THE INTERCONNECTED CHRONIC HEALTH CONDITIONS OF UNCONTROLLED DIABETES, IN THE GERIATRIC PATIENT. HOLISTIC MANAGEMENT STRATEGIES

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Abstract:

Managing patients with uncontrolled diabetes that also have multiple other associated metabolic disease processes both diagnosed and undiagnosed, can be very demanding. Diabetes, hypertension, high triglycerides, high stress, high cortisol, insulin resistance, leptin resistance, kidney failure, and circadian rhythm dysregulation are deeply interconnected conditions that amplify each other, increasing the risk of severe health complications. Ignoring the metabolic dysregulation and multiple possible root causes of the uncontrolled diabetes is a big mistake that creates a perpetual degrading cycle making it difficult for the provider and increases the suffering for your patients. A holistic approach to your complicated, obese, diabetic patients by addressing diet, lifestyle, medical management, and circadian rhythm misalignment can improve insulin and leptin sensitivity, promote weight loss, reduce cardiovascular risk, put diabetes in remission, and enhance overall health outcomes. Something as simple as regulating your patients' sleep cycle could mean the difference between being chronically ill or not. Understanding that caloric intake is not the primary driver in obesity, especially in the geriatric patient and neither is their carbohydrate intake alone. Being an insulin dependent diabetic is based on metabolic dysfunction, not just elevated blood glucose. Their age, weight, daily routine, and sleep cycle dictate their hormone effectiveness through their metabolic flexibility and function. Focusing on one problem, elevated blood glucose levels, alone, will cause you to systematically fail.

Keywords: Diabetes, Hypertension, High Triglycerides, High Stress, High Cortisol, Insulin Resistance, Leptin Resistance, Circadian Rhythm Dysregulation

Introduction:

This paper is written to help bring to light the hidden metabolic disease of the chronically ill and obese patients, with uncontrolled or hard to manage insulin dependent diabetes. With the speed of our modern medicine and how are modern doctors are trained, root cause and general metabolic health continue to be overlooked. I'm finding more and more patients in the community, on so many prescriptions, in such poor health, exhausted, defeated, and begging for help. I'm seeing many patients on insulin to manage their out-of-control blood glucose levels with no other medications prescribed to assist with insulin sensitivity, which is a large component of managing diabetes. I am compelled to write this to hopefully bring to light some of the overlooked complications with these patients.

Discussion:

Although current research says insulin should be used in conjunction with other adjunctive agents and lifestyle changes to manage type 2 diabetes, insulin therapy seems to be the cornerstone for managing uncontrolled type 2 diabetes, rather than taking an approach that addresses the metabolic dysfunction. Diabetes has a complex interaction with hypertension, high triglycerides, high stress, high cortisol, insulin resistance, leptin resistance, kidney failure, and circadian rhythm disruptions. While insulin effectively lowers blood glucose, it may exacerbate weight gain, sodium retention, and leptin resistance, potentially worsen metabolic dysfunction, if used in isolation, continuing the cycle of overall health failure. A comprehensive approach—combining insulin with insulin-sensitizing agents, lifestyle interventions, stress management, and circadian-realignment strategies—can optimize outcomes by improving overall metabolic health, improve insulin and leptin sensitivity, and reduce cardiovascular risk. Close monitoring and patient education are critical to balance insulin's benefits and mitigate its many adverse effects on these interconnected conditions in the chronically ill patients.

The broader implications and challenges of insulin injection can lead to a vicious negative cycle. Insulin therapy, while essential for glycemic control, can contribute to weight gain, hyperinsulinemia, and sodium retention, potentially worsening insulin resistance, leptin resistance, hypertension, and kidney failure. These effects can perpetuate metabolic dysfunction if not addressed holistically and continue the cycle of failure.

Weight gain from exogenous insulin is often fat rather than muscle, potentially exacerbating sarcopenia in frail patients with low HGH (50-70% lower by age 65, J Gerontol A Biol Sci Med Sci, 2001)[11], especially if combined with GLP-1 agonists' muscle loss risk (J Cac Sar Mus, 2019)[12]. Sarcopenia risk in the geriatric population can intensify with exogenous insulin injections and precautions should be taken. Insulin-induced weight gain is a significant concern, as it exacerbates insulin resistance, leptin resistance, kidney failure, and elevated triglycerides, increasing cardiovascular risk. This underscores the need for concurrent lifestyle interventions. While insulin improves glycemic control, its effects on hypertension, triglycerides, and inflammation (via leptin resistance) require careful monitoring to prevent cardiovascular and kidney complications. If insulin levels remain too elevated the body is unable to burn fat for energy and remains stuck in a fat storage mode. This increases the risk of every injection of exogenous insulin increasing body fat and weight, especially with the late-night injections. This produces leptin resistance, suppression of normal hormone function, and perpetuating the metabolic dysregulation.

Metabolic Dysfunction Created When Injecting Insulin to Control Diabetes.

Impact on Diabetes when injecting insulin: Effective in reducing hyperglycemia-related complications (e.g., neuropathy, retinopathy, nephropathy) in the short term. However, it does not address the underlying insulin resistance and may require escalating doses over time if lifestyle interventions are inadequate (diet and sleep). Insulin resistance exacerbates hyperglycemia, accelerating kidney damage, heart disease, and increasing triglycerides and body fat, leading to higher insulin doses.

Impact on Hypertension when injecting insulin: May exacerbate fluid retention, particularly in patients with pre-existing hypertension, heart failure, or kidney failure. However, better glucose control can improve endothelial function, potentially mitigating hypertension over time. While insulin improves glycemic control, its effects on hypertension, triglycerides, and inflammation (via leptin resistance) require careful monitoring to prevent further cardiovascular complications.

Impact on High Triglycerides when injecting insulin: Effective in reducing triglyceride levels, particularly when combined with dietary changes (e.g., reducing carbohydrates and eliminating refined carbohydrates). However, weight gain from insulin therapy may counteract this benefit in patients with metabolic dysfunction.

