

## HOW HUSBANDRY PRACTICES MAY CONTRIBUTE TO THE COURSE OF INFECTIOUS DISEASES IN PIGS

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### Introduction

During the last decades a profound changing has been observed in pig production structure and techniques. Most change is a result of competitive pressures driven by the quest for better income. The new knowledge made available by animal scientists was turned into new technologies which were rapidly implemented at the different stages of the pig production chain. Pig farming practices followed the learning curve and tremendous improvements could be obtained in productivity. Hence, where a virtual average sow could wean about 15 piglets per year in 1960, the same average animal raised 25 piglets in 2000. Where average daily gain in growing-finishing pigs was barely 500 g, it is now close to 800 g; Obvious modifications came into practice in the field of nutrition, housing, herd management, prophylaxes and general hygiene routines. In the mean time the size of the herds dramatically increased looking for economies of scale and due to targeted breeding programmes, the pig itself has been modified. In this field the most visible changings are probably those related to carcass composition where leanmeat proportion has been seriously increased. All these changings did not take place without interfering with the patterns of the diseases encountered. In this paper, the contribution of husbandry practices to disease expression will be assessed.

### A brief retrospective viewing of diseases impact in parallel to intensification in western Europe.

The process of intensification of European pig production has been underway for approximately 40-50 years. The process had three different components :

- Increase in intensity. It means intensification in the use of resources. The archetype of change is the number of farrowings obtained per sow per year which has been increased mainly in relation to a reduction of lactation length and of non-productive periods.
- Increase in scale
- Farm specialisation and geographical concentration of pig production

Because these changes are interrelated they are often confused. In particular the effects of changes in scale and those of intensification "per se". With the increased pressure on the physiology of the animal, and the progressive changes in its environment it could be expected that parallel changes would be observed in disease. Prior to intensification in Europe, the major animal health problems were both endemic and epidemic (9). In pigs, whereas major diseases like CSF were cause for concern, diarrhoea in piglets, parasitism, Erysipelas and peripartum disorders were the main grounds for daily veterinary intervention. Where tuberculosis did occur in cattle, the infection could be transmitted to pigs by feeding of unpasteurized milk and dairy by-products. In addition, faeces could also contain viable tubercle bacilli, a hazard where pigs and cattle were raised together which was often the case in European family farms at that time. Similar epidemiological links were found between avian tuberculosis and pigs (6).

At the first step to intensification in Europe, the efforts directed at genetic improvements lead to large-scale animal movements sometimes over long distances without any serious concern for biosecurity. Aujeszky's disease (AD), already active in Eastern Europe (4) spread widely to the west and transmissible gastro-enteritis (TGE, (25)) decimated

thousands of young piglets during the 1960s and 1970s. In addition to these "new" epidemics, "old" diseases such as CSF (22) and FMD (in UK : 1967-68, France 1974) continued to pose problems, although now more sporadically as specific policies were adopted in different countries. In the meantime, technical guidelines for intensive livestock production were progressively learned and implemented. Changes in herd management and husbandry including hygiene and prophylaxes (e.g. vaccination, de-worming...) resulted in a profound modification of the overall animal health and disease scenario. Severe health problems related to internal parasites could be solved and endemic diseases such as Erysipelas nearly disappeared. Eradication plans were set-up and the major notifiable diseases like FMD, ASF and CSF became increasingly sporadic whilst AD tended to establish endemically in some areas. The overall situation improved regarding those well-defined monofactorial diseases, but other disorders became progressively more prevalent, particularly those related to pathogen transfer through animal trading and inadequacy of on-farm environmental conditions. Respiratory disorders for example increased in pigs kept in large groups in confined housing systems (as they did also in other species : veal calves and beef cattle). The occurrence of digestive disorders also rose (at weaning and during growing-finishing phase). It slowly became obvious that multifactorial diseases or syndromes with a quantitative expression develop on farms in cases of poor biosecurity, inadequate housing, feeding and hygiene. In certain cases only some of the technical elements of intensive livestock farming were known. However in others the preliminary knowledge was there but it was not properly used, opening the field for endemic pathogen expression (31).

