Banner University Medicine

BUMC Phoenix

Update on Obesity and Bariatric Surgery

Your Role on the Interdisciplinary Team

Christine Lovato MD, FACS, FASMBS

Friday, August 3, 2018

BUMCP Center of Excellence

Banner University Medicine

Our Team

Melissa Davis, DNP

Heather Noriega, FNP

Banner University Medicine

Our Team

Jessica Arroyo RD, CSP

Megan Flores RD, MS

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Our Team

Robin Blackstone MD, FACS, FASMBS

Christine Lovato MD, FACS, FASMBS

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Objectives

1. Update on the pathophysiology of obesity and the role of surgery.

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2. Review outcomes of Bariatric Surgery including improvement of co-morbid disease and the PCP role on the interdisciplinary team.

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2. Review outcomes of Bariatric Surgery including improvement of co-morbid disease and the PCP role on the interdisciplinary team.
3. Get to know the Bariatric Surgery program at BUMCP – how can I get my patients to surgery?

Defining Obesity – Body Mass Index (BMI)

Normal weight:	18.5-24.9
Overweight:	25-29.9
Obese:	30-39.9
Morbidly Obese:	≥ 40
Super Obese:	≥ 50

Snapshot of Obesity in the US

- 38% of adults in the United States are considered obese.

Source: Centers for Disease Control and Prevention (CDC)

Snapshot of Obesity in the US

- 38% of adults in the United States are considered obese.
- 17% of our children are obese.

Source: Centers for Disease Control and Prevention (CDC)

Snapshot of Obesity in the US

- We spend over $270 Billion per year on obesity related health problems.

Source: New Society of Actuaries Study 2013

Associated Comorbidities

- High blood pressure
- Heart failure
- Venous stasis/blood clots
- Pulmonary hypertension
- Obstructive sleep apnea
- Hypoventilation syndrome
- Asthma
- Type II Diabetes
- High cholesterol
- Fatty liver disease
- Depression/Anxiety
- Reflux/heartburn
- Gallstones
- Degenerative joint disease
- Degenerative disk disease
- Osteoarthritis
- Ventral hernias
- Urinary incontinence
- Irregular periods/PCOS
- Skin infections
- Headaches

Metabolic Syndrome

Defined by the presence of at least three of the following:

1. Waistline > 40”(male) OR > 35”(female)
2. Fasting blood sugar > 100 mg/dl
3. Triglycerides > 150 mg/dl
4. Blood pressure > 130/85 mm Hg
5. HDL < 40 male, < 50 female

1863: Rudolf Virchow proposed the link between Cancer and Inflammation

- Uterine
- **Breast**
- Gastric
- Esophageal
- Hepatic
- **Colon**
- Renal
- **Pancreatic**

1863: Rudolf Virchow proposed the link between Cancer and Inflammation

- Uterine
- **Breast – 12% per 5 BMI**
- Gastric
- Esophageal
- Hepatic
- **Colon – 30% higher**
- Renal
- **Pancreatic – 1.5x higher**

How did we get here??

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- Genetics

Estimated to contribute 40-70%.

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Rarely monogenic. Most often a complex interaction between multiple genes.

Genetics

- A child with:

-One obese parent has a 25-50% chance of being obese

Genetics

- A child with:

-One obese parent has a 25-50% chance of being obese

-Two obese parents has a 75% chance of being obese

Genetics

A Twin Study of Human Obesity

Stunkard, Albert J MD, Foch, Terryl T PhD, Zdenek, Hrubec ScD

JAMA. 1986;256(1):51-54.

- Height, weight, and BMI were assessed in a sample of 1974 monozygotic and 2097 dizygotic male twin pairs.
- Concordance rates for different degrees of overweight were twice as high for monozygotic twins as for dizygotic twins.
- Classic twin methods estimated a high heritability for height, weight, and BMI, both at age 20 years (.80, .78, and .77, respectively) and at a 25-year follow-up (.80, .81, and .84, respectively).
- Height, weight, and BMI were highly correlated across time, and a path analysis suggested that the major part of that covariation was genetic. These results are similar to those of other twin studies of these measures and suggest that human fatness is under substantial genetic control.

Genetics

The New England
Journal of Medicine

Volume 314 JANUARY 23, 1986 Number 4

AN ADOPTION STUDY OF HUMAN OBESITY

ALBERT J. STUNKARD, M.D., THORKILD I.A. SØRENSEN, DR.MED., CRAIG HANIS, PH.D., THOMAS W. TEASDALE, M.A., RANAJIT CHAKRABORTY, PH.D., WILLIAM J. SCHULL, PH.D., AND FINI SCHULSINGER, DR.MED.

Abstract We examined the contributions of genetic factors and the family environment to human fatness in a sample of 540 adult Danish adoptees who were selected from a population of 3580 and divided into four weight classes: thin, median weight, overweight, and obese. There was a strong relation between the weight class of the adoptees and the body-mass index of their biologic parents — for the mothers, $P<0.0001$; for the fathers, $P<0.02$. There was no relation between the weight class of the adoptees and the body-mass index of their adoptive parents. Cumulative distributions of the body-mass index of parents showed similar results; there was a strong relation between the body-mass index of biologic parents and adoptee weight class and no relation between the index of adoptive parents and adoptee weight class. Furthermore, the relation between biologic parents and adoptees was not confined to the obesity weight class, but was present across the whole range of body fatness — from very thin to very fat. We conclude that genetic influences have an important role in determining human fatness in adults, whereas the family environment alone has no apparent effect. (N Engl J Med 1986; 314:193-8.)

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- Epigenetics

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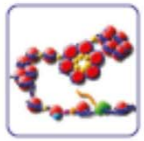
- Epigenetics

Factors that affect how genes are translated, expressed and regulated

Includes DNA methylation, histone tail modifications, and chromatin remodeling.

How did we get here??

van Dijk *et al. Clinical Epigenetics* (2015) 7:66
DOI 10.1186/s13148-015-0101-5



CLINICAL EPIGENETICS

REVIEW Open Access

Recent developments on the role of epigenetics in obesity and metabolic disease

Susan J. van Dijk[1], Ross L. Tellam[2], Janna L. Morrison[3], Beverly S. Muhlhausler[4,5†] and Peter L. Molloy[1*†]

How did we get here??

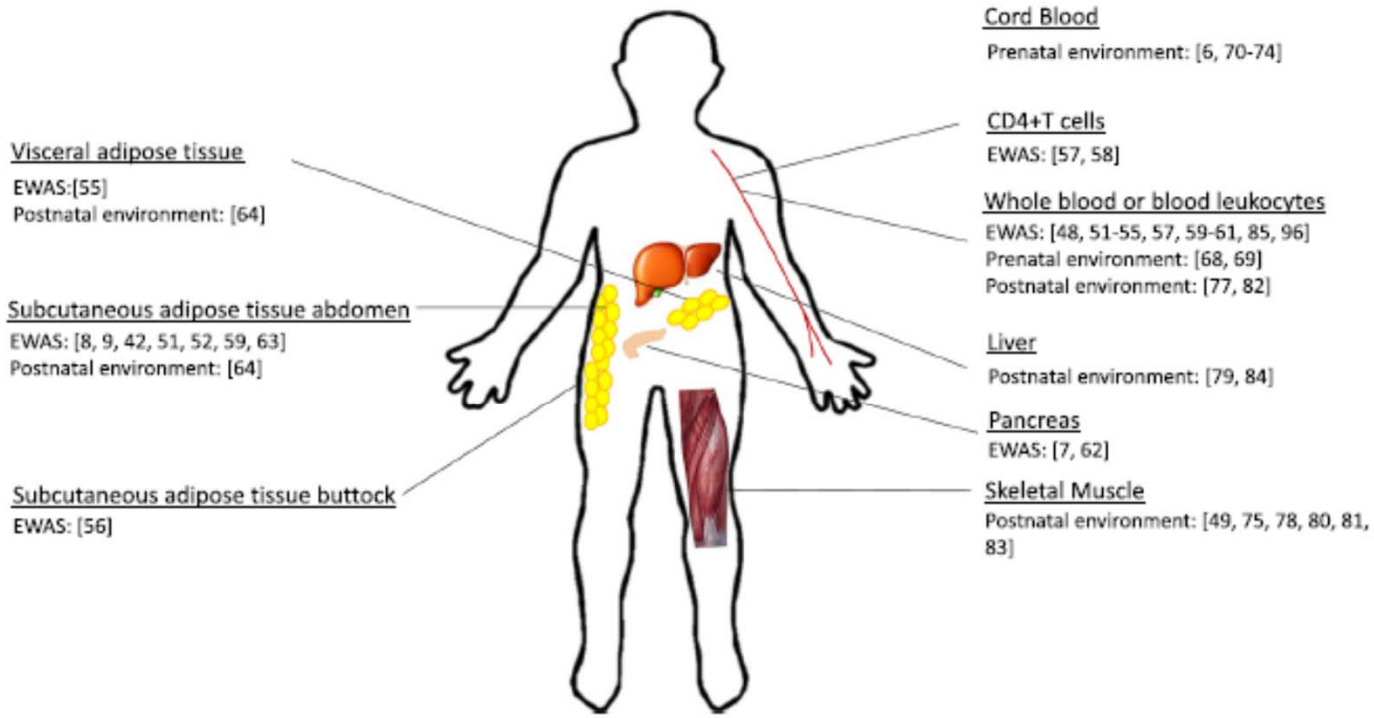


Fig. 2 Overview of human tissues used for studies into the role of epigenetics in obesity. For each tissue, studies are grouped by study type; epigenome-wide association studies (EWAS) and pre- and postnatal interventions. *Numbers* represent the reference number

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How did we get here??

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- Epigenetics

Factors that affect how genes are translated, expressed and regulated.

- Culture/Environment

Food products, sugar intake, eating too fast and relative inactivity compared to previous generations.

