UPCOMING ACTIVITIES

August 23, 2016, Tuesday

PASOO 22nd ANNUAL CONVENTION

Theme: Focusing on Fat

EDSA Shangri-La Hotel, Mandaluyong City

September 1-7, 2016

Obesity Awareness and Prevention Week

8th Obesity Workshop

Iloilo / 4th Quarter 2016

October 11, 2016

World Obesity Day

PASOO BOARD OF DIRECTORS

(Not in photo are Dr. Ramon F. Abarquez, Jr and Dr. Celeste C. Tanchoco, RD, DrPh)

OUR MISSION

Pioneer in the prevention & control of obesity & its complications through education, research & advocacy

OUR VISION

An obesity risk-free nation

Philippine Association for the Study of Overweight and Obesity
Member of the World Obesity Federation

OBESITY ALERT
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Study of Overweight and Obesity

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Philippine Association for the Study of Overweight and Obesity
Member of the World Obesity Federation

PRESIDENT'S MESSAGE

Roberto C. Mirasol, MD, FPCP, FPSEDM
President, PASOO
Chief, Section of Endocrinology Diabetes and Metabolism
St. Luke's Medical Center, Quezon City
Manila Doctors Hospital

Today, I challenge all of you to help us fight obesity! PASOO has been in existence for twenty-two years and yet we still see an incredible rise in the prevalence of overweight and obesity in our midst. The recent surveys attest to this. If left untreated we might see an epidemic happening in the next few years. We at the PASOO therefore have launched awareness campaigns to increase the knowledge and understanding of this disease.

First week of September is Obesity Awareness Week where this year we emphasize the value of Exercise as a key in the management of overweight and obesity. Through our advocacy program **EXERCISE IS MEDICINE (EIM)** we are able to bring to light the advantages of doing regular safe exercise. The goals of the EIM initiative is to assess and record Physical Activity as a vital sign during patient visits and to conclude each visit with an exercise prescription. We have done several courses in the past and will continue to do so in the coming year. Help us spread the mantra that Exercise is indeed medicine and we cannot overemphasize its advantages.

October 11 is World Obesity Day. Now on its second year, our focus is **Overcoming Childhood Obesity**, aligning with the WHO Commission's Report on Ending Childhood Obesity. We know that childhood obesity is on the rise. We need to take action to decrease the complications and these are worse and occur early on if they have been overweight or obese since childhood.

What can you do?

You can help us by organizing a lay forum on obesity. You can disseminate key messages to your social networks (face book, twitter, etc). You can call on our new government leaders to take urgent action to tackle childhood obesity. You can raise awareness in your community and the workplace. You can spread the news through radio, television or newspapers. Put up posters and have a lobby display in your hospitals, clinics or offices. With concerted efforts we can create awareness and hopefully behavior could change. Let's join hands to FIGHT OBESITY!

Roberto C . Mirasol, MD, FPCP, FPSEDM
PASOO President, 2014-2016

member of WORLD OBESITY federation

What's inside?

What's inside

EDGARDO L. TOLENTINO, JR, MD, FPPA
Secretary, PASOO
Chairman, Dept. of Psychiatry and Mental Health
Quirino Memorial Medical Center

From Concept to Patient

At the heart of what we do at PASOO is our obese or overweight patient, friend, family member or...even ourselves. We are an association of specialists from very diverse fields of medicine as well as the disciplines of diet and nutrition and kinesiology working harmoniously and cooperatively utilizing our triad of key strategies: education, research and advocacy. Obesity, after all, has multiple interacting causes including biological, genetic, behavioral and socioeconomic factors.

Reflective of the multi-dimensional problem of obesity, the concepts, research, management/intervention, and advocacy efforts all focus on the obese patient. Towards this end, the different contributors have shared their take on the problem based on their expertise. PASOO's strength is that it can take on the problem of obesity, slice it open, and examine it from different scopes of its many experts. Our 2016 edition of the Obesity Alert newsletter is a testament to this. As a peek, you have a bevy of interesting and thought-provoking articles from Concept on...with the obese patient at heart. Read on....

CONCEPTS:

- *Are You Happy With Your Weight?*
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- *MEASURING OBESITY: Body Composition vs BMI*
  - Rosa Allyn G. Sy, MD, FPCP, FPSEDM

RESEARCH

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- Eating Breakfast May Improve the Health of Obese Individuals!
  - Nemencio A. Nicodemus Jr., MD, FPCP, FPSEDM
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- PAVS – A Simple Clinical Tool to Promote Physical Activity
  - Rodolfo F. Florentino, MD, PhD
- Fat Replacement Strategies - Celeste C. Tanchoco, RND, DrPh
- LOVE and Attain Healthy Weight - Sioksoan Chan-Cua, MD, MSc

We enjoin other members to contribute articles that will enrich discussion, inform the public, educate fellow members , or just provoke new thinking in the field of obesity. You may submit articles, as well share your comments and suggestions at our email address: sec@obesity.org.ph

HAPPY READING!

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Are You Happy With Your Weight?

Sanirose S. Orbeta, MS, RD, FADA
Board Member, PASOO
Consulting Clinical and Sports Nutritionist

What is "Healthy Weight"?

A person's "healthy" weight is unique to that person. Various factors influence weight such as genes (which play a role in determining body size and shape), physical activity and the food one eats. Whether or not one's weight is "healthy" depends on where the body fat is located, how much of the weight is fat and whether he or she has weight-related health problems such as diabetes and high blood pressure.

How Do You Determine "Healthy Weight"?

Several indicators can be used to determine a person's weight. Available weight tables and charts can be used to give information on one's weight in relation to his age and/or height.

Body Mass Index or BMI is an accepted and most practical and simple index of measuring the degree of body fat. It gives weight range classification or category if the person is obese, overweight or of acceptable weight regardless of age, gender, and ethnicity. It is measured by dividing the weight in kilograms by the height in meters. A BMI of 25 or below is acceptable, 27.3 (women) and 27.8 (men) is categorized as overweight and 30.0 is considered obese. Another way to estimate BMI is to multiply the weight in pounds by 704, divided by the height in inches.