The current situation in the EU can be summarized :

- Major diseases that were formerly of primary importance have been mostly eradicated. Sporadic outbreaks may occur if the related pathogens are introduced into the herds. Biosecurity including the role of the wild boar must be here worthconsidering.
- Major zoonoses (tuberculosis, brucellosis) have been eradicated, though there may be possible sporadic recurrences.
- Pre-eminence of enzootic disorders relating to inadequate management conditions and shortcomings in hygiene maintenance. Conventional enzootic disorders were present prior to intensification but they are somewhat different now and they are becoming much more apparent and detectable in the current context.
- Persistence of epidemic diseases like Influenza and emergence of new and largely unpredictable problems like PRRS in 1990 (33) and more recently PMWS (3, 27).

Figure 1 tries to illustrate the main trends in pig health issues over time. The situation regarding disease is never fixed (50). The mechanisms responsible for emerging or re-emerging diseases and the circumstances surrounding the occurrences are multiple.

### The role of husbandry on a bacterial problem: The case of enteric disorders at weaning.

Early and abrupt weaning which is the rule in intensive farming systems is recognised as a critical hurdle to the piglet. Very often enteric disorders occur after weaning and this stage is a major target for drug usage. A strong relationship was early established between the digestive

disorders (mainly diarrhoea, sometimes oedema disease) and toxigenic *E. coli* bacteria (5, 44). A limited range of the latter are involved, and studies determined the pathogenesis (43). From literature the following statements can be listed :

- Field case studies show that *E. coli* strains, especially those producing toxins, are most often (if not always) involved in the pathogenic process.
- But on the other hand :
  - Those pathogenic strains of *E. coli* can be found in non-diseased as well as in diseased pigs or herds (21, 29) as far as appropriate detection means are dedicated to the purpose.
  - Post-weaning enteric disorders as encountered in the field cannot be reliably reproduced under experimental condition solely by the administration of toxigenic *E. coli* (13, 40).

The three points clearly show that, in addition to the presence of the toxigenic *E. coli*, multiple factors are needed to induce typical disease. Several experimental trials were designed to look at the role of different elements of the environment with a special care taken to the diet (26, 51, 52). It is noteworthy that similar observations were made on the role of diet composition on the severity of swine dysentery. The soluble non-starch polysaccharides were found to play an important role on spirochaetes colonization (18).

Regarding enteric weaning problems an adverse climatic environment was also studied and found to have an impact (53). Our own observations demonstrated the complexity of the pathogenic process and the major role of the environment. Groups of piglets randomly selected on severely affected farms and moved to our experimental facilities on the day of weaning (at 4 weeks of age) did not express any significant sign of post-weaning enteric disease, whereas the weaners remaining on the home farm fell ill. In each paired-group the diet was the same at the two locations. From the various measurements that were made (including feed consumption, feeding behaviour, climate...) at the two locations of each trial, it was concluded that the difference between the absence and presence of disease was due to intricate inter-relationships between factors that were active on the farms but not in our facilities. In other words, despite being present, the toxigenic *E. coli* bacteria could not exert their harmful effect in our facilities. A survey was then designed and carried out in more than a hundred farms and the risk-factors could be obtained (34). They are shown in Table 1. They mainly relate to feed intake (prior to weaning and during the very first days post-weaning), housing, climate and hygiene. Figure 2 is an attempt to schematically build up the sequence of events taking place around weaning and it shows how husbandry can interfere (36).

#### **The role of husbandry on a viral disease: the case of PMWS.**

Post-weaning Multisystemic Wasting Syndrome (PMWS), first described in North-America (11, 27) has now been recognised in most of the pig-producing countries around the world. The laboratory investigations rapidly pointed out the regular presence of a circovirus (PCV2, Porcine Circovirus type 2) in the damaged lymphoid tissues (3, 20) whereas serological tests targeted on PCV2 antibody detection (8) showed that PCV2 was widespread in the pig population. Furthermore it was shown from retrospective studies on archival sera that the prevalence was already high far before the first descriptions of PMWS and antibodies were found in affected as well as in non-affected herds (42, 46). PCV2 genome sequencing did not reveal, to the author's current best knowledge any regular significant discrepancies among PCV2 isolates found in affected farms compared to isolates found in farms with no history of PMWS. Thus the situation is

rather confusing since on the other hand symptoms and lesions of PMWS could be obtained experimentally through PCV2 inoculation. In these experiments the virus could be recovered from the damaged tissues. However a more detailed examination of the protocols shows that the typical expression of the syndrome in growing pigs as observed in the field can hardly be obtained with the sole PCV2, inoculated in a way which is close to the expected natural one and many attempts failed. Nevertheless the role of the PCV2 in the pathogenic process is more than likely since its neutralization by vaccination with ORF2 protein could avoid the clinical signs of PMWS in experimentally infected pigs (30). At last, no other pathogen could be found to reproduce PMWS when inoculated solely.

