infection in cattle has always been pigs infected by the respiratory route. Cattle at risk need not necessarily be those placed near the infected pigs, as virus in an animal house has been seen transmitted by air currents determined by ventilators over distances of 10-20 meters, in some cases even from pigs in a neighbouring room.

In swine, the infection was considered to be exclusively respiratory for decades, and naturally occurring genital infection was not demonstrated until 1981. In herds with outbreaks in cattle characterized by pruritus on the hindquarters (vaginal infection), respiratory infection of pigs was never observed, but a sow had characteristically been served by a foreign boar from 5-14 days earlier. In some such cases investigated, genital infection was confirmed in sows, which clearly linked cases in cattle with a posterior localization of pruritus to a concurrent genital infection of swine. Naturally occurring genital infection has been found also in wild pigs in the USA.

Respiratory infection of pigs was demonstrated late in the history of the disease. The first outbreak in Denmark was in 1964, and in the preceding 33 years there had been only 3 outbreaks in cattle showing an anterior localisation of pruritus indicative of respiratory infection, but close to 60 outbreaks, where the cattle showed a posterior localization of pruritus indicative of genital infection.

The development of the disease situation in pigs in Denmark has clearly illustrated that SuHV1 has the ability to change in degree of pathogenicity over time. The changes occurred in two steps. In the early 1960's respiratory strains developed, which were spread rapidly between herds due to animal contacts, mainly by trade, and later in the 1970's strains developed, which had an even higher degree of pathogenicity to both cattle and swine. These new strains were found to be syncytial in contrast to earlier isolates from traditional outbreaks. And that the new respiratory strains and syncytial strains had not been introduced from abroad was confirmed with absolute certainty by restriction fragment pattern analyses of virus DNA.

That syncytial virus strains were more virulent than non-syncytial strains was later substantiated by results from examination of isolates from England and Northern Ireland.

As concluded from the studies of respiratory SuHV1 infection in cattle, the decisive spread of a respiratory infection among swine in a ventilated animal house will be by air currents over many

meters determined by the ventilation system and not by close animal-to-animal contacts. This feature also illustrates that during an acute outbreak in a swine herd considerable amounts of virus will be blown out into the surroundings by ventilators, exposing neighbouring herds at a risk of infection. Early observations in Denmark allowed the conclusion that airborne spread between swine herds might occur over several kilometres, but after eradication of the indigenous infection, new infections were introduced from abroad, which demonstrated that airborne spread of virus between herds had taken place over even 10 to 30 kilometres. Syncytial strains of SuHV1 have shown to possess a pronounced tendency to airborne transmission among swine herds, which implies that if a syncytial strain is first introduced into an area, it is likely to be the prevailing type in that area within a few years.

The special manifestation of Aujeszky's disease in cattle showing pruritus on the hindquarters has regularly been associated with use of a boar from a boar centre for natural service of a sow shortly before appearance of the clinical disease. In most cases examined, virus was found in the vagina of the affected bovine animals, although in low titres, and in three outbreaks – the only cases investigated early enough to be successful - virus was demonstrated in the vagina of a sow. It was found that animal sodomy seemed to play a role in the transmission of the genital infection from swine to cattle, and this conclusion is further substantiated by comprehensive supplementary information given in this review paper. From the fact that genital infection in cattle is closely correlated with contemporary genital infection in swine on the same premises and from the many reports over the years on infection in cattle showing pruritus on the hindquarters, it can additionally be concluded that the SuHV1 infection in a great part of the 20th century was maintained as a porcine genital infection in many European countries.

1. Introduction

Suid herpesvirus 1 (SuHV1) infection or Aujeszky's disease was first identified as a new specific infectious disease in Hungary by *Aujeszky* (1902). The disease was found in a steer, a dog and a cat from different premises, and all showed pruritus in the nose and mouth area. In the following years the infection was reported from many different countries, especially in cattle.

In Denmark, the infection was first seen in 1931 in cattle on two neighbouring farms (*Bang* 1932). Five cows, one heifer and two calves were affected. Five had pruritus on their heads, two on one side of the chest, while in one calf no itching was noticed. The source of infection for cattle was not identified. In 1932, *Burggraaf and Lourens* reported on a great number of affected cattle on 5 farms in Holland situated within a few kilometres from each other. They observed that the disease in cattle had been preceded by symptoms in swine. Pruritus was seen in the head or chest regions of the cattle. In 1935, *Köves and Hirt* reported natural, wide-spread infection in swine in Hungary, but already 1913 the infection had been demonstrated in that country in a domestic sow suspected of rabies and in a wild pig forwarded for examination because of unspecific disease in the wild swine population in a certain wood district. Here, a similar disease problem had been observed the previous year. No wild pigs were then submitted for laboratory examination, but Aujeszky's disease was diagnosed in a dog, a badger and a fox from the district (*Ratz* 1914). *Shope* (1935a, b) found the infection to be widespread among swine in some areas in the USA and concluded that pigs were the source of infection for cattle. He had observed that the affected cattle had been kept in close contact with pigs and suggested that infection took place, when nasal secretions from infected pigs were rubbed into abraded skin of the cattle. This notion of cutaneous or percutaneous infection of cattle was concordant with the general experience from experimental infection of cattle, rabbits, etc. that pruritus would appear at the

site, where virus had been injected.

Denmark is the country, where Aujeszky's disease has been surveyed and studied most intensively, not only right from its first appearance in 1931 until the completed eradication of the indigenous infection by the end of 1985, but also in the following years, when new infections were introduced from abroad. The results here-from concerning the infection in cattle and swine were comprehensive and of extreme importance, not only for the understanding of the epidemiology of this particular infection, but also for the evaluation of the possibilities of its control and for the elaboration of adequate eradication schemes.[1] This article dealing with the principal epidemiological features of Aujeszky's disease will therefore naturally focus on results from Danish observations and investigations, but will include all relevant results from other countries as well.

Latency of the SuHV1 infection in swine is well documented, and its significance with regard to maintenance of the infection in herds and transmission into other herds is well understood. It will consequently not be dealt with here as a separate epidemiological characteristic.

2. Infection in cattle

2.1. The background

In 1965, *Bendixen et al.* reported on 64 outbreaks recorded in cattle since its first appearance in 1931. Until 1964 they all occurred on the island of Zealand. (*Figure 1*), and a considerable number of affected animals could therefore be brought to the veterinary clinic at the Royal Veterinary College in Copenhagen. This institute had been contacted in each case either directly by the veterinary practitioner or the National Veterinary Service, and detailed information on the outbreaks was carefully journalized.

Figure 1. Outbreaks of Aujeszky's disease in cattle in Denmark 1931-1964. Up to 1964 all outbreaks occurred on the island of Zealand (Bendixen et al. 1965).

It was striking that in 61 of the 64 outbreaks affected animals showed pruritus on the hindquarters. Many outbreaks in cattle have been reported without information about the exact location of pruritus, but all cases observed by *Burggraaf and Lourens* (1932) in 5 herds in Holland (cf. above) were associated with an anterior localization of pruritus, i.e. on head or chest. From questionnaires, however, returned from veterinary practitioners *Jansen* (1957) concluded that most Dutch cases observed had shown posterior pruritus, as did also animals from 3 outbreaks examined by *Jansen and Kunst* (1957). *Rosenberger* (1940) reported on nine outbreaks occurring 1925 to 1940 in the Hanover area in Germany,

1 Epidemiological characteristics such as (1) airborne and (2) genital infection of cattle, (3) airborne spread with air currents among swine and from swine to cattle within herds, (4) airborne spread between swine herds, (5) correlation between virulence of virus strains and their ability to form syncyntia in cell cultures, (6) higher risk of airborne herd-to-herd spread for syncytial virus strains, (7) naturally occurring genital infection of pigs, and (8) long-distance airborne spread between swine herds over 10-30 kilometres were original Danish findings, all by this author except for the last-mentioned long-distance airborne spread. This particular feature was a conclusion from investigation of outbreaks introduced from herds south of the Danish-German border after 1985, i.e. after that the indigenous infection had been eradicated, and after that this author had left the National Veterinary Research Institute.

where most animals showed posterior pruritus. Two to four animals were affected in each outbreak, and in the individual herds the animals showed disease at practically the same time. *Lamont* (1947) referred to outbreaks in cattle in Northern Ireland, in a few of which the animals showed pruritus on head or chest, but in most of them pruritus was seen in the anal/vulvar region. In Sweden, Aujeszky's disease was not seen until 1965 (*Estola* **et** *al.* 1965). In the first outbreak some litters of piglets died, and several cows and heifers became infected showing head pruritus. Also in that country posterior pruritus was seen in later outbreaks in cattle (*Rockborn and Hugoson* 1972).

