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CAUSES AND CONTROL OF SPONTANEOUS CARDIOMYOPATHY OR ROUNDHEART DISEASE IN UTAH TURKEYS

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INTRODUCTION

Circulatory disturbances in turkeys are likely to become increasingly prevalent because of the economic need to continue to produce fast-growing strains of turkey. The consequences of breeding for rapid growth have undoubtedly affected the occurrence of circulatory-related mortality in commercial turkey flocks. Spontaneous turkey cardiomyopathy (STC), also known as roundheart disease, is a prevalent circulatory disturbance afflicting turkeys raised at moderate to high altitudes. A condition in turkeys with enlarged hearts was first described in 1962. Since then possible genetic, management factors, and nutritional causes of the round-heart syndrome have been implicated. The objective of this fact sheet is to briefly describe STC as seen in Utah, and to propose possible ways to minimize economic loss associated with this disease.

Most commercial flocks of turkeys raised in Utah are grown at an elevation of 5000 to 5600 feet above sea level. Average flock mortality caused by STC is 1.5% to 3.0%; however, losses range from 0% to greater than 15%. At least 60% to 80% of all flocks placed yearly in Utah experience some degree of STC-associated mortality. Generally, hen flocks have a higher incidence of STC than toms. It is conservatively estimated that STC costs the Utah turkey industry \$250,000 per year.

Spontaneous turkey cardiomyopathy in Utah is seen at two major periods. The first occurs early in brooding between 10 and 17 days of age. The prevalence and incidence rates are greater in hen flocks at this age. Poults that succumb at this stage are usually large with well-developed musculature. We have shown that in a typical two-week old hen flock with 2% STC mortality, the actual prevalence of turkeys with non-lethally damaged hearts may be as high as 80% to 90%. The second spike of STC mortality is seen during late brooding and early growout. Toms four to eight weeks old are most likely to show this type of mortality pattern. Usually the dead turkeys are smaller than the flock average. Sometimes mortality continues at a low rate for weeks after the initial spike. Examination of these turkeys reveals that the heart damage occurred long before actual death—probably during early brooding or before.

CONTRIBUTING FACTORS

Altitude is a definite predisposing factor. Researchers in Canada have reported that STC can be induced in chambers mimicking high altitude conditions (1). Most turkeys in Utah are raised in the Sanpete Valley, where the average elevation is approximately 5500 feet above sea level. Our field studies

indicate flocks in brooders with an oxygen level less than 20.3%^a are at greater risk of experiencing significant roundheart mortality. We have shown that turkeys hatched from the same flock but raised at different altitudes will exhibit very different responses to STC. The poult raised at approximately 500 feet above sea level showed no clinical signs of STC, yet flockmates kept at 5500 feet had significant STC mortality (2).

The time of year when turkeys are raised also plays a significant role. Broods placed in winter have a higher seasonal index^b of STC than those placed in late spring and summer (Figure 1). Brooder buildings are more likely to have suboptimal air exchange during colder times of the year because of a tendency to conserve heating costs. However, not only is oxygen availability likely to be lower in under-ventilated brooders, but levels of carbon monoxide and carbon dioxide also tend to increase. Carbon monoxide is produced by incomplete combustion of stoves and through exhaust of internal combustion engines. Our research indicates that exposure over a period of at least a few hours to carbon monoxide above 25 to 30 parts per million (ppm) may increase the risk of a STC outbreak.

