PREVALENCE AND CAUSALITY OF OBESITY

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OBESITY IN THE MANKIND HISTORY

Venus of Věstonice
30,000 B.C.

Baroque sculpture
17. century
THE GLOBAL EPIDEMIC OF OBESITY

Report of a WHO Consultation on Obesity. WHO/NUT/NCD, Geneva No 894, 2000,

Data 2005:
400 million adults obese (BMI ≥ 30)
1.6 billion adults overweight (BMI ≥ 25)

Projection 2015:
700 million adults obese
2.3 billion adults overweight
CHARACTERISATION OF OBESITY AND DEFINITION OF BODY MASS INDEX (BMI)

• Obesity is a chronic disease characterised by an increase of body fat stores (>25% in men, >30% in women)

• In clinical practice, body fatness is assessed by the body mass index

• Body mass index (BMI) is calculated: measured body weight (kg)/measured height (m²)
**CLASSIFICATION OF OVERWEIGHT AND OBESITY (BMI) AND BODY FAT DISTRIBUTION (WAIST CIRCUMFERENCE)**

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>BMI kg/m²</th>
<th>DISEASE RISK* Waist Circumference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>&lt;102 cm (men)</td>
</tr>
<tr>
<td>Underweight</td>
<td>&lt;18.5</td>
<td>Increased</td>
</tr>
<tr>
<td>Normal range</td>
<td>18.5 – 24.9</td>
<td>Low</td>
</tr>
<tr>
<td>Overweight</td>
<td>25.0 – 29.9</td>
<td>Increased</td>
</tr>
<tr>
<td>Obesity (grade I)</td>
<td>30.0 – 34.9</td>
<td>High</td>
</tr>
<tr>
<td>Obesity (grade II)</td>
<td>35.0 – 39.9</td>
<td>Very High</td>
</tr>
<tr>
<td>Obesity (grade III)</td>
<td>&gt;40.0</td>
<td>Extremely High</td>
</tr>
</tbody>
</table>

* International Diabetes Federation (2005) declares more strict cut-off points for waist circumference: ≥94 cm in men and ≥80 cm in women.
WAIST CIRCUMFERENCE AS A MEASURE OF VISCERAL FAT

Waist circumference is measured midway between the upper iliac crest and the lower rib.
## Country-Ethnicity-Specific Values for Waist Circumference Defining Abdominal Obesity

*International Diabetes Federation (2005)*

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Europids</td>
<td>≥94 cm</td>
<td>≥80 cm</td>
</tr>
<tr>
<td>South Asians</td>
<td>≥90 cm</td>
<td>≥80 cm</td>
</tr>
<tr>
<td>Chinese</td>
<td>≥90 cm</td>
<td>≥80 cm</td>
</tr>
<tr>
<td>Japanese</td>
<td>≥85 cm</td>
<td>≥90 cm</td>
</tr>
</tbody>
</table>
PREVALENCE OF OBESITY in most European countries is in the range 10-20% in men and 15-30% in women (IOTF 2005)
PREVALENCE OF OVERWEIGHT IN CHILDREN AND ADOLESCENTS IS INCREASING

IOTF/EASO Obesity in Europe Report 2002; IOTF 2004
TRENDS IN OBESITY PREVALENCE (IOTF 1999)
HEALTH RISKS OF OBESITY

Obesity - besides smoking, the most important cause of death which could be prevented

**METABOLIC**
- Diabetes type 2
- Dyslipidaemia
- Hyperuricaemia, gout

**CARDIOVASCULAR**
- Hypertension
- CHD, heart failure
- Cerebral stroke
- Thromboembolism

**BRONCHOPULMONARY**
- Asthma
- Sleep apnoea syndrome
- Hypoventilation syndrome

**GASTROINTESTINAL**
- Gastroesophageal reflux disease
- Non-alcoholic hepatic steatosis
- Hernias

**TUMOURS**
- Breast, uterine, ovarian cancer
- Colon cancer, prostatic cancer

**OTHERS**
- Polycystic ovary syndrome
- Infertility/amenorrhoea
- Urinary incontinence
- Osteoarthritis, spondylitis
- Depression, anxiety
- Eating disorders
- Dermatological diseases