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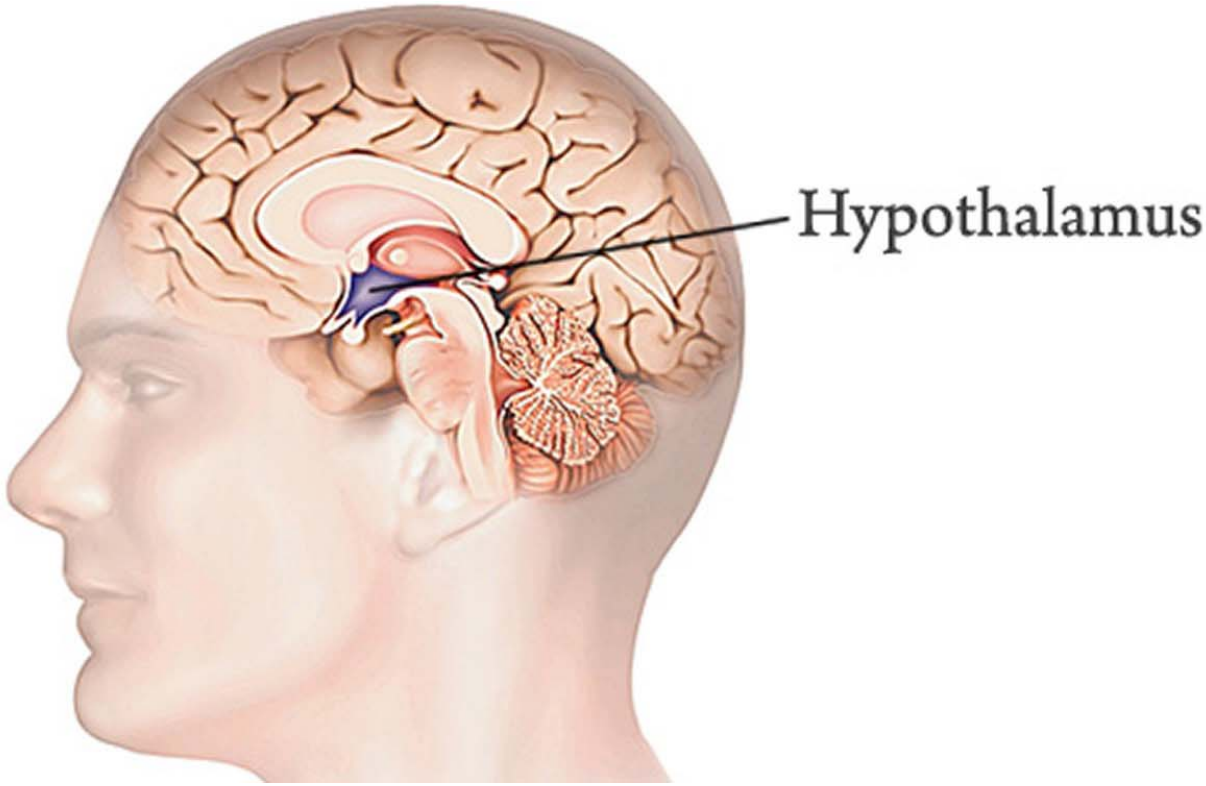
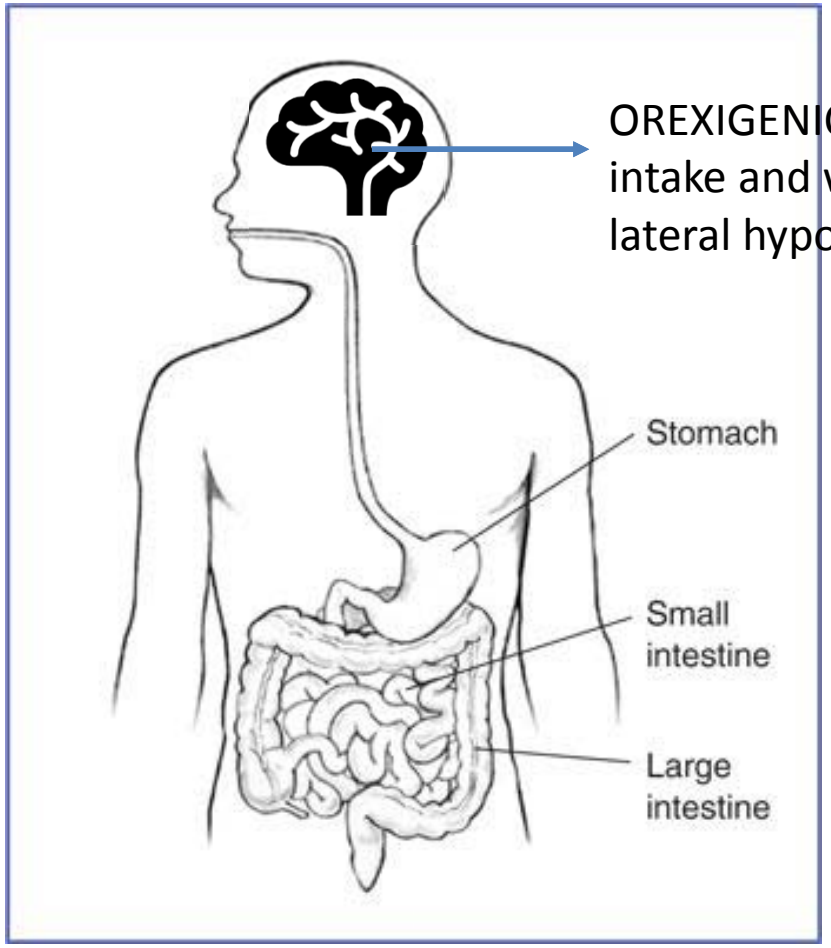
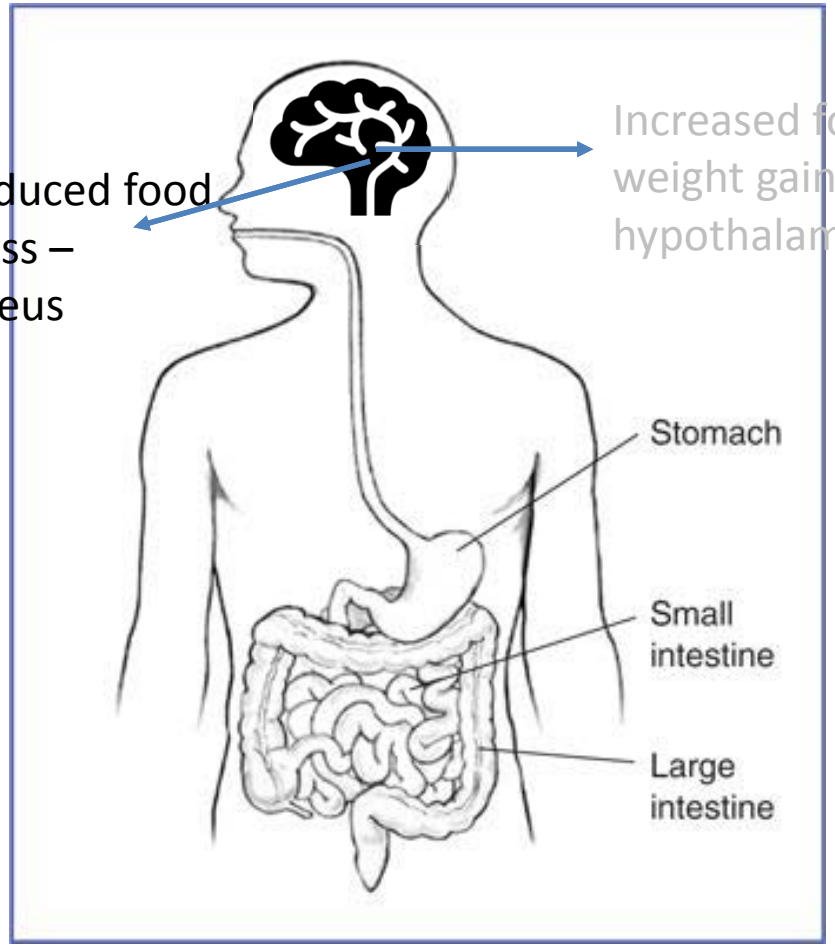
Neural regulation of hunger and satiety





OREXIGENIC - Increased food intake and weight gain - lateral hypothalamic area

ANOREXIGENIC - Reduced food intake and weight loss – paraventricular nucleus



Cell

Leading Edge
Review

Gut-Brain Cross-Talk in Metabolic Control

Christoffer Clemmensen,[1,2] Timo D. Müller,[1,2] Stephen C. Woods,[3] Hans-Rudolf Berthoud,[4] Randy J. Seeley,[5] and Matthias H. Tschöp[1,2,*]

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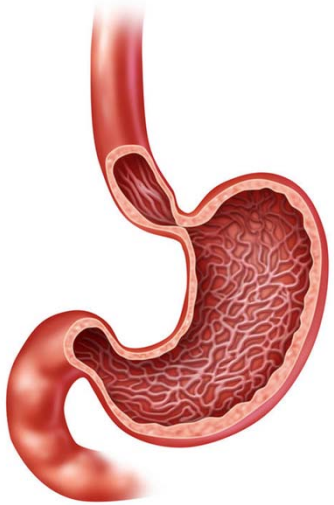
[2]Division of Metabolic Diseases, Department of Medicine, Technische Universität München, 80333 Munich, Germany
[3]Department of Psychiatry and Behavioral Neuroscience, University of Cincinnati, Cincinnati, OH 45220, USA
[4]Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, LA 70803, USA
[5]Departments of Surgery, Internal Medicine, and Nutritional Sciences at the University of Michigan, Ann Arbor, MI 48109, USA
*Correspondence: tschoep@helmholtz-muenchen.de
http://dx.doi.org/10.1016/j.cell.2017.01.025

Because human energy metabolism evolved to favor adiposity over leanness, the availability of palatable, easily attainable, and calorically dense foods has led to unprecedented levels of obesity and its associated metabolic co-morbidities that appear resistant to traditional lifestyle interventions. However, recent progress identifying the molecular signaling pathways through which the brain and the gastrointestinal system communicate to govern energy homeostasis, combined with emerging insights on the molecular mechanisms underlying successful bariatric surgery, gives reason to be optimistic that novel precision medicines that mimic, enhance, and/or modulate gut-brain signaling can have unprecedented potential for stopping the obesity and type 2 diabetes pandemics.

Neural regulation of **hunger** and satiety

Ghrelin

Produced by the endocrine cells of the gastric fundus



Neural regulation of **hunger** and satiety

Ghrelin

Neural regulation of hunger and **satiety**

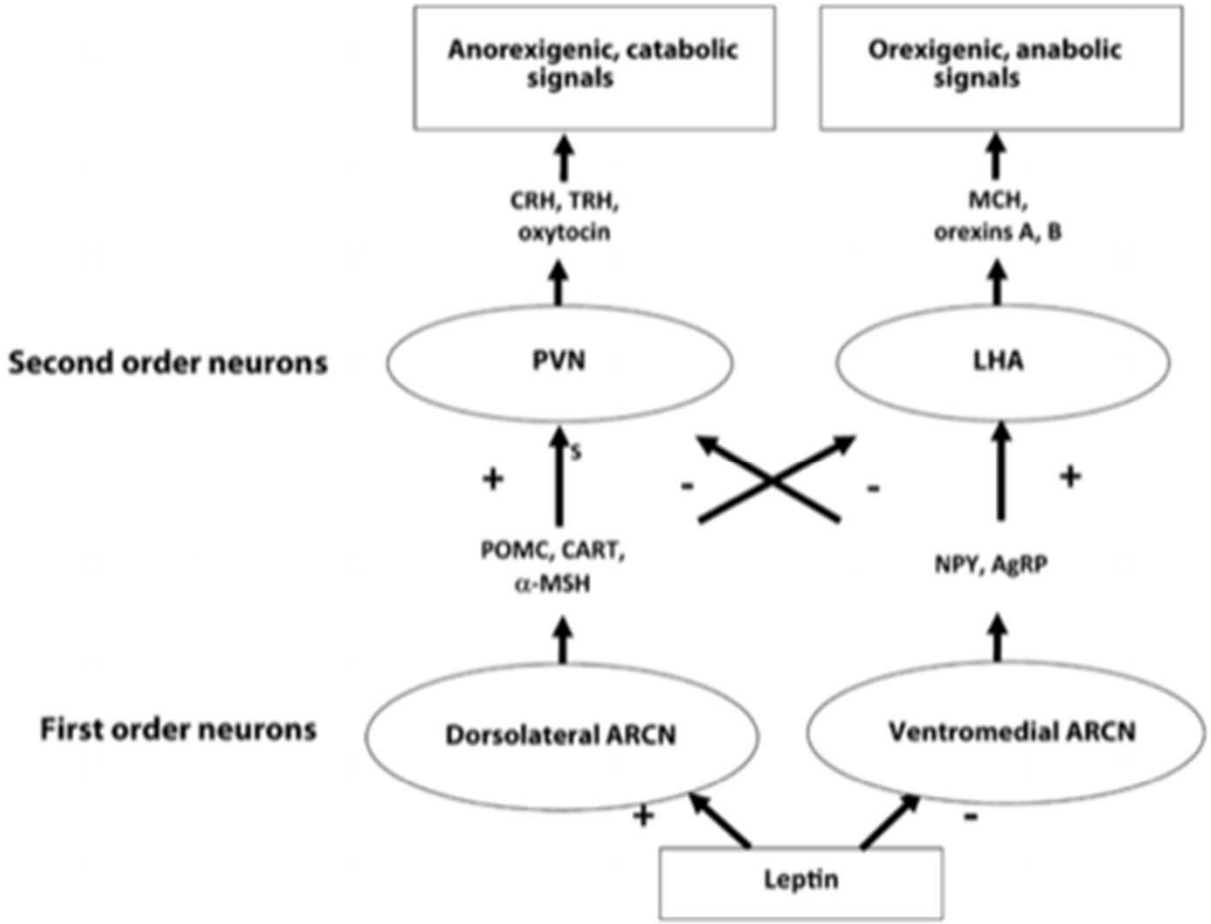
Leptin

Produced by peripheral adiopose tissue in proportion to adipose tissue mass

Stimulates the hypothalamus (both the satiety and reward centers)

Leptin increases energy expenditure and decreases food intake

Neural regulation of hunger and satiety


Anorexigenic, catabolic signals
Orexigenic, anabolic signals
CRH, TRH, oxytocin
MCH, orexins A, B
Second order neurons
PVN
LHA
+
-
-
+
POMC, CART, α-MSH
NPY, AgRP
First order neurons
Dorsolateral ARCN
Ventromedial ARCN
+
-
Leptin

Neural regulation of hunger and **satiety**

Leptin

Hyperleptinemia in obesity – decreased sensitivity vs "resistance?"

Neural regulation of hunger and **satiety**

CCK (cholecystokinin)

Produced by the duodenum

Stimulates release of digestive enzymes, slows gastric emptying.

