Scientific Support for Chapter 13:

What Every Diabetic and "Pre-Diabetic" Needs to Know!

Kolata, Gina, "Research Questions Benefit of Low-Salt Diet, Drawing Criticism From C.D.C.," May 4, **2011**, page A17:[1]

- "A new study found that **low-salt diets** ***increase the risk*** of death from heart attacks and strokes and ***do not prevent high blood pressure***...
- "The investigators found that the **less salt** people ate, the **more likely they were to die** of heart disease...
- "'If the goal is to prevent hypertension' with lower sodium consumption, said the lead author, Dr. Jan A. Staessen, a professor of medicine at the University of Leuven, in Belgium**, 'this study shows it does not work.'**
- "...But, Dr. Alderman said, the new study is not the only one to find **adverse effects of low-sodium diets**. His own study, with people who had high blood pressure, found that **those who ate the** ***least salt*** **were** ***most likely to die***.
- "...***Lowering salt consumption***, Dr. Alderman said, has consequences beyond blood pressure. It also, for example, ***increases insulin resistance***, which can increase the risk of heart disease."

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1 Kolata, Gina, "Research Questions Benefit of Low-Salt Diet, Drawing Criticism From C.D.C.," May 4, **2011**, page A17.

The New York Times, **Gary Taubes, "Salt, We Misjudged You," June 3, 2012, pages 7-8":**[2]

- "Salt consumption **is said to** raise blood pressure, cause hypertension and increase the risk of premature death. This is why the **Department of Agriculture's** dietary guidelines still **consider salt Public Enemy No. 1**, coming before fats, sugars and alcohol. It's why the director of the **Centers for Disease Control and Prevention** has **suggested that reducing salt consumption is as critical** to long-term health **as quitting cigarettes**.

- "'You can say without any shadow of a doubt,' as I was told then by Drummond Rennie, an editor for *The Journal of the American Medical Association*, that the ***authorities pushing the eat-less-salt message*** had "made a commitment to salt education that goes ***way beyond the scientific facts***."

- "A 1972 paper in *The New England Journal of Medicine* reported that the **less salt** people ate, the higher their levels of a substance secreted by the kidneys, called *renin*, which set off a physiological cascade of events that seemed to end with an **increased risk** of heart disease. In this scenario: ***eat less salt*, secrete more renin, get heart disease, *die prematurely***.

- "When several agencies, including the Department of Agriculture and the Food and Drug Administration,

2 "Salt, We Misjudged You," June 3, **2012**, pages 7-8.

held a hearing last November to discuss how to go about getting Americans to eat less salt (as opposed to whether or not we should eat less salt), ***these proponents argued that the latest reports suggesting damage from lower-salt diets should simply be ignored***." [Note: Once again, we see opinion trumping valid medical science.]

Garg, R., et al., *Metabolism: clinical and experimental***, 60 (2011), 965-968, "Low-salt diet increases insulin resistance in healthy subjects":**[3]

- "Low dietary salt is recommended as one of the public health measures to decrease risk of cardiovascular disease. ***However, low salt intake stimulates aldosterone production*** ...We recently demonstrated an association between **aldosterone** and ***insulin resistance*** in healthy patients.
- "**Our study shows that low salt intake is associated with higher IR (insulin Resistance**)."

***The New York Times*, Kolata, Gina, October 19, 2012, page A17,** "Diabetes Study Ends Early With a Surprising Result":[4]

- "A large federal study of whether diet and weight loss can prevent heart attacks and strokes in overweight and obese people with Type 2 diabetes has **ended two years**

3 Garg, R., et al., *Metabolism: clinical and experimental*, 60 (**2011**), 965-968.

4 *The New York Times*, Kolata, Gina, October 19, **2012**, page A17.

ahead of schedule because the intensive program did not help.

- "...Like many, she had **assumed diet and exercise would help** ... "The study **randomly assigned 5,145** overweight or obese people with Type 2 diabetes to either a rigorous diet and exercise regimen or to sessions in which they got general health information. The diet involved 1,200 to 1,500 calories a day for those weighing less than 250 pounds and 1,500 to 1,800 calories a day for those weighing more. ***The exercise program was at least 175 minutes [nearly 3 hours] a week of moderate exercise***.

- "But **11 years after** the study began, researchers concluded it was **futile to continue —** the two groups had **nearly identical rates of heart attacks, strokes and cardiovascular deaths**."

Diabetes induced Eczema and dry skin take a hike...:[5]

- "**Dermatitis** is consistently the first sign of EFA [PEO] deficiency in both animals and humans. **Itch** is the symptom that seems to respond most to GLA [an omega-6 derivative]. **There is also a significant reduction in the need for potentially harmful steroids**.