**OBESITY PREVENTS**
- Osteoporosis

OBESITY PREVENTS OSTEOPOROSIS
OBESITY INCREASES RISK FOR DM2
MOST (BMI $\geq$35 vs <22 kg/m$^2$)

77,690 females and 46,060 males adjusted for age, smoking, race, 10-year risk

Field AE, Arch Intern Med 2001; 161: 1581-1586
RELATIONSHIP BETWEEN BMI AND MORTALITY
American Cancer Society Prevention Study

METABOLIC SYNDROME WORLDWIDE DEFINITION

International Diabetes Federation (2005)

- **Waist circumference (ethnicity specific)** + Any two of the following

- ↑ Triglycerides ≥ 1.7 mmol/l (150 mg/dl) or treated dyslipidaemia

- ↓ HDL-cholesterol or treated dyslipidaemia
  - Males < 1.0 mmol/l (40 mg/dl)
  - Females < 1.3 mmol/l (50 mg/dl)

- ↑ Blood pressure ≥ 130 / ≥ 85 mm Hg or treated hypertension

- ↑ fasting plasma glucose ≥ 5.6 mmol/l (100 mg/dl) or previously diagnosed type 2 diabetes
ABDOMINAL (VISCERAL) OBESITY AND CARDIOVASCULAR HEALTH RISKS

Visceral obesity

- Dyslipidaemia
  - HDL-cholesterol ↓
  - Triglyceride ↑
  - Total cholesterol ↑
  - LDL-cholesterol ↑
  - Apo-B ↑

- Endothelial dysfunction

- Trombogenesis
  - Fibrinogen ↑
  - PAI-1 ↑

- Inflammatory response
  - Cytokines ↑ (TNFα)

- Liver and muscle fatty infiltration

- Hypertension
  - Left heart hypertrophy
  - Heart failure

- Insulin resistance
  - Hyperglycaemia
  - DM2

- Renal hyperfiltration
  - Albuminuria

Sharma AM 2002
SYMPTOMS AND SECONDARY DISORDERS ASSOCIATED WITH ABDOMINAL OBESITY EVALUATED BY WAIST CIRCUMFERENCE IN MEN

HIGH PREVALENCE OF TYPE 2 DM AND METABOLIC SYNDROME IN MAURITIUS

Recent Studies

- Nyamdorj R et al.: BMI Compared With Central Obesity Indicators as a predictor of Diabetes Incidence in Mauritius. Obesity 2008 Nov 13
- Cameron AJ et al.: Central Obesity as a Precursor to the Metabolic Syndrome in the AusDiab Study and Mauritius Obesity 2008 Sept 25
ADIPOSE TISSUE
LARGEST ENDOCRINE ORGAN IN THE BODY
CYTOKINES PRODUCED BY ADIPOCYTE ARE RELATED TO THE RISKS OF METABOLIC SYNDROME
PATHOGENESIS OF OBESITY
ACCUMULATION OF BODY FAT STORES RESULTS FROM IMBALANCE BETWEEN ENERGY INTAKE AND ENERGY EXPENDITURE

ENVIRONMENTAL & SOCIETAL INFLUENCES

INDIVIDUAL / BIOLOGICAL SUSCEPTIBILITY
GENES

DIETARY & PHYSICAL ACTIVITY PATTERNS

ENERGY BALANCE
IN
CHO
FAT
P

OUT
EE-PA
DIT
RMR

stable

+ stable -

BODY FAT STORES
INTERACTION BETWEEN BIOLOGICAL (GENES, HORMONES ETC.), PSYCHOBEHAVIOURAL AND ENVIRONMENTAL FACTORS IN BODY WEIGHT REGULATION AND HEALTH RISKS OF OBESITY
OBESITY RELATED TO MENDELIAN SYNDROMES - RARE
• Prader-Willi (prevalence 1:25,000), Bardet-Biedl etc.
• 2005 - 49 loci relevant to human obesity have been mapped

