GLP-1 Agonists: The Ecstasy. The Agony. Nutritional Support Protocols for your Patients that **Get Results**.

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August 14, 2026

Speaker Disclosure

Simon Agger D.C. is a licensed Chiropractic Physician, and presents Clinical innovations that have not been endorsed by the FDA to licensed healthcare practitioners. He does not claim to cure any known diseases. Cases discussed are his patient experiences over the last 28 years in practice. None of this lecture is a substitute for proper medical advice. Please consult with your personal licensed physician outside of this event - even if it turns out to be me!!

Speaker Disclosure, cont.

- Full time private practice, 1998-present: Portland, OR
- Adjunct faculty, University of Western States ‘Human Nutrition & Function

  Medicine’ Masters of Science Program: 2014 – 2019.
- Affiliate faculty, UWS Chiropractic Program: 2005 - present.
- Faculty, Integrative Medical Nutrition, Rocky Vista University Osteopathic Medicine program, CO & UT Campuses 2023- 2025
- Speaker, Standard Process & MediHerb US/AUS: 2015-present
- Consultant SP & MediHerb US/AUS, 2019-present
- Researcher, Nutritional Innovation Center, Kannapolis, NC, 2021 – present
- Attending Physician, Oregon Ballet Theatre: 2005 - present

Objectives

- Dr Agger dives into what you need to know about incretins as a clinician.
- Your role with co-managing these patients on & off medications,
- offering natural alternatives
- understanding the reality of patients utilizing GLP-1 & GIP agonists. Opportunities & profound responsibilities for clinicians.
- current research,
- case studies from his clinic, and of course,
- nutritional diet/lifestyle, botanical & manual medicine essentials to support patients on & off these drugs.

Goals

- After attending this session, attendees will
- (1) have a solid grounding on what incretins are;
- (2) how to modulate them using natural medicine;
- (3) understanding what to expect from patients on GLP-1 & GIP agonist drugs;
- (4) understand how to educate & manage them successfully.

What the heck are Incretins?

- **Peptide hormones derived from your gut**, released in response to incoming nutrients
- IN – CRET – TIN
- Intestine – Secretion – Insulin
- **Stimulate insulin secretion**
- Glucagon-like peptide 1 (GLP-1)
- Glucose-dependent insulinotropic polypeptide (aka Gastric inhibitory polypeptide (GIP)

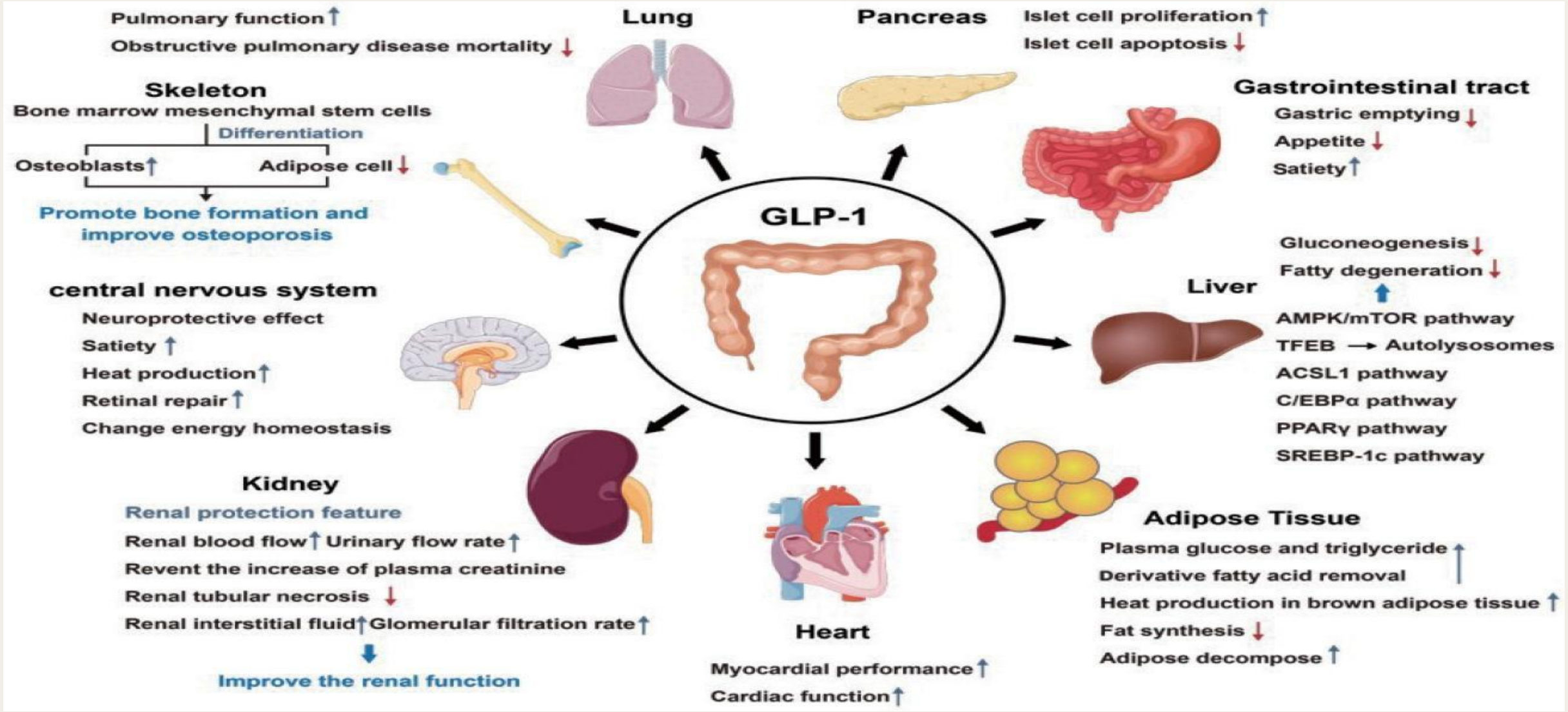
What is their Function?

- GLP-1 is a natural hormone **made in your gut** after you eat that helps keep your blood sugar, appetite, and weight in balance.
- **It tells your pancreas to release insulin when sugar is in your blood**, stops your liver from making too much sugar, **slows how quickly food leaves your stomach**, and **signals your brain that you're full.**
- When this system gets disrupted—by gut issues, inflammation, or insulin resistance—it can cause problems with hunger control, fat storage, and blood sugar regulation.

GLP-1 graphic

Glucagon-Like Peptide-1: New Regulator in Lipid Metabolism

Diabetes & Metabolism Journal 2024;48(3):354-372. DOI: https://doi.org/10.4093/dmj.2023.0277



Glucagon Like Peptides (GLP-1) agonists

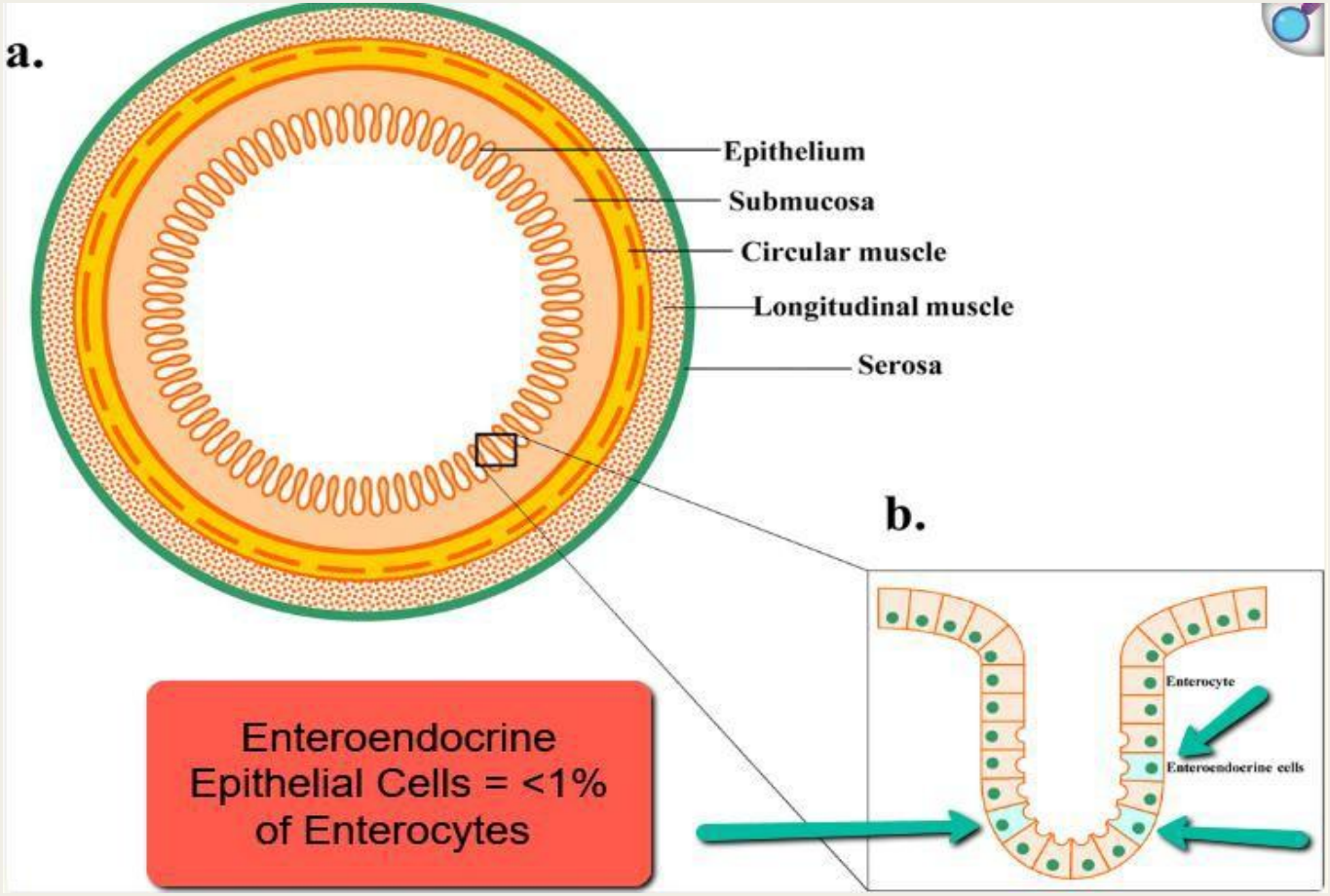
How does GLP-1 work?

