Summary: Viral haemorrhagic disease (VHD) is a new and severe infectious disease of rabbits, with a high rate of morbidity and mortality. The disease occurs throughout the year, affecting only adult rabbits and not other domestic animals, fowls or laboratory rodents. The transmission is horizontal, by direct or indirect contact, and through all routes. There is no evidence of congenital infection or biological vectors.

The causative agent, viral haemorrhagic disease virus (VHDV), is present in all tissues, excretions and secretions. It is an icosahedral and non-enveloped parvovirus. The genome, as determined by classical methods, high performance chromatography and in vitro synthesis of double-stranded DNA, is linear, single-stranded DNA. VHDV can agglutinate human erythrocytes at very high titres, irrespective of blood groups, and has a stable reaction to many physical and chemical factors. VHDV has been adapted to grow on rabbit kidney cell strain (DJRK) culture and to produce cytopathic effect (CPE). Inactivated cell culture can protect inoculated rabbits against virulent VHDV.

The disease is now effectively controlled in the People’s Republic of China, but has not yet been completely eradicated.


INTRODUCTION

Viral haemorrhagic disease (VHD) is a new, peracute and highly communicable disease, affecting adult rabbits only. It is characterised by sudden death, haemorrhages in various tissues, bloody exudates in the trachea and around the nostrils, and by haemagglutination (HA) of human red blood cells at a very high titre. The aetiological agent is a unique virus which was not reported in worldwide literature before 1984. Owing to contradictory results reported on the nature of the genome, no consensus has been reached concerning its classification as a virus. The author bases his assumption that it is a parvo-like virus on the type of genomic nucleic acid involved and on cross-reaction with certain other parvoviruses.

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CHARACTERISATION OF 
VIRAL HAEMORRHAGIC DISEASE VIRUS

Morphology

The morphology of viral hemorrhagic disease virus (VHDV) was examined under the electron microscope by negative staining with phosphotungstic acid after a sample from the liver emulsion of an infected rabbit had been concentrated and purified by differential centrifugation and polyethylene glycol (PEG) sedimentation, or further by extraction with chloroform and other organic solvents. VHDV is approximately 32-34 nm in diameter and varies slightly in different preparations (2, 3, 4, 6, 9, 13, 16, 18, 20, 26, 29). The virion is icosahedral and nonenveloped and has a core of 18-20 nm (4, 9, 13). The capsid is composed of 32 capsomeres (9, 18) (Fig. 1). The virions in thin sections of rabbit liver and kidney cell strain (DJKR) from infected cell culture are slightly smaller and are found in the cell nuclei and around the nuclear membrane (8, 14, 17).

FIG. 1
Viral haemorrhagic disease virus, negative staining,
× 216,000

Sedimentation coefficient and buoyant density

The purified virus preparation is layered on a continuous sucrose gradient from 20 to 60% and centrifuged at 18,400 rpm. The sedimentation coefficient of VHDV, determined by ultraviolet scanning, is 162S. The purified virus preparation layered
on 35% CsCl is centrifuged at 40,000 rpm for 24 h. The virus band is collected and examined with Abbé’s refractometer. The buoyant density of VHDV is 1.36-1.38 g/cm$^3$ (2, 10).

Nucleic acid

Because the virus is difficult to grow on cell culture and to purify without contamination of host cell components, there was much disagreement among Chinese scientists between 1984 and 1986 over the nature of the nucleic acid of VHDV. The dispute subsided after highly purified preparations and modern techniques, complementing classical methods, had been used.

Type

The partially purified virus preparation is further treated with ribonuclease (RNase) and deoxyribonuclease (DNase) to degrade the contaminating nucleic acids of host cells adsorbed on the virion capsid. The highly purified virus preparation is examined by diphenylamine and orcinol reactions and the nuclease degradation test (2, 18, 20). The electrophoretically pure nucleic acid is also analysed by high performance liquid chromatography for its base composition (20, 21). The results are summarised in Table I and Figure 2.