OBESITY DUE TO SINGLE-GENE MUTATIONS - RARE
• 1997 - 3 cases involving mutations in two genes
• 2000 - 47 cases involving mutations in six genes
• 2002 - 89 cases involving mutations in six genes
• 2005 – 173 cases involving mutations in 10 genes

OBESITY DETERMINED BY MULTIPLE GENES: POLYGENIC - COMMON
• More than 600 genes, markers, and chromosomal regions have been associated or linked with human obesity phenotypes
• Genetic variations in the first intron of the FTO gene (Frayling TM et al. 2007) and downstream of the MC4R gene (Loos RJ et al. 2008) - might greatly contribute to the general predisposition to obesity
ADIPOSE TISSUE
LARGEST ENDOCRINE ORGAN IN THE BODY

CYTOKINES PRODUCED BY ADIPOCYTE ARE RELATED TO THE RISKS OF METABOLIC SYNDROME AND REGULATION OF ENERGY BALANCE
ADIPOSE TISSUE HORMONE LEPTIN REGULATES ENERGY BALANCE AND REPRODUCTION

LEPTIN

• Increases thermogenesis by activateing SNS

• Decreases food intake by
  – by inhibition of orexigenic pathway: neuropeptide Y (NPY) and Agouti Related Peptide (AGRP)

• Initiates puberty and facilitate fertility by activating gonadoliberin secretion (LHRH)
MUTATION OF LEPTIN GENE

CONGENITAL LEPTIN DEFICIENCY IS ASSOCIATED WITH SEVERE EARLY-ONSET OBESITY IN HUMANS

TREATMENT OF INBORN LEPTIN DEFICIENCY BY RECOMBINANT LEPTIN

Child B before leptin
(wt = 42kg at 3yrs)

Child B after leptin
(wt = 32kg at 7yrs)
MUTATION OF MELANOCORTIN4 RECEPTOR (MC4R) GENE

9 yr - homzygote

16 yr – his brother without mutation

MUTATION OF MELANOCORTIN4 RECEPTOR (MC4R)

• most frequent form of monogenic obesity
• prevalence in early onset obesity 0.5-6.0 %, Czech cohort: 2.4%
• missense, frameshift, in-frame deletion, nonsense mutations (> 70)

<table>
<thead>
<tr>
<th>Mutation</th>
<th>Gender (F/M)</th>
<th>Age (years)</th>
<th>Onset (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arg7Cys*</td>
<td>M</td>
<td>17</td>
<td>5</td>
</tr>
<tr>
<td>Ser19fsdelA</td>
<td>F</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>Cys84Arg*</td>
<td>F</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>Gly181Asp</td>
<td>M</td>
<td>15</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Phe51Leu</td>
<td>M</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Phe51Leu</td>
<td>F</td>
<td>17</td>
<td>3</td>
</tr>
<tr>
<td>Ser127Leu</td>
<td>M</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>

* new
ESTIMATES OF HERITABILITY OF COMMON FORMS OF OBESITY RANGE FROM 40 to 70%.

THE FOLLOWING FACTORS ARE GENETICALLY DETERMINED

- Control of food intake (appetite regulation)
- Perception of hunger and satiety
- Eating behavior (dietary disinhibition)
- Hormones controlling food intake, energy expenditure and fat stores
- Food preferences
- Food ingestion
- Nutrient absorption and oxidation
- Energy expenditure (metabolic rate, diet-induced thermogenesis, spontaneous physical activity)
- Nutrient deposition in energy stores
GENES ASSOCIATED WITH COMMON OBESITY IN HUMANS

Association confirmed by $\geq 5$ studies:

