Obesity, Nutrition and Nutrigenomics

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School of Public Health and Community Medicine
University of Washington
Learning Objectives

1. Be familiar with the evidence for genetic influences on obesity

2. Understand how genetic factors can influence obesity, both directly and indirectly

3. Be familiar with concept of nutrigenomics

4. Be familiar with one current application of genomic information for public health practice
Outline

1. Background
2. Genetics of Obesity
   • Animals
   • Humans
   • Pharmacogenomics
3. Indirect genetic influences
   • Genetics of Taste
4. Nutrigenomics
5. Family History
6. Summary
Public Health Importance

• **Mortality**
  - Increased risk of premature death

• **Morbidity**
  - Diabetes, Heart disease, Hypertension, some Cancers, Breathing Problems, Ischemic Stroke, Arthritis, and Reproductive Complications

• **Prevalence**
  - 59 million (30%) Americans are obese (BMI >= 30)
  - Rates are increasing faster than ever (epidemic proportions)
Trends in Obesity

From the CDC website: BRFSS Trends Data
Trends in Obesity

Figure 2. Age-adjusted* prevalence of overweight and obesity among U.S. adults, age 20-74 years

%  

Overweight or obese (BMI ≥ 25.0)  
Obese (BMI ≥ 30.0)  

NHANES II (1976-80)  
(n=11,207)  

NHANES III (1988-94)  
(n=14,468)  

NHANES (1999-00)  
(n=3,601)  

47  
15  
23  
64  
31  

*Age-adjusted by the direct method to the year 2000 U.S. Census estimates using the age groups 20-39, 40-59, and 60-74 years.

From the CDC  
Website: NHANES  
Study Data
Trends in Obesity

*Source: Behavioral Risk Factor Surveillance System, CDC.*
Risk Factors for Obesity

- Diet: high calorie and low nutrient dense foods
- Physical Inactivity
- Age
- Socioeconomic status
- Certain medical conditions and medications
- Race
- Quitting smoking
- Family History
- Genetic susceptibility
Rodent Models of Obesity
Parabiosis Experiment: \textit{ob} and \textit{db} mice

Summary of parabiosis experiments with the \textit{ob/ob} and \textit{db/db} mice.

\textit{ob/ob}: leptin deficiency

\textit{db/db}: mutation in leptin receptor

By Pamela Hunt, Amgen Inc.
Available at: http://www.biotech-medecine.com/archives/review14/point
RODENT STRAINS WITH MONOGENIC FORMS OF OBESITY

<table>
<thead>
<tr>
<th>Strain</th>
<th>Inheritance</th>
<th>Encoded Protein</th>
<th>Defect</th>
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<tbody>
<tr>
<td>Obese (<em>ob</em>)</td>
<td>Mouse</td>
<td>Recessive</td>
<td>Leptin</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Stop codon/promoter defect in leptin</td>
</tr>
<tr>
<td>Diabetes (<em>db</em>)</td>
<td>Mouse</td>
<td>Recessive</td>
<td>Leptin receptor</td>
</tr>
<tr>
<td>Agouti yellow (<em>ay</em>)</td>
<td>Mouse</td>
<td>Dominant</td>
<td>Agouti</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ectopic expression of melanocortin receptor antagonist</td>
</tr>
<tr>
<td>Tubby (<em>tub</em>)</td>
<td>Mouse</td>
<td>Recessive</td>
<td>Phosphodiesterase</td>
</tr>
<tr>
<td>Fat</td>
<td>Mouse</td>
<td>Recessive</td>
<td>Carboxypeptidase E</td>
</tr>
<tr>
<td>Zucker/fatty (<em>fa</em>)</td>
<td>Rat</td>
<td>Recessive</td>
<td>Leptin receptor</td>
</tr>
<tr>
<td>Koletsky (<em>kol</em>)</td>
<td>Rat</td>
<td>Recessive</td>
<td>Leptin receptor</td>
</tr>
<tr>
<td>Corpulent (<em>cp</em>)</td>
<td>Rat</td>
<td>Recessive</td>
<td>Leptin receptor</td>
</tr>
</tbody>
</table>

