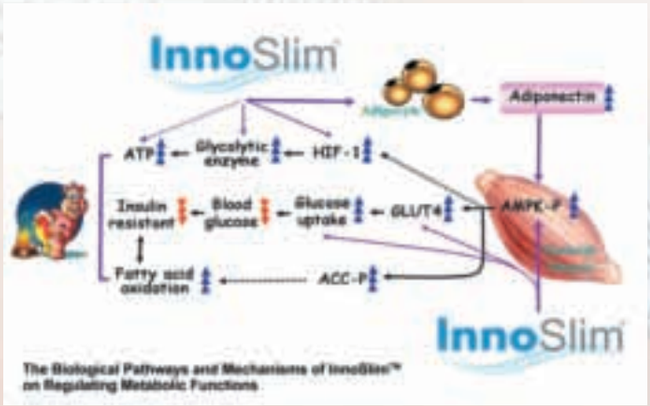
InnoSlim®

Product Dossier
Effective and Stimulant Free Weight and Blood Sugar Management Catalyst
Optimizes Glucose and Fat metabolism



InnoSlim™'s Biological Pathways/Mechanisms on Regulating Metabolic Functions

I. What Is InnoSlim™

InnoSlim™ is a proprietary blend of highly fractionated Panax notoginseng and Astragalus membranaceus extracts using a proprietary pharmaceutical extraction and processing technology. NuLiv's in vitro, in vivo, and human studies have demonstrated that InnoSlim™ can effectively regulate glucose, fat cell, and muscle cell metabolisms that have positive effects in reducing glucose absorption, and in increasing fat cell breakdown and muscle cell glycogen synthesis. These effects make InnoSlim™ an ideal foods and dietary ingredient for weight, blood sugar, and cholesterol and blood lipid management as well as other food and dietary supplement products.

2. How InnoSlim™ Works

InnoSlim™ decreases the glucose absorption rate in the intestine by down regulating both the protein and mRNA expression levels of SGLT1. InnoSlim™ may increase metabolic rate, possibly through mildly modulating the activity of sympathetic nerve. Confirmation of this effect requires further research. InnoSlim™ up-regulates adiponectin, which is a well known wellbeing & Slim Marker, while down-regulates the inflammatory & insulin resistance markers, i.e., TNF-alpha, RBP-4, PAI-1. InnoSlim™ decreases circulating glucose and reduces fat accumulation through AMPK- HIF-1-GLUT4 Pathway and results in a favorable metabolic milieu for correcting and supporting type 2 diabetic conditions.

3.Specific Functions of InnoSlim™

- Decreasing glucose absorption in intestinal cells (42%) (Section 6.1)
- Increasing glucose absorption in muscle cells (Section 6.2)
- Increasing glucose absorption and fatty acid breakdown in fat cells (Section 6.3)
- Decreasing glucose levels (Section 7.2)
- Increasing insulin sensitivity (Section 7.3)
- Reducing the activity of inflammatory bio markers (Section 6.4)
- Increasing ATP production in liver cells (Section 6.5)

4. Applications

Blood Sugar Health

Cardiovascular Health
Fitness & Energy
General Health & Wellness
Weight Management
And countless other health categories

5. Research Summary

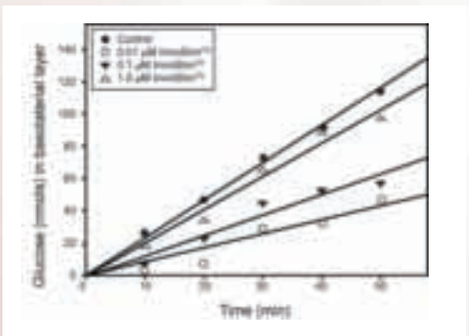
In vitro - InnoSlim™ decreased the intestinal glucose absorption rate by 42% when compared to the control group. The in vitro study on InnoSlim™ is similar to the in vitro study published in J. Agric. Food Chem., Vol. 55, No. 5, 2007 by T.C. Chang, etc. (1, 2) that was sponsored and funded by NuLiv Science. InnoSlim™ also elevated cellular level ATP by 18% when compared to the control group. (Section 6)