**Table I**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>VHDV-NA</th>
<th>Yeast RNA</th>
<th>Thymus DNA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphenyl amine</td>
<td>Blue</td>
<td>No change</td>
<td>Blue</td>
</tr>
<tr>
<td>Orcinol</td>
<td>No change</td>
<td>Dark green</td>
<td>Light green</td>
</tr>
<tr>
<td>DNase 1</td>
<td>Degraded</td>
<td>Resistant</td>
<td>Degraded</td>
</tr>
<tr>
<td>RNase A</td>
<td>Resistant</td>
<td>Degraded</td>
<td>Resistant</td>
</tr>
</tbody>
</table>

Since VHDV nucleic acid turns blue in the diphenylamine reaction and light green in the orcinol reaction, is degraded by DNase 1 but resistant to RNase A, and is composed of four nucleoside bases, adenine (A), thymine (T), guanine (G) and cytosine (C), there is no doubt that it is a DNA.

Strand

The highly purified virus preparation has been subjected to several tests. In the melting point (Tm) test, the purified nucleic acid is dissolved in standard saline-citrate buffer (0.15 M NaCl and 0.015 M sodium citrate, pH 7.0). The optical density (OD) values of the solution are recorded every 5 min from 18°C to 95°C by ultraviolet spectrometry at a wavelength of 260 nm. The OD values increase only slightly from 0.25 to 0.28. This indicates that the VHDV nucleic acid cannot be separated by heat, unlike that of the double-stranded (ds) DNA. In acridine orange staining the nucleic acid band in agarose gel, after electrophoresis, gave a red flame when examined under a high pressure mercury lamp. In the nuclease degradation test, the nucleic acid was degraded by Alu 1, but resistant to DNase S1 (2, 20). In high performance liquid
FIG. 2

High performance liquid chromatogram of hydrolysates of VHDV nucleic acid

Left: VHDV nucleic acid
Middle: calf thymus DNA
Right: yeast RNA

Note the difference in the four nucleotide bases between DNA and RNA

C = cytosine
T = thymine
U = uridine
G = guanine
A = adenine
chromatography, $A = 24.4\%$, $T = 31.9\%$, $C = 23.7\%$ and $G = 20.8\%$ (19). Since $G$ is paired with $C$, and $T$ with $A$, the mole percentages of $G$ and $C$, or $T$ and $A$, have to be approximately equal in dsDNA. Here $G$ and $C$, and $T$ and $A$, are unequal, which provides indirect evidence that the nucleic acid tested is single-stranded. The results are summarised in Table II.

**Table II**

Identification of the nature of the nucleic acid strand of VHDV

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Nucleic acid of VHDV</th>
<th>Phage λ ssDNA</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heat denaturation</td>
<td>No change</td>
<td>No change</td>
<td>OD value increase</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>at 260 nm</td>
</tr>
<tr>
<td>Acridine orange</td>
<td>Red</td>
<td>Red</td>
<td>Green</td>
</tr>
<tr>
<td>DNase S1</td>
<td>Degraded</td>
<td>Degraded</td>
<td>Resistant</td>
</tr>
<tr>
<td>Alu 1</td>
<td>Resistant</td>
<td>Resistant</td>
<td>Degraded</td>
</tr>
<tr>
<td>Base composition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>by HPLC</td>
<td>C = 23.7</td>
<td>C = 21.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>G = 20.8</td>
<td>G = 21.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>T = 31.9</td>
<td>T = 27.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>A = 24.4</td>
<td>A = 28.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Shape**

After elimination of the contaminating cellular nucleic acid with nuclease, the purified virions are treated with urea to elicit the release of the nucleic acid from the capsid. After being spread on water, stained with uranium acetate and shadowed with platinum, the specimen is observed under the electron microscope. As shown in Figure 3, the nucleic acid is not segmented and is linear in shape, with an average length of 1.92 µm (1.84-2.2 µm) (28).

The results of polyacrylamide gel electrophoresis (PAGE) and agarose gel (AGE) also indicate that the nucleic acid of VHDV is not segmented (2).

**Molecular weight**

The molecular weight of VHDV nucleic acid is $2.4 \times 10^6$ when compared with the distance moved by phage M 13 single-stranded DNA after PAGE and AGE. The molecular weight calculated from the length of nucleic acid, by multiplying by $1.2 \times 10^6$ per µm, is $2.3 \times 10^6$. These two values are very close.