- "It was first reported in the **1950**s that **diabetic animals** ***required much more*** **linoleic acid [Parent omega-6]**

5 Horrobin, David, F., "Fatty acid metabolism in health and disease: the role of Δ6-desaturase," *American Journal of Clinical Nutrition*, *American Journal of Clinical Nutrition*, 1993:57(suppl):732S-737S.

than did normal animals. Linoleic acid [Parent omega-6] concentrations are almost always normal or slightly above normal in **diabetic patients**, whereas the concentrations of linoleic acid **metabolites are consistently below normal**.

▶ "Perhaps the main problem in the management of diabetes is the development of **long-term damage to the retina, the kidneys, the cardiovascular system, and the peripheral nerves**. Although there are many hypotheses, none have found universal acceptance and treatment is generally unsatisfactory. Good control of blood glucose may be beneficial, **but many well-controlled diabetics develop severe complications** whereas some poorly controlled diabetics do not. [Note: good control alone is insufficient as clearly demonstrated in recent studies like ACCORD presented at the end of this section.]

- "It is possible that the **increased requirement for EFAs is an important factor in the development of diabetic complications**. Neurophysiologically detectable ***damage to nerve function occurs in more than 90% of diabetics***. **The neuropathy leads to many further complications** including skin ulceration, limb amputation, impotence, and bladder."

Dr. Horrobin continues in another medical journal article:

- "There are ***multiple abnormalities of EFA-eicosanoid metabolism in diabetes*** because 5-desaturation is also impaired and there is a block in the conversion of DGLA

to PGE1. Because of this impaired 6-desaturation, **diabetics require higher amounts of EFAs than non-diabetics**. There have been several successful attempts to manage diabetic complications by the provision of very high levels of linoleic acid [Parent omega-6] intake.

▶ "These have shown convincingly that the development of ***cataract***, of ***retinopathy*** and of **cardiovascular damage** can all be ***slowed or stopped*** by the administration of large **daily doses of LA [Parent omega-6]**.

• "Of all the complications of diabetes, the **nerve damage** (**neuropathy**) seemed the one most likely to respond to GLA [**Parent omega-6 derivative**] treatment. This is because EFAs are required for neuronal structure, because second messengers derived from EF As such as PGE1 or diacylglycerol are required for normal neuronal function, and because nerve **microcirculation is also important in nerve function**. **Stiff red cells, such as might result from reduced amounts of EFAs in the membranes**, would impair nerve circulation and lead to a **reduced supply of oxygen** and nutrients.

▶ "**No one reviewing this literature** can have any doubts that GLA [Parent omega-6 derivative] is a **potent treatment** for experimental **inflammatory and auto immune disorders**…."

Controlling blood glucose alone (even with tight control of BP and cholesterol) wasn't sufficient, as this quote from *The New England of Medicine* publishing the seminal ACCORD study states:[6]

> "**Medical experts were stunned**. '**It's confusing and disturbing that this happened**,' said Dr. James Dove, president of the American College of Cardiology. 'For 50 years, we've talked about getting blood sugar very low. Everything in the literature would suggest this is the right thing to do,' he added. Among the study participants who were randomly assigned to get their blood sugar levels to nearly normal, ***there were 54 more deaths than in the group whose levels were less rigidly controlled***. The patients were in the study for an average of *four years* when investigators *called a halt* to the intensive blood sugar lowering and put all of them on the less intense regimen."

Dr. Horrobin has more to say:[7]

> "*Diabetes.* The decline of 6-desaturation in animal models of diabetes has been repeatedly and thoroughly documented both *in vitro* and *in vivo*. ***In human diabetics, the fatty acid compositions of plasma, serum, red cells and platelets are also consistent with impaired formation of GLA*** **[Parent omega-6 derivative]**. The

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6 *The New England Journal of Medicine*, 2008; 358:2545-2559.

7 Horrobin, D.F., "Nutritional and medical importance of gamma-linoleic acid," *Prog. Lipid Res.*, Vol. 31, No. 2, pages 163-194, 1992.

low levels of unsaturated fat in blood in diabetes were actually noted as long ago as 1928! Administration of insulin to human diabetics changes the plasma fatty acid composition in a manner consistent with stimulation of 6-desaturation."

Cellular insulin Sensitivity Increases:[8]

"EPO (Evening Primrose oil) [**Parent omega-6 and GLA**] feeding for 4 months **significantly reduced erythrocyte membrane microviscosity** in the diabetic patients. EPO feeding also **dramatically increased the prostaglandin E_1** (PGE_1) on diabetic erythrocyte membranes to normal levels.

"We have also shown that **prostaglandin E_1** (PGE_1) receptor in human erythrocyte **membrane positively modifies the insulin effect on membrane microviscosity by lowering the physiological concentration of insulin needed** to produce a given effect.

