ADVANCED CUTTING EDGE CONCEPTS IN OBESITY RESEARCH AND TREATMENT

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- Exercise Physiologist for ACE
- Doctorate in Exercise Physiology and Education Administration
- M.S.-Masters in Physiology
- Masters in Exercise Physiology
- BS- Kinesiology and Psychology- UCLA
- Infomercial Chief Scientist- 10 studies
- Primary author- personal training manuals
- Nationally ranked duathlete (1993)
- Currently competitive runner- road & trail

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Obesity Productions by ACE

- The Skinny on Fatness: Epidemiology on Obesity
- The Etiology of Obesity: The Causes of Weight Gain
- Weapons of Fat Mass Destruction: Roles of Diet, Exercise and Science to Battle the Bulge
- Advanced, Cutting Edge Concepts in in Obesity Research and Treatment

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Learning Objectives

1. Understand the new holistic, dynamic approach to obesity management vs. an isolated, static perspective
2. Understand what epigenetics is and how life events/behaviors can alter genetic outputs
3. Appreciate how the endocrine system may promote obesity and disease
4. Discuss how inflammatory factors may promote obesity and disease and strategies to lower these factors
5. Realize how visceral adipose and metabolic syndrome are interlinked and associated with adipokines/ hormones
6. Understand the effects of diet and exercise in altering hormonal milleau and adipokine activity to reduce obesity and counter disease effects

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Quote from Dr. Luxy John

"Obesity is being revolutionized by our greater understanding of the disease. Knowledge in this field is still limited to a few culprit factors that might be involved in the perpetuation of the disease, while less is known about the precise role of the individual factors in the causation or initiation of obesity. Discovery of up to 20 different peptides including Ghrelin, Leptin, and GLP-1 have given obesity research a boost, opening up areas for further studies'

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Chewing the Fat…On Fat

- Since 1970 in the U.S., the rates of obesity have doubled to 35.8% and more than 2/3rd overweight.
- Economic impact of obesity (2005)- estimated at $190.2 Billion (B).
- Medical care costs in U.S.(2008) was $147B*. This figure went up 47% (almost double) from 1998- $78.5 B
- 75 cents for every $1 goes to treating chronic diseases, many of which can be decreased by lifestyle choices.

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**Finkelstein, EA, Trogdon, JG, Cohen, JW, and Dietz, W. Annual medical spending attributable to obesity: Payer- and service-specific estimates. Health Affairs 2009; 28(5): w822-w831.*

Chewing the Fat…On Fat

- One study found those ≥30 BMI had 36% higher health care costs.
- Annual direct costs of childhood obesity is estimated at $14.3 B. It is believed that it may increase to $45B by 2050.
- Estimated annual expenditures on diet products $40-$100 Billion in the US alone! (Cummings- Banking on Failure- BBC News- 03
- Marketdata estimates in 2007 at $55B and now over $60B spent on fat loss products or programs
- Its estimated that 400,000 American deaths/ yr are attributable to poor diet and physical inactivity

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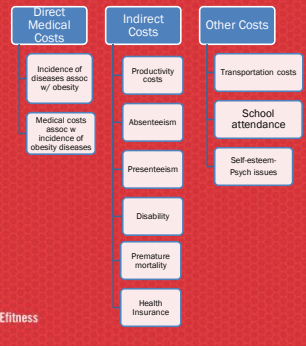
Cost of Obesity- Comes in Many Forms



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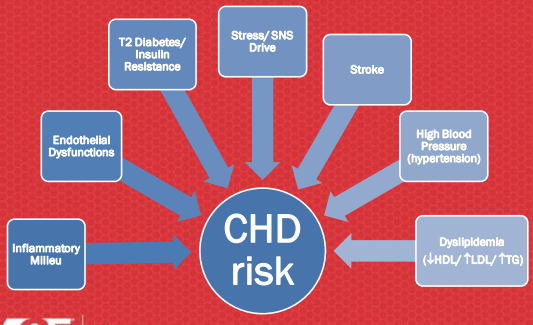
Being Overweight Itself- Isn't Dangerous



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New Frontiers in Obesity Research

- Gut Hormones
- Adipokines/ Cytokines
- Visceral Adipose Tissue vs Sub-cutaneous
- Epigenetics/ Genomics/ Obesogens
- Insulin Resistance/Metabolic Syndrome
- Role of Exercise in modulating cellular and genetic factors

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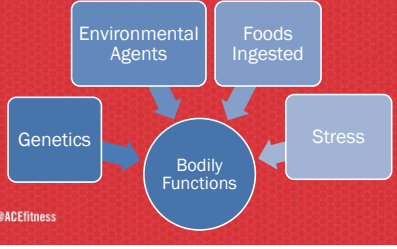
Systems Biology Approach with Nutrition