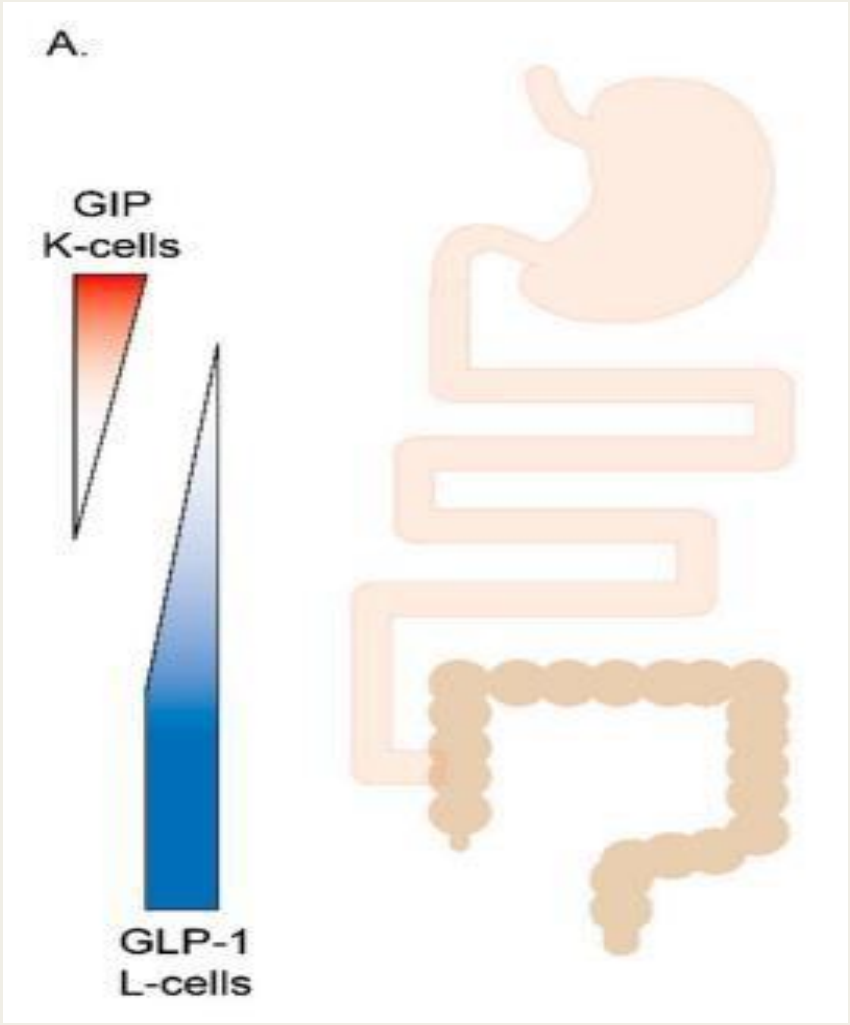
- GLP-1 works via the hypothalamus to decrease appetite when satiety is stimulated.
- Slows the gastric emptying of the stomach
- Stimulates the pancreas to promote insulin secretion & suppress Glucagon.
- GLP-1 is released by the small intestine in response to food. It is one of 2 incretins (GLP-1, GIP).

Common drugs:

- Ozempic (semiglutide), GLP-1 agonist
- Tirzepatide (mounjaro) GLP-1 and GIP agonist.
- Both slow down gastric emptying & can cause worse dysbiosis. (Use a probiotic, support gastric secretions & bile production). See next slide.

Specialised Cells in gut





What you need to know

- GLP-1 receptor agonists **mimic or amplify the body's natural GLP-1 signaling.**
- They suppress appetite, slow gastric emptying (how quickly food leaves your stomach)
- They **enhance insulin release when sugar is present**
- They **reduce glucagon** (a hormone that raises blood sugar),
- They produce significant weight loss and better blood sugar control in clinical trials.

Big Picture

- GLP-1 receptor agonist therapy is therefore *not* a replacement for system repair — **it is a temporary intervention designed to give the body metabolic breathing room while deeper restoration unfolds**.

- If you do not do any functional medicine support then refer them to a clinician that does.

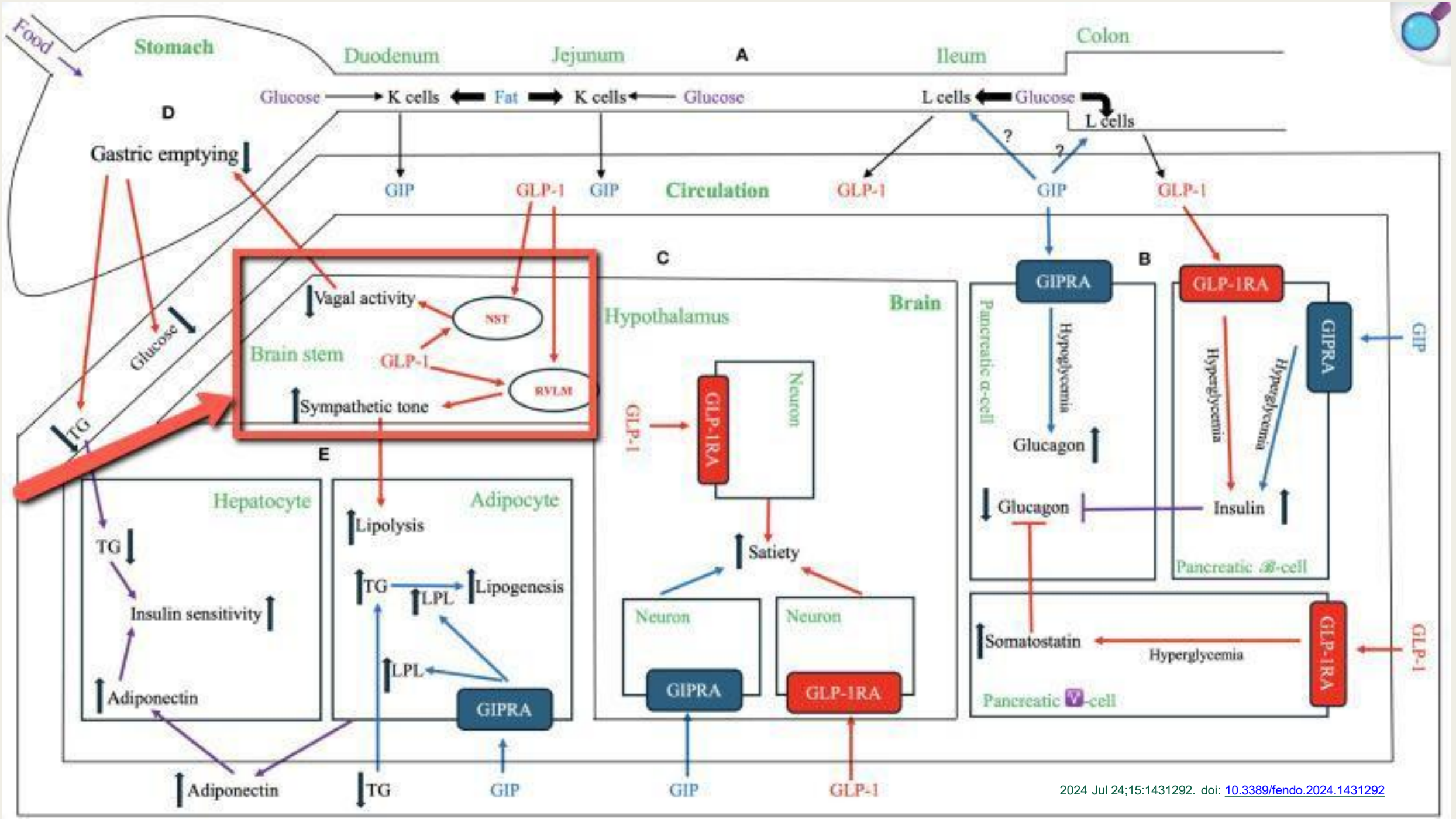
The Promise of GLP-1/GIP Agonist drugs

- GLP-1
- GLP/GIP
- These provide ‘the magic bullet’, but do not address the underlying cause of the IR/DM or obesity – that is, **they treat the symptoms**.
- **We have to look at IR/DM whether they are on the drug or not!**
- Hormones
- Metabolic dysfunction/inflammation
- Circadian patterns
- Detoxification

Effects on ANS

- Increase Sympathetic Tone
- Decrease Parasympathetic tone

- = Classic chronic Pain/inflammatory syndromes that we see.

