

Runting and Stunting in Broiler Chickens

Guillermo Zavala and Taylor Barbosa

Poultry Diagnostic and Research Center
Department of Population Health, University of Georgia

Summary. Runting-stunting syndrome (RSS) is a condition affecting young broiler chickens and is characterized by growth depression, decreased uniformity and poor economic performance. Affected chickens may express watery diarrhea, thin-walled and pale intestines with undigested feed in their lumina, enlarged gall bladders and relatively small livers. The feathers follow a relatively normal growth pattern but the growth of the body and muscle development is severely reduced. Microscopically, the pancreas may be infiltrated with mononuclear cells or may be atrophic and fibrotic. The intestinal crypts are severely distended and cystic and the intestinal wall may be severely infiltrated with lymphocytes and other inflammatory cells. The etiology or etiologies of RSS have never been identified, albeit a number of viruses and bacteria have been proposed. Reovirus, astrovirus and other enteric viruses as well as bacteria such as *E. coli* have been isolated from chickens affected with RSS. However, RSS has not been reproduced consistently in the laboratory by monovalent inoculations with any of these agents. Therefore, it is critical to attempt the identification of: a) Possible etiologies; and b) Effective intervention strategies. The isolation, identification, and characterization of an infectious agent would allow the development of diagnostic reagents and possibly vaccines. Effective intervention strategies would contribute to curb the detrimental effects of RSS in poultry integrations. This manuscript summarizes our efforts to investigate the pathogenesis of RSS in young broilers and to identify possible intervention strategies.

A disease model for RSS. As stated above, a variety of enteric viruses have been proposed as possible etiologies for RSS, but none have been confirmed as being directly responsible for the reproduction of the syndrome. Since we lacked a specific disease agent (or agents) to study RSS, or a laboratory approach to reproduce RSS, we have developed a simple model for reproduction of RSS by exposure to a contaminated environment. Poultry litter and chickens were obtained from a broiler house containing severely affected broilers. The affected broilers and the contaminated litter were used to seed colony houses (small enclosed isolated pens) to be used to attempt to reproduce the syndrome. The seeder chickens were maintained for 2 weeks in the colony houses and then euthanatized and replaced with a second batch of day-old broiler chickens. The second batch was grown for 2 weeks and then immediately replaced by a third batch of day-old broiler chickens. This approach was repeated 18 times with no significant downtime between batches of flocks. The body weights and intestinal lesions of the exposed chicks were compared each time to the weights and intestinal lesions of hatchmates grown in a clean and disinfected colony house. This system has been able to

reproduce consistently the clinical signs, lesions and severe body weight depression commonly observed in field cases of RSS. Every growout was done always with progeny from a single young donor breeder flock, most frequently from a high-yielding, slow-feathering breed cross. This model has been able to consistently induce approximately 50% body weight depression, severely decreased uniformity, and a high frequency of intestinal lesions that are typical of RSS. This RSS reproduction research model has been used to study pathogenesis, possible intervention strategies, application of autogenous inactivated vaccines for use in broiler breeders, and possible etiologies. Using this model (and complementary *in vivo* and *in vitro* assays), the following conclusions have been produced at the University of Georgia.

Body weights are severely depressed. The most dramatic impact is a severe growth depression observable as early as 6 days of age. By 14 days of age, exposed chickens weigh as little as 50% of the control unexposed chickens.

Uniformity is severely depressed. One of the most relevant features of RSS in the field is a severe loss of uniformity. In our research model, the 14-day-old body weight uniformity of control unexposed broiler chickens averages approximately 70%, in contrast with approximately 35% uniformity in the RSS-affected chickens.

Cold brooding enhances RSS. Early growth, body weight and body length uniformity are adversely affected when early brooding is done at suboptimal temperatures. Rearing broiler chicks at 85°F instead of 94°F translated into lower body weights and lower uniformity by 14 days of age. Today's higher yielding breeds require less heat in incubation and more heat in the brooding period.

Various breeds and genetic levels are susceptible. We have tested the 3 most popular 3 breed crosses in the United States and all breeds or breed crosses are susceptible. However, certain high-yielding, slow-feathering breed crosses were affected more severely than others. We have also tested grandparent off-sex "byproduct" which was also susceptible, demonstrating that more than one generation can be affected by RSS.