- *"Systems biology…is about putting together rather than taking apart, integration rather than reduction. It requires that we develop ways of thinking about integration that are as rigorous as our reductionist programs, but different….It means changing our philosophy, in the full sense of the term" Noble, Dennis (2006). The music of life music of life: Biology beyond the genome. Oxford: Oxford University Press. pp. 176*



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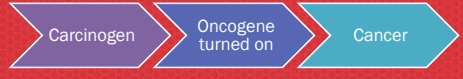
Paradigm Shift with Nutrition and Health Care- Static to Dynamic

- The old healthcare system used to isolate the body parts and symptoms (reductionist approach). A sickness was due to some system breaking down and the symptoms needs to be reduced or removed
- The new healthcare system looks at not only the interactions of various physiological systems but the environmental and genetic influences. A new view that "sickness" is likely the normal reaction of the body to a given stimulus (often a toxin)



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A Holistic View- Inside and Out

- Mother's Weight
- Breast feeding
- Television
- Family meals
- No outdoor play
- Dieting too early
- Poverty

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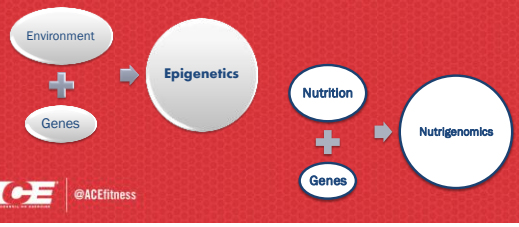
A Holistic and Dynamic Approach

- Seeing things in the "totality" is critical to be able to discern "true functionality" or dysfunction
- Scientists must dissect things to understand independent function (in vitro), but to understand how a system works, we must observe "the system" as a whole (in vivo)



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Examples of Epigenetics and Health

Person A	Person B	Person C	Person D
Genetic Code- 1	Genetic Code-1	Genetic Code-1	Genetic Code-1
Bad health lifestyle	Bad health- lifestyle	Good health-lifestyle	Good Health- lifestyle
Diseases develop	No diseases develop	No diseases develop	Diseases develop

Environmental agent exposure can be substituted for lifestyle

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Epigenetics, Drugs, and Health Effects- Pharmacogenomics

Person A	Person B	Person C	Person D
Drug- 1	Drug-1	Drug-1	Drug-1 + Lifestyle
Bodily reaction	No Bodily reaction	Side Effect Reaction	Interactive Reactions
Symptoms Relieved	No symptoms relieved	Side Effect Expression	Symptoms relieved or not

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Communication Systems in the Body

Neuro-Endocrine System
- Electrical Impulses
- Neurotransmitters
- Hormones
  - Endocrine
  - Exocrine

Peripheral Intercellular
- Hormones (Gut, other organs)
- Cytokines- non specificied cell
- Adipokines- Adipocyte cytokine
- Myokines- Skeletal muscle cell

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Factors That May Alter Hormone Action

- Food additives- preservatives and sweetners
- Food pesticides- environmental pollutants
- Plastic byproducts
- Drugs
- Infections
- Diseases- insulin resist., cancers, liver & kidney
- Lack of exercise
- Irregular meal consumption (skipping breakfast)
- Extreme Dieting
- Smoking
- Stress & Depression & anger
- Pregnancy/menopause
- Vitamin deficiencies
- Excess caffeine
- Excess alcohol
- Excess sugar

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Hormone Imbalances in America?

- 25.8 million Americans have diabetes- 8.3% of population (2011)
  - 18.8 million diagnosed
  - 7 million undiagnosed
  - 215,000 people under 20 have diabetes- 1 in 400 children-adolescents
- 79 million Americans have pre-diabetes (2011)
- 1 in 3 people have metabolic syndrome (criteria as listed in 2003-06 NHANES sample)
- 1 in 10 have an underactive thyroid gland
- 1 in 10 have polycystic ovarian syndrome
- 1 in 13 have severe PMS

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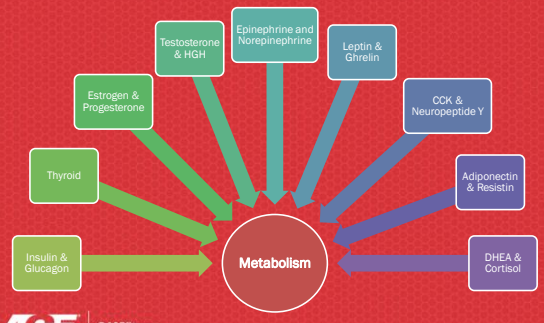
Endocrine System and Metabolism



