

Glucerna 1.2 Cal Monograph

Glucerna

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Glucerna 1.2 Cal	List #	NDC Code
8 fl oz can	50904	70074-0509-05
1 L Ready to Hang	50906	70074-0509-07
1.5 L Ready to Hang	50902	70074-0509-03

1.0 Product Description

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5.0 Notes

Glucerna

Smart nutrition for people with diabetes.™

Product Description

Glucerna 1.2 Cal is a calorically dense, diabetes-specific enteral product featuring a unique carbohydrate blend for enhanced glycemic control and fish oil to support cardiovascular health. This product is clinically demonstrated to produce a superior glycemic response compared to a standard calorically dense enteral product in individuals with type 2 diabetes.¹

- For patients with type 1 or type 2 diabetes
- For patients with impaired glucose tolerance resulting from metabolic stress, such as illness, trauma, and infection
- For tube feeding or oral use
- For supplemental or sole-source nutrition
- For use under medical supervision

Features and Benefits of Glucerna 1.2 Cal

Features	Benefits
Clinically demonstrated benefit	Clinically demonstrated to produce a superior glycemic response compared to a standard calorically dense enteral product in individuals with type 2 diabetes (Data on File. Clinical Study BK06. Comparison of Nutritional Products for People with Type 2 Diabetes. Abbott Nutrition, Columbus, Ohio, 2008.)
Calorically dense	At 1.2 Cal/mL, can meet patient needs with less volume
Unique blend of carbohydrates	Contributes to blunted postprandial glucose response due to digestive/metabolic properties of the carbohydrates
Optimal fat blend	Low in saturated fatty acids (SFA) and rich in monounsaturated fatty acids (MUFA) for improved glycemic control and blood lipid profiles. Provides both plant-based omega-3 fatty acids from canola oil (3 g of ALA per 1500 calories) and fish oil omega-3 fatty acids to support circulatory and heart health
Fish oil	As part of the fat blend, Glucerna 1.2 Cal provides the omega-3 fatty acids EPA + DHA (1 g per 1500 Cal) from fish oil, which meets American Heart Association recommendations for reducing the risk of cardiovascular disease ²
High in protein	20% of calories from protein to promote anabolism and support wound healing
NutraFlora® scFOS	Fructooligosaccharides are prebiotics and a source of soluble fiber to help maintain digestive tract health. NutraFlora FOS is the only short-chain FOS
Complete and balanced	Macronutrient distribution: 35% carbohydrate, 45% fat, and 20% protein. 1500 Cal provide at least 100% of the RDIs for 24 key vitamins and minerals
Chromium picolinate	Chromium picolinate is the most bioavailable form of chromium and is clinically shown to lower fasting, postprandial glucose, and A1C levels ³
Fortified with conditionally essential nutrients	Supplemented with the conditionally essential nutrients carnitine and taurine
Lactose- and gluten-free	Does not contribute to risk of lactose-induced diarrhea in lactose-intolerant patients; may be used by people who are gluten-intolerant

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Energy Distribution of Glucerna 1.2 Cal

Glucerna 1.2 Cal		
Calories	285 Cal/237 mL 1200 Cal/L 1800 Cal/1.5 L	
	(g/L)	(% energy)
Protein	60.0	20
Carbohydrate	114.5	35
Fiber	17.0	—
Fat	60.0	45

Carbohydrate and Fiber Profile of Glucerna 1.2 Cal

Glucerna 1.2 Cal	
Carbohydrate Content	27.1 g/237 mL 114.5 g/L 171.8 g/1.5 L
Carbohydrate Sources	Corn maltodextrin, isomaltulose, fructose, sucromalt, glycerine, fiber, and Fibersol®
Fiber Content/Sources	<p>4.1 g/237 mL 1.7 g of total dietary fiber from soy, oat, and corn fibers (Fibersol); 2.4 g of scFOS</p> <p>17.0 g/L 7.0 g of total dietary fiber from soy, oat, and corn fibers (Fibersol); 10.0 g of scFOS</p> <p>25.5 g/1.5 L 10.5 g of total dietary fiber from soy, oat, and corn fibers (Fibersol); 15.0 g of scFOS</p>

Fibersol® is not a registered trademark of Abbott Laboratories Inc.

Fat Profile of Glucerna 1.2 Cal

Glucerna 1.2 Cal	
Fat Content	14.2 g/237 mL 60 g/L 90 g/1.5 L
Sources of Fat (Listed as % of total fat blend)	
High-oleic sunflower oil	48%
Canola oil	43%
Fish oil	4%
Soy lecithin	5%
Fatty Acids*	
Monounsaturated Fatty Acids (MUFA)	36 g (27%) [†]
Polyunsaturated Fatty Acids (PUFA)	17 g (13%) [†]
Saturated Fatty Acids (SFA)	4 g (3%) [†]

* Fatty acids equal approximately 95% of total fat.

[†] Percent of total energy; total energy per liter is 1200 Cal.

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Protein Profile of Glucerna 1.2 Cal

Glucerna 1.2 Cal	
Protein Content	14.2 g/237 mL 60.0 g/L 90.0 g/1.5 L
Protein Source	Sodium caseinate, soy protein isolate, and milk protein concentrate
Total Cal/g Nitrogen Ratio	125:1
Nonprotein Cal/g Nitrogen Ratio	100:1

Vitamin and Mineral Profile of Glucerna 1.2 Cal

Vitamins	8 oz	% RDI†	1 L	% RDI†	1.5 L	% RDI†
Total Vitamin A (IU)*	1840	37	7730	155	11600	230
Vitamin D (IU)	82	21	345	86	515	130
Vitamin E (IU)	9.2	31	39	130	58	195
Vitamin K (mcg)	29	36	120	150	180	225
Vitamin C (mg)	62	105	260	435	390	650
Folic acid (mcg)	76	19	320	80	480	120
Thiamin (Vitamin B1) (mg)	0.29	19	1.2	80	1.8	120
Riboflavin (Vitamin B2) (mg)	0.33	19	1.4	82	2.1	125
Vitamin B6 (mg)	0.38	19	1.6	80	2.4	120
Vitamin B12 (mcg)	1.2	20	4.8	80	7.2	120
Niacin (mg)	3.8	19	16	80	24	120
Choline (mg)	105	‡	440	‡	660	‡
Biotin (mcg)	57	19	240	80	360	120
Pantothenic acid (mg)	1.9	19	8.0	80	12	120
Minerals						
Sodium (mg)	265	‡	1110	‡	1670	‡
Sodium (mEq)	11.5	‡	48.3	‡	72.6	‡
Potassium (mg)	480	‡	2020	‡	3030	‡
Potassium (mEq)	12.3	‡	51.8	‡	77.7	‡
Chloride (mg)	305	9	1280	38	1920	56
Chloride (mEq)	8.7	‡	36.6	‡	54.9	‡
Calcium (mg)	190	19	800	80	1200	120
Calcium (mEq)	9.5	‡	39.9	‡	59.9	‡
Phosphorus (mg)	190	19	800	80	1200	120
Phosphorus (mEq)	12.3	‡	51.6	‡	77.4	‡
Magnesium (mg)	76	19	320	80	480	120
Magnesium (mEq)	6.3	‡	26.3	‡	39.5	‡
Iodine (mcg)	29	19	120	80	180	120
Manganese (mg)	0.38	19	1.6	80	2.4	120
Copper (mcg)	0.38	19	1.6	80	2.4	120
Zinc (mg)	2.9	19	12	80	18	120
Iron (mg)	3.5	19	15	83	22	120
Selenium (mcg)	14	20	56	80	84	120
Chromium (mcg)	38	32	160	135	240	200
Molybdenum (mcg)	15	20	60	80	90	120
Other						
M-Inositol (mg)	200	‡	845	‡	1270	‡
Taurine (mg)	32	‡	135	‡	200	‡
L-Carnitine (mg)	42	‡	175	‡	265	‡

* Includes 830 IU/8 fl oz of Vitamin A Activity supplied by 0.63 mg of beta-carotene.

* Includes 3570 IU/L of Vitamin A Activity supplied by 2.7 mg of beta-carotene.

* Includes 5300 IU/1.5 L of Vitamin A Activity supplied by 4.0 mg of beta-carotene.

† For adults and children 4 or more years of age.

‡ RDI Not Established.

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Ingredient Listings

Glucerna 1.2 Cal 237 mL/8 fl oz

Water, Sodium caseinate, Corn maltodextrin, High oleic safflower oil, Isomaltulose, Canola oil, Fructose, Soy protein isolate, Sucromalt, Fructooligosaccharides, Glycerine, Milk protein concentrate, Oat fiber, Soy lecithin, Soy fiber, Potassium citrate, Marine oil (may contain one or more of the following: anchovy, menhaden, salmon, sardine, tuna), Magnesium phosphate, Natural and artificial flavors, Potassium chloride, m-Inositol, Calcium carbonate, Calcium citrate, Sodium citrate, Ascorbic acid, Choline chloride, Sodium chloride, L-Carnitine, Taurine, Carrageenan, Ferrous sulfate, dl-alpha-tocopheryl acetate, Zinc sulfate, Niacinamide, Calcium pantothenate, Manganese sulfate, Cupric sulfate, Vitamin A palmitate, Thiamine chloride hydrochloride, Pyridoxine hydrochloride, Beta-carotene, Riboflavin, Sodium selenate, Chromium picolinate, Folic acid, Biotin, Sodium molybdate, Potassium iodide, Phylloquinone, Cyanocobalamin, and Vitamin D3.

Contains milk and soy ingredients. Gluten- and lactose-free.

Glucerna 1.2 Cal 1 Liter Ready-To-Hang

Water, Sodium caseinate, Corn maltodextrin, High oleic safflower oil, Isomaltulose, Canola oil, Fructose, Soy protein isolate, Sucromalt, Fructooligosaccharides, Glycerine, Milk protein concentrate, Oat fiber, Soy lecithin, Soy fiber, Potassium citrate, Marine oil (may contain one or more of the following: anchovy, menhaden, salmon, sardine, tuna), Magnesium phosphate, Potassium chloride, m-Inositol, Calcium carbonate, Calcium citrate, Sodium citrate, Ascorbic acid, Choline chloride, Sodium chloride, L-Carnitine, Taurine, Carrageenan, Ferrous sulfate, dl-alpha-tocopheryl acetate, Zinc sulfate, Niacinamide, Calcium pantothenate, Manganese sulfate, Cupric sulfate, Vitamin A palmitate, Thiamine chloride hydrochloride, Pyridoxine hydrochloride, Beta-carotene, Riboflavin, Sodium selenate, Chromium picolinate, Folic acid, Biotin, Sodium molybdate, Potassium iodide, Phylloquinone, Cyanocobalamin, and Vitamin D3.

Contains milk and soy ingredients. Gluten- and lactose-free.

Glucerna 1.2 Cal 1.5 Liter Ready-To-Hang

Water, Sodium caseinate, Corn maltodextrin, High oleic safflower oil, Isomaltulose, Canola oil, Fructose, Soy protein isolate, Sucromalt, Fructooligosaccharides, Glycerine, Milk protein concentrate, Oat fiber, Soy lecithin, Soy fiber, Potassium citrate, Marine oil (may contain one or more of the following: anchovy, menhaden, salmon, sardine, tuna), Magnesium phosphate, Potassium chloride, m-Inositol, Calcium carbonate, Calcium citrate, Sodium citrate, Ascorbic acid, Choline chloride, Sodium chloride, Carrageenan, L-Carnitine, Taurine, Ferrous sulfate, dl-alpha-tocopheryl acetate, Zinc sulfate, Niacinamide, Calcium pantothenate, Manganese sulfate, Cupric sulfate, Vitamin A palmitate, Thiamine chloride hydrochloride, Pyridoxine hydrochloride, Beta-carotene, Riboflavin, Sodium selenate, Chromium picolinate, Folic acid, Biotin, Sodium molybdate, Potassium iodide, Phylloquinone, Cyanocobalamin, and Vitamin D3.

