Weight-loss myths

Some of the most firmly held beliefs about weight loss are unproven or downright untrue, according to an analysis comparing concepts promoted in the popular media with data from the scientific literature. The findings were published online January 30, 2013 in the New England Journal of Medicine.
Table 1. Seven Myths about Obesity.\textsuperscript{a}

<table>
<thead>
<tr>
<th>Myth</th>
<th>Basis of Conjecture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small sustained changes in energy intake or expenditure will produce large, long-term weight changes</td>
<td>National health guidelines and reputable websites advertise that large changes in weight accumulate indefinitely after small sustained daily lifestyle modifications, (e.g., walking for 20 minutes or eating two additional potato chips)</td>
</tr>
<tr>
<td>Setting realistic goals in obesity treatment is important because otherwise patients will become frustrated and lose less weight</td>
<td>According to goal setting theory, unsuitable goals impair performance and discourage goal-attaining behavior; in obesity treatment, incongruence between desired and actual weight loss is thought to undermine the patient’s perceived ability to attain goals, which may lead to the discontinuation of behaviors necessary for weight loss</td>
</tr>
<tr>
<td>Large, rapid weight loss is associated with poorer long-term weight outcomes than is slow, gradual weight loss</td>
<td>This notion probably emerged in reaction to the adverse effects of nutritionally insufficient very-low-calorie diets (&gt;800 kcal per day) in the 1980s; the belief has persisted, has been repeated in textbooks and recommendations from health authorities, and has been offered as a rule by dietitians</td>
</tr>
<tr>
<td>Assessing the stage of change or diet readiness is important in helping patients who seek weight loss treatment</td>
<td>Many believe that patients who feel ready to lose weight are more likely to make the required lifestyle changes</td>
</tr>
<tr>
<td>Physical education classes in their current format play an important role in preventing or reducing childhood obesity</td>
<td>The health benefits of physical activity of sufficient duration, frequency, and intensity are well established and include reductions in adiposity</td>
</tr>
<tr>
<td>Breast-feeding is protective against obesity</td>
<td>The belief that breast-fed children are less likely to become obese has persisted for more than a century and is passionately defended</td>
</tr>
<tr>
<td>A bout of sexual activity burns 100 to 200 kcal for each person involved</td>
<td>Many sources state that substantial energy is expended in typical sexual activity between two adults</td>
</tr>
</tbody>
</table>

\textsuperscript{a} We define myths as beliefs held true despite substantial evidence refuting them. A list of articles in which these myths are espoused is provided in the Supplementary Appendix.

Casazza K et al. N Engl J Med 2013;368:446-454

Table 2. Presumptions about Obesity.\textsuperscript{b}

<table>
<thead>
<tr>
<th>Presumption</th>
<th>Basis for Conjecture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regularly eating (vs. skipping) breakfast is protective against obesity</td>
<td>Skipping breakfast purportedly leads to overeating later in the day</td>
</tr>
<tr>
<td>Early childhood is the period during which we learn exercise and eating habits that influence our weight throughout life</td>
<td>Weight-for-height indexes, eating behaviors, and preferences that are present in early childhood are correlated with those later in life</td>
</tr>
<tr>
<td>Eating more fruits and vegetables will result in weight loss or less weight gain, regardless of whether one intentionally makes any other behavioral or environmental changes</td>
<td>By eating more fruits and vegetables, a person presumably spontaneously eats less of other foods, and the resulting reduction in calories is greater than the increase in calories from the fruit and vegetables</td>
</tr>
<tr>
<td>Weight cycling (i.e., yo-yo dieting) is associated with increased mortality</td>
<td>In observational studies, mortality rates have been lower among persons with stable weight than among those with unstable weight</td>
</tr>
<tr>
<td>Snacking contributes to weight gain and obesity</td>
<td>Snack foods are presumed to be incompletely compensated for at subsequent meals, leading to weight gain</td>
</tr>
<tr>
<td>The built environment, in terms of sidewalks and park availability, influences obesity</td>
<td>Neighborhood environment features may promote or inhibit physical activity, thereby affecting obesity</td>
</tr>
</tbody>
</table>

\textsuperscript{b} We define presumptions as unproven yet commonly espoused propositions. A list of articles in which these presumptions are implied is provided in the Supplementary Appendix.

Casazza K et al. N Engl J Med 2013;368:446-454
The myths and presumptions about obesity are just a sampling of the numerous unsupported beliefs held by many people, including academics, regulators, and journalists, as well as the general public. Yet there are facts about obesity of which are reasonably certain.

This provocative paper is followed by very sharp comments in the NEJM correspondence.
Lp(a) gene variant associated with aortic stenosis

An international consortium has identified a variation in the gene coding for lipoprotein (a) (Lp[a]), which appears to be strongly associated with aortic-valve calcification. People carrying this single nucleotide polymorphism (SNP) had a doubling of the risk of valve calcification on computer tomography (CT) compared with those without the variation. (February 7, 2013 issue of the *New England Journal of Medicine*. [1,2].)