In vivo – InnoSlim™ increased insulin sensitivity by 38% and decreased blood sugar level by 19% when compared to the control group. (Section 7)

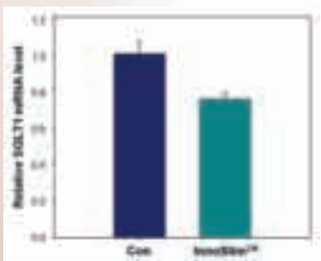
6. In Vitro Studies

6.1 Glucose Absorption in Intestinal (Caco-2) Cells (details available)

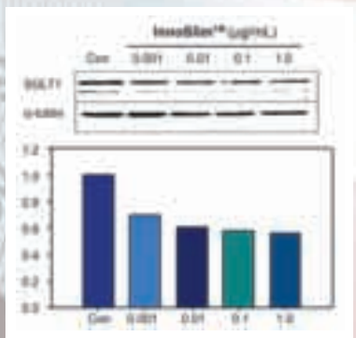
InnoSlim™ decreases glucose absorption in intestinal (Caco-2) cells by 42%



InnoSlim™ Down-regulates Transcription of SGLT1

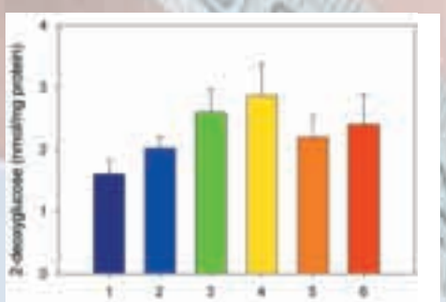


InnoSlim™ Down-regulates Protein Expression level of SGLT1 in Intestinal (Caco-2) Cells

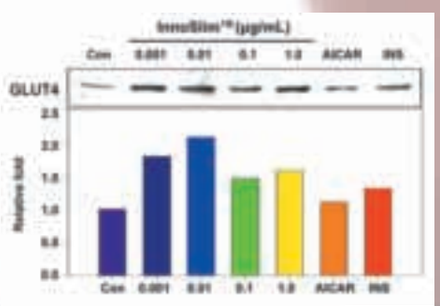


6.2 Glucose Absorption in Muscle (HSMMT) Cells (details available)

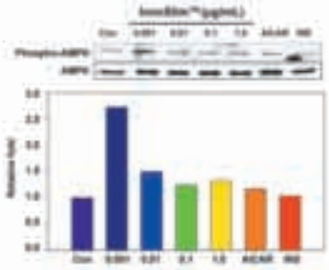
InnoSlim™ Up-regulates Glucose Uptake in Muscle (HSMMT) Cells



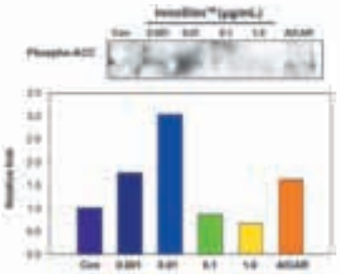
InnoSlim™ Up-regulates Protein Expression level of GLUT4 in HSMMT Cells



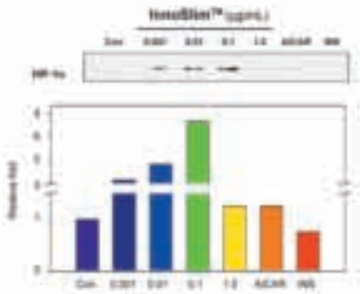
InnoSlim™ Up-regulates Protein Expression Level of Phospho-AMPK in HSMMT Cells