Contains milk and soy ingredients. Gluten- and lactose-free.

1.0 References

1. Data on File. Clinical Study BK06. Comparison of Nutritional Products for People with Type 2 Diabetes. Abbott Nutrition, Columbus, Ohio, 2008.
2. Kris-Etherton PM, Harris WS, Appel LJ: Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. *Circulation* 2002;106:2747-2757.
3. Broadhurst CL, Domenico P: Clinical studies on chromium picolinate supplementation in diabetes mellitus—a review. *Diabetes Technol Ther* 2006;8:677-687.

Ingredient Descriptions

Carbohydrates

Hyperglycemia is implicated in both short- and long-term complications of diabetes, and thus, managing postprandial plasma glucose (PPG) is critically important to individuals with diabetes.^{1,2} Postprandial glycemic response can be improved by consuming carbohydrates that are digested relatively slowly, thus releasing glucose over a longer length of the gastrointestinal tract than simpler forms of carbohydrates (Figure 2.1).

Including slowly digested carbohydrates in the diet permits individuals with diabetes to absorb glucose more evenly than when simple carbohydrates are consumed or when the carbohydrate source is rapidly digested and absorbed. The altered linkages of the carbohydrate structure lead to this slowed digestion. The slowly digested carbohydrate sources in Glucerna 1.2 Cal include fibersol, sucromalt, and isomaltulose. These ingredients are described in detail in this section.

Figure 2.1 Points of absorption of rapidly and slowly digested carbohydrate in the gastrointestinal tract

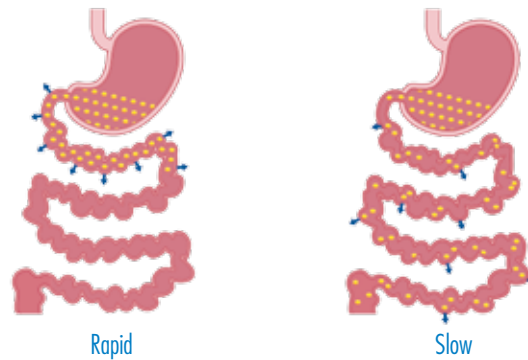
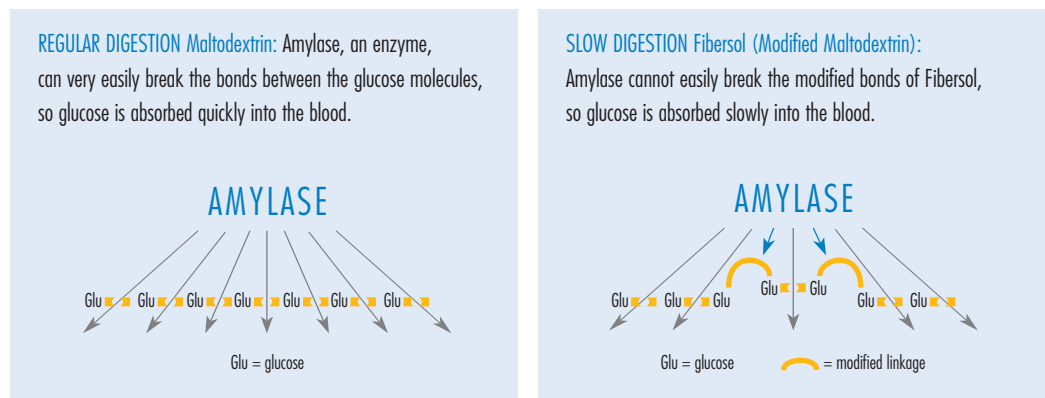


Diagram taken from: Jenkins DJ, Taylor RH, Wolever TM: The diabetic diet, dietary carbohydrate and differences in digestibility. *Diabetologia* 1982;23:477-484.

Fibersol®

Fibersol is a modified maltodextrin with glucose linkages that are more resistant to the digestive enzyme amylase than are the glucose linkages of standard maltodextrin (Figure 2.2).^{3,4} Because glucose from Fibersol is slowly digested, a portion of Fibersol proceeds to the colon, where it is fermented to short-chain fatty acids, similar to digestion of other soluble fibers. This slowed absorption gives Fibersol a lower glycemic index. Glucerna 1.2 Cal contains 3 g/L.

Figure 2.2 The modified maltodextrin of Fibersol slows its digestion, thus blunting glycemic response



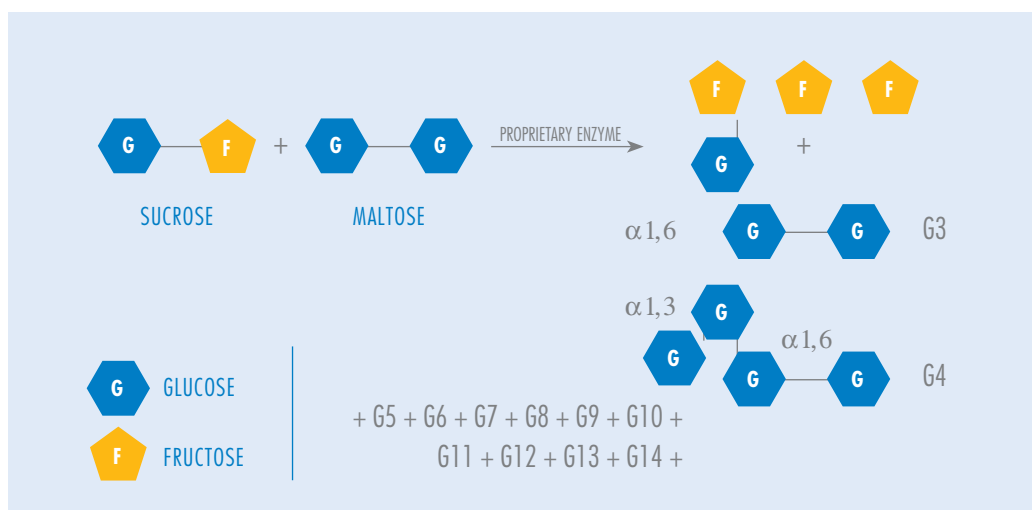
Sucromalt

Origin and Molecular Structure

Sucromalt is a slowly digested carbohydrate that contributes to a lower blood glucose response. Sucromalt is a fully digestible, 4 Cal/g, low-glycemic carbohydrate that provides sweetness and body (product attribute) in one ingredient.

Figure 2.3 below depicts the production of sucromalt. In this process, sucrose (glucose + fructose) and maltose (glucose + glucose) are combined. Sucrose is derived from either sugar cane or sugar beets. Maltose is derived from grains. A proprietary enzyme is added to cleave off fructose and to rearrange the glucose molecules. The glucose molecules are then linked with α -1,3 and α -1,6 linkages. These altered linkages are key to the lower glycemic response because it takes the body more time to identify and break the bonds into individual glucose molecules. As a result, there is less of a rise in blood glucose and eventually, the sucromalt is completely digested. Sucromalt is GRAS (Generally Recognized as Safe) for the general population. Glucerna 1.2 Cal contains 9.5 g/L.

Figure 2.3 Sucromalt production process



Blood Glucose Response

Glycemic Index Determination of Sucromalt

To date, six studies have been conducted to determine the glycemic index (GI) of sucromalt. The first two studies were conducted at NutriScience Limited (Maastricht University Holding, The Netherlands). The other four studies were conducted at Glycaemic Index Testing, Inc. (Toronto, Ontario).

Overnight-fasted subjects without diabetes were tested on two different occasions, once after ingesting sucromalt and once after ingesting glucose. Blood samples were obtained at baseline, 15, 30, 45, 60, 90, and 120 minutes post ingestion. Serum levels of glucose were measured and the incremental areas under the response curves were calculated (any area below baseline was ignored) and used to determine glycemic index. Additional information on the general methods can be found in Wolever TM, et al: The glycemic index: methodology and clinical implications. *AJCN* 1991; 54: 846-854.

The mean GI of sucromalt from the six different studies was calculated in order to arrive at a value for the GI of sucromalt. The GI of sucromalt is 53.3 ± 4.8 , which is classified as low.⁵

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Isomaltulose

Isomaltulose is a carbohydrate derived from beet sugar (sucrose). Like sucrose, it is fully available to the body but slowly released, thus, resulting in a much slower, lower, and longer-lasting blood glucose response compared to sucrose. In other words, isomaltulose provides glucose in a more balanced way. Isomaltulose is GRAS by the FDA. Glucerna 1.2 Cal contains 24.7 g/L.

Origin and Molecular Structure

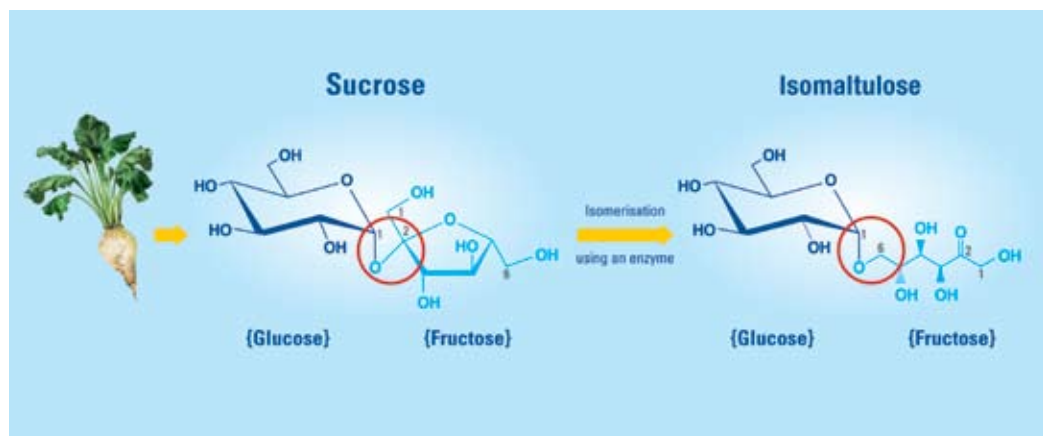
This carbohydrate naturally occurs in foods such as honey and sugar cane juice; however, the amounts are too small to be extracted. Therefore, an enzymatic process was developed by which isomaltulose is made from sucrose from sugar beets. Like sucrose, isomaltulose is a disaccharide carbohydrate consisting of the monosaccharides glucose and fructose. The difference from sucrose lies in the binding between those two monosaccharides, which is an α -1,6 linkage (compared to the α -1,2 linkage in sucrose) and therefore more difficult for gastrointestinal enzymes to split. Further details are summarized in Table 1 and the structure is shown in Figure 2.4.