Therefore it could be concluded at this stage that for PMWS to be fully expressed and become detrimental to productivity, PCV2 is indeed needed but specific circumstances are also needed in addition to PCV2 infection. In this context, PCV2 can be considered as an "associated cause" just as the toxigenic *E. coli* was in enteric disorders at weaning. In other words the PCV2 is involved in a chaining process finally resulting in more or less clearly expressed PMWS in certain herds, at a certain period and especially in certain pigs in those herds. PMWS can be mild and transient (54). There is growing and converging evidence that husbandry at large is making the difference.

Unless if new major developments surface from the investigations which are underway especially in the field of virology, the latter allegation can be backed to the following arguments :

- The PCV2 is rather widespread with and without PMWS and it is so probably since decades. Roughly speaking, it means that it is possible to live in peace with PCV2.
- When SPF pigs are experimentally infected by PCV2, a milder disease is obtained than for pigs of conventional health status (1). The microbial load the pigs are carrying plays a role.
- When looking in detail at husbandry in severely affected farms, shortcomings were early noticed. Recommendations were proposed and where the farmers could comply to most of them, the problem declined (37). A recent case-control survey involving 149 farrow-to-finish herds has pointed out a combination of risk-factors to PMWS expression (Table 2). These two studies emphasize the role of different components of animal husbandry, housing, hygiene, management and prophylaxes in particular. Indirectly those husbandry deviations were found to interact and facilitate concurrent pathogen proliferation like parvovirus in the growing-finishing compartment and that was found to be a further at-risk condition to PMWS.
- When compared to control herds, in PMWS herds, the profile regarding PCV2 seroprevalence significantly differed. Seroprevalence was higher in growing pigs (12-14 weeks of age) in cases than it was in the controls, attesting an earlier massive seroconversion in the cases (47).
- PMWS is horizontally transmissible (1). But sentinel PCV2-negative pigs kept since the beginning of the experiment among the inoculated pigs, started to show clinical signs only 16 days after the inoculated pigs had already clearly expressed signs of illness. A certain PCV2 pressure seems to be necessary to launch the discernible clinical signs.
- A litter effect could be observed in the affected farms. Some litters were decimated whereas others were totally spared (35). It would mean that part of the game is already played at weaning. The status of the sow herd regarding PCV2 and other infections might be involved, and subpopulations of sows in respect to health and

immune status might exist within the herds. The type of housing, of management, of general care including vaccination schedules implemented are here involved.

Taken together those listed observations allow to build up a preliminary and rather schematic diagram showing husbandry elements of the fallacious process resulting in PMWS (Figure 3). The immune system is known to be pivotal in the process (32). Specific (subtle) stimulations of the latter at critical stages during the cascade of events mentioned in Figure 3 are suspected to prepare the field for PCV2 replication that finally result in PMWS when the pig cannot handle a too heavy microbial load.