Neural regulation of hunger and **satiety**

GLP-1 (glucagon like peptide-1)

Produced by small intestine

Enhances insulin secretion

Inhibits gastric emptying

Neural regulation of hunger and **satiety**

GLP-1 (glucagon like peptide-1)

Produced by small intestine

Byetta, Victoza, Trulicity

Enhances insulin secretion

Inhibits gastric emptying

Neural regulation of hunger and **satiety**

GIP (gastric inhibitory peptide)

Produced by small intestine

Enhances insulin secretion

Neural regulation of hunger and **satiety**

PYY (peptide YY)

Produced by distal small intestine and colon

Inhibits gastric emptying

Neural regulation of hunger and **satiety**

PP (pancreatic polypeptide)

Produced by pancreas

Regulates pancreatic secretion

Neural regulation of hunger and **satiety**

Adiponectin

Apelin

CCL-2

Lipcalin 2

PAI-1

Resistin

RBP-4

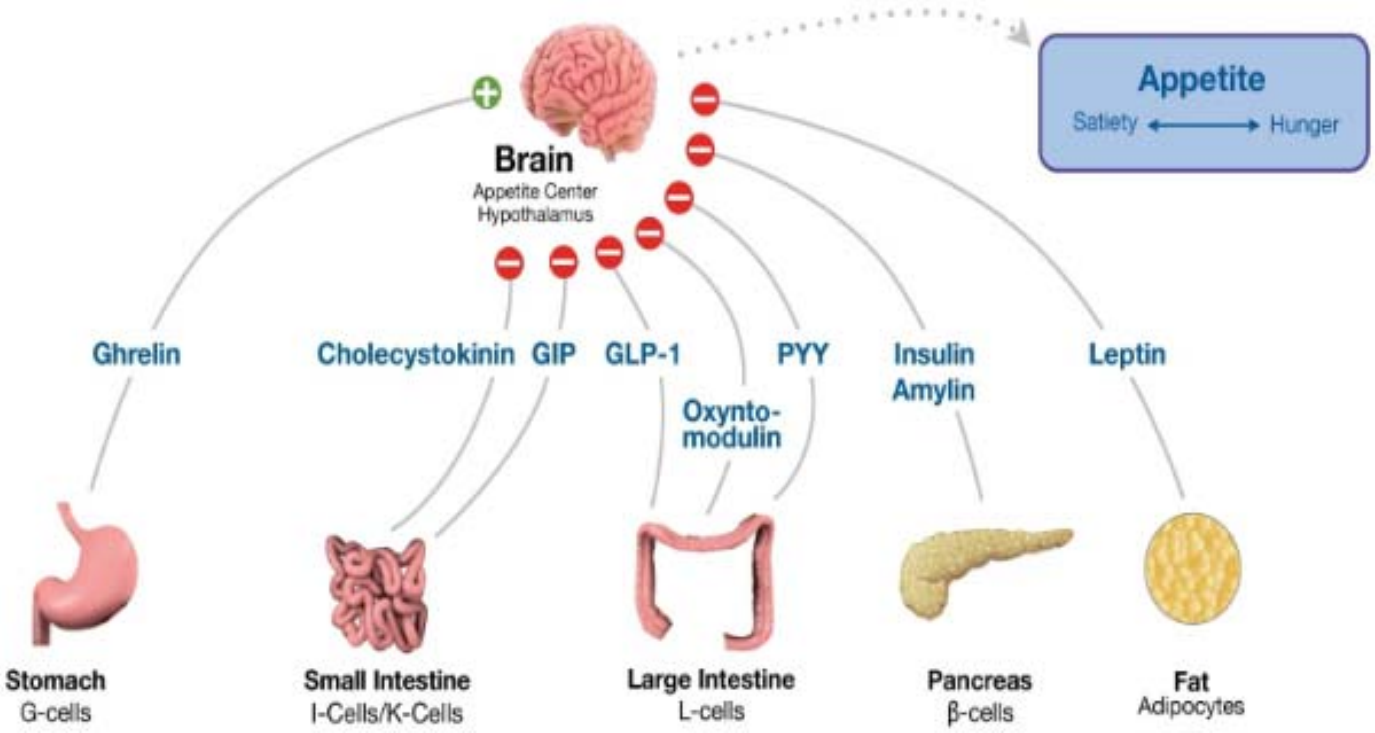
SFRP5

Visfatin

Neural regulation of hunger and **satiety**

Adiponectin	Amylin
Apelin	Glucagon
CCL-2	Oxyntomodulin
Lipcalin 2	
PAI-1	IFN gamma
Resistin	IL-1
RBP-4	IL-6
SFRP5	IL-10
Visfatin	TNF alpha

Neural regulation of hunger and satiety



Source: Genes, emotions and gut microbiota: The next frontier for the gastroenterologist.
World Journal of Gastroenterology: 23(17):3030-3042; May 2017.

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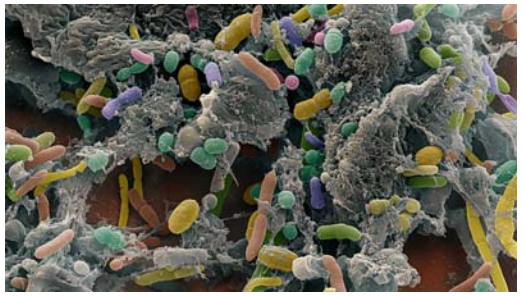
Gastrointestinal Flora in Obesity

Human Microbiome Project 2012



Ratio of microbial (bacteria) to human cells is 1.3 to 1!

Most live in the GI tract.

In mice, gut flora present in obese mice showed an increased capacity for harvesting energy.

Highly processed foods with numerous additives have been shown to decrease diversity in the gut microflora.

- **Acidulents**
- Confer sour or acid taste. Common acidulents include vinegar, citric acid, tartaric acid, malic acid, fumaric acid, and lactic acid.
- **Acidity regulators**
- Acidity regulators are used for controlling the pH of foods for stability or to affect activity of enzymes.
- **Anticaking agents**
- Anticaking agents keep powders such as milk powder from caking or sticking.
- **Antifoaming and foaming agents**
- Antifoaming agents reduce or prevent foaming in foods. Foaming agents do the reverse.
- **Antioxidants**
- Antioxidants such as vitamin C are preservatives by inhibiting the degradation of food by oxygen.
- **Bulking agents**
- Bulking agents such as starch are additives that increase the bulk of a food without affecting its taste.
- **Food coloring**
- Colorings are added to food to replace colors lost during preparation or to make food look more attractive.
- **Fortifying agents**
- Vitamins, minerals, and dietary supplements to increase the nutritional value
- **Color retention agents**
- In contrast to colorings, color retention agents are used to preserve a food's existing color.
- **Emulsifiers**
- Emulsifiers allow water and oils to remain mixed together in an emulsion, as in mayonnaise, ice cream, and homogenized milk.
- **Flavors**
- Flavors are additives that give food a particular taste or smell, and may be derived from natural ingredients or created artificially.

- **Flavor enhancers**
- Flavor enhancers enhance a food's existing flavors. A popular example is monosodium glutamate. Some flavor enhancers have their own flavors that are independent of the food.
- **Flour treatment agents**
- Flour treatment agents are added to flour to improve its color or its use in baking.
- **Glazing agents**
- Glazing agents provide a shiny appearance or protective coating to foods.
- **Humectants**
- Humectants prevent foods from drying out.
- **Tracer gas**
- Tracer gas allow for package integrity testing to prevent foods from being exposed to atmosphere, thus guaranteeing shelf life.
- **Preservatives**
- Preservatives prevent or inhibit spoilage of food due to fungi, bacteria and other microorganisms.
- **Stabilizers**
- Stabilizers, thickeners and gelling agents, like agar or pectin (used in jam for example) give foods a firmer texture. While they are not true emulsifiers, they help to stabilize emulsions.
- **Sweeteners**
- Sweeteners are added to foods for flavoring. Sweeteners other than sugar are added to keep the food energy (calories) low, or because they have beneficial effects regarding diabetes mellitus, tooth decay, or diarrhea.
- **Thickeners**
- Thickening agents are substances which, when added to the mixture, increase its viscosity without substantially modifying its other properties.

The Set Point

Calories consumed

Calories burned

Defending the set point

Resting Metabolic Rate

Defending the set point

Resting Metabolic Rate

"Energy required by your body to perform the most basic functions when your body is at rest. Including breathing, circulating blood or basic brain functions."

Defending the set point

What affects RMR?

Defending the set point

What affects RMR?

Age

Gender

Muscle Mass (or FFM)

Thyroid hormones

Genetics/Epigenetics

Defending the set point

What affects RMR?

Age

Gender

Muscle Mass (or FFM)

Thyroid hormones

Genetics/Epigenetics

Dietary Intake

Defending the set point

Dietary intake

Thermogenic Effect of Food (TEF)

or

Dietary Induced Thermogenesis (DIT)

The amount of energy expenditure expelled in order to process food for use and storage.

Defending the set point

Defending the set point

Dietary intake

Proteins

Fats

Carbohydrates

Defending the set point

What affects RMR?

Age

Gender

Muscle Mass (or FFM)

Thyroid hormones

Genetics

Dietary Intake

Physical activity

Defending the set point

Physical activity

Glucose

Defending the set point

Metabolic Adaptation

The Biggest Loser Study

Defending the set point

Persistent metabolic adaptation 6 years after "The Biggest Loser" competition. *Obesity.* 2016 Aug;24(8):1612-9.

Defending the set point

Average age: 34

Average BMI: 49.5

Average weight at start of program: 325 lbs

Average **weight loss** at end of program (30 weeks): 198 lbs

Defending the set point

Average age: 34

Average BMI: 49.5

Average weight REGAIN at 6 years: 90 lbs

Average weight at start of program: 325 lbs

Average **weight loss** at end of program (30 weeks): 127 lbs

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Defending the set point

Average RMR at start of program: 2607 kcal/day

Defending the set point

Average RMR at start of program: 2607 kcal/day

Average RMR at end of program: 1996 kcal/day

Defending the set point

Average RMR at start of program: 2607 kcal/day

Average RMR at end of program: 1996 kcal/day

That's a difference of 610 kcal/day

Defending the set point

Average RMR at start of program: 2607 kcal/day

Average RMR at end of program: 1996 kcal/day

That's a difference of 610 kcal/day

This decrease in metabolic rate persisted at the 6 year follow up.

Defending the set point

This permanent decrease in RMR was <u>not</u> observed in Gastric Bypass surgery patients. (RMR normalized after one year).

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What can be done?

What can be done?

Modify epigenetics

DNA methylation, histone tail modifications, and chromatin remodeling

What can be done?

Modify epigenetics

Modify culture/environment

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Nilla
HERSHEY'S
Hungry Jack


HALF & HALF
SKIM

What can be done?

Modify epigenetics

Modify culture/environment

Drugs

What can be done?

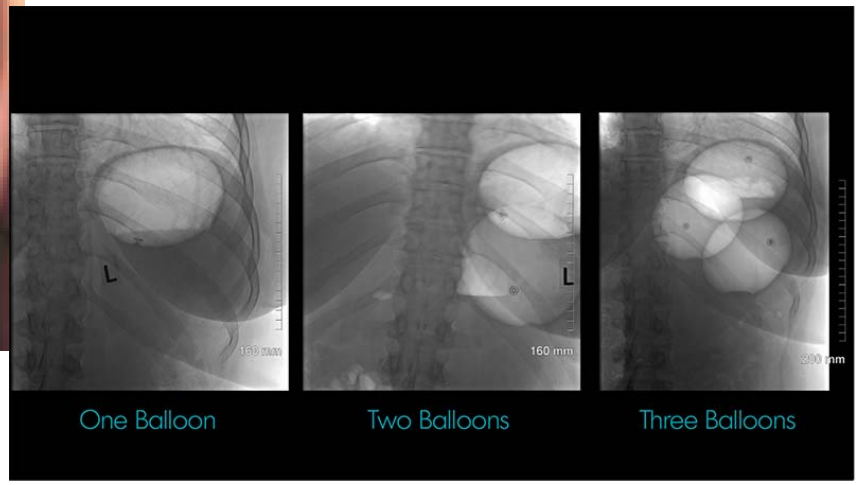
Modify epigenetics

Modify culture/environment

Drugs

Weight loss balloon

Weight loss balloon



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What can be done?

Modify epigenetics

Modify culture/environment

Surgery

Drugs

Weight loss balloon

With diet, exercise, and physician support,

Success rate for patients of BMI > 30 kg/m²

to keep weight off for ≥ 5 years: ?

With diet, exercise, and physician support,

Success rate for patients of BMI > 30 kg/m²

to keep weight off for ≥ 5 years: ~6%

With diet, exercise, and physician support,

Success rate for patients of BMI > 30 kg/m²

to keep weight off for ≥ 5 years: ~6%

Success rate following Bariatric Surgery: ?

With diet, exercise, and physician support,

Success rate for patients of BMI > 30 kg/m²

to keep weight off for ≥ 5 years: ~6%

Success rate following Bariatric Surgery: 80%

Where did we begin?