For example, a man who weighs 165 lbs. and stands 5'8" will have an acceptable body mass index of 25.

Body Shape

Are you apple-shaped or pear-shaped? apple-shaped bodies have more fat on the upper body portion around the abdomen. This is commonly seen in males. Individuals with fat gathering on the lower portion of the body like the buttocks and thighs are often pear-shaped. This is common among females. Excess weight below the waist, creating a pear-shaped body, does not pose as much risk for weight-related problems as does weight carried above the waist.

To determine your body shape, use the waist-to-hip ratio. The waist-to-hip measures the waist circumference divided by your hip measurement. The acceptable waist-to-hip ratio is less than 0.8 for females and less than 0.9 for males. If you measure below the acceptable values, you are considered okay and at less risk for heart diseases and diabetes.

For example, a woman whose waist and hip measurements are 29 inches and 32 inches respectively has a waist-to-hip ratio of 0.9 (above acceptable value for women). If she manages to lose some of those extra body fats through a modified diet or exercise and bring down her measurement of waist and hip to 27 inches and 32 inches respectively, she'll have a waist-to-hip ratio of 0.8, which is acceptable.

Are You Ready to Lose Weight?

If you've determined that you weigh more than your body can carry, it's time to decide whether you should lose weight or not.

The following is a weight loss readiness quiz which can interpret one's readiness for weight loss. Mark each item true or false.

_____ 1. I have thought a lot about my eating habits and physical activities to pinpoint what I need to change.

_____ 2. I have accepted the idea that I need to make permanent change, not temporary, changes in my eating style and activities to be successful.

_____ 3. I will only feel successful if I lose a lot of weight.

_____ 4. I accept the idea that it's best if I lose weight slowly.

_____ 5. I'm thinking of losing weight now because I really want to, not because someone else thinks I should.

_____ 6. I think losing weight will solve other problems in my life.

_____ 7. I am willing and able to increase my regular physical activity.

_____ 8. I can lose weight successfully if I have no "slip-ups"

_____ 9. I am ready to commit some time and effort each week to organizing and planning my food and activity programs.

_____10. Once I lose some initial weight, I usually lose the motivation to keep going until I reach my goal.

_____ 11. I want to start a weight loss program, even though my life is unusually stressful right now.

Scoring the Weight Loss Readiness Quiz:

To score the quiz:

1. Look at your answers to items 1, 2, 4, 5,7, and 9. Score "1" if you answered "true" and "0" if you answered "false".

2. For items 3, 6, 8, 10, 11, score "0" for each "true" answer and "1" for each "false" answer.

3. To get your total score, add the scores for all questions.

4. To interpret your scores:

8 or higher – you probably have good reasons for wanting to lose weight now and a good understanding of the steps needed to succeed

5 to 7 – you may need to re-evaluate your reasons for losing weight and the methods you would use to do so.

4 or less – now may not be the right time for you to lose weight. While you may be successful in losing weight initially, your score suggests you are unlikely to sustain sufficient effort to lose all the weight you want or to keep off the weight that you do lose. You need to reconsider your weight loss motivations and methods and perhaps learn more about the pros and cons of different approaches to reducing.

MEASURING OBESITY: Body Composition vs BMI

Rosa Allyn G. Sy, MD, FPCP, FPSEDM
Endocrinology, Diabetes, Metabolism and Nutrition
Cardinal Santos Medical Center

The World Health Organization has defined overweight and obesity as a 'disease in which excess body fat has accumulated to such an extent that health may be adversely affected'. In the early 1900s the Metropolitan Life Insurance Company used individual's weight versus the reference value weight (ideal body weight, desirable body weight) to determine longevity and mortality risk. In 1985, the relation of BMI and risk of morbidity and mortality was introduced. In 2010, the WHO International Classification of Weight based on BMI level was adopted not only in the US population but also worldwide and became the standard criteria for defining and assessing overweight and obesity.

To date there are several ways to determine body fat. It is divided into 2 main categories, namely: the "field method" and the "reference measurements".

BMI is the most basic method, the most common, simple, inexpensive and easy to measure field method in determining body fat. It has been analyzed and validated by accurate methods to be strongly correlated with body fat levels. Hundreds of studies show that a high BMI predicts higher risk of chronic disease and early death. Other field methods include waist circumference, waist to hip ratio, skinfold thickness and bioelectrical impedance all considered useful in clinics and community settings, as well as in large research studies.

However; in recent years, the use of body weight and BMI to define obesity have been criticized since **body weight and BMI do not depict the different proportions of lean versus adipose tissues** (i.e. body composition). It has been shown by studies that individuals may have similar BMI but with different disease risk because of body composition variability. Hence the use of body composition analysis with body fat percentage (BF%) determination may be more useful in determining all-cause mortality.

The more sophisticated methods referred to as the reference measurements such as the magnetic resonance imaging or dual absorptiometry are the accurate techniques in determining body composition. But since these methods are expensive and not readily available, they are typically used only in research studies to confirm the accuracy body measurement techniques. Methods like the Underwater weighing Densitometry, Air-Displacement Plethysmography, Dilution Method (Hydrometry) are safe and can be used in individuals with a BMI of 40 or higher except underwater weighing densitometry. However, although accurate, these tests provide some limitations. Dual Energy X-ray Absorptiometry cannot be used in pregnant women because of the small radiation exposure, while the underwater weighing densitometry is time consuming and requires individuals to be submerged in water and is generally not a good option for children and older adults.

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Weight Management Philippine Style 2016

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College of Medicine, Endocrinology
Past President, PASOO
Editor-in-Chief, JAFES

Weight management continues to be a challenge in the Philippines at the current time.

We all agree that achieving a healthy weight for every individual is a good goal.