### Discussion

The role of husbandry is definitely essential in herd health maintenance. In this paper the vocable has been used in its broad acceptance to include the different aspects of animal care which fall within the farmer's responsibility. There can be debate on whether or not veterinary aspects like vaccination programmes should be considered. Our point of view was in favour of their consideration since they did not concern notifiable disease or official regulation. The same debate could concern biosecurity. The latter point was also considered since it is an element of animal health care which is, on routine basis, part of farmer's duty. The role of biosecurity is obviously of primary importance in infectious diseases and not only for monofactorial ones. Obviously the safety margin in herd health maintenance strongly depends on the pathogens being present on the farm. When the undesirable ones, responsible of notifiable diseases have been kept aside, the challenge that both the farmer and the vet are taking up is to handle properly the remaining ones. Beside vaccines and drugs, diverse technologies are now available in this respect. Segregated early weaning was developed trying to obtain cleaner piglets (2, 12, 15). Where permitted by regulation, it becomes easier to implement thanks to appropriate diets, adequate accommodation and hygiene practices, in a word thanks to well-adapted husbandry. Artificial insemination is another technology avoiding boar movements. On the other hand the possibility of pathogen shedding into semen must not be forgotten especially for viruses (10). The consequences on pathogen spread can be considerable. On the farm the conditions "enveloping" the pigs are of paramount importance. Two examples of diseases are given in this paper but several other demonstrations can be found in literature like those related to respiratory tract health. The percentage of lungs severely affected by pneumonia increased in direct proportion to the number of deviation in the climatic parameters within the pig facilities (14). High concentrations of ammonia were found to increase the severity of atrophic rhinitis (17) and pneumonia (49). A positive relationship between the mean score of lung damage in pigs and concentrations of respirable dust was found (45). The author observed the same trend for the bacteria concentration in the air of the studied piggeries. A significant negative association was demonstrated between the prevalence of enzootic pneumonia and several factors among which the number of pigs per pen and per room (higher number being unfavourable), the frequency of manure removal, the type of floor (48). Non infectious factors like type of housing were also put forward in other studies (38). The structure of the sow herd (shape of parity pyramid) and in many cases the size of the herds have been found to increase the risk of getting disease and of disease impact. This issue has been recently discussed and the main reasons pointed out (23). In this field, especially when results are compared, an adequate attention needs to be paid to the methods used to assess the herd size effect. And obviously the way the pigs are housed and managed strongly interfere

(degree and type of compartmentalization, size of rooms, of pens, age-segregated rearing, strictness of all-in/all-out hygiene policy..... In line with the purpose, in large-size operations where the microbial pressure is high, the multisite production systems showed advantages and they were promoted (28). When properly managed the multisite systems can also lead to reduce drug consumption (16). In relation to viral diseases expression, the role of husbandry is also documented. The clinical effects of PRRS virus infection vary widely among farms (55) and, the "colorful" epidemiology of the syndrome was even outlined (7). In many herds, infection is asymptomatic with no effect on productivity. Some infected herds show occasional respiratory disease outbreaks in young pigs or periodic outbreaks of reproductive disease. Only a few herds experience severe problems. Despite in the US, differences in virulence were reported in PRRS virus strains (41), the level of severity of PRRS and the type of prevailing clinical signs were found to be associated to different factors like herd size and husbandry, all-in/all-out management practice in particular (24). Reducing crossfostering was found to be beneficial during an acute outbreak of PRRS (39). On our side, in a cross-sectional study about Swine Influenza outbreaks, a broad variation was found in the severity of the clinical expression in finishing pigs depending on the level of pre-existing enzootic respiratory problems, which are known in turn, as said earlier, to highly depend on husbandry. Intercurrent respiratory tract infections have been reported for years to be complicating factors of influenza (19).

### Conclusion

Undoubtedly, animal husbandry practices contribute to disease expression. Their role is of particular importance in multi-factorial enzootic diseases. Herd health results from complex interactions, cascades of events and permanent adjustments. The introduction of a given husbandry practice can easily disturb the balance in a herd and lead to troubles albeit not necessarily through the introduction of a new pathogen. A proper control of the on-farm population of potential pathogens needs a careful attention and a good skill. The ever-ending quest for better productivity and profit, the constantly evolving technologies in animal farming and the ever-increasing exchanges in a market now open to the world, might favour exposure to hazards. New and largely unexpected so-called production or "man-made" diseases will come along after those we already experience. At the research stage, more integrative programmes should be encouraged including the different disciplines of animal sciences. Furthermore, before large-scale spread of innovations they should be backed to both experimental and epidemiological investigations, due to the role of interactions. A given practice (e.g. an ingredient of a diet, a vaccine...) may give positive results in perfectly controlled experimental conditions in the short run whereas it might induce directly or indirectly unexpected problems in certain field conditions. Another point to be mentioned is the too-rapid causative links which tend to be established from diagnostic laboratory results, between pathogen detection and disease. When further investigations are directed at the search of those pathogens in healthy herds, there can be surprises. Appropriate husbandry practices are the corner stones for good herd productivity. Despite it can challenge our peace of mind, we can add that they can also make several pathogens remain silent ! In this respect PMWS could sound like a wake-up call to give better consideration to husbandry when facing disease. In many cases husbandry needs at least the same attention as the pathogen itself.