Table 1: Chemical Description of Isomaltulose

GENERAL OR USUAL NAME:	Isomaltulose
TRADE NAME:	Palatinose™
CHEMICAL CLASSIFICATION:	Carbohydrate (Disaccharide)
TOTAL MOLECULAR FORMULA:	$C_{12}H_{22}O_{11} \times H_2O$

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Figure 2.4 Molecular structure of isomaltulose



Physiological Properties

As a result of the stronger bond between the two monosaccharides, isomaltulose distinctly differs in its nutritional and physiological properties from those of sucrose:^{6,7}

Digestion and absorption. Isomaltulose is slowly hydrolyzed in the small intestine, about 4 to 5 times more slowly than sucrose, as demonstrated by enzyme kinetic studies. The same enzyme system as for other carbohydrates is used to hydrolyze isomaltulose (sucrase-isomaltase complex). Absorption does not only take place in the upper parts of the small intestine (as is the case for quickly absorbed sugars), but along the entire small intestine. This means that isomaltulose still supplies glucose (thus fuel or energy) for the body at a time when the digestion and absorption of sucrose is already completed.⁷

It has been demonstrated as well that the overall digestion of isomaltulose is essentially completed in the small intestine and no significant amounts of isomaltulose reach the large intestine.⁸ Thus, isomaltulose provides the same amount of calories as all digestible carbohydrates (sugars and starches: 4 Cal/g) and is equally well tolerated.

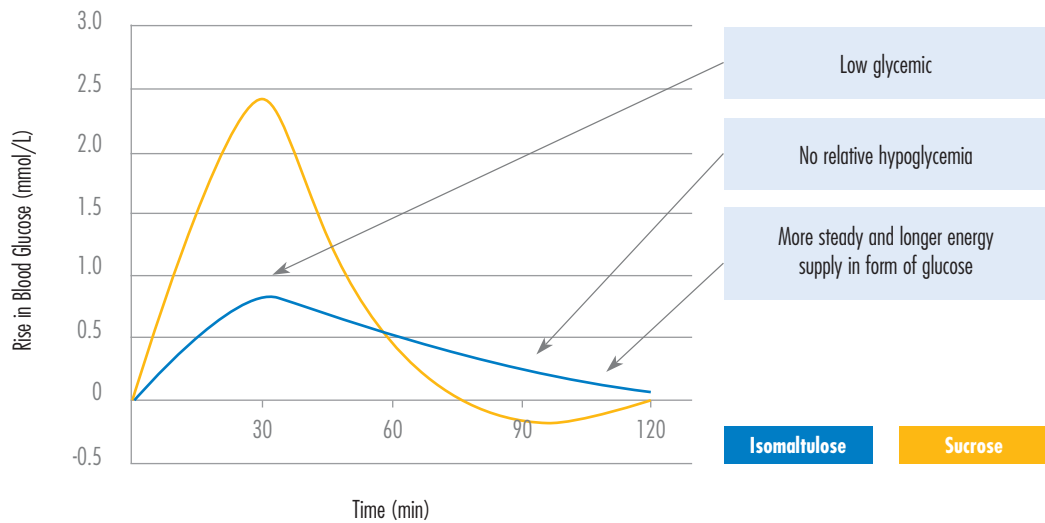
Blood glucose response and prolonged energy release. The slow but complete hydrolysis and absorption of isomaltulose is reflected in its characteristic blood glucose response with a slow, low, and sustained rise in blood glucose levels (Figure 2.5) and a correspondingly low insulin demand. The Glycemic Index (GI) of isomaltulose has been determined at Sydney University's Glycemic Research Service according to internationally recognized standard methodology, which yielded a GI of 32.⁹ In comparison, a GI of 68 has been determined for sucrose, and glucose has a GI of 100.

Insulin response and fat oxidation. The hormone insulin plays a key role in the regulation of metabolism. Among others, it down-regulates high blood glucose levels by "opening the door" for glucose uptake into cells. At the same time, it promotes the utilization of carbohydrates and the storage of fat and inhibits fat burning. High levels of insulin over a longer period of time are thought to contribute to obesity and the development of diabetes. Isomaltulose with its lower effect on blood glucose levels and subsequent lower insulin release thus shows increased fat oxidation (demonstrated by measurements of the respiratory quotient).¹⁰⁻¹² A study with metabolic syndrome subjects and studies with active sportsmen showed an increase in fat oxidation of as much as 28%.^{11,12}

Effect on long-term parameters of blood glucose control. A human intervention study over 12 weeks suggests that the regular intake of a liquid formula with isomaltulose by persons with impaired glucose tolerance would have beneficial effects on metabolic syndrome-related parameters: In this long-term study with persons with impaired glucose tolerance, the intake of an isomaltulose-based formula as part of breakfast was associated with improvements in 2h plasma glucose after OGTT and serum free fatty acid levels; moreover, in viscerally obese persons, the visceral fat accumulation was decreased ($p < .05$).¹³

2.0

Figure 2.5 Characteristics of the glycemic response of ismaltulose in comparison to sucrose



Fructose

Fructose is a simple sugar, (4 Cal/g), that occurs naturally in fruits and honey. Glucerna 1.2 Cal contains 22 g/L of fructose. The fructose in Glucerna 1.2 Cal facilitates hepatic glucose clearance and thus helps blunt a postprandial blood glucose rise.¹⁴

When fructose is metabolized in the liver, glucokinase is activated, which allows more glucose to enter the liver from the blood, thus lowering blood glucose levels (Figure 2.6). Normally, glucose is taken up by the liver and converted to glucose-6-phosphate by the enzyme glucokinase; glucose-6-phosphate is further metabolized or stored in the liver as glycogen. Glucose-6-phosphate is isomerized to fructose-6-phosphate, which in turn provides normal feedback deactivation of glucokinase.

In people with type 2 diabetes, glucokinase activity is inhibited; glucose uptake by the liver is reduced, and blood glucose levels are increased.¹⁵ However, when dietary fructose is consumed, liver uptake of fructose results in formation of fructose-1-phosphate, a metabolite that reduces the inhibition of glucokinase caused by fructose-6-phosphate (Figure 2.7). Thus, in the presence of dietary fructose, glucokinase activity can be at least partly restored to facilitate further uptake of glucose from the blood.

Glycerine

Glycerine (sometimes called glycerol) is a low-glycemic carbohydrate that functions as a sweetener. Glycerine is classified as a sugar alcohol. However, unlike other sugar alcohols such as maltitol and sorbitol, which are only partially metabolized and provide 2 Cal/g, glycerine is completely metabolized like a carbohydrate and provides 4.3 Cal/g. This difference in metabolism is key to minimizing the risk for GI side effects. The GI tolerance of sugar alcohols depends on how they are digested. The more completely a sugar alcohol is digested (like glycerine), the less potential there is for GI side effects.

Glycerine is listed as GRAS by the FDA and has an upper threshold of 125 g/day. Glycerine is listed with the ingredients on Glucerna products. The amount of glycerine in Glucerna is included in the total carbohydrate grams claimed on the product label. Glucerna 1.2 Cal contains 10 g/L.

Figure 2.6 Glucose metabolism without dietary fructose

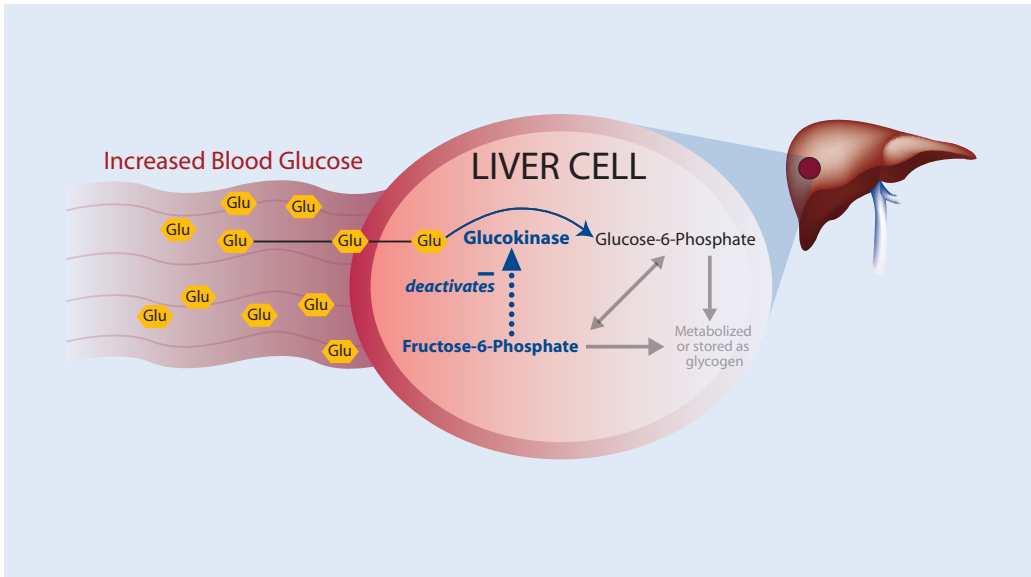
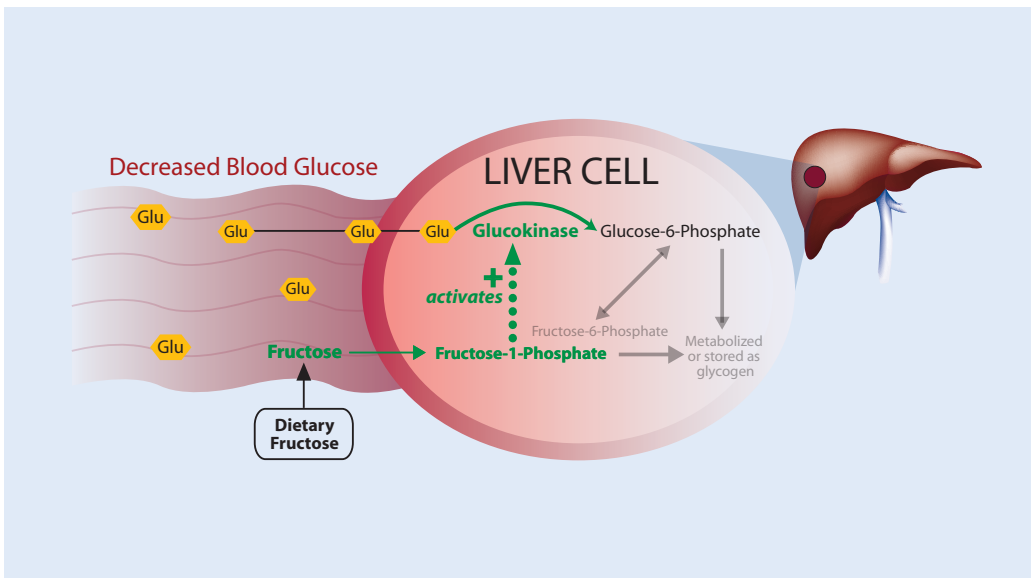


Figure 2.7 Glucose metabolism with dietary fructose



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Role of Fat Intake and Diabetes

Patients with diabetes are at an increased risk for dyslipidemia (abnormal lipid levels, eg, high cholesterol, triglycerides, and LDL cholesterol and low HDL cholesterol levels) and macro- and microvascular disease. Type 2 diabetes and chronic hyperglycemia increase the risk of cardiovascular disease (CVD) mortality between 40% and 200%.¹⁶ Individuals with diabetes have a three- to-fourfold increase in CVD risk compared with the general population.¹⁷ It is known that dietary fat composition plays an important role in the prevention and treatment of CVD and influences glucose metabolism. Therefore, the composition of the fat blend in nutritional products designed for people with diabetes is key to providing optimal metabolic control in patients with diabetes to reduce the risk for CVD or slow the progression of diabetes- and CVD-related complications.