For many years the significance of the localization of pruritus with respect to route of infection was not understood, and percutaneus infection was thought to be most likely (cf. *Lamont* 1947). Actually *Kretschmar*, in his monograph on Aujeszky's disease from 1970, expressed the opinion that this mode of infection in cattle could definitely be taken for granted and suggested that biting insects like *Stomoxys calcitrans* were responsible.

2.2. The two-year study 1971-1973 and relevant results from later outbreaks

From the point of view that the port of entry for a virus like SuHV1 was likely to be mucous membranes, this author decided in 1971 to study more intensively future outbreaks in cattle.

Three possible sites of primary infection were considered, namely the respiratory tract, the alimentary tract, and the genital tract. Unfortunately, only in a few cases did veterinary practitioners contact the State Veterinary Serum Laboratory (the National Veterinary Research Institute) before submitting material for laboratory examination, so only seldom material of interest in addition to the head or the thoracic or lumbar parts of the vertebral column necessary for the diagnosis could be examined. Results from the two years from December 1971 through November 1973 comprising 29 outbreaks were published 1975 (*Bitsch* 1975a, 1975b) together with results from a few selected outbreaks from 1974 and 1975 (*Bitsch* 1975c).

2.2.1. Outbreaks with an anterior localization of pruritus

It was of extreme significance that the outbreaks could be divided into two separate groups on basis of the localization of pruritus: (1) the outbreaks where affected animals showed an anterior localization of pruritus , and (2) outbreaks where animals showed a posterior localization of pruritus. In none of the herds were both manifestations seen.

In 12 outbreaks animals showed anterior pruritus. Of the 31 animals affected, 19 showed pruritus of the head and 7 on one side of the chest, while 5 showed no signs of pruritus (*Table 1*). Four animals without pruritus were examined virologically, and three had virus in the medulla thoracalis and not at all in brain material, and one animal had virus in brain material and samples from mucous membranes of the head, but not in the medulla thoracalis.

Table 1. Outbreaks in cattle from December 1971 through November 1973.

	No. of outbreaks	No. of animals affected	
		total	average
Anterior pruritus	12	31*	2.6
Posterior pruritus	17	23	1.4
Total	29	54	1.9

*Pruritus in head area: 19; on chest: 7; no pruritus: 5.

Mucous membranes of the heads of 11 animals with head pruritus and one without pruritus but with brain involvement were examined for presence of virus, and and samples from eight animals were found positive: a strong indication of infection by the respiratory route (*Bitsch* 1975a). The virus titres were usually low, demonstrating that cells of these membranes of this species do not readily support uptake and growth of virus. In one of the cases, however, as well as in one later case (*Bitsch* 1975c), the titres in mucous membranes and in swabs here-from were remarkably high (approx. $10^{2.5}$ $TCID_{50}$ per 0.1 ml of the tissue or swab material suspensions).

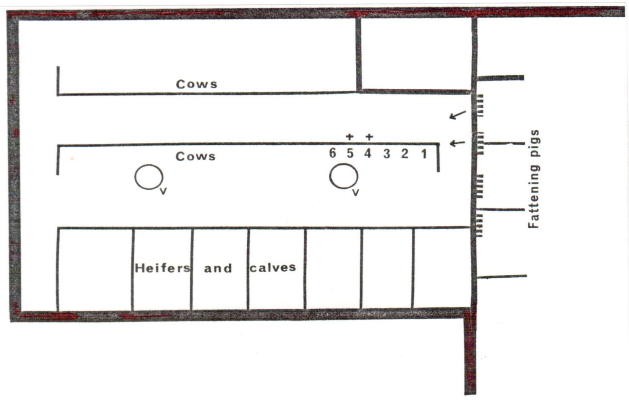

Figure 2. Plan of a cowhouse, where two cows received a respiratory SuHV1 infection from pigs in a neighbouring room. Arrows indicate air currents through two windows with broken panes created by a ventilator (V). Virus was isolated from lungs of one of the cows showing chest pruritus (Bitsch 1984a).

In none of the cases from the two-year study with chest pruritus and/or medulla thoracalis involvement were lung material forwarded for examination. In an outbreak shortly after, however (*Bitsch* 1975c), a cow showing beginning symptoms of disease was transported to the veterinary clinic at the Royal Veterinary College (together with the cow with head pruritus mentioned above with relatively high titres of virus in mucous membranes). This cow showed a slight itching on the right side of its chest. It was euthanized after two days of disease, and when it was skinned, the right side of the chest was found remarkably hyperaemic. Several samples collected aseptically from the lungs by use of cauterization were found virus-positive (*Bitsch* 1975c). Later, virus was found present in lung material from a further six cases in cattle and one case in a goat, all showing chest pruritus (*Bitsch* 1984a). So altogether virus has been demonstrated in the lungs of eight ruminants showing chest pruritus.

The results from the virological examinations provided strong evidence of the facts (1) that outbreaks with anterior localization of pruritus are due to respiratory infection and (2) that the sites of virus entry and of pruritus are not identical but related to a common part of the central nervous system (*Bitsch* 1975a). In fact, many years earlier Hurst (1933) observed that after intradermal, subcutaneous or intramuscular injection of virus (mainly into rabbits), histological degenerative lesions could be found in the corresponding part of the central nervous system and ganglia, and that symptoms of pruritus started shortly after the appearance of these changes. On these grounds he suggested that pruritus was not caused by a stimulus of nerve endings but by an involvement of parts of the central nervous system.

The circumstances observed in the herds, where cattle showed an anterior localization of pruritus, were in full accordance with this conclusion of a respiratory infection mode (*Bitsch* 1975b). Practically always were the cattle housed together with swine, most often feeder pigs, although not in close contact with them. *Three different situations* could be observed: (1) if the room was equipped with a ventilator expelling the air, the affected animals were typically placed between the ventilator and the pigs; (2) if there was only a simple air duct in the ceiling without a fan, affected animals were often placed close to the air duct with pigs just opposite, as if the air had circulated around the duct before leaving through it (like water let out of a sink or a bathtub); and (3) if there were no ventilation installations in the room, cases in cattle might appear almost everywhere. A special situation was seen in two later outbreaks, where the source of infection obviously was feeder pigs in an adjacent room: in one case there was a permanent aperture in the separating wall, while in the

other one (*Figure 2*) the wall was supplied with windows, where some panes were broken (*Bitsch* 1984a). The affected animals, five and two, respectively, were placed just between these wall openings and ventilators, which actually were found to expel air from the rooms with swine through the apertures described.

On average, 2.6 animals were infected in each outbreak. In one of the herds, however, where the shared cattle and swine house was poorly ventilated, an outbreak had occurred one year earlier affecting 14 head of cattle. In none of these outbreaks did the animals show a posterior localisation of pruritus, and in no case was transmission of the infection among cattle found likely to have occurred. But the finding of relatively high virus titres in mucous membranes or swabs here-from in the few cases mentioned above may be taken to indicate that cattle-to-cattle transmission may occasionally take place.

The duration of clinical disease is shorter in animals with a head involvement than in animals with a lung infection. Typically the first group died after approximately 24 hours, while the second group showed symptoms for a couple of days (*Bitsch* 1975a).

