High-altitude disease or high-production disease? - Part 2

By **Joe Neary, MA, MS, Colorado State University** January 07, 2014 | 12:37 pm EST

A widespread misconception is that bovine pulmonary hypertension (BPH) is only problematic at high altitude; this is not true! The second leading cause of mortality behind pneumonia in dairy heifers on one facility in northern Colorado at the modest

Bovine pulmonary hypertension, commonly called “high-altitude disease,” can occur at moderate elevations and even in feedyards at well below 5,000 feet in elevation.

altitude of 5,250 feet was identified to be BPH. Multiple feedlots in Nebraska, at approximately 4,000 feet, have reported BPH to occur with an incidence of up to 3 percent. The disease has also been anecdotally reported to occur in feedlots at 3,000 feet in Montana and the Texas Panhandle. Pulmonary hypertension is highly costly to the feedlot producer as it typically occurs within a few months of finishing after considerable expense has been invested in the animal.

In humans, pulmonary hypertension is a syndrome consisting of five distinct disease categories, of which alveolar hypoxia represents one of those categories. It is likely that the pathogenesis of BPH is more complex than we have believed up to now. Pulmonary hypertension in broiler chickens was originally believed to be a high-altitude disease problem when it was first reported to occur in flocks at altitudes over 10,000 feet in the 1960s. By the 1990s, pulmonary hypertension was reported to occur in fast-growing chickens at sea level. Due to the short generation interval and high selection intensity in the poultry industry, the disease went from being a high-altitude curiosity to a disease associated with high production that is now estimated to cost the broiler industry $1 billion per year.

An extensive amount of research has been undertaken on avian pulmonary hypertension. It is now widely accepted that cardiopulmonary insufficiency is the leading risk factor for pulmonary hypertension in broiler chickens. A broiler’s risk of pulmonary hypertension is positively associated with growth rate and oxygen demand. Insufficient cardiac reserve and/or insufficient pulmonary capacity to accommodate a high cardiac output are believed to predispose fast-growing

broilers with high oxygen requirements to right-sided heart failure. Poultry producers can reduce the risk of pulmonary hypertension in their flock by controlling feed consumption. Altitude exacerbates the imbalance of oxygen supply and demand, which is why the disease was first identified in flocks at high altitude.

In the 1960s and 1970s, studies of bovine pulmonary physiology indicated that, like broiler chickens, European cattle also have small cardiopulmonary systems for their body size and oxygen requirements. The alveolar surface area of the bovine is approximately half of what it should be for a mammal of equivalent size. Our research indicates that, like fast-growing broiler chickens, cardiopulmonary insufficiency may be a risk factor for BPH. Cardiopulmonary insufficiency is more likely in calves because their pulmonary system is not functionally mature until approximately 1 year of age. It would be foolhardy to ignore the parallels between avian and bovine pulmonary hypertension.

Combating an old problem with new technology

Over the last 100 years, since the discovery of brisket disease, we have learned a lot but must do more. We do not yet have the solution to this disease and must find a way to decrease the incidence of pulmonary hypertension within the cattle industry. For my doctorate studies at Colorado State University, under the mentorship of Drs. Frank Garry, Milt Thomas, Tim Holt, Mark Enns, Paul Morley and Chris Orton, we have been studying the epidemiological, physiological and genetic risk factors for pulmonary hypertension in beef cattle. We have been collaborating with Dr. Kurt Stenmark’s research group at the University of Colorado Health Sciences Center, Denver, in order to understand the complex cellular and genetic changes that occur within the cardiopulmonary system of calves. Pulmonary hypertension affects up to 100 million humans worldwide both as a primary disease and as a consequence of other diseases, such as obesity and parasitism. Because calves are one of the best animal models of pulmonary hypertension in humans, an added benefit to our ongoing collaborative research could be an improvement in the treatment of humans for this condition.

Sidebar: Limitations to the PAP test

The pulmonary arterial pressure (PAP) test is a diagnostic test but is primarily used as a screening tool in cattle since mean PAP (mPAP) is considered to be a moderate to highly heritable trait. In brief, flexible saline-filled catheter tubing is passed through a large bore needle inserted into the jugular vein down through the right atrium, into the right ventricle, and then into the pulmonary artery. A pressure transducer connecting the catheter to an oscilloscope provides a reading of the mean, systolic and diastolic pulmonary artery pressures. The jugular vein, atrium, ventricle and pulmonary artery have distinct pressure waveforms from which it is possible to determine catheter placement.

The failure rate of the PAP test increases with altitude. For this reason an mPAP result obtained at high altitude is more informative than an mPAP result obtained at low altitude. An mPAP value obtained at an altitude of 5,000 feet does not predict what that animal's response will be at 10,000 feet. If the mPAP was high at 5,000 feet then the animal should be taken to a lower altitude. However, if the PAP was average or low, this does not mean that the PAP will be average or low at a higher altitude.

Editor's note: Part 1 of this series, titled "Bovine pulmonary hypertension: 100 years of heartache," appears in the November/December 2013 issue of Bovine Veterinarian and is available online at bovinevetonline.com.

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