Aujeszky's disease: factors important for epizootiology and control

G. WITTMANN**

Summary: Aujeszky's disease (AD) is found all over the world in regions with dense pig populations. In Europe it is widespread and only a few countries are free from the disease.

AD virus (ADV) is fairly resistant to heat and very stable at normal temperatures and in the cold. Its pH stability ranges from 5.0 to 12.0.

The most efficient disinfectants are chemicals that liberate chlorine, and formaldehyde. NaOH is less effective. Lime is recommended for disinfection of slurry.

Because of its pH and temperature stability, ADV is very resistant to natural environmental conditions.

Pigs are the primary host of the virus, although a large number of other species can be infected naturally and experimentally.

Infection chiefly takes place by nasal and/or oral routes. The virus can further be transmitted to the fetus, and by mating or by artificial insemination. The virus is not very contagious.

Primary virus multiplication occurs in the naso-pharyngeal region and the respiratory tract. ADV enters the CNS by the neural pathway. The virus is disseminated within the body by lymphocytes and macrophages.

The virus is mainly excreted by nasal and oral excretions, but it is also present in the ejaculate, vaginal secretion and milk.

ADV infection evokes antibody production and cell-mediated immunity.

The ADV genome persists in infected pigs in a latent state for life. It can be reactivated by stress, and virus excretion then occurs.

Vaccination of pigs is successful in controlling clinical outbreaks. Inactivated and live vaccines are used, though the latter carry more risks. Vaccinated pigs are protected only against small quantities of the virus. With larger quantities, virus multiplication takes place and latent ADV infection is established. Vaccination of other animal species has given very contradictory results.

AD is endemic in areas having a dense pig population and intensive, specialized farming management, which involves much movement of animals. Therefore the distribution of AD can differ within a country.

From the epidemiological point of view, infected pigs are the main source of the spread of AD over large distances. ADV-infected breeding and artificial insemination centres are additional sources. For virus spread over short distances various sorts of vectors are involved. Air transmission is still controversial.

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** Präsident und Professor, Bundesforschungsanstalt für Viruskrankheiten der Tiere, Paul-Ehrlich Str. 28, P.O. Box 1149, 7400 Tübingen, Federal Republic of Germany.
Preconditions for the control of AD are compulsory notification of outbreaks and serological surveys. Further legislative measures should be taken for control. Vaccination does not eradicate AD. It prevents economic losses, but does not prevent the circulation of ADV in vaccinated herds.

The only effective way of eradicating AD is to slaughter all the pigs of seropositive herds. However, such eradication programmes are very expensive in heavily-infected countries. Sanitation programmes over large areas are also very costly, time consuming and their success is uncertain.

For preventing the introduction of ADV into AD-free countries or regions, trade in pigs must be carefully controlled, and only seronegative pigs should be imported. The same is true with regard to the import of semen. The risk of importing ADV with frozen meat is very slight.

KEY-WORDS : Swine - Aujeszky’s disease - Europe - Epidemiology - Control.

Aujeszky’s disease (AD) is a contagious illness which is characterized by encephalomyelitis and, in conjunction with this, involvement of the nasopharyngeal, tracheal and pulmonary regions. AD is caused by a DNA virus belonging to the herpes group. It occurs in a large number of mammals in natural conditions, the pig being of particular significance. In this paper I want to pick up some points which are directly and indirectly important for the epizootiology and the control of AD.

INCIDENCE OF AD

AD is found all over the world in regions with dense pig population. In Europe (8) outbreaks of AD have never been reported in Norway, Finland and Malta. The other countries show various degrees of incidence of AD: the disease is endemic in Belgium, the Federal Republic of Germany, France, Ireland, inclusive of Northern Ireland, and the Netherlands. It is of sporadic occurrence in Czechoslovakia, Denmark, the German Democratic Republic, Hungary, Italy, Luxembourg, Portugal, Sweden, United Kingdom and USSR. AD has occurred exceptionally in Albania, Austria, Bulgaria, Greece, Poland, Romania, Spain and Switzerland. In Cyprus no cases have been reported since 1967.