Fat Profile of Glucerna 1.2 Cal

Glucerna 1.2 Cal	
Fat Content	14.2 g/237 mL 60 g/L 90 g/1.5 L
Sources of Fat (Listed as % of total fat blend)	
High-oleic sunflower oil	48%
Canola oil	43%
Fish oil	4%
Soy lecithin	5%
Fatty Acids*	
Monounsaturated Fatty Acids (MUFA)	36 g (27%) [†]
Polyunsaturated Fatty Acids (PUFA)	17 g (13%) [†]
Saturated Fatty Acids (SFA)	4 g (3%) [†]
per 1500 kcal	
Total Fat, %	45
Saturated, %	3
Trans-Fatty Acids	0
MUFA %	27
PUFA, n6, %	8
LA, g	13
PUFA, n3, %	3
ALA, g	3
EPA + DHA, g	1
LA/ALA ratio	4.9:1
Total n6:n3 ratio	3.3:1

* Fatty acids equal approximately 95% of total fat.

† Percent of total energy in parentheses; total energy per liter is 1200 Cal.

Overview of the Fat Blend in Glucerna 1.2 Cal

The fat blend in Glucerna 1.2 Cal was developed with the goal of facilitating metabolic control in patients with diabetes, specifically, improved glycemic and lipidemic profiles.

Saturated Fatty Acids

Saturated fatty acids (SFA) are composed of carbons with no double bonds between adjacent carbon molecules (-C-C-). Glucerna 1.2 Cal is low in SFA by providing just 4% of the total energy. SFA are naturally occurring in the product's high-oleic safflower oil and canola oil. The American Diabetes Association (ADA) and American Heart Association (AHA) recommend that less than 7% of calories should come from SFA.^{18,19}

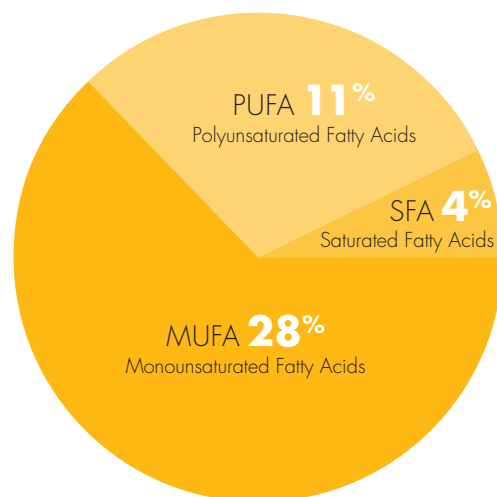
Monounsaturated Fatty Acids

Monounsaturated fatty acids (MUFA) are fatty acids made up of a carbon chain of varying lengths that contains a single double bond (-C=C-). Glucerna 1.2 Cal is high in MUFA, which contribute 28% of the total calories. The MUFA in Glucerna 1.2 Cal are provided from canola oil and high-oleic safflower oil and constitute 91% of the fat blend for improved glycemic control and blood lipid profiles.²⁰⁻²²

Dietary intakes low in SFA and rich in MUFA favorably influence blood lipid levels, specifically plasma triglycerides. Early clinical studies that examined the effect of MUFA on lipid parameters have shown that a high MUFA intake lowers total cholesterol and triglycerides and can raise HDL cholesterol and, therefore, favorably affect CVD risk in healthy individuals.^{23,24} Significant reductions were demonstrated in plasma triglyceride levels in subjects with type 2 diabetes.²⁵ A meta-analysis of ten, long-term, controlled clinical studies demonstrated that high MUFA intakes (22-32% of the total energy from MUFA and 37-50% of the total calories from fat) reduced fasting plasma triglycerides and VLDL cholesterol concentrations by 19% and 22%, respectively, compared to lower fat intakes (20-32% of the total calories from fat) in patients with type 2 diabetes.²¹

In addition to lowering triglycerides in patients with diabetes, a high MUFA intake can improve glycemic control. Consuming a high MUFA intake can lower fasting plasma glucose levels, as well as postprandial and mean 24-h plasma glucose concentrations compared to a high-carbohydrate, low-fat intake.²¹ Postprandial hyperglycemia is associated with outcomes such as increased risks for cardiovascular disease and thrombosis, and dyslipidemia.^{3,4} A high MUFA intake may help minimize postprandial hyperglycemia and, in turn, reduce the risk for CVD and thrombosis (blood clot formation).

Fat Blend Distribution*



*Fatty acids equal approx. 95% of total fat.

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n-3 (EPA + DHA and ALA) and n-6 Polyunsaturated Fatty Acids

Polyunsaturated fatty acids are fatty acids made up of a chain of carbon atoms with more than one double bond between adjacent carbons. The fat blend in Glucerna 1.2 Cal contains fish oil to provide an amount of eicosapentaenoic (EPA) and docosahexaenoic (DHA) recommended by the AHA. The AHA also recommends 1.5 to 3 g per day of plant-based alpha-linolenic omega-3 fatty acid (ALA).²⁶ Glucerna 1.2 Cal provides approximately 1 g EPA + DHA from fish oil sources (anchovy, menhaden, salmon, sardine, or tuna oils) and 3 g ALA per 1500 Calories.

The two subgroups of n-3 PUFA are ALA, found in plant sources such as canola and walnut oils, and EPA and DHA, which are found in oils from fatty fish. Linoleic acid (LA) is an n-6 fatty acid found in a many types of vegetable oils. ALA and LA are essential fatty acids, meaning that because the body is unable to produce them, they must be consumed in the diet. In the body, LA is elongated to arachidonic acid (AA), a metabolic precursor to eicosanoids, which are signaling compounds that can affect inflammation, platelet aggregation (involved in formation of blood clots), and vascular blood flow.

The eicosanoids produced from LA have greater proinflammatory and prothrombotic (blood clot formation) activity than those made from ALA or EPA. Increasing the intake of n-3 PUFA can partially substitute n-6 PUFA to interfere with the production of eicosanoids from AA and favor the production of weaker eicosanoids made from n-3 PUFA (Figure 2.8). The benefits of n-3 fatty acids are to reduce inflammation, vasoconstriction, and platelet aggregation.

Figure 2.8 n-3 PUFA and n-6 PUFA

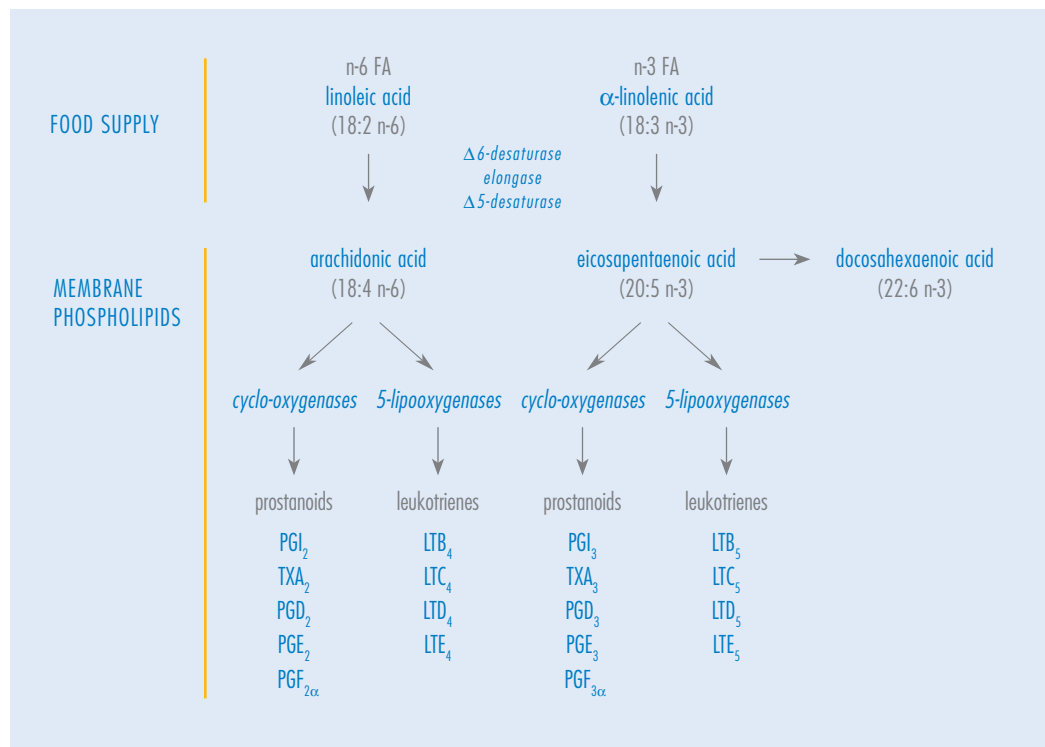


Diagram: Metabolism and nomenclature of the main PUFA of the linolenic series (left) and the α-linolenic series (right). The biosynthetic pathway is catalyzed by the reactions of elongase and desaturases (Δ5 and Δ6) and gives rise to eicosanoids (including prostanoids and leukotrienes) with distinct biological properties. LT, leukotriene; PG, prostaglandin; TX, thromboxane (see text for details).¹⁷

n-6:n-3 Ratio

The n-6:n-3 fatty acid ratio has received a lot of attention lately. Historically, n-3 fatty acids have been part of our diet since the dawn of humankind. The ratio of n-6 to n-3 fatty acids in the diet of humans back then has been estimated to be 1:1.²⁷ Today, that ratio has risen to 10:1 in the United States due to an increased use of n-6-rich vegetable oils along with a reduced intake of n-3 fatty-acid-rich foods. The n-6:n-3 ratio reflects the conversion efficiency of ALA to EPA + DHA in the body. The higher the LA content of the diet, the less the rise in EPA + DHA levels; the lower the ratio, the greater the rise in EPA + DHA levels. The Institute of Medicine has recommended a range of this ratio between 5:1 and 10:1.²⁸ However, the ratio becomes less important when EPA is in the diet because the ratio is only considering the conversion efficiency of ALA to EPA + DHA. The LA:ALA ratio of Glucerna 1.2 Cal is 4.9:1. The addition of preformed EPA and DHA ensures adequate provision of the bioactive fatty acids.

Reduced CVD risk and related complications, including improved lipid profile, reduced clot formations, and improved circulation, have been demonstrated in people with and without diabetes following diets rich in n-3 fatty acids.^{17,26} The main impact of n-3 fatty acids on improving dyslipidemia is a reduction in plasma triglycerides by 20-50% in healthy individuals, and more in patients with hypertriglyceridemia, including patients with diabetes.^{29,30} A second impact of n-3 fatty acids is on reducing LDL oxidation, a key factor in the early stages of atherosclerosis.³¹ These fatty acids have also been shown to decrease platelet activity and reduce platelet aggregation and the production of thromboxane A₂, a potent inducer of platelet aggregation and vasoconstriction.¹⁷ In addition to reducing triglycerides and platelet aggregation, n-3 fatty acids in combination with MUFA have been clinically shown to improve circulation in people with diabetes.³²

Chromium Picolinate

Mechanism of Action

Chromium is an essential trace mineral required by the body for normal carbohydrate metabolism. Chromium is considered essential because it is not made in the body and a certain level is needed in the diet to maintain health. Dietary form of chromium, also known as chromium III, is found in foods and supplements. Chromium enhances the biological action of insulin, the hormone that is critical for the normal regulation of carbohydrate, lipid, and protein metabolism. Chromium helps insulin work more efficiently to allow blood sugar to move from the blood into the cells.³³ The effectiveness of insulin is greater in the presence of chromium than in its absence.

