## **ABSTRACT**

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**Thesis title:** Astaxanthin and postbiotics – two promising agents to increase insulin sensitivity

in equine metabolic syndrome

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Equine Metabolic Syndrome (EMS) is an increasingly common endocrine disease caused by overfeeding horses with high-energy feeds, leading to obesity and insulin resistance. EMS is defined as a constellation of clinical symptoms such as: (i) insulin resistance, (ii) obesity in most cases, (iii) past or chronic laminitis, (iv) systemic and local inflammatory reaction and increased oxidative stress and hepatitis. Previous studies have shown that the liver of horses suffering from EMS is characterized by steatosis, overexpression of markers modulating the response to unfolded proteins, which lead to stress-dependent apoptosis of the endoplasmic reticulum.

This dissertation aimed to evaluate the use of astaxanthin as a therapeutic agent in restoring metabolic balance in EMS stromal stem cells by targeting abnormal oxidative stress and mitochondrial dysfunction. Effects of astaxanthin on cell viability, apoptosis, reactive oxygen levels, mitochondrial dynamics and metabolism, as well as its antioxidant capacity were assessed. The second analyzed research hypothesis assumed that the pre-incubation of insulin resistance induced HepG2 cell with a pro and postbiotic emulsion, would protect cells under normal and IR conditions from apoptosis, alleviate oxidative stress, as well as improve metabolism and dynamics of mitochondria, and reduce pathway activation Fetuin A / TLR4 / JNK / NF-κB. The results suggested that astaxanthin improves the metabolic status of equine ASC affected by metabolic syndrome. At the same time, the pro-and post-biotic emulsion is protective against inflammation, obesity and hepatic insulin resistance.

In conclusion, antioxidants in combination with microbiome stimulators may constitute a new dietary and therapeutic approach. The obtained results are the basis for the creation of potential biopharmaceuticals for the prevention of metabolic syndrome in horses.