- Jejunoileal Bypass, Kremen and Linner 1954
- Gastric Bypass, Mason 1967
- Biliopancreatic Diversion , Scopinaro 1978
- Vertical Banded Gastroplasty, Mason 1982
- Duodenal Switch, Marceau 1988
- Laparoscopic Gastric Bypass, Wittgrove 1993
- Lap-Adjustable Gastric Band, Belachew 1993
- Vertical Sleeve Gastrectomy, Johnston 2003

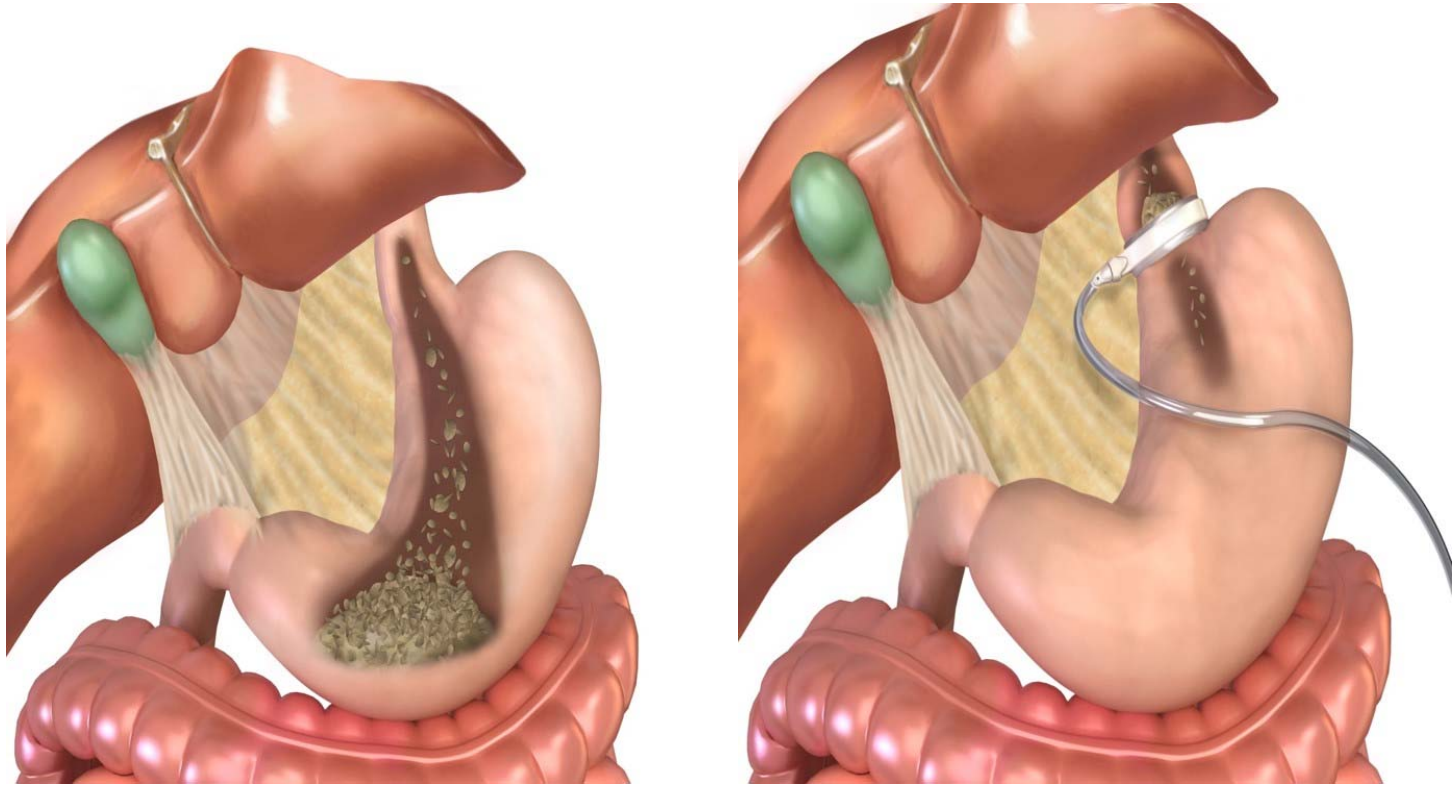
The Laparoscopic advantage

- Less post-operative pain
- Earlier return to bowel function
- Shorter hospital stay
- Earlier return to daily activities
- Fewer and less severe wound complications
- Fewer lung complications
- Minimal scarring

Bariatric Surgery

- Laparoscopic adjustable gastric band
- Laparoscopic Vertical Sleeve Gastrectomy
- Laparoscopic Roux-en-y Gastric Bypass
- Laparoscopic Biliopancreatic Diversion w/Duodenal Switch

Laparoscopic Adjustable Gastric Band



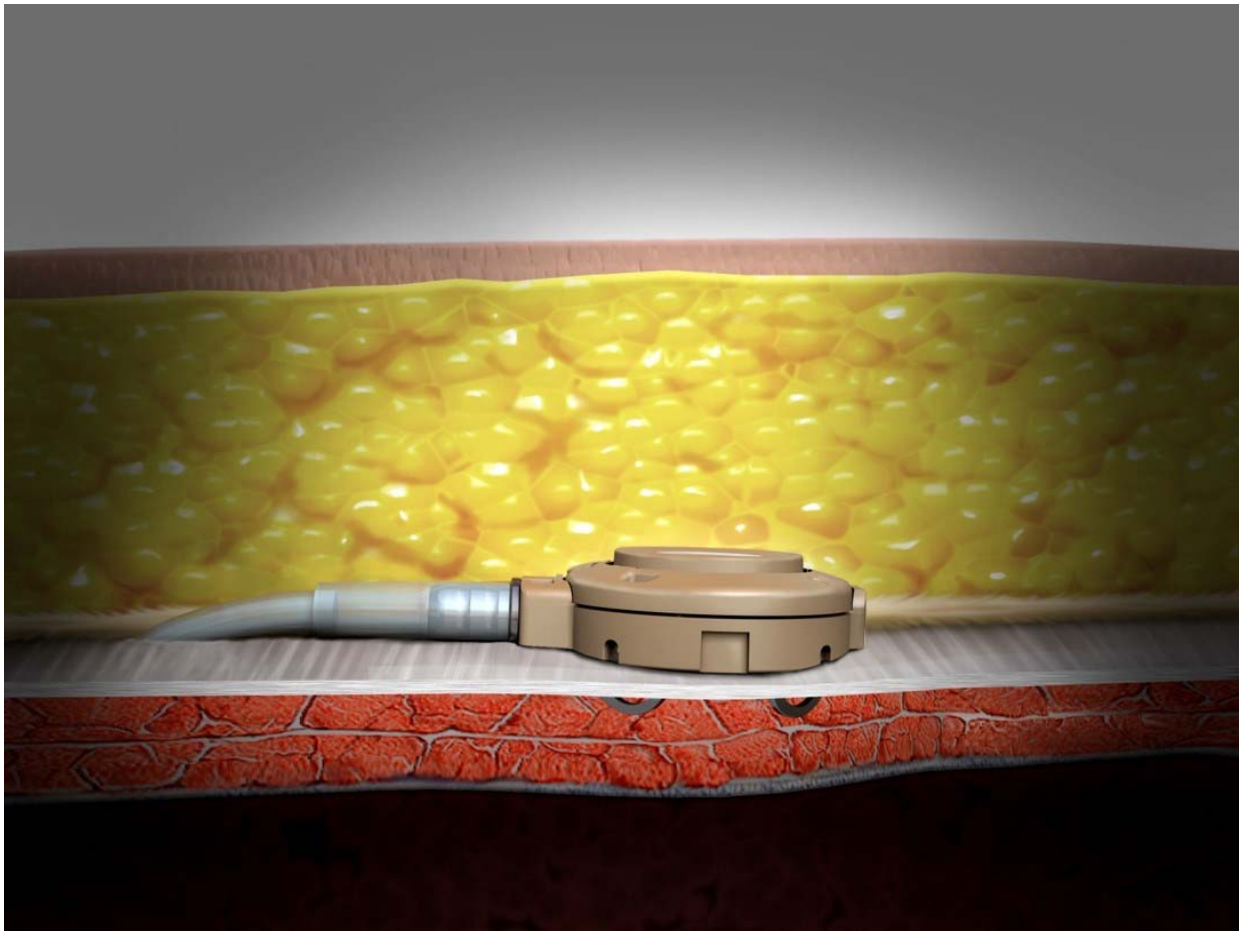
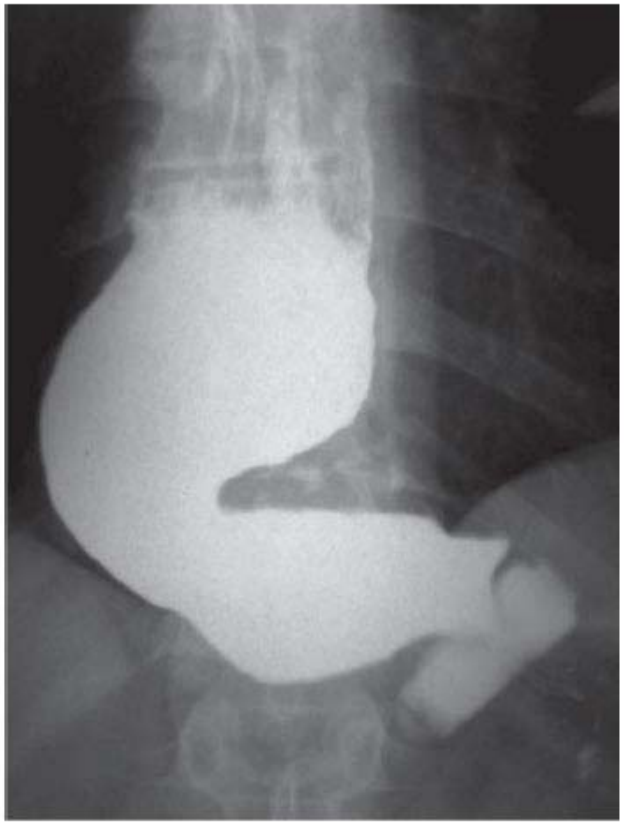


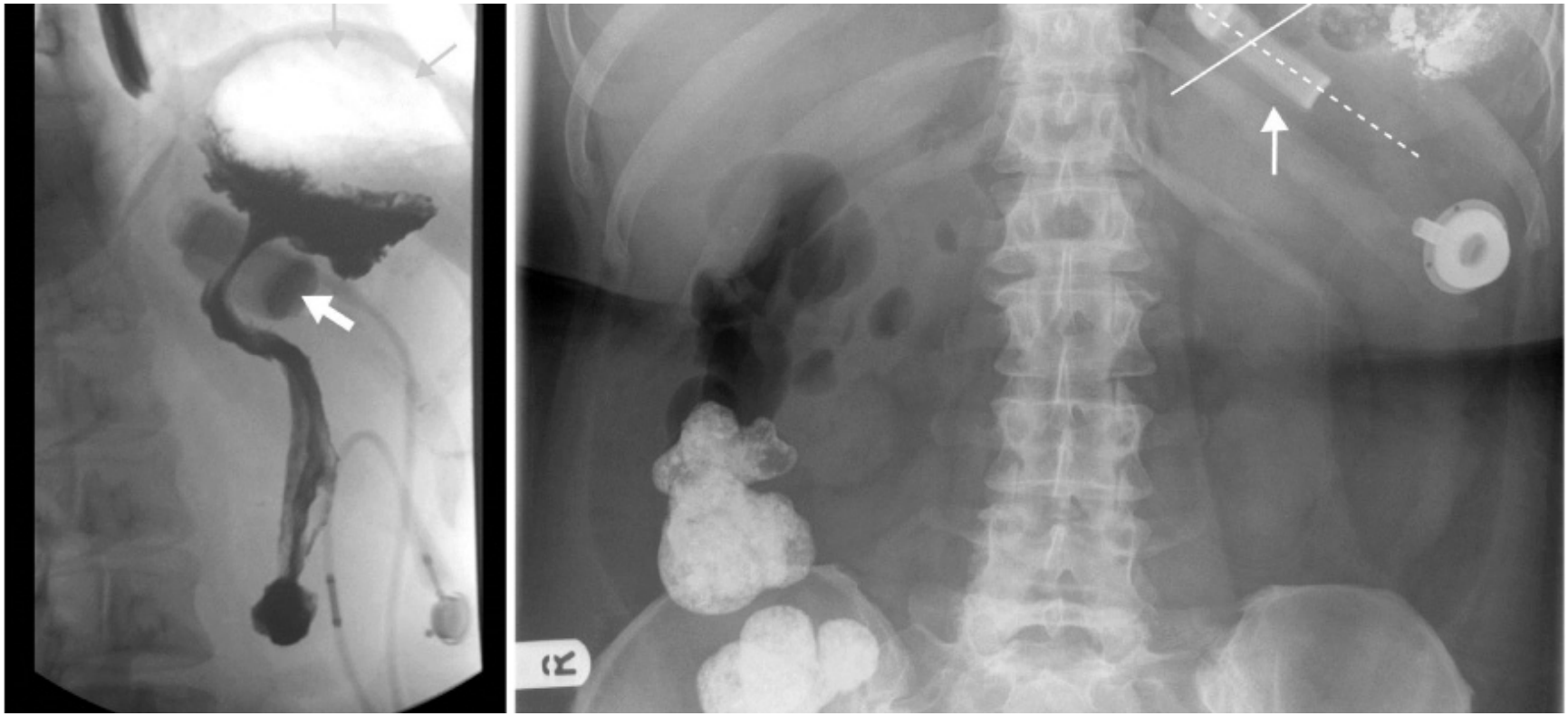
Table I
The profile of the gut hormones' changes after RYGB, BPD-DS, SG, AGB

