Block Absorption of Killer Carbohydrates

By Julius Goepp, MD

If you’re an American over 20 years old, you may already suffer from some form of impaired glucose control.

According to 2007 data from the National Institutes of Health, 25% of Americans 20 years and older had abnormally high levels of glucose in the blood—a pre-diabetic state. That number leapt to 35% in individuals 60 and older. Extrapolating from the total US population, nearly 60 million American adults may now be pre-diabetic.1

These statistics make it clear that the glut of excess carbohydrate calories lurking in the Western diet undermines our best efforts to maintain healthy metabolic function and body weight.

Far too many people have lost their metabolic capacity to properly process carbohydrates. The result is a looming public health disaster, as impaired glucose control initiates a series of metabolic disorders that sharply increases disease risk.

To reverse this lethal epidemic of impaired glucose control, aging individuals often require more aggressive measures than merely limiting carbohydrate intake.

Fortunately, researchers have discovered proven methods to limit one’s exposure to carbohydrate calories without dramatic changes in diet. It centers on a group of natural compounds called enzyme inhibitors.

These interventions block the action of sucrase, amylase, and glucosidase, digestive enzymes responsible for the breakdown of various forms of carbohydrate. Instead of absorbing and converting these sugars into higher than desired blood glucose levels, you may be able to safely eliminate many of these excess carbohydrate calories.

These natural “carb-blockers” provide a practical approach to help lower glucose levels, improve age-related markers of health, and regain glycemic control.2

COMBATING THE SCOURGE OF SUCROSE

Starting in early childhood, most Americans are exposed to far too much refined carbohydrates in the form of sucrose (table sugar). It is virtually omni-present in processed foods. It also happens to be an enemy to human health and an age-accelerator.

Many individuals do not even knowingly ingest it. According to a 2009 study in the Journal of Nutrition, only 3% of dietary sucrose is deliberately added by consumers; 82% is added by manufacturers.3 And while the World Health Organization (WHO) recommends that sucrose comprise only 10% of total energy intake, many people substantially exceed this level from the earliest years of life—usually at the expense of key nutrients.3,4
By slowing starch digestion also prolongs the amount of time it takes for the stomach to empty its contents, further reducing the amount of carbohydrate calories released at any one time into the intestine. Rapid absorption of sugars from a high-sucrose meal triggers a dangerous sequence of unfavorable hormonal and metabolic alterations that promote still greater consumption, especially in overweight individuals. The result is the dangerously high incidence of metabolic disease we see today—obesity, type 2 diabetes, and metabolic syndrome.

This alarming trend has led to a set of novel, evidence-based solutions. Aging individuals now have practical means at their disposal to slow or reverse the long-term consequences of chronic sucrose overexposure.

Sucrose is composed of two simple sugar molecules, glucose and fructose. It is poorly absorbed in the intestine in this form. In order to be utilized, it must first be broken down by the digestive enzyme sucrase. Blocking the enzymatic action of sucrase therefore limits uptake of sucrose.

### L-arabinose

Researchers have identified a potent sucrase inhibitor called **L-arabinose**. Although it is a simple plant sugar, L-arabinose is indigestible and cannot be absorbed into the blood. Instead it remains in the digestive tract and is eventually excreted. By blocking metabolism of sucrose, L-arabinose inhibits the spike in blood sugar and fat synthesis that would otherwise follow a sugar-rich meal. In animal models, L-arabinose virtually eliminates the rise in blood sugar following administration of sucrose, with blood glucose levels rising only 2% higher than in control animals that did not receive sucrose. L-arabinose did not exert any effect on serum glucose levels in control animals that did not receive sucrose.

L-arabinose has been proven safe in both short- and long-term studies, and may contribute to lowered levels of glycosylated hemoglobin (hemoglobin A1C), a measure of chronic exposure to sugar in the blood. A study combining L-arabinose and white bean extract (see next page) not only smoothed out postprandial glucose spikes and reduced insulin levels—it lowered systolic blood pressure.

### Chromium

The trace element **chromium** is another potent aid in the management of healthy blood sugar levels. Scientists have known for years that chromium aids in the management of type 2 diabetes, where it not only helps control blood glucose but also reduces total cholesterol levels. The combination of chromium with L-arabinose attacks multiple targets to prevent sucrose-induced blood sugar elevation and reduce sugar calorie exposure.