**Terminal hairpin**

The evidence of classical tests and high performance chromatography indicates that the genome of VHDV is single-stranded DNA. Among vertebrate viruses, only the genome of the Parvoviridae belongs to this type. If VHDV is a parvovirus, there should be hairpin structures at the 3' and 5'-terminals of the nucleic acid chain. To examine this, the VHDV nucleic acid is extracted from the highly purified viral preparation and reacted with *Escherichia coli* DNA polymerase 1 Klenow large
Electron micrograph of DNA of viral haemorrhagic disease virus, \( \times 20,000 \)

Fragment. Double-stranded DNA is synthesised \textit{in vitro} and then integrated into the Bluescript plasmid. The recombinant plasmids collected from transformed \textit{E. coli} are identified by agarose electrophoresis and restriction enzyme cleavage. They could hybridise with VHDV nucleic acid by dot blot hybridisation after being labelled by nick translation (24). The evidence indicates that there is a hairpin at the 3'-terminal of VHDV nucleic acid.

**Polypeptides**

The emulsion of infected liver was purified by a process of repeated freezing and thawing, chloroform extraction, PEG sedimentation and differential centrifugation. The partially purified VHDV preparation was chromatographed on a Sepharose 4B column. Three fractions were eluted. The purified and concentrated VHDV was in fraction two when identified by HA and AGE. This fraction was analysed for viral polypeptides with sodium dodecyl sulphate (SDS)-PAGE. Four polypeptides were detected; their respective molecular weights, as compared with those of marker proteins, were as follows: VP1 = 58.3-61.1 kDa, VP2 = 54.7 kDa, VP3 = 51-53 kDa and VP4 = 25.9 kDa. When the electrophoretic gel was scanned with a densitometer, the average component percentages, calculated from the 4-test integral area, were: VP1 = 54.7 ± 1.2%, VP2 = 17.7 ± 0.6%, VP3 = 22.7 ± 1.1% and VP4 = 4.9 ± 0.1% (7, 11).

In Western blotting, the polypeptides were transferred from the electrophoretic gel onto nitrocellulose filters and reacted with:

a) VHD antiserum and peroxidase-labelled staphylococcal protein A, or
b) monoclonal antibodies from several hybridoma cell strains and peroxidase-labelled antimouse IgG goat serum.

All four polypeptides were detected by VHD antiserum and three monoclonal antibodies (25, 27) (Table III). It is suggested that polypeptides VP1, VP2, VP3 and VP4 share common epitopes, similar to those found in Aleutian mink disease virus.

**TABLE III**

*Immunological reactions between VHDV specific monoclonal antibodies and VHDV polypeptides, according to Western blotting*

<table>
<thead>
<tr>
<th>Name of MAb</th>
<th>VP1</th>
<th>VP2</th>
<th>VP3</th>
<th>VP4</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA8-1</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>CF2-1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CG4-1</td>
<td>+</td>
<td></td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>CH3-1</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RM-14</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RM-17</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

**Haemagglutination**

*Spectrum*

Various tissues collected from infected rabbits immediately before and after death can agglutinate human red blood cells at very high titres. No obvious difference is observed among blood groups A, B, AB and O. Erythrocytes of the chicken, goose and sheep seem to be agglutinated at low titres (10×10^2 - 10×10^4), while those of other animals, including the cow, goat, pig, rabbit, rat, mouse, guinea pig, duck and quail, cannot be agglutinated. HA is inhibited by specific antiserum.

*Distribution*

The virus exists in all tissues and body fluids of infected animals. The HA titres in the liver, spleen and serum can reach 10×10^10 - 10×10^18, much higher than those of other tissues (Fig. 4). There is some evidence that the HA titre is related to the content of blood in the tissue.

*Physical and chemical factors*

HA is not significantly affected at different temperatures (4°C-37°C). There is no strict pH value requirement; the HA titre, optimal at pH 6.0, is basically the same between pH 4 and 7.2. When the agglutinated red blood cells (RBC) are suspended in borate buffer at pH 9.2 and incubated at 22°C-37°C for 30 min, the viral particles are released. The released RBC and virus are able to reagglutinate when the pH value lowers. Two percent NaCl in 0.01 M phosphate buffer is usually used as a diluent of RBC in the HA test; this can enhance the HA titre to a value 1-2 times higher than in 0.85% NaCl (15, 23).
The HA activity can be destroyed by 0.5% trypsin, 2% sodium borohydride, or 2% chloramine T, but not by receptor destroying enzyme (RDE), sodium metabisulphite, potassium periodate, chloroform, ether or formaldehyde. The HA activity is rather stable to heat. When the infected tissue is kept at 4°C and 15°C for three months, the HA titres decrease by $2^{2-4}$ and $2^{4-5}$, respectively. If it is kept in a waterbath at 56°C for 24 h, the titres decrease by $2^{4-6}$ (23).

