Few things are well defined regarding runting-stunting syndrome. Starting by its very name, “runting-stunting” is poorly defined and will probably be changed in the future depending on the etiological agent or agents identified. In poultry, a “runt” is defined as an animal that is unusually small, especially the smallest of a flock. “Stunting” relates to the hindering of the normal growth of an individual. Thus, “runting-stunting” can be defined as a syndrome in which a number of individuals in a flock appear considerably small due to delayed growth. The larger the number of chickens that are unusually small, the lower the body weight average for the flock.

Runting-stunting syndrome (RSS) has been recognized for many years and was first described in the 1970s. One can think of a seemingly endless list of possible causes for RSS, including faulty poultry genetics, management, environmental challenge, feeding and nutrition, infectious disease and perhaps other reasons and any possible combination of them. A syndrome is in the eyes of the beholder... Several candidate infectious agents have been described in the past, including reoviruses and small round viruses. Although some features of RSS have been reproduced upon experimental infection with reovirus, the general consensus is that these viruses are unlikely to be the sole culprit in RSS. Small round viruses (small within the context of general virology) have been visualized many times in the intestines and intestinal contents of affected broiler chickens using electron microscopy, but such viruses can also be found in non-affected flocks or individual chickens. Bacteria have not been considered a major player in RSS, albeit intuitively one would think that bacteria must play a role at least as secondary pathogens after viruses have damaged the intestinal wall. The digestive tract is one of the most important organs from the economic point of view, and yet it is one of the least understood regarding poultry pathology. Due to the complex nature of the microbial populations in the intestine, isolating potential pathogens and sorting them out is often a daunting task. Culture methods have not been developed or are too cumbersome for some of the viruses visualized in cases of RSS. As a result, essential diagnostic assays are lacking or they are simply impractical. Additional confounding issues include the sheer fact that RSS is a syndrome and it may manifest itself in a variety of forms, it may result in different outcomes or it can simply be interpreted in diverse ways in the field. The poultry industry has used multiple intervention strategies attempting to control RSS, often without any palpable success.

During 2003-2005, RSS has caused economically significant problems at least in the Southeastern United States and particularly during winter and spring. As observed in this geographical region, RSS is a condition associated with delayed growth, increased need for discarding runts during the first 2-3 weeks of age, reduced livability, increased feed conversion and days to market, and reduced efficiency at the processing plant should the runts reach it. Some integrators have reported up to 20 points increased feed
conversion and up to 30 points lower body weights. Clinically, affected flocks show large numbers of immobile chicks huddling around the feeders and drinkers within hours after placement. Some may peck incessantly at the walls. The litter quickly becomes damp and chicks may exhibit matted down in the abdominal area as a result of resting on wet litter. Consumption of chick starter feed typically lasts a day or two longer than usual. As early as 6-7 days of age many chicks will already appear stunted, pale and sometimes disoriented, but the usual peak of the problem occurs at around 10-12 days of age. The bodies of affected chicks will look small relative to the length of the primary feathers of the wings and beak. A small number of affected chicks may display “helicopter” feathers in their wings and other feather abnormalities.

Figure 1. “Helicopter” wing feathers in a 26 day old runt (left panel). The right panel illustrates the vast range of body weights observed in a 26-day-old flock.

Surprisingly, fat soluble vitamin deficiencies have not been observed consistently. If allowed to remain in the flock, stunted chickens do not recover, although mortality may remain relatively within normal limits. However, the need for culling runts will translate usually into increased mortality as the flock is settled.

Some of the internal gross lesions or changes that have characterized recent cases of RSS include (not exclusively): small livers with a grossly enlarged gall bladder; pale, thin, almost translucent intestinal wall with wet pellets of undigested food that is visible through the intestinal wall; large amount of fluids inside the small and large intestines; infrequent feed impaction in the cloaca; occasional increased amount of pericardial fluid; sporadic white or cream-colored plaques in individual proventricular glands.