Males of high-yielding breeds are more severely affected. Male broilers are consistently more severely affected than females. This applies for chickens as early as 10 days of age and later ages.

Mortality and lesions peak at 6-12 days of age. A peak in mortality with RSS-associated lesions is frequently observed at 6-12 days of age (depending on many poorly understood factors). Generally, intestinal lesions associated with RSS tend to be more severe during the same age range. Mortality involves more male broilers over females, but female broilers are also involved. RSS-related lesions usually tend to diminish in severity and frequency after 14 days of age and most gross lesions tend to disappear by 21 days of age. However, microscopic lesions can remain detectable as late as 35 days of age in field chickens that have experience RSS at an earlier age.

The effects of RSS may be mitigated with increased downtime, cleaning and disinfection and house heating. Results of our experiments have suggested that: a) RSS is exacerbated in flocks raised on built up litter; b) short downtime facilitates RSS expression and severity; c) house (and litter) heating at least 100°F for 100 hours mitigates the detrimental effects of RSS; d) cleaning and disinfection effectively prevents RSS from significantly affecting broilers, and certainly prevents expression of microscopic RSS-related lesions.

Antibacterial and antiprotozoal drugs have no effect. Sulfa drugs and other antibacterial drugs have been unsuccessful in the field. Experimentally, metronidazole administered at a therapeutic dose to broilers was also unsuccessful. Metronidazole was used experimentally only to determine if this antiprotozoal and anti-anaerobic bacterial drug would mitigate the effects of RSS, but certainly not to use it in the field. Metronidazole has antibacterial and antiprotozoal effects, especially against *Clostridium*, *Fusobacterium*, *Peptococci* and *Peptostreptococci*, as well as *Giardia*, *Entamoeba* and *Trichomonas*. Metronidazole failed to reduce the negative impact of RSS on body weight depression, uniformity loss, mortality or RSS-related lesions. Work performed at Iowa State University has shown that the use of virginiamycin produced some benefits in turkeys expressing PEMS (a similar syndrome affecting turkeys). However, virginiamycin has not been tested recently in university trials involving broiler chickens.

Role of maternal antibodies against CAV, IBDV and Reovirus. Experiments performed at the University of Georgia have demonstrated that there is no significant correlation between maternal antibody titers at hatch and body weights attained at 10 days of age. This was observed for CAV, IBDV and reovirus. Thus, it is unlikely that any of these three disease agents may play a direct role in the expression of RSS in young broilers. However, it is logical to think that adequate protection against any or all of them constitutes a sound criterion for indirect protection against any other infectious agent.

Etiological agents. Numerous agents have been reported as possible candidates. In our experience, field samples and in-house experiments have revealed a variety of viruses being present in the intestinal contents of chickens affected with RSS, including reovirus, enterovirus-like particles, rotavirus and astrovirus. Most of these (with the exception of reovirus) are difficult to propagate in the laboratory and thus it is difficult to demonstrate their role in RSS, and to develop diagnostic reagents. Unusual reoviruses have been identified recently at the University of Georgia and are currently being evaluated. However, at least one of such reoviruses has been characterized, purified and used as an autogenous inactivated vaccine for broiler breeders. Early evaluations have suggested that there was no significant benefit from the use of such reoviruses as autogenous vaccines, but further research is needed to conclusively evaluate such vaccines.

RSS is currently regarded as one of the most economically important diseases affecting the broiler industry in the United States. The etiology or etiologies are not known, but certain simple intervention strategies may contribute to mitigate the detrimental effects of RSS and include: a) avoid cold brooding; b) cleaning and

disinfection of at least the brood chambers in rearing may diminish the effects of RSS; c) adequate downtime of at least 12 days may contribute to mitigate RSS; d) in operations that use built up litter, heating the houses and the litter to at least 100°F for 100 hours may reduce the negative impact of RSS; e) avoid the use of built up litter in operations where RSS has become a significant problem; f) in-house composting of contaminated litter before its reutilization may prove highly beneficial, although this has not been evaluated yet.