Sufficient evidence has been accumulated for the following conclusions concerning outbreaks of Aujeszky's disease in cattle with an anterior localization of pruritus:

(1) Cases regularly result from respiratory infection;
(2) Cases with head pruritus are associated with primary infection of mucous membranes of the nasal or pharyngeal cavities;
(3) Chest pruritus is associated with a primary lung infection;
(4) Cases of primary infection of both upper and lower respiratory tract may proceed without signs of pruritus;
(5) Animals infected by the upper respiratory tract typically die after one day of disease, while animals suffering lung infection show symptoms for approximately 2 days;
(6) Individual cases in cattle regularly receive the infection directly from swine;
(7) Respiratory Aujeszky's disease is practically speaking a dead end infection in cattle;
(8) Animals at risk are those exposed to air currents from pigs infected by the respiratory route;
(9) The site of pruritus, iatrogenic cases disregarded, is not the site of virus entry, but those two sites are related to a common part of the central nervous system, most probably also involving sensory ganglia. Pruritus in Aujeszky's disease therefore appears to be a neurological condition due to damage or stimulation of the central nervous system, and the intense itching of a skin area must be considered a phantom sensation.

2.2.2. Outbreaks with a posterior localization of pruritus

In 61 of the 64 outbreaks occurring 1931-1964 described by *Bendixen et al.* (1965) all 114 infected animals showed a posterior localization of pruritus. In 60 per cent of these outbreaks only one animal was infected, the average number being 1.7. In two herds there were outbreaks with a one-year interval, and in a third herd an outbreak reappeared after 5 years. For 16 affected calves under one year of age, their sex was not recorded, but the remaining 98 animals were all females. The cattle and swine herds were relatively small. In 39 cases the veterinary practitioners had informed about the size of the swine herd. All had sows, and in 19 cases was additionally informed about regular use of boars from boar centres for natural service. From the size of the swine herds, however, the authors judged that such boars had regularly been used in a large majority of the herds.

In the material from the two-year study only 17 of the 29 outbreaks were associated with posterior pruritus (*Table 1*). Again, most often only one animal was infected in each outbreak, the average number being as low as 1.4. If more than one animal was infected, they usually showed symptoms at the same day, in a few cases up to two days apart (*Bitsch* 1975a). One outbreak deviated from the rest by the finding that the infection had

obviously been transmitted by the veterinary practitioner, just as it was also found in a few later cases (*Bitsch* 1975c). This outbreak was on a farm with both a big cattle and a big swine herd. The remaining 16 outbreaks all occurred on farms with small swine herds. And in none of these herds were symptoms of the disease in swine observed. Of special interest was that the affected animal on one farm was a bull calf. Samples were collected from rectum and several sections of the intestines of that calf, but no virus could be found (see later).

Only in three cases from two herds were vaginas received for examination, but virus was found in all three of them, although at low titres. Veterinary practitioners were therefore encouraged to contact the laboratory before forwarding material for examination, and over the following years vaginal samples from a further 22 animals from 21 outbreaks were found virus-positive, while vaginal samples from 14 animals from 13 outbreaks were found virus-negative (*Bitsch* 1984a). Generally, however, the titres demonstrated in vaginal samples were very low, indicating that a negative result did not exclude the possibility of a vaginal infection. These additional results strongly indicate that in the majority of outbreaks in cattle with a posterior localization of pruritus, animals are infected by the vaginal route. It was characteristic in these cases that there were relatively few sows in the herds and that boars from boar centres had been used for natural service of the sows. (More details are given below in the section on genital infection in swine).

Animals showing pruritus on the hindquarters typically die after 2 days of disease just as the animals suffering from lung infection *(Bitsch* 1975a).

In one case from the two-year study (*Bitsch* 1975a, 1975b) and three later cases (*Bitsch* 1975c) the findings strongly indicated *iatrogenic infection.* Two of these animals showed pruritus on their hindquarters, while the remaining two showed pruritus in the flank area, in one of them involving a surgical wound.

The possibility of *alimentary infection* was left open after the early studies 1971-1973, especially because the case in a bull calf mentioned above (most probably the only natural case ever in a male bovine animal with posterior pruritus) could not easily be explained otherwise,[2] but also because the evidence of vaginal infection in the majority of other cases at that time was still not overwhelming. But if alimentary infection of bovine animals resulting in posterior pruritus was a real possibility, such cases should definitely also be found in combined cattle and swine herds, where pigs were infected by the respiratory route. And such cases do not seem ever to have been observed. It will therefore be justifiable to conclude that *the possibility* of *posterior localization of pruritus resulting from alimentary infection is totally unrealistic.*

The conclusions concerning cases in cattle with a posterior localization of pruritus are therefore:

(1) Cases have regularly been associated with vaginal infection. The vagina must accordingly be considered to be the port of virus entry;
(2) Clinical disease typically lasts two days;
(3) Outbreaks have regularly occurred in combined swine and cattle herds, where boars from boar centres have been used for natural service;
(4) Generally one or two animals are infected in each outbreak, in the majority of cases only a single animal;
(5) When several animals are infected, they have usually become infected at practically the same time;
(6) A sow has regularly been served by a centre boar a short time, most often 5-8 days, before symptoms;
(7) Some extreme cases have been iatrogenic;
(8) Percutaneus infection is unlikely except in iatrogenic cases;
(9) Cases have never been reported to occur in association with respiratory infection of swine;

2 According to the farmer's wife this particular calf was their special pet animal. It was not tied up, and followed persons around in the cowhouse just like a dog.

(10) Alimentary infection is not a realistic possibility.

3. Infection in swine

3.1. Pathogenicity of virus strains

3.1.1. Syncytial and non-syncytial virus strains

SuHV1 exhibits a virus strain-specific cytopathogenic effect (CPE) in susceptible tissue cultures of mainly two types: where the infected cells (1) form syncytia (syncytial virus strains) or (2) become rounded or ballooned (non-syncytial virus strains), cf. *Kaplan* 1969.

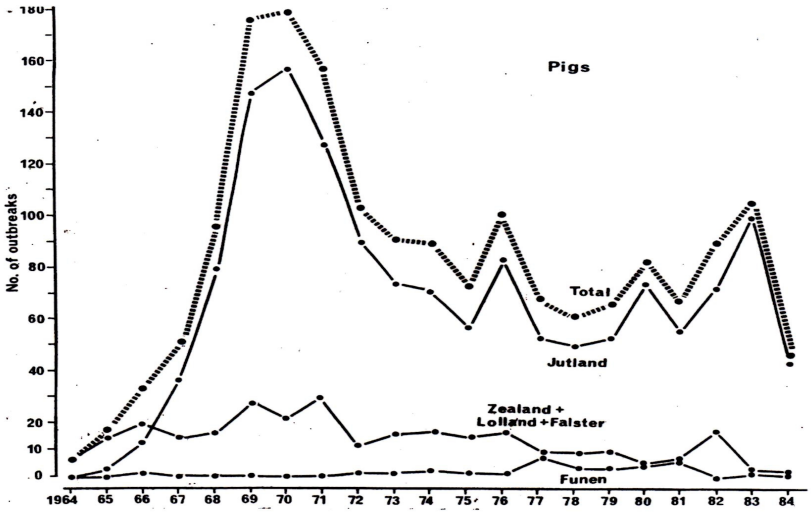

Figure 3. Outbreaks of Aujeszky's disease in swine herds in Denmark 1964-1984. The spread in the 1960's followed the development of respiratory virus and the increased number of outbreaks 1982-1983 in Jutland was caused by the spread of syncytial virus strains.

All cases in cattle in Denmark from 1931 up to 1964 occurred on the island of Zealand (*Figure 1*) (*Bendixen et al. 1965)*. But in 1964 three outbreaks were diagnosed in Jutland. The structure of the swine production was changing. From 1950 to 1964 the annual number of pigs slaughtered gradually rose by 300 per cent (*Bendixen et al.* 1965), and this tendency continued over the following years. Conventional breeding herds and herds with specialized production of fattening pigs became steadily larger.

In Denmark, as in many other countries in Europe, Aujeszky's disease was not seen as a disease problem in pigs until many years after its clinical appearance in cattle. The first Danish outbreak in swine was seen 1964 on Zealand (*Andersen et al.* 1964), and over the next years the infection was spread rapidly, especially in Jutland (*Figure 3*) (*Bitsch and Andersen* 1982, *Bitsch* 1983). But clinical symptoms other than fever were seen only in piglets, and generally only litters under two weeks of age were clinically affected. On average, two litters died in each outbreak.