<table>
<thead>
<tr>
<th>Gene</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE</td>
<td>6 studies</td>
</tr>
<tr>
<td>ADRB3</td>
<td>29</td>
</tr>
<tr>
<td>DRD2</td>
<td>5</td>
</tr>
<tr>
<td>GNB3</td>
<td>15</td>
</tr>
<tr>
<td>IL6R</td>
<td>6</td>
</tr>
<tr>
<td>LEP</td>
<td>13</td>
</tr>
<tr>
<td>LIPE</td>
<td>6</td>
</tr>
<tr>
<td>NR3C1</td>
<td>12</td>
</tr>
<tr>
<td>PPARGC1A</td>
<td>8</td>
</tr>
<tr>
<td>UCP1</td>
<td>11</td>
</tr>
<tr>
<td>VDR</td>
<td>7</td>
</tr>
<tr>
<td>ADRB2</td>
<td>20</td>
</tr>
<tr>
<td>APOE</td>
<td>5</td>
</tr>
<tr>
<td>FABP2</td>
<td>5</td>
</tr>
<tr>
<td>HTR2C</td>
<td>12</td>
</tr>
<tr>
<td>INS</td>
<td>10</td>
</tr>
<tr>
<td>LEPR</td>
<td>19</td>
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<tr>
<td>MC4R</td>
<td>8</td>
</tr>
<tr>
<td>PLIN</td>
<td>9</td>
</tr>
<tr>
<td>PLIN</td>
<td>9</td>
</tr>
<tr>
<td>PPARG</td>
<td>30</td>
</tr>
<tr>
<td>PPARGC1A</td>
<td>8</td>
</tr>
<tr>
<td>RETN</td>
<td>5</td>
</tr>
<tr>
<td>TNFA</td>
<td>11</td>
</tr>
<tr>
<td>UCP2</td>
<td>14</td>
</tr>
<tr>
<td>UCP3</td>
<td>15</td>
</tr>
<tr>
<td>VDR</td>
<td>7</td>
</tr>
<tr>
<td>FTO</td>
<td>6</td>
</tr>
</tbody>
</table>

*The Human Obesity Gene Map*
<table>
<thead>
<tr>
<th>Trait</th>
<th>Genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thriftiness</td>
<td>ADRβ2, ADRβ3, UCP1, UCP2, UCP3</td>
</tr>
<tr>
<td>Hyperphagia</td>
<td>DRD2, HTR2C, LEP, LEPR, MC4R, NR3C1</td>
</tr>
<tr>
<td>Sedentaryism</td>
<td>DRD2, MC4R</td>
</tr>
<tr>
<td>Low Fat Oxidation</td>
<td>ACE, ADIPOQ, GNB3, IL6, INS, LDLR, LIPE, RETN, TNFA</td>
</tr>
<tr>
<td>Adipose Tissue Hyperplasia</td>
<td>PPARγ, VDR</td>
</tr>
</tbody>
</table>

According to Bouchard C. 2006
## FAMILY BACKGROUND OF OBESITY

Mean BMI $39.9 \text{ kg/m}^2$

<table>
<thead>
<tr>
<th>Obesity in family</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$n = 206$</td>
<td>$n = 782$</td>
</tr>
<tr>
<td>Father</td>
<td>42.7%</td>
<td>30.4%</td>
</tr>
<tr>
<td>Mother</td>
<td>56.3%</td>
<td>54.0%</td>
</tr>
<tr>
<td>Both parents</td>
<td>23.3%</td>
<td>15.9%</td>
</tr>
<tr>
<td>One or both parents</td>
<td>72.8%</td>
<td>68.5%</td>
</tr>
<tr>
<td>Brother</td>
<td>18.4%</td>
<td>14.6%</td>
</tr>
<tr>
<td>Sister</td>
<td>21.4%</td>
<td>14.8%</td>
</tr>
<tr>
<td>None</td>
<td>15.5%</td>
<td>18.4%</td>
</tr>
</tbody>
</table>