The most bioavailable form of chromium is called chromium picolinate.^{34,35} Chromium picolinate is the most efficacious form to use for chromium supplementation. During digestion, carbohydrates are normally broken down to glucose, the body's main source of energy. Glucose is then released into the bloodstream. A rise in blood glucose causes the pancreas to release insulin. Healthy insulin function requires adequate chromium levels. Chromium is also needed for normal insulin function, and insulin is the key metabolic hormone that influences carbohydrate metabolism.

Chromium helps insulin bind to the insulin receptors that line cell membranes. These bonds stimulate glucose transporters, which move to the surface of the cell and allow glucose to enter the cell to be converted into energy.

Daily chromium levels are often compromised, due to consumption of highly processed foods and generally sub optimal nutrition, especially with age. The American diet contains small to moderate amounts of chromium, and a high intake of sugars is correlated to an increase in urinary excretion of chromium and decreases chromium bioavailability.^{32,34} Without supplementation, it is difficult to get enough chromium and inadequate chromium levels contribute to decreased stimulation of glucose transporters. Therefore this can be a factor in glucose not entering cells as readily and rising to unhealthy levels in the bloodstream.

The RDI for chromium is 120 mcg and there is no established Upper Limit.

Clinical Studies

Chromium picolinate was added to Glucerna 1.2 Cal due to the body of literature supporting the effects of chromium picolinate in people with diabetes.

Amounts of 200-1000 mcg of chromium/day as chromium picolinate have been found to improve blood sugar control in people with diabetes. In 2006, Broadhurst and Domenico conducted a review of 15 studies with results supporting the safety and therapeutic value of CrPic for the management of hyperglycemia in subjects with diabetes. The chart on page 21 outlines these 15 studies, and Figure 2.9 outlines studies showing a difference in A1C.

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Figure 2.9 The mean difference in A1C from baseline for the chromium picolinate arm in each clinical study (nine studies total) is shown

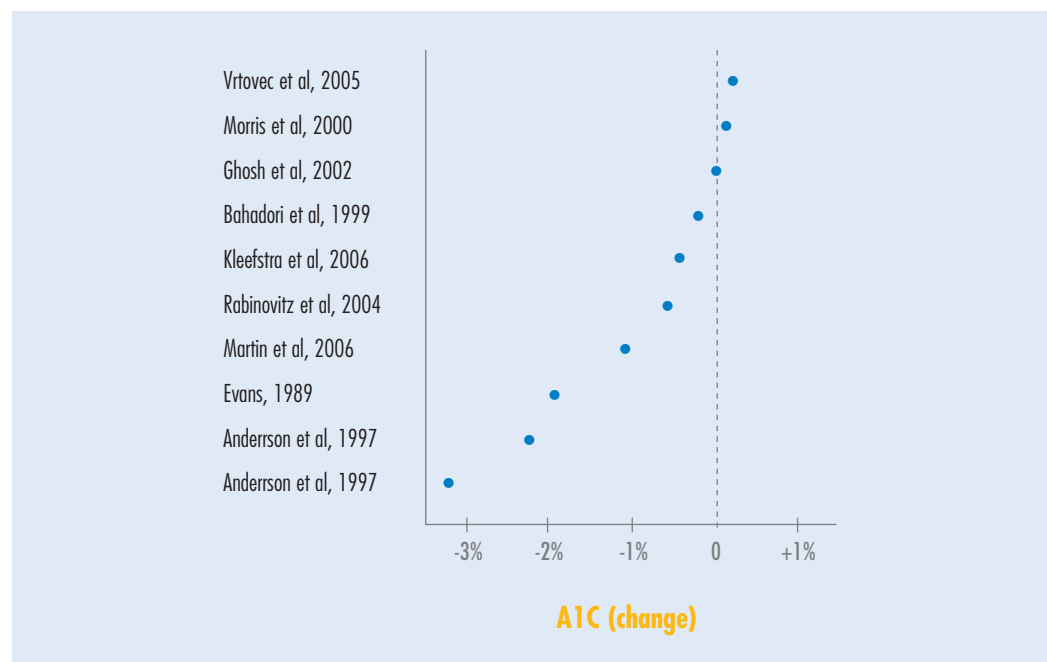


Diagram taken from: Broadhurst CL and Domenico P: Clinical studies on chromium picolinate supplementation in diabetes mellitus—a review. *Diabetes Technol Ther* 2006;8:677-687.³⁵

The chart below outlines studies that have investigated the role of chromium picolinate^{34,36-49}

All results listed were statistically significant unless noted otherwise.

Primary Author	Year	Study Design	Subject Char.	N	Amount per day (mcg)	Study Duration	Results	Concomitant Medications
Ravina	1995	Open label	Types 1 and 2	162	200	3 months	↓ fasting blood glucose levels and ↑ insulin sensitivity	Insulin sulfonylurea or metformin
Anderson	1997	RCT	Type 2	105	200/1000	4 months	↓ fasting blood glucose, postprandial glucose, insulin, and A1C	Glibendamide or glipizide
Lee	1994	RCT	Type 2	28	200	2 months	↓ triglycerides	Insulin, oral meds, diet
Evans	1989	RCT	Type 2	6	200	1.5 months	↓ fasting blood glucose, and A1C	Hypoglycemic meds
Jovanovic– Peterson	1999	RCT	Gestational diabetes	20	300-800	2 months	↓ fasting and postprandial blood glucose, insulin ↓ A1C	8 women on insulin
Rabinovitz	2004	RCT	Type 2	39	400	3 weeks	↓ fasting blood glucose ↓ A1C	Insulin/oral hypoglycemic meds
Ghosh	2002	RCT	Type 2	43	400	3 months	↓ fasting and postprandial blood glucose, insulin ↓ A1C	Hypoglycemic meds
Morris	2000	Open label	Type 2	5	400	3 months	↑ insulin sensitivity ↓ A1C (not significant)	None
Feng	2002	RCT	Type 2	136	500	3 months	↓ fasting and 2-hour glucose	Insulin
Cheng	1999	Open label	Type 2	833	500	9 months	↓ fasting blood glucose	Hypoglycemic meds
Kleefstra	2006	RCT	Type 2	29	500/1000	6 months	↓ A1C (not significant)	Oral insulin/oral hypoglycemic meds
Martin	2006	RCT	Type 2	16	1000	6 months	↑ insulin sensitivity ↓ A1C	Sulfonylurea
Vrtovec	2005	RCT	Type 2	56	1000	3 weeks- 9 months	↓ fasting insulin levels ↓ A1C (not significant)	ACE inhibitors, A2 antagonists Loop diurectics statins, aspirin
Bahadori	1999	Open label	Type 2	16	1000	4 months	↑ fasting insulin	Sulfonylurea and metformin
Cefalu	1999	RCT	Insulin resistance	29	1000	8 months	↑ insulin sensitivity	None

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Conditionally Essential Nutrients

Taurine

Taurine is a conditionally essential amino acid made from cysteine and methionine. It is an abundant free amino acid in the body but is not incorporated into body proteins. It possesses antioxidant properties and is important in bile acid conjugation, cell volume regulation, neural and retinal function, platelet aggregation, membrane stabilization, calcium homeostasis, and neuromodulation.

Taurine is present in low but adequate levels in the diet. Supplemental taurine may be required in patients being fed a defined formula diet when fed as a sole source of nutrition for prolonged periods of time. The mean daily intake is 58 mg but varies widely (17-1000 mg/d).

Low urinary and plasma taurine levels are found in adults in catabolic states (eg, cancer, chemo/radiation therapies, inflammatory processes, trauma, sepsis, burns, etc.); plasma levels are elevated in renal failure.

No studies have identified any beneficial effects of taurine supplementation for adult patients. Standard enteral formulas contain 0-211 mg taurine/1000 Cal.⁵⁰ Glucerna 1.2 Cal contains 32 mg/8 fl oz, 135 mg/L, and 200 mg/1.5 L.

Carnitine

Carnitine and taurine are present in low but adequate levels in a normal diet. Supplementation with these conditionally essential nutrients may be required under some circumstances, especially if a defined formula diet is consumed for prolonged periods.

Carnitine deficiency has been observed in patients with sepsis and trauma, during long-term total parenteral nutrition, and with long-term enteral nutritional support. Evidence of taurine depletion has been demonstrated after surgical trauma, during prolonged total parenteral nutrition, and in healthy adults fed taurine-free enteral diets.²⁴⁻²⁶ Glucerna products are fortified with carnitine and taurine.

Carnitine is an amine synthesized in the body from lysine and S-adenosylmethionine, and is found in diets containing animal products. The typical Western diet provides between 100–300 mg carnitine/day. Carnitine functions to transfer long-chain fatty acids into mitochondria for β -oxidation. It also transports other acyl groups and coenzyme A in many biochemical/metabolic reactions.

Carnitine deficiency can be caused by an inadequate intake, accelerated losses (eg, as in renal failure), and reduced synthesis (eg, in hepatic or renal failure). Carnitine needs are increased during critical illness or hemodialysis. The optimal level of carnitine in these states has not been identified, but many enteral products provide between 0–150 mg carnitine/1000 Cal.⁷ Glucerna 1.2 Cal contains 42 mg/8 fl oz, 175 mg/L and 265 mg/1.5 L.

M-inositol (myoinositol, inositol)

M-inositol, the most abundant stereoisomer of inositol, is an intracellular component of most plant and animal cells. M-inositol is the major nutritionally active form of inositol. M-inositol functions in nerve conduction by modulating the enzyme sodium-potassium-ATPase. Humans can make myoinositol from glucose. Dietary intake can influence the levels of circulation and bound m-inositol in the body, and intake from an average diet is approximately 1 gram daily.⁵¹ The major dietary sources include cereals and legumes.⁵²

Because glucose and m-inositol are similar in structure, the two substances compete for transport into tissues, resulting in m-inositol depletion in peripheral nerves and renal glomeruli. Additionally, people with diabetes have been found to lose excessive amounts of m-inositol in their urine.⁵³ Deficiency has been proposed to play a role in the pathogenesis of diabetic neuropathy.⁵⁴

More specifically, in the nerve, glucose is metabolized through the polyol pathway to sorbitol via the enzyme aldose reductase (AR). There is a strong relationship between AR levels and neuropathy. Sorbitol can cause osmotic stress and can lower nerve myoinositol and taurine levels, which decreases sodium-potassium-ATPase.⁵⁵ Furthermore, the hyperglycemia of diabetes leads to an increased flux through the polyol pathway, resulting in elevated levels of sorbitol.

Glucerna 1.2 Cal contains m-inositol at 200 mg/8 fl oz, 845 mg/L, and 1270 mg/1.5 L.

Other Characteristics of Glucerna 1.2 Cal

Glucerna 1.2 Cal		
Osmolality	720 mOsm/kg water	
Osmolarity	579 mOsm/kg	
Viscosity	Thin (room temperature), nectar-like (chilled) Not Low-Residue	
Minimum Tube Size for Gravity/ Pump Feeding, FR	10/8	
Exchanges* (per 8 fl oz)	2 starches, 2 medium-fat meat OR 2 starches, 1 medium-fat meat, 1 fat	
Carbohydrate Choices	2	
Renal Solute Load	479 mOsm/L Renal solute load (RSL) represents the solutes excreted per liter of product consumed. The major determinants of renal solute load are dietary protein and electrolytes. Each milliequivalent (mEq) of sodium, potassium, and chloride contributes approximately 1 mOsm to the renal solute load; in adults, each gram of protein contributes approximately 5.7 mOsm.	
Water	8 fl oz: 192 g/mL/cc, 1 L: 805 g/mL/cc, 1.5 L: 1210 g/mL/cc	
Electrolyte Content (per Liter)		
Contribution to RSL (mOsm/L)		
Sodium	48.3 mEq	48.3
Potassium	51.8 mEq	51.8
Chloride	36.6 mEq	36.6
Protein Content	60 g	342
Total RSL		479

*Calculated using Exchange lists for Diabetes, American Diabetes Assn, American Diabetic Assn, 2008.