**Receptors on RBC**

The viral haemagglutinin receptors on the surface of human RBC are sensitive to chloroform and ether, and stable to trypsin, potassium periodate, RDE an
sodium borohydride. They are highly resistant to heat, unaffected after heating at 95°C for 15 min. The RBC fixed with 1% KMnO₄ remain agglutinable by the virus at 4°C, with a clear and stable agglutination pattern. Comparative study indicates that HA results with either fixed or fresh RBC correlate well (22).

**Resistance**

The 10% liver emulsion of infected rabbits was centrifuged at 10,000 x g for 30 min. The supernatant was collected and treated in each of the following ways:

a) in a waterbath at 50°C for 60 min;

b) in 1.0 M MgCl₂ solution in a waterbath at 50°C for 60 min;

c) in pH 3.0 Hanks’ solution at room temperature for 30 min;

d) in 20% ether stored overnight at 4°C;

e) in 10% chloroform, vibrating vigorously at room temperature for 10 min;

f) control, without any treatment.

Samples of each preparation were inoculated peritoneally into six or seven susceptible rabbits. Rabbits of the six groups all became infected and died within 36 h; however, one rabbit in Group 3 (preparation c) survived, as did another in Group 4 (preparation d) (4).

Alternately, the supernatant of the liver emulsion was mixed with an equal volume of 2% NaOH or 2% formalin and was incubated at 37°C for 1 h. Each mixture was diluted tenfold by phosphate buffered saline (PBS) and inoculated into six susceptible rabbits, all of which survived and remained healthy, while the control rabbits inoculated with untreated sample died within 36 h (20).

The virus is very stable at low temperatures. Infected liver samples kept at -5°C for 413 days or -20°C for 560 days did not have significant drops in their infective titres. Those kept at -70°C for 4.5 years after lyophilisation remained infectious without any loss (16). For safety purposes rabbit sheds, including the cages, floors, walls and utensils, should be cleaned and thoroughly disinfected with 10% bleaching powder or 3% formalin followed by 2% NaOH, and kept empty for two weeks in summer, or for two months in colder seasons.

**Relationship to some parvoviruses**

In indirect ELISA, when purified VHDV is coated on polystyrene microtitration plate wells and treated with VHDV reference antiserum and peroxidase-conjugated staphylococcal protein A, or with VHDV monoclonal antibodies and peroxidase-conjugated antiserum to GPV, the titre of VHDV is 1:6400. The results have been compared with those of the canine (CPV), feline (FPV), mink (MPV), pig (PPV), goose (GPV) and mouse (MVM) parvoviruses. When VHDV antiserum is used, the ELISA titres of these viruses are 1:160, 1:80, 1:40, 1:80, 1:160 and 1:160, respectively. When purified GPV is coated on microtitration plate wells and reacted with antiserum to GPV or VHDV, the respective titres are 1:12800 and 1:320. When purified MVM is used as antigen and reacted with antisera to MVM and VHDV, the titres are 1:9600 and 1:6400, respectively. If the coated antigen is PPV and is reacted with antisera to PPV and VHDV, the respective titres are 1:12800 and 1:320. If the coated antigen
is MEV and is reacted with antisera to MEV and VHDV, the titres are, respectively, 1:5120 and 1:40. The results indicate that VHDV may, to some extent, be related in antigenicity to MVM, PPV and GPV.

Cell culture

VHDV is very difficult to grow in cell culture. Since 1984, the author has inoculated emulsions of liver, spleen, lung and serum collected from experimentally infected rabbits onto various cell cultures, including primary rabbit cells (kidney, liver, lung and testis) and cell lines (PK-15, BHK-21, MA-104, IBRS-2, HeLa and VERO). Blind passages were made and every passage was checked for HA and cytopathic effect (CPE). None of them manifested any evidence of growth. The author also modified the method of inoculation; for example, by inoculating just after adsorption of cells on a glass surface, inoculating at phase S1 of cell division, treating the sample with trypsin and adding trypsin to the cell medium, substituting rabbit serum for calf serum, and using cells together with culture fluid in passage. None of the attempts succeeded. In 1988, a rabbit kidney cell strain, designated DJRK, was used. Regular CPE was observed at the third passage; this was characterised by the rounding and aggregating of cells which finally detached from the glass surface (Fig. 5). Thread-like intercellular bridges were also observed (8). Fluorescence, principally in the cytoplasm around the cell nuclei and, to some extent, in the nuclei at an early stage, was detected by the fluorescent antibody technique. The cell cultures of the fifth and tenth passages were inoculated into susceptible rabbits, all of which died within 5 to 11 days. HA appeared to titres of $2^2$-$2^6$ at 1-3 days after inoculation, $2^6$-$12^2$ at 2-8 days after inoculation and increased abruptly to $2^{14}$-$2^{17}$ just before death. The pathological changes were similar to the natural VHDV infection. Numerous virions accumulated adjacent to the nuclei and a few were observed in the nuclei themselves, in thin sections of cultured cells of the tenth passage (Fig. 6). Susceptible rabbits, inoculated with cell culture of the tenth or fifteenth passages inactivated by 0.35% formaldehyde, could produce HI serum antibodies to titres of $2^9$-$2^{11}$ at day 10 post inoculation and could resist the challenge of virulent VHDV at day 15 post inoculation.