V.Hainer et al., 1998
INTRAPAIR CORRELATION OF BMI IN TWINS REARED TOGETHER AND APART

STUDIES ON GENES x ENVIRONMENT INTERACTION CONDUCTED IN IDENTICAL TWINS

• Response to positive energy balance induced by overfeeding in male normal weight identical twins
  

• Response to negative energy balance induced by exercise in male identical twins with modestly elevated weight
  

• Response to negative energy balance induced by VLCD in female obese identical twins
  
  V. Hainer et al. Int. J. Obes. 2000, 24:1051-1057
STUDIES IN IDENTICAL TWINS DEMONSTRATED THAT GENES AFFECT WEIGHT GAIN IN RESPONSE TO A POSITIVE ENERGY BALANCE


SIMILARITY WITHIN PAIRS WITH RESPECT TO CHANGES IN BODY WEIGHT IN 12 PAIRS OF MALE TWINS IN RESPONSE TO 100 DAYS OF OVERFEEDING

![Graph showing correlation between change in body weight and similarity in weight gain among twins.](image-url)
STUDIES IN IDENTICAL TWINS DEMONSTRATED THAT GENES AFFECT FAT LOSS IN RESPONSE TO A NEGATIVE ENERGY BALANCE

Hainer V et al. Int. J. Obes. 2000, 24:1051

INTRAPAIR RESEMBLANCE IN VLCD - INDUCED FAT LOSS

Twin A

$r = 0.88$

$F = 17.0$ (p < 0.001)
ENVIRONMENT STRONGLY AFFECTS BMI IN OBESITY PRONE INDIVIDUALS: MEAN BMI IN AGE-MATCHED PIMA INDIANS LIVING IN ARIZONA AND MEXICO

p<0.001 Arizona vs. Mexico

Ravussin E. Metabolism 1995, 9 (Suppl. 3): 12
GENE - GENE INTERACTION
GENE(S) - ENVIRONMENT INTERACTION

leptogenic resistant to obesity

leptogenic (restrictive)

↑ magnitude of weight loss
prone to weight loss maintenance

slim

normal weight

obese

obesigenic prone to obesity

obesigenic (toxic)

↓ magnitude of weight loss
prone to weight regain

Hainer V et al. Essentials of Clinical Obesity, Grada-Avicenum, Prague, 2004
CURRENT EPIDEMIC OF OBESITY

• cannot be explained by changes in genes during the past two decades although they do play an important role in individual susceptibility to development of obesity

• reflects the failure of human genes to cope with an obesigenic (toxic) environment characterized by a positive energy balance due to
  – high energy density diet
  – low physical activity and adoption of a sedentary lifestyle
OBESIGENIC ENVIRONMENT: HIGH ENERGY DENSITY DIET

- Increased consumption of fatty items
- Increased consumption of soft drinks and sweetened juices
- Decreased consumption of fruits and vegetables
- Decreased consumption of low fat dairy products

FAST FOOD

traditional Czech: 3335 kJ

Mc Donald’s: 3344 kJ
DIETARY FAT IS A MAJOR RISK FACTOR FOR WEIGHT GAIN AND OBESITY

- High energy density (fat: 38 kJ/g vs. carbohydrate or protein: 17 kJ/g)
- Weak satiety
- Low postprandial thermogenesis (Diet Induced Thermogenesis – DIT)
- Taste preferences for fat (and sugar - high palatability substances) – hedonic responses
EFFECTS OF MACRONUTRIENTS ON SATIETY AND DIET-INDUCED THERMOGENESIS

- proteins +++
- carbohydrates ++
- fats +
OBESIGENIC ENVIRONMENT
DIMINISHES HABITUAL PHYSICAL ACTIVITY AND CONTRIBUTES TO DEVELOPMENT OF OBESITY IN SUSCEPTIBLE INDIVIDUALS