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Table 1- The risk factors to post-weaning enteric disorders and the corresponding preventive measures

(1) Adapted from (34)

(2) Additional requirements

(3) The arrows dictate the relationship between an increase in the risk factor and the risk of disease onset.

e.g. : the risk of disease increases when hygiene declines, ..., when the numbers of litters and of pigs per pen increase ...etc...

The risk factors	Least risky value of the factor(1)	Relationship with the disorders(3)
- Hygiene status in post-weaning room at the arrival of the piglets.	All-in/all-out + perfect cleaning + empty pit below slatted floor + disinfection + dry floor + warm (24°).	↘
- Creep-feed intake/piglet last week prior to weaning (g).	> 470 g.	↘
- Air quality (throughout the 4 weeks after weaning).	NH3: < 10 ppm; CO2: < 0,15% + average air speed: < 0,10 m/sec., no draught + no turbulence of the flow + no air flowing up from the slatted floor + RH: < 85%.	↘
- Temperature (2), 1 <sup>st</sup> week post-weaning	28°C (weaning at 4 weeks)	↘
2 <sup>nd</sup> week post-weaning	27°C	↘
- Water supply (2)	Waterers: •easy to access, to operate, to maintain clean •provision of potable water.	↘
- Feed intake/piglet during the first week post-weaning.	> 1700 g	↘
- Age at weaning (days).	≥ 28 days	↘
- Live weight at weaning (kg).	≥ 9 kg	↘
- Number of litters of origin per pen (post-weaning room).	< 4	↗
- Number of piglets/pen.	< 13	↗
- Space available at the feeder (cm/pig).	≥8	↘
- Stocking density (pigs/m <sup>2</sup> )	□ 3	↗
- Severity of concurrent respiratory disorders.	- absence of cough - average sneezing counts: n < 2 (for 100 pigs, mean of 3 counts of 2 min.)	↗
- Available man power (sow/person) farrow-to finish farms	□ 80	↘
- Overall farm disease level (Post-weaning enteric disorders excluded)	A score was calculated based on : -annual mortality rate in growing-finishing pigs, -annual mortality rate in sows, -influenza-like episodes/year in growing-finishing pigs, -PRRS infection in growing-finishing pigs, -diarrhoea prior to weaning, -diarrhoea during growing-finishing phase.	↗

Table 2: Variables and variable categories in the final logistic regression model for risk factors to PMWS expression in farrow-to-finish farms (comparison « CASES » / « CONTROLS#1 »<sup>a</sup>, n= 114 farms, France, 2000-2001).

Variables	% of CASES	Logistic regression model <sup>b</sup>	
		OR	Confidence interval (90%)
PPv status of 20 week-old fattening pigs			
Negative	47.4	1.0	-
Positive (>20% positive results/farm)	76.5	4.4	1.1 ; 18.3
PRRSv status of 20 week-old fattening pigs			
Negative	32.2	1.0	-
Positive (>50% positive results/farm)	72.7	6.5 <sup>c</sup>	2.6 ; 16.1
Purchase of the semen from an insemination centre			
More than 95%	44.4	1.0	-
On-farm collection or natural mating	58.3	4.6 <sup>c</sup>	1.8 ; 11.9
Vaccination scheme against parvovirus and Erysipelas			
Only associated vaccines	37.2	1.0	-
Separated vaccines (at least for the gilts)	63.5	2.5	1.1 ; 5.9
Average pen surface in post-weaning rooms			
Less than 5.9 m <sup>2</sup>	40.0	1.0	-
between 5.9 m <sup>2</sup> and 7.8 m <sup>2</sup>	41.4	1.3	0.4 ; 4.3
More than 7.8 m <sup>2</sup>	63.6	4.1 <sup>c</sup>	1.4 ; 11.4
Duration of the empty period in weaning facilities			
Less than 4 days	64.4	1.0	-
More than 4 days	38.2	0.2 <sup>c</sup>	0.1 ; 0.6
Duration of the empty period in farrowing facilities			
Less than 5 days	64.7	1.0	-
More than 5 days	32.6	0.2 <sup>c</sup>	0.09 ; 0.7
Group housing of sows during pregnancy			
No	55.6	1.0	-
yes	42.4	0.3	0.1 ; 0.9
Treatment against external parasites of the breeding herd			
Never	51.5	1.0	-
Yes, regularly	31.8	0.1 <sup>c</sup>	0.04 ; 0.5
Number of sows per farm	-	0.99	0.99 – 1.0

<sup>a</sup>«CASES» : PMWS-affected farms; «CONTROLS#1»: Farms with no history of PMWS

<sup>b</sup>Logistic regression model : Intercept = -0.94, model deviance = 96.9, model df. = 11 (p<0.001).

