Glucagon-Like Peptide-1

Glucagon-like peptide -1 (GLP-1) is a 30 amino acid polypeptide. It is an incretin hormone, which increases postprandial insulin secretion, produced by L cells of the small intestine and colon. Fasting plasma GLP-1 levels range between 5-10 pmol/L and peaks at 50 pmol/L 20-30 minutes after meal consumption depending on the size and nutrient composition of the meal. (Baggio and Drucker, 2007) GLP-1 receptors are found on pancreatic cells, adipose tissue, parietal cells of the stomach, pylorus, and brain.

Bo Ahren Nature Reviews Drug Discovery 8, 369-385 (May 2009)

GLP-1 level rises after nutrient ingestion especially glucose and fatty acids (Roberge and Brubaker, 1993) and rapidly decreases due to renal clearance and degradation by dipeptidyl peptidase IV (DPP-4) (Kieffer et al., 1995).
GLP-1 promotes cell insulin gene transcription and glucose dependent insulin release (Drucker et al., 1987). Exogenous GLP-1 stimulates islet cell proliferation in vitro (Buteau et al., 1999) and promotes cell neogenesis and increases islet size in diabetic db/db mice (Stoffers et al. 2000). Additionally, GLP-1 inhibits cell apoptosis, and glucagon secretion, gastric emptying and food intake (Drucker, 2000). GLP-1 has been shown to reduce blood glucose, hemoglobin A1c, increase insulin secretion, and insulin mRNA in diabetic rodents (Stoffers et al, 2000; Greig et al., 1999; Wang et al., 1997). In diabetic patients GLP-1 infusion has been shown to promote satiety, reduce fasting plasma glucose and postprandial plasma glucose and increase insulin levels (Toft-Nielsen et al., 1999).

Baseline levels of GLP-1 in diabetic and normal subjects are the same however glucose stimulated GLP-1 secretion is blunted in diabetic individuals compared to matched normal subjects (Mannucci et al., 2000). GLP-1 receptor agonists and DPP-IV inhibitors can enhance the action of GLP-1 in diabetics. However, nutrient can also stimulate GLP-1 secretion especially pea protein (Geraedts et al., 2011). Moreover, in vitro studies have shown that essential amino acids compared to non-essential amino acids increase GLP-1 secretion via extracellular signal-regulated kinase (ERK1/2) activation (Reimer, 2006). Furthermore, consumption of high protein diet containing 30%, 40%, and
30% of energy from protein, carbohydrate and fat respectively, elevated GLP-1 concentration and increased satiety, diet-induced thermogenesis, and fat oxidation, in healthy subjects (Lejeune et al., 2006). Also, breakfast rich in monounsaturated fatty acids (Mediterranean diet) increased GLP-1 response compared to carbohydrate rich breakfast in obese and insulin resistant subjects given respective diets for 28 days in a crossover study (Paniagua et al., 2007). This formulation contains macronutrient distribution of 30/40/30 of protein/carbohydrate/fat, high monounsaturated fatty acids, high essential amino acids and pea protein thereby potentially causing increased GLP-1 secretion thus minimizing postprandial glucose excursions.

Reference:-
Roberge JN, Brubaker PL 1993 Regulation of intestinal proglucagon-derived peptide secretion by glucose-dependent insulinotropic peptide in a novel enteroendocrine loop.Endocrinology 133:233–240