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>EXAMPLES</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRANSPORTATION</td>
<td>- private cars</td>
</tr>
<tr>
<td></td>
<td>- motorcycles</td>
</tr>
<tr>
<td></td>
<td>- elevators</td>
</tr>
<tr>
<td></td>
<td>- escalators</td>
</tr>
<tr>
<td></td>
<td>- moving sidewalks</td>
</tr>
<tr>
<td></td>
<td>- automatic door openers</td>
</tr>
<tr>
<td>COMMUNICATIONS</td>
<td>- mobile phones</td>
</tr>
<tr>
<td></td>
<td>- e-mail</td>
</tr>
<tr>
<td></td>
<td>- fax</td>
</tr>
<tr>
<td>WORK SITE ACTIVITIES</td>
<td>- computers</td>
</tr>
<tr>
<td></td>
<td>- automatisation</td>
</tr>
<tr>
<td></td>
<td>- robotisation</td>
</tr>
<tr>
<td></td>
<td>- remote control</td>
</tr>
<tr>
<td>BODY CARE</td>
<td>- electric shavers</td>
</tr>
<tr>
<td></td>
<td>- hair dryers</td>
</tr>
<tr>
<td></td>
<td>- massage apparatus</td>
</tr>
<tr>
<td>COOKING, HOMEWORK AND</td>
<td>- food processors</td>
</tr>
<tr>
<td>HOUSEKEEPING</td>
<td>- electric knifes, juicers, can openers</td>
</tr>
<tr>
<td></td>
<td>- electric beaters and blenders vysavače</td>
</tr>
<tr>
<td></td>
<td>- microwaves</td>
</tr>
<tr>
<td></td>
<td>- dish washers</td>
</tr>
<tr>
<td></td>
<td>- washing machines and dryers</td>
</tr>
<tr>
<td></td>
<td>- vacuum cleaners</td>
</tr>
<tr>
<td></td>
<td>- snow blowers</td>
</tr>
<tr>
<td></td>
<td>- electric drills and screwdrivers</td>
</tr>
<tr>
<td>GARDENING</td>
<td>- motor or electric saws</td>
</tr>
<tr>
<td></td>
<td>- motor or electric lawnmowers</td>
</tr>
<tr>
<td></td>
<td>- electric sanders</td>
</tr>
<tr>
<td>ENTERTAINMENT</td>
<td>- TV and video sets</td>
</tr>
<tr>
<td></td>
<td>- remote control of TV/audio/video</td>
</tr>
<tr>
<td></td>
<td>- video games</td>
</tr>
</tbody>
</table>
Cellular phones and remote controls deprive us from walking!

20 times daily x 20 m = 400 m

Walking distance lost/year
400x365 = 146,000 m

146 km = 25 h of walking

1 h of walking = 113-226 kcal
Energy saved = 2800-6000 kcal

→ + 0.4-0.8 kg adipose tissue per year
FACTORS CONTRIBUTING TO OBESITY EPIDEMIC BESIDES HEREDITARY PREDISPOSITION & EXCESS INTAKE OF ENERGY DENSE FOOD WITH SEDENTARISM

- Intrauterine programming and epigenetic events
- Epigenetic events during postnatal life
- Assortative mating
- Smoking cessation
- Infection (adenovirus)
- Decrease (or increase) in the number of hours of sleep
- Increase in home temperature during winter
- Air conditioning availability in warm climates
CONCLUSION

• Obesity is associated with increased morbidity, mortality and impaired quality of life

• Production of hormones and inflammatory cytokines by adipose tissue contributes to development of the cardiometabolic health risks

• Obesity epidemic reflects the failure of human genes to cope with an obesigenic environment characterized by a positive energy balance

• Involvement of hereditary factors in the development of obesity is estimated about 40 – 70%
CONCLUSION

• Common obesity is polygenic disease determined by interaction of multiple genes with environmental factors; among them consumption of energy dense food and sedentary lifestyle play a crucial role.

• Obesity has been included in the International Classification of diseases more than half a century (code E66).