Structure of the Lp(a) particle

The same SNP has previously been identified as a risk factor for increased Lp(a) levels and coronary artery disease (CAD). The cumulative findings support the proposition that a common genetic defect in lipid metabolism underlies the
Aortic stenosis is the third most prevalent form of cardiovascular disease in the Western world, after hypertension and CAD, and is caused by calcification and hardening of the aortic valve, impeding blood flow from the heart to the rest of the body, leading to chest pain, loss of consciousness, and shortness of breath. In severe cases, patients require aortic-valve replacement. Currently, there are no medical treatments to prevent this disease, which mainly affects people over the age of 60.

In their genomewide association study, the team first looked at 2.5 million SNPs among more than 6900 people of white European descent and found that a variant in the Lp(a) locus—rs10455872—reached genomewide significance for the presence of aortic-valve calcification on CT scan (odds ratio per allele 2.05; p=9x10^-10). This finding was replicated in an additional 2000 people of Hispanic origin, about 2500 African Americans, and more than 700 Germans (p<0.05 for all comparisons). In further prospective analyses, the researchers were also able to demonstrate an association between the variant and incident aortic stenosis (hazard ratio per allele 1.68) as well as aortic-valve replacement (hazard ratio 1.54) among more than 28 000 people in Sweden. The association with aortic stenosis was also replicated in an independent Danish cohort.

This research not only shows that a common variant in Lp(a) is implicated but that this was indeed "a driver of the process."
Associations between Each Single-Nucleotide Polymorphism (SNP) and Aortic-Valve Calcium or Mitral Annular Calcium, According to Chromosomal Position.

Manhattan plots show that a single SNP on chromosome 6 (rs10455872) has genomewide significance for aortic-valve calcium ($P=9.0 \times 10^{-10}$) (Panel A) and that two SNPs on chromosome 2 (rs17659543 and rs13415097) have genomewide significance for mitral annular calcium ($P=1.5 \times 10^{-8}$ and $P=1.8 \times 10^{-8}$, respectively).
Although increased levels of Lp(a) have previously been associated with aortic-valve disease, prior observational studies couldn't prove that it was a contributing factor rather than just a marker. These results provide the first evidence for a causal relationship between Lp(a) and calcific aortic-valve disease.

The main implications of this are understanding better about the biology of what might lead to aortic-valve calcification and potentially progression to clinical aortic stenosis. This may lead to new therapies that could perhaps delay progression of aortic stenosis.

Since Lp(a) predicts risk for myocardial infarction and now we know it also predicts risk for aortic stenosis, we are hoping that maybe pharmaceutical companies will advance their research into compounds that can lower Lp(a), because drugs like statins don't."

One compound that does lower Lp(a) is niacin but this has recently suffered a huge setback in the wake of the HPS-2 THRIVE results. Estrogen also lowers Lp(a) but again has suffered from controversy, she notes.

Sources

ABSTRACTS ON VARIOUS TOPICS
The first abstract is a summary of an article published by our colleague and BLC member, M.B. Twickler.

Maternal lipid profile during early pregnancy and pregnancy complications and outcomes: the ABCD study.

J Clin Endocrinol Metab. 2012 Nov;97(11):3917-25

Vrijkotte TG, Kruksiener N, Hutten BA, Vollebregt KC, van Eijsden M, Twickler MB. Department of Public Health, Academic Medical Center, University of Amsterdam, P.O. Box 22660, 1100 DD Amsterdam, The Netherlands. T.Vrijkotte@amc.uva.nl

CONTEXT: Elevated lipid levels during late pregnancy are associated with complications and adverse outcome for both mother and newborn. However, it is inconclusive whether a disturbed lipid profile during early pregnancy has similar negative associations.

OBJECTIVE: Our objective was to investigate whether nonfasting maternal total cholesterol and triglyceride levels during early pregnancy are associated with six major adverse pregnancy outcomes.

METHODS: Data were derived from the Amsterdam Born Children and Their Development (ABCD) cohort study. Random blood samples of nonfasting total cholesterol and triglyceride levels were determined during early gestation (median = 13, interquartile range = 12-14 wk). Outcome measures were pregnancy-induced hypertension (PIH), preeclampsia, preterm
birth, small/large for gestational age (SGA/LGA), and child loss. Only nondiabetic women with singleton deliveries were included; the baseline sample consisted of 4008 women. Analysis for PIH and preeclampsia were performed in nulliparous women only (n = 2037).