In the 1970's the situation was found to have changed further, when more severe outbreaks appeared in especially big herds. Also litters over 2 weeks of age died, as might also older pigs, and sows were seen to abort infected fetuses.

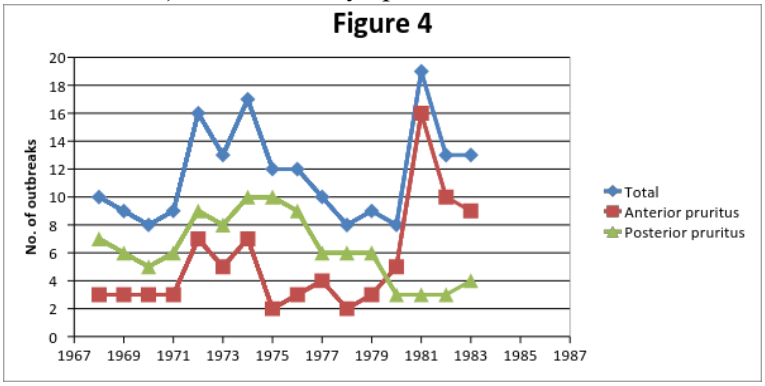

Figure 4. Outbreaks in cattle in Denmark 1968-1983. Note the increased number of outbreaks with anterior pruritus (respiratory infection) associated with the appearance and spread of syncytial virus strains after 1979.

This new situation with more severe respiratory disease in pigs was reflected also by a higher frequency of outbreaks in cattle with an anterior localization of pruritus caused by respiratory infection (*Figure 4*).

At the same time it was observed that the degree of pathogenicity of the virus isolates could be related to their cytopathic effect (CPE) in tissue cultures. Not only were virus isolates from cattle with pruritus on the hindquarters consistently non-syncytial, while most isolates from cases with anterior pruritus were syncytial

(*Table 2*), but isolates from severe outbreaks in swine herds were also - in contrast to isolates from most other outbreaks in swine - found to be syncytial (*Bitsch* 1980[3]). In the following years, syncytial strains became predominating (*Figure 5*), especially in Jutland (*Bitsch and Andersen* 1982, *Bitsch* 1983, 1984a). This correlation between virulence and type of CPE was not especially Danish. *Harkness and Sands* (1985) found results accordingly with British isolates, and a series of coded virus isolates from Northern Ireland received from *J.B. McFerran* examined by this author gave similar results[4] . Also all the highly virulent virus strains introduced by the airborne mode into Denmark from herds south of the Danish-German border after 1985 were found to be syncytial (see later).

Table 2. Prevalence of syncytial and non-syncytial virus in outbreaks of Aujeszky's disease in cattle up to 1980 (Bitsch 1980).

	No. of outbreaks*	Type of virus strain involved	
		syncytial	non-syncytial
Anterior pruritus	17	14	3**
Posterior pruritus	25	0	25***
Total	42	14	28

*Isolates from all 29 outbreaks in 1978 and 1979 were supplemented with 13 preserved isolates from outbreaks in the period 1972-1977.

**Only one animal was infected in each of these three outbreaks.

***All isolates from the many later outbreaks of this type were also non-syncytial.

3.1.2. Restriction fragment pattern analysis of virus DNA

It was evident from the results in 1978 and 1979 that Aujeszky's disease had become a more severe disease over the last few years. This was a main reason for the decision in 1980 to control and eradicate the infection. An important question was the origin or the cause of the new more virulent strains. In this context it should be noted that import of swine was prohibited for many years up to 1972, when Denmark joined the European Common Market, most probably in order to protect the Danish landrace from foreign genes, and after 1972 all animals imported were subjected to control of Aujeszky's disease, both before import but also after a quarantine period as required by the farmers' organizations. So infected pigs had not been imported for decades.

Gielkens and Berns and *Ludwig et al.* reported 1982 on differentiation of SuHV1 virus isolates by use of restriction fragment pattern analysis of the virus DNA. *A.L.J. Gielkens* agreed to test some Danish isolates and received four isolates: a syncytial respiratory and a non-syncytial genital strain from cattle, and a syncytial and a non-syncytial respiratory strain from swine, all from outbreaks in 1982. He responded immediately (May 1982) with the information that the four isolates appeared identical but differed from all other European strains, he had tested so far. One characteristic difference was that fragment/band 9 from the BamHI analysis

3 Of the isolates from swine evaluated, four were judged to be of an intermediary type. For practical reasons they were later included among the non-syncytial strains.

4 The CPE produced by one of the isolates, which turned out to be the NIA 4 virus of obscure origin and similar - if not identical - to the Hungarian modified Bartha vaccine strain, differed completely from what had been seen with all other SuHV1 virus strains.

of the Danish isolates was missing. These results were published in a later report (*Gielkens et al.* 1985).

H. Ludwig received a syncytial respiratory strain from swine and a non-syncytial genital strain isolated in 1981 from cattle and found them to be identical to a Danish cattle isolate from1962 (posterior pruritus) and a Swedish pig isolate from 1982, but again different from all other SuHV1 strains investigated (*Herrmann et al.* 1984). They found that European isolates could be divided into three major groups, which were designated I, II and III. The group III comprised exclusively the Danish-Swedish strains. *Todd and McFerran* (1985) tested two syncytial and two non-syncytial isolates (one of which was the first genital isolate directly from pigs) from 1980 and 1981 with identical results. *Christensen and Sørensen* (1988) tested approx. 70 Danish isolates, which all - apart from six isolates from herds infected within one month in the winter of 1985 in an area not far from the German border (*Figure 6,* cf. section on airborne transmission) - were found to be Group III strains.

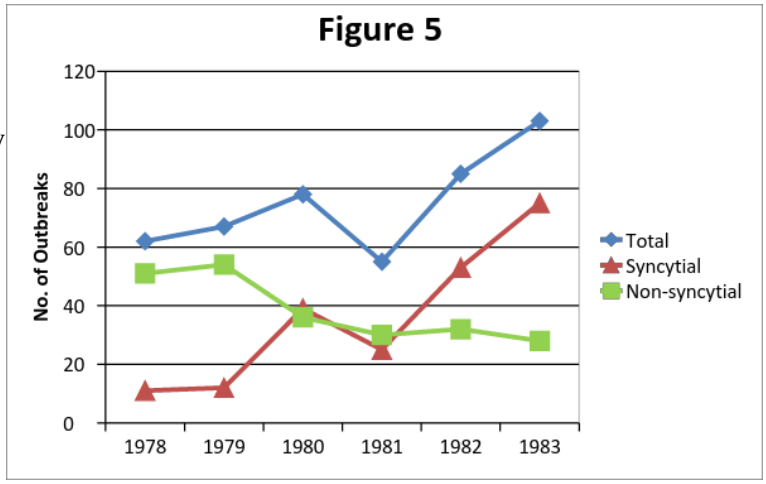

Figure 5. Outbreaks of Aujeszky's disease in pigs 1978-1983 caused by syncytial and non-syncytial virus (Bitsch 1984a). In 1985 there were 38 outbreaks, of which 32 (84%) were caused by syncytial strains (see Figure 6).

The important conclusion made already 1982 after the response from *A.L.J. Gielkens* after his preliminary analyses that the new virulent Danish strains had not been introduced from abroad but had developed from Danish strains of lower pathogenicity (or virulence) was in this way substantiated by the results from many later investigations.

In summary, the main conclusions concerning the degree of pathogenicity (virulence) of SuHV1 strains are:

(1) Syncytial virus strains possess a higher degree of pathogenicity to both cattle and swine than non-syncytial strains ;

(3) Isolates from cattle exhibiting posterior pruritus (iatrogenic cases not considered) are regularly non-syncytial;

(4) SuHV1 appears to possess the ability to vary in degree of pathogenicity. Over time virus may become more virulent along with changes in the pig breeding and production structure;

(5) Danish virus strains are unique and constitute a separate group of SuHV1 viruses. No foreign strains were introduced into Denmark before 1985.

(6) Changes in virulence occurred in two major steps. The first step was the establishment about 1964 of respiratory virus strains, which were spread vigorously over the following years causing outbreaks in pig herds and death of piglets a few weeks old, and the second step was associated with the development and spread in the 1970's of the more virulent syncytial, respiratory strains.