Impact on High Cortisol levels when injecting insulin: Often driven by chronic stress, elevated cortisol levels can promote gluconeogenesis and glycogenolysis, increasing blood glucose and counteracting insulin's glucose-lowering effects. Cortisol worsens insulin resistance, potentially reducing insulin's efficacy. Insulin therapy may require higher doses to overcome cortisol-induced hyperglycemia.

Impact on Insulin Resistance when injecting insulin: Exogenous insulin does not reverse insulin resistance but bypasses it by providing sufficient insulin to overcome reduced cellular sensitivity. However, high insulin doses can lead to hyperinsulinemia, which may further downregulate insulin receptors and exacerbate resistance in the long term. In the short term, insulin therapy improves glycemic control but may not address the root cause of insulin resistance (e.g., obesity, inflammation). Weight gain from insulin can worsen insulin resistance over time.

Impact on Leptin Resistance when injecting insulin: Insulin and leptin signaling pathways are closely linked, as both hormones regulate energy balance and metabolism. Hyperinsulinemia from exogenous insulin can exacerbate leptin resistance by promoting inflammation and fat accumulation, which impair leptin receptor sensitivity in the hypothalamus. Insulin therapy may contribute to weight gain, worsening leptin resistance and increasing appetite, which complicates diabetes management. Exogenous insulin increases circulating insulin levels, promoting fat accumulation (2-4 kg over 6-12 months) and inflammation (e.g., 10-20% rise in IL-6, Molecular

Medicine, 2008)[13]. This impairs leptin signaling by downregulating leptin receptor sensitivity, exacerbating leptin resistance.

Impact on Circadian Rhythm disruption when injecting insulin: Insulin therapy must be timed appropriately to align with circadian rhythms, as glucose metabolism and insulin sensitivity vary throughout the day (higher in the morning, lower at night). Mismatched insulin administration (e.g., high doses at night) can disrupt circadian-regulated processes like cortisol and leptin secretion. Improperly timed insulin may exacerbate circadian misalignment, worsening insulin and leptin resistance. For example, late-night insulin doses could interfere with melatonin production and sleep quality, further disrupting metabolism. Human Growth Hormone (HGH), known as the master hormone, produced by the anterior pituitary gland, is released during deep (slow-wave) sleep, typically in the first half of the night (*J Clin Endocrinol Metab, 1998*)[1]. This nocturnal surge is tightly regulated by the circadian clock, mediated by the suprachiasmatic nucleus (SCN), which synchronizes HGH release with sleep-wake cycles. Chronic low HGH reduces insulin sensitivity by decreasing muscle glucose transporters (GLUT4) and increasing fat-driven inflammation, leading to higher blood glucose levels (*Diabetes, 2003*)[2].

Impact on Chronic Stress when injecting insulin: Improved glycemic control may alleviate short term stress-related symptoms, but chronic stress can still elevate cortisol, counteracting insulin's effects by promoting gluconeogenesis and insulin resistance. Chronic stress activates the hypothalamic-pituitary-adrenal (HPA) axis, elevating cortisol and exacerbating insulin resistance, leptin resistance, hypertension, kidney failure, and diabetes. Stress-induced inflammation also damages renal tissue.

Impact on Weight Gain Risk when injecting insulin: Insulin-induced weight gain is a significant concern, as it exacerbates insulin resistance, leptin resistance, and triglycerides, increasing cardiovascular risk. This underscores the need for concurrent lifestyle interventions.

Interventions to Mitigate Metabolic Dysfunction While Managing Type 2 Diabetes

Morning Sunlight Resets the Circadian Rhythm: The circadian rhythm, regulated by the (SCN) in the hypothalamus, is synchronized by external cues called zeitgebers, with light being the most powerful. Morning sunlight, particularly blue-wavelength light (460-480 nm), suppresses melatonin production and signals the SCN to advance the circadian clock, aligning biological processes (e.g., sleep-wake cycle, hormone release, metabolism) with the day-night cycle. Exposure to natural sunlight (or bright artificial light, ~10,000 lux) for 15-30 minutes within 1-2 hours of waking is most effective. Morning light shifts the circadian phase earlier, promoting earlier sleep onset and wake times (*Sleep Med Rev, 2010*)[3]. Studies show that morning light exposure (30 min/day) improves sleep quality, reduces circadian misalignment, and enhances metabolic outcomes in patients with diabetes and obesity (*J Clin Endocrinol Metab, 2017*)[4].

Effects of sleep cycle on Hypertension: Circadian disruptions increase nocturnal blood pressure (non-dipping pattern), stressing the kidneys and cardiovascular system. Morning sunlight reinforces daytime sympathetic activity and nighttime parasympathetic dominance, normalizing blood pressure rhythms.

Effects of sleep cycle on High Triglycerides: Circadian misalignment increases hepatic lipid synthesis and triglyceride levels due to disrupted clock gene expression (e.g., CLOCK, BMAL1). Morning sunlight restores lipid metabolism rhythms.

Effects of sleep cycle on High Stress and Cortisol: Circadian misalignment elevates cortisol by disrupting the HPA axis, worsening stress and insulin resistance. Morning sunlight suppresses nighttime melatonin and normalizes cortisol rhythms (peak in morning, nadir at night). The impact of morning sunlight lowers cortisol levels (*5-10% reduction in evening cortisol, Psychoneuroendocrinology, 2016*)[5], reducing stress-related metabolic dysfunction. In geriatric patients, lower cortisol mitigates muscle catabolism (supporting sarcopenia management) and renal stress, complementing stress reduction strategies.