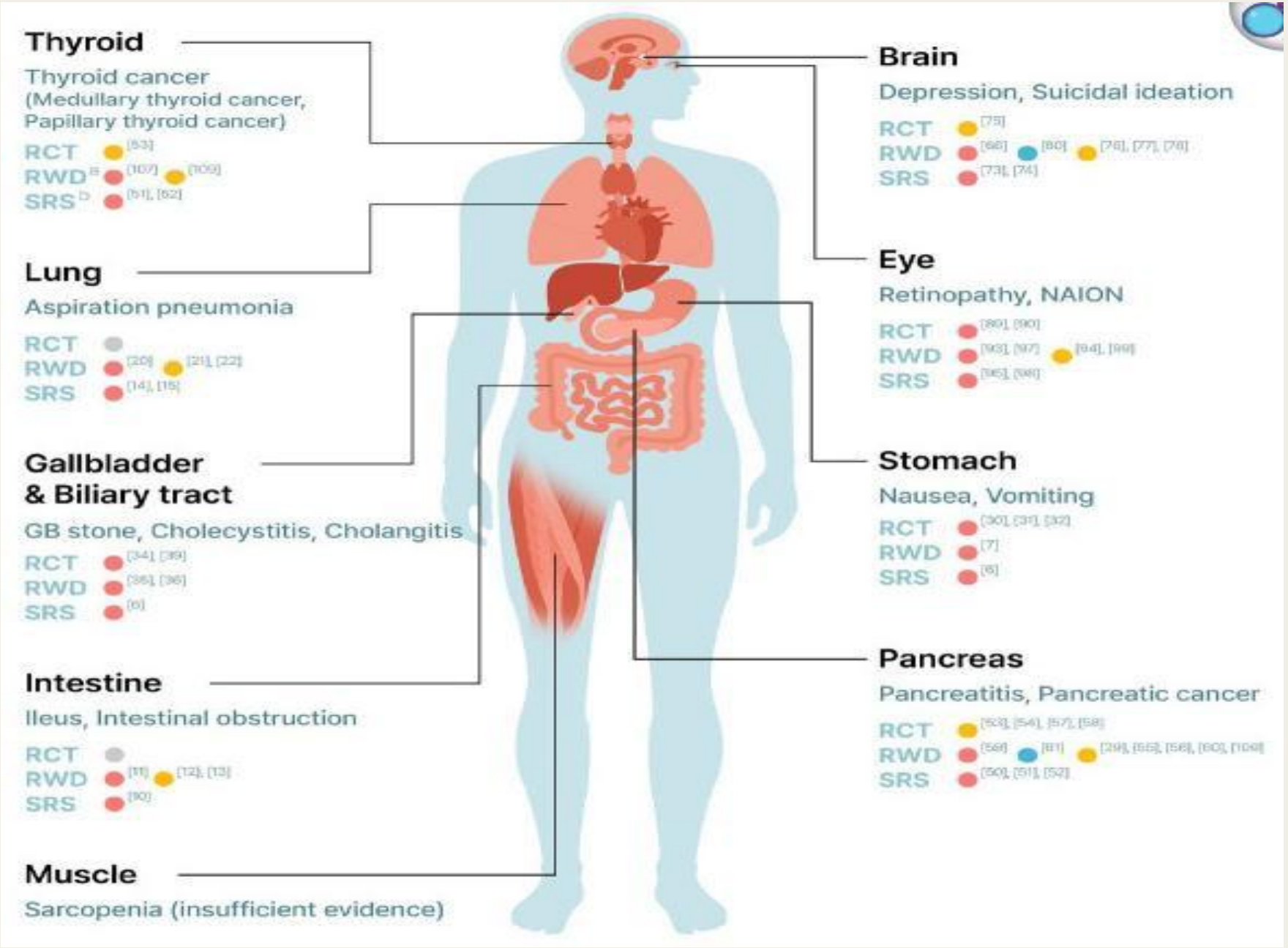


2024 Jul 24;15:1431292. doi: 10.3389/fendo.2024.1431292

Exploring side effects of GLP-1 Agonists 2025

- GLP-1 RAs demonstrate metabolic efficacy but present diverse adverse events.
- GI effects are common; obstruction or perioperative aspiration are major concerns.
- GLP-1 RAs link to gallbladder/biliary diseases without increasing pancreatitis risk.
- **Close monitoring is essential for depression and suicidal ideation during therapy.**
- **Retinopathy, NAION, cancer, and sarcopenia warrant careful consideration,** even though some are ‘rare’ side-effects.
- doi: 10.4093/dmj.2025.0242

Current Key Concerns



Sarcopenia/muscle lean mass loss

- **Approximately 40% of the weight reduction associated with GLP-1 RAs is derived from a lean body mass**, which raises significant concerns regarding its potential negative impact on skeletal muscle functionality [110].
- Current evidence, including magnetic resonance imaging studies, suggests that **skeletal muscle alterations** resulting from GLP-1 RA-induced weight loss, such as enhanced insulin sensitivity and reduced muscle fat infiltration, **are adaptive**, thereby improving muscle quality despite concurrent reductions in lean mass [112].
- **Future research** on GLP-1-based medicines for weight reduction must prioritize the precise evaluation of muscle mass, composition, and functionality **to clarify their impact on muscular health**.

Key Patient Selection –

- Their baseline health often determines how ‘well’ they do

- This is especially true of their ‘gut health’. Many patients selected for GLP-1 therapy will have significant dysbiosis, often bacterial/fungal/yeast overgrowth.

- Many patients have ***significant co-morbidities***.

- Many patients (asking to be) put on this have significant body dysmorphia

Before Your Patient starts GLP-1/GIP Agonists……

Bloodwork (a) Glycemic/metabolic load

- HOMA-IR
- **Fasting Insulin**
- **Fasting Glucose**
- **HbA1c**
- C-peptide
- **Uric Acid** (< 5.5 - 6.5)
- **CMP** (incl. Ca, Na, BUN, creatinine, GFR, Phosphoros, K)
- **Vit D**
- **RBC Mg**
- **Omega-3 index**

Bloodwork (b) Inflammatory markers

- **CBC** w/differrential
- **Lipid Panel w/oxidised LDL**/Lp(a)
- Homocysteine (<6); (>15 = Hhcy)
- Neutrophil/Lymphocyte ratio <2.24
- **HsCRP** (<1-3.0)
- **Ferritin** (80-200)
- Fibrinogen (<350)
- Anti-CPP/Raf; ANA
- **TSH; TPO; Tga** (Auto-antibodies)

UA

- Protein (or trace)
- Glucose
- Ketones
- pH (want to be acidic)
- Bacteria/WBC esterase
- UCA ratio (<25)
- Ntx/creatinine ratio (telopeptide loss)

GUT Check

- Palpation – Ridler’s points
- Intestinal palpation (including Chapman reflexes)
- Murphy’s point
- Palpate for any gas & bloating (along with history).
- PCR Bacterial/Myco biome test (SIBO/SIFO)
- Any history of IBS (Constipation or loose/urgent stools?)

Toxicity estimation

- Past work history
- Life history exposure
- Toxicity questionnaires
- Choice chemical & metal checks (labwork, resonance testing (AK), UA, hair tissue mineral analysis).
- Digestion & elimination history (constipated, rabbit droppings?)
- Bowel characteristics
- Bloating characteristics, belching
- History of biliary colic

Sleep/stress patterns

- Get down & stay down?
- If wake up when? How often? #of days a week?
- Do you take any foods/supplements to help sleep?
- Perimenopause?
- Drugs that affect sleep (i.e. SSRI’s, amphetamines)
- Take drugs to help sleep?
- Use Apnea machine; How long?

Estimate ‘ballpark mitochondrial health’

- Regular exercise?
- Balanced diet?
- Low to moderate stress?
- Stable relationships?
- Good sleep hygiene?
- **Overall health** (Good/ Fair/ Rubbish?)
- Anemic? Fibroids?
- Abrupt Hormonal Changes (hot flashes/night Sweats/PMS)
- Medications (statins)

Assess Body Composition

- BMI & percent body Fat
- Bone density (DEXA)
- Specialised equipment for measuring body composition (InBody etc)
- Measure extremity girth (3” from knees & elbows & around biceps/calf muscles). Can also measure waist.

- Always perform measurement with same control parameters (Same hydration, etc)

Do baseline metrics on the following symptoms BEFORE THEY START ANY DRUG TX

- Nausea
- Headache
- Biliousness/colic
- Dizziness/vertigo
- Light headedness
- Syncope
- Shortness of breath/chest pain & tightness
- Lower extremity cramping & Charlie Horses
- Constipation & straining/Hemorrhoids.

Standard Process

SYSTEMS SURVEY FORM

Restricted to Professional Use

NAME:

AGE:

HEALTH CARE PROFESSIONAL:

DATE:

Circle the corresponding number.

1	MILD symptom (occurs rarely)	2	MODERATE symptom (occurs several times a month)	3	SEVERE symptom (occurs almost constantly)

GROUP 1

1. 1 2 3 Acid foods upset
2. 1 2 3 Get chilled often
3. 1 2 3 "Lump" in throat
4. 1 2 3 Dry mouth, eyes, nose
5. 1 2 3 Pulse speeds after meal
6. 1 2 3 Keyed up, fail to calm
7. 1 2 3 Gag occasionally
8. 1 2 3 Unable to relax, startle easily
9. 1 2 3 Extremities cold, clammy
10. 1 2 3 Strong light irritates
11. 1 2 3 Occasionally weak urine flow
12. 1 2 3 Heart pounds after retiring
13. 1 2 3 "Nervous" stomach
14. 1 2 3 Appetite reduced occasionally
15. 1 2 3 Cold sweats often
16. 1 2 3 Get heated easily
17. 1 2 3 Nerve discomfort
18. 1 2 3 Staring, blink little
19. 1 2 3 Sour stomach frequent

