Childhood Obesity: A Lifelong Course

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Outline

- Childhood obesity and risk of adult obesity
- Childhood obesity and chronic disease risks (e.g. CVD risks)
- Obesity and cancer risk
- Examples of national efforts to curtail the obesity epidemic (e.g., NIH Obesity Task Force, WE CAN program)
- Potential opportunities for Maryland
Childhood Obesity—
a Lifelong Course
DEFINING OBESITY IN CHILDHOOD

Body mass index-for-age percentiles:
Boys, 2 to 20 years

A 10-year-old boy with a BMI of 23 would be in the obese category (95th percentile or greater).

A 10-year-old boy with a BMI of 21 would be in the overweight category (85th to less than 95th percentile).

A 10-year-old boy with a BMI of 18 would be in the healthy weight category (5th percentile to less than 85th percentile).

A 10-year-old boy with a BMI of 13 would be in the underweight category (less than 5th percentile).

BMI = \frac{\text{Weight (kg)}}{\text{Height (m}^2\text{)}}

OR

BMI = \frac{\text{[Weight (lbs)] \times 703}}{\text{[Height (in}^2\text{)]}}

BMI 30 or above = obese; BMI 25-29.9 = overweight; BMI 18.5-24.9 = normal
Figure 1.

Childhood Obesity Epidemic

Percent Overweight

<table>
<thead>
<tr>
<th>Year</th>
<th>Boys</th>
<th>Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td>1966-1970</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>1971-1974</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>1976-1980</td>
<td>5</td>
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<td>1988-1994</td>
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<td>1999-2000</td>
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<tr>
<td>2001-2002</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>2003-2004</td>
<td>20</td>
<td>20</td>
</tr>
</tbody>
</table>

Multiple Factors Contributing to Childhood Obesity

Energy Intake greater than Energy Output

- Excessive Increase in Body fat Leading to Obesity
  - Risk for heart disease, cancer, other chronic diseases

Factors:
- Adverse media influences
- Individual factors: e.g., poor sleep patterns
- Cultural & Socio-Economic factors
- Built environment: e.g., walkability and safety of neighborhoods
- School and neighborhood environments: e.g., peer, sibling, and social networks
- Home and family environments: (TV, sedentary behavior, sweetened beverages, fast foods)
- Individual biologic and physiologic factors

Diseases: e.g., type 2 diabetes and high blood pressure
Does being overweight in childhood increase your disease risks in adulthood?

The Bogalusa Heart Study (BHS)

- **Objectives:** To investigate the early natural history of cardiovascular disease in a cohort of children and young adults in a biracial, semirural community in Louisiana; More than 16,000 individuals. [http://www.nhlbi.nih.gov/resources/deca.descriptions/bhs.htm](http://www.nhlbi.nih.gov/resources/deca.descriptions/bhs.htm)

- Systolic and diastolic blood pressure, total cholesterol, LDL cholesterol, HDL cholesterol all track from childhood through adolescence to adulthood.

- Weight change (normal weight to obese) exerts powerful effect

- **Answer:** YES; about 50%-60% of obese children become obese adults

Does being overweight in childhood increase your disease risks in adulthood?

**Harvard Growth Study**

- Risk of mortality of males and females overweight during high school years; 1800 children.
- In Males: All cause mortality 1.8; CHD 2.3, Stroke 13.2, Colorectal cancer 9.1
- In Females: Risks are lower than males: All cause mortality 1.0; CHD 0.8, Stroke 0.4, Colorectal cancer 1.0, Breast cancer 0.9.
- Psychosocial consequences: fewer years of education, lower family incomes, higher rates of poverty, lower self-esteem.

Obesity and Health Risks

- Heart disease
- High Blood Pressure
- Type-2 Diabetes
- Asthma & Sleep apnea
- Obesity Health Risks
- Musculoskeletal Disorders - e.g., osteoarthritis
- Depression
- Low self esteem
- Renal diseases
- Cancer
- Left Ventricular Hypertrophy
- Type-2 Diabetes
- High Blood Pressure
- Asthma & Sleep apnea
- Cancer
- Left Ventricular Hypertrophy
YOUNG LIVES AT RISK: Our Overweight Children

How Obesity Harms A Child's Body

For the first time in history, American children could have a shorter life span than their parents. The cause: obesity. With about a third of all youths overweight or worse, adverse health effects are being seen in alarming proportions. And medical experts fear those problems foreshadow what tomorrow’s young adults will face as the years of excess pounds add up.

So what happens inside a child or teen carrying this kind of load? As this organ-by-organ summary shows, obesity kills slowly, causing damage from head to toe, with painful lasting effects.