<sup>c</sup>Significant also at p<0.05 (likelihood-ratio  $\chi^2$  test)

Figure 1: Schematic retrospective view of health problems affecting the pig in western Europe in relation to intensification process.

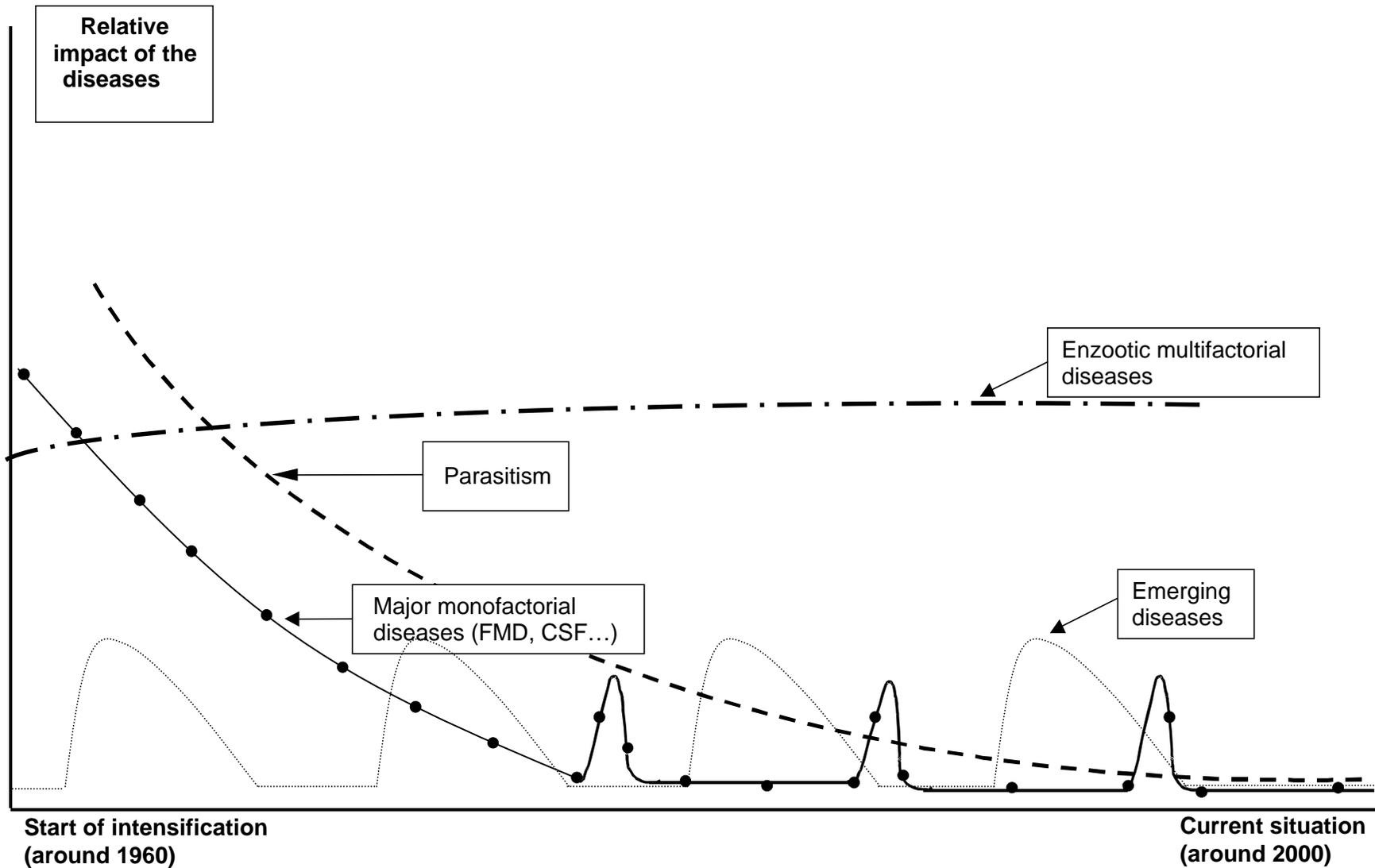


Figure 2 : The cascade of events and inter-related risk factors intervention in post-weaning enteric disorders in pigs.(36)

