factsheet
Pork Information Gateway

Swine Stress and Pathogen Shedding

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Background

Meeting consumer concerns about food safety and animal welfare have been identified as key points for future livestock production. In particular, meat safety has emerged as an absolute but minimum requirement for the future success of livestock and meat production. There are pathogens in raw meat that have long been traced to farm animals and can cause human illness. In swine, these primarily include *Salmonella* and *Campylobacter* species (CDC, 2000). It has been established that modern housing and management practices (mixing, handling, isolation, transport, weaning) can be stressful under some circumstances and that stress may cause decreased well-being, reduced growth, and increased disease rates through immunosuppression (Kelley, 1980). A void in current knowledge exists in the cause and effect relationship between the animal's response to stress and the shedding and transmission of these pathogens from food producing animals. There is also a lack of science demonstrating production practices that effectively decrease the likelihood of swine harboring these pathogens.

How are Stress and Food Safety Related?

The scientific literature contains many examples where "stress" is identified as a factor that increases pathogen shedding in animals (Corrier et al., 1990; Hussein et al., 2001; Isaacson et al., 1999; Schwartz, 1991). In their risk management program for Salmonella Noordhuizen and Frankena (1999) indicate some of the risk factors for presence of pathogens include transport, the environment (rodents/ birds, farm density), dust, stressors, group size and mixing of animals. In most cases, stress was not measured throughout the course of the experiments.

Possible Mechanisms

One reason anecdotal and correlative information has led us to believe that there is a direct link between stress and food safety is that "stress" is a nebulous term. While the concept of stress is readily understood, an accepted definition of stress within the scientific community still eludes us. Most of us understand that stress is an everyday occurrence and that all organisms live in a more or less stressful environment.

Stress is a normal experience for swine and is not necessarily detrimental to the animal if it is able to cope with that stressor. It is in cases where the pig cannot cope with a stress that detrimental effects are seen. We understand a great deal about the physiological stress response. This response begins by the pig perceiving that it is in a stressful situation. The hypothalamus, (a brain structure involved in the stress response) releases a hormone called corticotropin-releasing hormone (CRH). CRH begins a cascade of other endocrine and immunological changes (Moberg, 2000). What is less well understood is how factors like the genetics of an animal; its prior experiences and its perception of the stressor influence the initial release of CRH in the brain.

How the immune system responds to stress has been studied for a number of years, as it is sensitive to stressful situations. The classic view of stress has been that it is immunosuppressive. Recently it has been demonstrated that stress can enhance or suppress the immune response (Tachscherer et al., 1998). Unequivocal immunosuppressive effects of acute stress may be doubtful (Wrona et al., 2001) but there is greater consensus that chronic stress is immunosuppressive.

Research on Typical Production Stressors

Armed with information about how stress operates and how that stress can directly and indirectly affect the pig's immune system and thus it's defense mechanism against microorganisms, it is understandable why an increase in pathogens caused by stress is assumed. However, applied studies have not always indicated that this assumption is correct. Transportation is one type of production stress that has been associated with pathogen shedding. Williams and Newell (1970) demonstrated that the rate of *Salmonella* shedding in pigs, as measured by rectal isolation, increased markedly during transport from farm to abattoir, and proposed that stress precipitated the fecal shedding phenomenon. Recently, however, Hurd (2001) has indicated that stress associated with transportation of finisher pigs, by itself, did not contribute to an increased likelihood that *Salmonella* would be detectable in intestinal contents at the time of slaughter. Furthermore, Hurd et al., (2001) indicated that factors beyond stress, for example simply placing the pigs in a holding pen, appear to be more important in increased infection rates of pigs with *Salmonella*.

Jones et al., (2001) reported that weaning, mixing and cold stress in piglets resulted in increased shedding of enterotoxigeic *E. coli* (in a disease challenge model) without affecting humoral immunity. The authors concluded that increased fecal shedding of this strain of *E. coli* due to stress might not involve modulation of the immune response. These reports certainly question the hypothesis that stress induced immunosuppression is predominantly and directly associated with increased pathogen shedding. Other alternative mechanisms may exist. Weaning stress may be one example of a complex stressor with multiple changes affecting an animal. Weaning is associated with a change in diet type and volume as well as changes in the morphology of the intestine. It has recently been shown that there are also changes in the motility pattern of the small intestine of the pig following weaning to a solid diet (Lesniewska et al., 2000). CRH has been shown to have a direct effect on gastric motility indicating at least the potential for an effect of stress independent of the immune response.

Lack of food may also influence gastric function and shedding of pathogens. Following a 48-h fast, pigs (miniature) were found to have increased numbers of *Campylobacter jejuni* in their gastrointestinal tract. These differences were no longer present 5 days after the fast. No difference was seen in *C. jejuni* following a 4h transport (Harvey et al., 2001). Fasting has also been observed to increase cecal concentration of *E. coli* (Nattress and Murray, 2000) and *Salmonella* in pigs (Isaacson et al., 1999).

Conclusion

While there is evidence linking stress with pathogen shedding in pigs and swine production, lacking is research evidence demonstrating a cause and effect relationship between what happens physiologically during the stress response and what happens to growth and shedding of pathogens that are of importance in food safety. At the same time, approaches that reduce stress in swine production need to be tested for their effect on the prevalence of pathogenic microorganisms. Only by knowing the cause and effect relationship and actual mechanism involved in stress induced pathogen increases can we begin to provide swine producers with real management solutions to solve this important challenge.

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