Our national survey shows that as of 2013, one out of every 3 Filipinos is overweight and obese (30%), using Body Mass Index (BMI) of 25 as a criterion. Trends show increasing prevalence through the years.

It is important to note a few basic points.

First, **obesity is a problem that we face alongside malnutrition.** The same survey reports one out of every 10 Filipinos have chronic energy deficiency (10%) at a BMI <18.5. This is important to note as national programs would need to address both extremes of the spectrum of this problem.

Second, obesity with its accompanying cardiovascular risks present differently among Asians including Filipinos as compared to populations in the US and Europe. Even if our obesity rates using BMI of 30 as cut-off are lower, it appears that Filipinos cannot be satisfied with this statistic. Studies on Filipinos both here and from Filipino colleagues internationally have demonstrated that we need to be concerned even at a lower BMI. **Filipinos have been shown to have a greater proportion of fat deposits at the abdomen, called visceral adiposity brings about increased risk for heart attacks and stroke.** This can be seen from increasing waist circumference and increasing waist to hip proportions.

Third, severe obesity does exist in some Filipinos. With increasing BMI, aggressive strategies have to be applied.

We as a country should put our act together in the prevention of obesity and diabetes that can place a great toll on our resources, work capacity and quality of life of our patients.

What are the specific prevention and management strategies?

Intensive lifestyle change is the cornerstone of prevention. The national program should include education on these basic facts from the primary school level. Regular physical activity should be promoted starting at this educational level and should proceed all the way to the workplace. Some companies now provide their employees with pedometers to count the number of steps each day. We need safe sidewalks, free from pollution to encourage the citizenry to walk a part of their commute.

Drug therapy is acceptable for those who continue to fail lifestyle intervention. We need to bear in mind though that weight loss successes with drugs may be limited, that is, about 10% reduction and we have to aware of safety issues. Newer diabetes drugs have also given preference to those that are able to reduce weight in addition to the blood sugar lowering.

Bariatric surgery is being used as a treatment option in severe obesity. Various management centers in the metropolis provide multi-disciplinary teams that are able to provide holistic approaches to weight loss. Clearly, a successful weight reduction improves metabolic parameters and reduce cardiovascular risk. Let us continue to assist these teams in providing safe and cost-effective treatments for those that have failed weight reduction.

We thank the Lord for another year of activities at the Philippine Association for Overweight and Obesity (PASOO) and the Philippine Society of Endocrinology, Diabetes and Metabolism (PSEDM) towards the objective of having an obesity risk-free nation.

The Hormones of FAT

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I have always misconstrued perivascular fat as just depots of adipose tissue, providing insulation to the body. What I have learned is that perivascular fat is in fact a metabolically active endocrine and paracrine organ secreting hormones which serve either as protagonist or antagonists of the atherosclerotic process. Fat surrounds the heart in its pericardial sac, the coronaries on the epicardial surface, around the aorta. It is interesting how fat cells surrounding the heart and blood vessels, through its hormonal secretions, influence blood flow and in fact, every heart beat. Fat cells secrete many hormones which are counter regulatory to each other, but in this article **let us focus on two: leptin which is pro-inflammatory and proatherogenic, and adiponectin which is antiatherogenic.** The leptin /adinopectin ratio has been described as an index in to measure the atherogenic process among diabetics.

LEPTIN

Leptin is akin to a thermostat that regulates body fat, instead of monitoring heat. The level of leptin reflects the amount of fat in the body. When a person is full, his leptin levels are high; when hungry, his leptin levels are low. Leptin, was originally identified in 1994 by Friedman and was considered to be the gene defect product that was responsible for the obesity syndrome. Leptin is encoded by the ob gene located on chromosome 17. It is primarily produced in fat cells. Its primary physiological role is to communicate with the brain the level of available energy stores and to restrain food intake and induce energy expenditure. The absence of leptin or the resistance to its effect, leads to increased appetite and food intake that result in morbid obesity. Leptin receptors in the brain are abundant in the hypothalamus, a region which regulates appetite. Leptin works by inhibiting the activity of neurons that contain neuropeptide Y and the agouti-related peptide as well as by increasing the activity of neurons expressing α-melanocyte-stimulating hormone The neuropeptide is important in the regulation of appetite, while the latter hormone is a significant mediator of satiety. The net action of leptin is to inhibit appetite, stimulate thermogenesis, enhance fatty acid oxidation, decrease glucose, and reduce body weight and fat. Circulating leptin levels correlate well to body fat mass, sex hormone levels, dietary fat, and age. Its expression is increased by overfeeding, insulin, glucocorticoids, endotoxin, and cytokines and is decreased by fasting, testosterone, thyroid hormone, and exposure to cold temperature.

In humans, leptin levels are usually very high in obesity as a result of leptin resistance rather than leptin deficiency; disruption of leptin signaling in the satiety centers in the brain results in obesity. Although described in literature, mutations of the gene for leptin appear to be a very rare cause of extreme obesity in humans. Leptin resistance is part of the maladaptation of the biological system for weight maintenance that makes it extremely difficult to lose or maintain weight. Leptin levels are up but fail to signal the brain's appetite and satiety centers.

The high levels of leptin in the obese can cause hypertension. Leptin centrally activates the sympathetic nervous system. It significantly increases plasma norepinephrine and epinephrine concentrations via the ventromedial hypothalamus. Although the effects of leptin signaling in the satiety centers may be disrupted, its sympathetic excitatory effects are maintained in obesity. In the periphery, the hypertensive effect of leptin can be due to the release of vasoconstrictors like endothelin I and angiotensin II from endothelial cells, macrophages, fibroblasts, and cardiomyocytes. Leptin also enhances the release of reactive oxygen species that counteract the effects of endogenous nitric oxide.

