SEROLOGICAL SURVEY OF A PARVOVIRUS INFECTION IN A PIG HERD.

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Forcine Parvovirus (PPV) causes reproductive failure in swine and is widespread throughout the world. Several studies were performed in pig herds [2, 6]. In this study, the antibodies titers in the sera of sows in one pig herd have been examined for 4 years on the same animals to observe the evolution of antibodies levels and of the PPV infection among the breeders.

MATERIALS AND METHODS : in a breeding and fattening mig unit housing 100 sows, serum samples of 31 to 53 sows of different ages were regularly collected each month during 4 years (from 1977 to 1980). The sows were individually identified and the sera samples were collected from the same sows at each time. Of course, during these 4 years, some sows have been culled and gilts introduced. In this herd, the gilts are bred in the farm and, in usual conditions, no breeders are coming from outside. Antibodies were detected by haemagglutination inhibition test (HIT) [1, 5]. The antibodies titers were expressed as the reciprocal of the highest dilution of serum inhibiting haemagglutination of 4 HA units of virus. A serum is considered as positive if antibodies have been detected after the dilution 1/320th. Antibodies or haemagglutinating antigen were detected in fetuses by the technics previously described [3, 5]. Lungs collected from fetuses were also tested for PPV antigen by immunofluorescence microscopy [4].

RESULTS: In October 1977, the table I shows that, among the sows included in it, some of them have high titers of PPV antibodies in their sera (sows n° 3, 4, 5, 7), whereas others are not infected as no antibody is detected in the sera (sows 1, 2, 6). From December to February, a seroconversion is observed on several sows and the sow I is a good example of the phenomenon. But, numerous sows were not infected at this time as no PPV haemagglutination inhibiting antibody appeared in their sera. This case is illustrated by the sows 2 and 6. At this period, reproductive disorders (mummified fetuses) had been observed in some litters, but the problem was limited in the herd. No etiological investigation was performed. Then, all the gilts, bred in the fattening units and introduced in the breeding units since March 1978, have no PPV antibody in their sera. No seroconversion is observed in these sows in May 1978 and until December 1979 as it is shown on the table I (sows 8, 9, 10). Besides, the table 2 shows that, in proportion as young sows are introduced in the breeding units and old sows are culled, the percentage of the infected sows in the herd progressively diminishes until December 1979. This is only due to the replacement of the old infected sows by non infected gilts and not to a decrease of the level of serum antibodies. Indeed, the table I shows

clearly that HI antibodies titers are remarkably constant in the same sows during 4 years by taking account of technical variations. So, it seems that PPV haemagglutination inhibiting antibodies are persisting, without any variation, a very long time in the sera of the sows. Roughly, in January 1980 HI antibodies appeared in the sera of some previously non infected sows: 2, 6, 8, 9,10. In the herd, numerous mummified fetuses have been observed in the litters from February to May and PPV was identified in the fetuses. The sows did not show a seroconversion at the same moment; anyway, as it is shown in the table 2, 97 p. cent of the tested sows were infected in May 1980.

DISCUSSION: The results of this study show that PPV infection seemed to stop in the herd after the infectious period in December 1977. Indeed, all the gilts whose serum was free of antibodies did not show any seroconversion even after a contact with older infected sows. The epidemiological factors responsible of the spread of the virus of a herd are still unknown; some work demonstrated that the conditions of herd management could have an influence on the probability of the infection of gilts or sows from infected breeders [6]. In the case previously described, the conditions of rearing are intensive with fastened pregnant sows from the 4th week of gestation. In the service unit, the sows are unfastened by group of 3 sows, in a pen with partially stlatted areas. It is difficult to evoke these intensive conditions of rearing to explain the stop of the PPV infection as, in December 1979, the infectious episod spread very quickly in the whole herd. So, two hypothesis could be advanced in the case of this herd : - After the infectious period in December 1977, PPV disappeared form the herd which was, again, infected from the the outside, in December 1979.

- After the infectious period in December 1977, PPV was still present in animals, in a latency state, but the infection did not run its course any more, stopped by the immunity of the herd. Two years later, in December 1979, spontaneous reactivation of the virus could have been occured in the herd. The outcome of the infection could be due, at this moment, to a too low immunity level of the herd or to still unknown epidemiological factors.

SELECTED REFERENCES:[1] CARTWRIGHT, S. F. et al.: J. Comppath. 1969, 79, 371-377. [2] JOHNSON, R. et al.: Aust. Vet. J. 1976, 52, 80-84. [3] JOO H. S. et al.: Aust. Vet. J. 1976, 52, 51-52. [4] MENGELING W. L. et al.: Am. J. Vet. Res. 1976, 37, 1393-1400. [5] VANNIER et al.: Rec. Med. Vet., 1979, 2, 151-158. [6] WALTON J.R. et al.: I. P. V. S. Congress 1980, Copenhagen, 60.

TABLE 1 : Parvovirus antibodies titers in the sera of ten sows of the herd collected during 4 years

	Months and		recto in the					also also tono	
No of sows	Oct. 1977	Dec. 1977	Feb. 1978	May 1978	Nov. 1978	May 1979	Dec. 1979	Jan. 1980	May 1980
1	20	640	640	2560	1280	5120	5120	5120	2560
2	40	160	80	80	80	160	160	80	5120
3	2560	5120	2560	2560	1280	5120	≥ 10240	5120	10240
4	5120	2560	2560	5120	1280	1280	Cu.	Cu.	Cu.
5	> 10240	≥ 10240	≥ 10240	≥ I0240	> 10240	N.D.	Cu.	Cu.	Cu.
6	40	20	20	40	20	< 20	20	160	10240
7	≥ 10240	N.D.	≥ 10240	5120	5120	> 10240	Cu.	Cu.	Cu.
8	N.P.	N.P.	N.P.	40	40	20	40	40	≥ 10240
9	N.P.	N.P.	N.P.	80	40	20	160	10240	≥ 10240
10	N.P.	N.P.	N.P.	N.P.	40	20	160	80	≥ 10240
N.1	P. : Not Pre	sent - N.D.	: Not Done -	Cu. cuiled					

TABLE 2: Number of infected and non infected sows in the herd during 4 years

Mont	hs and year	S								
	Oct. 1977	Dec. 1977	Feb. 1978	May 1978	Nov. 1978	May 1979	Dec. 1979	Jan. 1980	May 1980	
Number of	23	23	29	21		9		24	30	
infected Sows	(64 %)	(70 %)	(30 %)	(40 %)	(39 %)	(29 %)	(20 %)	(61 %)	(97 %)	
Number of non	13	10	13	32	28	22	31	15	1	
infected sows	(36 %)	(30 %)	(30 %)	(60 %)	(61 %)	(71 %)	(80 %)	(39 %)	(3%)	