Public Abstract

First Name:Raja

Middle Name:S.

Last Name:Settivari

Adviser's First Name:Donald E.

Adviser's Last Name:Spiers

Co-Adviser's First Name:

Co-Adviser's Last Name:

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Title:Temporal Effects of Fescue Toxicosis and Heat Stress on Rat Physiology and Hepatic Gene Expression

Fescue toxicosis is caused by consumption of tall fescue grass infected with a fungus, Neotyphodium coenophialum. It affects many animal species, with estimated annual loss to the US cattle industry exceeding $600 million. Animal responses to fescue toxicosis depend on ambient temperature. The present studies used a rat model to identify time-related changes associated with fescue toxicosis (E+) under short- and long-term exposures to comfortable thermoneutral (TN) and heat stress (HS) conditions. Short-term E+ decreased feed intake and growth rate; whereas long-term exposure resulted in adaptation with greater recovery at TN. Core temperature during E+ did not change at TN, but increased above normal during HS. Short-term E+ at TN decreased serum glucose, urea nitrogen, alkaline phosphatase, and cholesterol; whereas long-term E+ under these conditions resulted in adaptation. In contrast, only long-term E+ at HS decreased these serum parameters. Serum prolactin was decreased with E+ under all conditions. Antioxidant gene expression decreased with E+, with even greater reduction during HS. Long-term E+ and HS increased gene expression of enzymes that breakdown toxins. Genes associated with immune response increased with long-term E+ at TN, but decreased at HS. Recovery observed with fescue toxicosis at TN could be attributed to increased gene expression for detoxification and immune response, whereas decreased antioxidant and immune response genes could contribute to distress associated with fescue toxicosis during heat stress.