Excess leptin in obesity also can cause blood vessels to harden or calcify. The differentiation of marrow osteoprogenitor cells is regulated by leptin, it was subsequently shown that leptin could stimulate the calcification of vascular cells. When leptin attaches to its receptor in the artery wall, the effect is osteogenic differentiation of a subpopulation of vascular cells, called calcifying vascular cells. Leptin receptors were found in the endothelium of the adventitial vessels but not in the aortic endothelium. Leptin receptors were identified in atherosclerotic coronary arteries, predominantly in the endothelial cells of intimal neovessels, macrophages/foam cells and vascular smooth muscle cells.

ADIPONECTIN

To counterbalance the effect leptin, adipocytes also produce adiponectin. Adiponectin is the most abundant cytokine produced by fat cells, it was originally identified by four independent groups in the mid-1990s. The adiponectin gene is located on chromosome 3q27 in humans, a locus that has been linked with diabetes susceptibility. The secretion of adiponectin by fat cells appears to be hormone regulated. Its level declines following stimulation with insulin, tumor necrosis factor - α, endothelin-1, and glucocorticoids, whereas levels increase with insulin-like growth factor – 1 treatment. Initially, adiponectin was thought to be exclusively synthesized by adipocytes; however, a recent study suggests that it is also synthesized and secreted by human heart muscle cells.

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The Fat and Fit Myth

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You see an obese patient with normal vital signs, but you tell him, "You need to lose weight. Your bad cholesterol might be high already, making you at high risk for heart disease." Then, you order for routine laboratory exams that include lipid profile and fasting blood glucose. They all come out normal! So, should you still tell your patient to lose weight?

"Metabolically healthy obesity" (MHO) is a medical condition characterised as in patients who are obese but without the metabolic abnormalities such as dyslipidemia, and impaired glucose tolerance. Though there is no universal definition for MHO, similar to standards of obesity (such as body mass index vs. Waist Circumference vs. Waist:Hip ratio, etc), whether this can actually decrease a patient's risk for heart disease or decrease mortality is under debate.

In Northern Sweden, a substudy of the WHO Multinational MONItoring of Trends and Determinants in CArdiovascular Disease (MONICA) was conducted from 1986 to 2009 in 8,874 subjects to confirm whether the incidence of cardiovascular disease was decreasing despite increasing BMI. **Metabolic health was defined as a total cholesterol level below 5.0 mmol/l, blood pressure below 140/90 mmHg and not having diabetes.** By 2009 the age range was 25 to 74 years.

Results from the study showed the following: The prevalence of metabolic health among obese subjects reached 21.0% in 2009. For overweight subjects metabolic health increased to 18%, whereas for the normal-weight subjects, the increase was 39% in 2009. The prevalence of metabolic health among subjects with abdominal obesity reached 17.3% in 2009. The prevalence of metabolic health among subjects with abdominal obesity reached 17.3% in 2009. Among those with no abdominal obesity the increase was 38% in 2009 (p = <0.001 for all groups). Only among non-obese men and obese women did the increase continue between 2004 and 2009. In the other groups a slight decline or levelling off was noted.

A study done in Finland (Korhonen, P et al, 2015) which aimed to determine the lifestyle of MHO individuals showed that they were mostly women, cohabitating, slightly younger, more educated, took less alcohol and had more physical activity or exercise.

The Whitehall Cohort Study (2014), however, disputed the idea that MHO's were at less risk for cardiovascular mortality. (Fig. 1) The study concluded that MHO have higher risk of CVD and this risk is no different with that in the metabolically unhealthy obese. This suggests that **obesity outweighs the impact of metabolic status for risk of cardiovascular disease.** A meta-analysis (Eckel, N et al. 2015) was determined whether MHO or MHOverweight conditions were actually "benign" conditions, rendering less cardiovascular mortality.Restricting analysis only to studies (Fig. 2) with at least 10 years of follow-up, the MHO group indeed had increased mortality and cardiovascular risk compared with the metabolically healthy normal-weight group (RR, 1.24; CI, 1.02 to 1.55; I2 33.6%). These data indicate that, with long- term follow-up, metabolically healthy obesity is associated with increased mortality and CV risk.

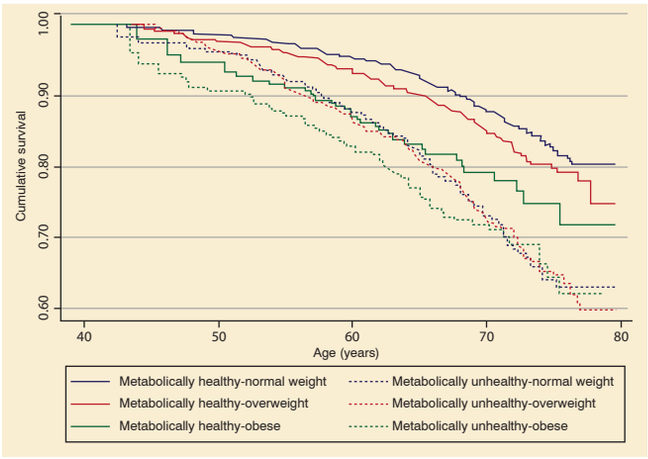
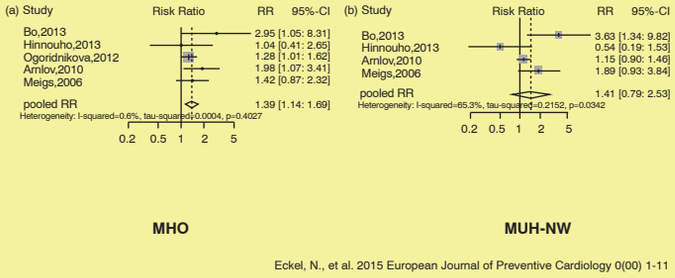


Fig. 1. Kaplan–Meier survival curves showing the association between body mass index-metabolic status phenotypes and cardiovascular disease events.