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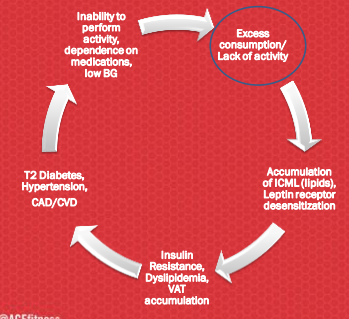
Hormones Effects on Appetite

Hormone	Source	Factors Increasing	Factors Decreasing	Actions
Gherlin	Stomach Fundus	Protein, Fiber, Smoking	Glucose, Fat, Insulin, PYY	↑Appetite, GH release, ↑ Gastic Emptying
GLP-1	Distal ileal (sm intestine) cells	Glucose, FFA, Epi, Atropine,	Insulin, Somatostatin	↓Appetite, ↓ glucagon, delay gastric empty, Insulinotropic
Oxyntomodulin	L (oxyntic)cells-fundic mucosa	It binds to GLP-1 and glucagon Rs		↓Appetite, Inhib acid secrtn, no change gastic empty, (-) plasma gherlin
Peptide YY	Colo-Rectal- L cells	Fiber, short chain FA, Bile salts, Glucose, AAs, CCK, CGRP	GLP-1	↓Appetite, Inhib gastric acid secrtn, ↓gastic empty, stim intenst H20 absorption
Cholecystokinin (CCK)	Intestinal 1 cells	Fats, Proteins, Bulimia		↓Appetite, Stim gall bladder contrxn, relax sphincter Oddi, delays gastric emptying, (-) gastric acid secretion
Amylin	Pancreatic Beta Cells (P cells)	Blood glucose rise, TNF and fatty acids		↓Appetite, Reduces glucagon, delays gastic emptying, ↑renin & aldosterone
Enterostatin	From proenzyme pancreatic procolipase	Fat in diet		↓Appetite- esp. fat intake, body fat/weight, increase sympath drive to BAT.

Negative Interactions in Systems



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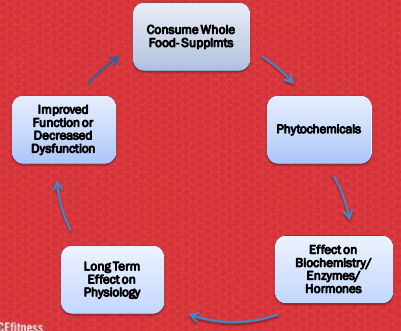
Positive Interactions in Systems



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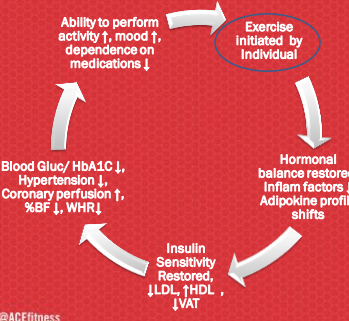
More Positive Interactions in Systems



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Factors Associated with Inflammation

- **Activators/Markers**
  - C-Reactive Protein
  - Homocysteine
  - Tumor Necrosis Factor (TNF-α)
  - InterleukinIL-1, 2,6, 18
  - E-selectin
  - Serum amyloid A
  - Cell adhesion molecule-1 (CAM-1, VCAM-1)
- **Inhibitors**
  - Adiponectin
  - Omega 3 FA (to ↓ 6:3 ratio)
  - Dietary Fiber
  - Carotenoid rich foods
  - Nuts (esp. ones rich in arginine & monosat. fats

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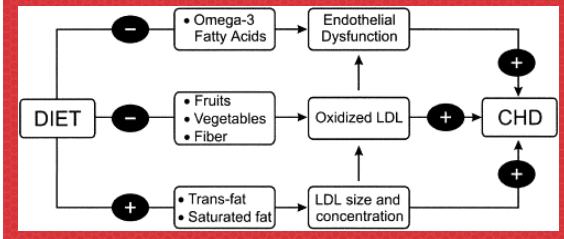
Ways Unhealthy Dietary Patterns may lead to Coronary Heart Disease



Taken from- Giugliano, D, Criello A, Esposito K (2006). The Effects of Diet on Inflammation: Emphasis on the Metabolic Syndrome. J or Am College of Cardiology 48(4): 677-685