From: Diabetes Mellitus A Fundamental and Clinical Text (3rd Ed.) D. LeRoith, S.I. Taylor and J.M. Olefsky, Lippincott Williams & Wilkins, 2004, Philadelphia
Evidence for genetic influences: Humans

- Familial aggregation
  - familial clustering of obesity in families

- Twin Studies
  - greater concordance among MZ twins compared to DZ twins

- Family Studies
  - variety of “statistical models” consistent with genetic influences
The Search for Obesity Susceptibility Genes

“I found one! I found one!”

Kenneth M. Weiss & Joseph D. Terwilliger
nature genetics • volume 26 • October 2000
Candidate Genes and Single Gene Disorders: Chromosomal Location

Image adapted from:
### MAJOR PHENOTYPIC FEATURES OF MONOGENIC FORMS OF HUMAN OBESITY

<table>
<thead>
<tr>
<th>Gene</th>
<th>Obesity</th>
<th>Birth weight</th>
<th>Endocrine abnormalities</th>
<th>Hyperphagia</th>
<th>Inheritance</th>
<th>Chromosome</th>
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<tr>
<td>LEP</td>
<td>Severe</td>
<td>Normal</td>
<td>Low leptin</td>
<td>+</td>
<td>Recessive</td>
<td>7q31.1</td>
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<td></td>
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<td></td>
<td>Hypogonadism</td>
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<td></td>
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<td></td>
<td>High thyroid-stimulating hormone</td>
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<td></td>
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<td></td>
<td>High insulin</td>
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<td></td>
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<td>High leptin</td>
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<td>Pituitary dysfunction</td>
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<td>LEPR</td>
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<td>Hypogonadotrophic hypogonadism</td>
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<td>Recessive</td>
<td>1p31</td>
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<td>High Insulin</td>
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<td>POMC</td>
<td>Severe</td>
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<td>Red hair pigmentation</td>
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<td>ACTH deficiency hypocortisolism</td>
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<td>Low α-MSH</td>
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<tr>
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<td></td>
<td>Hypogonadotrophic hypogonadism</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypocortisolism</td>
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<tr>
<td>PC1</td>
<td>Severe</td>
<td>?</td>
<td>High proinsulin, low insulin</td>
<td>?</td>
<td>Recessive</td>
<td>5q1.5-2.1</td>
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<td>Postprandial hypoglycemia</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>High POMC</td>
<td></td>
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<td>MC4-R</td>
<td>Severe</td>
<td>Normal</td>
<td>Not observed</td>
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<td>18q22</td>
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<td>NROB2</td>
<td>Mild</td>
<td>High</td>
<td>Mild hyperinsulinemia</td>
<td>-</td>
<td>Dominant</td>
<td>1p36.1</td>
</tr>
</tbody>
</table>

LEP, leptin; LEPR, leptin receptor; POMC, pro-opiomelanocortin; PC1, prohormone convertase1; MC4-R, melanocortin-4 receptor; ACTH, adrenocorticotropic hormone; α-MSH, α-melanocyte-stimulating hormone.

From: Diabetes Mellitus A Fundamental and Clinical Text (3rd Ed.) D. LeRoith, S.I. Taylor and J.M. Olefsky, Lippincott Williams & Wilkins, 2004, Philadelphia
Leptin Therapy: From mice to humans

Left: Ob mouse 6 weeks post leptin therapy
Right: Ob mouse 6 weeks post saline injections

A child with a mutation in the leptin gene before and after leptin therapy


Pharmacogenomics

• Tailoring of drug treatment according to individual genotype

• Certain medications are known to be more (or less) effective in patients with a particular genetic profile

• Example: Leptin therapy for obese patients with a mutation in their leptin gene

• Concerns:
  - Access
  - Increase in health disparities
Genetics of Human Obesity

• Common form(s) of obesity are likely due to complex interactions between genes and environment
  - body fat pattern
  - appetite regulation
  - other pathways

• Rare monogenic forms do not account for majority of cases

• Genes can also indirectly influence obesity through a variety of mechanisms
Supertasters: Food Dislikes

Sensitivity to bitter taste is heritable among nontasters, regular tasters and supertasters.