(7) The ability of a SuHV1 isolate to cause syncytium formation in cell cultures is a valuable marker of virulence.

3.2. Airborne transmission within and between herds

The results from the study of Aujeszky's disease in cattle referred above demonstrated clearly not only that cases in cattle exhibiting pruritus on the head or chest were due to respiratory infection, but also that the

individual cases received the infection directly from swine in practically all instances. At the same time it was illustrated that virus from respiratorily infected pigs was carried away by air currents over relatively long distances, not even to animals in the same room, but also in some instances to animals in adjacent rooms (*Bitsch* 1975a, *Bitsch and Andersen* 1982, *Bitsch* 1984a). Transmission of an infectious agent by air currents does not seem to have been demonstrated that clearly before. Up till then, spread of a respiratory infection like Aujeszky's disease in pigs was considered to require close or relatively close contact between individuals. However, the findings with Aujeszky's disease in cattle, which was found relatively resistant to infection by the respiratory route, implied that the predominating or decisive spread also among swine in ventilated rooms would be the transmission by air currents over many meters rather than transmission among animals in close contact.

Donaldson et al. (1983) demonstrated under controlled experimental conditions that virus could be sampled from the air in loose boxes with infected animals, and that infection also could be transmitted by air from infected pigs in one box through an air duct to uninfected pigs in another box.

The Danish observation that air containing considerable amounts of virus was moved by ventilators and blown out into open air furthermore implied that the infection might also be spread by the airborne mode to swine farms in the surroundings. The risk of infection for other herds would naturally depend on (1) the effect and construction of the ventilation system and (2) the size of the infected herd as well as that of those at risk, (3) of weather conditions, (4) length of time of exposure for herds at risk, (5) characteristics of the virus strain and (6) distance from the source of infection. After the appearance of syncytial virus strains (*Bitsch* 1980) a growing number of especially closed big herds became infected. Some outbreaks concluded to have been caused by airborne transmission of virus were described by *Bitsch and Andersen* (1982), and others later (*Bitsch* 1984a). Transmission had been noted to occur over distances of up to 3 kilometres. In one of the early cases, where an SPF herd was concluded to have received the infection from a very large herd with sows and fattening pigs 500 meters away, the air blown out by ventilators of the latter could actually under certain weather conditions be observed and followed over considerable distances as relatively thin "wisps of smoke". Most of the herds found likely to have been the source of airborne infection for other herds were with feeder pigs (*Bitsch 1984a*).

Figure 6. Outbreaks in swine herds 1985.
Symbols:
Encircled dots: 6 outbreaks caused by non-syncytial virus.
Triangles: 32 outbreaks caused by syncytial virus. The 6 outbreaks in the south of Jutland were by a foreign syncytial virus strain.

In 1984 *Gloster et al.* reported on the possibility of airborne herd-to-herd spread of Aujeszky's disease between a number of herds in Yorkshire and concluded that meteorological and epidemiological data suggested that airborne transmission might have occurred in some of the cases.

In 1987 *Christensen et al.* reported on restriction pattern analyses of virus DNA from all isolates of SuHV1 from 1985 *(cf. Figure 6*). The analyses were extended in a subsequent paper (*Christensen and Sørensen* 1988),

which is referred to above in the section on pathogenicity of SuHV1 strains. The main finding was that all Danish isolates - apart from those from six outbreaks in swine herds in the south eastern part of Jutland , which occurred early in 1985 within a period of one month - belonged to the group III of the SuHV1 virus. Virus of this group has been recovered only from outbreaks in Denmark and Sweden, cf. above. The six deviating isolates were group II viruses and were from herds situated within 10 kilometres from each other. It was concluded that the primary source of infection for these six herds were herds south of the Danish-German border[5].

As seen in *Figure 6,* the outbreaks in 1985 caused by syncytial virus appeared in clusters, which was due to airborne spread of the infection among the herds.

In two later papers (*Christensen et al.* 1990, *Christensen et al.* 1993) was reported on introductions of Aujeszky's disease virus identified as various subtypes of group II viruses into Denmark in the winters of 1986/1987, 1987/88, 1988/89 and 1989/90. On basis of comparison with isolates from Northern Germany, and of meteorological data and other special conditions it was concluded that the infection had been transmitted by the airborne mode from German herds to some Danish herds in the south of Jutland, from where it was further spread similarly to other herds. In the winters of 1987/1988 and 1989/90 it was even spread more than 30 kilometres over waters to the island of Funen[6].

The most important conclusions related to airborne transmission of SuHV1 are hereafter:

(1) Within a herd the decisive transmission of the respiratory virus will follow air currents over many meters, in ventilated rooms determined by the ventilation system. Spread from close animal-to-animal contacts will generally be of minor importance;

(2) Risk of herd-to-herd transmission will be high in areas with big swine herds with effective ventilation systems. An infection may be spread over distances of many kilometres;

(3) Syncytial virus strains are more likely to be spread by the airborne mode than are non-syncytial

5 All isolations of SuHV1 from outbreaks in 1985 were made by this author, and these isolates together with a great number of earlier isolates were forwarded to the Danish Veterinary Virus Research Institute by the end of 1985 for further investigations. Unfortunately, in the two papers giving the results (*Christensen et al.* 1987 and *Christensen and Sørensen* 1988) it is erroneously mentioned that one of the six isolates differing from the traditional Danish virus strains originated from a herd in the mid part of Jutland. But all six deviating isolates were from herds situated in a small area in the south of Jutland, cf. *Figure 6*.

6 Another respiratory disease of domestic animals, infectious bovine rhinotracheitis, IBR, caused by *Bovine herpesvirus 1* was eradicated in Denmark from 1984 to 1991. During the eradication period, several acutely infected herds were concluded to have received the infection by the airborne mode from a neighbouring acutely infected herd. And already in the winters of 1990 and 1991, some new infected herds close to the German border were concluded to have been infected from herds south of the border. But also in the years 1992 to 1995 and in 2001 after the final eradication, a number of herds in the border county were found infected in the winter period. Airborne transmission from German herds was concluded to be the only possible explanation. Most of the infected herds were situated within a few kilometres from the border, but two were 10 and 25 kilometres away. In some instances neighbouring herds got infected subsequently. In one particular case, a dairy herd 300 meters from an infected herd showed a low reaction in bulk tank milk shortly after the outbreak, but only one animal had got infected in this herd. Of special interest was that this particular cow was placed in the cowhouse just between a ceiling ventilator and air-intake in the wall facing the infected farm. The risk of airborne herd-to-herd transmission of IBR is considerable, although not of quite the same magnitude as the risk of herd-to-herd spread of syncytial strains of SuHV1. (All investigations on IBR were performed by this author).

strains.

3.3. Genital infection of pigs

Aujeszky's disease in cattle showing pruritus on head or chest is due to respiratory infection, and after appearance of the highly pathogenic, syncytial virus strains, such outbreaks become predominating (cf. Figure *4).* But up to 1964 the large majority of the outbreaks in cattle was associated with pruritus of the hindquarters. In later such cases, virus was demonstrated, although in low titres, in the vagina of most of the animals examined, and the obvious conclusion was that the vagina was the port of virus entry (cf. *Section 2* above) . But still, up to 1980, the way of transmission from infected swine remained to be demonstrated. The fact, however, that cases regularly appeared in small herds using centre boars for natural service (at least in Denmark and Sweden, where this particular characteristic had been observed) made it logical to anticipate that these boars had introduced the infection to sows in a genital form.

Akkermans (1963) (see also *Akkermans* 1964) tested 40 boars aged from 6 months to several years from 13 different boar centres in the Netherlands. He concluded that they originated from uninfected herds and found that practically all had become antibody-positive by the age of one year. Furthermore, in one experiment he infected a boar nasally with a respiratory strain isolated from a dead pig and found that the infection was transmitted to the vagina of two sows served 6 and 16 days later, although preputial swabs consistently were virus-negative. In another experiment a sow was infected by the vagina using the same virus strain. Virus was demonstrated in vaginal swabs for 8 days, and after four days she was mated with a boar, after which virus could be found in preputial swabs from this boar for nine days. But because no genital strain was used in the experiments, no particular conclusions can be made from these results. And naturally occurring genital infection has never been demonstrated in the Netherlands.