**RESULTS:** Mean (sd) triglyceride and total cholesterol levels were 1.33 (0.55) and 4.98 (0.87) mmol/liter, respectively. The incidence of pregnancy complications and perinatal outcomes were as follows: PIH, 4.9%; preeclampsia, 3.7%; preterm birth, 5.3%; SGA, 9.3%; LGA, 9.3%; and child loss, 1.4%. After adjustments, every unit increase in triglycerides was linearly associated with an increased risk of PIH [odds ratio (OR) = 1.60, P = 0.021], preeclampsia (OR = 1.69, P = 0.018), LGA (OR = 1.48, P < 0.001), and induced preterm delivery (OR = 1.69, P = 0.006). No associations were found for SGA or child loss. Total cholesterol was not associated with any of the outcome measures.

**CONCLUSIONS:** Elevated maternal triglyceride levels measured during early pregnancy are associated with pregnancy complications and adverse pregnancy outcomes. These results suggest that future lifestyle programs in women of reproductive age with a focus on lowering triglyceride levels (i.e. diet, weight reduction, and physical activity) may help to prevent hypertensive complications during pregnancy and adverse birth outcomes.
**Effect of Combination Therapy With Fenofibrate and Simvastatin on Postprandial Lipemia in the ACCORD Lipid Trial**


Gissette Reyes-Soffer, Henry N. Ginsberg, MD

Corresponding author: Henry N. Ginsberg, hng1@columbia.edu

**OBJECTIVE** The Action to Control Cardiovascular Risk in Diabetes lipid study (ACCORD Lipid), which compared the effects of simvastatin plus fenofibrate (FENO-S) versus simvastatin plus placebo (PL-S) on cardiovascular disease outcomes, measured only fasting triglyceride (TG) levels. We examined the effects of FENO-S on postprandial (PP) lipid and lipoprotein levels in a subgroup of ACCORD Lipid subjects.

**RESEARCH DESIGN AND METHODS** We studied 139 subjects (mean age of 61 years, 40% female, and 76% Hispanic or black) in ACCORD Lipid, from a total 529 ACCORD Lipid subjects in the Northeast Clinical Network. PP plasma TG, apolipoprotein (apo)B48, and apoCIII were measured over 10 h after an oral fat load.

**RESULTS** The PP TG incremental area under the curve (IAUC) above fasting (median and interquartile range [mg/dL/h]) was 572 (352–907) in the FENO-S group versus 770 (429–1,420) in the PL-S group (*P* = 0.008). The PP apoB48 IAUC (mean ± SD [μg/mL/h]) was also reduced in the FENO-S versus the PL-S group (23.2 ± 16.3 vs. 35.2 ± 28.6; *P* = 0.008). Fasting TG levels on the day of study were correlated with PP TG IAUC (*r* = 0.73 for FENO-S and *r* =
0.62 for PL-S; each $P < 0.001$). However, the fibrate effect on PP TG IAUC was a constant percentage across the entire range of fasting TG levels, whereas PP apoB48 IAUC was only reduced when fasting TG levels were increased.

**CONCLUSIONS** *FENO-S lowered PP TG similarly in all participants compared with PL-S. However, levels of atherogenic apoB48 particles were reduced only in individuals with increased fasting levels of TG. These results may have implications for interpretation of the overall ACCORD Lipid trial, which suggested benefit from FENO-S only in dyslipidemic individuals.*

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**Television Viewing and Low Leisure-Time Physical Activity in Adolescence Independently Predict the Metabolic Syndrome in Mid-Adulthood**

*Diabetes Care 2013 Jan 22*

Patrik Wennberg, PHD

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**OBJECTIVE** We investigated whether television (TV) viewing and low leisure-time physical activity in adolescence predict the metabolic syndrome in mid-adulthood.
RESEARCH DESIGN AND METHODS TV viewing habits and participation in leisure-time physical activity at age 16 years were assessed by self-administered questionnaires in a population-based cohort in Northern Sweden. The presence of the metabolic syndrome at age 43 years was ascertained in 888 participants (82% of the baseline sample) using the International Diabetes Federation criteria. Odds ratios (ORs) and CIs were calculated using logistic regression.

RESULTS The overall prevalence of the metabolic syndrome at age 43 years was 26.9%. Adjusted OR for the metabolic syndrome at age 43 years was 2.14 (95% CI 1.24–3.71) for those who reported “watching several shows a day” versus “one show/week” or less and 2.31 (1.13–4.69) for leisure-time physical activity “several times/month” or less compared with “daily” leisure-time physical activity at age 16 years. TV viewing at age 16 years was associated with central obesity, low HDL cholesterol, and hypertension at age 43 years, whereas low leisure-time physical activity at age 16 years was associated with central obesity and triglycerides at age 43 years.

CONCLUSIONS Both TV viewing and low leisure-time physical activity in adolescence independently predicted the metabolic syndrome and several of the metabolic syndrome components in mid-adulthood. These findings suggest that reduced TV viewing in adolescence, in addition to regular physical activity, may contribute to cardiometabolic health later in life.