- raw cruciferous vegetables and some green vegetables (broccoli, cabbage, Brussels sprouts, spinach, kale)
- sharp cheeses
- dark chocolate
- Japanese green tea
- grapefruit and lemon juice
- dry wine
- beer
- black coffee / tea
- tonic water
Link between genetic taste markers and BMI?

• Inconsistent evidence for an association between taster status and BMI
  - no association between taster status and BMI
  - nontasters and regular tasters were heavier than supertasters after accounting for dietary restraint

• Many factors influence food preference and dietary intake

Public Health Implications:
Dietary intervention strategies aimed at improving diet quality should include taste preferences, as well as a wide range of demographic, economic and sociocultural variables
Nutrigenomics

Integrates genomics and nutrition

- **Goal:** Improving health and preventing disease through tailored diet and lifestyle prescriptions

- **Concerns:**
  - Private companies offering “genetic personalization products”
    - Personalized advice on nutritional requirements and optimal exercise/training programs
    - No evidence for efficacy
    - Misleading claims
    - Biobanking DNA
Nutrigenomics

The study of how different foods can interact with particular genes to increase the risk of diseases such as type 2 diabetes, obesity, heart disease and some cancers

Goal: Use of personalized diets to prevent or delay the onset of disease and optimize and maintain human health

http://nutrigenomics.ucdavis.edu/pressarticles.htm
Nutritional Genomics

Why the interest?

- Improve health of populations
  - United States
  - Globally

- Improve athletic performance

- Weight loss

- Potential economic impact
  - Functional food and dietary supplements is currently a $40 billion industry
  - The focus on nutrigenomics could mean an $80 billion dollar industry in 7-10 years
What is the evidence?

• Single Gene Disorders
  • PKU
  • Lactose intolerance

• Complex conditions
  • Genes involved in susceptibility to complex diseases have been identified
  • Nutritional environment modifies the expression of genes
  • Metabolism of nutrients may vary by genotype, ultimately affecting health
What is the public health application?

- Can we use this information along with our increasing knowledge of the genetics of obesity for public health applications?
  - Obesity epidemic
Direct to Consumer Marketing

- Health Clubs
- Vending Machines
- Internet
- Retail stores
Carolyn Katzin

Carolyn Katzin, MS, CNS is an internationally acclaimed nutritionist with twenty years of experience. Her advice is smart, practical, intuitive and individualized. Born in London, she has degrees in nutrition from University of London and from UCLA’s School of Public Health. Carolyn is a licensed nutritionist and a certified nutrition specialist.

Carolyn has created The DNA Diet™ a cutting edge health program providing you with highly specific information about your own unique molecular identity. Nutritional genetics provides a new standard of excellence in dietary advice. A safe, non-invasive screening tool combined with Carolyn’s expertise allows you to modify your diet based on your unique nutritional genetic profile. Carolyn helps you to optimize your risk of common diet related diseases such as heart disease, stroke and certain cancers. Personalized Nutrition or Nutritional Genetics is the most cutting edge way to be proactive about your health.

Special introductory offer: A confidential, safe, non-invasive DNA test is used to determine variations in genes involved in detoxification processes, bone health, heart health, antioxidant activity, inflammatory processes and insulin sensitivity. Based on these genetic results, dietary recommendations Carolyn creates a specific personalized diet action. The results are presented in a boxed, keepsake book. Carolyn can also provide you with an exceptionally effective six week diet program, the Rapid Results Diet, should you need to lose weight. The DNA Diet test and written action plan including menu and recipes is $600. The combined program is available for $1200.