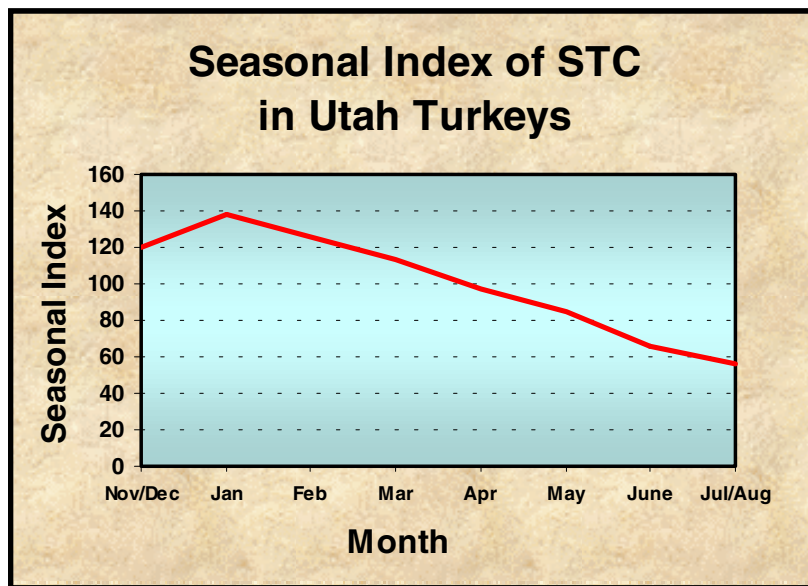


Figure 1. Occurrence of STC in Utah in relation to time of year when flocks are placed.

Carbon dioxide is of special concern. Levels above 2500 ppm have been linked with significant STC mortality. Carbon dioxide is formed through normal combustion of stoves and poult respiration. It is heavier than air and will displace it from low areas. A buildup of carbon dioxide will accumulate in the building at poult level if adequate air circulation is not maintained. Because carbon dioxide is normally exhaled by the poults and is formed as a by-product of combustion, it is always present in an enclosed brooder building. Outside air contains about 300 ppm carbon dioxide. Typical brooder readings in our studies range from 700 to 1500 ppm. Levels fluctuate freely, and vary considerably from one area of a brooder to another. A characteristic of roundheart mortality is that more turkeys die during the night. A possible explanation for this is that microenvironments form as the poults bed down and crowd together. Carbon dioxide builds up and oxygen availability lessens. If air movement in the building is impaired or reduced, these pockets of low oxygen/high carbon dioxide don't disperse rapidly enough to keep sufficient fresh air available. We have obtained carbon dioxide readings over 3000 ppm in clusters of

a In relation to monitoring equipment zeroed to 20.9% O₂ immediately outside the brooder building.

b A seasonal index is expressed as a percent deviation from one. A seasonal index of 100 means the actual incidence is equal to the expected incidence for that period. For example, if the seasonal index were twice the average for that month it would be 200%.

poults crowded together, yet one foot away the level was only 900 ppm. Interior circulation fans properly placed and operated can help alleviate dead air pockets.

Slowing the growth rate during the first four weeks will decrease incidence of STC. We have studied the application of low protein and reduced salt (NaCl) diets in the control of STC mortality. Feeding a less than conventional protein level for the first three weeks decreased STC losses, but at the same time significantly depressed growth rate. It may not be economically worthwhile to use lower than recommended protein level because the decreased mortality did not offset the lost weight gain. Subsequent studies using a reduced salt (NaCl) diet during the first three weeks of life significantly decreased STC mortality while only slightly inhibiting growth rate (Table 1). Particularly in hens, the low salt groups out performed the controls by yielding more pounds of marketable product at processing age. The greater livability offset the slight weight gain reduction.

Table 1. Percent mortality attributable to STC by field trial, treatment, and gender through three weeks of age. (3)

Trial Number	Gender	Diet (% Added NaCl)	STC Mortality (%)
1	Hens	0.200	6.5
		0.150	0.7
2	Toms	0.200	1.0
		0.175	0.0
3	Hens	0.200	3.6
		0.175	1.4
	Toms	0.200	1.8
		0.175	0.6

CONTROL

Various ways to reduce losses from STC have been identified. Not all work in every circumstance, and some require very careful management by the grower.

1. Ignite stoves at least 12 hours before poults arrive to be sure they are burning cleanly. Check for proper combustion of methane or propane brooder stoves. Flame should burn mainly blue without accompanying smoke or excessively yellow flames.

2. Make sure air is circulating within the brooder building. Ceiling or horizontally mounted circulation fans should be used soon after placement of the brood. Start them at a very slow speed (just enough to keep the air tumbling but no noticeable breeze at poult level). Adjust fan speed accordingly as poults get older and can tolerate more air movement.