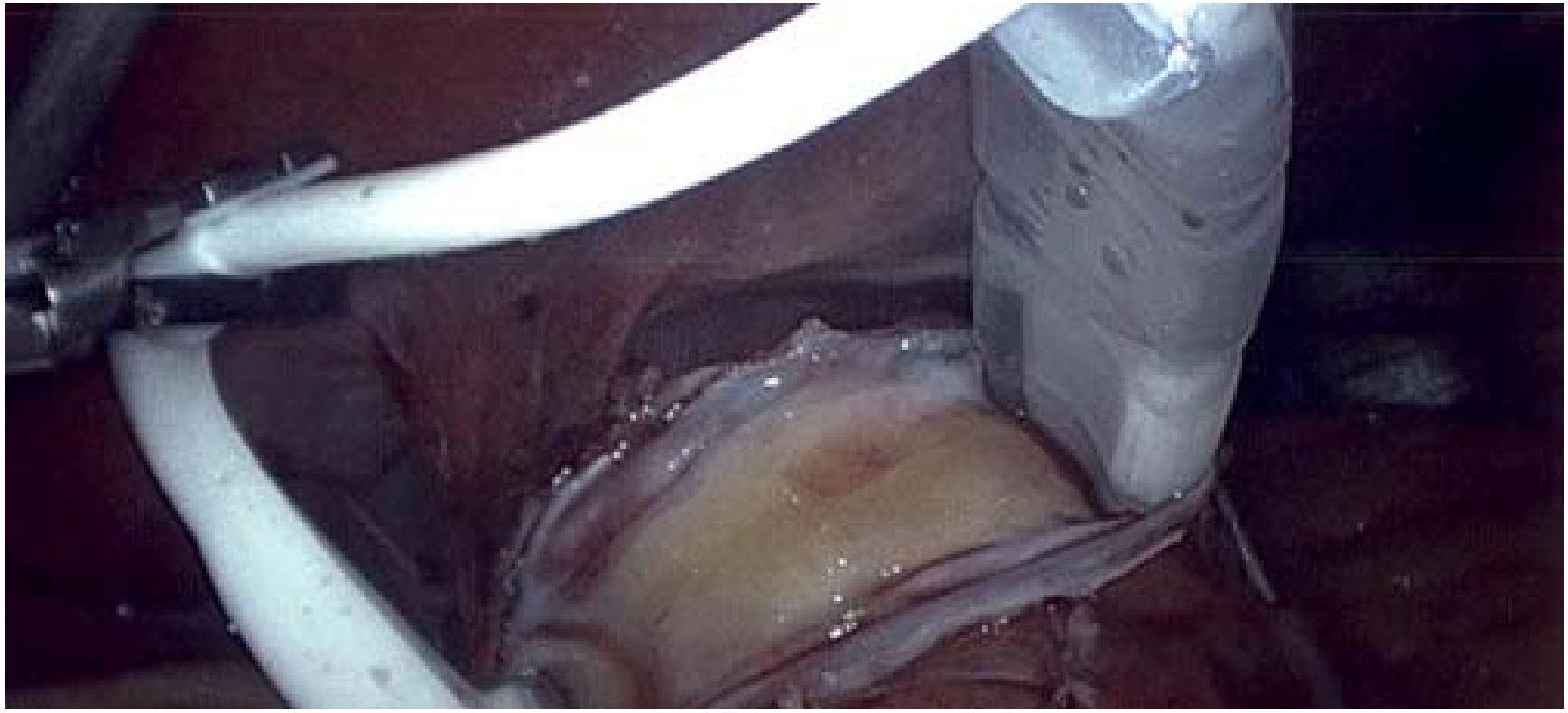
	RYGB	*BPD*	*SG*	*AGB*	*BPD-DS*
Fasting GLP-1	↔	↑	↔	↔	–
GLP-1 AUC	↑	↑	↑	↔	–
Fasting PYY	↔	↑	↑ or ↔ or ↓	↔	↑
PYY AUC	↑	–	↑	↔	↑
Fasting GIP	↔	↓	–	↔	–
GIP AUC	↔	↓	–	↔	–
Fasting ghrelin	↔ or ↓ or ↑	↔ or ↑	↓	↑ or ↔	↓
Ghrelin AUC	↔ or ↓	↑	↓	↔	–

↔: No significant change in the majority of studies; ↑: Significant increased in the majority of studies; ↓: Significant decreased in the majority of the studies; –: No studies for this parameter; GLP-1: Glucagon Like Peptide-1; PYY: Peptide YY; GIP: gastric inhibitory polypeptide/glucose – dependent insulinotropic polypeptide; RYGB: Roux- en-Y Gastric Bypass; BPD: Biliopancreatic Diversion; SG: Sleeve Gastrectomy; AGB: Adjustable Gastric Banding; BPD-DS: Biliopancreatic Diversion with Duodenal Switch; AUC: Area Under the Curve.

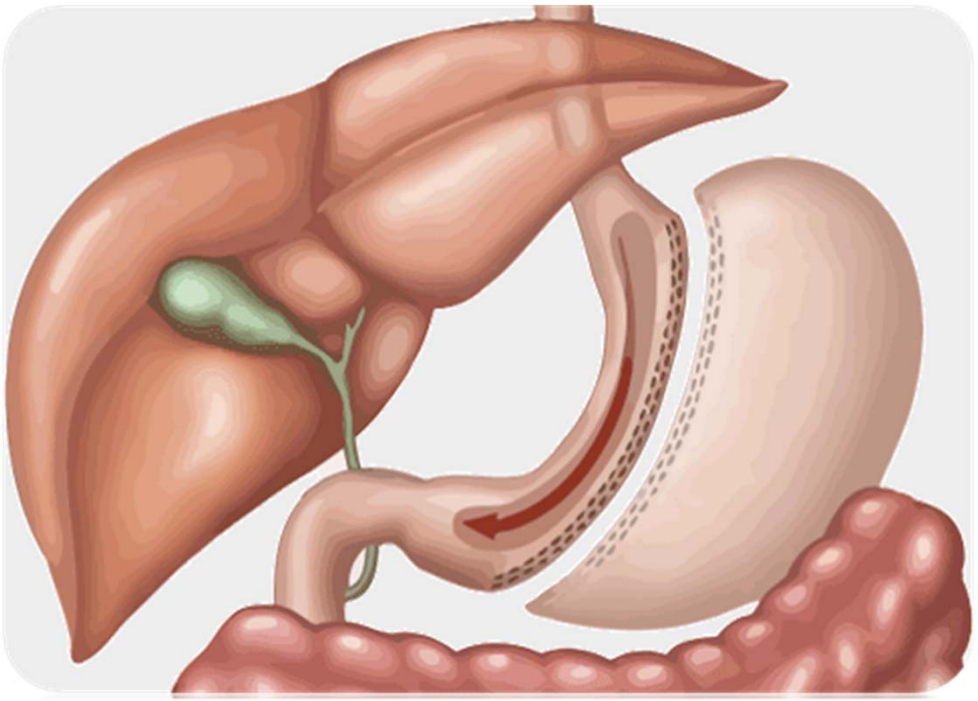
Papamargaritis, D, Miras, AD, LeRoux, CW. Influence of diabetes surgery on gut hormones. *Nutricion Hospitalaria*. 2013; 28(2):95-103.







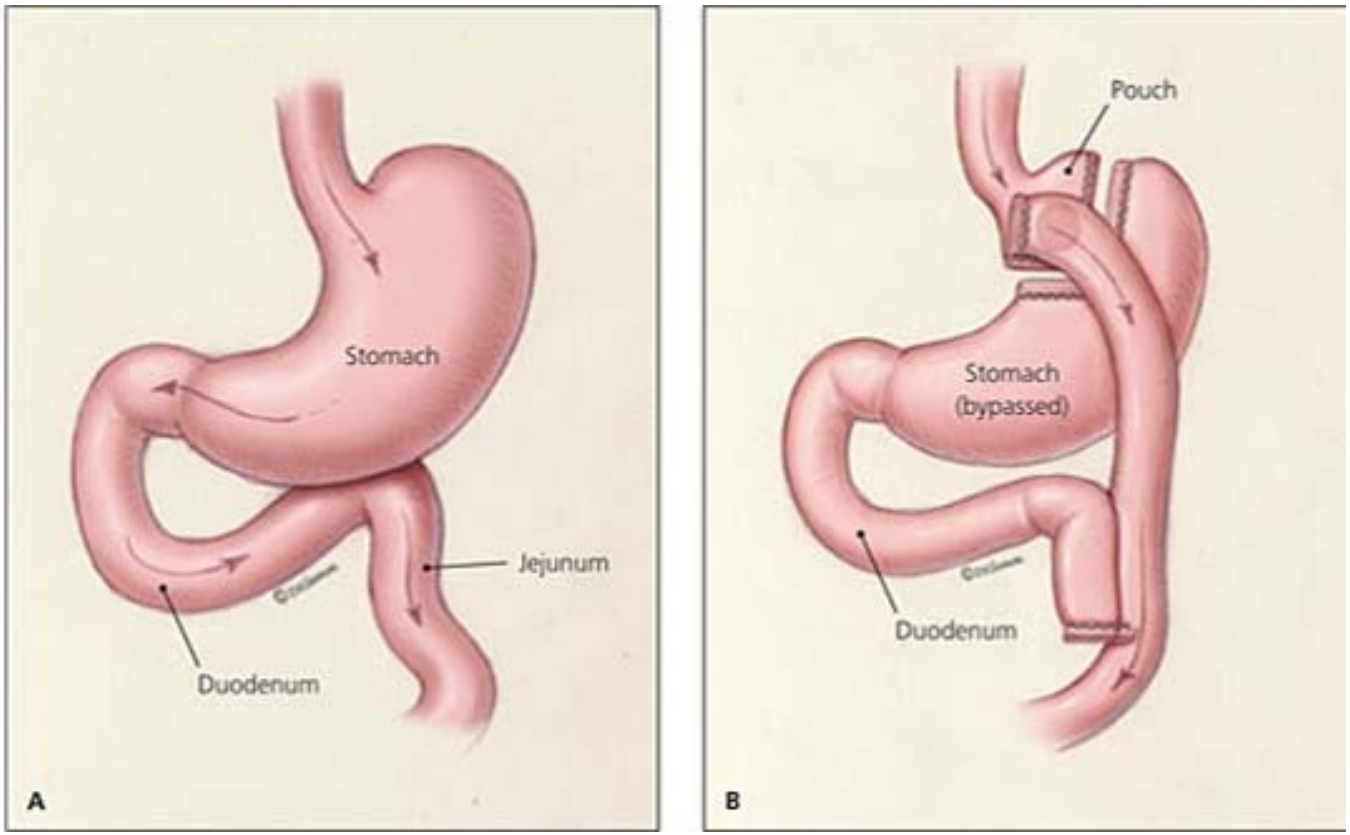
Vertical Sleeve Gastrectomy



Vertical Sleeve Gastrectomy Advantages

- Excess weight loss of >50% for 3-5+ year data.
- Requires no foreign objects and no bypass or re-routing of the food stream
- Causes favorable changes in gut hormones that suppress hunger, reduce appetite and improve satiety

Roux-en-y Gastric Bypass



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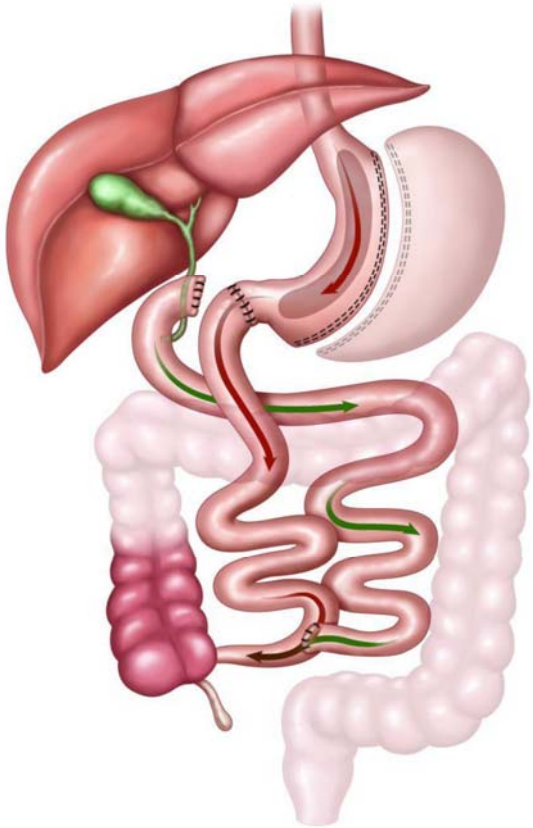
Roux-en-y Gastric Bypass Advantages

- Long-term excess weight loss 60-70%
- May lead to conditions that increase energy expenditure
- Produces favorable changes in gut hormones that reduce appetite and enhance satiety