___ ___ ___ TOTAL
1 2 3

GROUP 2

20. 1 2 3 Joint stiffness after arising
21. 1 2 3 Muscle, leg, toe cramps at night
22. 1 2 3 "Butterfly" stomach, cramps
23. 1 2 3 Eyes or nose watery
24. 1 2 3 Eyes blink often
25. 1 2 3 Eyelids swollen, puffy
26. 1 2 3 Indigestion soon after meals
27. 1 2 3 Always seem hungry, feel "lightheaded" often
28. 1 2 3 Digestion rapid
29. 1 2 3 Vomit occasionally
45. 1 2 3 Get "shaky" if hungry
46. 1 2 3 Fatigue, eating relieves
47. 1 2 3 "Lightheaded" if meals delayed
48. 1 2 3 Heart palpitates if meals missed or delayed
49. 1 2 3 Fatigue in afternoon
50. 1 2 3 Overeating sweets upsets
51. 1 2 3 Awaken after few hours sleep, hard to get back to sleep
52. 1 2 3 Crave candy or coffee in afternoon
53. 1 2 3 Moods of "blues" or melancholy
54. 1 2 3 Craving for sweets or snacks

___ ___ ___ TOTAL
1 2 3

GROUP 4

55. 1 2 3 Hands and feet go to sleep easily, numbness
56. 1 2 3 Sigh frequently, "air hunger"
57. 1 2 3 Aware of "breathing heavily"
58. 1 2 3 High-altitude discomfort
59. 1 2 3 Open windows in closed room
60. 1 2 3 Immune system challenges
61. 1 2 3 Afternoon "yawner"
62. 1 2 3 Get "drowsy" often
63. 1 2 3 Swollen ankles worse at night
64. 1 2 3 Muscle cramps, worse during exercise; get "charley horse"
65. 1 2 3 Difficulty catching breath, especially during exercise
66. 1 2 3 Tightness or pressure in chest, worse on exertion
67. 1 2 3 Skin discolors easily after impact
68. 1 2 3 Tendency to anemia
69. 1 2 3 Noises in head or "ringing in ears"
84. 1 2 3 Skin peels on foot soles
85. 1 2 3 Discomfort between shoulder blades
86. 1 2 3 Occasional laxative use
87. 1 2 3 Stools alternate from soft to watery
88. 1 2 3 Sneezing attacks
89. 1 2 3 Dreaming, nightmare-type bad dreams
90. 1 2 3 Bad breath (halitosis)
91. 1 2 3 Milk products cause upset
92. 1 2 3 Sensitive to hot weather
93. 1 2 3 Burning or itching anus
94. 1 2 3 Crave sweets

___ ___ ___ TOTAL
1 2 3

GROUP 6

95. 1 2 3 Loss of taste for meat
96. 1 2 3 Lower bowel gas several hours after eating
97. 1 2 3 Burning stomach sensations, eating relieves
98. 1 2 3 Coated tongue
99. 1 2 3 Pass large amounts of foul-smelling gas
100. 1 2 3 Indigestion ½-1 hour after eating; may be up to 3-4 hours after
101. 1 2 3 Watery or loose stool
102. 1 2 3 Gas shortly after eating
103. 1 2 3 Stomach "bloating"

___ ___ ___ TOTAL
1 2 3

GROUP 7A

104. 1 2 3 Difficulty sleeping
105. 1 2 3 On edge
106. 1 2 3 Can't gain weight

https://my.standardprocess.com/Products/Literature/Systems-Survey-Form?aliaspath=%2fProducts%2fLiterature%2fSystems-Survey-Form

Are they a candidate for therapy with drugs?

- Are they morbidly obese with significant comorbidities?
- Are they just wanting a ‘quick, possibly temporary, fix?’
- Do they understand what this drug will do & how it works?
- Do they understand the benefits vs risks?
- **Can they do the work needed** to assist the ‘drugs promise’ & then recover as the drug is withdrawn?
- We’re looking at a minimum 12 month commitment, even if they only use the drugs for 1-3 months.

Your patient is now taking GLP-1/GIP Agonists

Clinicians should Focus On the following….

- **Adequate Protein** – a natural GLP-1/GIP agonist
- Exercise daily to **maintain muscle mass**.
- Support digestion & peristalsis
- Support absorption
- Support elimination
- Support possible mineral uptake loss
- Support possible nutrient malabsorption
- Support the microbiome
- Supporting ANS PSNS
- **Decreasing inflammation with Chiropractic & allied health care**.

Nutrient Deficiency Risks with GLP-1 Therapy

Nutrients Depleted by Metformin

- ☑ Vitamin B12*
- ☑ Calcium
- ☑ Vitamin D

- ☑ Calcium
- ☑ CoQ10
- ☑ Iron
- ☑ Vitamins A,D,E,K
- ☑ Protein
- ☑ Omega-3's

Protein

- Intake – How much?
- Assimilation – can they break them down into amino acids for fuel?
- Absorption – do their intestines work well?
- Excretion – Can they get rid of proteins that have been assimilated & absorbed? – or are these proteins half digested creating extra histamine reactions, putrefaction and inflammation?

Measuring Protein Mass is tricky

- Systemic inflammation not only reduces albumin synthesis but increases its degradation and promotes its transcapillary leakage.
- Albumin has been criticized as a player in nutritional assessment due to its lack of specificity and long half-life (approximately 20 days)
- Albumin 3.5 - 5.0mg/dl.
- Prealbumin <10mg/dL = malnutrition. Half life 2-3 days.
- Blood Urea Nitrogen (BUN) 7- 20mg/dL High levels may indicate high protein metabolism/breakdown or renal issues.
- Urinary Creatinine measures the breakdown of skeletal muscle, but varies over 24 hrs & should be collected over 24hr period.
- Urinary N-telopeptide/creatinine ratio measures largely type 1 collagen in bone.
- See next slide……

Measuring Muscle Mass

- Because DEXA & bioimpedance have their limitations, there are other methods.
- Whole body MRI is good but has limitations (size of pt, etc)
- D_3creatine (D_3Cr) dilution method.(Oral dosage & urinary collection 48-96 hrs later). See Appendix.
- **Physical measurements may be your best bet**.
- Focus on **muscle strength** more than muscle mass.
- Dr A see footnotes…..

Exercise – Get a baseline

- Cardio.
- HIIT
- Walking
- Lifting weights
- **Can they do this without injury?**
- **Can they maintain exercise frequency & intensity over several months?**
- Recovery – What does this look like?

Recovery

- Minerals are not getting absorbed as well, thus, muscles often do not fully contract & relax well.
- Gastroparesis is a significant side effect. **Stretching abdominal area in all directions will be helpful.**
- Massage will be helpful in working out lactate & pyruvate in muscle spindles. (re: nutrients may not be absorbed into muscle well on GLP-1/GIP therapy)
- Chiropractic will assist in helping to ramp down inflammation & tissue tenderness & can be used to stimulate PSNS. **Chiro is mandatory for improving mobility & functional capacity**.

GLP-1 agonist risk vs benefits

What to watch in your patients on GLP-1 agonists

- "Compared to ***usual care***, GLP-1RA use was associated with a reduced risk of substance use and psychotic disorders, seizures, neurocognitive disorders (including Alzheimer's disease and dementia), coagulation disorders, cardiometabolic disorders, infectious illnesses and several respiratory conditions. There was an **increased risk of gastrointestinal disorders, hypotension, syncope, arthritic disorders, nephrolithiasis, interstitial nephritis and drug-induced pancreatitis** associated with GLP-1RA use ***compared to usual care***. *[usual care = usual western medical approaches].*

- **Support digestion** (bitters, digestive enzyme, Betaine, Okra Pepsin, PSNS, Sit & Chew food).
- **Hypotension/syncope**: Check Ragland's, Cuff test, regularly. B –vitamins, licorice, sns support (Phosfood, B6,RNA)
- **Arthritic disorders**: Blood tests; **Boswellia**, fish oils, Turmeric, Choline, Betacol, Glucosamine)
- **Kidney function:** Blood/urine tests, Hydration
- **Support pancreas**: Blood work: Check pancreatic function, PMG's, Gymnema

https://doi.org/10.1038/s41591-024-03412-w

Case study: Ozempic Patient Elizabeth

40 yr old Ozempic for 8 months pt 10/24

Chief Complaints:

- Aches and pains getting worse
- Digestion alternating constipation/diarrhea 1 yr
- Excessive belching & heartburn
- Distress with fatty foods
- Hungry hour after eating, increased thirst
- Frequent urination/increased thirst/difficulty losing weight/ vaginal dryness/dis-interest in sex post Celexa (SSRI) 10 yrs ago
- Irritable if meals missed
- Sleep poor since pandemic (65% of time takes 2 hrs to get to sleep at 9:30pm, 50% of time wakes at 2-3am; able to get back to sleep 65% of time) Insomnia.
- Dizziness when stands up last 20 yrs.
- Under high stress; Stress headaches
- Weak nails, dry skin
- Sweats at night last 20 years
- Hair falling out
- Mental sluggishness
- Nervous legs at night, last 20 years

Case study: Ozempic Patient Elizabeth

40 yr old Ozempic for 8 months pt 10/24

Clinical Findings:

- **Blood work**: **Chol 145mg/dl**; HDL 42mg/dl; tg 67, LDL 89. **CRP 2.2**; **Fibrinogen 386**; Ferritin 141; T4 8.0; WBC 8.3; Neut. 71%; Lympho. 21%; **Neutrophil/lymphocyte ratio:3.38**; **Potassium & calcium low; phosphorus low**; Mg 2.1; No Hashimoto's Ab; Vit D 42; **B12 381; Omega index 5.8**; **UA epithelial cells**.
- **Cuff test** 80; **Ragland's** – 109/68 | 113/77
- **Palpation**: GB ¾; R & L Ridler ¾, Thyroid non tender.
- **HRV** :Blocked & switched SNS/PSNS dysregulation -breast scars/metals: platinum (jewelry); Milk cheese eggs – major foods. Primary = gallbladder w/Chemical stress (all) Secondary: R adrenal – no stressors