► "…[E]rythrocyte membranes prepared from diabetic patients **show only 42% of the PGE_1 binding activity found in controls."**

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8 Dutta-Roy, Asim, "Effect of Evening Primrose Oil Feeding on Erythrocyte Membrane Properties in Diabetes Mellitus," *Omega-6 Essential Fatty Acids: Pathophysiology and Roles in Clinical Medicine*, 1990, pages 505-511 (out of print).

"The Gracey HYPO-thesis" *for the CAUSE and CURE of Diabetes...*

After studying the field for over a decade, I have come to the conclusion that "Diabetes is caused by ***eating high glycemic foods too*** often which reduces **brain/nerve insulin production**. Glucose entry is increased into [and storage in] any 'peripheral tissues' having insulin receptors. Overeating of carbohydrates (*glycemic* insulin-generating foods) is a main cause of the worldwide-obesity epidemic. **Prof. Peskin's PEO discovery helps patients decrease their carbohydrate craving and cravings for sweets**, along with **improving cell membrane functionality so LESS INSULIN is REQUIRED.** It was previously thought that the brain was insulin-insensitive. This was wrong, very wrong. Neural cells require it.

Relative **hypoglycemia is** caused by eating too often—especially meals relatively high in *glycemic* [raising blood glucose] carbohydrates. ***Neuroglycopenia*** is a shortage of glucose (glycopenia) in the brain, usually ***due to hypoglycemia*** (a "relative" glucose low)**.** When anyone eats, especially carbohydrates/glucose, the bloodstream becomes flooded with it. As a supra-physiologic response, the pancreas releases insulin in response to increasing concentrations of glucose. Insulin forces glucose into [and storage in] many 'peripheral tissues.'

As a result, *blood glucose concentration drops*, often resulting in *relative* hypoglycemia. *Relative* hypoglycemia results in **brain & nerve cells being deprived** of the glucose that has been 'drained' out of the bloodstream, by the insulin, for storage and use in other tissues.

► This is why the typical very poor "high/low" glycemic control in patients is disastrous. For example, a 300 mg/dl reading dropping to 120mg/dl is a definite "relative low."

In an effort to allow sufficient glucose to the brain, the body compensates, by becoming '*insulin resistant*'—the body is protecting neural synapses—but at a large price. With this mechanism, there is now a higher glucose concentration remaining in the bloodstream to help better supply the glucose-fuel needs of the brain & nerves. I term that process ***'compensatory hyperglycemia.'*** Whenever anyone trying to prevent relative hypoglycemia consumes excess glycemic carbohydrates, their blood glucose concentration rises temporarily, to an above-the-national-average concentration [transient supernormal glycemia *aka* TSG] in order to help prevent relative-hypoglycemia particularly in **type 1/ type 2** diabetics.

When anyone, especially children, eats too often, the **pancreatic beta-cells** become **'inflamed'** [less functional] in order to help **reduce insulin production**

and increase 'compensatory hyperglycemia' attempting to best protect the brain/nervous system [e.g., type 1A diabetes]. **Eating less OFTEN** increases 'localized' brain/nerve insulin production ['fine-tuning' of brain/nerve glucose metabolism] without the typically associated blood glucose "lows."

Therefore, the CURE for diabetes is to eat less OFTEN—along with optimum PEO consumption— ensuring maximum cellular insulin response. This guarantees fewer rapid reductions in brain/nerve glucose concentration and therefore **fewer episodes of *relative* hypoglycemia so the pancreatic beta cells don't become "inflamed**."

When an aging adult eats too often too, the brain/nerve cells can become chronically 'starved & inflamed' from lack of 'localized' brain/nerve insulin production, and increase 'compensatory loss-of-appetite' / 'loss-of-memory-to-eat' / 'eating less often' [type 3A diabetes], to help protect the brain from potential SEVERE chronic *relative* hypoglycemia.

Diabetes can now be seen as a protective cycle that can be controlled / mitigated and/or cured, by intermittent-fasting [which also increases liver 'digestion efficiency'], a ketogenic / low glycemic diet ensuring enough energy from patients' stored body fat, and eating high glycemic foods less frequently—all of which the **PEO Solution** assists with.

Arteries and Insulin Resistance: *Medical News Today* **reported:**[1]

> "Earlier studies showed that in the context of systemic *insulin resistance*, blood vessels become resistant, too. But it wasn't clear if arteries become diseased because ***they can't respond to insulin*** or because they get exposed to too much of it. Now comes **evidence in favor of the former explanation**.
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> "Insulin-resistant blood vessels don't open up as well, and levels of a protein known as VCAM-1 increase, too. VCAM-1 belongs to a family of **adhesion** molecules… The animals' [mice] **insulin-resistant arteries** develop **plaques that are twice the size of those on normal arteries**."

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1 "Arteries and Insulin Resistance," May 5, **2010**, http://www.medicalnewstoday.com/articles/187720.php.