RESISTANCE OF AUJESZKY’S DISEASE VIRUS (ADV) TO TEMPERATURE AND pH

The ADV is fairly resistant to heat. It is inactivated at 60°C within 30-60 min, at 70°C within 10-15 min, at 80°C within 3 min and at 100°C within 1 min (42). The virus is very stable at normal temperatures and in the cold. It stays alive at 25°C about 6 weeks, at 15°C about 9 weeks, at 4°C about 20 weeks and at -40°C for years. However, the virus is relatively unstable at -18°C to -25°C, where inactivation occurs within 12 weeks (24, 95).

Between pH values of 5.0 and 12 the virus is stable and even at pH values of 2.0 and 13.5 it takes 2 to 4 hours before the virus is completely inactivated (13).

By combining low or high pH levels with elevated temperatures the inactivation time is significantly reduced (24).
DISINFECTANTS

The effect of disinfectants depends on the resistance of ADV to chemical influences and on the environmental conditions, such as temperature, protective factors, etc. The following data are based on laboratory experiments (21, 74). The use of caustic soda solution is not advised, since the virus is not sufficiently inactivated by 1% NaOH even after 6 hours (74). A 3% formalin solution kills the virus in about 3 hours. Chemicals that give off chlorine are the most effective disinfectants. A 3% chloramine solution inactivates the virus within 10 min, a 1% solution within 30 min. Quaternary ammonia compounds are also very effective. When disinfecting on a large scale, cheaper disinfectants are adopted: calcium chloride milk, calcium chloride preparations which dissolve in water, crude chloramine and agents containing at least 1% active formaldehyde. For disinfecting slurry lime (20 kg Ca(OH)₂ per m³) is recommended (37).

NATURAL ENVIRONMENTAL CONDITIONS

Because of its pH and temperature stability the ADV is very resistant to natural environmental conditions. However, it must be realised that the environment is of a complex nature; adverse and favourable factors concerning virus survival are involved. Therefore, the data given must be taken only as clues to the result.

The virus is not killed in the course of the maturation of pig meat at 4°C (77); however it is inactivated in the meat at -18°C within 35 to 40 days (25, 26). In urine the virus survives for 3 weeks in summer and 8-15 weeks in winter (11). In slurry the virus is thought to live about 2 months in winter and about 1 month in summer (39, 40), in biothermically treated slurry the virus is inactivated in summer within 5 days and in winter within 12 days (56), in aerated slurry (pH 9.6, temperature up to 44°C) the virus is inactivated within 50 hrs (18). In packed-down dung the virus is inactivated in 8 to 15 days (40). In soil the virus was found for 5 to 6 weeks (39). In hay and straw the virus can survive 15 days in summer and 40 days in winter (67) while virus dried on sacks and wood survived a somewhat shorter time, about 10 days in summer and 15 in winter (40, 67). No data are available on untreated edible waste material, but in material fermented by Lactobacillus acidophilus ADV was inactivated at 20°C and 30°C within 24 hrs, but survived for at least 48 hrs at 10°C and at least for 96 hrs at 5°C (93).

SUSCEPTIBLE ANIMAL SPECIES

Pigs are the primary host of the virus, although a large number of other species can be infected naturally or experimentally. The most important of these are cattle, sheep, goats, dogs, cats and foxes in fur farms, rats and wild mice. On the other hand, it is very difficult to infect horses and birds; large virus doses are necessary and they must be injected either intracerebrally, subcutaneously or intramuscularly. Man is considered to be not susceptible to the disease.

In pigs the morbidity and the mortality rates are dependent on the age of the animals and because they decrease with rising age, piglets and young pigs are most at risk from the disease. In other species the course of infection is usually lethal at all ages and recovery is an exception.
INFECTION

Pigs are infected chiefly through the nasal route by aspirating virus aerosols or by sniffing sick animals. Oral infection occurs by taking in food or mother's milk containing virus. The virus can be transmitted in the uterus from the mother to the foetus. It can be also transmitted during mating or by artificial insemination. Dogs, cats and other carnivores as well as mice and rats are infected orally by eating ADV contaminated meat, slaughter offals or carcases.

Susceptibility to infection is dependent on several factors (12, 15, 25, 34, 43, 59, 60, 84, 86): the degree of virulence of the virus strain, the amount of virus taken in, the route of infection, the species of animal, the age of the pigs, stress, and individual condition of the animal. For example, larger quantities of virus are necessary for oral infection than for nasal infection, rats require high doses of virus to become perorally infected but low doses when infected intramuscularly, piglets need less virus than adult pigs and cattle need more virus than pigs. For intranasal infection, piglets require between 10^1 and 10^3 TCID_{50}, young pigs about 10^4 TCID_{50} and grown-up pigs 10^5 TCID_{50} (3, 12, 34). Cattle require at least 10^5 TCID_{50} (15, 84).