• General public, health policy makers and many health care providers do not accept obesity as a serious health problem although its pathogenesis, prevalence and health consequences are comparable with other complex diseases as hypertension.
Established in 1986 to promote obesity research and actions that tackle the current epidemic of obesity
EASO INCLUDES 29 MEMBER COUNTRIES

- Austria
- Belgium
- Bulgaria
- Croatia
- Czech Republic
- Denmark
- Finland
- France
- Germany
- Greece
- Georgia
- Hungary
- Iceland
- Ireland

Northern Region
Middle Region
Southern Region

- Israel
- Italy
- Netherlands
- Norway
- Poland
- Portugal
- Republic of Macedonia
- Romania
- Serbia
- Slovakia
- Spain
- Sweden
- Switzerland
- Turkey
- United Kingdom
ECO IS THE MOST IMPORTANT ANNUAL SCIENTIFIC EVENT ON OBESITY IN EUROPE

The ECO provides information about recent research advances in the field of obesity.

PREVIOUS ECOs

- 1988 Stockholm, Sweden
- 1991 Nice, France
- 1992 Noordwijkerhout, Netherlands
- 1993 Ulm, Germany
- 1995 Copenhagen, Denmark
- 1996 Barcelona, Spain

FUTURE ECOs

- 2009 Amsterdam, Netherlands
- 2011 Istanbul, Turkey
- 1997 Dublin, Ireland
- 1999 Milan, Italy
- 2000 Antwerp, Belgium
- 2001 Vienna, Austria
- 2003 Helsinki, Finland
- 2004 Prague, Czech Republic
- 2005 Athens, Greece
- 2007 Budapest, Hungary
- 2008 Geneva, Switzerland

EASO
European Association for the Study of Obesity
TO REALIZE ITS GOALS THE EASO ESTABLISHED THREE TASK FORCES AND ITS OFFICIAL JOURNAL

- OBESITY MANAGEMENT & EDUCATION TASK FORCE (OMTF)
- OBESITY PREVENTION AND PUBLIC HEALTH TASK FORCE
- CHILDHOOD OBESITY TASK FORCE
- OBESITY FACTS HAS BEEN LAUNCHED AS AN EASO OFFICIAL JOURNAL THIS YEAR
EASO OBESITY MANAGEMENT & EDUCATION TASK FORCE (OMTF)

- Creation of network of obesity specialists across the EASO member countries
- Implementation of educational programmes on obesity management
- Guidelines on bariatric surgery (developed together with the European Chapter of the International Federation for the Surgery of Obesity) - published in the International Journal of Obesity, Obesity Surgery and Obesity Facts
- Updated version of the European Guidelines on Obesity Management in Primary Care - published in the 2nd issue of Obesity Facts
- Continuous monitoring of obesity management strategies in the EASO member countries
- Definition of the EASO Collaborating Centres in Obesity Management
EASO CHILDHOOD OBESITY TASK FORCE (COTF)

• Promotion of educational activities for healthcare providers dealing with obese children across Europe

• Current goal: To prepare a template for childhood obesity management

• Satellite symposia at ECOs

• Collaboration with other childhood obesity groups (EAROC, ECOG, ECOPA)
EASO PREVENTION AND PUBLIC HEALTH TASK FORCE

• Joint action with WHO Europe ‘Lifestyle Strategies for Primary Care for the Prevention of Overweight and Obesity’ (workshop at ECO in Geneva)

• Establishing a network of public health experts and epidemiologists representing the national associations for the study of obesity in Europe

• Participation in the EU Platform on Diet, Physical Activity and Health

• Monitoring overweight and obesity in Europe

• Improvement of the epidemiological perspective on obesity in Europe
INVITATION

WELCOME TO THE 17th ECO IN AMSTERDAM, MAY 6-9th 2009
Greetings from Prague - Castle
Prague

seat of the Charles University
“Golden”, “steepled” ...
Prague – historical city
Prague – city of churches...
Prague by night
Old Town Square
Christmas tree 2008
Thanks for your attention!

Prague – Charles Bridge