METABOLICALLY HEALTHY OBESITY AND CARDIOVASCULAR EVENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS (BMI AND HO,MA-IR)



Eckel, N., et al. 2015 European Journal of Preventive Cardiology 0(00) 1-11

Fig. 2. Meta-analyses of risk for cardiovascular events of participants with (a) metabolically healthy obesity, (b) metabolically unhealthy normal weight compared with metabolically healthy normal-weight individuals; phenotypes defined by body mass index categories and a pre-defined cut-off for HOMA-IR.

To determine which factors can influence the transition form an MHO to metabolically unhealthy obesity, in Spain a cohort of 3,052 individuals, aged 25 to 74 years were screened (Schroder H. et al, 2013) were followed up for 10 years (year 2000 to 2009). At baseline, 20.8% were MHO and overweight, but at the endof the study, 49.2% of these individuals were already classified as MUO and overweight. Risk factors identified at

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EATING BREAKFAST MAY IMPROVE THE HEALTH OF OBESE INDIVIDUALS!

Nemencio A. Nicodemus Jr., MD, FPCP, FPSEDM
President: Philippine Society of Endocrinology, Diabetes and Metabolism (PSEDM)
Board Member, PASOO & Asian Alliance for the Study of Neuroendocrine Tumors and Philippine Thyroid Association

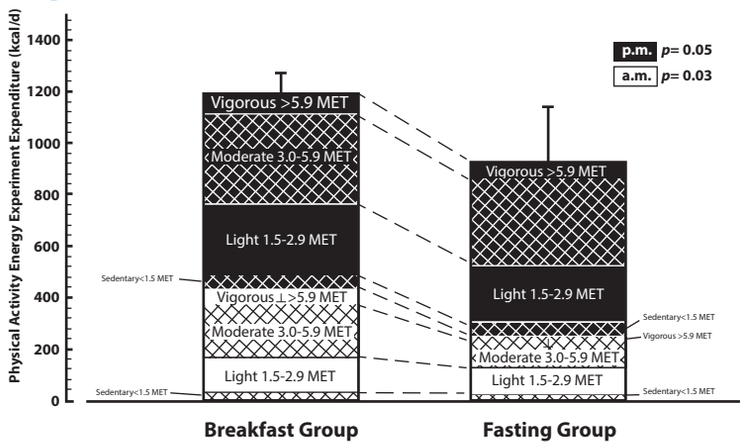
Since we were children, we would hear our grandparents and parents tell us to eat breakfast in order to be healthy. Most health advocates say it is the most important meal of the day. The importance that we put on breakfast is summed up in the famous quote **"Eat breakfast like a king, lunch like a prince and dinner like a pauper!"**

However, because of their busy lifestyle, some people are not able to eat breakfast regularly. Rather, they skip this meal then have heavy brunch once already in their workplace. For obese individuals, skipping a meal makes them believe that they will be healthier since they are cutting from their daily calories. But will cutting calories from breakfast really be better?

TO FAST OR TO BREAKFAST?

This particular concept was tested in a randomized controlled clinical trial of obese individuals aged 21 to 60 years old who were randomized to either daily breakfast before 11 AM or extended fasting with no food intake until 12 noon. The subjects were followed up for 6 weeks with these regimens. At the start and end of the study, the following were measured: resting metabolic rate, physical activity thermogenesis, diet-induced thermogenesis and energy intake. Several blood tests for metabolic control and cardiovascular disease risk were also obtained.

At the end of 6 weeks, those who took regular breakfast did not have significantly higher weight gain compared to those who did extended fasting. Daily energy intake was also not significantly greater in the breakfast group versus the fasting group. In contrast, those who did extended fasting had compensatory increases in their dietary intake later in the day. There were no differences in the metabolic factors and cardiovascular health markers that were tested (i.e., lipid profile, IL-6, CRP, fasting glucose, fasting insulin). But **a significant effect of eating regular daily breakfast was a greater physical activity thermogenesis in the morning.**[1]



A meta-analysis of randomized controlled trials looking at the impact of exercise on cardiorespiratory fitness and a variety of cardiometabolic biomarkers in adults without cardiovascular disease showed that exercise significantly improved cardiorespiratory fitness. Lipid profiles were improved in exercise groups, with lower levels of triglycerides and higher levels of the good cholesterol (high-density lipoprotein cholesterol). In addition, the meta-analysis showed that people aged < 50 years, men, and people with type 2 diabetes, hypertension, dyslipidemia, or metabolic syndrome (where obesity is a central feature) appeared to benefit more.[2]

Physical activity plays an important role in cardiovascular health. With increased physical activity, obese individuals may be able to achieve better overall health.

Thus, the advice for the general population is much more important to the obese individuals: **Eat breakfast regularly for better health!**

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2. Lin X et al. J Am Heart Assoc. 2015;4:e002014 doi: 10.1161/

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baseline were: increase in abdominal adiposity as defined by BMI, waist circumference and waist:height ratio. It was, however, mentioned that one unit change in healthy lifestyle index (exercise, diet and quitting smoking) was associated with a 33% lower risk of shifting to MUO. It appears that MHO individuals cross-sectionally and over brief periods of time may be at less risk of metabolic dysfunction, including diabetes, cardiovascular disease, and obesityrelated cancers compared to metabolically unhealthy normal weight (MUH-NW), but these effects are transient. In long-term studies, despite "healthy" metabolic profiles, these individuals may still be at increased risk for cardiovascular morbidity and mortality.

HOW TO SURVIVE A HEART ATTACK: BE 'LEAN'

Acd. Ramon F. Abarquez, Jr. MD, NAST, EFACC, FAsCC, FPCP, FPCC, CSPSH
Professor Emeritus, College of Medicine, University of the Philippines
System Academician, National Academy of Science and Technology
Board Member, PASOO

Heart attack (myocardial infarction) or coronary arterial lumen narrowing (by blood clot from an acute ruptured atherosclerotic chronically formed plaque/plaques) and/or vascular occlusion (by chronic vessel wall eccentric or concentric narrowing leading to a vessel stenosis, with/without blood clots) or both pathology, is the actual cause of morbidity and mortality in atherosclerotic disease.