Biliopancreatic Diversion w/Duodenal Switch



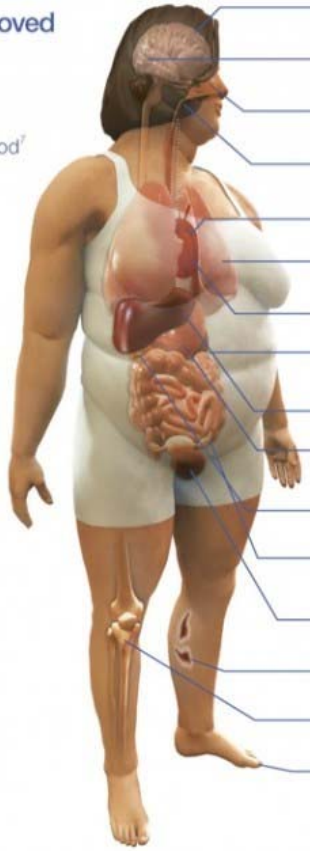
Biliopancreatic Diversion w/Duodenal Switch Advantages

- Greater weight loss than RYGB, LSG, or AGB (65 – 70% EWL)
- Reduces the absorption of fat by 70%
- Causes favorable changes in gut hormones to reduce appetite and improve satiety

Resolution of Comorbidities after Bariatric Surgery

Quality of Life Improved
in 95% of patients[7]

Relative Risk of Mortality Reduced
by 89% in a five-year period[7]



- Migraines
  57% resolved[2]
- Depression
  47% reduced[2]
- Pseudotumor cerebri
  84% resolved[8]
- Obstructive sleep apnea
  74% to 98% resolved[2,5]
- Hypercholesterolemia
  63% resolved[2]
- Asthma
  69% improved[2]
- Hypertension
  62% to 70% resolved[1,2]
- Nonalcoholic fatty liver disease
  37% resolution of steatosis[6]
- Metabolic syndrome
  80% resolved[6]
- GERD
  72% to 95% resolved[2,3]
- Type 2 diabetes
  82% to 98% resolved[2,5,9]
- Polycystic ovarian syndrome
  75% resolution of hirsutism[10]
  100% resolution of menstrual dysfunction
- Urinary stress incontinence
  44% resolved[2]
- Venous stasis disease
  95% resolution of venous stasis ulcers[4]
- Osteoarthritis/Degenerative joint disease
  41% resolved[2]
- Gout
  72% resolved[2]

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 AUGUST 23, 2007 VOL. 357 NO. 8

Effects of Bariatric Surgery on Mortality in Swedish Obese Subjects

Lars Sjöström, M.D., Ph.D., Kristina Narbro, Ph.D., C. David Sjöström, M.D., Ph.D., Kristjan Karason, M.D., Ph.D., Bo Larsson, M.D., Ph.D., Hans Wedel, Ph.D., Ted Lystig, Ph.D., Marianne Sullivan, Ph.D., Claude Bouchard, Ph.D., Björn Carlsson, M.D., Ph.D., Calle Bengtsson, M.D., Ph.D., Sven Dahlgren, M.D., Ph.D., Anders Gummesson, M.D., Peter Jacobson, M.D., Ph.D., Jan Karlsson, Ph.D., Anna-Karin Lindroos, Ph.D., Hans Lönroth, M.D., Ph.D., Ingmar Näslund, M.D., Ph.D., Torsten Olbers, M.D., Ph.D., Kaj Stenlöf, M.D., Ph.D., Jarl Torgerson, M.D., Ph.D., Göran Ågren, M.D., and Lena M.S. Carlsson, M.D., Ph.D., for the Swedish Obese Subjects Study

Swedish Obese Subjects Study

- Prospective trial with matched control patients
- Mean age: 48
- Mean BMI: 41
- 2010 bariatric surgery, 2037 conventional medical treatment

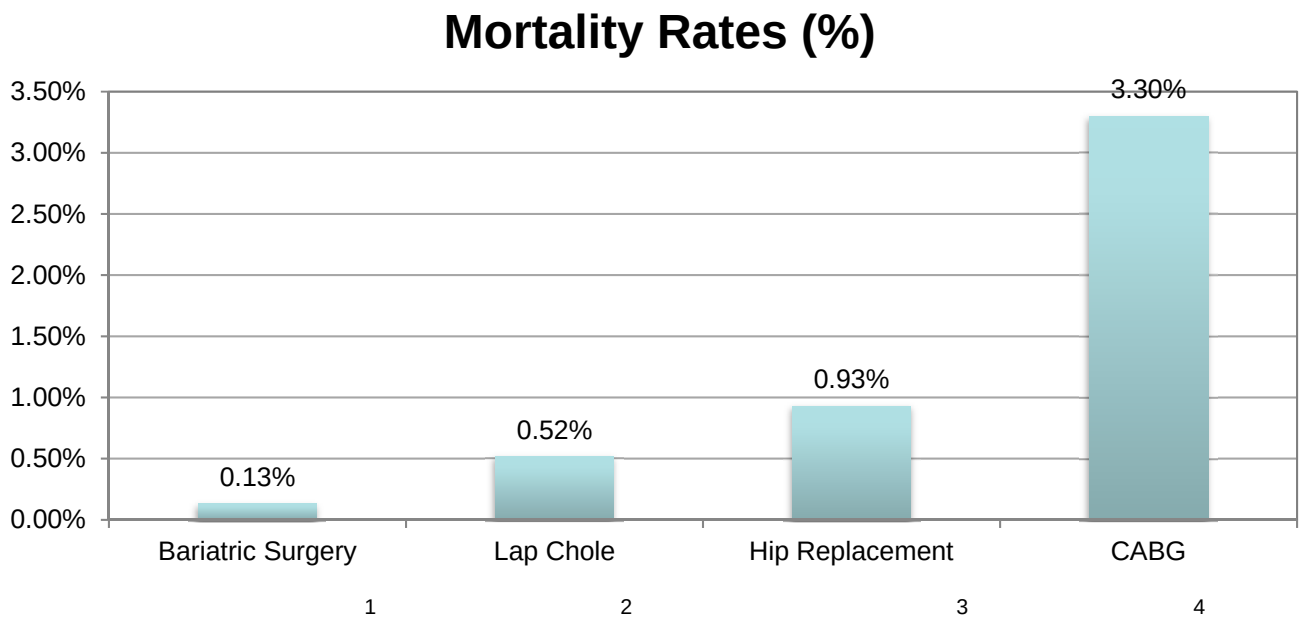
- Weight change:
  - After two years: control group +0.1%; surgery group -23.4%
  - After ten years: control group +1.6%; surgery group -16.1%

Swedish Obese Subjects Study

- 129 deaths in the control group at 10 years
- 101 deaths in the surgery group at 10 years

- Adjusted overall hazard ratio in surgery group 0.76

Bariatric Surgery Has a Low Incidence of Mortality



[1]Mortality rate when performed at a Bariatric Surgery Center of Excellence; Bariatric Surgery: DeMaria EJ, Pate V, Warthen M et al. Baseline data from American Society for Metabolic and Bariatric Surgery-designated Bariatric Surgery Centers of Excellence using the Bariatric Outcomes Longitudinal Database, Surgery for Obesity and Related Diseases. Article in Press.

[2]Dolan JP, Diggs BS, Sheppard BC et al. The National Mortality Burden and Significant Factors Associated with Open and Laparoscopic Cholecystectomy: 1997–2006. J Gastrointest Surg. 2009; 13:2292-2301

[3]Lie SA, Engesaeter LB, Havelin LI et al. Early postoperative mortality after 67,548 total hip replacements. Acta Orthopaedica 2002; 73(4):392-399

[4]Ricciardi R; Virnig BA, Ogilvie Jr. JW. Volume-Outcome Relationship for Coronary Artery Bypass Grafting in an Era of Increasing Volume. Arch Surg. 2008;143[4]:338-344

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The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Bariatric Surgery vs. Intensive Medical Therapy in Obese Patients with Diabetes

Philip R. Schauer, M.D., Sangeeta R. Kashyap, M.D., Kathy Wolski, M.P.H., Stacy A. Brethauer, M.D., John P. Kirwan, Ph.D., Claire E. Pothier, M.P.H., Susan Thomas, R.N., Beth Abood, R.N., Steven E. Nissen, M.D., and Deepak L. Bhatt, M.D., M.P.H.

Schauer et al

- RCT 150 patients (Med Only vs RNY or Sleeve)
- BMI 27-43 and HgbA1c >7
- ALL underwent intensive medical therapy
- Primary study endpoint HgbA1c <6
- Secondary endpoints: CRP, fasting glucose, insulin, lipids, adverse events, change in medications
- 93% Follow up

Table 2. Primary and Secondary End Points at 12 Months.*

End Point	Medical Therapy (N=41)	Gastric Bypass (N=50)	Sleeve Gastrectomy (N=49)	P Value		
				Gastric Bypass vs. Medical Therapy	Sleeve Gastrectomy vs. Medical Therapy	Gastric Bypass vs. Sleeve Gastrectomy
Glycated hemoglobin						
≤6% — no. (%)	5 (12)	21 (42)	18 (37)	0.002	0.008	0.59
≤6% with no diabetes medications — no. (%)	0	21 (42)	13 (27)	<0.001	<0.001	0.10
Baseline — %	8.9±1.4	9.3±1.4	9.5±1.7			
Month 12 — %	7.5±1.8	6.4±0.9	6.6±1.0	<0.001	0.003	0.23
Change from baseline — percentage points	−1.4±1.5	−2.9±1.6	−2.9±1.8	<0.001	<0.001	0.85
Body weight — kg						
Baseline	104.4±14.5	106.7±14.8	100.6±16.5			
Month 12	99.0±16.4	77.3±13.0	75.5±12.9	<0.001	<0.001	0.50
Change from baseline	−5.4±8.0	−29.4±8.9	−25.1±8.5	<0.001	<0.001	0.02
High-density lipoprotein cholesterol						
Percent change from baseline	11.3±25.7	28.5±22.7	28.4±21.9	0.001	0.001	0.98
Triglycerides						
Median percent change from baseline (interquartile range)	−14 (−40 to 3)	−44 (−65 to −16)	−42 (−56 to 0)	0.002	0.08	0.17
High-sensitivity C-reactive protein						
Median percent change from baseline (interquartile range)	−33.2 (−71 to 0)	−84 (−91 to −59)	−80 (−90 to −63)	<0.001	<0.001	0.59

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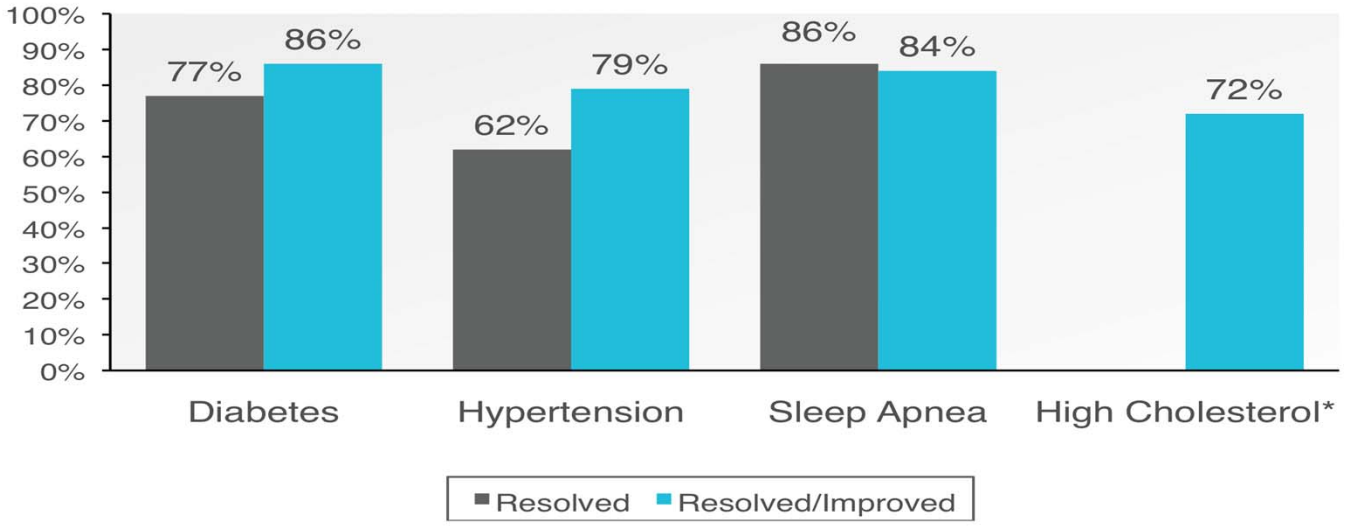
IDF Position Regarding Surgery to Treat DMII in Obese Patients

- "Bariatric intervention is a health and cost-effective therapy for type 2 diabetes and obesity with an acceptable safety profile"
- "Bariatric surgery for severely obese people with type 2 diabetes should be considered much earlier in management rather than held back as a last resort"
- "It should be incorporated into type 2 diabetes treatment protocols"
- "Should be considered in patients with BMI > 30 when diabetes cannot be controlled with optimal medical regimen"

Surgical Therapy for Morbid Obesity

Resolution* of Co-morbidities Following Gastric Bypass Surgery



Buchwald H, Avidor Y, Braunwald E, et al. Bariatric Surgery–A Systematic Review of the Literature and Meta-analysis. JAMA 2004 Oct 13;292(14).