3. Air exchange is just as critical as air movement. Poults should receive a minimum of 0.2 cubic feet per minute (cfm) of *incoming* air at placement. Calculate how much fan capacity is necessary to satisfy the cfm per poult need. Then, turn on exhaust fans at least 12 hours before poult arrival in order to stabilize the environment. Static pressure should be maintained between 0.05" and 0.08" of water column. Adjust fan cycles according to temperature *and* air needs—not just temperature. During cold months an economic evaluation of fuel cost vs. air exchange rate is recommended to achieve optimum efficiency. However, keep in mind that some sacrifice in energy cost may be necessary to assure adequate fresh air.

Although increasing air exchange rate reduces STC mortality, we still encounter flocks (particularly hens) that exhibit significant loss even when the air exchange rate exceeds 0.2 cfm per poult. Perhaps the negative pressure generated by running exhaust fans (which would slightly decrease the partial pressure of O₂) further affects O₂ availability to the poults. We intend to research this concept in greater detail.

4. Poults should not be unnecessarily disturbed during the second and third weeks of life. Through prevalence studies, we discovered that only a few poults with abnormal hearts actually die. In a flock with 2% mortality caused by STC, it is likely that 80% to 90% of the poults have some degree of heart damage. This suggests that the way we handle a flock, even after STC mortality starts, may have a dramatic impact on how many birds remain alive until the body can compensate and get them through this critical period. To limit poult activity, circles should be combined gradually. Some growers have found that combining two circles before completely turning the poults loose has controlled STC to some extent. Another method is to take the wire circles and "S"-shape them up and down the length of the building when the poults are turned loose. This doesn't inhibit the caretaker from freely moving around inside, yet it gives the poults the illusion they are still partially penned up.

5. Minimize the risk of turkeys becoming ill with poult enteritis. Caked litter around feeders and waterers should be removed daily for at least the first 10 days. This will help keep bacterial numbers from building up to a point where the ability to fight off infection is overwhelmed. A risk study conducted in Utah turkeys indicated that a flock contracting poult enteritis within the first 10 days of life had three times greater risk of experiencing substantial STC mortality at two to three weeks of age than an age-matched flock that had no earlier outbreak of poult enteritis. When other risk factors were also considered (i.e., gender and season), the probability dramatically increased. For example, the risk of suffering significant STC loss was 21 times greater for a flock of hens brooded in winter with poult enteritis compared to a tom flock brooded in July with no poult enteritis.

6. Keep poults from becoming chilled or overheated. Overheating will cause an increase in metabolic rate, which in turn creates a higher demand for oxygen. Chilling also increases the body's demand for oxygen. Lighting stoves at least 12 hours before poult arrival will help warm the room and bedding.

7. Light reduction programs through three weeks of age have shown beneficial results. Facilities and experience need to be considered. Darkening a brooder should never be done at the expense of compromising air movement and/or air exchange.

8. Feeding a reduced salt diet for the first two to three weeks of life may be beneficial, particularly where consistently high STC mortality is encountered.

Spontaneous turkey cardiomyopathy is an extremely prevalent disorder afflicting turkey production in Utah. Because of the variability in incidence, multiple factors undoubtedly contribute to clinical field outbreaks. Not all factors are under direct control by the grower, such as the elevation at which turkeys are raised. This fact sheet outlines approaches that might be employed from the live production end to minimize losses. Future research will be directed at further reducing the economic damage caused by STC.

REFERENCES

1. Julian, R. J., S. M. Mirsalimi, L. G. Bagley, and E. J. Squires. Effect of hypoxia and diet on spontaneous turkey cardiomyopathy (round-heart disease). *Avian Dis.* 36:1043-1047. 1992
2. Frame, D. D., F. D. Clark, R. E. Warnick, R. A. McMillan, S. E. Poe, and G. L. Anderson. Observations on morbidity, mortality, and selected pathologic features associated with spontaneous cardiomyopathy or roundheart disease in turkeys. (unpublished data).
3. Clark, F. D., D. D. Frame, R. E. Warnick, E. J. Kelly, and B. L. Roeder. Effects of lowered sodium chloride on spontaneous cardiomyopathy mortality in turkeys. *Proceedings of the 44th Western Poultry Disease Conference*, pp. 22-23. 1995.

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