In Sweden, *Rockborn and Hugoson* (1972) noticed in 1967 in a herd with a case of Aujeszky's disease in a cow that a centre boar had visited the herd two weeks earlier. Of 9 boars from the centre, 5 were found antibody-positive. Sows from herds, where boars from that centre had been used, were then blood-tested at slaughterhouses during 1968. Of 424 sows from 149 herds, 123 (29%) from 64 herds (45%) were found antibody-positive. No clinical signs of the infection had been seen in pigs in these herds.

The close correlation between outbreaks in cattle and use of centre boars was reported already by *Bendixen et al.* (1965). The first 60 outbreaks up to 1964 all occurred on the island of Zealand (F*igure 1*). In early1984 one third of boars from boar centres on that island were found serologically positive, and when small herds in the area were tested, a usual finding was single reactors among the sows.

Demonstration of porcine genital infection was attempted in 5 outbreaks (*Bitsch* 1984b, Outbreaks 1 to 5), the first time (Outbreak 1) in 1981. A sow had been served by a centre boar (Day 0) before appearance of disease symptoms in two calves (Day 5). Virus was demonstrated in the vagina of both calves and also in a vaginal swab collected on Day 7 from the sow at a titre of $10^{6.2}$ $TCID_{50}$ per ml of the swab fluid (further details of this outbreak are given in *Section 4*).

In an outbreak from 1983, where virus was demonstrated in the vagina of a cow (*Bitsch* 1984b, Outbreak 4), virus was found in a vaginal swab collected from a sow served by a centre boar close to one week before death of the cow. The virus titre was $10^{5.5}$ $TCID_{50}$ per ml swab fluid. It is interesting that the affected cow was reported to be in oestrus on the day of insemination of the sow (cf. suspicion of sodomy, *Section 4*).

In an outbreak from 1982 (*Bitsch* 1984b, Outbreak 3) a sow (Sow 1) had been served by a centre boar on November 15 (Day 0), and a cow showed clinical disease on Day 15. There were only 2 sows on the premises, and vaginal swabs were taken from these and the cow on Day 17. The samples from Sow 1 and the cow were negative, while the sample from the second sow showed a virus titre of $10^{-3.5}$ $TCID_{50}$ per ml of the swab fluid. Unfortunately, only the lumbar spinal cord and not additionally the vagina of that cow was forwarded to the laboratory for examination. The boar mentioned was the only contact with swine from other herds for several

months, and a logical conclusion was that virus had been transmitted from the vagina of Sow 1 to the other sow and the cow at practically the same time approximately one week after the mating of Sow 1 (for more details, see *Section 4*).

In a further two outbreaks (*Bitsch 1984b, Outbreaks 2 and 5*) vaginal swabs from sows were examined virologically, but with a negative result. In Outbreak 2 from 1982, two heifers showed clinical disease on February 23, and virus was found in the vagina of the one examined. Two sows had been served by centre boars 7 and 11 days earlier, but vaginal swabs taken from the sows five days after the clinical outbreak, i.e. 16 and 12 days after service, respectively, were found negative. In Outbreak 5, where a calf showed signs of Aujeszky's disease on September 19, 1983, virus was found in the vagina of the calf. Vaginal swabs collected from two gilts, which had been served by a centre boar nine days before disease in the calf, were found negative, but the sampling was not performed until 12 days after service. Blood samples, however, taken of both of them at the same time showed low antibody titres. Apart from the gilts, there were only some feeder pigs in this herd. So with respect to these two outbreaks, it must be concluded, that although vaginal infection was most likely introduced as a genital infection approximately one week prior to the disease in cattle, the samples from the sows had not been collected early enough, as the development of antibody will hamper virus recovery after more than one week.

The main conclusion from the investigation of the five outbreaks reported in 1984, where centre boars were suspected to have introduced a genital infection into swine herds, was that the genital infection could be confirmed in sows in three outbreaks, while in the remaining two, samples were collected too late for confirmation of the expected vaginal infection of the sows. The results closely linked cases of Aujeszky's disease in cattle with pruritus on the hindquarters to genital infection in swine on the same premises. At the same time they convincingly documented two special epidemiological features of Aujeszky's disease in pigs, namely (1) natural occurrence of a true porcine genital infection and (2) spread of this manifestation of infection between swine herds by centre boars used for natural service.

*Romero et al. (*1997) subjected naturally infected wild pigs in the USA to immunosuppression using dexamethasone and isolated SuHV1 primarily from the vagina and prepuce and less frequently from the respiratory tract of these animals. When 6 naturally infected boars were placed together with 5 uninfected and non-pregnant gilts and examined over eight weeks, virus could be demonstrated in vaginal samples, but not in nasal swabs from any of the animals. In later experiments (*Romero et al.*, 2001), when infected non-pregnant sows were kept together with uninfected boars for six weeks, virus could be demonstrated in preputial swabs and not in nasal swabs from the boars. Likewise, when infected boars were kept together with uninfected gilts, virus was demonstrated in vaginal swabs and not in nasal swabs taken from the gilts. Their conclusion was that the SuHV1 infection in wild pigs was primarily maintained as a venereal infection.

According to various web-sources, wild pigs in the USA were originally escaped domesticated swine, but about a century ago wild pigs were imported into the USA from Europe, and the wild pigs to-day are considered to be a mixed breed population. It is noteworthy that the SuHV1 infection appears to occur in wild pigs in USA in the form that was prevailing in domestic swine in Europe before the development and intensive spread of respiratory virus strains.

The main conclusions related to occurrence of porcine genital infection with SuHV1 are hereafter:

(1) Genital infection is a specific epidemiological entity of the infection;

(2) It has been maintained in domestic swine in several countries for long periods by centre boars used for natural service of sows from different herds;

(3) It has always been asymptomatic in infected pigs;

(4) Indications of respiratory spread of an infection introduced in a venereal mode have never been observed;

(5) In Denmark the many genital isolates from swine and cattle have consistently been found non-syncytial, and there is no reason to suspect that this should not account also for genital strains from other countries;

(6) Natural genital infection has been demonstrated also in wild pigs.

4. Selected outbreaks in cattle with a posterior localization of pruritus: supplementary details pointing to involvement of animal sodomy in transmission of the infection

Sodomy is a delicate subject, so when investigating outbreaks, it will be unacceptable to suggest a such action to have taken place, also to the local veterinary practitioner. Relevant information therefore had to be collected indirectly and with much care. Naturally, the most valuable sources of information were the farmer or his wife and their practising veterinarian.

The first outbreak, where sodomy was found likely to have been involved, also was the first case ever, where virus was demonstrated in the vagina of an affected bovine animal (*Bitsch* 1975b, Outbreak 14). The farmer had brought a sow to a boar centre for service about 5 days before the disease appeared in the calf. From the veterinary practitioner I learned (1) that the owner was a bachelor, and (2) that he - early in the afternoon, shortly after having returned home from the centre – had brought the affected calf in from the field and placed it in the house next to the sow, where it remained until it got sick. When talking to the farmer I suggested that he might have got his hands contaminated by pulling the tail of the sow, and that he thereafter by dragging the calf by its tail might have transferred virus to the perineal region. But the farmer did not respond at all hereto.

A second outbreak was the very first case of proven naturally occurring genital infection in pigs (*Bitsch* 1984b, Outbreak 1). Fortunately, the practitioner phoned to me early about two suspected cases of Aujeszky's disease in calves showing posterior pruritus. Without hesitation I then told him, (1) that the two calves would be females, (2) that they would be placed next to each other and that the neighbouring calves would be males, and (3) that a sow would have been served by a centre boar approximately one week earlier. He immediately confirmed the two first postulates. In fact, 11 calves were stalled in a row, where No. 2 and No. 3 - the affected ones - and No.10 were females, while the rest were males. He promised to forward the vaginas of the calves and a vaginal swab from the sow for laboratory examination. Virus was found in both vaginas, and a high virus titre was demonstrated in the vaginal swab from the sow, cf. *Section 3.3*. According to the farmer's wife, sows to be served were usually pointed out to the one bringing the boar by herself or her husband, but in this particular case they had both been away and had been substituted by their son. According to the practising veterinarian their son was “15 years old or so”. And what was known for sure was that when the boar had left, their son was quite alone at home.