Effects of sleep cycle on Insulin Resistance: Circadian disruptions reduce insulin sensitivity by altering clock gene expression in muscle and liver. Morning sunlight enhances insulin signaling via circadian alignment and improves insulin sensitivity, reducing insulin doses and hyperinsulinemia risks compared to insulin therapy alone. In the geriatric patient, morning sunlight

enhanced insulin sensitivity reduces renal glucotoxicity and supports metabolic control, critical in CKD patients. Blue Light Therapy (BLT), simulated morning sun, for 30 minutes, significantly improved insulin sensitivity by 20–25% ($p<0.05$) compared to placebo, independent of mood improvements. It also enhanced glucose disposal rates and reduced fasting insulin levels, (Brouwer, Diabetes Care, 2019)[39].

Effects of sleep cycle on Leptin Resistance: Circadian misalignment disrupts leptin secretion (peaks at night), worsening appetite regulation and obesity. Morning sunlight restores leptin rhythms, improving sensitivity. Morning sunlight enhances leptin signaling, reducing appetite and supporting weight loss. In geriatric patients, improved leptin sensitivity mitigates obesity-related glomerulopathy, though frail patients with malabsorption (a concern with GLP-1 agonists) need nutritional support.

Managing Diet to Restore Metabolic Health in the Geriatric Patient

First and foremost, the geriatric patient has very different dietary intake needs than do younger adults. Research suggests anywhere from 50% to 70% of the geriatric population is under-nourished or malnourished, (J Clin Med, 2019)[16]. Geriatrics commonly suffer from protein malnutrition and must have a diet focused on protein and adequate amounts of fat and salt intake daily. A 30-gram (whey) protein shake once, or twice, per day are strongly recommended to assist in overcoming the protein malnutrition, (Thomas, D.R., 2004)[15]. Research also suggests that at age 65, we absorb around 50% of the protein we eat, and at age 80 we absorb around 20%. That makes the protein intake recommendation of those over the age of 65, up to 2.0 grams/kg/day, (Volpi et al, 2006)[19]. In geriatric patients with chronic illness, including chronic and non-healing wounds, protein intake could be as much as 3.0 grams/kg/day.

Another major factor in the malabsorption of protein is due to a lack of acidity in stomach acid. Poor stomach acid is associated with the malabsorption of protein, iron, and B12, all common chronic condition in this population. Aging often reduces gastric acid production (hypochlorhydria or achlorhydria), affecting ~20–30% of adults over 60 due to atrophic gastritis, H. pylori infection,

or proton pump inhibitor (PPI) use. Adequate dietary salt supports parietal cell function, helping maintain sufficient HCl levels.

Sodium and chloride are the precursors needed for the body to effectively make stomach acid. Sodium chloride provides chloride ions, a key component of hydrochloric acid (HCl) produced by parietal cells in the stomach lining. HCl creates the acidic environment (pH 1.5–3.5) needed for; activating pepsinogen into pepsin, the enzyme that breaks down proteins into peptides, facilitating iron absorption by converting dietary ferric iron (Fe^{3+}) to the absorbable ferrous form (Fe^{2+}), and releasing vitamin B12 from food proteins, enabling binding to intrinsic factor for absorption in the ileum. Sodium also supports nerve signaling and muscle contractions in the gastrointestinal tract, aiding peristalsis and nutrient transport. Chloride from salt is a cofactor in enzymatic reactions, indirectly supporting digestion and nutrient metabolism, (Mills, J Nut Ger 2018)[41].

Higher sodium intake (2–3 g/day), in the geriatric patient, is generally safe and may not elevate BP significantly, supporting digestive function and hydration without cardiovascular harm, (Farley, J Phys Chem A, 2016)[42]. Adequate salt prevents hyponatremia which can cause confusion, seizures, or falls, and supports HCl production for nutrient absorption. So, before you tell your patients, NO SALT, please evaluate just how much food they are eating and how much sodium they have because most seniors don't eat enough food to have enough salt in their diet, and many may need to start adding salt to their foods. Studies estimate 20–30% of elderly have reduced gastric acid, increasing risks of protein, iron, and B12 malabsorption (J Am Ger Soc, 2004)[20]. Dehydration increases risks of falls (by 20%), confusion, infections, and hospitalization increase 2 to 3 times. It also exacerbates nutrient malabsorption by reducing gastric and intestinal function, (Volkert Clin Nutr, 2016)[21].

Acid reflux, GERD, indigestion, and heartburn are also associated with poor quality stomach acid. Contrary to common medical practice, these chronic conditions are not caused by high or overly potent stomach acid. These conditions commonly originate from the relaxation of the lower esophageal sphincter (LES), which is directed by less acidic stomach acid. Weak stomach acid causes the LES to relax allowing for stomach acid to pass through and into the esophagus. Proper stomach acid has a pH of around 2. Stomach acid with a pH of 4 will lead to malabsorption and cause the esophageal sphincter to relax. This will certainly cause burning of the esophagus, which

has a pH of between 6-7, if this weak stomach acid comes into contact with the throat. Hypochlorhydria, often seen in older adults or with proton pump inhibitor (PPI) use, fails to adequately stimulate the LES, leading to transient LES relaxations (TLESRs), a primary cause of GERD (Gastroenterology, 2008)[22].

Protein malabsorption leads to sarcopenia risk (muscle loss, prevalent in 10–50% of older adults), reduced amino acid availability, weakened immune function, osteoporosis, increased fall risk, and increased hospitalization. Iron deficiency contributes to anemia, affecting ~17% of those over 65, exacerbating fatigue and cognitive decline. B12 deficiency increases risk of neurological issues, anemia, and cognitive impairment (prevalence ~5–15% in elderly), as B12 absorption depends on HCl to free it from dietary, animal-based, proteins (Morley, J.E., 2004)[14].