FAT AND THE BODY
A fat cell is like a plastic bag that holds a drop of fat. The number of fat cells a person has is determined by his adolescence — overeating in childhood creates more. The cells increase in size depending on how much fat they store.

So although overweight children can become lean (as their fat cells shrink), they do not lose the extra fat cells no matter how much weight they lose.

LOSEING WEIGHT
Weight is determined by the rate at which the body stores energy from the food one eats and the rate at which that energy is used. When one is not eating, food is not absorbed. However, the body is always using energy, and the energy must come from internal reserves.

WHAT IS BMI?
Body mass index is a measure of weight in relation to height that is used to estimate a person’s body fat and, by extension, health risks. BMI is the most widely accepted method used to screen for overweight in children and adolescents, but it is not an actual measure of body fat. Being “normal” means that a child has a BMI at or above the 50th percentile for his or her age. A BMI of at or above the 85th percentile is labeled “overweight.”

SOURCES: Jennifer Miller, assistant professor of pediatrics, University of Florida; Jeffrey Goldsmith, pediatric endocrinologist, University of California at San Diego; surgeon Matt Hummert, Children’s National Medical Center; Lou Kandel, pediatric orthopedic surgeon, Texas Scottish Rite Hospital for Children; Robert E. Layman, pediatric endocrinologist, University of California at San Francisco; Angela Deak, pediatric endocrinologist, Washington University St. Louis; New England Journal of Medicine; William Mandarca, senior vice president, Theorem Research, Stephen Curry, professor of pediatrics, University of Rochester; Paul Koplowitz, chief of endocrinology, Children’s National Medical Center; William H. Dietz, director of the Edean of Nutrition, Physical Activity and Obesity, federal Center for Disease Control and Prevention; David S. Ludwig, obesity program director, Children’s Hospital Boston; Matthew Gillman, associate professor of ambulatory care and prevention, Harvard Medical School; Steven Doerrakis, professor of health policy, Harvard School of Public Health; Michael Rosen, professor of preventive medicine, University of Southern California; Dania Caprio, pediatric endocrinologist, Yale University School of Medicine; Francis Newcomb, director of the Comprehensive Child Health Behavior Center, Children’s Hospital Los Angeles; David S. Katz, professor of medicine, University of Medicine and Dentistry, John Melmon, associate professor of pediatrics, Brook Center at Cincinnati Children’s Hospital Medical Center; Fred W. Stawicki, chief of the division of metabolism, John Hopkins Children’s Center; Institute of Medicine, National Institutes of Health; Journal of the American College of Physicians.

REPORTER: Susan Levine, Andrea Maloney, Peggy Scheller and Rob Stein, WASHINGTON POST - The Washington Post

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Childhood obesity and chronic disease risks

Psychosocial\(^{17-20}\)
- Poor self-esteem
- Depression
- Eating disorders

Pulmonary\(^{22-24}\)
- Sleep apnoea
- Asthma
- Exercise intolerance

Gastrointestinal\(^{30,31}\)
- Gallstones
- Steatohepatitis

Renal\(^{36}\)
- Glomerulosclerosis

Musculoskeletal\(^{37-40}\)
- Slipped capital femoral epiphysis
- Blount’s disease
- Forearm fracture
- Flat feet

Cardiovascular\(^{25-29}\)
- Dyslipidaemia
- Hypertension
- Coagulopathy
- Chronic inflammation
- Endothelial dysfunction

Neurological\(^{21}\)
- Pseudotumor cerebri

Endocrine\(^{32-35}\)
- Type 2 diabetes
- Precocious puberty
- Polycystic ovary syndrome (girls)
- Hypogonadism (boys)

Courtesy of Linda Nebeling
Childhood obesity, risk of adult obesity, and chronic disease risk

Metabolic
- Type 2 diabetes mellitus
- Metabolic syndrome

Orthopedic
- Slipped capital femoral epiphysis
- Blount’s disease

Cardiovascular
- Dyslipidemia
- Hypertension
- Left ventricular hypertrophy
- Atherosclerosis

Psychological
- Depression
- Poor quality of life

Neurological
- Pseudotumor cerebri

Hepatic
- Nonalcoholic fatty liver disease
- Nonalcoholic steatohepatitis

Pulmonary
- Obstructive sleep apnea
- Asthma (exacerbation)

Renal
- Proteinuria

Source: Daniels et al. Circulation, 2007
Obesity and cancer risk
Established or suspected obesity-related cancers:

**Sufficient evidence in humans**
- Colon
- Breast (postmenopausal)
- Endometrium
- Kidney (renal cell)
- Esophagus (adenocarcinoma)