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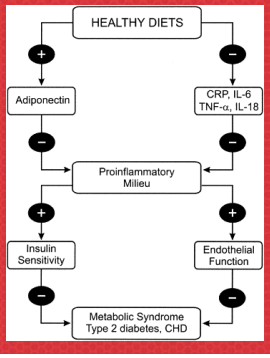
Healthy Diets affects on Proinflammatory Milieu & Metabolic Syndrome

Taken from- Giugliano, D, Criello A, Esposito K (2006). The Effects of Diet on Inflammation: Emphasis on the Metabolic Syndrome. J or Am College of Cardiology 48(4): 677-685



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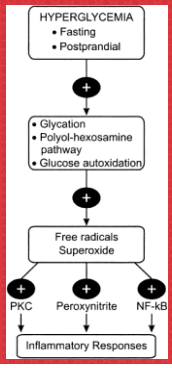
Hyperglycemia on Inflammatory Responses



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The Effects of Diet on CHD Risk

- Serum lipids- the obvious (LDL /HDL / TG)
- Oxidative Stress
- Sub-clinical Inflammation
- Endothelial Dysfunction
- Insulin Sensitivity
- Blood Pressure
- Thrombotic Tendency

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General Dietary Recommendations to Lower CHD Risk Factors

1) Increase consumption of Ω3 fatty acids
2) Substitute non-hydrogenated unsaturated fats for saturated and trans fats
3) Consume a diet high in:
   a) Fruits and vegetables- especially colorful ones
   b) Nuts- especially those with linolenic acid
   c) Whole grains and low in refined grains

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Metabolic Syndrome- Description

- Metabolic Syndrome identified in 1988 by Gerald Reaven
- Metabolic syndrome is characterized by:
  - the presence of obesity and insulin resistance,
  - a reduced responsiveness of the body tissues to insulin.
- Associated disorders of metabolic syndrome are:
  - high blood pressure
  - two lipid problems: high blood levels of triglycerides and low blood levels of high-density lipoprotein cholesterol (HDL).
- Having metabolic syndrome:
  - increases the individual's chance of developing type 2 diabetes 4-5x,
  - doubles the chance of developing coronary heart disease. (2008)

- *Grundy SM. Metabolic Syndrome Pandemic. ArteriosclerThrombVascBiol. 2008 April; 28: 629-36.*

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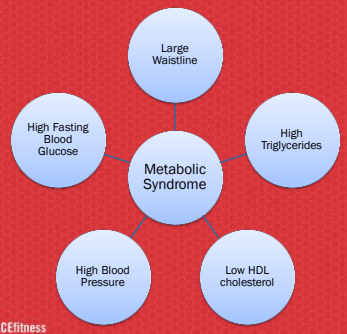
Metabolic Syndrome Factors



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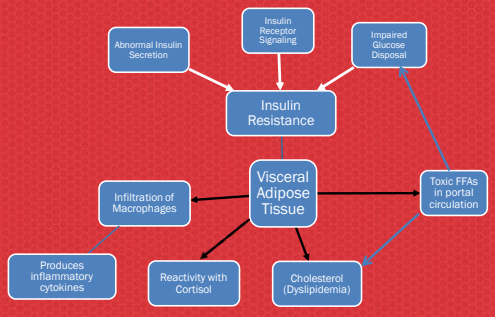
Metabolic Syndrome Overview



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Metabolic Syndrome & CVD Risk Factors

- Abdominal obesity
- Atherogenic dyslipidemia (elevated triglyceride, small LDL particles, low HDL cholesterol)
- Raised blood pressure
- Insulin resistance (with or without glucose intolerance)
- Proinflammatory state
- Prothrombotic state

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Metabolic Syndrome Criteria- Age and Weight Matters

- Being in the 40-59 yr- ↑ 3x more likely than 20-39 yrs to meet criteria
- 40% of people over 60 meet criteria
- Males 60+ yrs- ↑ 4x, Females- ↑ 6x- as youngest group
- Overweight males 6x & Obese males 32x more likely than normal weight males to meet the criteria
- Overweight females 5x and obese females 17x more likely than normal weight females to meet the criteria
- 1 million (4.2%) 12-19 yrs met the criteria.

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Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among U.S. adults. *Diabetes Care*. Oct 2004;27(10):2444-9.

The SYNDROME of Modern Society

- **Factors for Metab Syndrome include:**
- 20-30% of U.S. population have at least 3 symptoms
- 1/3 rd of U.S. population have at least 1 symptom
- Highest among Mexican Am (31.9%) then African American women- explaining high incidence of other symptoms.
- About 1/4th of European & Latin American people have MS
- Increased from 50 mil in 1990, to 64 million in 2000.
- MS doubles risk of CAD

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Does your Shape indicate your Risk?