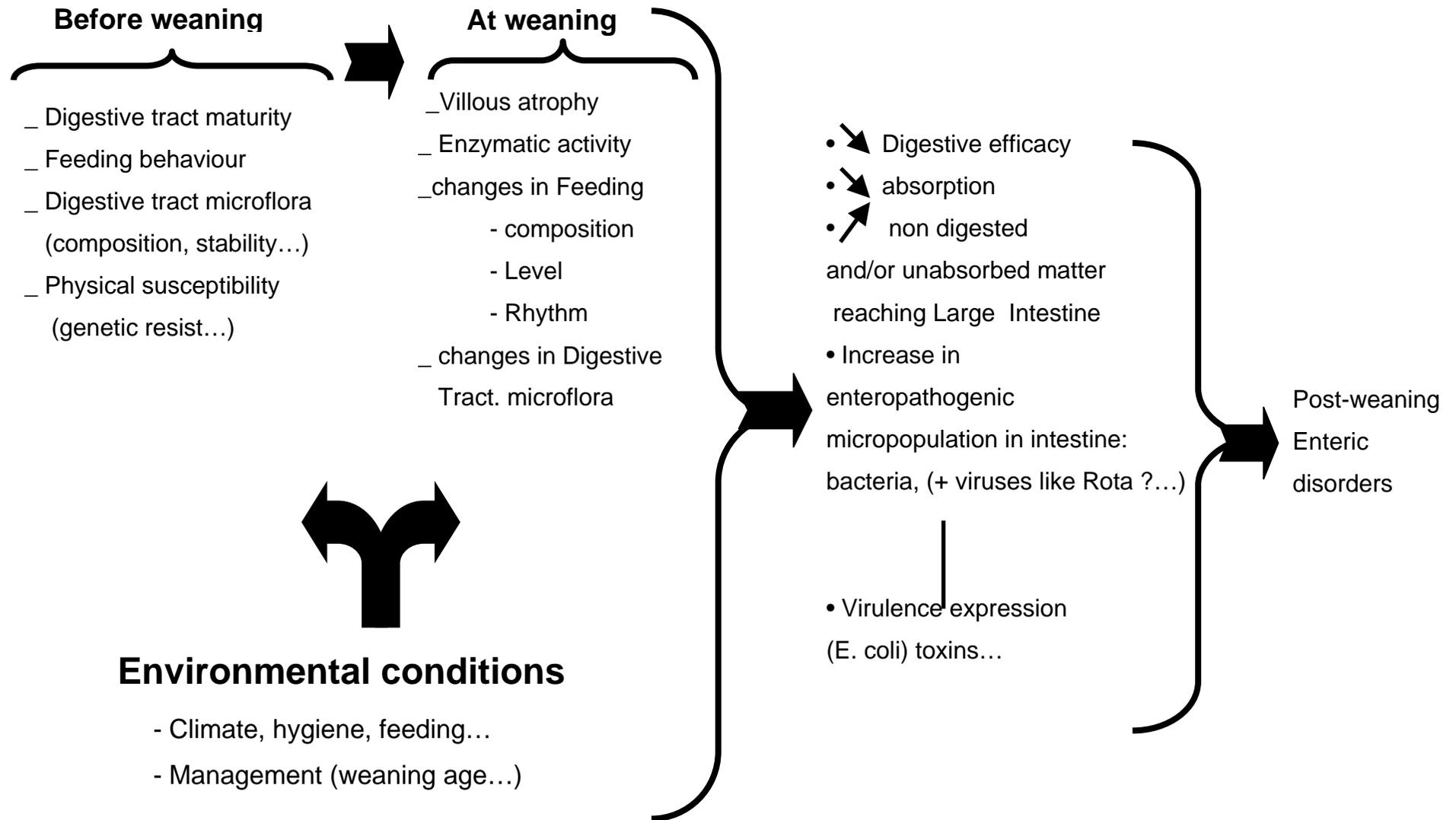


Figure 3 : Schematic preliminary diagram of the fallacious process resulting in PMWS in PCV2 infected farrow-to-finish herds