Be the one of the first to have your own DNA diet. Contact Carolyn at carolyn@carolynkatzin.com or toll-free (866) 471 0529 for more information about this test.

"I was so impressed by my consultation with you. I know you have made a huge difference in my life and perhaps longevity."

Lynn P. May, 2005

KCBS - The DNA Diet feature piece during sweeps week May, 2005. Produced by Don Damrath and syndicated nationwide.

Los Angeles Times Health Section - Front page story "Are the clues to diet success in your genes?" by Hilary MacGregor, April 11, 2005

Washington Post - "Labs turn DNA into Personal Health Forecasts" includes several quotes from
DNA Diet Builds Customized Weight-Loss Plan
One-size-fits-all diets could be a thing of the past. NBC station KNSD in San Diego reported that a handful of bio-tech companies are promising a high-tech recipe for losing weight and eating better. The newest weight-loss plan is a customized diet based on your DNA. The DNA diet is a personalized meal plan that claims to be based on your unique genetic blueprint.

Katzin claims that based on your DNA profile she can “determine whether someone should increase the amount of folic acid, B-6 or B-12, for example. So, we would choose foods that are rich in those supplements.”

… “interprets the data and makes a customized meal plan. Her suggestions range from “taking more vitamins to eating more meat.”
Sciona

- International company previously based in the UK
  - Personalized health and nutrition recommendations
  - Products were available through retail stores
- GeneWatch UK called on retail stores to stop offering these tests
- Currently based in Boulder, Colorado
  - Launching a campaign in 4 test markets
  - Partnerships with retail stores and local health care system
In store sales

Sciona
Optimal health through genetics

About Sciona  Products  Scientific Leadership  Commercial Leadership  Ethical Leadership

Current Focus  Industry News & Events  Future Applications  Retail Partners

Home » Commercial Leadership » Retail Partners

Retail Partners

www.pharmacacom
www.eq-life.com
www.prairiestonerx.com
www.lundsmarket.com
www.byerlys.com
www.hy-vee.com
www.ukrops.com
Lund Foods CEO: “…plan is to create a link between the evaluations performed by Sciona and his stores’ food experts, which have long provided consumers with diet and nutritional advice and information.”

Today Food Editor: “The idea, which is a good one, is to help shoppers understand what they can do in their daily food choices to either maintain their good health or help correct certain genetic defects that the test may have identified.”
Heart Health

• “Analyzes thirteen of your genes that may play an important role in determining how your body manages overall heart health”

• “…assesses nine key diet and lifestyle action areas”

<table>
<thead>
<tr>
<th>Gene Analyzed</th>
<th>Role of the Gene in Heart Health</th>
<th>Genetic Variation Screened For Variations Found in Your Gene</th>
<th>Percentage of Population with this Gene Variation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>MTHFR</td>
<td>Use of Folic Acid for DNA Synthesis or DNA Repair</td>
<td>C677T A1298C</td>
<td>28.7 30.0</td>
</tr>
<tr>
<td>MS_MTRR</td>
<td>Metabolism of Vitamin B12</td>
<td>A66G</td>
<td>47.3</td>
</tr>
<tr>
<td>MTR</td>
<td>Removal of Homocysteine</td>
<td>A2756G</td>
<td>17.4</td>
</tr>
<tr>
<td>CBS</td>
<td>Metabolism of Vitamin B6 and Removal of Homocysteine</td>
<td>C699T</td>
<td>28.0</td>
</tr>
<tr>
<td>MnSOD</td>
<td>Antioxidant Defense</td>
<td>C(-28)T</td>
<td>54.2</td>
</tr>
<tr>
<td>SOD3</td>
<td></td>
<td>T175C</td>
<td></td>
</tr>
<tr>
<td>IL-6</td>
<td>Inflammatory Response</td>
<td>G(-174)C</td>
<td>36.3</td>
</tr>
<tr>
<td>TNF-α</td>
<td></td>
<td>G(-308)A</td>
<td>16.5</td>
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<tr>
<td>APOC3</td>
<td>Triglyceride Metabolism</td>
<td>C3175G</td>
<td>12.6</td>
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<tr>
<td>CETP</td>
<td>Cholesterol</td>
<td>G279A</td>
<td>37.0</td>
</tr>
<tr>
<td>LPL</td>
<td>Metabolism</td>
<td>C1595G</td>
<td>9.9</td>
</tr>
<tr>
<td>eNOS</td>
<td>Blood Flow</td>
<td>G894T</td>
<td>35.6</td>
</tr>
<tr>
<td>ACE</td>
<td></td>
<td>DEL</td>
<td>61.0</td>
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</tbody>
</table>