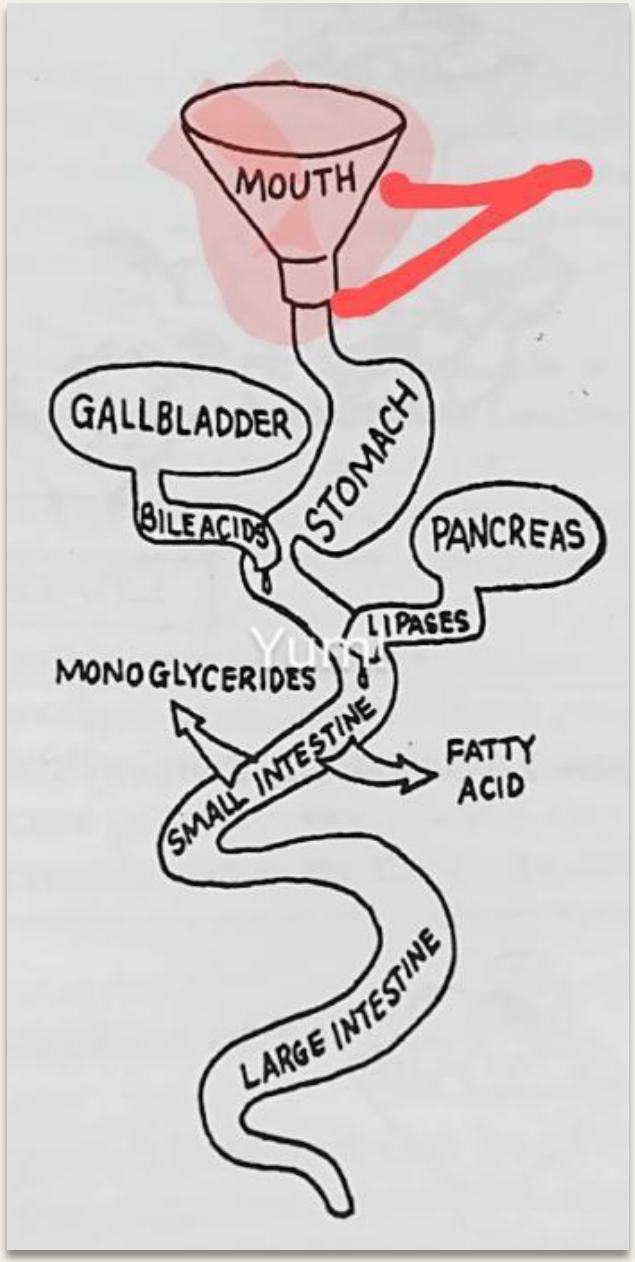
Starting program:

- **Ashwagandha Forte** 2,2
- **Boswellia Complex** 2,2
- **GI Adsorb** 3 before bed
- **Hemp oil complex** 1,2
- **Livaplex** 3,3,3
- **Magnesium** 200mg to **bowel tolerance**
- **Min Chex** 2,2,2
- **Olprima** EPA/DHA
- **Turmeric forte** 1,1
- **Wheat Germ Oil** on **scars** at night
- **Zypan** 2,2,2

Supporting Digestion

Sufficient HCl

- Chewing food
- PSNS while eating – ***remember digestion begins in the brain. The body and mind must be relaxed to fully digest food.***
- Saliva contains amylase (and salivary lipase)
- **What if our patient is on a blood pressure medication or similar that inhibit saliva production?** (see next slide)
- '90% of patients tested had low HCl by Heidelberg telemetry' Jonathan Wright, MD.



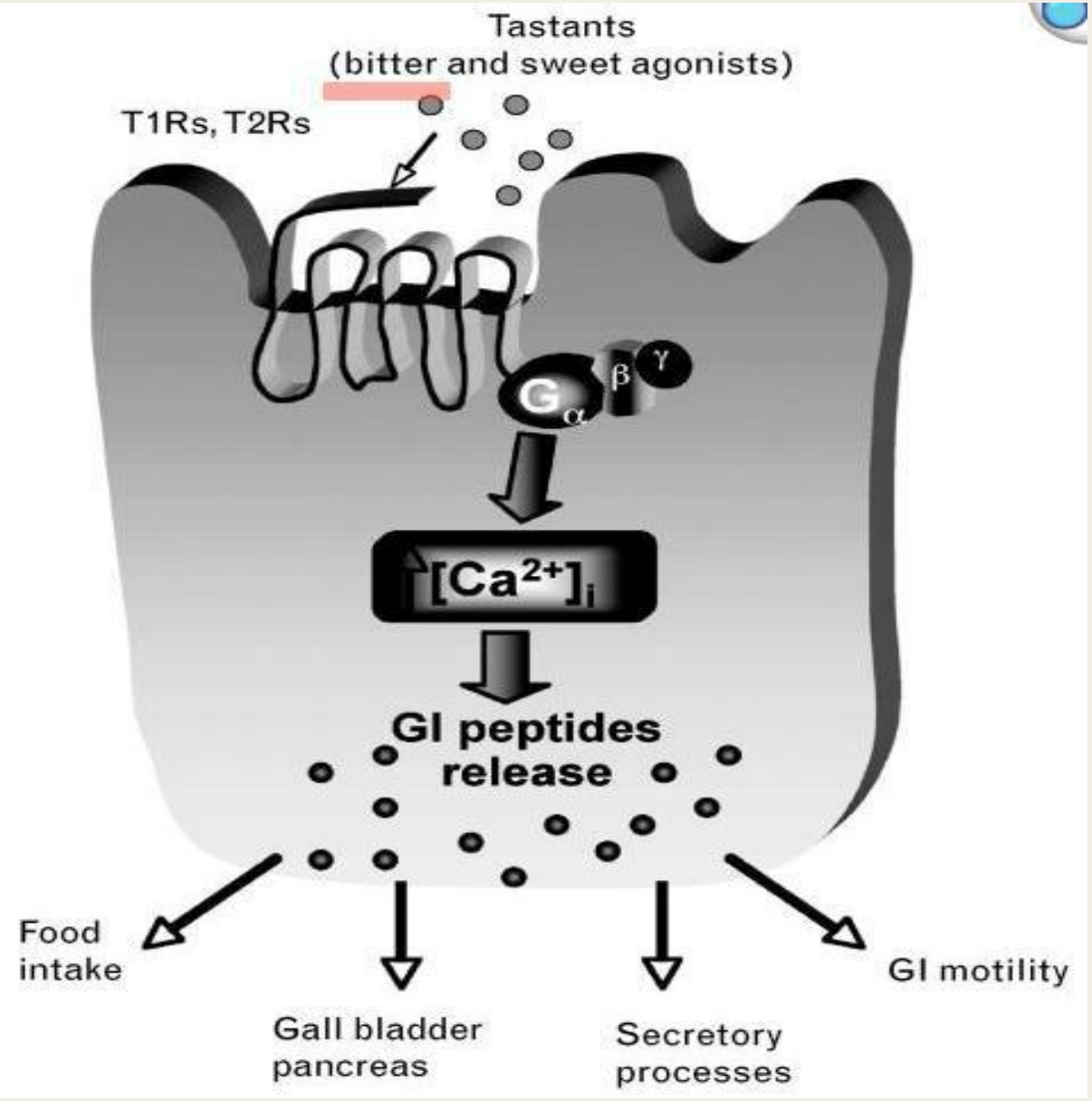
Stomach pH @0.8

Patient Education

- When the stomach attains **the right pH** (@ 0.8 pH), it signals the pyloric valve to open and empty contents into the duodenum.

- **Improper pH signalling** due to **too little Hydrochloric acid** will mean that **gastric emptying will be delayed** – sometimes for hours – *resulting in the fermentation and/or putrefaction of stomach contents and potentially generating reflux symptoms*.

- **Improperly digested chyme** that is still retained in the stomach **becomes rancid, putrefies and ferments to create toxicity**



Clinical: HCl

Subjective signs (gas, bloating, belching, heartburn, esophagitis (food sticks))	**Observation** (enamel erosion on teeth, breath, upper GI bloating)	**Objective testing** (Ridler's point, Chapman reflexes, Oral pH paper, telemetry, endoscopy, other tests)	**Clinical solutions**:
HCl betaine	Digestive Enzyme	Upper GI support	Apple Cider Vinegar and other **bitters**
Aloe juice	Detox	Okra pepsin	PSNS Stimulation

Supporting Absorption

- **Can we get the stomach to empty fully?**
- Digestive enzymes, (HCL/pancreatic proteases; bitters)
- Fasting pre=GLP-1/GIP therapy
- Purification Programs prior to starting any GLP-1/GIP therapy.
- See also above slide.
- **Can we get fats to assimilate? Bile to be manufactured?**
- Beetroot leaf juices, bile salts with Collinsonia, Cholinergic precursers (choline, Lecithin, B2 vitamin complexes), Globe Artichoke leaf, Dandelion root, Milk Thistle seed, Bupleurum root, and Fringe Tree bark extracts.

The Problem of ‘Thick bile’

- Betaine, which is naturally occurring in the leaf and root of the whole red beet plant, acts to flush the bile. Using Beet leaf juice concentrates can cause pain if the patient’s bile is too thick
- Vitamins A and F will thin the bile and make the flushing action of bile more tolerable if it is too thick. Vitamin A assists in fat metabolism. **Lecithin** thins the bile.
- **Bile salts** thin the bile.
- Cholesterol thickens the bile.

The Role of Bile Salts

- Bile salts prime the biliary system for proper bile production.
- **Bile salts detoxify the liver in some states**.
- Common indications of low bile-salt production: sore tongue, metallic/bitter taste on tongue

- The most common bile salts are Taurine (Taurocholic acid) and Glycine (Glycocholic acid).

The patient who needs bile salts:

Those that are **not getting adequate absorption of fat-soluble dietary factors, (ADEK), especially Vitamin A.**

Deficiencies of Vitamin A can also lead to irritation such as itching of the epithelial tissues.