From this one can conclude that ADV is not very contagious. This is shown by the fact that usually not all the pigs or cattle within a house become infected. With pigs the percentage of animals infected fluctuates between 50% and 90% and with cattle between 3% and 60%. The spread of infection within a herd mostly depends therefore on the opportunity of direct contact between animals. Spread of infection is highest within a pen but lower from pen to pen.

VIRUS MULTIPLICATION

(17, 45, 59, 60, 83, 86)

Primary virus multiplication in pigs takes place in the naso-pharyngeal region and in the respiratory tract. From here the virus invades the CNS by the neural pathway. The virus seems also to be disseminated throughout the body to certain organs and tissues in pigs by leukocytes in blood and lymph (83). It can also be assumed that virus spread also occurs centrifugally from CNS via the nerves to other parts of the body.

The intensity of virus multiplication varies in different parts of the body. The largest amounts of virus are always found in the sites of primary virus multiplication, especially in the tonsils and pharyngeal lymph nodes. Smaller amounts of virus are present in the lungs, and fairly small amounts of virus are found in the CNS and in other organs. The more the virus multiplies in the organs, the longer it can be isolated there. Virus from throat lymph nodes and tonsils, therefore, can be isolated for up to 35 days, from the lungs for up to 14 days, from the CNS for up to 10 days and from the other organs for up to 7 days.

In cattle, primary virus multiplication also occurs in the naso-pharyngeal region and the thymus. However, virus is thereafter only found in the CNS (47, 84).

VIRUS EXCRETION

The virus can be isolated from nasal swabs of infected pigs for 8 to 17 days with maximum titres of between 10^{5.8} and 10^{8.3} TCID_{50} per swab (30, 44, 45, 87). From oropharyngeal swabs virus can be isolated for 18 to 25 days with titres up to 10^6 TCID_{50} (20). At the peak of virus excretion one pig excretes 10^{5.3} TCID_{50} into the
air during a 24 hrs period (25). Virus is found in vaginal secretions and foreskin secretions (ejaculate) for up to 12 days (3, 48, 50) and for 2 to 3 days in the milk (39). The virus occasionally occurs in the urine but it has not been isolated from the faeces (3, 39), though it was found in rectal swabs up to 10 days (25). It is important to note that virus excretion starts before the onset of clinical symptoms.

In nasally-infected cattle the virus can be isolated from the nasal secretion (84). The amount of virus excreted is less than in pigs but reaches up to $10^4$ infectious units per nasal swab, which is sufficient to infect other animals.

In dogs and cats the virus is excreted in the saliva, but the amount of virus excreted is very small, amounting to about 10 to 100 infectious units (75). In sick rats the virus is found in the nasal and oral mucosa (43).

**IMMUNITY**

ADV infection evokes antibody production and cell-mediated immunity in pigs (10, 29, 51, 65, 66, 79, 80, 88, 90, 91, 92). However, immunity is not total; after reinfection the virus can multiply to a limited degree despite a previous immune response. In cattle, sheep, dogs and cats the disease takes such a rapid course that no antibodies are produced.

**LATENT PERMANENT INFECTION**

The ADV persists in the infected animal in a latent state all its life (14, 23, 31, 32, 57, 61). It makes no difference whether the infection is accompanied by clinical symptoms or not, or what degree of immunity the animal possesses. A latent infection also occurs when pigs with colostral antibodies (52, 53) or pigs which have been vaccinated either with live vaccines (49, 53) or inactivated vaccines (88) are infected with virulent ADV.

The latent virus can no longer be directly identified as it is not present as a complete virus but only as DNA, the genetic substance of the virus. To detect this DNA special biological methods such as tissue fragment cultivation, co-cultivation or biochemical methods are needed (14, 31, 57, 61, 88). Virus DNA can be present in cells of the tonsils, thymus, lymph nodes, lungs, ganglion trigeminale, brain, spinal cord, bone marrow, and in macrophages and lymphocytes.