InnoSlim™ Up-regulates Protein Expression Level of Phospho-ACC in HSMMT Cells

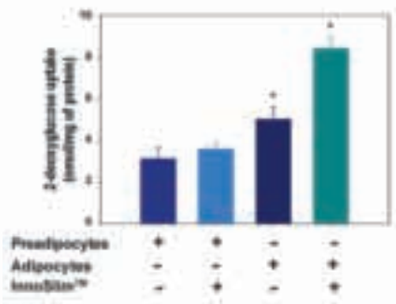


InnoSlim™ Up-regulates Protein Expression Level of HIF-1

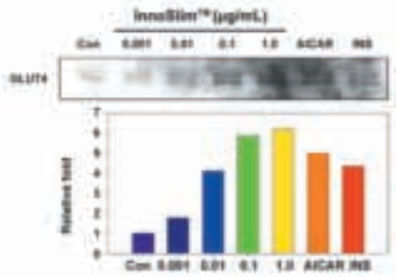


6.3 Glucose Absorption and Fatty Acid Breakdown in Fat (3T3-L1) Cells (request details)

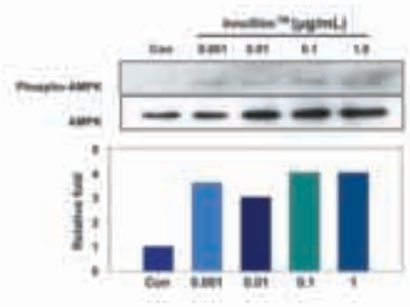
InnoSlim™ Up-regulates Glucose Uptake in fat Cells



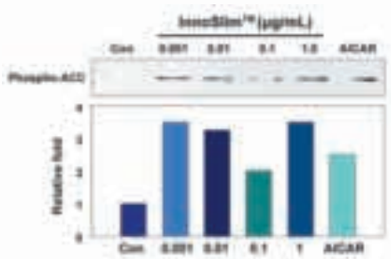
InnoSlim™ Up-regulates Protein Expression level of GLUT4 in Adipocytes



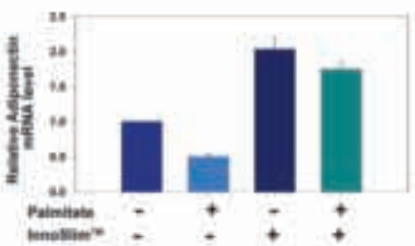
InnoSlim™ Up-regulates Protein Expression level of Phospho-AMPK in Adipocytes



InnoSlim™ Up-regulates Protein Expression Level of Phospho-ACC in Adipocytes

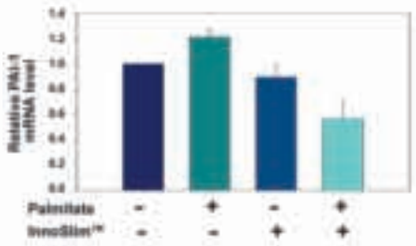


InnoSlim™ Up-regulates Transcription of Adiponectin

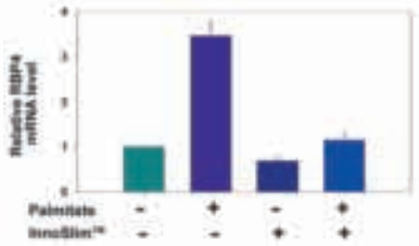


6.4 Regulation of Inflammatory Biomarkers (Details available)

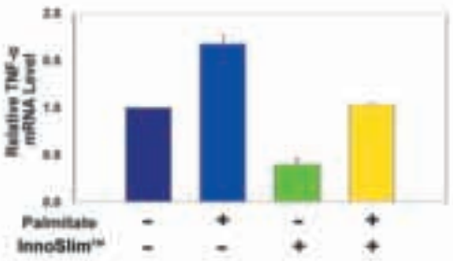
InnoSlim™ Down-regulates Transcription of PAI-1