*The population frequencies given are normalized for the U.S. population data from the U.S. 2002 Census Report. Population frequencies can vary for different ethnic groups, so for more detailed information, please turn to the Population Frequency Data Table in the Reference Section of your report.
Insulin Resistance

- “Analyzes five of your genes that may play an important role in determining how your body manages overall insulin resistance”
- “...assesses five key diet and lifestyle action areas”

<table>
<thead>
<tr>
<th>Gene Analyzed</th>
<th>Role of the Gene in Insulin Resistance</th>
<th>Genetic Variation Screened For Variations Found in Your Gene</th>
<th>Percentage of Population with this Gene Variation*</th>
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<tbody>
<tr>
<td>VDR</td>
<td>Mechanism of Insulin Secretion</td>
<td>CTAqIT</td>
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<tr>
<td>VDR</td>
<td></td>
<td>TBsm1C</td>
<td>69.6</td>
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<td>IL-6</td>
<td>Inflammatory Response; Response of Cells to Insulin</td>
<td>G(-174)C</td>
<td>36.3</td>
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<tr>
<td>TNF-α</td>
<td></td>
<td>G(-308)A</td>
<td>16.5</td>
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<tr>
<td>PPARγ</td>
<td>Glucose and Lipid Metabolism</td>
<td>Pro12A</td>
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<td>ACE</td>
<td>Blood Pressure Regulation</td>
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Inflammation Health

• “Analyzes six of your genes that may play an important role in determining how your body manages inflammation”

• “...assesses four key diet and lifestyle action areas”

<table>
<thead>
<tr>
<th>Gene Analyzed</th>
<th>Role of the Gene in Inflammation</th>
<th>Genetic Variation Screened For Variations Found in Your Gene</th>
<th>Percentage of Population with this Gene Variation*</th>
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<tbody>
<tr>
<td>GSTM1</td>
<td>Detoxification</td>
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<td>GSTP1</td>
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<td>34.8</td>
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<tr>
<td>GSTT1</td>
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<td>(DEL)</td>
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<tr>
<td>MnSOD</td>
<td>Destroys Free Radicals</td>
<td>C(-28)T</td>
<td>54.2</td>
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On the internet

Genovations™ Profiles

CardioGenomic™ Profile
Evaluates genetic variations (SNPs) that modulate blood pressure regulation, lipid balance, nutrient metabolism, inflammation, and oxidative stress.

OsteoGenomic™ Profile
Evaluates SNPs that modulate bone formation (collagen synthesis), bone breakdown (resorption), and inflammation, including key regulatory mechanisms affecting calcium and Vitamin D3 metabolism.

DetoxiGenomic™ Profile
Evaluates SNPs associated with increased risk of impaired detoxification capacity especially when exposed to environmental toxins. It also identifies individuals potentially susceptible to adverse drug reactions.

ImmunoGenomic™ Profile
Evaluates SNPs that modulate immune and inflammatory activity. Polymorphisms affect the levels and activity of the cytokines. These variations can affect balance between cell (TH-1) and humoral (TH-2) immunity, reveal potential defects in immune system defense, and stimulate mechanisms leading to chronic, overactive inflammatory responses.

NeuroGenomic™ Profile
Evaluates SNPs that modulate methylation, glutathione conjugation and oxidative protection.