The virus DNA in the cell can be reactivated. This reactivation can be induced experimentally by using immuno-suppressants (52, 88). Natural reactivation has been reported in sows at farrowing (23). Presumably other stress situations such as fever, extreme temperatures, transportation, etc. also act as triggers. After reactivation, complete virus particles are formed which multiply in the cells and spread through the body. The virus is excreted and a booster effect is evoked on antibody production (52, 88).

Latent infection can occur only in animals which survive the illness. For this reason latency has been described so far only in pigs. In other animal species no suitable test material is available because, as a rule, the sick animals die.
VACCINATION

Vaccination of pigs against AD is successful in controlling clinical outbreaks. Inactivated vaccines and live vaccines are available (5, 33, 36, 71, 73, 81). The question of using inactivated or live vaccines is a scientific and a political problem. In some countries live vaccines are forbidden whereas in other countries they are allowed. Live vaccines certainly offer more risks than inactivated vaccines. The attenuated virus multiplies in the body and can be excreted, so that transmission to other animals, reverse mutations and genetic recombination with field strains cannot be excluded. Many of the live vaccines remain pathogenic for cattle, small ruminants, dogs and cats (5, 63, 78). Cases have been reported where cattle died as a result of being infected with residual attenuated virus on syringes and hypodermic needles which had not been sterilized after vaccinating pigs (22, 38). In Germany cattle and dogs died after illegal vaccination with a so-called innocuous live vaccine. Inactivated vaccines, on the other hand, provide at least as good immunity as live vaccines without risk.

With both sorts of vaccines two vaccinations in an interval of 4 to 6 weeks are necessary for optimal immunity, which lasts about 5 to 6 months; however, with fattening pigs one vaccination gives sufficient protection until slaughter. With breeding pigs vaccination must be repeated every 6 months or 4 to 6 weeks before farrowing.

In pigs which have received colostrum from ADV-immune sows antibodies are demonstrable maximally until 12 weeks (68). However, the protection level of antibodies vanishes much earlier, namely after 4 to 10 weeks, depending on the amount of antibodies transferred. These colostral antibodies inhibit the development of active immunity after vaccination (7, 55, 71, 72, 76, 85, 96). But the antibody blockade can be partially overcome by vaccination, when the antibody level has dropped to a low one. Therefore, vaccine producers recommend vaccinating these pigs first when 6 to 8 weeks old, and, very importantly, a second time 4 to 6 weeks later.

Vaccinated pigs with optimum immunity are protected against an Aujeszky virus infection only if they contract only small quantities of the virus, e.g. $10^4$ TCID$_{50}$ intranasally. With larger virus quantities virus multiplication takes place in the body (6, 83, 86). This is less than in non-vaccinated pigs but the virus spreads through the nerve system to other parts of the body. Slight clinical symptoms can appear. Virus is excreted with nasal and pharyngeal fluids, though the intensity of virus excretion (between $10^{2.8}$ to $10^{4.3}$ TCID$_{50}$) and the duration (4 to 7 days) is lower than with non-vaccinated pigs (87). As with non-vaccinated animals, the virus genome persists in the cells of the central nervous system and of other parts of the body, can be reactivated by stress and is excreted (49, 52, 53, 88). Therefore, after being infected with ADV vaccinated animals behave similarly to non-vaccinated animals. The same is true when passively immune piglets are infected with ADV.

Reports on vaccination of cattle, small ruminants, dogs and cats are contradictory. These animals responded to vaccination by the development of neutralizing antibodies. When challenge infection was performed intramuscularly, subcutaneously or intradermally, most of these animals were protected (4, 27, 28, 58). However, no protection was observed after nasal or oral infection (15, 54, 89). An
explanation for this discrepancy may be that in the first cases ADV was neutralized by antibodies at the site of infection, before nerves were reached. After nasal or oral infection the virus apparently enters the nerves directly and reaches the CNS, being thereby protected against the action of antibodies which cannot cross the nerve barrier.

**EPIZOOTIOLOGY**

AD is endemic in areas with dense pig population and intensive, specialized farming management which affords a lot of animal movement between the farms. Therefore the distribution of AD within a country differs from one part to another. In districts where small pig farms, which produce their own stock, are predominant, AD is not endemic, unless the boar station is infected. When single outbreaks of AD occur in these districts they usually originate from pigs which have been imported from infected areas.