An **acid stool** may be the cause of pruritus ani (stools should be more alkaline). Bile salts can assist in such cases.

Visual disturbances such as flashes of light (scotomas), if retinal detachment is ruled out (by an Ophthalmologic exam), they will benefit from bile salts.

Bile salts should ***not*** be taken long term in large doses (unless your patient does not have a gallbladder!), as they will inhibit the liver from further bile production.

We want the body to produce its own bile salts.

A quick reminder: Cholesterol and bile salts

- Cholesterol from general circulation is converted to bile salts.
- During this process, much cholesterol is removed from the circulation, **and serves as THE major route of cholesterol's elimination** from the body.

This conversion requires:

- **Adequate Vitamin A and C**,
- **A good balance of fatty acid families**,
- **Adequate levels of niacin/niacinamide, betaine and choline**.
- Adequate amounts of **taurine**, glycine and copper.

Garlic can also break down cholesterol.

Address Gastric bacterial stress

- Teeth hygiene program (educate them)
- Do they need a PPI?
- **Berberine** can be used concurrently with a PPI until you can transition them off it.
- Myrrh Forte, Artemisinin, Thymus and Spleen support.
- Take out stressors (sugar, caffeine, alcohol, processed food, sometimes fruits, heavy carb and protein meals).

Can you imagine what a PPI would do to this upper GI tract?

- Whether ‘over the counter’ or a prescription medication?
- ***You have a duty to lead your patients.***
- Many may be too far gone with significant cellular hyperplasia and dysplasia in their esophageal/cardiac sphincters (which can be pre-cancerous – monitor these patients with endoscopies).
- Most of them you will be able to assist in their rehab very successfully.

Clinical Pearl: Gas and Bloating

- If within 30 minutes of eating: Low Hcl. Needs Hcl.
- If within 30 to 120 minutes after eating needs gallbladder support
- If using bitters, ***suck them before*** eating foods

Clinical Pearls

- What is your patient's mineral status?
- What is their calcium cuff test score? Do their muscles cramp?
- Think minerals.
- Do you think they can get their major minerals out of their blood stream and into their tissues?
- We may need EFAs to assist this process (Cat F, Linum B6, Fish oils)
- Do they have good smooth muscle tone? (make an educated guess)
- Do they have good cholinergic pre-cursers?
- **Do they have GERD**? Protocols are like GI sphincter rehab (below)

GI Sphincter Rehab

- Is there reflux supine? Is it relieved by raising their torso?
- Can they handle some betaine/digestive enzyme/apple cider vinegar?
- **Do we need to soothe something else first?**
- Aloe Vera/HiPepDGL/ginger/chamomile teas
- After 2-8 weeks, can they handle Betaine, etc?
- **If ulcer present**, also add **Gotu Kola**,
- They are chewing food/sitting to get vagus engagement/are avoiding irritants (caffeine, alcohol, processed food, sugars, white breads, large boluses of protein)
- **Physical stretching** (somato-visceral reflexes)
- Chapman Reflexes manipulation, visceral manipulation
- **Ligaplex, Sulphur derivatives** (garlic, Spanish Black Radish, onions)
- Heal epithelial cells

Rapid fat loss in first 2 months on GLP-1/GIP

- **Toxins are lipophilic**
- **Rapid fat loss means large oxidative stress** in the form of lipid peroxidation (esp. as most patients on these have poor lipid profiles & often poor mitochondrial health.).
- This will stress mitochondria, leading to poor metabolic recovery unless fat assimilation, digestion, elimination & lipid peroxidation are handled.
- Astaxanthin has 100x the lipid peroxidation of Vitamin E.
- Anti-oxidants in the form of Rosemary, Bioavialable turmeric, Green Tea, Selenium Yeasts, wheat germ oil, NAC, pulsed Ascorbate, etc.

Astaxanthin: a red-colored xanthophyll carotenoid

- Exerts an **anti-oxidative and anti-inflammatory** effect on various cell lines.
- In this manner astaxanthin maintains **mitochondrial integrity under various pathological conditions**.
- Mitigates oxidative stress, lipid accumulation, and inflammation in skeletal muscle cells by **promoting mitochondrial biogenesis**, thereby preserving muscle structure and function.
- Shows **potential** as a therapeutic agent for **skeletal muscle protection in metabolic stress conditions.**

Astaxanthin studies

- **Astaxanthin** [ASX] 2021 study inhibits MMP's, inflammatory cytokines >cholesterol efflux, >reverse cholesterol transport. Pereira et al, int J Mol Med 2021
- **6mg/d ASX for 10 days** <blood transit time; >microvascular Flow; >tissue oxygenation, nutrient delivery & vascular efficiencyJ Clin. Biochem Nutr 2008 Miyawaki et al Effects of ASX on Human blood rheology.
- **8mg ASX /day for 8weeks**. < fasting Glucose & HbA1c > improved insulin sensitivity.< blood pressure w/ T2DM 2018 Mashhadi et al.
- **ASX as a metabolic regulator of Glucose & lipid homeostasis** .A number of **'preclinical studies'** support insulin sensitivity, secretion Beta cell function, stimulation of glucose transport, while supporting lipid homeostasis. Journal of functional foods 2024 Medoro et al.
- **ASX 12mg/day12 wks**. RCT (6 red food/day) **enhances metabolic adaption w/aerobic training in the elderly** 2021 Liu et al

Supporting Elimination

Gut Microbiome support

- The gut microbiome — the community of bacteria living in your digestive tract — directly influences GLP-1 secretion, and **dysbiosis** (an imbalance in those bacteria) **can blunt GLP-1 effectiveness.**
- Endotoxins — toxins released by certain bacteria — can leak into the bloodstream when the gut barrier is damaged, triggering body-wide inflammation making it harder for GLP-1 to work.
- **Probiotics**, (Lactobacillis, acidophilus), pre-biotics, (2-FL, inulin) biofilm busters (garlic, berberine, Cats Claw, Red Kale)
- **Binders** (zeolite, chlorella) for mycotoxins

Candida/Mycotoxin support

- **Remove** – dietary: (sugar, high glycemic fruits); enviromental: (house/work myco-stressors, chemicals)
- **Remove** pathogenic Yeasts: Kill off pathogenic yeasts (Garlic, Spanish Black Radish, Oregano oil, Caprylic Acid, Black Walnut, Slippery elm, Clove oil, Artimisia annua, myrrh),
- **Restore** – (**Replace, Re-innoculate, Repair**) - increase beneficial yeasts (lactic acid Yeasts, fermented foods, lactobacillis, acidophilus, bifido-bacter, etc)
- **Heal intestinal barriers**/villi (okra, aloe, Rutin, Gotu Kola, L-Glutamine, slippery elm, DGL, Marshmallow root); Reduce intestinal inflammation: Bioavailable turmeric, Boswellia resins.

Decreasing Sympathetic Dominance

ANS Dysregulation –Decreasing SNS stress (driving hypertension, insulin resistance, poor digestion, absorption & elimination)

- SNS creates vasoconstriction, creating increased pressure
- SNS drives up insulin resistance because patient is stuck in ‘fight-flight’
- SNS ruins digestion, teeth & sleep.
- SNS contributes to shallow breathing (apnea)

Clinical Pearl: Signs of SNS Overload

- High levels of cortisol/nor-adrenalin in urine (DDX Cushings, pituitary/adrenal tumor, severe depression)
- Hypertonic muscles (upper trapezius, lumbar erectors, etc)
- Humoral cortisol spill-over…Saliva, serum measurements in lab tests
- HRV graph readings in real time at a +2 or higher
- Tachycardia in the absence of heart chamber dysregulation or failure and COPD.
- Positive Ragland's (although sympathetica-tonia can also exist with a negative Ragland's)
- Pupil size: Constricted = PSNS; Dilated = SNS.
- History and observation (fighty/flighty, domestic situation, etc.)