Male	Female	Health Risk- WHR based
= or < 0.90	= or <0.8	Low
0.9 to 1.0	0.81 to .85	Moderate
>1.0	>0.85	High

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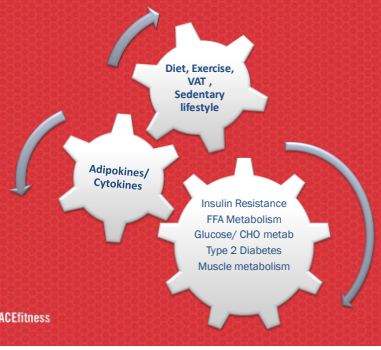
What are the links between cause and effect for metabolic syndrome?



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Comparison of VAT to SCAT

Visceral Adipose Tissue	Sub-Cutaneous Adipose Tissue
Major predictor of Metab Syndr- IR	Preadipocytes have greater differentiation capac.
Less responsive to insulin's adipogenic effects	May replenish VAT
Produces more IL-6, PAI-1	Produces Leptin
More glucocorticoid receptors	Estrogen promotes
High density of androgen receptors	May be protective
May enhance truncal SCAT lipolysis	

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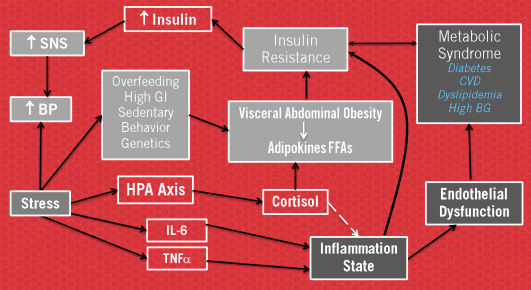
Overview of Factors involved with VAT and Metabolic Syndrome



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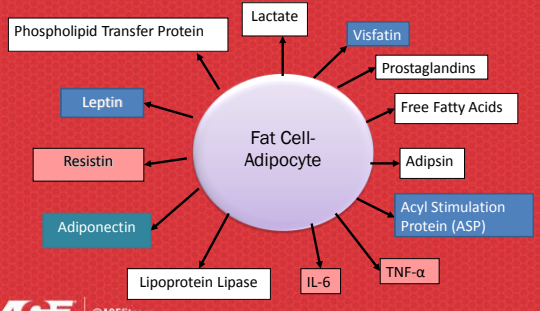
Various Chemical Signals to Adipocytes



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Known Adipokines

Specific Adipokines
- adiponectin
- adipsin
- estrogen
- angiotensin II
- angiotensinogen
- leptin
- plasminogen activator I (PAI-1)
- agouti protein
- resistin
- acylation stimulating protein (ASP)
- IGF-1
- tumor necrosis factor alpha ($TNF\alpha$)
- transforming growth factor (TGF)-B
- bone morphogenic protein (BMP)

Non-Specific Adipokines
- prostaglandins
- various IGF binding proteins
- interleukins (ILs)
- fibroblasts
- FFAs

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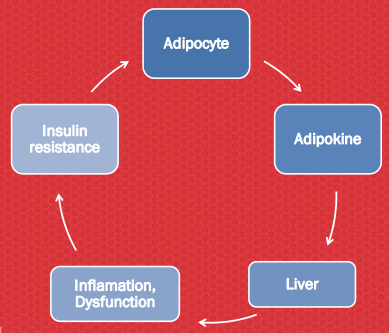
Simplified View of Adipocyte Interactions



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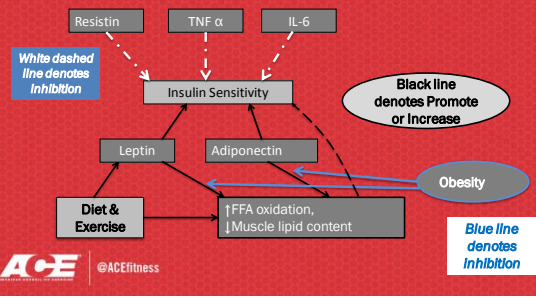
Role of Adipokines as regulators of skeletal muscle fatty acid metab. and insulin sensitivity

- Leptin and Adiponectin vs Resistin and TNFα



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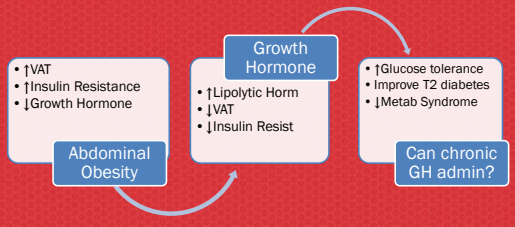
Visceral Obesity, Impaired Glucose Tolerance, Metabolic Syndrome and Growth Hormone Therapy