HEART ATTACK: The heart muscles deprived of nutrients and oxygen contained in the blood, will fail to function adequately as a pump to eject sufficient blood to the entire body. During 'systole', the heart muscles contract sending blood into the 'epicardial' vessels, conduit vessels outside, surrounding the entire heart. Blood cannot enter the blood vessels penetrating the entire heart muscles because during 'systole', the heart is contracted. During 'diastole,' once the heart relaxes, to allow blood to enter the heart chambers, like a 'waterfall effect,' blood enters into the intra-myocardial (heart muscles) vessels to perfuse the myocardium. If the myocardium is deprived of sufficient blood supply needed for normal heart pumping action, a heart attack can follow.

LIFE-SAVER: The coronary collateral circulation (CCC) provides an alternative source of blood supply to the myocardium jeopardized by INADEQUATE BLOOD SUPPLY (ischemia), thus allowing bulk blood flow to be deliver by the native, pre-formed collateral extension (already sizeable capacity enlarged vessels). The protective effect of a well-developed CCC translates into relevant improvements in all-cause and cardiac mortality in the acute and/or chronic phase of coronary artery disease and a reduction of future adverse cardiovascular events. In acute coronary occlusion, the extent of collateral pathways is a significant determinant of the severity of myocardial infarction.

STABLE – CHRONIC - REPEATED – 'SILENT' HEART ATTACKS: In the chronic phase of CORONARY ARTERY DISEASE (CAD), the CCC compensates for the flow-limiting features of stenotic lesions. Effective blood flow across collaterals to jeopardized myocardium is induced when normal antegrade blood supply is compromised. Concurrently, the pressure drop across the stenosis leads to a pressure gradient along the pre-existing collaterals connecting to the constricted vessel, leading to their recruitment. Effective flow is induced from the donor artery across the collateral network to the recipient coronary artery, thereby sharing blood supply ensues. Consequently, repeated 'heart attacks' do occur, un-noticed, mild enough to pay attention to, thus, disregarded as an innocent symptom, felt but not so seriously recognized, or lasting a few minutes not to be considered as a 'killer-situation. Thus, the chest discomfort (angina) remains as a non-documented episode/nuisance. If recognized, a stable, chronic, repeated, but 'silent' heart attacks or ischemic episodes may precede an acute attack or may persist as chronic CAD.

COLLATERAL ACTIVATION MECHANISMS: 'Arteriogenesis' is growth of pre-existing collaterals while 'angiogenesis' is triggered by acute ischemia. Blood flow can increase maximally 10- to 20- fold by arteriogenesis, but only 1.5- to 1.7 fold by angiogenesis, (the sprouting of new, minute, high-resistance, low-flow capillaries). Thus, arteriogenesis has the capacity to compensate for an occluded artery, whereas angiogenesis does not. [Swiss Med Wkly. 2015;145:w14154]

THE 'PARADOX' OF CHRONIC TOTAL CORONARY OCCLUSIONS: In patients with chronic total occlusion (CTO), collateral function improves over a time period of, at least, 12 weeks after occlusion. The collateral remodeling in these patients relates to chronic stable CAD in a setting of a more or less gradual progression of coronary obstruction, (CORONARY VESSEL STENOSIS) which allows sufficient time for the growing collaterals to preserve myocardial viability. Thus, repeated effort – related but stable angina or recurrent 'silent' ischemia is associated with adequate developing CCC. With or without diabetes, 'silent' ischemia can be normalized by CCC. [Int J Cardiol. 2011 Mar 3;147(2):319]

ACUTE HEART ATTACK: In patients with acute coronary occlusion, the timeframe for insufficient collaterals to remodel (further) is generally too short. Plaque rupture (composite inflammatory cells and fat particles) leading to thrombus formation (blood clot) is the most common cause of acute coronary syndromes, which, when followed by

atherothrombosis, (clot in occluded vessel) leads to rapid and insidious thrombotic occlusion. In this context, it is of note that most of the coronary lesions ultimately responsible for major adverse cardiovascular events (MACE) have been shown to be angiographically mild [NEJM. 2011;364:226]. Therefore, the culprit lesion itself is unlikely to cause relevant collateral remodeling. In the absence of stenosis-induced CCC enlargement, the protective effect from acute ischemia is entirely dependent on the extent of the native CCC network. Therefore, the native collateral circulation is pertinent not only in the healthy state, but also in coronary artery disease (CAD). Notably, myocardial viability is not a prerequisite for the CCC remodeling. Thus, well-developed collaterals can be observed to grow only after its dependent myocardium has become entirely necrotic, again consistent with the notion that collateral growth can occur independently of ischemia.

COLLATERAL CIRCULATION PREDICTOR: Consequently, in patients with CAD, the severity of the underlying arterial obstruction has been found to be the only independent predictor for good collateral function. Furthermore, collateral flow was found to be higher in CTO than in non-occlusive obstructions [Catheter Cardiovasc Interv. 2014;83:9] However, there is still considerable inter-individual variability in the level of collateral function for a given stenosis severity, which highlights the modulation of the arteriogenic response by other factors.