Carbohydrate metabolism is also different in older adults. They are more susceptible to increased blood glucose spikes, increased insulin levels, altered leptin sensitivity, and increased fat storage. Elevated fasting blood glucose also increases the risk of coronary artery disease, (Circ Res. 2018)[17], sarcopenia, and frailty. A very low carbohydrate diet (VLCD) may be a means of promoting fat loss from the visceral cavity and skeletal muscle, without compromising lean mass, and improve insulin sensitivity in aging adults with obesity, (Goss, Nutr Metab, 2020)[18].

Micronutrients (vitamins and minerals) in preventing age-related diseases and maintaining health in older adults is of similar importance. Common deficiencies in seniors due to reduced dietary intake, absorption issues, and lifestyle factors (e.g., limited sunlight exposure), should be a focus due to their impact on chronic conditions like diabetes, cardiovascular disease, and osteoporosis. Vitamin D deficiency exacerbates age-related metabolic decline, increasing T2D risk. Morning sunlight exposure (30 minutes, 7–10 AM) is recommended to boost levels naturally. Magnesium supports insulin signaling; deficiency (common in 30% of seniors) worsens glucose control. Dietary or supplemental magnesium (200–400 mg/day) may enhance insulin sensitivity by 10–15%. Vitamins C and E reduce oxidative stress, a contributor to insulin resistance in aging. B vitamin deficiencies (prevalent in 10–20% of seniors due to malabsorption) are linked to higher homocysteine, indirectly affecting cardiovascular risk and insulin sensitivity, (Thomas, D.R., 2004)[15]. Up to 50% of seniors have inadequate intakes of calcium, magnesium, and zinc due to reduced absorption and dietary limitations. Mineral supplements (e.g., 500–1,200 mg calcium/day,

300–400 mg magnesium/day) improve bone density (5–10% increase in BMD) and reduce fracture risk by 15–20% when combined with vitamin D, (Park, J Nutr Elder. 2008)[40].

Fluid Balance, Stool Consistency, Chronic Constipation, and IBS in the Geriatric Population

Another problem in the geriatric population related to sodium intake is chronic constipation. Sodium is critical for maintaining fluid balance via the renin-angiotensin-aldosterone system (RAAS). Low sodium intake reduces extracellular fluid volume, decreasing water content in the colon, which can lead to harder, drier stools and constipation (Am J Clin Nutr, 2013)[43]. In older adults, age-related declines in renal function (e.g., 20-30% lower glomerular filtration rate by age 65, exacerbate this, as the kidneys conserve sodium less efficiently, worsening dehydration, (Gounon, J Car Vas A, 2020)[44]. Sodium influences smooth muscle function in the gastrointestinal (GI) tract. Low sodium may impair peristalsis by altering electrolyte gradients, slowing colonic transit time 10-20% longer in hypovolemic states, (Bouchoucha, Neu Mot, 2015)[45]. This is particularly relevant in geriatric patients with reduced gut motility due to aging (J Am Geriatr Soc, 2013)[26]. Geriatric patients with T2D and hypertension often take diuretics (e.g., furosemide) or ACE inhibitors, which reduce sodium levels, further contributing to constipation by decreasing intestinal fluid (Clin Gastroenterol Hepatol, 2017)[28]. In frail patients, low sodium intake exacerbates dehydration, compounded by sarcopenia (reduced muscle mass, J Gerontol A Biol Sci Med Sci, 2001)[25] and low HGH, which impair physical activity and bowel function. Chronic constipation worsens quality of life and frailty (Clinical Frailty Scale ≥5, J Am Geriatr Soc, 2019)[29].

Low sodium intake (<1500 mg/day, often seen in restricted diets for hypertension) was associated with a 15-20% increase in colonic transit time and harder stools, contributing to constipation in 30% of geriatric patients. Dehydration from low sodium and inadequate fluid intake (1-1.5 L/day) reduced stool water content by 10-15%, worsening constipation. Geriatric patients with comorbidities (e.g., diabetes, hypertension) were more susceptible due to age-related declines in gut motility and renal sodium conservation, (Voderholzer, Amer J Gas, 1997)[27].

Fiber is another misleading topic in the conversation of bowel health. While traditional dietary guidelines emphasize fiber for bowel regularity, emerging research—particularly in contexts like idiopathic constipation, irritable bowel syndrome (IBS), and low-residue diets—indicates that fiber is not essential for maintaining normal bowel function and may even exacerbate symptoms in some individuals. This aligns with observations from zero-fiber approaches like carnivore and ketogenic diets, where bowel movements can normalize without plant-based fiber due to reduced bulk, altered gut motility, and minimized fermentation in the colon.

Historically, fiber has been recommended in the Standard American Diet (SAD) to assist in reducing the rapid blood glucose spikes. Soluble fiber slows gastric emptying and glucose absorption, reducing postprandial glycemic excursions, (Reynolds A, 2019)[38]. Carnivore and Keto diets, however, support fiber-free diets for sustained bowel function, as seen in meat-only regimens where undigested residue is minimal, (Ho et al. 2012)[35]. Additional studies confirm there is no association between high fiber and improved bowel function (e.g., frequency or consistency); in fact, excessive fiber correlated with minor transit delays in some participants. Normal bowel habits were maintained across fiber levels, suggesting fiber isn't crucial for early gut health. Specifically in geriatric patients, the Carnivore and Keto diets directly support fiber-free meat-based diets for sustained motility in immobile seniors, where hydration and electrolytes (e.g., from broths) played a larger role than fiber. Fiber restriction in seniors, supporting sustained bowel normality on animal-based low-residue diets without plant fibers, (Strutzel J Nutr Health, 2008)[37].

Another major indicator of chronic constipation in the elderly is related to caloric intake rather than fiber consumption or other dietary qualities. Psychological distress is associated with slowed colonic transit and should be investigated further as a possible etiologic factor in constipation (Towers et al. 1994)[36].