©2002 Genovations™, a product of Great Smokies Diagnostic Laboratory
## Gene Analyzed

<table>
<thead>
<tr>
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*The population frequencies given are normalized for the U.S. population data from the U.S. 2002 Census Report. Population frequencies can vary for different ethnic groups, so for more detailed information, please turn to the Population Frequency Data Table in the Reference Section of your report.
Consumer Demand?

• Sciona claims to have sold 10,000 kits in Europe, Asia and the US

• Current use is likely limited to those who can afford to pay

• HealthSyles Survey indicates that only 14% of US population are aware of these tests, and only 0.6% have used a test
  - age and income are associated with awareness (Goddard et al., GIM 2007;9:510-7)
Why the Concern?

• Not regulated by the Food and Drug Administration (FDA)

• Genetic information is unlike other health information, in that it also provides information about your family members

• Some companies make dubious claims about how the kits not only test for disease but also serve as tools for customizing medicine, vitamins, and foods to each individual's genetic makeup (doegenomes.org)
“Buyer Beware”

A recent report by the Government Accountability Office highlighted a few of the concerns with DTC nutrigenomic tests.

• may mislead consumers by making unsound and ambiguous predictions about health risks

• purchase dietary supplements that may be significantly overpriced compared with similar products available through a supermarket or pharmacy

• supplement use may be harmful for some people
Potential Benefits

• Increased focus on a healthy diet and lifestyle
• Motivate positive behavior change
• Increased awareness of risk of certain conditions
• Improved health and quality of life
• Focus on prevention
• Decreased morbidity and premature mortality
• Reduced health care costs
• Identify subgroups who might be particularly responsive or resistant to environmental (dietary) intervention
• Better understanding of the mechanisms involved in disease susceptibility
Potential Harms

• Attention is drawn away from other modifiable risk factors
• Decreased use of other services
• False sense of security
• Focus on specific nutrients/foods
• Ineffective or harmful
• Misleading claims
• Dilute or contradict public health messages
Potential Harms, cont.

• Increased costs associated with personalized diets and designer foods
• Targeting vulnerable populations
• Concerns surrounding confidentiality, insurance
• Biobanking of samples, informed consent
• Unintended consequences
Prevention Efforts

1. Dietary modification coupled with regular physical activity
   - Modest enough to be acceptable, but robust enough to produce meaningful changes
   - Maintain healthier habits

2. Approach
   - All Inclusive population approach?
     - general educational campaign
   - Target specific sub-groups?
     - incentive to follow regime should be higher than for general population

3. Genomic Tools
   - Family history as a bridge from genetics to genomics
Positive Family History and Risk of Obesity

Relative Risk

Study

(1st author, at risk group, affected relative)
Rationale for using Family History as a Public Health Tool

1. Screening for single major gene(s) is unlikely
2. Reflects unique Genomic information
   - genomic, ecologic, behavioral and interactions
3. Effective interventions
4. Identify individuals for targeted intervention
5. Family-Centered approaches
Family Medical History

• Several family medical history initiatives
  - CDC Family History Initiative
  - National Coalition for Health Professional Education in Genetics (NCHPG)
  - Surgeon General’s public health campaign

• “Surgeon General’s American Family Health Initiative”
  - Thanksgiving
  - web and paper based tool to collect family medical history information
Conclusions

• Obesity is influenced by both genes AND environment

• Obesity is associated with poverty, SES and education

• Diet is important
  • High-fat energy-dense foods are often the cheapest options for the consumer
  • Health foods cost more

• Nutrigenomic testing is not ready for prime time
Implications

Potential Benefits of genetic information:
• Identify subgroups who might be particularly responsive or resistant to environmental or pharmacologic intervention

• Better understanding of the mechanisms involved in obesity

• Motivate positive behavior change

Potential Harms of genetic information:
• Focus away from environmental factors can have negative consequences

• Focus on “genetic” factors can lead to fatalism

• Potential for increase in health disparities
UW Center for Genomics and Public Health

http://depts.washington.edu/cgph

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Centers for Disease Control and Prevention