

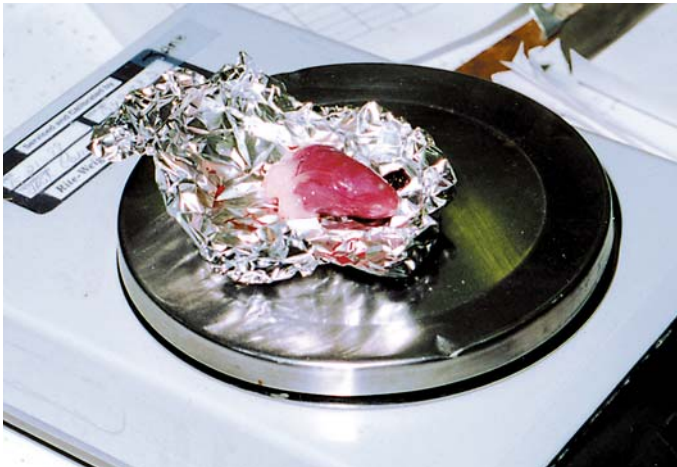
# Chicken Model May Provide Clues for PROBLEMS

Marco Nicovich



*Jeanetta Tankson, left, who recently earned her Ph.D. at Mississippi State, and Davis Wilbourn, a graduate student, harvest chicken hearts to screen for bacterial species in the organs.*

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*Researchers weigh a heart as one measurement of pulmonary hypertension syndrome development after infection with *E. faecalis*.*

*By Charmain Tan Courcelle*

Work conducted by a research team at Mississippi State University suggests that chickens and humans have more in common in matters of the heart than might first meet the eye.

MAFES poultry scientist J. Paul Thaxton and former MSU graduate student Jeanetta Tankson have found that *Enterococcus faecalis*, a common bacterial inhabitant of animal and human intestines, is a cause of pulmonary hypertension syndrome in chickens. The researchers also discovered that chicken and human sufferers of this disease share similar symptoms, leading them to believe they have an animal model that may provide answers to the development of this condition in humans.

Pulmonary hypertension syndrome is marked by a prolonged buildup of pressure in the pulmonary artery that results from an obstruction in the small blood vessels of the lung. The blockage leads to an increased resistance to blood flow from the heart to the lungs. To overcome this resistance, the right ventricle of the heart works harder to push enough blood through the lungs. Over time, the overworked right ventricle becomes weak and damaged, and eventually can fail, leading to death.

About 8 percent of chickens reared in the U.S. are affected by pulmonary hypertension syndrome every year. The annual cost to the industry worldwide due to this condition has been estimated at \$1 billion. Chickens with pulmonary hypertension can develop ascites — an accumulation of fluid in the abdominal cavity, called “water belly” — and either die prematurely or are condemned at processing.

“A number of theories have been put forward to explain the cause of this condition,” Thaxton said. “Some people said it results from birds being fed too much and selected for rapid growth. Others said it’s due to inadequate ventilation and exposure to ammonia. We don’t disagree with any of these explanations;

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however, we propose that another cause of pulmonary hypertension syndrome is bacterial invasion.”

Working with MAFES poultry microbiologist Yvonne Vizzier-Thaxton, the team isolated bacteria from the heart and lungs of chicks from early embryonic stage to three weeks of age. They then analyzed the samples for the types of bacterial species present and their numbers.

“Of the 41 different bacterial species we identified in the hearts and lungs of chicks, only one, *Enterococcus faecalis*, was present at every single sampling time in either the heart or lungs, or both organs simultaneously,” Thaxton said.

Overall, the scientists found that the heart and lungs of young chicks only transiently harbored bacteria, including *E. faecalis*. But they chose to focus on *E. faecalis* as a possible cause of pulmonary hypertension syndrome because it can become pathogenic under the right conditions.

“*E. faecalis* can cause endocarditis (inflammation of the innermost layer of the heart’s valves) if it inadvertently enters the circulation and infects the heart,” Thaxton said. “That made this bacterium a logical candidate for a cause of pulmonary hypertension in chickens.”

The researchers tested this theory by looking for the incidence of damage to the right ventricular wall — cavity formation — in chicks infected with three doses of *E. faecalis*.

“About 90 percent of birds challenged with *E. faecalis* in this study developed visual signs of pulmonary hypertension syndrome, including a depression in the external wall of the right ventricle,” Thaxton said.

The study confirmed that *E. faecalis* is one cause of pulmonary hypertension syndrome in chickens. In other experiments, the scientists wanted to gain a better understanding of pulmonary hypertension by studying the changes to the heart and lungs of birds experiencing this condition.

Thaxton and his colleagues assessed the physical characteristics of the heart and lungs of chickens infected with *E. faecalis* — weight, length and diameter of the heart; the thickness of right and left ventricular walls; right and left ventricle weight and right and left lung weight — and compared them with heart and lung measurements from control birds. They also evaluated the cellular and tissue structure of the heart and lungs.

The group found *E. faecalis*-induced pulmonary hypertension caused the right ventricle to increase in size (hypertrophy). They also observed changes to the structure of the lung’s blood vessels in chickens infected with *E. faecalis*, including epithelial cell injury and death and pulmonary congestion, which together with hypertrophy are common hallmarks of primary pulmonary hypertension in humans.

In humans, primary pulmonary hypertension is a diagnosis of exclusion, meaning other lung and heart diseases are ruled out before this condition is confirmed. Because of this, diagnosis of pulmonary hypertension often comes too late, when severe or fatal symptoms are present.

Thaxton said he hopes the chicken model of pulmonary hypertension syndrome will provide an understanding of disease progression in humans.

In addition, his group is working to develop a detection system for pulmonary hypertension syndrome in live chickens. The scientists have found decreases in the levels of protein and cholesterol in the serum (the liquid portion of blood) and increases in percentages of certain immune cells (monocytes and basophils) are good indicators of pulmonary hypertension syndrome caused by *E. faecalis*.

These results could eventually lead to a diagnostic tool for pulmonary hypertension syndrome, which would go a long way in helping the poultry industry limit losses to this condition, Thaxton said.