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Osmolality

Background

Osmolality is a measure of the concentration of particles/solutes in solution.⁵⁰ Osmolality is defined as milliosmoles per kilogram of solvent (ie, mOsm/kg). Osmolality is the appropriate term for describing solutes in enteral formulas. The major contributors to osmolality in enteral formulas are electrolytes, minerals, and organic compounds, such as protein and carbohydrate. The higher the caloric density, the less water in the formula, therefore the higher the osmolality. Smaller molecules contribute to osmolality, so products with hydrolyzed macronutrients tend to have the highest osmolality. For example, the carbohydrates in Glucerna 1.2 Cal contribute to the higher osmolality. Enteral products available have osmolalities ranging from 270 mOsm/kg to about 720 mOsm/kg H₂O, depending on the concentration of water-soluble components.

Clinical Application

Osmolality was once considered to be a major factor in gastrointestinal intolerance to enteral feeding.⁵⁶ An isotonic formula has the same osmolality of blood (~300 mOsm/L), which leads to the assumption that less water will be drawn from the body into the gut lumen and not cause diarrhea. High-osmolality formulas are sometimes diluted in the belief that the GI tract needs to reacclimate to luminal nutrients after a period of receiving nothing by mouth. This practice is not supported to date and can lead to bacterial contamination through manipulation of the formula as well as inadequate nutrient intake. However, in some patients, a period of adaptation (slow rate of delivery) of an enteral formula may be considered to reacclimate the GI tract. When chyme (stomach content) is released from the stomach, bile salts, pancreatic enzymes, bicarbonate, and water are secreted to increase the pH and to make the solution isoosmolar. This function, referred to as “autoisotonicity,” is a function of the small bowel.

Although osmolality has been shown to slow gastric emptying in various subject populations, clinically it is insignificant.⁵⁷ For comparison and perspective purposes, Table 2 lists the osmolalities of common foods/liquids used in the hospital setting. Consider that a clear liquid diet, with an osmolality of greater than 1200 mOsm/kg, is the first post-op diet initiated when the stomach is first reacclimating. Other examples include sherbet, with an osmolality of approximately 1225 mOsm/kg, and juices, which are 990 mOsm/kg (see Table 2). Patients are rarely intolerant to these fluids. Furthermore, many medications have an osmolality higher than any food or beverage, but the volume of these medications is generally small. For example, metoclopramide, a commonly used agent for gut motility, has an osmolality of 8350 mOsm/kg (see Table 3 for additional values of common medications).

Table 2: Osmolality of Select Liquids Frequently Used in the Hospital Setting

Typical Liquids	mOsm/kg
Milk	275
Gelatin	535
Broth	445
Sodas	695
Ice Pops	720
Juices	990
Ice Cream	1150
Sherbet	1225

Table 3: Osmolality of Common Medications⁵⁷

Drug	Common Single Dosage (mL)	Osmolality (mOsm/kg)
Acetaminophen elixir	15	5400
Diphenoxylate suspension	10-20	8800
Chloral hydrate syrup	2.5-10	4400
Furosemide (oral liquid)	2-8	3938
Metoclopramide liquid	5-15	8350
Cimetidine liquid	5-10	4035

Zarling et al fed one of two hypertonic formulas (650 and 690 mOsm/kg H₂O) over 8 hours for four consecutive days.⁵⁸ The aims of this study were to assess the effect of flow rate, osmolality, and composition of tube feedings on clinical tolerance. Ten normal subjects received full-strength enteral nutrition at 50 mL/hr on day 1, 100 mL/hr on day 2 and 150 mL/hr on day 3. The subjects were given half-strength formula at 100 mL/hr on day 4. Another ten normal subjects received the same regimen but in the reverse order, starting at 150 mL/hr and ending with the same half-strength formula at 100 mL/hr. There was no significant difference in tolerance found between subjects or approaches. Even at the maximal flow rate and osmolality, the result demonstrated that both types of enteral formulas were well tolerated as assessed by frequency of abdominal pain, bloating, passage of rectal gas, and stooling.

More specifically, two studies have shown that hypertonic formulas (ranging from 503 to 620 mOsm/kg H₂O) infused either into the stomach or at the ligament of Treitz achieve isotonicity or near isotonicity by the time they reached the ligament of Treitz⁵⁹ or 35 cm further down into the jejunum.⁶⁰ [note: The ligament of Treitz marks the point where the duodenum and jejunum meet.] Rees et al studied the delivery of undiluted, hypertonic (630 mOsm/kg H₂O) elemental enteral nutrition by continuous nasogastric infusion (87 mL/hr) over 24 hours in twelve patients with impaired gastrointestinal function due to inflammatory bowel disease and short-bowel syndrome.⁶¹ All received standard medical treatment in addition to enteral nutrition. The delivery of nutrition was well tolerated, and a slower rate of administration was not necessary, based on patients' symptoms.

Table 4: Osmolality of Some Abbott Nutrition Tube Feeding Products

Formula	mOsm/kg H ₂ O
Jevity® 1.2	450
Jevity 1.5	525
Nepro®	600
TwoCal® HN	725
Vital® HN	500
Glucerna®	355
Glucerna® Select	470
Glucerna® 1.2 Cal	720
Pulmocare®	475

Conclusion

Osmolality is just one minor characteristic of enteral formulas. Tolerance to tube feeding delivery is dependent on a variety of other factors, including, but not limited to, the following which must be taken into consideration: Overall clinical status of the patient, gastrointestinal function, mode of delivery (tube placement/location), appropriate feeding regimen for patient needs (continuous, nocturnal, bolus, intermittent), infusion rate, formula composition, concurrent medications, and proper, safe delivery of product to avoid contamination. Abbott Laboratories does have a system in place to monitor the safety of all products on the market to collect information related to gastrointestinal issues.

“Hypertonic enteral formulations have frequently been blamed for formula intolerance (eg, diarrhea). However, the osmolality of an enteral formulation has little to do with formula tolerance. Formula tolerance or diarrhea is most often related to severity of illness, comorbid conditions, enteric pathogens, or the concomitant use of medications administered through the enteral access device. In addition, the osmolality of several items on a clear liquid diet and many medications given via the enteral route is much higher than the osmolality of enteral formulations.”⁶²

–The A.S.P.E.N Nutrition Support Core Curriculum. 2007, p. 212

“The success of enteral tube feeding very much depends on the engagement of the person who delivers the nutrition.”⁶³

–Stephen McClave, MD

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1. de Vegt F, Dekker JM, Ruhe HG, et al: Hyperglycaemia is associated with all-cause and cardiovascular mortality in the Hoorn population: the Hoorn Study. *Diabetologia* 1999;42(8):926-931.
2. Monnier L, Lapinski H, Colette C: Contributions of fasting and postprandial plasma glucose increments to the overall diurnal hyperglycemia of type 2 diabetic patients. *Diabetes Care* 2003;26:881-885.
3. Fibersol-2: A soluble, non-digestible, starch-derived dietary fibre. In: McCleary B, Prosky L, eds. *Advanced Dietary Fibre Technology*. Oxford, UK: Blackwell Science; 2001:509-523.
4. Tokunaga K, Matsuoka A: Effect of a [FOSHU] which contains indigestible dextrin as an effective ingredient on glucose and lipid metabolism. *J Jpn Diabetes Soc* 1999;42:61-65.
5. International Table of Glycemic Load Values: 2002. *Am J Clin Nutr* 2002;76:5-56.
6. Sentko A, Willibald-Ettle I: Palatinose™ (isomaltulose). *Leatherhead Ingredients Handbook Sweeteners*, 2007, 3rd ed.
7. Lina BAR, Jonker D, Kozianowski G: Isomaltulose (Palatinose™) — a review of biological and toxicological studies. *Food Chem Toxicol* 2002;40:1375-1381.
8. Gostner, et al: Intestinal digestibility of isomaltulose (Palatinose™) in patients with ileostoma [Intestinale Verdaulichkeit von Isomaltulose (Palatinose™) bei Patienten mit Ileostoma]. *Z Gastroenterol* 2006;44:1073-1094. [Abstract P 18].
9. Sydney University's Glycaemic Research Service (SUGiRS) (2002) GI Database at www.glycemicindex.com.
10. Arai H, Mizuno A, Sakuma M, et al: Effects of a palatinose-based liquid diet (Inslow) on glycemic control and the second-meal effect in healthy men. *Metabolism* 2007;56:115-121.
11. König, et al: Metabolic effects of low-glycemic Palatinose™ during long-lasting endurance exercise. *Ann Nutr Metab* 2007;51 (suppl 1):69.
12. Kozianowski G: Physiological functionalities of the novel low glycemic carbohydrate isomaltulose (Palatinose™). *Ann Nutr Metab* 2007;51 (suppl 1):157.
13. Oizumi T, Daimon M, Jimbu Y, et al: A palatinose-based balanced formula improves glucose tolerance, serum free fatty acid levels and body fat composition. *Tohoku J Exp Med* 2007;212:91-99.
14. Hawkins M, Gabriely I, Wozniak R, et al: Fructose improves the ability of hyperglycemia per se to regulate glucose production in type 2 diabetes. *Diabetes* 2002;51:606-614.
15. Caro J, Triester S, Patel V, et al: Liver glucokinase: decreased activity in patients with type II diabetes. *Horm Metab Res* 1995;27:19-22.
16. Nettleton JA, Katz R: n-3 long-chain polyunsaturated fatty acids in type 2 diabetes: a review. *J Am Diet Assoc* 2005;105:428-440.
17. De Catarina R, Madonna R, Bertolotto A, et al: N-3 fatty acids in the treatment of diabetic patients. *Diabetes Care* 2007;30:1012-1026.
18. American Diabetes Association. Nutrition recommendations and interventions for diabetes. *Diabetes Care* 2008;31:S61-S78.
19. American Heart Association Nutrition Committee. Know Your Facts. Available at: <http://www.americanheart.org/presenter.jhtml?identifier=532>. Accessed January 3, 2008.
20. American Diabetes Association. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Diabetes Care* 2003;26:S51-S61.
21. Garg A: High-monounsaturated-fat diets for patients with diabetes mellitus: a meta-analysis. *Am J Clin Nutr* 1998;67:577S-582S.
22. Ros E: Dietary cis-monounsaturated fatty acids and metabolic control in type 2 diabetes. *Am J Clin Nutr* 2003;78:617S-625S.
23. Mensink RP, Katan MJB: Effect of monounsaturated fatty acids versus complex carbohydrates on high-density lipoproteins in healthy men and women. *Lancet* 1987;329:122-125.
24. Grundy SM, Florentin L, Nix D, et al: Comparison of monounsaturated fatty acids and carbohydrates for reducing raised levels of plasma cholesterol in man. *Am J Clin Nutr* 1988;47:965-969.
25. Parillo M, Rivellese AA, Ciardullo AB, et al: A high-monounsaturated fat/low-carbohydrate diet improves peripheral insulin sensitivity in non-insulin-dependent diabetic patients. *Metabolism* 1992;41:1373-1378.
26. Kris-Etherton PM, Harris WS, Appel LJ: Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. *Circulation* 2002;106:2747-2757.
27. Simopoulos AP: Evolutionary aspects of omega-3 fatty acids in the food supply. *Prostaglandins Leukot Essent Fatty Acids* 1999;60:421-429.
28. Food and Nutrition Board. Institute of Medicine: Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids, 2002.
29. Harris WS: n-3 fatty acids in human lipoprotein metabolism: an update. *Lipids* 1999;34:S257-S258.
30. Krauss RM, Eckel RH, Howard B, et al: AHA dietary guidelines: revision 2000: statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* 2000;102:2284-2299.
31. Appel LJ, Miller ER III, Seidler AJ, et al: Does supplementation of diet with "fish oil" reduce blood pressure? A meta-analysis of controlled clinical trials. *Arch Intern Med* 1993;153(12):1429-1438.
32. West SG, Hecker KD, Mustad VA, et al: Acute effects of monounsaturated fatty acids with and without omega-3 fatty acids on vascular reactivity in individuals with type 2 diabetes. *Diabetologia* 2005;48:113-122.
33. Anderson RA: Chromium, glucose intolerance and diabetes. *J Am Coll Nutr* 1998;17:548-555.
34. Ghosh D, Bhattacharya B, Mukherjee B, et al: Role of chromium supplementation in Indians with type 2 diabetes mellitus. *J Nutr Biochem* 2002;13:690-697.
35. Broadhurst CL, Domenico P: Clinical studies on chromium picolinate supplementation in diabetes mellitus—a review. *Diabetes Technol Ther* 2006;8:677-687.