*Resolution was observed in the confines of the study, and EES has no independent data to suggest permanent resolution.

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Reduction in Healthcare Utilization

Five-Year Healthcare Utilization

	BARIATRIC MEAN (SD)	CONTROLS MEAN (SD)	P-VALUE
Hospitalizations	2.75 (3.44)	3.17 (3.22)	0.001
Hospital Days	21.05 (38.97)	36.59 (25.41)	0.001
Physician Visits	9.62 (15.8)	17.00 (21.74)	0.001

Christou NV, Sampalis JS, Liberman M, et al. Surgery Decreases Long-Term Mortality, Morbidity, and Health Care Use in Morbidly Obese Patients. Annals of Surgery 2004;240(3):416-424.

Possible Surgical Complications

Dehydration

DVT/PE

Leak

Bleeding

Marginal ulcer

Stricture

Dumping syndrome

Protein/mineral deficiencies

Possible Surgical Complications

Dehydration

DVT/PE

Leak

Bleeding

Marginal ulcer

Stricture

Dumping syndrome

Protein/mineral deficiencies

Possible Surgical Complications

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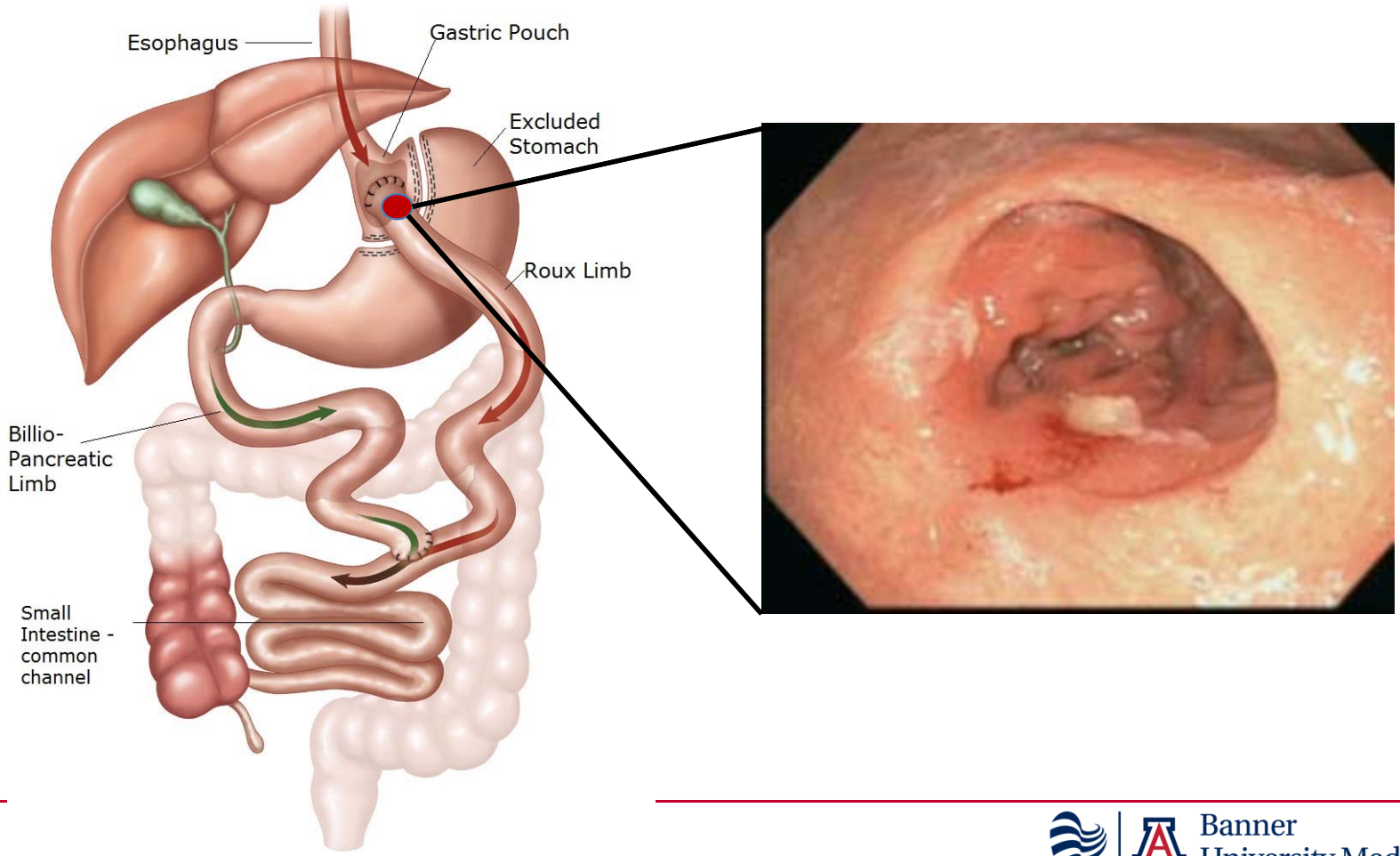
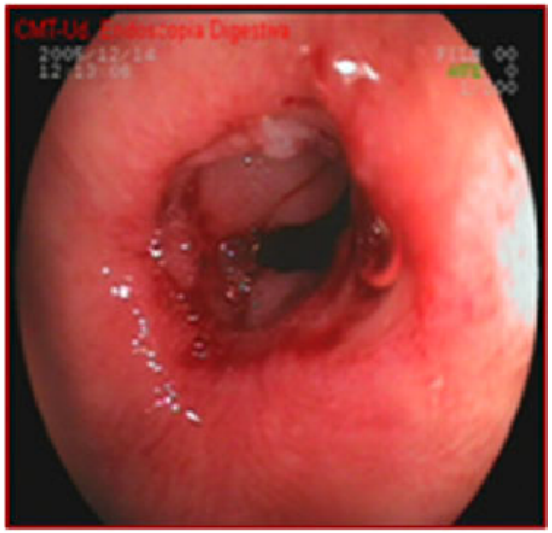

Esophagus
Gastric Pouch
Excluded Stomach
Roux Limb
Billio-Pancreatic Limb
Small Intestine - common channel

Possible Surgical Complications

Dehydration
DVT/PE
Leak
Bleeding
Marginal ulcer
Stricture
Dumping syndrome
Protein/mineral deficiencies

Anastomotic stricture

Balloon dilation



Stricture following dilation

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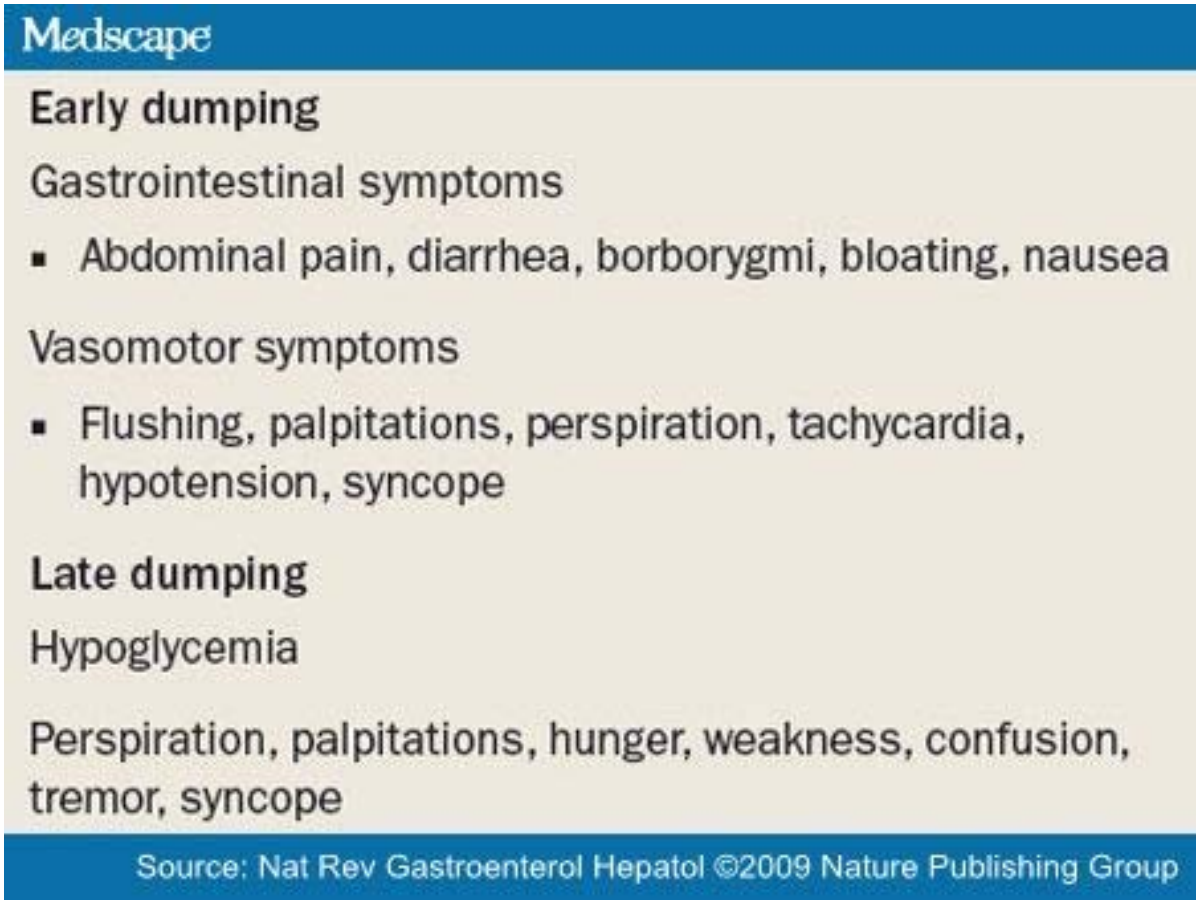
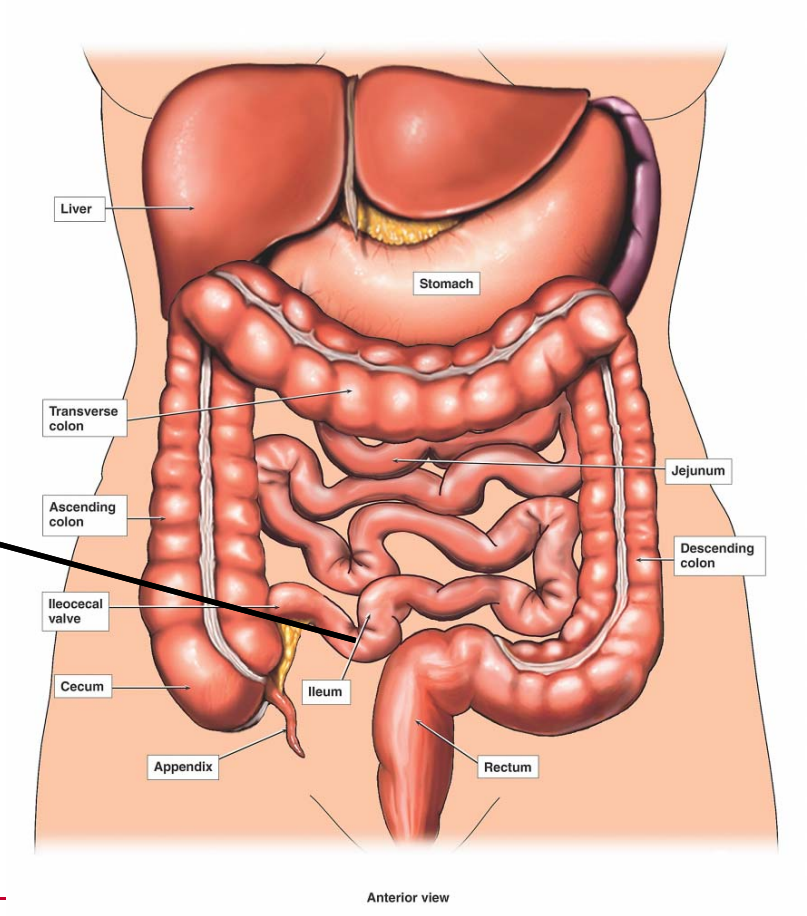
Possible Surgical Complications

Dehydration
DVT/PE
Leak
Bleeding
Marginal ulcer
Stricture
Dumping syndrome
Protein/mineral deficiencies


Medscape
Early dumping
Gastrointestinal symptoms
Abdominal pain, diarrhea, borborygmi, bloating, nausea
Vasomotor symptoms
Flushing, palpitations, perspiration, tachycardia, hypotension, syncope
Late dumping
Hypoglycemia
Perspiration, palpitations, hunger, weakness, confusion, tremor, syncope
Source: Nat Rev Gastroenterol Hepatol ©2009 Nature Publishing Group