Various microscopic lesions have been described but the most frequent one is the presence of multiple cysts involving the intestinal crypts. The early lesion can described as “cystic enteropathy” and as it evolves into an inflammatory lesion it turns into “cystic enteritis”. The nature of the inflammatory responses suggests a viral etiology rather than bacterial. As the lesion progresses it may result in shortening and clubbing of the intestinal villi. Other microscopic lesions are important observations but they are not always present in cases of RSS. Viruses, nutritional deficiencies and parasites have been proposed as possible etiologies of cystic enteropathy. Until now, only deficiencies of some complex B vitamins (thiamin, riboflavin, pantothenic acid, and niacin) have consistently reproduced cystic enteropathy.
At the Poultry Diagnostic and Research Center, University of Georgia (PDRC) we have performed a series of experiments designed to reproduce the syndrome, isolate potential pathogens and identify mitigating factors:

Several experiments involving gavage inoculations with intestinal contents from affected chickens have resulted in severe weight depression. Inoculations with filtered gut contents have also resulted in RSS, suggesting that viruses may be involved since the filters used discriminate bacteria. Multiple viruses were isolated initially but most have been eliminated after further characterization revealed that they were unlikely to participate in RSS. After a substantial amount of work in the laboratory, two groups of viruses isolated from cases of RSS are currently being studied at PDRC. These involve *reoviruses* and *astroviruses* that appear to have unusual characteristics. All experiments conducted with bacteria at PDRC have failed to reproduce RSS.

RSS has also been reproduced at PDRC by placing broiler chicks on contaminated litter obtained from an affected local farm. Simple exposure to contaminated litter has resulted consistently in a dramatic body weight depression. Several groups of broilers have already been grown sequentially on such contaminated litter with control chickens grown in a separate facility on fresh litter. A very short downtime has been allowed between each growout, which has resulted in a progressive loss of early uniformity and severe depression of body weight gain. Most affected chicks display lesions consistent with RSS.
Figure 3. Blue data points in the left and right panels correspond to individual body weights of broiler chicks raised on fresh litter (150 chicks per group). The red and orange data points correspond to body weights of broilers raised on built up litter obtained from an RSS-affected farm (two replicate groups of 150 chicks each). The left panel illustrates the body weights as they were collected at 18 days of age and the right panel illustrates the body weights sorted ascending by group to demonstrate the body weight spread in each group. All chickens were produced by the same breeder flock and hatched in the same machine. Average body weight for the controls = 455.2 grams (standard deviation = 59.2); average body weight for RSS group 1 = 218.1 g (standard deviation = 93.9); average body weight for RSS group 2 = 242.1 (standard deviation = 97.8). Clearly, body weights and uniformity were severely affected in the RSS groups.

Based on the results of these experiments, it can be concluded that built up litter and short downtime may contribute to RSS in a detrimental way. Additional experiments conducted at PDRC have confirmed that proper brooding temperature is critical to mitigate early poor uniformity and delayed growth. Cold brooding induced a loss in uniformity and depressed body weight gain during the first 7-14 days of age. Furthermore, it is now known that RSS-associated agents may be controlled if exposed to heat. Experiments conducted at PDRC have demonstrated that heat effectively reduces the detrimental effects of infectious agents in the intestines of affected chicks. A practical application of these findings would be to apply heat to affected chicken houses in order to mitigate any agents associated with RSS. Thus along the lines of intervention, research performed at PDRC has suggested the following:

1. Built up litter is clearly infectious and can cause severe body weight depression and loss of uniformity.
2. Short downtime may exacerbate RSS.
3. RSS agents are clearly sensitive to heat. Thus heat treatment of affected houses during downtime is likely to mitigate RSS.
4. Proper brooding temperatures minimize the effects of RSS (delayed body weight gain and chick uniformity).

Research on RSS continues to be an important subject at PDRC and also in other research institutions. Some enteric viruses (reoviruses and astroviruses) are actively being characterized and explored as candidates for vaccines and to develop diagnostic tests. In addition, the possibility of vertical transmission of RSS and maternal immunity are now being explored at PDRC. Studying RSS in broiler breeder pullets is also another priority among the list of items being researched at the University of Georgia.