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Attallah H, Friedlander AL, Hoffman AR Growth Hormone, IGF Research 2006. Jul: 16 Suppl A:S62-7. Epub 2006, Apr 18

Overview of Adipokines/Cytokines on Insulin

- Leptin and Adiponectin
  - Increase insulin sensitivity
  - Increase free fatty acid oxidation (via AMP-activtd prot. kinase)
  - Decrease skeletal muscle (sk. mm.) lipid content (storage)
- Resistin, Tumor Necrosis Factor-Alpha (TNFα), Interleukin-6 (IL-6)
  - Decrease Insulin Sensitivity
- Obesity seems to impairs leptin and adiponectin to stimulate fat oxidation in muscle, which leads into insulin resistance
- Resistin and TNF α have been shown to directly impair insulin signaling thus insulin stimulated glucose uptake.
- Diet & Exercise restores the sensitivity of skeletal muscle to leptin

Dyck, DJ, Heigenhauser, GJ, Bruce, CR. The role of adipokines as regulators of skeletal muscle fatty acid metabolism and insulin sensitivity. *Acta Physiol (Oxf), 186(1): 5-16, 2006.*

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Description of Adiponectin- Affects and Effects

- Adiponectin- anti-diabetic, anti-atherosclerotic, anti-inflammatory functions. Widespread influences: Insulin resistance, T2 Diabetes, obesity, vascular disease,
- Gene expression is higher in SCAT than VAT (lean & obese)
- Obese patients have lower Adiponectin mRNA levels
- Plasma levels negatively correlated to VAT amount.
- TNFα known to inhibit Adiponectin promoter activity

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Description of Adiponectin- Affects and Effects- cont.

- Its believed adiponectin receptors are in skeletal mm and exercise may increase it.
- Insulin reduces levels of Adiponectin mRNA
- Interleukin- 6 (IL-6) also lowers gene expression and secretion from adipocytes.
- Adiponectin are lower in ischemic HD and hypertensive patients. Adiponectin inhibits ICAM-1 va

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Description of Leptin- Affects and Effects

- Data shows that leptin deficiency causes major pathologies, but excess in the blood not much effect. Thus- primarily a regulator of food intake.
- Leptin uses a receptor (LROb) which then translocates a STAT3 transcription factor, which then activates a POMC transcription but also SOCS3.
- The SOCS3- *Suppressor of Cytokine signaling* - knocks out the Leptin signaling. Leptin can thus alter itself in obese states.

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Description of Leptin- Affects and Effects- cont.

- It seems that IL-6, TNF-α, fatty acids are regulators of SOCS3 and thus may explain leptin resistance in the obese.
- Leptin therapy has been shown to improve hypertriglyceridemia, insulin resistance, hyperphagia, and morbid obesity.
- Leptin receptors have been identified in the hypothalmus (feed & satiety center). A signal case of leptin receptor mutation was described in an obese human. Often seen in rat (fa/fa) models.

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Resistin- Affects and Effects

- Sometimes called FIZZ3- it is an adipokine. As named- it RESISTS insulin, causes glucose intolerance, and has been a link between obesity and insulin resistance.
- Resistin impairs insulin-stimulated glucose uptake in cultured adipocytes
- Administration of anti-resistin antibody prevented development of insulin resistance & hyperglycemia assoc with excess fat intake.
- Resistin levels in mice- decrease with fasting, and increase with re-feeding. Resistin also increases with high fat feeding.
- There has been no consistent findings with resistin levels in adipose tissue or circulation and insulin resistance or adiposity.
- Jury is still out on its being a causative factor or bystander in insulin resistance.

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Actions of Acyl Stimulation Protein (ASP)

1. A factor controlling adipose tissue function. When injected in lean mice it reduces TG levels 40%, and enhances TG clearance in hypertriglyceridemic and hyperinsulinemic mice (genetic model).
2. ASP stimulates TG synthesis in human adipocytes and skin fibroblasts. Synergistic with (and more potent than) Insulin in stimulating TG synthesis.
3. It acts to clear plasma TG in portion to its concentration, primarily through enzyme (diacylglycerol acyltransferase) activation.
4. Thus, ASP may enhance signaling between circulation and adipose tissue to modulate storage in fatty tissue.
5. ASP increases glucose transport via GLUT-1,3,4 transporters.
6. ASP is higher postprandial and higher in morbidly obese, in T2 diabetes. ASP levels correlated to lipids and BG in non-diabetic only, suggesting a possible dysregulation in ASP production or metabolism.
7. HyperApoB (lipoprotein) pts. have dysfunction in ASP pathway