GLP-1



Anterior view

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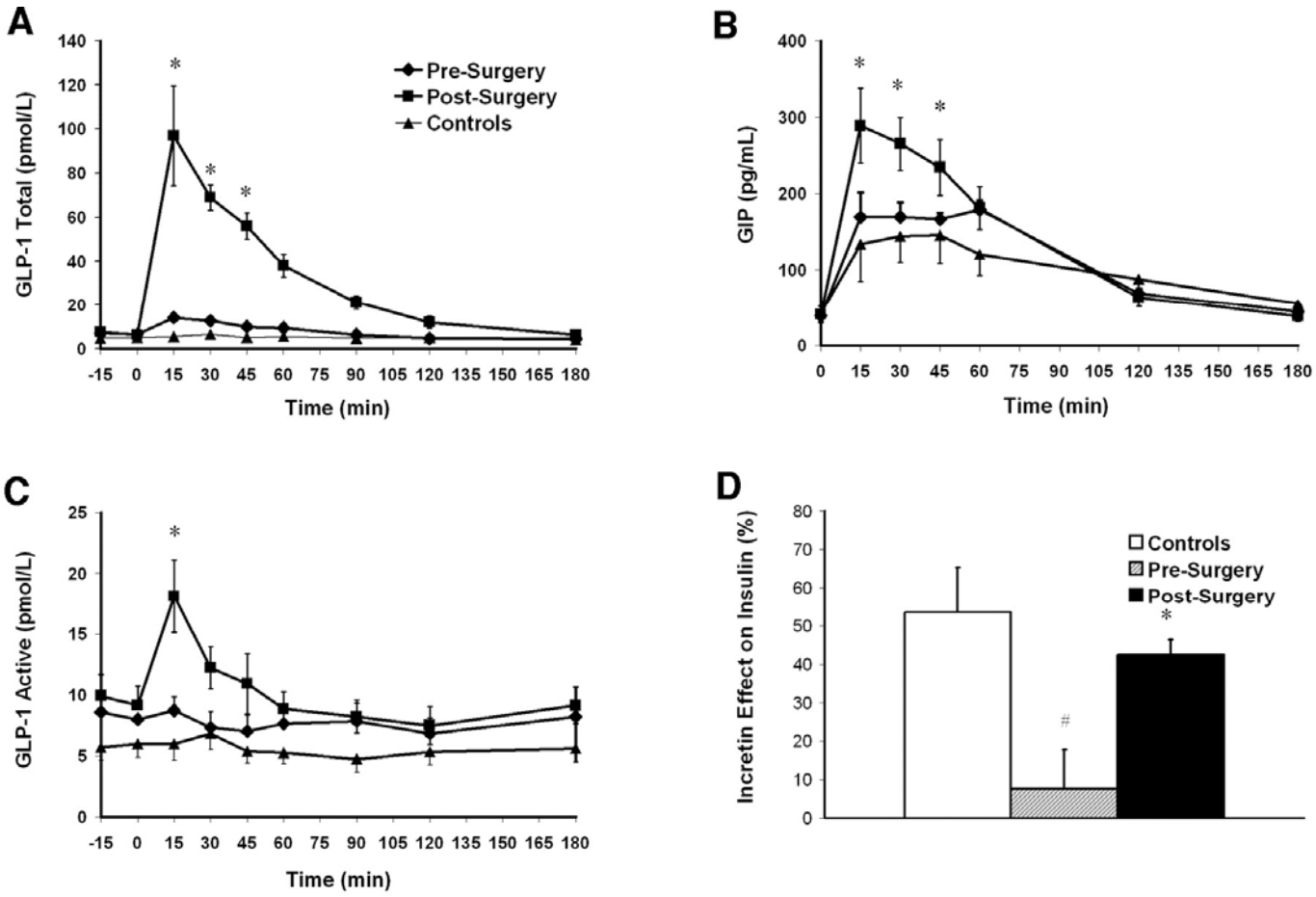

Source: Laferrere, B, Heshka, S, Wang, K, et al. Incretin Levels and Effect Are Markedly Enhanced 1 Month After Roux-en-y Gastric Bypass Surgery in Obese Patients with Type 2 Diabetes. *Diabetes Care 2007*; 30 (7): 1709-1716.

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Possible Surgical Complications

Dehydration
DVT/PE
Leak
Bleeding
Marginal ulcer
Stricture
Dumping syndrome
Protein/mineral deficiencies

BUMC-P Bariatric
VITAMIN PROTOCOL

*Please bring your supplements to **all** post-operative visits to be verified by a Provider*

Bariatric Multivitamin

*For dosing instructions, please read the label
*Must contain at least **20 nutrients** and at least **45mg of iron** *per day*
*No gummies or patches

- ProCare Health Bariatric Complete Multivitamin chewable/capsule (with 45mg iron) (procarenow.com)
- Bariatric Advantage Ultra Multi with Iron or Advanced Multi EA (bariatricadvantage.com)
- Celebrate Multi-Complete (with 45mg iron) chewable/capsule (celebratevitamins.com)

Calcium Citrate (*may or may not contain Vitamin D3*)

***Total of 1,500mg per day**
*Your body can only absorb about **500mg at a time**, so spread it out in 3 doses
*Please read the label carefully to determine correct dose. Must be in the form **"citrate"**
*Do not take at the same time as multivitamin because it will complete with iron for absorption

- Bariatric Advantage Calcium Citrate Chewables or Chewy Bites (bariatricadvantage.com)
- GNC Calcium Citrate Caplets -- *with* or *without* vitamin D3 (GNC)
- Bariatric Fusion Calcium Soft Chews (bariatricfusion.com)
- Ultra Plan Ultimate Calcium Citrate Wafers (Hi-Health)
- Bluebonnet Liquid Calcium Magnesium Citrate Plus Vitamin D3 (Sprouts, Amazon)

Vitamin D3

***A *total* of 3,000 International Units (IU) per day**
*** Note: your multivitamin and/or calcium citrate may have D3 in them, so you must account for this when calculating the amount of additional D3 you need (if any) to total 3,000 IU ***
Example: 3,000 IU needed each day
- 1,000 IU total in your multivitamin
- 1,000 IU total in your calcium citrate
1,000 IU vitamin D3 needed additionally each day

- Bluebonnet Earthsweet Vitamin D3 Chewables (Sprouts, Amazon)
- Natrol Vitamin D3 Fast Dissolve Tablets (CVS, Walgreens, Wal-Mart)
- Webber Naturals Vitamin D3 Liquid Drops (Sprouts, Costco, Wal-Mart)
- CVS Vitamin D3 Softgels (CVS)

*If you decide to take a non-bariatric specific multivitamin, it must contain at least 20 different nutrients and at least 18mg of iron per serving (you will need to take *2 servings* per day)

*If you have had a ***duodenal switch*** procedure, please speak with your bariatric dietitian to tailor your vitamin regimen due to increased nutritional needs

**PROMO CODE For online Bariatric Advantage orders: banner (10% discount)

Last Revised 3/7/2018

Post-Bariatric Surgery Vitamin Screening

Vitamins A, B1, B12, D
Iron panel
Zinc, Selenium

Vitamin K, E (DS)
Copper (DS)

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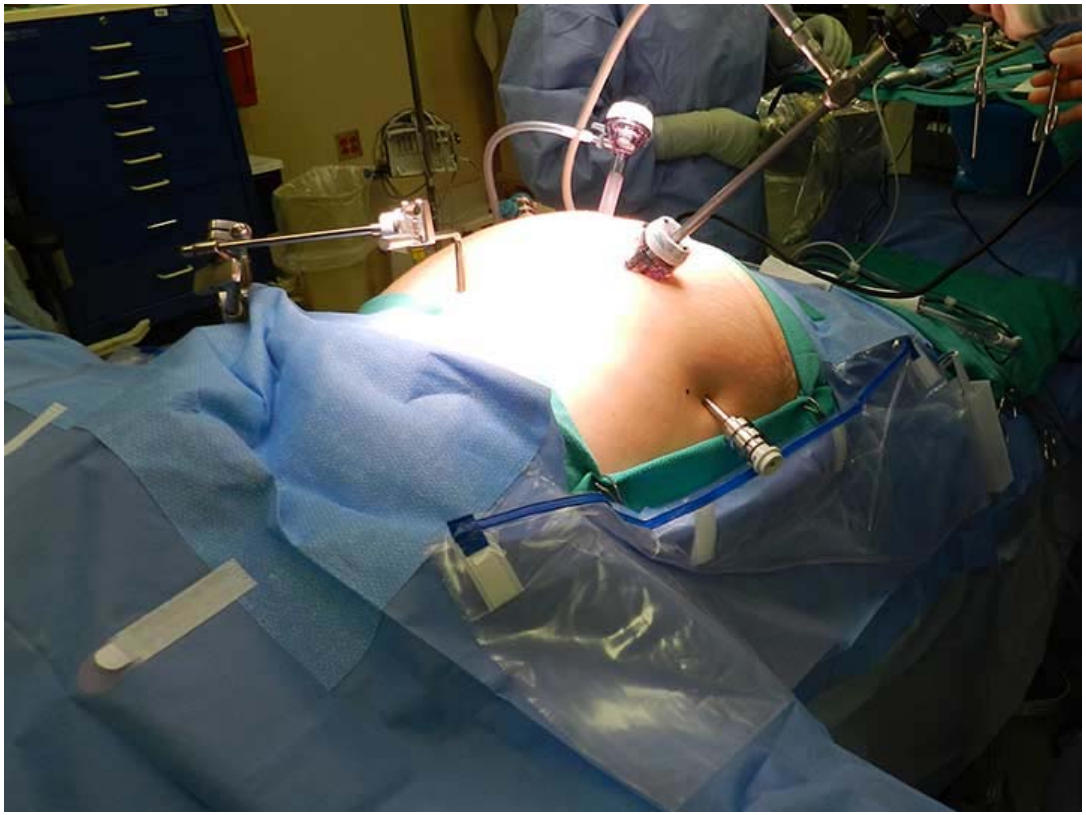
Who qualifies for Bariatric Surgery?



Qualifying for surgery

NIH Guidelines for eligibility:

1. Failed dietary therapy

2. BMI > 40 kg/m²

   OR

   BMI > 35 kg/m² with associated comorbidity (DM, HTN, OSA)

Qualifying for surgery

Other eligibility requirements

3. Optimization of other co-morbid conditions
4. Non-smoker
5. Psychological disorder undergoing treatment
6. Drug/alcohol addiction in remission
7. Health maintenance/screening

Bariatric Surgery Program BUMCP

Seminar

Initial Consultation

Insurance Requirements

Surgeon Requirements

Banner University Medicine

Bariatric Surgery Program BUMCP

Seminar

2nd, 4th Tuesdays, 6pm
Sandstone Conference Center

Initial Consultation

Insurance Requirements

Surgeon Requirements

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Insurance Requirements

Medically supervised weight loss (0-6 months)

Psychological evaluation

Letter of medical necessity (Bariatric surgeon)

Food/activity log

Support group participation

Surgeon Requirements

Dietary counseling
UGI vs EGD
Labs, CXR, EKG

Sleep Study with CPAP/BiPAP titration
Cardiac evaluation/optimization
Venous duplex
Pulmonary evaluation/optimization
Physical Therapy evaluation
Health maintenance screening
Out of town patients to stay in town until post-op class.

Peri-operative course

Pre-operative:

Pre-operative class and pre-operative visit with surgeon

Post-operative:

Post-operative class, 30 day appointment, 60 day class, 6 month and 1 year visits.

Peri-operative Course

Hospital Stay 1 night for most patients

Fluid - 64oz/day

Ambulation

IS for home

Vitamin supplementation

Protein goal - 60-80 Grams/day

Bariatric soft diet starts in the first week post-op

Lifelong follow up

- Once yearly visits
- Vitamin labs
- Registered dietitian visits
- Monthly support groups
- Social media/Community

How to get your patient started

- Referral required
- Fax to 602-521-3046
- We will call to schedule Seminar and send out new patient packet
- Seminars (2nd and 4th Tuesdays, 6pm Sandstone Auditorium)
- First consultation within 2 weeks of seminar

How to get your patient started

Center for Obesity and Bariatric Surgery

1441 N. 12th Street
Phoenix, AZ 85006
Phone: 602-521-3050
Fax: 602-521-3046

Practice Supervisor: Jennifer Molina 602-521-5950
Jennifer.Molina@bannerhealth.com

Christine Lovato 480-202-9449
Christine.Lovato2@bannerhealth.com

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Objectives

1. Update on the pathophysiology of obesity and the role of surgery.
2. Review outcomes of Bariatric Surgery including improvement of co-morbid disease and the PCP role on the interdisciplinary team.
3. Get to know the Bariatric Surgery program at BUMCP – how can I get my patients to surgery?

QUESTIONS?

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