36. Ravina A, Slezak L, Rubal A, et al: Clinical use of the trace element chromium (III) in the treatment of diabetes mellitus. *J Trace Elem Med Biol* 1995;8:183–190.
37. Anderson RA, Cheng N, Bryden NA, et al: Elevated intakes of supplemental chromium improve glucose and insulin variables in individuals with type 2 diabetes. *Diabetes* 1997;46:1786–1791.
38. Lee NA, Reasner CA: Beneficial effect of chromium supplementation on serum triglyceride levels in NIDDM. *Diabetes Care* 1994;17:1449–1452.
39. Evans GW: The effect of chromium picolinate on insulin controlled parameters in humans. *Int J Biosocial Med Res* 1989;11:163–180.
40. Jovanovic-Peterson L, Gutierrez M, Peterson CM: Chromium supplementation for women with gestational diabetes mellitus. *J Trace Elem Med Biol* 1999;12:91–97.
41. Rabinovitz H, Friedensohn A, Leibovitz A, et al: Effect of chromium supplementation on blood glucose and lipid levels in type 2 diabetes mellitus elderly patients. *Int J Vitam Nutr Res* 2004;74:178–182.
42. Morris BW, Kouta S, Robinson R, et al: Chromium supplementation improves insulin resistance in patients with Type 2 diabetes mellitus. *Diabet Med* 2000;17:684–685.
43. Feng J, Lin D, Zheng A, et al: Chromium picolinate reduces insulin requirement in people with type 2 diabetes mellitus [abstract]. *Diabetes* 2002;51:A469.
44. Cheng N, Zhu X, Hongli S, et al: Follow-up survey of people in China with type 2 diabetes mellitus consuming supplemental chromium. *J Trace Elem Med Biol* 1999;12:55–60.
45. Kleefstra N, Houweling ST, Jansman FG, et al: Chromium treatment has no effect in patients with poorly controlled, insulin-treated type 2 diabetes in an obese Western population: a randomized, double-blind, placebo-controlled trial. *Diabetes Care* 2006;29:521–525.
46. Martin J, Wang ZQ, Zhang XH, et al: Chromium picolinate supplementation attenuates body weight gain and increases insulin sensitivity in subjects with type 2 diabetes. *Diabetes Care* 2006;29:1826–1832.
47. Vrtovec M, Vrtovec B, Briski A, et al: Chromium supplementation shortens QTc interval duration in patients with type 2 diabetes mellitus. *Am Heart J* 2005;149:632–636.
48. Bahadori B, Wallner S, Hacker C, et al: Effects of chromium picolinate on insulin levels and glucose control in obese patients with Type-II diabetes mellitus [abstract]. *Diabetes* 1999;48:A349.
49. Cefalu WT, Bell-Farrow AD, Stegner J, et al: Effect of chromium picolinate on insulin sensitivity in vivo. *J Trace Elem Exp Med* 1999;12:71–83.
50. Materese L, Gottschlich M: *Contemporary Nutrition Support Practice: A Clinical Guide* 2003.
51. Colodney L, et al: *Alternative Med Rev* 1998;3:432-447.
52. www.pdrhealth.com Accessed April 2, 2007.
53. Larner J, Allan G, Kessler C, et al: Phosphoinositol glycan derived mediators and insulin resistance. Prospects for diagnosis and therapy. *J Basic Clin Physiol Pharmacol* 1998;6:127-137.
54. Boulton AJ, Malik RA, Arezzo JC, et al: Diabetic somatic neuropathies. *Diabetes Care* 2004;27:1458-1486.
55. Bloomgarden Z: Neuropathy, womens' health, and socioeconomic aspects of diabetes. *Diabetes Care* 2002;25:1085-1094.
56. Parrish C: Enteral feeding: the art and the science. *Nutr Clin Pract* 2003;18:76-85.
57. Parrish C, McCray S: Nutrition intervention in the patient with gastroparesis. *Pract Gastroenterol* 2003;53-66.
58. Zarling EJ, Parmar JR, Mobarhan S, et al: Effect of enteral formula infusion rate, osmolality, and chemical composition upon clinical tolerance and carbohydrate absorption in normal subjects. *JPEN* 1986;10:588-590.
59. Miller LJ, Malagelada JR, Go VL: Postprandial duodenal function in man. *Gut* 1978;19:699-706.
60. Hecksweiler P, Vidon N, Emonts P, et al: Absorption of elemental and complex nutritional solutions during a continuous jejunal perfusion in man. *Digestion* 1979;19:213-217.
61. Rees RP, Keohane PP, Grimbale GK, et al: Tolerance of elemental diet administered without starter regimen. *Br Med J* 1986;290:1869-1870.
62. The A.S.P.E.N Nutrition Support Care Curriculum, p. 212.
63. McClave SA, Sexton LK, Spain DA, et al: Enteral tube feeding in the intensive care units: Factors impeding adequate delivery. *Crit Care Med* 1999; 27:1252-1256.

Clinical Management of Diabetes

Glycemic Control as It Relates to In-Hospital Management

Diabetes is the fourth most common comorbid condition complicating all hospital discharges. In 1997, diabetes was present in 9.5% of all hospital discharges and 29% of patients undergoing cardiac surgery. Diabetes causes a twofold to fourfold increase in rates of hospitalizations and increases hospital length of stay by 1 to 3 days, depending on the admitting diagnosis. Studies have clearly shown that hyperglycemia in hospitalized patients complicates numerous illnesses and is an independent risk factor for adverse outcomes. More intense effort at managing glycemic control may improve short-, intermediate-, and long-term outcomes in patients with diabetes or impaired glucose tolerance in the hospital for therapeutic procedures as well as for treatment of the complications of this disease.¹

Fortunately, the risk of developing chronic microvascular and neuropathic complications, as well as acute complications such as immune dysfunction, cardiovascular changes (eg, increased blood pressure), thrombosis, inflammation, and oxidative stress, can be dramatically reduced through improved glycemic control.^{2,5} In general, for every percentage reduction in A1C, the risk of chronic complications can be expected to decrease by 40%.⁶ The benefits of tight glycemic control extend to the acute care setting, where hyperglycemia is common, secondary to metabolic stress or diabetes. Studies have demonstrated improved outcomes in medical and surgical intensive care unit (ICU) patients treated with intensive insulin therapy to attain tight glycemic control, specifically reductions in risk for multisystem organ failure, postoperative length of stay in the ICU, and infection.⁶⁻⁸ Although intensive treatment with insulin or oral glucose-lowering agents can be of great benefit to the patient, these therapies have been associated with an increased risk of hypoglycemia and related outcomes.^{3,4,9} The concomitant use of medical nutrition therapy (MNT) as adjunctive therapy for glycemic control may further enhance the quality of life and reduce risk of hypoglycemia in patients with diabetes. Within MNT, the development of diabetes-specific products designed to attenuate postprandial glycemic excursions should enhance the use of nutrition to achieve glucose control in people with diabetes mellitus.

Medical Nutrition Therapy

Nutrition therapy for people with diabetes is aimed at improving health with healthy food choices, while also meeting individual needs. Individual needs are based on disease state, physical status, personal and cultural food preferences, lifestyle, and attitude.¹⁰ Specific goals of nutrition are to prevent or treat chronic complications by attaining and maintaining optimal metabolic outcomes.

Goals of Medical Nutrition Therapy for Best Possible Metabolic Outcomes

- Improve overall health through optimal nutrition
- Maintain blood glucose levels that are as near normal as possible by balancing food intake with medication (if applicable) and physical activity levels
- Achieve optimal blood lipid levels
- Provide appropriate amounts of calories to achieve and maintain reasonable body weight

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Nutritional Needs

Enteral nutrition support is recommended for patients who are unable to meet their nutritional needs through voluntary oral intake, but have a functioning, intact gastrointestinal tract. There are two schools of thought surrounding the choice of enteral products for patients with diabetes. For some clinicians, the use of a standard enteral product coupled with close blood glucose monitoring and adjustments of exogenous insulin is sufficient to provide acceptable glycemic control.¹¹ Standard enteral products are generally high in low-molecular-weight carbohydrates and low in fat and contain moderate amounts of protein. When ingested, the carbohydrate is rapidly absorbed, which raises blood glucose levels. Diabetes-specific enteral products, on the other hand, possess macronutrient profiles designed to provide better glycemic control. They have lower levels of carbohydrate, are higher in fat, and contain between 16 and 22% of calories from protein. The carbohydrates found in diabetes-specific products are a blend of slowly digested carbohydrates, including fiber, to help modulate the glycemic response. The fat is a lipid blend rich in monounsaturated fatty acids. Because of this macronutrient profile, some clinicians prefer to use diabetes-specific enteral products for their patients with diabetes so as to circumvent concerns that standard products may compromise glycemic control.

A prospective, randomized, crossover meal tolerance test designed to simulate tube feeding compared the effects of a standard product to a diabetes-specific product in 10 subjects with type 1 diabetes consuming 20 mL of product every 15 minutes while receiving continuous intravenous insulin over 4 hours. Serum glucose levels were consistently lower and total urinary glucose excretion was significantly lower after consuming the diabetes-specific product compared to the standard product.¹²

A study by Sanz-Paris et al compared the 2-hour postprandial effects of a low-carbohydrate (LCF) and a high-carbohydrate, low-fat formula (HCF), both designed for patients with diabetes.¹³ Fifty-two patients with type 2 diabetes were randomly assigned to consume one of the two products after taking their diabetes medications (either insulin or sulfonylureas). The glycemic response to the HCF was significantly greater than that of the LCF. In addition, insulin and C-peptide levels were greater with the HCF than the LCF products. The researchers concluded that partial replacement of complex digestible carbohydrates with monounsaturated fatty acids in the lower-carbohydrate product may improve glycemic control better than the HCF diabetes product in patients with type 2 diabetes.