In a third case (*Bitsch* 1984b, Outbreak 3) I was contacted by the practitioner, who reported that a cow in a herd had shown symptoms of Aujeszky's disease with pruritus in the tail region. I explained to him that a sow in the herd would have been served by a centre boar about a week earlier, and asked him to collect a vaginal swab from this sow for examination. However, at the farm he phoned back and told me that the sow (Sow 1) had been served two weeks ago. There were only two sows on the premises, so he agreed to take a vaginal swab also from the second sow (Sow 2). The vaginal swab from Sow 1 was found negative, as expected, because it was collected too late after service. But the vaginal swab from Sow 2, which had not been served recently, showed a relatively high virus titre (see *Section 3.3*). It appears unquestionable that the infection originated from the boar, but virus had not been transmitted to the cow shortly after the mating. Actually, the findings were consistent with a transmission of the infection from the vagina of Sow 1 to the vagina of Sow 2 and the affected cow approximately one week later. In this respect it is of interest that Sows 1 and 2 were

housed in a separate building, while the infected cow and other bovine animals were in the cowhouse on the other side of the farmyard. The centre boar had been the only contact with swine from other herds for several months.

In a later outbreak we came as close to a confession of animal sodomy as one can get without using that particular word. Virus was found in the vagina of a heifer, which was placed immediately next to the sow served a week earlier by a centre boar. When I phoned to the farmer some days after delivery of the laboratory results, I got into contact with his wife. I explained to her about the situation: that virus would have been introduced by the boar, and that the heifer would have received a vaginal infection. Furthermore I suggested that if a person later that day had brushed the hindquarters of the sow and then in immediate succession with the same brush also the tail region of the heifer, I would realize a risk of transmission of the infection to the heifer. I then heard her speak to someone in the room, saying: "Peter (name changed), it is the laboratory veterinarian. He tells me, that if one has brushed the hindquarters of the the sow and thereafter the hindquarters of the heifer, one may have transmitted the infection from the sow to the heifer. I saw you were out there in the evening. The lights were on". I could not hear the other person speak, but after a while she returned and said shortly: "I was speaking to our assistant. He tells me that he has done so". It was striking that she did not hesitate at all. She knew immediately which day the sow had been served, and that the lights had been on in the animal house in the evening on that particular day. Most probably, she and her husband had already talked about what might have taken place. And that the assistant that evening should have brushed the hindquarters of those two animals is definitely unlikely.

Finally, one particular outbreak deserves to be mentioned. A cow and her calf, both showing posterior pruritus, died close to one week after delivery of the calf. We demonstrated virus in the vagina of the cow, but the calf was not examined. When I later visited the farm, I inspected the farm buildings together with the owner and his young male assistant. In the barn there was in one corner a pen with a sow, which had been served by a centre boar some days before appearance of the disease, and in another one a calving box, where the cow had been with her calf. And there were no other animals in that room.

As mentioned earlier, one or two animals were usually infected in each outbreak with posterior pruritus, which fits with the notion of involvement of sodomy. But in extreme cases several more animals have been infected. *Shope* (1931) mentions an outbreak, where 9 animals showed pruritus on the hindquarters. *Bendixen et al.* (1965) informed of an outbreak with 7 cases and in a later Danish outbreak even 10 cows got infected. But from the details of this last outbreak given in the footnote below, it will be seen that also here sodomy may very well have been involved.[7]

Concluding remarks on sexual transmission of SuHV1 from swine to cattle by man:

Although animals in the first outbreaks in cattle in European countries (Aujeszky 1902, Bang 1932, Burggraaf and Lourens 1932) showed pruritus on head or chest (respiratory infection,

7 Unfortunately, the laboratory was not contacted during that acute outbreak, and just lumbar CNS material of one cow was received from an incineration plant several days after the outbreak. The animals got sick at the end of one week. Later, we received detailed information from the veterinary practitioner about the precise time of onset of disease and time of death of the individual animals as well as their location in the animal house, where 34 dairy cows and calves and pigs were housed. According to the farmer's wife, a sow had been served by a centre boar on the preceding Saturday. When I later phoned to the farmer to have finally confirmed that no pigs had been loose in the house that particular weekend, I talked with his daughter, who explained to me that to their knowledge no pigs had been loose. But they still needed to ask her brother, because that weekend he had been home on leave from military service. And particularly that Saturday evening, he had been the only one at home.

cf. above), the manifestation associated with pruritus on hindquarters was predominating in cattle in Europe for many years. But the port of virus entry and the way of transmission of the infection from swine to cattle remained obscure. One step forward in the understanding of these questions was the finding that vaginal infection could readily be demonstrated in most of the bovine cases, and a second important step was that such cases - which already early had been found to be linked with use of centre boars for natural service of sows in the herd - were associated with genital infection of sows in the same herd. The evidence of the fact that the genital infection had been sexually transmitted to the cattle by man must be considered substantial, but from the findings and circumstances described here, it appears justified to conclude that it is overwhelming. Considering the known and widely accepted diversity of the human – and not least male - sexual behaviour, this conclusion, although controversial, can not be surprising. And no other explanation seems possible. It is thought-provoking that this explanation of the cause of this manifestation of the infection in cattle also leads to another conclusion, namely that animal sodomy was not uncommon in rural areas in the 20th century.

References

Akkermans, J.P.W.M., 1963:
Ziekte van Aujeszky bij het varken in Nederland (Aujeszsky's disease in swine in the Netherlands). Dissertation. Centraal diergeneeskundig Instituut, Rotterdam 1963.

Akkermans, J.P.W.M., 1964:
Ziekte van Aujeszky bij het varken in Nederland (Aujeszky's disease in swine the Netherlands).
Tijdschr. Diergeneesk., 1964, 89, 146-159.

Andersen, H.K., Basse, A., Møller, T., 1964:
Enzootisk optrædende morbus Aujesky hos pattegrise (Aujeszky's disease in pigs in Denmark). Medlbl. danske Dyrlægeforen. 1964, 47, 968-978.

Aujeszky, A., 1902:
Über eine neue Infektionskrankheit bei Haustieren (a new infectious disease in domestic animals). Zentralbl. Bakt. Parasitenk. Infektionskr. Hyg., I. Abt. Orig., 1902, 32, 353-357.

Bang, O., 1932:
Pseudowut (acute infectiöse Bulbärparalyse) beim Rind in Dänemark (Aujeszky's disease in cattle in Denmark).
Acta path. microbiol. scand., Suppl. 11, 1932, 180-182.

Bendixen, H.C., Bendixen, H.J., Christensen, N.O., 1965:
Morbus Aujeszkyi's forekomst og optræden hos kvæg i Danmark (Aujeszky's disease in cattle in Denmark). Nord. Vet.-Med. 1965, 17, 249-279.

Ben-Porat, T., Deatly, A.M., Easterday, B.C., Galloway, D., Kaplan, A.S., McGregor, S., 1984:
Latency in pseudorabies virus.
In: Wittmann, G., Gaskell, R.M., Rziha, H-J. (eds), *Latent herpesvirus infections in veterinary medicine*. Curr. Top. Vet. Med. Anim. Sci. 1984, 27, 365-385. Martinus Nijhof Publ., The Hague, Boston 1984.

Bitsch, V., 1975a:
A study of outbreaks of Aujeszky's disease in cattle. I. Virological and epidemiological findings.
Acta vet. scand. 1975, 16, 420-433.

Bitsch, V., 1975b:
A study of outbreaks of Aujeszky's disease in cattle. II. Further investigations on the routes of infection.
Acta vet. scand. 1975, 16, 434-448.

Bitsch, V., 1975c:

A study of outbreaks of Aujeszky's disease in cattle. III. Further outbreaks of special interest regarding epidemiology.
Acta vet. scand. 1975, 16, 449-455.

Bitsch, V., 1980:
Correlation between the pathogenicity of field strains of Aujeszky's disease virus and their ability to cause cell fusion – syncytia formation – in cell cultures.
Acta vet. scand. 1980, 21, 708-710.