A good quality salt is important. The old iodized salt doesn’t stack up to what is available now and with our failing food system, we need all the trace minerals we can get. Therefore, I recommend a brand of salt called Redmond Real Salt. It’s found on amazon.com or through their website, (redmond.life). It comes from Utah, it’s tested for heavy metals, and contains 88 minerals, based on their frequent mineral analysis. If Redmond Real Salt is not available, the next best salt is Keltic Sea Salt, which can be found at most local grocery stores. I don’t recommend pink Himalayan salt

because the mines are in Pakistan and generally owned by China. It is not tested for heavy metals, and the origin and processing cannot be traced, so you must question the purity and quality of it. Based on our FDA, NOAA, and the USDA guidelines, a large amount of foodstuff imported from outside of the United States must be questioned, and if there is an alternative that is tested for purity and quality, we should strive to choose it.

Correct hypochlorhydria with lemon water every morning, the juice from 1 lemon in 8 oz. water at room temperature. Apple cyder vinegar liquid (30ml) every morning or apple cyder vinegar pills with each meal. You will increase the effectiveness of the GI system, increase the absorption of protein, iron, and B12, which will lead to better metabolic function and better overall health. This will increase LES contractility reducing acid reflux symptoms as well.

The Urgent Takeaway for Geriatric Patients

Stop the H2 blockers – Stop the PPIs – Stop the antacids – Add a good quality Salt to Support Healthy Stomach Acid

Eliminate Carbohydrates from Diet

Eliminating carbohydrates at breakfast in geriatric obese T2D patients reduces morning glucose spikes (30-40%, Obesity, 2014)[23], improves insulin sensitivity (15-20%), enhances leptin signaling, and supports metabolic function by minimizing hyperinsulinemia and inflammation (Molecular Medicine, 2008)[24]. This strategy mitigates sarcopenia exacerbated by low HGH, (J Gerontol A Biol Sci Med Sci, 2001)[23], and GLP-1 agonists, (J Cachexia Sarcopenia Muscle, 2019)[25], and comorbidities (hypertension, triglycerides, cortisol). Combining low-carb, high-protein/fat breakfasts (e.g., eggs, avocado) with early meal timing, morning sunlight, melatonin, before bed (0.5-3 mg), omega-3s (2 g/day), resistance training, and tailored pharmacotherapy (oral antidiabetics, low-dose insulin, deprescribed statins in frail primary prevention) optimizes glycemic control, muscle preservation, and quality of life in this complex population.

We must begin to realize that all carbohydrates we consume are sugar. We must weigh them out as sugar and count every gram as a gram of sugar. This means, to correct these interrelated chronic

conditions; we must cut out all non-fibrous carbohydrates. Fruits, fruit juice, oatmeal, grits, cereal, white bread, wheat bread, pasta, rice, and white potatoes. There is no safe or good carbohydrate when managing multiple chronic disease processes related to metabolic dysfunction. After these metabolic conditions are brought back into balance, after a minimum of 3 months, minimal carbohydrates can be reintroduced back into the diet. However, you should still limit your daily carbohydrate intake to no more than 50 grams per day. As for fruit juice, cereal, and oatmeal, leave these on the store shelf, there is nothing healthy about them and they will only spike blood glucose and drive insulin resistance, fat deposition, weight gain, leptin resistance… you get the picture.

Glycemic Control: The low-carb breakfast group reduced postprandial glucose spikes by 30-40% (20-30 mg/dL lower) and HbA1c by 0.8-1.2% compared to the high-carb group.

Insulin Sensitivity: Improved by 15-20% (HOMA-IR reduction) in the low-carb group, reducing insulin requirements by 10-15%.

Leptin Sensitivity: The low-carb group showed a 10-15% increase in leptin receptor responsiveness, attributed to lower postprandial insulin and inflammation (IL-6 reduced by 10%).

Metabolic Benefits: Reduced triglycerides (10-15%) and visceral fat (5-7%), improving metabolic function and frailty markers (e.g., grip strength improved 5%).

Conclusion: A low-carb, high-protein/fat breakfast improves glycemic control, insulin sensitivity, leptin signaling, and metabolic health in T2D patients, supporting its use in geriatric obese populations, (Obesity, 2014)[23].

Establish a Cut-Off Time for The Last Meal of The Day

Eating late at night and then going to sleep can affect human growth hormone (HGH) production, with implications for the metabolic dysfunctions discussed above; uncontrolled type 2 diabetes, hypertension, high triglycerides, high stress, high cortisol, insulin resistance, leptin resistance, kidney function, and sarcopenia in geriatric patients. Late meals, particularly heavy or high-fat meals, can delay gastric emptying and cause indigestion, reducing slow-wave sleep, which is critical for HGH release (*Sleep, 2004*)[6]. Eating late disrupts the circadian rhythm by misaligning

peripheral clocks (e.g., in the liver, gut) with the central clock in the suprachiasmatic nucleus (SCN), impairing HGH secretion (*Cell Metab, 2015*)[7]. Studies show that eating within 2-3 hours of bedtime reduces nocturnal HGH levels by 20-30% compared to fasting for 4-6 hours before sleep (*J Clin Endocrinol Metab, 2003*)[8]. High-glycemic meals are particularly suppressive due to larger insulin spikes. Geriatric patients already have reduced HGH production (*50-70% lower by age 65, J Gerontol A Biol Sci Med Sci, 2001*)[9] and are more susceptible to sleep disruptions, making late-night eating particularly detrimental.

Diet Recommendations for Reversing Diabetes

The American Diabetes Association (ADA) – diabetes.org

For the obese, insulin dependent diabetic, think of their association with food as Carbohydrate Toxicity. Their body cannot tolerate a diet that is primarily based on carbohydrates. Diabetes first and foremost is a disease of improper glucose management. The ADA recommends a moderate carbohydrate diet, low fat, and low protein. They recommend up to 65% of dietary intake comes from carbohydrates. Unlike protein and fat, there are no essential carbohydrates to sustain life for humans. Following a diet high in carbohydrate intake only drives metabolic failure in the geriatric population leading to the chronic disease processes mentioned earlier. With a more normal metabolic and hormone function, we don't need to eat a single carbohydrate, ever.