The epizootical course of AD shows seasonal cycles. During the warm season the number of outbreaks drops and reaches a minimum from July to September. During the cold season the number of outbreaks rises, showing a maximum from January to April. The reasons for this are unknown, but survival conditions for the virus are certainly better in winter than in summer.

From the epizootiological point of view ADV infected pigs are the main source of the spread of AD. The other species are less important since they usually die and virus spread is thus interrupted. The most important cause of virus spread over long distances is the trade with clinically inapparently infected pigs. Even the stress of transportation might be sufficient to evoke reactivation of the virus genome. ADV-infected boar stations and artificial insemination centres are also sources of virus spread, as well as ADV-contaminated vehicles used for transportation of pigs. Theoretically, ADV may also be transmitted by embryo transfer (19), however, in practice this seems unlikely (35).

Further sources of virus spread, especially over short distances, e.g. to neighbouring stables or within a village, can be by man, implements, vehicles, food, offals, cats, dogs and rats. However the epizootiological role of these latter animals must not be overestimated. If they transmit ADV it is more mechanically as vectors than by virus secretion (43, 75). Experimentally no contact transmission was found between sick cats and pigs (75). Insects are apparently not involved in virus spread since they are not virus hosts and, furthermore, not mechanical vectors of the virus. This is shown by the fact that outbreaks of AD are reduced in the warm season and rise in the cold season.

Aerial transmission of ADV is a matter of discussion. An infected pig excretes up to $10^{5.8}$ TCID$_{50}$ of ADV at the peak of virus excretion during a 24 hour period (25). Thus air-borne infection occurs within a pig-house and exceptionally, under unfavourable conditions the virus may be transmitted e.g. by the air stream of a ventilator, to nearby houses or pastures over a distance of 10 to 20 metres, eventually up to 500 m (16). However, it is very dubious whether ADV can be transmitted by air over longer distances, when one realizes that the virus is not very contagious.
CONTROL AND ERADICATION OF AD

Preconditions for the control of AD are the duty of notification of AD outbreaks and serological surveys on a large scale to find infected herds, especially with regard to latently-infected pigs. In this connection the question arises whether latently-infected pigs can be seronegative. In my opinion, this is not the case. We have never found experimentally ADV infected pigs which became seronegative during an observation period up to 2.5 years. This is apparently due to a booster effect on antibody production which occurs at each reactivation (52, 87, 88). However, it might be that foetuses which had been infected in the uterus and survived establish immunological tolerance. After birth they are latently infected but do not have antibodies.

Besides notification further legislative measures can be taken for control, e.g. quarantine of the infected farm, killing of the infected herd, disinfection, heat treatment of meat, restrictions on transport and markets, etc. However, most of these measures are apparently not very effective unless all the infected herds are eliminated. But this latter procedure is very expensive and therefore it is limited to countries or areas where AD is not endemic. The United Kingdom, with a low incidence of AD, is going this way. Up to September 1983 £19 million had been paid for compensation (9). In the Federal Republic of Germany, with a high incidence of AD, DM 61 million were paid for compensation between 1980 and 1982. Nevertheless, AD increased in the Republic during this period from 631 to 1,290 new outbreaks per year.

The failure of the classical legislative measures of veterinary policy, except killing-out, for controlling AD is not surprising. Firstly, AD is a latent permanent virus infection and secondly modern animal husbandry depends on management and trade conditions which are far away from the conditions 50 years ago, which are still the base of veterinary legislation.

For example, a piglet producer would be ruined, if he is not allowed to sell his piglets as long as there are seropositive animals in the herd. The only possibility of overcoming these difficulties is the killing-off of AD-sick animals and vaccination of the clinically healthy pigs of the infected herd; the ban could be lifted 5 weeks later. However, it must be realized that a part of these pigs would have been apparently ADV infected before vaccination and would have converted, despite vaccination, to latent infection with all its consequences, namely reactivation, virus excretion and infection of vaccinated and non-vaccinated animals.