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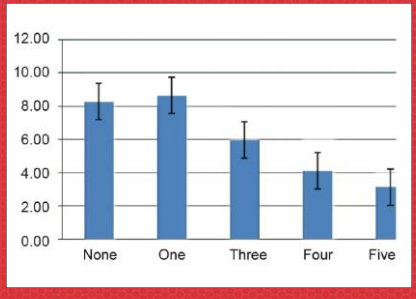
Adiponectin PAI-1 and Metabolic Syndrome



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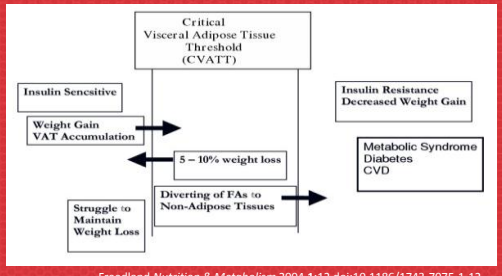
Critical Visceral Adipose Tissue Threshold (CVATT)



Freedland *Nutrition & Metabolism* 2004 **1**:12 doi:10.1186/1743-7075-1-12.

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Metabolically Obese vs BMI Obese

- Eric Freedland describes two forms of obesity and body weight is only one factor .
  - Metabolically obese normal weight (MONW)-apparently lean individuals with metabolic syndrome.
  - Metabolically normal obese (MNO), those with normal metabolic profiles- i.e."fit and fat."
- Exercise and Diet can have much more dramatic effects on metabolism than actual body weight.

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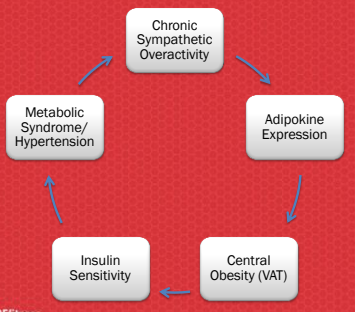
Obesity and Adipokines: Effects of Sympathetic Overactivity



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Why SNS Overdrive in Obesity?

- Part of the counter-regulatory reaction to excess feeding. Increased intake= increased peripheral tissue SNS activation to burn additional calories.
- With increased SNA→ stim β-adrenergic thermogenesis to prevent further fat storage.
- ↑ SNS also stimulate lipolysis to ↑ non-esterified FFA, contributing to insulin resistance.
- Adipose tissue may release adipokines that increase SNA. If chronic, it impairs β-adrenergic signaling, thus reducing metabolic stimulation = obesity, insulin resistance and other issues detrimental to health.

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What is Insulin Resistance? SNS role?

- Insulin Resistance is the eventual failure of the GLUT-4 to translocate to the cell membrane to uptake glucose into peripheral tissue.
- Evidence shows normal GLUT-4 levels in insulin resistant people- thus problem is a signaling issue.
- Thus glucose rises→ triggers more insulin stim. and eventually a "hyperinsulinemic state".
- High SNS→ ↓GLUT-4 activity via activating a phospholipase C- β, impairing dephosphorylation and the intrinsic activity of GLUT-4.

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Exercise & Diet Effects

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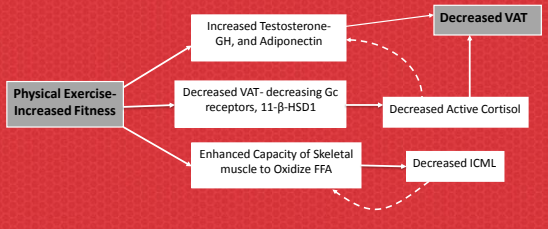
Possible Effects of Exercise in Countering VAT and Metabolic Syndrome



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Effect of Exercise on Circulating Adipokine Levels in Obese Young Women

Obese people had:

- Body wt.
- BMI
- % BF
- LBM
- hsCRP
- Leptin
- TNFα
- L-scale
- Adiponectin

After exercise- reversed

- Body Wt
- BMI
- % BF
- LBM
- hsCRP
- Leptin
- TNFα
- L-scale
- Adiponectin
- VO2max
- HDL-Chol

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Effect of low and high intensity exercise on circulating growth hormone in men

1. Males 18-35 volunteered for ramp-type progressive cycle-ergometer exercise to determine LT and VO2max.
2. They performed 1, 5, or 10 min constant work > or < LT.
3. Results showed only 10 minutes of high intensity exercise could raise GH significantly above baseline.
4. This increase occurred despite simultaneous increases in Insulin and Glucose
5. The HI 10 min bout also significantly raised Epi, NE, and lactate.