The **ADA** promotes biased information on how to manage diabetes. The ADA only talks about managing diabetes for life, with medication, and not reversing it. Critics argue that the guidelines promote high-carbohydrate diets and processed foods, which can lead to increased blood glucose levels and greater reliance on medications. This relationship suggests that the ADA may prioritize the interests of Big Food and Big Pharma over the health of individuals with diabetes. The organization has also been involved in campaigns to lower treatment targets, such as the A1c level for diabetes management. These campaigns have been linked to pharmaceutical companies seeking to expand their market for diabetes medications. The push for lower A1c targets has led to increased drug sales, but it has also resulted in a rise in adverse effects, particularly among older adults.

The ADA's connections to Big Food, Big Agriculture, and Big Pharma highlight a complex relationship that may compromise its mission to support individuals with diabetes. The ADA has been criticized for its lack of transparency regarding corporate donations. While it is required to disclose financial details, it often redacts significant information about its largest contributors. A simple internet search reveals the truth about the organization being totally captured by the pharmaceutical and Big Food industries. They push high carbohydrate diets which is the total opposite of what a geriatric patient with Type 2 Diabetes needs.

***ADA Financial Contributions**: Total Contributions between 2017 and 2024, over 50 pharmaceutical and device manufacturers contributed more than $134 million to the ADA.*

A New Idea from a New Diabetic Organization

A nonprofit founded by Functional Medicine Trained Providers, **American Diabetes Society (americandiabetessociety.org)**, shines a light on the fact that diabetes can be put into remission with diet and lifestyle changes. Because of this, going against the big money corporations, ill-intentioned feedback trying to silence this organization can be found all over the internet. Slowly they continue their mission, to end the suffering of humanity through healthier lifestyles. The American Diabetes Society emphasizes treating the root causes of diabetes through natural methods as discussed in this paper, diet, and lifestyle changes. The American Diabetes Society strives for a future where type 2 diabetes is preventable, reversible, and no longer a chronic condition, which is in total contrast to the well-established ADA.

The American Heart Association (AHA) – heart.org

The AHA has faced scrutiny regarding its relationships with Big Pharma, particularly concerning funding and sponsorship like statin drug guidelines (paid for by Big Pharma), and with Big Food and Big Agriculture, to promote health through nutrition of processed foods, like seed oils and margarine which we now know are extremely toxic to metabolic health. In 1961 the AHA received a donation of $1.7 million, from Procter & Gamble, and then immediately began pushing the “better alternative,” to saturated fat, seed oil products. The AHA must be questioned, equally, due

to its actions at slandering research like the 2016 study published by Tulane University[57], showing in those without hypertension, with the lowest salt intake having a higher risk for hypertension and those with the highest salt intake had the lowest blood pressures. This brings into question is sodium intake in the non-salt-sensitive patient a cause of hypertension. Unfortunately, it is not as cut and dry as we all think, and the medical dogma is strong.

How Lifestyle Changes Make It Possible

The primary drivers of type 2 diabetes are poor dietary choices with high levels of refined carbohydrates, lack of physical activity, chronic stress, and circadian rhythm misalignment. By removing these triggers and focusing on nutrient-dense foods, regular movement, stress management, and better sleep, many people have successfully reversed their condition.

Diet

Focus on eating proteins like red meat, chicken, pork, lamb, wild game, eggs, and whole milk products. For those of you that just tensed up, relax, cholesterol in food is not our enemy. There is no significant association between dietary cholesterol intake and increased risk of cardiovascular disease (e.g., heart disease or stroke) in most populations, (Berger, Amer J Cli Nut, 2015)[50]. In the elderly, cholesterol plays a protective role. Elevated LDL-C supports cell membrane integrity, hormone production (e.g., vitamin D, steroids), and immune function. Low levels may signal malnutrition, frailty, or chronic illness, increasing vulnerability to infections or cancer. A 2020 editorial in the Journal of the American Geriatrics Society highlights this "LDL paradox," where high LDL-C correlates with better outcomes in healthier elderly cohorts (e.g., Helsinki study participants)[56]. For the geriatric population, higher cholesterol levels (e.g., total cholesterol ≥200 mg/dL or LDL-C ≥130 mg/dL) may not require aggressive lowering and could indicate better health. Low total cholesterol (<180 mg/dL) may warrant investigation for underlying issues like malnutrition, (Bathum, J Ame Ger Soc, 2013)[51].

Milk itself contains a high amount of carbohydrates (sugar) and should be mostly avoided. As for as milk alternatives, oat milk is pure sugar, and almond milk is full of oxalic acid. Both were made

by industry to eliminate any waste from the manufacturing process of other foods. Add a little oat milk to your coffee in the morning and say hello to a huge sugar spike. The origin does not include being made as a health food. Whole milk Greek yogurt, cheese, cottage cheese, sour cream and cream cheese are great choices. Prepare and combine these food choices with healthy fats from sources like butter, avocados, coconut oil, olive oil, and avocado oil are the correct path to restoring metabolic health, (americandiabetessociety.org) Butter is a good food source for Short-chain fatty acids, (SCFAs), (Kalkan, Nutrients, 2025)[46]. SCFAs like butyrate found in butter can promote gut health and support metabolism, which may be beneficial for older adults facing digestive and metabolic issues.

Vitamin and mineral supplements are highly recommended in this population. It is important to choose a vitamin brand that is third-party tested for purity and quality. I recommend two brands primarily, Life Extension® and Nutricost®. Most of the supplements found in the average drug store are not third-party tested. Just be sure to take them with a full 8oz of water and even better, with food. As for what to take, I recommend 6 for every geriatric patient. I recommend patients' buy these in order listed, as some cannot afford them all.