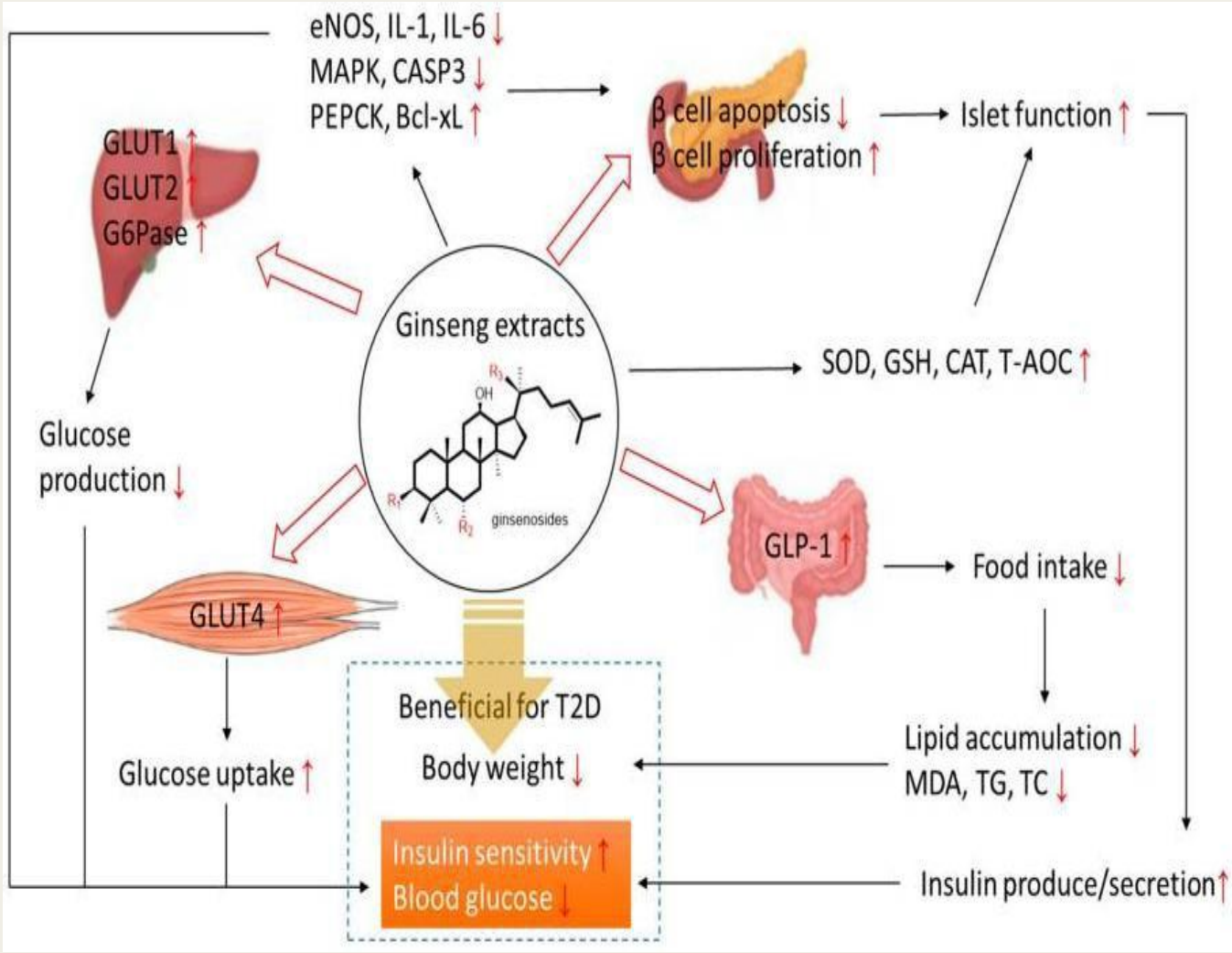
Physiological SNS downregulation

- **Orchic Extracts,** especially for short term responses to stress, such as palpitations, temporary high systolic readings (before pressure has become chronically high). So called ‘enzymatic spreading factor’ postulated for results.
- **Trace Minerals** (Kelp, alfalfa,) **Organically Bound Mineral Complexes**
- Microminerals: **Magnesium, calcium, potassium (Mg Salts, Whole Food Mag, Cal-Mag); Calcium Salts, Natural sources of Trace minerals.**
- **Herbs: Skullcap, St Johns Wort, Schisandra, Saffron**
- **Valerian, Kava, Hemp oil, [Corydalis, California Poppy, Jamaican Dogwood]**
- **Relaxation techniques, meditation, counselling, exercises, sleep.**
- **Specific Chiropractic Adjustments.**
- **Low Level Laser Therapy – Vagal toning**

Ozempic GLP-1 agonist alternatives

Some Common Alternatives

- **Berberine** (w/silymarin), 800-1200mg/day.
- **Fenugreek seed** – slows transit time; Fen Greek seed (1.2g/tab); Fenugreek & Choline (686mg/tab).
- **Bitters - Appetite regulation, Digestive support**
- **Protein > GLP-1 signalling, reduces cravings**
- **Fiber > satiety, stabilize post prandial glucose.**
- **Exercise** (150mins moderate/wk)
- **Korean Red Ginseng** (for T2DM), Korean Ginseng **Liquid 2 tsps./d or 5ml BID**), Rhodiola/Ginseng (6-8/day)
- **Green Tea/Astaxanthine/Turmeric** – Anti-oxidants



Supporting Dysglycemia

Berberine is anti-glycemic

2022 SRMA 37 papers on berberine/T2DM

- **Berberine** can inhibit the voltage-gated k^+ channels of **pancreatic β cell membrane** and **promote insulin secretion without causing hypoglycemia**, because….
- **The glucose-lowering effects of berberine are only under hyperglycemic conditions or in a high-glucose-dependent manner.**
- **Safe & effective** both stand alone & in conjunction with Oral Hyperglycemic Agents. (Metformin, Glypizide, Gliclazide, Sulfonylureas, etc)

- Can **reduce HbA1c by** between 0.82; 0.63; 1.16; 0.73 mmol/L (**14.7mg/dl;11.3mg/dl; 20mg/dl;13.1mg/dl**) in some of the studies.(median values)
- **A 1% Change in A1c is equal to 11 mmol/mol.**
- Stand alone Berberine dosages varied between 600mg/**900mg/1000-1,500mg**/9000mg/**day TID** (mostly herbal derivatives (Chinese studies))
- Many studies which **combined BBR with MET** dosage was **usu. 900mg/1500mg TID**.
- Most studies **included lifestyle modification**
- Most study times were **90-180 days (3-6 months)**

Berberine vs Metformin glucose regulation

2009 study 500mg TID 3 months

- 2009 study n=36; 500mg TID 3 months
- **HbA1c dropped by 7.5% in both groups**
- Fasting glucose dropped by 6.9%
- Post prandial glucose dropped by 11.1%
- **Metformin & berberine were equivalent**

- **Pregnancy & lactation**: Information is very limited as there have been **no trials** on this. (Huang lian TCM, 218 pregnancies & no birth defects; bilirubin ‘can potentially build up in brain’ – 1 study) **Best to avoid**.

Berberine

Standard daily dose: 1g- 1500mg/day

- Berberine - a potential drug to treat **obesity** by **downregulation of adipogenesis and lipogenesis**.
- Berberine **exerts its long-term body weight losing effect** through enhancing AMPK-mediated ATGL expression, which **increases the basal lipolysis state of triglycerides in adipocytes**
- Berberine has been shown to **exert antitumor effects through multiple routes**, for example, suppressing cell proliferation, metastasis and angiogenesis in numerous tumour types, including breast cancer, gastric cancer, melanoma, and hepatoma [>600 publications in Pub Med].
- **Anti-inflammatory effects** of berberine has been acknowledged for long history. [<nf-kB, IL-8, etc]

Gymnema

Sweet Suppression in the G.I. Tract – Tongue to small intestine

- **Anti-diabetic** – results best achieved by **6-12 month administration.**
- **Anti-obesity -** Weight loss and dieting since Gymnema (in liquid form) anesthetizes the sweet taste buds (also in tablet form, to an extent [suck tablet]).
- **Hypo-lipidemic**
- **Anti-microbial**
- **Free radical scavenging**
- **Anti-inflammatory**

- 400mg leaf extract containing 100mg Gymnemic acids /tablet.
- No adulteration problems
- Inhibits sweet taste sensation
- Ayurveda = gurmar aka “sugar destroyer”

Fenugreek 3-9/d

According to recent biochemical studies, 2023 SRMA Review

- Anti-diabetic properties by **prolonging gastric emptying time and lowering glucose uptake in the small intestine because of their high fiber content**, thereby slowing down carbohydrate metabolism and lowering blood glucose levels.
- **Restoration of pancreatic cells** by safeguarding β-cells [32,33]
- An increment in serum insulin levels by **stimulation of islet cell regeneration or insulin release from pre-existing islet cells.**
- Fenugreek also stimulates glycogen synthase activity and **promotes the production of liver and muscle glycogen** [34]. This promotes the regeneration of depleted glycogen, decreases pro-inflammatory cytokines and pancreatic enzymes, and adjusts the serum lipid profiles and the activity of insulin-sensitive carbohydrate metabolic enzymes [34].
- Fenugreek **may increase insulin sensitivity** by enhancing insulin action at the cellular level, lowering HbA1c levels by using glucose in the peripheral tissues and maintaining blood glucose levels [32,33,34,35].
- Not only does fenugreek affect blood sugar, but multiple reviews also suggest a role in the **improvement of lipid profiles**, making it suitable for metabolic disorders [4,9,24,35,36]. ***[< Cholesterol, LDL's, tg's]***
- It 'could be' possible that there are interrelated mechanisms of action of fenugreek where its biochemical properties could be related to the metabolism of sugar, lipid, or both [35,36].

doi: 10.3390/medicina59020248

Case Study: High A1c _ Ronnie 66 yr old

High Fasting Glucose 261/HbA1c 11

- **History**: Been prescribed Metformin 10 yrs prior didn't like it so stopped it. Had been on Lisinopril for HTN, but didn't like chronic cough & dizzy side effects, so stopped.
- Difficulty getting an erection. **Highly stressed 7-8/10**
- **Cc**: recurrent tinnitus (hearing test negative)
- **Ragland's: |173/94, - 146/89**
- Cuff test:220
- **+2/4 R Ridler's point; stomach, transverse colon, pancreas area**.
- Heavy fungus on toes. Bloated abdomen
- HRV: PSNS/SNS 'switched'
- Kinesiology: Small Parasites; Thyroid Primary.
- Cardio Plus Blood panel ordered:
- **Fasting Glucose: 261mg/dl**
- **HbA1c: 11.1%**
- B12: 335
- **UA: Protein, Glucose & Ketones +1/+3/+2.**
- GFR:104; CRP 2.19; Vit D 52; T4 8.1; TSH 0.18

Program high HbA1c

- Referred to PCP for opinion. Pt wants to go as natural as possible. Declined meds.
- **Starting Program**:
- **Zymex II** (1st month only)
- **Ashwaganda Forte** 1,1,1
- **Gymnema** 2,2,2
- **Pancreatrophin PMG** 3,3,3
- **Hydrate daily: 8 glasses/day**.
- Apple cider vinegar daily, Brazil Nuts, Watermelon rind (L-Citrulline)
- Pt declined adding Berberine the following month. Also Saw A PCP who Rx. METF. Pt declined
- Pt slept a lot. 'Did not exercise' but had very physical job on printing press, 10hr shifts.
- **5 months later**:
- Fasting glucose: 129mg/dl
- **Hb A1c: 7.1%**
- **Bp:110/72**

Case Study: 40 yr old Jennifer

Goals: '*Heart & Mind Health'*

- 'High Cholesterol' Dx by PCP
- Lower back health
- Better energy
- **Wants to lose 80#** (is 240#) [waist girth >hips]
- Recovering EtoH 1.5 yrs (gives her heartburn)
- Gave up sugar 6 months ago (used to have fatigue after meals, 'this has gone away' in last 2 months)
- **Fatigued in afternoon & 'after bread'** (doesn't eat it much)
- **High amounts of stress, 5-7/10** (daughter on spectrum, husband EtoH)
- Occasional difficult bowel movements
- Heart Palpitations (when falling asleep & thirsty)
- Ankles swell if sit too long.
- Diminished Sex Drive (no trust w/husband, no desire)
- Peri-menopausal via her PCP (2 yrs ago 'no progesterone' via blood work).
- Has regular cycle, (scant start, heavy finish).
- Taking supplements still that I had given her a year or 2 ago.