InnoSlim™ Down-regulates Transcription of RBP4

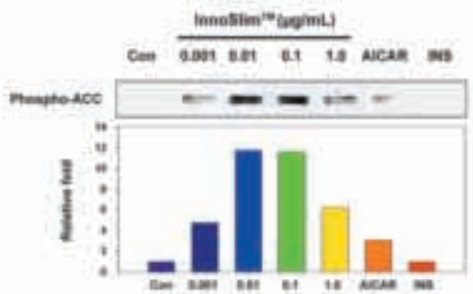


InnoSlim™ Down-regulates Transcription of TNFα

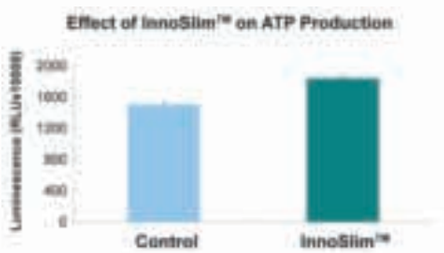


6.5 Liver Cell Studies (Details available)

InnoSlim™ Up-regulates Protein Expression Level of Phospho-ACC in HepG2 Cells



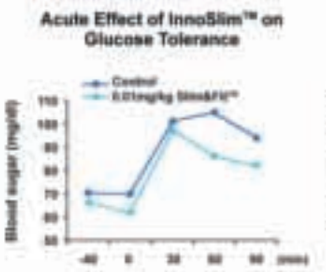
InnoSlim™ Increases ATP Production in Liver Cells

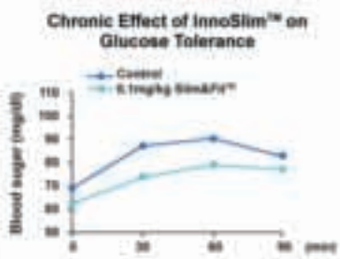


7. In Vivo Studies

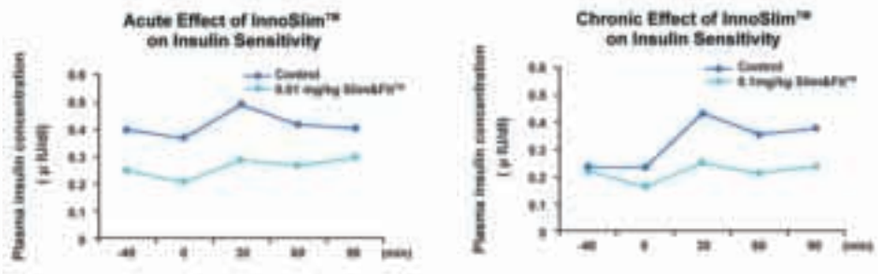
7.1 Animal Subjects (details available)

7.2 Oral Glucose Tolerance Tests (OGTT) (details available)





7.3 Insulin Assay (details available)



8. Discussion

Obesity is simply the consequence of an imbalance between energy intake and expenditure in human body. The main sites of energy utilization, skeletal muscle and adipose tissue, play significant roles in the regulation of energy homeostasis. Skeletal muscles are one of the major organs responsible for insulin-mediated glucose disposal and maintenance of glucose homeostasis of the body. Adipose tissue is now considered to be an active endocrine organ that secretes various humoral factors (adipokines). Several lines of evidence have shown that obesity triggers dysregulation of the endocrine function of adipose tissue. This inflammation, in turn, has been linked to the pathology of several metabolic and cardiovascular disorders, such as insulin resistance, type 2 diabetes, hyperlipidemia, and atherosclerosis.

Adiponectin is secreted by adipocytes and is the only adipose-specific protein known to date that is negatively regulated in obesity. Arita et al showed that mean plasma adiponectin levels were 3.7 mg/ml in a group of obese patients, whereas in non-obese subjects these values reached a mean of 8.9 mg/ml. Adiponectin has been implicated as a mediator of insulin sensitivity. Adiponectin increases insulin sensitivity by increasing fatty acid oxidation and decreasing the buildup of triglycerides in skeletal muscle.

HIF-1 is a stress sensor, a heterodimeric transcription factor composed of alpha and beta subunits. The alpha subunit is stable in hypoxic conditions but is rapidly degraded in normoxia. The function of HIF-1 is also modulated by several molecular mechanisms that regulate its synthesis, degradation, and transcriptional activity. Upon stabilization or activation, HIF-1 translocates to the nucleus and induces transcription of its downstream target genes.

9. Safety Data (details available)

10. Conclusion

AMP-activated protein kinase (AMPK) functions as an energy sensor to provide metabolic adaptation under the ATP-deprived conditions such as hypoxia. Activation of AMPK in skeletal muscle, liver, and adipose tissue results in a favorable metabolic milieu for the support and correction of type 2 diabetic conditions, i.e., decreased circulating glucose, reduced plasma lipid, and ectopic fat accumulation, as well as enhanced insulin sensitivity.

11. Reference (details available)