COLLATERAL CIRCULATION UNIQUENESS: Notably though, collateral arteries and arterioles do not seem prone to atherosclerosis [Eur Heart J. 2013 Sep;34(34):2674] In patients with CTO with quantitatively assessed collateral function, hypertension was a predictor for better developed collaterals in a multivariate analysis. In the human heart without CAD, the contrary has been found to be true: better collateral function in the absence of arterial hypertension, TG/HDL-C ratio (0.27 to 14.33), after multivariate analysis, the highest tertiles of ACUTE MYOCARDIAL INFARCTION had a 5.32-fold increased risk of mortality. And, multivariate analysis among stable CTO CAD cases, revealed that **no diabetes, no dyslipidemia, nor renal dysfunction, but with multi-vessel disease and HTN history were independently associated with better coronary collateralization,** [J Zhejiang Univ Sci B. 2013 Aug;14(8):705] **Collateral vessel development is poorer in obese patients (BMI > 30 kg/m2) with ischemic heart disease** compared to normal range BMI, and the risk of having poor collateral vessel development is significantly increased. However, this might be **reflecting the cluster of risk factors, associated with metabolic syndrome, in which insulin resistance plays a major role** [Int J Obes Relat Metab Disord. 2003 Dec;27(12):1541]

REVASCULARIZATION CAUTIONS IN CCC: Percutaneous coronary intervention (PCI) of a coronary stenosis removes its associated resistance to antegrade coronary flow and the pressure gradient across the collateral network driving effective collateral flow is also diminished leading to regression of collateral function over time after revascularization. And, **re-canalize occlusive lesions was associated with a greater decrease in collateral function immediately after revascularization** compared with nonexclusive lesions [Heart. 2001;86:438] and at 6 months follow-up, only 4% of patients with subtotal or total occlusions at baseline showed sufficient collaterals, whereas this was observed in 18% of patients exclusively with a CTO after a mean of 5 months. Incidentally,10% of patients showed reocclusion after PCI of a CTO. In these patients collateral function was not different at follow-up when compared with the baseline value. A meta-analysis, of seven studies with angiographic or functional collateral assessment **good collaterals were predictive for re-stenosis, with a relative risk increase of 40% (9% to 80%, p = 0.009).** [BMC Medicine. 2012;10:62.] Thus, in patients with CAD, a well-functioning coronary collateral circulation is independently associated with a reduction in infarct size, left ventricular dysfunction and cardiovascular events, which translates into a relevant improvement in survival.

THUS, INVEST ON YOUR COLLATERALS. SLIM DOWN. YOUR LIFE IS AT STAKE.

From Page 6...

Adiponectin acts through its receptors which are primarily expressed in skeletal muscle and liver, endothelial cells, cardiomyocytes, and pancreatic cells. Despite being the most abundant adipokine secreted by adipose tissue, obese subjects have significantly lower levels of adiponectin when compared with nonobese subjects, and the adiponectin levels were negatively correlated to body mass index in both male and female subjects.

Adiponectin, via its anti-inflammatory actions on the vascular endothelium, is a molecular regulator of atherosclerosis. **Adiponectin appears to protect against all stages of atherosclerotic plaque formation, maintaining a functional, healthy endothelium; preventing plaque initiation, formation, and progression; and protecting against plaque rupture and thrombosis.** Mutations within the adiponectin gene appear to be associated with obesity, insulin resistance, and diabetes.

There are many other hormones and adipokines secreted by fat cells surrounding vascular tissue, each one contributing or deterring the atherosclerotic process. Leptin and adiponectin form but one axis on which this process evolves. We have much to learn about obesity, perivascular fat, and the cardiovascular disease.

Adipose tissue is a major source of energy for the human body. It is also a source of major adipocytokines, adiponectin and leptin. Insulin resistance is a condition in which insulin action is impaired in adipose tissue and is more strongly linked to intra-abdominal fat than to fat in other depots. The expression of adiponectin decreases with increase in the adiposity. Adiponectin mediates insulin-sensitizing effect through binding to its receptors AdipoR1 and AdipoR2, leading to activation of adenosine monophosphate dependent kinase (AMPK), PPAR-α, and presumably other yet-unknown signalling pathways. Weight loss significantly elevates plasma adiponectin levels. Reduction of adiponectin has been associated with insulin resistance, dyslipidemia, and atherosclerosis in humans.

PAVS

A Simple Clinical Tool to Promote Physical Activity

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It is said that that physical inactivity is one of the most important public health problems of the 21st century, and may even be the most important.[1] In their longitudinal study analyzing the effect insufficient physical activity on all-cause mortality among more than 60 thousand adults, it was shown that low cardiorespiratory fitness contributes 5 to 8 times more than obesity, diabetes, and hypercholesterolemia to all-cause mortality. Indeed, WHO says that those with insufficient physical activity have 20% to 30% increased risk of all-cause mortality and is one of the 10 leading risk factors for global mortality. Globally in 2010, 23% of adults were insufficiently active, higher in women than in men.[2] In the Philippines, according to the 2013 National Nutrition Survey of the Food and Nutrition Research Institute, insufficient physical inactivity as measured by IPAQ was 37% in adult men and 53% in adult women. On the other hand, physical activity (PA) is not only helpful in losing and maintaining weight among obese individuals, but more importantly reduces the risk of dozens of diseases – cardiovascular disease, diabetes II, abnormal lipid profile, metabolic syndrome, colon and breast cancer, early death and others. **Moreover, physical activity improves cardiorespiratory fitness and muscle mass, healthier body mass and composition, and even better cognitive function.**

With these growing evidence on the ill-effects of physical inactivity and the beneficial effects of physical activity, **it would do well for health care providers particularly physicians to promote physical activity and exercise at every client visit.** Physicians have the unique opportunity to encourage physical activity among their patients by way of a simple physical activity assessment and monitoring tool: **PAVS or Physical Activity Vital Sign.** In other words, as the initial part of the Exercise is Medicine™ initiative recommends, the level and type of the patient's physical activity should be considered as a "vital sign", similar to measuring the patient's weight and blood pressure. Simply asking patients about what physical activity or exercise the patient is engaged in, will open the opportunity for brief counseling and advice on a healthy lifestyle. Such questioning and can even be done by the physician's medical assistant and results recorded on the patient's written or electronic record. PAVS is relatively simple to implement, requires a very brief period of time by the physician or medical assistant, and may lead to additional PA counselling and referral.