Bitsch, V., Andersen, J.B., 1982:
On the epidemiology of Aujeszky's disease in Denmark and the possibilities of its control.
In: Wittmann, G., Hall, S.A. (eds.), *Aujeszky's disease*. Current topics in veterinary medicine and animal science, Vol. 17, 227-236. Martinus Nijhof Publ., The Hague, Boston, London 1982.

Bitsch, V., 1983:
Aujeszky's sygdom.
Proc. Almänt veterinärmöte, Stokholm 1983, 262-271.

Bitsch, V., 1984a:
The main epidemiological features of Aujeszky's disease.
Proc. 11th Conference of the OIE Commission for Europe, Vienna 1984, 381-391.

Bitsch, V., 1984b:
Naturally occurring genital infection with Aujeszky's disease virus in pigs.
Proc. 8th International Pig Veterinary Society Congress, Gent 1984, 23.

Burggraaf, A., Lourens, L.F.D.E., 1932:
Infectieuse bulbair-paralyse (ziekte van Aujeszky) (Aujeszky's disease).
Tijdschr. Diergeneesk. 1932, 59, 981-1002.

Christensen, L.S., Sørensen, K.J., Lei J.C., 1987:
Restriction fraction pattern (RFP) analysis of genomes from Danish isolates of Suid herpesvirus 1 (Aujeszkyi's disease virus).
Arch. Virol. 1987, 97, 215-224.

Christensen, L.S., Sørensen, K.J., 1988:
Introduction of foreign strains of Suid herpesvirus 1 (Aujeszky's disease virus) documented by restriction fragment pattern (RFP) analysis.
Arch. Virol. 1988, 100, 109-113.

Christensen, L.S., Mousing J., Sørensen, K.J., Strandbygaard, S.B., Henriksen, C.A., Andersen, J.B., 1990:
Evidence of long distance airborne transmission of Aujeszky's disease (pseudorabies) virus.
Vet. Rec. 1990, 127, 471-474.

Christensen, L.S., Mortensen, S., Bøtner, A., Strandbygaard, S.B., Rønsholt, L., Henriksen, C.A., Andersen, J.B., 1993:
Further e*vidence of long distance airborne transmission of Aujeszky's disease (pseudorabies) virus.*
Vet. Rec. 1993, 132, 317-321.

Donaldson, A.I., Wardley, R.C., Martin, S., Ferris, N.P.. 1983:
Experimental Aujeszky's disease in pigs: excretion, survival and transmission of the virus.
Vet. Rec. 1983, 113, 490-494.

Estola, T., Obel, A.-L., Rutqvist, L., 1965:
Ett utbrott av Aujeszkys sjukdom i Sverige (an outbreak of Aujeszky's disease in Sweden).
Nord. Vet.-Med. 1965, 17, 649-656.

Gielkens, A.L.J., Berns, A.J.M., 1982:
Differentiation of Aujeszky's disease virus strains by restriction endonuclease analysis of the viral DNA.

In: Wittmann, G., Hall, S.A. (eds.). *Aujeszky's disease*. Current topics in veterinary medicine and animal science, 17, p. 1-13. Martinus Nijhof Publ., The Hague, Boston, London 1982.

Gielkens, A.L.J., Oirshott, J.T. van, Berns, A.J.M., 1985:
Genome differences among field isolates and vaccine strains of pseudorabies virus.
J. Gen. Virol. 1985, 66, 69-82.

Gloster, J., Donaldsen, A.I., Hough, M.N., 1984:
Analysis of a series of outbreaks of Aujeszky's disease in Yorkshire in 1981-1982: the possibility of airborne disease spread.
Vet. Rec. 1984, 114, 234- 239.

Herrmann, S-C., Heppner, B., Ludwig, H., 1984:
Pseudorabies virus from clinical outbreaks and latent infections grouped into four major genome types.
In: Wittmann, G., Gaskell, R.M., Rziha, H-J. (eds), *Latent herpesvirus infections in veterinary medicine*.
Curr. Top. Vet. Med. Anim. Sci. 1984, 27, 387-401. Martinus Nijhof Publ., The Hague, Boston 1984.

Harkness, J.W., Sands, J.J., 1985:
Quantitative evaluation of syncytium formation in cell culture by British isolates of Aujeszkyi's disease virus.
Br. vet. J. 1985, 141, 151-159.

Hurst, E.W., 1933:
Studies on pseudorabies (infectious bulbar paralysis, mad itch). I. Histology of the disease, with a note on the symptomatology.
J. exp. Med. 1933,58, 415-433.

Jansen, J., 1957:
De ziekte van Aujeszky in Nederland (Aujezky's disease in The Netherlands).
Tijdschr. Diergeneesk. 1957, 82, 631-634.

Jansen, J., Kunst, H., 1957:
On the localization of the virus of Aujeszky in the central nervous system.
Tijdschr. Diergeneesk. 1957, 82, 674-679.

Kaplan, A.S., 1969:
Herpes simplex and pseudorabies viruses
Virol. Monogr. 5, 1969. Springer Verlag, Wien, New York.

Kretschmar, C., 1970:
Die Aujeszkysche Krankheit (Aujeszky's disease).
VEB. Gustav Fischer Verlag, Jena 1970.

Köves, J., Hirt, G., 1935:
Über die Aujszkysche Krankheit der Schweine (On Aujeszky's disease in swine).
Arch. f. Tierheilk. 1935, 68, 1-23.

Lamont, H.G., 1946/1947:
Observations on Aujeszky's disease in Northern Ireland.
Vet. Rec. 1946, 58, 621-625 and 1947, 59, 1-3.

Ludwig, H., Herrmann, S-C., Heppner, B., 1982:
The genomes of different field isolates of Aujeszky's disease virus.
In: Wittmann, G., Hall, S.A. (eds.). *Aujeszky's disease*. Current topics in veterinary medicine and animal science, 17, p. 15-20. Martinus Nijhof Publ., The Hague, Boston, London 1982.

Ratz, S. von, 1914:
Die Empfänglichkeit der Tiere für Paralysis bulbaris infectiosa (susceptibility of various animal species to Aujeszky's disease).

Zeitschr. Infektionskrh. parasit. Krh. Hyg. Haust. 1914, 15, 99-106.

Rockborn,G., Hugoson, G., 1972:
On the occurrence of pseudorabies in Sweden. I. Incidence of latent infections in sows and boars..
Zentralbl. Vet.-Med., Rh. B, 1972, 633-640.

Romeo, C.H., Meade, P., Santagata, J., Gillis, K., Lollis, G., Hahn, E.C., Gibbs, E.P.J., 1997:
Genital infection and transmission of pseudorabies virus in feral swine in Florida, USA.
Vet. Microbiol. 1997, 55, 131-139.

Romero, C.H., Meade, P.N., Schultz, J.E., Chung, H.Y., Gibbs, E.P., Hahn, E.C., Lollis, G., 2001:
Venereal transmission of pseudorabies viruses indigenous to feral swine.
Journ. Wildlife Dis. 2001, 37, 289-296.

Rosenberger, G., 1940:
Beobachtungen über die Aujeszkysche Krankheit in Deutschland (observations on Aujeszky's disease in Germany).
Deutsche Tierärztl. Wschr. 1940, 48, 485-488.

Shope, R.E., 1931:
An experimental study of „mad-itch“with especial reference to its relationship to pseudorabies.
J. Exp. Med. 1931, 54, 233-248.

Shope, R.E., 1935a:
Experiments on the epidemiology of pseudorabies. I. Mode of transmission of the disease in swine and its possible role in its spread to cattle.
J. exp. Med. 1935, 62, 85-99.

Shope, R.E., 1935b:
Experiments on the epidemiology of pseudorabies. II. Prevalence of the disease among Middle Western swine and the possible role of rats in herd-to-herd infections.
J. exp. Med. 1935, 62, 85-99.

Todd, D., McFerran, J.D. 1985:
Restriction endonuclease analysis of Aujeszky’s disease (pseudorabies) virus DNA: comparison of Northern Ireland isolates and isolates from other countries.
Arch. Virol. 1985, 167-176.