**Sufficient or limited evidence in animal studies**
- Liver
- Mammary gland
- Pituitary gland (adenoma)
- Prostate
- Multiple Myeloma

## Risk of cancer associated with a 5 kg/m² increase of BMI

<table>
<thead>
<tr>
<th>Women Relative Risk</th>
<th>Men Relative Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Endometrial</td>
<td>1.59</td>
</tr>
<tr>
<td>• Gallbladder</td>
<td>1.59</td>
</tr>
<tr>
<td>• Renal</td>
<td>1.34</td>
</tr>
<tr>
<td>• Esophageal</td>
<td>1.51</td>
</tr>
<tr>
<td>• Leukemia</td>
<td>1.17</td>
</tr>
<tr>
<td>• Thyroid</td>
<td>1.14</td>
</tr>
<tr>
<td>• Post-menopausal</td>
<td>1.12</td>
</tr>
<tr>
<td>• Breast cancer</td>
<td>1.12</td>
</tr>
<tr>
<td>• Pancreas</td>
<td>1.12</td>
</tr>
<tr>
<td>• Non-Hodgkin’s</td>
<td>1.07</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>• Oesophageal</td>
<td></td>
</tr>
<tr>
<td>adenocarcinoma</td>
<td>1.52</td>
</tr>
<tr>
<td>• Thyroid</td>
<td>1.33</td>
</tr>
<tr>
<td>• Colon</td>
<td>1.24</td>
</tr>
<tr>
<td>• Renal</td>
<td>1.24</td>
</tr>
<tr>
<td>• Malignant melanoma</td>
<td>1.17</td>
</tr>
<tr>
<td>• Multiple myeloma</td>
<td>1.11</td>
</tr>
<tr>
<td>• Rectal cancer</td>
<td>1.09</td>
</tr>
<tr>
<td>• Leukemia</td>
<td>1.08</td>
</tr>
<tr>
<td>• Non-Hodgkin’s</td>
<td></td>
</tr>
<tr>
<td>lymphoma</td>
<td>1.06</td>
</tr>
</tbody>
</table>

Mortality from cancer according to BMI for US men in the cancer prevention Study II, 1982-1998

Mortality from cancer according to BMI for US women in the cancer prevention Study II, 1982-1998

Possible Mechanisms

Source: Calle EE & Thun MJ. Obesity and Cancer, Oncogen, 23, 6365-6378, 2004

- Obesity, especially abdominal obesity
- High Blood Insulin and Insulin Resistance
- Reduced Sex Hormone Binding Proteins
- ↑Free Circulating Estrogen
- Breast and Colon Cancer

Mostly in men and post-menopausal women

Through Insulin Growth Factors
Examples of National Efforts to Curtail the Obesity Epidemic
We Can!™ An Evidence-based Program to Help Children and Families Maintain Healthy Weight

NIH Science working through Communities, Partnerships and Media…

Communities
- Curricula
- Local Partnerships
- Local Media
- Outreach Events

Partnerships
- Federal
- Clinical
- Outreach
- Media
- Corporate

Media
- Web
- Print
- Television

…to help children and families maintain a healthy weight.
**We Can! Around the Country**

- **We Can!** is currently running in 920 Community Sites in 50 states, the District of Columbia, Puerto Rico, the Northern Mariana Islands, and 9 other countries (Australia, Bangladesh, Canada, Fiji, Greece, India, Israel, Nigeria, the Philippines, and Uganda). Settings include schools, park and recreation departments, hospitals, health systems and public health departments (12 different settings).

13 **We Can!** cities, including Boston, Pittsburgh, and Las Vegas are coordinating intensive We Can! programming for employees, parents and youth.

- More than 40 National and Corporate Partners
We Can! Resources to Mobilize Communities

Science-based information around We Can’s 4P’s

From a National Web Platform
http://wecan.nhlbi.nih.gov
Resources to Curtail the Obesity Epidemic

- NIH Obesity Research: Strategic Plan
  http://obesityresearch.nih.gov
- Centers for Disease Control and Prevention
  http://www.cdc.gov/nccdphp/dnпа/obesity/state_programs/index.htm
Resources to Curtail the Obesity Epidemic

Continued

- Robert Wood Johnson Foundation (Goal: to reverse childhood obesity epidemic by 2015)
  http://www.rwjf.org/childhoodobesity/index.jsp

- Evidence-based Physical Activity Guidelines from DHHS

- USDA/DHHS Dietary Guidelines for Americans
  www.healthierus.gov/dietaryguidelines
Potential Opportunities for Maryland

- Implement the physical activity guidelines in all schools and communities. Encourage children to spend 60 minutes (1 hour) or more on moderate to vigorous physical activity daily. [http://www.cdc.gov/HealthyYouth/physicalactivity/guidelines.htm](http://www.cdc.gov/HealthyYouth/physicalactivity/guidelines.htm)

- Encourage healthy eating habits in school, home and when eating out—[www.healthierus.gov/dietaryguidelines](http://www.healthierus.gov/dietaryguidelines)

- Adopt the We CAN program in schools, homes and communities in Maryland.