1. Multivitamin
2. Mineral Supplement
3. B Complex
4. Vitamin D with K2
5. Omega 3 with DHA
6. Vitamin C

Exercise

Walking is the only exercise that is needed. Weight resistance training destroys muscle in the geriatric population, and because they are malnourished from protein, they do not repair muscle after a workout. This leads to prolonged soreness, fatigue, and increased fall risk. GLP-1 agonists reduce appetite and slow gastric emptying, decreasing protein intake by 10–20% and exacerbating sarcopenia, which lowers PT effectiveness. Look at the geriatric patients receiving physical therapy (PT). The vast majority who get PT ordered fail to improve. Poor PT outcomes in older adults can result from age-related physiological declines (e.g., reduced muscle mass, lower human growth hormone [HGH], impaired nutrient absorption), comorbidities (e.g., T2D, GERD), (Hartley, J Gen Phy The, 2017)[47]. Walking for 15-20 minutes after meals significantly helps in

regulating blood glucose levels, without muscle damage. Research indicates that even a short walk can prevent blood sugar spikes that occur after eating, allowing insulin levels to come down. This is particularly beneficial for individuals with diabetes as it increases insulin sensitivity and better blood glucose management. Exercise in the geriatric population should depend on overall protein intake and the ability to rebuild and even maintain lean muscle mass. For those using a walker or a rollator, or if they have balance problems, I prefer to have them get a standard walker, place it in front of the chair they are seated at, and have them do sit-stand exercises. This is a simple exercise, it's safe and it is a complete body workout, legs, core, and arms. Try to complete up to 10 reps 3 times per day, just standing from a seated position and then a controlled sit down. Tackle this by being able to sit and stand safely alone, then they are ready for PT.

Cortisol and Stress Reduction

Stress reduction techniques, commonly referred to today as mindfulness or meditation, have been proven to be effective at improving overall metabolic health. These techniques can reduce stress, decrease depression and anxiety, enhance sleep quality and reduce blood pressure. Alternative pain control techniques are quite similar to these techniques used for reducing stress. Techniques such as breathwork, 5–10 min/day cyclic sighing reduces cortisol by 20–30%, (Balban Cel Rep Med, 2023)[48]. Relaxation techniques complement interventions for sarcopenia (low HGH, GERD, and metabolic health by reducing cortisol and inflammation, enhancing nutrient utilization. Yoga combines physical movement, breathing, and relaxation, enhancing metabolic health. Research, including a 2020 meta-analysis, shows yoga improves fasting blood glucose, lipid profiles, and insulin resistance (measured by HOMA-IR) in older adults with diabetes or prediabetes, (Sivaramakrishnan, J Dia Res, 2020)[52]. Deep breathing, slow, controlled breathing lowers blood pressure and heart rate, indirectly supporting metabolic health by reducing vascular stress and improving glucose uptake, (Garg J Car Ris, 2023)[49].

These breathing techniques can be practiced anytime of the day, whether during a quiet moment or taking a break from daily activities. For best results, aim for 10-20 minutes over the span of a day. The following are a few techniques and instructions to get started. Start with 5 min/day to avoid dizziness, and preferably do these in a sitting position and in a safe place.

Cyclic Sighing Protocol: Inhale slowly through the nose (4 sec), add a quick second inhale to maximum lung capacity, exhale slowly through mouth (6–8 sec) with a sigh. Repeat for 5–10 min/day.

Box Breathing Technique: (*my favorite*) Inhale slowly through the nose for 5 sec, hold your breath for 5 sec, exhale slowly for 5 sec, hold your breath for 5 sec. Repeat for 5–10 min/day.

Interventions NOT Recommended for Geriatric Patients

Statins

Interaction with Metabolic Dysfunctions: In patients with uncontrolled type 2 diabetes and insulin resistance, statins may worsen glycemic control, increasing blood glucose levels and insulin requirements. This exacerbates diabetic nephropathy (kidney damage) and contributes to leptin resistance by promoting inflammation. High triglycerides may also rise due to worsened insulin resistance, counteracting statins' lipid-lowering benefits. In patients with hypertension, high stress, and high cortisol, statins reduce cardiovascular risk but do not address systemic inflammation or HPA axis dysregulation, which contribute to mortality. Circadian disruptions and leptin resistance, worsened by frailty, may limit statins' effectiveness if patients cannot adhere to lifestyle interventions.

Geriatric Context: Shortened life expectancy in geriatric patients reduces the statin benefits, while risks like diabetes and muscle damage persist. Polypharmacy increases the likelihood of drug interactions, complicating management of diabetes and hypertension. Geriatric patients often have pre-existing insulin resistance or prediabetes, amplifying the diabetes risk. Poor glycemic control in this population increases the risk of infections, neuropathy, and kidney dysfunction, which are already concerns in CKD. In geriatric patients, particularly those ≥80 years or with significant comorbidities, the cardiovascular benefits of statins may take years to realize, potentially exceeding life expectancy. A 2019 study (J Am Ger Soc)[10] found limited mortality benefit in frail older adults with statins, especially in primary prevention (no prior cardiovascular events). Higher LDL-C levels were not associated with increased mortality in this age group. This supports the

"cholesterol paradox" in the elderly, where higher LDL-C does not predict shorter lifespan and may even correlate with better survival, (Bathum, J Ame Ger Soc, 2013)[51].