**COMPLEX CARBOHYDRATE ENZYME INHIBITORS**

### White Bean Extract

Extracts from the common white kidney bean, *Phaseolus vulgaris*, are powerful blockers of the enzyme **alpha-amylose**, which is secreted by the pancreas. Alpha-amylose breaks down long-chain, complex starch molecules into simple sugars and short-chain **oligosaccharides** for absorption in the small intestine. Blocking alpha-amylose inhibits the metabolism of starches and slows the rate at which free sugars are absorbed.

White bean extract shows enormous potential for preventing the blood sugar and insulin spikes that are associated with many chronic health disorders. Slowing starch digestion also prolongs the amount of time it takes for the stomach to empty its contents, further reducing the amount of carbohydrate calories released at any one time into the intestine.

White bean extracts operate along numerous overlapping pathways in multiple, related physiological systems. Laboratory research shows that supplementation with white bean extract promotes weight loss in obese animals, with dramatic reduction in fat accumulation without loss of muscle mass. Plasma insulin levels also dropped substantially following a high-carbohydrate meal including white bean extract in pre-clinical studies, reflecting a much more gradual rise in blood sugar levels.

**Amylase** inhibition with white bean extract has proven particularly effective in reducing glycemia (sugar load in the blood) in studies on diabetic animals. Supplementation in diabetic rats not only substantially lowered mean blood sugar levels; it also reduced the animals’ total food and water intake (water intake is increased in untreated diabetes because of the amount lost in sugar-laden urine). White bean extract has also been shown to boost levels of an intestinal hormone called **cholecystokinin** (CCK), which produces the sensation of satiety following a meal and reduces the urge to continue eating. Research further indicates it can restore overactive intestinal sugar-digesting enzymes to nearly normal levels, further reducing the sugar load.
White bean extract has yielded equally compelling results in human studies. It has been shown to diminish the effects of high-glycemic index foods (like white bread) that are notorious for producing sharp, potentially dangerous postprandial blood sugar spikes, helping to alleviate metabolic burden throughout the body. In obese but otherwise healthy patients, supplementation with white bean extract produces weight loss more than twice as fast as in controls. Even in people who are only slightly overweight, white bean extract reduces body weight, body mass index (BMI), fat mass, fat tissue thickness, and waist circumference, while maintaining lean body mass—key elements that are linked with metabolic syndrome and cardiovascular disease risk.

White bean extract is highly effective even in aging individuals whose diets contain the highest amounts of carbohydrates. By blocking amylase activity and slowing starch digestion, it increases the amount of intact carbohydrates reaching the lower bowel, where they are consumed by beneficial intestinal bacteria. It has also been shown to induce a three-fold reduction in serum triglycerides compared to controls.

White bean extract has an excellent safety profile, even at high doses. As with all the carbohydrate inhibitors reviewed here, it does not reduce blood sugar to below normal levels—an important feature for diabetics in whom large swings in glucose levels are especially dangerous.

It should be noted that ingestion of white bean extract may result in minor intestinal protein losses, requiring additional protein intake.

Irvingia

Extract of the wild African mango Irvingia gabonensis can produce weight loss by inhibiting calorie absorption and storage through multiple mechanisms. It also exerts potent anti-diabetic and anti-obesity effects. Studies from Africa in the mid-1980’s showed that Irvingia extract normalized blood sugar in diabetic patients while increasing the activity of enzymes involved in cellular energy metabolism. Irvingia extract also lowers blood levels of dangerous LDL and VLDL in diabetics, while increasing beneficial HDL levels. It mobilizes liver enzyme activity in favor of glucose storage and away from its release into the bloodstream, thereby improving glucose control.

Irvingia works in part by inhibiting the amylase enzyme, and thus provides support for those attempting to restrict their total carbohydrate exposure and lose weight. In one double-blind, placebo-controlled study of obese but otherwise healthy adults, one month of supplementation produced a 5.3% body weight loss in supplemented patients, compared with only a 1.3% loss in the control group. These individuals also saw significant improvement in their lipid profiles. Additional studies confirm these findings, demonstrating significant reductions in body fat content, waist circumference, blood sugar levels, and markers of fat tissue regulation.

In addition to blocking amylase activity, Irvingia extract acts directly on fat cells to reduce lipid formation and storage. Its operation is multi-modal—downregulating genes that promote fat production—while upregulating factors that suppress it, including adiponectin, a glucose-regulating hormone that lowers fat-mediated inflammation and enhances insulin sensitivity. Adiponectin levels correlate with a reduction in risk for metabolic syndrome.