- Learn from other states, for example, Arkansas.

- Leverage resources and funds from other national programs: The CDC and RWJF obesity programs.
Other Resources: Dissemination into Practice

http://cancercontrolplanet.cancer.gov
Links to comprehensive cancer control resources for public health professionals
There is a lot to do!

To move from this.......
To this........
Acknowledgement

- Linda Nebeling, PhD, MPH, RD, FADA
  National Cancer Institute, nebelinl@mail.nih.gov

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- Eileen Charneco, MS (Epidemiology Intern), Division of Prevention and Population Sciences, National Heart, Lung, and Blood Institute
Risk estimates by cancer sites in Men

Risk estimates by cancer sites in Women

<table>
<thead>
<tr>
<th>Cancer site and type</th>
<th>Number of studies</th>
<th>RR (95% CI)</th>
<th>P</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endometrium</td>
<td>19</td>
<td>1.59 (1.50-1.68)</td>
<td>&lt;0.0001</td>
<td>77%</td>
</tr>
<tr>
<td>Gallbladder</td>
<td>2</td>
<td>1.59 (1.02-2.47)</td>
<td>0.04</td>
<td>67%</td>
</tr>
<tr>
<td>Oesophageal adenocarcinoma</td>
<td>3</td>
<td>1.51 (1.31-1.74)</td>
<td>&lt;0.0001</td>
<td>0%</td>
</tr>
<tr>
<td>Renal</td>
<td>12</td>
<td>1.34 (1.25-1.43)</td>
<td>&lt;0.0001</td>
<td>45%</td>
</tr>
<tr>
<td>Leukaemia</td>
<td>7</td>
<td>1.17 (1.04-1.32)</td>
<td>0.01</td>
<td>80%</td>
</tr>
<tr>
<td>Thyroid</td>
<td>3</td>
<td>1.14 (1.06-1.23)</td>
<td>0.001</td>
<td>5%</td>
</tr>
<tr>
<td>Postmenopausal breast</td>
<td>31</td>
<td>1.12 (1.08-1.16)</td>
<td>&lt;0.0001</td>
<td>64%</td>
</tr>
<tr>
<td>Pancreas</td>
<td>11</td>
<td>1.12 (1.02-1.22)</td>
<td>0.01</td>
<td>42%</td>
</tr>
<tr>
<td>Multiple myeloma</td>
<td>6</td>
<td>1.11 (1.07-1.15)</td>
<td>&lt;0.0001</td>
<td>0%</td>
</tr>
<tr>
<td>Colon</td>
<td>19</td>
<td>1.09 (1.05-1.13)</td>
<td>&lt;0.0001</td>
<td>39%</td>
</tr>
<tr>
<td>Non-Hodgkin lymphoma</td>
<td>7</td>
<td>1.07 (1.00-1.14)</td>
<td>0.05</td>
<td>47%</td>
</tr>
<tr>
<td>Liver</td>
<td>1</td>
<td>1.07 (0.55-2.08)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastric</td>
<td>5</td>
<td>1.04 (0.90-1.20)</td>
<td>0.56</td>
<td>4%</td>
</tr>
<tr>
<td>Ovarian</td>
<td>13</td>
<td>1.03 (0.99-1.08)</td>
<td>0.30</td>
<td>55%</td>
</tr>
<tr>
<td>Rectum</td>
<td>14</td>
<td>1.02 (1.00-1.05)</td>
<td>0.26</td>
<td>0%</td>
</tr>
<tr>
<td>Malignant melanoma</td>
<td>5</td>
<td>0.96 (0.92-1.01)</td>
<td>0.05</td>
<td>0%</td>
</tr>
<tr>
<td>Premenopausal breast</td>
<td>20</td>
<td>0.92 (0.88-0.97)</td>
<td>0.001</td>
<td>39%</td>
</tr>
<tr>
<td>Lung</td>
<td>6</td>
<td>0.80 (0.66-0.97)</td>
<td>0.03</td>
<td>8.4%</td>
</tr>
<tr>
<td>Oesophageal squamous</td>
<td>2</td>
<td>0.57 (0.47-0.69)</td>
<td>&lt;0.0001</td>
<td>60%</td>
</tr>
</tbody>
</table>

Figure 4: Summary risk estimates by cancer sites in women