![Fig. 5](image)

**Fig. 5**

Cytopathic effect (CPE) produced in a monolayer of DJRK cell culture by VHDV

Rounding of cells and intercellular bridges can be observed.
Thin section of a monolayer of DJRK cells infected by VHDV, $\times 80,000$

Virions aggregate adjacent to the disappearing nuclear membrane; a few are seen in the nucleus.
EPIDEMIOLOGY

Host range

It is probable that only rabbits are susceptible to VHD. No evidence indicates that horses, mules, donkeys, cattle, buffalo, sheep, goats, swine, dogs, cats or fowls can be affected by the disease, though these animals have frequent contact with infected rabbits and rabbit carcasses, excretions and virus-contaminated materials in backyard rabbitries (5, 13, 16). Man is not susceptible to VHDV, as evidenced by the lack of disease in the millions of people who consume rabbit meat. No case of infection has been reported from large numbers of farmers, veterinarians and laboratory technicians who come in contact with VHDV daily.

Attempts to infect laboratory rodents and fowls have all been unsuccessful. It has been reported that white mice, rats (white, black, grey and brown), golden hamsters and guinea pigs have been inoculated subcutaneously, intramuscularly, peritoneally or orally with liver emulsions of infected rabbits. No symptom has manifested in these animals and HI antibodies cannot be detected in their sera (16).

A captive hare with pathological changes similar to VHD was found in 1986. Its liver emulsion was inoculated into rabbits and typical VHD resulted. The HA titre of that liver sample was $10 \times 2^6$, which could be inhibited by VHDV antiserum. Ninety-nine serum samples taken from hares had titres lower than $1:2^1-1:2^3$ (40 hares), $1:2^4$ and $1:2^5-1:2^7$ (25 hares), and $1:2^8-1:2^9$ (9 hares). Hunters have never seen any hares either infected or killed by a disease resembling VHD.

Susceptibility of rabbits

Domestic rabbits of all breeds appear to be fully susceptible. When Angora, Rex, Chinchilla, Japanese White and fur hybrids are inoculated experimentally with virus preparation, no significant difference in morbidity and mortality is observed. In natural outbreaks, however, wool rabbits may be more susceptible than meat and fur rabbits (16).

Susceptibility is clearly influenced by age. Rabbits less than one month of age cannot be infected clinically, and those between one and two months of age may be infected at low percentages. The majority of infections are found in rabbits older than three months. There is no difference in susceptibility between male and female adult rabbits (13).

Serum HI antibodies may exert effects on susceptibility. When the antibody level is above $2^4$, rabbits are usually resistant to experimental challenge (12). Forty-six samples of rabbit serum collected in Jiangsu Province from 1975 to 1983 were examined for HI antibodies to VHD. Titres of 44 samples were below $2^2$; those of only two samples reached $2^4$. This implies that VHD did not exist in Jiangsu province before 1984, but the source of antibodies is not clear. Naturally resistant rabbit flocks are not uncommon, even in isolated villages where VHD never occurs and vaccination is not carried out (1).

Transmission

The VHD virus is present in blood, organs, secretions, excretions and on skin and mucous membranes, principally during the late stage of infection and after death.
Direct or indirect contact with infected rabbits or carcasses, excretions and contaminated objects may transmit infection to susceptible animals.

Rabbit wool pedlars, who shear and purchase rabbit wool even from infected rabbits and carcasses, may play a significant role in the spread of the disease in the People's Republic of China. They come to villages by coaches and trains and the virus can be carried extensive distances on their hands, clothes, shoes and scissors. Through ignorance, farmers may carry the virus to their neighbours and relatives (19).