INITIAL Exam Findings

Following 21-day Purification Program

- Food Diary: **n/a – came in following a 21 Purification Program**
- **Ragland's**: Supine: 122/75 Standing: 124/86
- **Cuff test**: 160
- **HRV**: Blocked PSNS
- **Palpation**: asc. Colon 2/4, Ridler's ¼.bi. Thyroid non-tender.
- **Kinesiology**: PSNS block: Yeast/fungal. Liver Primary. (Colon 0 incl. IC valve) Adrenal, secondary.
- **Prior Labs 6 mo earlier**: **Vit D 15.5mg/dl**; Chol184; tg 64 HDL 59; LDL 110.**Lp(a)73mg/dl**; CRP 1.9, ferritin 17.

Initial Program

Purification Program –Completed ||

- **Walk** dog 2x/day (exercise)
- **Increase protein**
- Continue 3-4 servings of fish/wk.(was also doing **Olprima DHA/EPA** 2,2/d), since I last saw her.
- **CardioPlus**, 3/d
- **Cholaplex,** 6/d (Orchex/Cat G/Cat F/Cyruta/Cholacol)
- **Drenamin** 4/d
- **Organically Bound Minerals** 6/D
- Zymex Caps 6/d
- **Vit D** (continue).

Following 2 months on program:

- **Blood work following 2 months** on program: Chol 196mg/dl; tg 70; HDL 68; LDL 114
- **Lp(a) 30.4mg/dl**
- CRP 1.6
- Vit D 20
- ***All chest tightness & palpitations gone***.
- ***Discuss***.

Current patient stereo types on GLP-1 Agonists

- Lose #20 in first 2 months
- Gastroparesis in 70%
- Increased NMS Pain & tenderness in 90%
- Nausea & dizziness
- No appetite
- Muscle cramping esp. Lower Extremities.
- When come off or have REBOUND weight gain
- Put on some sort of daily exercise (to offset muscle mass loss)
- Unsure on length of time to take drug, some ‘forever’
- **A large portion ‘have no clue that they should do anything else than just take the drug’**

Increased NMS P & tenderness

- Boswellia & Turmeric resins
- Celery seed extracts
- Magnesium Salts & Foods
- Omega indexes/ Fish oils & dietary consumption
- Sort digestion
- Sort sleep
- Sort PNS/SNS balance
- Adjust them

Key Supplements - Summary

- Bitters, Betaine HCL, Pancreatic enzymes
- **Liver Phase 1 and 2, Kidney, intestinal Purification Programs w/Protein shake support**, 10-21 days.
- Beet leaf juice concentrates with vit A and F/Ox bile salts w/Collinsonia
- **Biofilm modulation (garlic red Kale, alpha amylase, bifido bacter, acidophilis)**
- Bind myco & other gut toxins chlorella/zeolite/Collinsonia
- **Antioxidant/lipid peroxitive/ blood sugar support** (astaxanthine/nitrites/chromium)
- **Boswellia Celery seed, turmeric (bioavailable)/Gota Kola/white willow (anti inflamm. & collagen support**
- Magnesium
- Minerals (Kelp/alfalfa).
- Mineral & Orchic Complexes to support PSNS
- Quality Gymnema/Berberine herbal extracts
- **Fiber Support Psylium husk, rice bran, pectin, etc**
- Hypothalamus support (Bovine nucleic organ support

Thank You!

Contact/Referrals

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Website: www.aggerchiro.com
Linkedin: www.linkedin.com/in/simon-agger-61494410
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Appendix - Part 1 FCA

- Common Causes of too little saliva (common Drugs).
- Dental Hygeine Program examples
- Stomach Peristalsis
- Migrating Motor Complex
- D_3creatine (D_3Cr) dilution method

Common causes of too little saliva ‘dry mouth’

- **Calcium Channel Blockers -** Amlodipine (Norvasc),Diltiazem (Cardizem, Cartia, dilasor, Diltia, Diltia XL, Tiazac), Felodipine, Isradipine, Nicardipine, Nifedipine (Adalat CC, Procardia), Nisoldipine (Sular), Verapamil (Calan, Covera HS, Isoptin, Verelan).
- **Anti-depressants** SSRI’S often cause dry mouth; Citalopram (**Celexa**), escitalopram (**Lexapro**), Paroxetine (**Paxil**). Fluoxetine(**Prozac**), Fluvoxamine (**Luvox**) and Sertraline (**Zoloft**).
- **Anti-anxiety drugs** - Valium, Lorazepam, and **Xanax**
- **Antihistamines** - Atarax and Vistaril (hydroxyzine hydrochloride), Clarinex (desloratadine), Xyzal (levocetirizine), **Allegra** (fexofenadine), **Benadryl** (diphenhydramine), **Claritin** (loratadine) and **Zyrtec** (cetirizine)
- **Chemotherapy medications**

Clinical Pearl: Know your patients meds!

- Have them fill out a medication form (next to their supplement form)
- When did they start them and **why?**
- **Ask them about NSAIDs, ASPIRIN AND ORAL STEROID INHALERS**
- How long have they been on them?
- Have **any dosages changed?** Have they increased or decreased?
- Do they take them regularly?
- Do they **feel any difference** when **not taking** or **taking them?** What **specifically**, do they notice?
- Are they interested in natural alternatives, **including diet and lifestyle?**
- **On a scale of 1-10 how much do they want to change their health?**

Dental hygiene program examples

- Floss 3 times daily after meals;
- Use throat spray throughout day (5-10 servings)
- Scrape tongue if coated and treat underlying cause
- Rinse mouth after meals (salt water, Listerine, doTerra ‘On Guard,’ herbal rinse (goldenseal/echinacea/Myrrh – then spit out)
- Salt water rinses
- Topical uses: Liquid echinacea, Goldenseal, licorice on gums. (see Herbal Compendium)
- Take gum and bone support for teeth (esp. if had had fillings 35+ years)
- Raw Veal bone products to support dentin
- Buckwheat leaf juice concentrates for bleeding gums.

Does stomach peristalsis affect absorption? YOU BET!!

- Motility is a summation on the enteric nervous system, ***vagal efferents and thus, requires the requisite minerals*** (for calcium depolarisation of the nerve cells, vagal efferents, and good smooth muscle tone).

- **Do your patients have adequate calcium supplies?**
- What does their expanded metabolic panel show?
- How many patients do you see with Calcium below 9.0? (should be 9.0 - 11.5mg/dl)

- **What makes good smooth muscle tone?** - Good downstream cholinergic pre-cursers (Choline, Lecithin, B2(G) vitamin complexes)

- When food is present, all patterns of gastrointestinal motility are based on **three fundamental motor events** programmed by the enteric nervous system:
  1. Peristalsis (propulsion),
  2. **Mixing movements (called segmentation),**
  3. Tonic contraction at the sphincters.
- Segmentation (mixing movement) are cyclic contractions of the muscle in local segments which force the chyme (food bolus) to slosh back and forth for short distances.
- **This ensures maximal absorption of nutrients by the epithelial cells**.
- Segmentation **occurs in the fed state**.

Motility ‘with’ and ‘without’ food….

- MIGRATING MOTOR COMPLEX (MMC) sweeps the GI tract clean **during periods of fasting.**

- The MMC performs a “housekeeping” function in that **it prevents bacterial overgrowth due to stagnant food contents.**

- The MMC **usually originates in the stomach but can begin in either the duodenum or the jejunum**.
- It occurs at a different frequency than the peristalsis associated with fed states.

Motility ‘with’ and ‘without’ food….

- The hormone, motilin, is thought to be responsible for the initiation of the MMC.
- **Motilin is secreted by an empty stomach**. If the stomach has undigested, putrefying, fermenting food still in it……..

- Finally, ***How are their sphincters***? (Endoscopy)

Oral tracer dose of deuterated creatine (D3Cr)

- Heymsfield et al. [12] estimated that about 98% (taken from Hunter [13]) of the body creatine pool is sequestered in the sarcomere; muscle has no capacity for creatine synthesis.
- Creatine is synthesized in the liver and kidney and transported against a huge concentration gradient into the sarcomere.
- The phosphorylation of creatine to high energy phosphocreatine represents an immediately available source of ATP for rapid muscle contraction.
- Because of its unique metabolic role for ATP synthesis, creatine and creatine phosphate are co-located with the contractile apparatus of muscle [14].

- The method uses an oral tracer dose (**30**, 15 and 2 mg for adults, children, or infants) **of deuterated creatine**.
- It is 100% bioavailable and after entering the circulation, D_3Cr is actively transported into all skeletal muscle cells.
- The method requires a **fasting urine sample** as consumption of foods with creatinine (mainly meat and meat products) results in increased urine creatinine excretion and dilution of the D_3creatinine enrichment.
- Thus, the method requires **knowledge of the tracer dose** of D_3Cr, **ingestion of the dose**, and **collection of a single fasting urine sample 48 to 96 h later.**