ASPALATHUS LINEARIS, COMMONLY KNOWN AS ROOIBOS, IS WELL KNOWN FOR ITS VARIOUS HEALTH PROPERTIES, SOME OF WHICH INCLUDE ANTI-OXIDANT AND STRESS-LOWERING ACTIVITIES. ROOIBOS IS ALSO RICH IN POLYPHENOLS AND IS THE ONLY PLANT KNOWN TO CONTAIN THE PHENOLIC ANTIOXIDANT, ASPALATHIN.

The aspalathin content of the rooibos plant varies depending on a number of factors, and its level in the plant material drops even further during the fermentation process due to oxidation needed to form the characteristic red-brown colour and aromatic flavour.

Unfermented (green) rooibos, which is not oxidised, contains much higher levels of aspalathin than the fermented rooibos, the product commonly prepared as a herbal tea.

The SAMRC and the Agricultural Research Council (ARC) have developed a method for the production of aspalathin-enriched green rooibos extract (GRT Extract), containing a minimum of 12% aspalathin, and having a number of beneficial effects in the management of conditions linked to glucose and lipid metabolism. The beneficial effects of aspalathin and aspalathin-rich green rooibos extracts (e.g. Afriplex GRT) are based on current and previous research conducted by the SAMRC and other organisations.

**BENEFITS AND ADVANTAGES:**

- Glucose Lowering Effect
- Ameliorated Insulin Resistance in Vitro
- Protects Pancreatic Beta Cells Against Oxidative Stress
- Protects Heart Cells and Lowers Cardiovascular Risk Factors
The proof of concept for aspalathin and aspalathin-rich green rooibos extract as an anti-diabetic and anti-atherogenic agent comes from both local and international studies. Kawano et al. (2009) reported that aspalathin increases glucose uptake in muscle cells and insulin secretion from pancreatic \( \beta \)-cells.

In our laboratory, we confirmed the hypoglycaemic activity of an aspalathin-rich green rooibos extract in vitro and in vivo (Muller et al., 2012). Mazibuko et al. (2013; 2015) demonstrated that, at a molecular level, green rooibos extract and aspalathin ameliorated insulin resistance by resensitising insulin signalling suppressed by palmitate culminating in increased levels of glucose uptake via GLUT 4 in muscle and fat cells.

These findings were independently confirmed by Son et al. (2012) who also reported that aspalathin, isolated from rooibos, increased glucose uptake in L6 myotubes by increasing AMPK phosphorylation and GLUT 4 translocation to the membrane. Furthermore Son et al. showed that aspalathin protected against ROS generated by artificial advanced glycation end (AGE) products in RIN-5F rat insulinoma cells.

Further, in the ob/ob mouse model, aspalathin reduced fasting blood glucose levels, increased adiponectin levels and reduced hypertriglyceridaemia and serum thiobarbituric acid reactive substances (TBARS) levels, a marker of ROS. In addition, enzymes related to gluconeogenesis, glycogenolysis and lipogenesis were reduced by aspalathin, while the mRNA expression of glycogen synthase was increased in the liver of these obese IR mice.

In the heart, Smit et al., (2017) demonstrated that aspalathin improved glucose uptake of the heart muscle in young and mature obese insulin resistant rats. Aspalathin also protected the diabetic heart against glucose induced oxidative stress and cell damage. Using H9c2 heart cells aspalathin was demonstrated to reduce oxidative stress and protect against high glucose induced cell apoptosis (Johnson et al., 2016). These findings were confirmed in diabetic db db mice whereby the diabetes-induced oxidative stress was improved by aspalathin via the downregulation of NRF2 a major regulator of cellular antioxidation (Dludla et al., 2017).

In addition, aspalathin was shown to reduce increased triglycerides and cholesterol, thereby reducing cardiovascular risk factors (Johnson et al., 2017). In vitro and in vivo studies on the beneficial effects of rooibos and aspalathin are continuing at the SAMRC and a clinical trial is planned for 2018.

REFERENCES:

7. Dludla et al., 2017. Aspalathin protects the heart against hyperglycemia-induced oxidative damage by up-regulating Nrf2 expression. Molecules 22: 129.