Infected rabbits may accidentally be mixed with healthy ones in rabbit processing plants, especially during the incubation period. The virus is difficult to detect in meat; however, by using ELISA, the author has detected it in bone marrow after viral antigens had been concentrated from samples (19).

Since 1986, new outbreaks have occasionally occurred in healthy rabbitries after the introduction of breeding rabbits from enzootic areas where vaccination with inactivated vaccine had been regularly carried out. Young rabbits and vaccinated adults may appear to be healthy when, in fact, they are subclinically infected (19).

Season

VHD is epizootic throughout the year, without marked seasonal differences, though outbreaks are comparatively more frequent during colder months.

PRESENT STATUS OF VIRAL HAEMORRHAGIC DISEASE OF RABBITS IN THE PEOPLE'S REPUBLIC OF CHINA

The first case of viral haemorrhagic disease, involving an Angora rabbit imported a few days before from the German Democratic Republic, appeared in the spring of 1984 in Wuxi City, Jiangsu Province, about 100 km from Shanghai. Except for the young and suckling animals, almost all the rabbits in the affected rabbitry had died of infection within a week. Pathogenic bacteria were not isolated and antibiotics were ineffective; no infectious disease with similar symptoms and pathological changes could be found by referring to the worldwide literature. In less than nine months, the disease had spread rapidly to an area of approximately 50,000 km², nearly destroying most of the rabbitries within that area. At first, local scientists felt helpless to control this horrible disease; before long, however, an inactivated vaccine was developed which induced solid and effective immunity in rabbits. By the end of 1986 the disease had gradually been brought under control, and there is now hope of eradication within a few years. However, the eradication programme is not always carried out as conscientiously as required by the veterinary authorities. Sporadic outbreaks, usually limited to a single rabbitry or a small village, are still found in other provinces, where vaccinations are not performed with the necessary vigour. The disease readily subsides after all the rabbits of infected rabbitries are slaughtered and the rabbits of the neighbouring rabbitries are vaccinated. The source of infection probably remains rabbits introduced for breeding purposes, even if they appear to be normal. These rabbits are not usually infected in an outbreak because they have been vaccinated, and high levels of HI antibodies are detected in their sera.

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LA MALADIE HÉMORRAGIQUE VIRALE DU LAPIN EN RÉPUBLIQUE POPULAIRE DE CHINE : ÉPIDÉMILOGIE ET CARACTÉRISATION DU VIRUS. – Wei-Yan Xu.

Résumé: Récemment reconnue, la maladie hémorragique virale (VHD) est une grave maladie infectieuse des lapins, qui entraîne des taux de morbidité et de mortalité très élevés. Elle peut se déclarer à n'importe quelle période de l'année et n'atteint que les lapins adultes à l'exclusion de tout autre animal domestique, volaille ou rongeur de laboratoire. Elle se transmet par voie horizontale, par contact direct ou indirect, et par toutes les voies. Il n'a pas été rapporté de cas d'infection congénitale ni de transmission par vecteurs vivants.

Le virus responsable de la maladie hémorragique virale (VHDV) est présent dans tous les tissus infectés, ainsi que dans les excréptions et sécrétions. Il s'agit d'un virus icosaédrique non enveloppé, proche des parvovirus. Les méthodes classiques d'analyse du génome, telles que la chromatographie haute performance et la synthèse in vitro de l'ADN double brin, ont permis de déterminer que le génome du VHDV était un ADN linéaire à un seul brin. L'auteur a observé que le VHDV agglutine à un très haut titre les globules rouges humains, quel que soit le groupe sanguin. Le virus reste stable en présence de nombreux agents physiques et chimiques. Le VHDV a été adapté en culture sur des souches cellulaires de rein de lapin (DJRK) où il entraîne un effet cytopathogène. La culture cellulaire inactivée peut protéger les lapins inoculés contre une souche virulente de VHDV.

La maladie est à présent bien contrôlée en République populaire de Chine ; elle n'a cependant pas encore été totalement éradiquée.

El cultivo celular inactivado puede proteger los conejos inoculados contra una cepa virulenta de VHDV.

Actualmente, la enfermedad está bajo control en la República Popular de China, a pesar de no haberse logrado todavía su total erradicación.


*  *

REFERENCES


