Cytokines, SOCS-3 signaling: causes of high blood pressure, high cholesterol, & diabetes….
The Autocrine and Paracrine Roles of Adipokines. K Karastergiou *Molecular and Cellular Endocrinology* 2009 doi:10.1016/j.mce.2009.11.011

"The secretions of these engorged (fat) adipose tissue macrophages (immune fighting cells), such as IL-6 (interleukin) and TNFα (tumor necrosis factor-alpha) along with MCP (monocyte chemo-attractant protein) and leptin from hypertrophied (enlarged) adipocytes (fat cells) regulate the pathological changes of obesity, such as insulin resistance, and endothelial dysfunction."

Diabetes, Metabolic Syndrome, and Obesity 2012; 5:175-189 online 7/8/12 I Spreadbury
Comparison with our **ANCESTRAL DIET** suggests dense **ACELLULAR** carbohydrates (no longer in the form in which it grows in the field = refined or processed starches) promote an **INFLAMMATORY** microbiota (the bacteria in our bowel) the primary dietary cause of LEPTIN resistance and OBESITY.
"…diet-related inflammation and evolutionary medicine. The obese guard their elevated weight…. In high-fat diet-induced obesity, leptin resistance is seen initially at VAGAL afferents, blocking the actions of satiety (feeling full) mediators, then centrally with GASTROINTESTINAL BACTERIAL-TRIGGERED SOCS3 signaling." …. Due to being made up of **CELLS**, virtually all "**ANCESTRAL**" foods have markedly lower carbohydrate densities. Thus, the "forgotten organ" of the gastrointestinal microbiotica (BOWEL FLORA) is markedly changed by **POSTPRANDIAL LUMINAL CARBOHYDRATE CONCENTRATIONS**. … **ACELLULAR** flours, sugars, and processed food produce an INFLAMMATORY MICROBIOTA in the gastrointestinal tract with **FAT** effecting a "**double hit**" by allowing increased absorption of (INFLAMMATORY) POLYSACCHARIDES. A diet free of refined grains and full of whole foods with carbohydrate from CELLULAR tubers, leaves, fruits, and nuts produces a gastrointestinal microbiota consistent with our evolutionary configuration.

Google "SOCS3" search 7/8/12: "HGNC is the gene that encodes a member of the STAT-induced STAT inhibitor (SSI) also known as suppressor of cytokine signaling (**SOCS**). This gene is induced by **INTERLEUKIN** 6, interleukin 10, and INTERFERON gamma. The protein encoded by this gene can bind to **JAK2 kinase**."

"**Signal transducers and activators of transcription (STAT)-induced STAT inhibitor-1 (SSI-1) suppressor of cytokine signaling-1 (SOCS-1)** suppresses **TUMOR NECROSIS FACTOR** alpha (TNF a) induced cell death. Y Morita et al *Proc Natl Acad Sci* 2000;97:5405-5410

Signal transducers and activators of transcription (STAT)-induced STAT inhibitor-1 [SSI-1; also known as a suppressor of cytokine signaling-1 (SOCS-1)] was identified as a negative feedback regulator of **JANUS KINASE STAT** signaling…. accelerated apoptosis… This sustains the activation of **p38 mitogen-activated protein kinase (MAPK**)... CYTOKINES play important roles in controlling … cell DIFFERENTIATION, PROLIFERATION (STAT3 inhibition of **NF-kB** induced **LVH** is attenuated by IL 10), and APOPTOSIS, and these effects are mainly brought about by Janus kinase-signal transducers and activators of transcription (**JAKSTAT**) signaling. Activation of suppression of cytokine signaling (SOCS-1) is influenced by IL2, IL3, IL4, IL6, IL 13, granulocyste-macophage colony-stimulating factor, erythropoeitin, IFN-g…affect all 4 JAKs."