Prophylactic vaccination is widely used for protecting non-infected pig herds in endemic areas. However, as already mentioned, vaccination does not in any case prevent ADV infection, which converts into latent infection. These animals can spread the disease despite their being vaccinated, and the virus circulates within the herd (62, 69, 94) and can be transmitted by these animals to other herds. Therefore, each vaccinated pig should be marked regardless of originating from an infected herd or a non-infected herd. Thus, everyone who wants can avoid buying a vaccinated animal. In addition, one must remember that not all pigs of a vaccinated herd are ever immune. Some vaccinated animals are always unable to develop sufficient immunity, especially after the first vaccination, and there are pigs in the herd which have not been immunised, either because they were too young or derived from immune sows. In the meantime their maternal immunity has been lost. Thus susceptible animals are among the immune ones and they can be targets for ADV infection.
In general one can say that vaccination prevents or reduces clinical disease and therefore economic losses, but it does not prevent ADV infection, virus excretion and establishment of latent infection. Therefore, though vaccination may indeed delay the spread of AD, the disease will never be eradicated by this means. Furthermore, serological surveys will be made difficult because it is impossible to distinguish between vaccinated, vaccinated/infected and unvaccinated/infected animals.

The question is, what can be done?

The most effective method to eradicate AD in a country is to slaughter all the pigs of seropositive herds within the shortest time possible. A precondition for this is repeated serological surveys of at least 10% of the pigs in every herd in the country.

In heavily infected countries such an eradication programme is very expensive. Based on the 1,249 clinical outbreaks of AD in Germany in 1983 it can be calculated that the costs of eradication would amount to about DM 175 million. The assumptions used were the following: (a) the number of seropositive herds would be three times higher than that of the clinically affected herds, (b) an assumed number of 100 pigs per herd, (c) that about 50% of the compensation paid would return by income from sales, (d) that about 6.4 million of pigs would be serologically examined and (e) that all the serologically positive herds would be slaughtered within four years.

Let us consider the next possible but less acceptable procedure, the killing of all the herds with clinical AD and all the seropositive animals in latently-infected herds. If it is assumed that about 30% of the animals in infected herds would be seropositive in the average and that this method would take five years, then about DM 123 million would be necessary. The reason for the reduction of costs by only one-third is that the costs for the serological surveys in both cases are the same.

However, nobody knows how these theoretical models work in practice. For example, with hog cholera, it took eight years to reduce the number of outbreaks by a similar eradication programme from 1,226 in the year 1974 to four outbreaks in 1981.

What about the possibility of eradicating AD by starting sanitation programmes in the infected herds? Several programmes exist (1, 2, 41, 64, 70, 97). Most of them are based on the rearing of a seronegative offspring. When the original breeding animals are not vaccinated, the seropositive ones must be separated from the seronegative ones and from their offspring and slaughtered as soon as possible. The smaller is the number of seropositive animals, the more promising this procedure is; but if the number of seropositive is around 50% or higher, this method will not work. Therefore, it is better to vaccinate the original herd and rear an unvaccinated, seronegative offspring. We have no time to go into details of sanitation procedures, but I want to say that the sanitation programme must be designed individually for each herd and each type of management.

Sanitation is also very costly and time-consuming. One has to assess two years until a herd is sanitized, provided that good isolation facilities are present and any introduction of ADV can be prevented. This will be difficult in heavily-infected districts. To eradicate AD in such districts by the herd-sanitation method is either completely impossible or it takes ten or more years (2, 41). The final costs of such a programme will clearly be higher than those of the first programme mentioned.
Finally, what can be done to prevent the introduction of ADV into AD-free countries or regions?

First of all, the trade in pigs must be carefully controlled. Only seronegative pigs should be allowed for import. The same is true with regard to the semen used in artificial insemination.

The question now arises: what about the import of meat from ADV-infected countries? It is true that ADV can be present in the meat (77); however, this seems to be a rather rare event, even during the acute phase of the disease (17, 25, 59, 60). It cannot be excluded that virus strain differences exist in this regard. Further, after reactivation of the latent virus the invasion of the meat might only occur rarely if at all. Again, ADV is not very stable at temperatures around −20°C and in the meat the virus is inactivated at −18°C within 35 to 40 days (25, 26). This is probably caused by enzymatic processes which proceed at this temperature and destroy the virus. Therefore, I personally believe that the risk of importing ADV with frozen meat is very small.

What is the outlook on the future situation of AD? With increasing intensity of pig-husbandry and trade in pigs, AD can spread further and remain endemic in infected countries and areas. Legislative measures, except the slaughter policy and vaccination will not prevent this tendency; they only veil it to a certain degree. Heavily infected countries with intensive and extensive production of pigs will probably have to live with AD in the future.

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REFERENCES

**Books and reviews:**


**Special papers:**