The Physical Activity Vital Sign (PAVS)

- The Physical Activity Vital Sign (PAVS) is a clinical tool designed to screen PA levels in adults
- The PAVS consists of **two** simple questions:

1. How many days a week do you engage in moderate to strenuous exercise (like a brisk walk)?

2. On average how many minutes per day do you exercise at this level?

***It is essential that these questions be asked with every patient AND that the responses recorded at EVERY patient visit to establish a continuing record of their PA levels**

References:
[1] Blair, SN. Br J Sports Med 2009;43:1-2
[2] http://who.int/gho/ncd/risk_factor/physical_activity_text/en/

Fat Replacement Strategies

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Currently, five types of fat replacements are available in the United States. These products are used by manufacturers to yield food products with lesser fat content.

The first and the simplest fat replacement is water. The addition of water yields a product, such as diet margarine, with less fat per serving than the normal product.

Starch derivatives such as Z-Trim, cellulose, Maltrim, Stellar and Oatrim, that bind water form a second type of fat replacement. The resulting gel replaces some of the mouth feel lost by the removal of fat. Starch derivatives are used in luncheon meats, salad dressings, frozen desserts, baked goods, spreads, dips and candies. They are not used for fried foods and most of starch derivatives contain calories . Gums extracted from plants can also be used to replace fat. They thicken the product and replace some of the body that fat provides.They are used in salad dressings.
Protein-derived fat replacements uses egg and milk.The proteins have been treated to produce microscopic, mist like protein globules. Simplesse is a fat replacement of this type which has low calorie value - about 1 to 2 kcal/g and the product has a high water content. Simplesse is used primarily in frozen desserts. It can not be used for cooking or frying.

Another protein-derived fat replacement is Dairy-Lo. It is a modified whey protein concentrate that has fat like properties.

The fifth form of fat replacement is the engineered fat. An example is Olestra, which is made by chemically linking fatty acids to sucrose. Olestra cannot be digested by either human digestive enzymes or bacteria that live in the intestines, and thus, yields no energy to the body. Olestra, however, binds the fat-soluble vitamins ADEK and carotenoids, thus, reducing their absorption. To compensate, the manufacturers add these vitamins, but not carotenoids, to Olestra. Other engineered fats are Salatrim, marketed under the name Benefat and Appetize.

Other than these fat replacement strategies, there are many ways one can avoid eating too much total fat and saturated fat such as:

- Steam, boil and bake rather than fry
- Season vegetables with herbs and spices rather than with sauces butter or margarine
- Limit baked goods made with large amounts of fat especially croissants, doughnuts, muffins, biscuits and butter rolls
- Use fruit puréed in place of fat when possible, such as in quick breads
- Replace whole milk with nonfat or reduced-fat milk
- Choose lean cuts of meat
- Trim fat from meat before and after cooking
- Remove skin from poultry
- Chill meat or poultry broth until the fat solidifies. Spoon off the fat before using the broth.
- Pay attention to the type and amount of fat of commercially prepared foods.
- Read food labels

LOVE and Attain Healthy Weight

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LOVE is defined as a feeling of strong or constant affection for a person. It can also denote a feeling of great interest, affection, or enthusiasm for something.

LOVE touches our heart and makes us feel good.

I see and feel LOVE when the mother breastfeeds her baby and the parents hug and kiss their children. A homeless boy loves his mother and wants a better life for his mother; so, he studies hard in the school, does his homework on a wooden stool placed close to a fast food restaurant to catch the light from the store (because he does not have a home with light). His photo was taken by a student, Joyce Torrefranca, and it appeared in the newspaper and social media. LOVE indeed makes life beautiful and worth living.

We love other person; we love things. However, we have to learn to love ourselves and take good care of our bodies.

LOVE ourselves to health. LOVE ourselves enough to live a healthy lifestyle.

L Limit food portion to just right size & choice
Avoid trans-fat

O Omit sugary drinks
Drink water – aim for more than 6 to 8 glasses a day

V Vegetables and fruits daily
Goal is 5 servings (fist size) daily or half of the plate each meal

E Exercise / physical activity daily
Aim for 60 minutes a day

Eat right and have a balanced diet. Don't overeat; otherwise, our stomach, intestines and also pancreas will overwork. Obese people have high risk of insulin resistance; their insulin does not work efficiently and diabetes mellitus occurs earlier in obese children and young adults, particularly those with big abdominal circumference. People with "central" (abdominal) obesity has increased visceral fat and higher risks for type 2 diabetes, hypertension, cardiovascular diseases, dyslipidemia (high triglycerides, and LDL, low HDL levels) and non-alcoholic fatty liver disease, early stroke and premature death.

Move more to be fit. Reduce sedentary activity and limit television watching, video game playing and computer time to 2 hours or less. Right type and amount of exercise can improve the endurance, flexibility and strength.

Having a healthy lifestyle leads to a healthy weight.

Health is wealth. According to an ancient Roman poet, Virgil, **"the greatest wealth is health."**

Be healthy and happy!

PASOO and Manila Doctors Hospital held an educational activity at Rafael Elementary School, Makati City on August 9, 2016 to highlight the importance of healthy lifestyle.

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PASOO IN ACTION

2015

Exercise is Medicine Prescription Course for Primary Care Physicians
November 3-4, 2015
Net Lima Bldg, Bonifacio Global City, Makati City

PASOO 21st Annual Convention
September 3, 2015
EDSA Shangri-La Hotel, Mandaluyong City

PASOO-AstraZeneca Healthy Cookbook 2nd Edition Launch
December 9, 2015
Guevarra's, San Juan, Metro Manila

2016

PASOO Lay Forum
July 29, 2016
Laoag City Auditorium, Laoag City

PASOO 7th Intensive Obesity Workshop for Health Professionals
July 30, 2016
Plaza Del Norte, Laoag, Ilocos Norte

PASOO Kids Lecture Series in cooperation with Manila Doctors Hospital
August 9, 2016
Rafael, Palma Elem. School, Makati City

1st World Obesity Day
October 11, 2015
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