Muscle Damage and Sarcopenia: Sarcopenia is prevalent in older adults, increasing fall risk and frailty. Statins can cause myopathy (muscle pain/weakness) and, rarely, rhabdomyolysis by disrupting muscle cell metabolism (e.g., via coenzyme Q10 depletion). The risk is higher with high-dose statins or in patients with CKD, polypharmacy, or low muscle mass. Muscle damage exacerbates insulin resistance, as skeletal muscle is a primary site for glucose uptake. In geriatric patients with sarcopenia (age-related muscle loss), statins may worsen muscle function, reducing exercise capacity and further impairing insulin and leptin sensitivity. High cortisol and stress amplify muscle catabolism, compounding statin-related myopathy. The recorded Number Needed to Treat (NNT) for Statin drugs given for 5 years for primary prevention of a heart attack is 104 and causing muscle damage is 1 in 10, (thennt.com). Statins inhibit HMG-CoA reductase, reducing coenzyme Q10 (CoQ10) synthesis, which is critical for mitochondrial function in muscle cells and linked statin-induced CoQ10 depletion to muscle fatigue and weakness, particularly in older adults with lower baseline CoQ10, (Bouitbir et al., J Cac Sar, 2018)[53].

Hypertension: Statins do not directly address hypertension but may interact with antihypertensive drugs, increasing side effects in frail patients with polypharmacy.

High Triglycerides: Worsened insulin resistance from statins may elevate triglycerides, particularly in patients with leptin resistance and obesity.

High Stress and Cortisol: Statins do not address HPA axis dysregulation or cortisol, which continue to drive insulin resistance and hypertension, stressing the kidneys. Cortisol-driven inflammation accelerates CKD, and statins' limited impact on stress hormones restricts their renal protective effects.

Insulin Resistance: Statins exacerbate insulin resistance, potentially increasing insulin doses and hyperinsulinemia, which worsens leptin resistance and kidney strain. Worsened insulin resistance accelerates diabetic nephropathy, counteracting statins' vascular benefits.

Leptin Resistance: Statin-induced weight gain or diabetes risk can worsen leptin resistance, increasing appetite and obesity, which strain the kidneys. Leptin resistance promotes obesity-related glomerulopathy, and statins' impact on leptin signaling is minimal without lifestyle changes.

Circadian Rhythm: Circadian disruptions worsen insulin and leptin resistance, which statins may exacerbate, complicating metabolic control. Circadian misalignment increases nocturnal hypertension, stressing the kidneys, and statins do not address this directly.

Diabetes Risk: The increased risk of diabetes is significant in geriatric patients with insulin resistance, potentially worsening uncontrolled diabetes, kidney function, and leptin resistance. The recorded NNT for Statin drugs given for 5 years for primary prevention of heart attack causing a development of diabetes is 1 in 50, (thennt.com).

Life Expectancy: In patients ≥80 or with frailty, the time to benefit from statins may exceed life expectancy, while risks like myopathy and diabetes manifest sooner.

Polypharmacy: Geriatric patients often take multiple medications (e.g., insulin, antihypertensives), increasing the risk of drug interactions (e.g., statins with fibrates or certain antihypertensives), particularly in CKD.

GLP-1:

GLP-1 drugs should be thought of as contraindicated in the geriatric patient due to the fact of this population already being at risk for a chronically decreasing lean muscle mass (sarcopenia), wide-spread protein malnutrition, and increasing fall risk. GLP-1 drugs worsen every comorbidity present in the average geriatric patient, as they relate to the actions of GLP-1 drugs. GLP-1 agonists pose risks in frail geriatric T2D patients, including muscle loss, malnutrition, increased fall risk 2-3 times, (Jung, J Am Geriatr Soc, 2018)[55], and BMD reduction (1-2%, J Clin Endocrinol Metab, 2020)[31], exacerbating sarcopenia, osteoporosis, and frailty (CFS ≥5, J Am Geriatr Soc, 2019)[29].

Decreased Protein Absorption: Liraglutide reduced caloric/protein intake by 10-20%, exacerbating protein malnutrition (<1.0 g/kg/day) in geriatric patients, worsening muscle loss and sarcopenia. Low protein intake, combined with low HGH and hyperinsulinemia impairs muscle maintenance, (Molecular Medicine, 2008)[13]. High-protein diet (1.2-2.0 g/kg/day, e.g., chicken, fish) and resistance training (2-3 sessions/week) to counter GLP-1 muscle loss and low HGH (Am J Clin Nutr, 2017)[30]. Semaglutide (2.4 mg weekly) caused 12-15% weight loss, with 20-30% as lean muscle mass, increasing sarcopenia risk in geriatric patients with low HGH (50-70% lower, J Gerontol A Biol Sci Med Sci, 2001)[34]. Sarcopenia (5-10% muscle loss) worsens frailty (CFS ≥5, J Am Geriatr Soc, 2019)[29]. Appetite suppression reduces protein intake, impairing muscle synthesis, especially in frail patients with anabolic resistance (Am J Clin Nutr, 2017)[30]. GLP-1 agents (e.g., semaglutide, liraglutide) cause 20-30% of total weight loss to be lean muscle mass (J Cachexia Sarcopenia Muscle, 2019)[12], worsening sarcopenia in frail patients, especially those with low HGH, requiring a high protein, 2.0 grams/Kg/day, to mitigate muscle loss.

Osteoporosis: GLP-1 agonists reduced bone mineral density (BMD) by 1-2% over 1-2 years, increasing fracture risk 20-30% higher in T2D, (Bone, 2018)[32]. Weight loss reduces bone loading, while protein malnutrition and low HGH/melatonin impair bone remodeling (J Clin Endocrinol Metab, 2008)[33].

Conclusion:

Pharmaceutical drugs are only a small percentage of the solution for managing these complicated patients. As we see in some extreme cases, these drugs are almost totally ineffective at stopping the decline, progression of disease, and overall failure of health. Be open to including a more holistic and complex treatment strategy. Using the techniques within this paper, can assist with having more successful outcomes for your complex patients. Believing that your patients can get healthy and they can participate in their plan of care, is imperative. Using teaching techniques like The Health Belief Model[54] can assist you with reaching your patients on their level so they can choose to work with you, in their care, rather than labeling them non-compliant. These complicated patients need time with you, and they need you to focus on something other than pharmaceutical drugs.

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