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Felsing NE, Brasel JA, and Cooper DM. Journ of Clin. Endocrin. & Metab, 75(1): 157-162

Insulin resistance with aging: Effects of diet and exercise

- Those with ↑ body fat, T2DM, ↑age tend to get IR
- Aging associated increases in body fat and IR
- Abnormal FFA metabolism has important role in IR
- Abdominal fat and VAT associated with IR
- Exercise and diet associated with ↓in body fat (wt loss) and ↑insulin sensitivity and glucose tolerance.
- Longitudinal studies show improved gluc metabolism w/ aerobic or resistance training in middle-aged and older men and women.
- Skeletal muscle changes, enhanced blood flow w/ exercise likely contribute to modifying insulin resistance.

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HIIE Training on Fat Loss and insulin Levels of Young Women

- 3 Groups of 15- High intensity intermittent exercise (HIIE), steady-state exercise (SSE), or control (cont) for 15 wks of exercise intervention. BMI 23.2±2.0, Age 20.2±2.0 yrs
- Both exercise groups significantly improve CV fitness, only HIIE had significant reduction in total body mass, fat mass, trunk fat, and fasting plasma insulin levels.
- ↓Leptin were - correlated with ↑VO_{2peak}, + correlated with decreases in TBM, no changes in adiponectin levels
- HIIE 3x/wk for 15 wks vs same SSE had signif↓ body fat, subcutaneous leg and trunk and insulin resistance.

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Trapp EG, Chisholm DJ, Freund J., Boutcher SH. (2008). Intl J of Obesity, 32, 684-691.

The Relationship of Selected Factors to Metabolic Syndrome in Young Females

- 69 healthy young females classifed according to BMI: lean<= 25, overweight (>25 and <30) and obese (>=30).
- Leptin, hs-CRP, insulin, and uric acid ↑ w/ ↑BMI.
- Only leptin vs waist circumference related in lean group
- 6 variables w/ signif correlations in obese group
  - hsCRP vs waist circumference
  - hsCRP vs systolic BP (-)
  - Insulin vs diastolic BP
  - Adiponectin vs blood sugar (-)
  - Uric acid vs waist circumference
  - Uric acid vs Triglycerides

Conclusion: Uric Acid seemed to be the most reliable variable to identify obese subjects with metabolic syndrome

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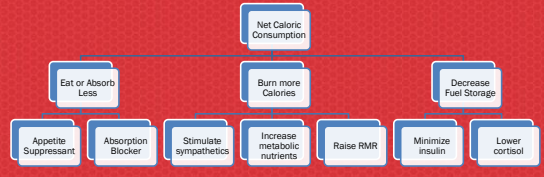
Dietary Strategies to Reduce Obesity



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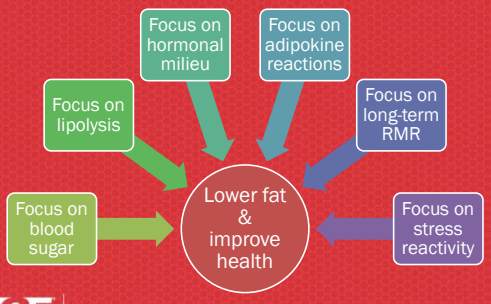
Newer Strategies to Battle the Buldge



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Don't Die first, Health Second, Looks and Fitness Third

- Reduce CVD risk factors to non-lethal levels
- Reduce negative health factors to safe (maybe high) levels
- Both reduce risks and gain factors leading to improved function
- Lose fat, gain muscle, and look hot!

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Conclusions

- By understanding all the factors involved with obesity, you can better address a holistic treatment plan for your client.
- Both hormones and intercellular messengers are affected by diet and exercise as well as stress, sleep, and the quality of food or drink we consume
- Inflammatory factors have a significant role in obesity and the various negative complications associated with it.
- Metabolic syndrome is an example of the interactive nature of physiological variables and disease states.
- Visceral adipose tissue seems to be uniquely associated with several CVD and Metabolic Syndrome disease factors

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Conclusions- cont.

- The Critical Visceral Adipose Tissue theory proposes that even small changes in VAT can restore insulin sensitivity and lipids.
- Exercise and diet can have profound affects on VAT, insulin resistance, hormones, inflammation, and adipokines, even if the cosmetic changes may be unnoticed.
  - HI exercise seems to be more effective to elicit these changes.
- Newer strategies in obesity management are concerned with the deadly factors and many forms they come in. Losing fat for cosmetic reasons should be a lower priority, but it is still important for health, mental outlook, and quality of life.

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