Another study compared the glucose and insulin responses of a standard and diabetes-specific product in 48 subjects with type 2 diabetes. The subjects were fed a bolus of each product on separate occasions following an overnight fast. Postprandial glucose and insulin levels were measured for four hours following consumption of the product. Results showed that glucose and insulin levels were significantly lower following consumption of the diabetes-specific product.¹⁴

In a study evaluating long-term glycemic control, lipid responses, and clinical outcomes, elderly, tube-fed, long-term-care patients with type 2 diabetes (n=27) were randomized to receive either a standard, high-carbohydrate product or a reduced-carbohydrate, diabetes-specific product for three months. Differences in fasting and serum glucose and capillary glucose levels demonstrated better control with the diabetes-specific product. In addition, the amount of insulin administered was consistently higher in the group fed the standard formula. The group who were fed the diabetes-specific product were reported to have improved clinical outcomes of reductions in the incidence of fevers, pneumonia, and urinary tract infections.¹⁵

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McCargar et al assessed the long-term effect of a standard or diabetes-specific product enriched with monounsaturated fatty acids on carbohydrate and lipid metabolism in 32 patients with type 2 diabetes.¹⁶ The products were consumed for 28 days at > 80% of daily energy intake, with subjects self-monitoring their blood glucose levels before and two hours after each meal. The postprandial rise in capillary blood glucose was significantly lower in the group fed the diabetes-specific product than the standard product. Trends of clinical interest, but not statistical significance, were greater decreases in fructosamine and insulin observed with consumption of the diabetes-specific product. There were no differences in triglycerides and cholesterol between the two groups, leading the authors to conclude that monounsaturated fatty acids do not present any risk to lipoprotein metabolism in patients with type 2 diabetes.

A randomized, double blind, prospective study investigated the long-term effects of a low-carbohydrate, high-monounsaturated-fatty-acid diabetes product and a standard enteral product on fasting and postprandial blood glucose, total daily insulin, A1C, and lipid profile in 78 insulin-treated tube-fed patients with type 2 diabetes over a 12-week period. The results demonstrated significantly lower levels of fasting blood glucose, total daily insulin requirements, and A1C levels in patients receiving the diabetes-specific product compared to the standard product. There was no difference in postprandial glucose or lipid levels between the groups.¹⁷

A recent meta-analysis of 23 studies was conducted to determine whether diabetes-specific products are superior to standard products by comparing their effects on glycemia, lipidemia, medication requirements, and complications.¹⁸ Sixteen of the studies involved oral supplementation and seven involved tube feeding. Based on their analysis, the authors concluded that, whether consumed orally or tube-fed, diabetes-specific products improve glycemic control. In addition, the longer-term feeding studies reported a reduced requirement for insulin and fewer complications with diabetes-specific products compared to standard nutritional products.

Management Strategies and Treatment Goals

The complexities of diabetes mean differences in management for each individual with the disease, but the overriding goals are to prevent or delay microvascular and macrovascular complications and to improve overall health. Reaching these broad goals requires meeting and maintaining specific targets — normal plasma glucose, A1C, and lipid levels; a normal range of blood pressure; and appropriate body weight.

Major Goals for Diabetes Management

- Achieve glycemic control to prevent or limit microvascular complications
- Control lipid metabolism and blood pressure to prevent or limit macrovascular complications
- Balance food intake with energy output to control weight and improve overall health

Glycemic Control

Glycemic control for people with diabetes is targeted to specific measurable goals, although certain goals are adjusted to meet specific individual needs.¹⁹ A1C is the preferred measure of long-term glycemic control and the primary management target. A1C testing one to four times per year, depending on the individual's medical condition, is recommended for persons with any type of diabetes. Routine self-monitoring of blood glucose (SMBG), three or more times per day, is recommended for persons with type 1 diabetes; the frequency of testing may be less in type 2 patients on oral antihyperglycemic therapy, but more for those who take insulin with or without oral agents.^{19,20} Glycemic goals should be individualized appropriately for the age, gender, and health status of the patient; certain populations (children, pregnant women, and the elderly) require special considerations. Less-intensive glycemic goals may be indicated in patients who experience severe or frequent hypoglycemia, while more stringent glycemic goals (eg, A1C \leq 6%; fasting plasma glucose of 4.0-7.0 mmol/L; 2-hour postprandial plasma glucose of 5.0-8.0 mmol/L) may further reduce complications in some patients.^{20,21}

A1C, the primary target for glycemic control, is a test measuring the amount of glycosylated hemoglobin in the blood. Hemoglobin becomes glycosylated when glucose molecules bond with hemoglobin molecules of the red blood cells. Because the glucose remains attached for the life of the cell (ie, about 120 days), a test to measure A1C shows the person's average blood glucose level for that period of time. Test results reflect the sum of the fasting and postprandial blood glucose measurements.

Correlation between A1C level and mean plasma glucose levels on multiple testing over 2-3 months

A1C (%)	Mean Plasma Glucose (mg/dL)
6	135
7	170
8	205
9	240
10	275
11	310
12	345

These estimates are based on DCCT data (34). An updated version of this table, based on final results of the ADAG trial, will be available at www.diabetes.org after publication of the study's findings in 2008.

Diagram taken from: American Diabetes Association: Standards of Medical Care in Diabetes. *Diabetes Care* 2008;31(suppl 1):512-554.

Targets for glycemic control^{19*}

Parameter	Target
A1C	< 7.0%*
Preprandial capillary plasma glucose	70-130 mg/dL
Peak postprandial capillary plasma glucose [†]	< 180 mg/dL

* These goals are for nonpregnant individuals and are referenced to a nondiabetic range of 4.0-6.0% using a DCCT-based assay.

† Postprandial glucose measurements should be made 1-2 h after the beginning of the meal, generally peak levels in patients with diabetes.

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American Diabetes Association Recommendations for Inpatient Glucose Targets¹⁹:

Critically ill: Close to 110 mg/dL as possible; generally < 140 mg/dL

Non-Critically ill: Fasting < 126 mg/dL; all random blood glucose < 180-200 mg/dL

AACE/ACE Recommendations for Inpatient Glucose Targets¹:

Intensive care units: Maintain blood glucose <110 mg/dL

Non-Critical care units: Maintain premeal blood glucose <110 mg/dL and peak postprandial blood glucose < 180 mg/dL

Blood Lipid Control

Lipid management aimed at lowering LDL cholesterol, raising HDL cholesterol, and lowering triglycerides is important for type 2 diabetes (see table below).¹⁹ Effective lipid control has been shown to reduce macrovascular disease and mortality in patients with a history that includes cardiovascular events.²² Diabetes specialists advise testing adult diabetes patients for lipid disorders at least once each year and more often if needed to achieve goals.¹⁹ The recommended initial therapy for managing lipid levels is behavioral modification, including diet modification, weight loss, and increased exercise.¹⁹ The addition of lipid-lowering agents may be necessary to achieve lipid targets.¹⁹

Recommended Blood Lipid Levels¹⁹

Recommended Lipid Levels	
Total cholesterol	< 200 mg/dL
LDL cholesterol	< 100 mg/dL [†]
HDL cholesterol	> 40 mg/dL for men > 50 mg/dL for women
Triglycerides	< 150 mg/dL

* Current NCEP/ATP III guidelines suggest that in patients with triglycerides \geq 200 mg/dL, the “non-HDL cholesterol” (total cholesterol minus HDL) be used. The goal is \leq 130 mg/dL.

† This is the goal for individuals with diabetes aged > 40 years with a total cholesterol \geq 135 mg/dL without overt cardiovascular disease. An LDL cholesterol level of < 70 mg/dL (1.8 mmol/L) is recommended by the ADA for persons with diabetes and overt cardiovascular disease who are at very high risk for further events.

Summary

Clinical management of diabetes requires a multifaceted approach that makes use of pharmacotherapy (oral anti-diabetic agents and insulin), medical nutrition therapy, and lifestyle modification (physical activity and weight management), along with patient education and ongoing support.

3.0 References

1. American College of Endocrinology Position Statement on Inpatient Diabetes and Metabolic Control. *Endocr Pract* 2004;10:77-82.
2. American Diabetes Association: National Diabetes Fact Sheet, United States 2005. Available at: www.diabetes.org/uedocuments/NationalDiabetesFactSheetRev.pdf. Accessed February 22, 2007.
3. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 1993;329:977-986.
4. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 1998;352:837-853.
5. Clement S, Braithwaite SS, Magee MF, et al: Management of diabetes and hyperglycemia in hospitals. *Diabetes Care* 2004;27:553-591.
6. Van den Berghe G, Wouters P, Weekers F, et al: Intensive insulin therapy in the critically ill patients. *N Engl J Med* 2001;345:1359-1367.
7. Van den Berghe G, Wilmer A, Hermans G, et al: Intensive insulin therapy in the medical ICU. *N Engl J Med* 2006;354:449-461.
8. Lazar HL, Chipkin SR, Fitzgerald CA, et al: Tight glycemic control in diabetic coronary artery bypass graft patients improves perioperative outcomes and decreases recurrent ischemic events. *Circulation* 2004;109:1497-1502.
9. Mechanick JJ, Handelsman Y, Bloomgarden ZT: Hypoglycemia in the intensive care unit. *Curr Opin Clin Nutr Metab Care* 2007;10:193-196.
10. American Diabetes Association Standards of Medical Care in Diabetes, 2006. *Diabetes Care* 2006;29(suppl 1):S4-S42.
11. Charney P: Glycemic control in hospitalized patients: The RD's role. *Today's Dietitian* 2006;January:51-56.
12. Peters AL, Davidson MB, Isaac RM: Lack of glucose elevation after simulated tube feeding with a low-carbohydrate, high-fat enteral formula in patients with type 1 diabetes. *Am J Med* 1989;87:178-182.
13. Sanz-Paris A, Calvo L, Guallard A, et al: High-fat versus high-carbohydrate enteral formulae: effect on blood glucose, C-peptide, and ketones in patients with type 2 diabetes treated with insulin or sulfonylurea. *Nutrition* 1998;14:840-845.
14. Fix BM. Randomized, double blind, three way crossover comparison of glucose and insulin responses during a meal glucose tolerance test in subjects with type 2 diabetes consuming disease-specific versus standard nutritional formulas, Study SRDB09, Abbott Nutrition, Columbus, Ohio, 2003.
15. Craig LD, Nicholson S, Silverstone FA, et al: Use of a reduced-carbohydrate, modified-fat enteral formula for improving metabolic control and clinical outcomes in long-term care residents with type 2 diabetes: Results of a pilot trial. *Nutrition* 1998;14:529-534.
16. McCargar LJ, Innis SM, Bowron E, et al: Effect of enteral nutritional products differing in carbohydrate and fat on indices of carbohydrate and lipid metabolism in patients with NIDDM. *Mol Cell Biochem* 1998;188:81-89.
17. Pohl M, Mayr P, Merit-Roetzer M, et al: Glycaemic control in type II diabetic tube-fed patients with a new enteral formula low in carbohydrates and high in monounsaturated fatty acids: a randomised controlled trial. *Eur J Clin Nutr* 2005;59:1221-1232.
18. Elia M, Ceriello A, Laube H, et al: Enteral nutritional support and use of diabetes-specific formulas for patients with diabetes: a systematic review and meta-analysis. *Diabetes Care* 2005;28:2267-2279.
19. American Diabetes Association: Standards of Medical Care in Diabetes. *Diabetes Care* 2008;31(suppl 1):S12-S54.
20. O'Connell BS. Diabetes Classification, Pathophysiology, and Diagnosis. In: Ross TM BJ, O'Connell BS. *Diabetes Care and Education Dietetic Practice Group, ed. American Dietetic Association Guide to Diabetes; Medical Nutrition Therapy and Education: American Dietetic Association.* 2005;39-48.
21. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association, 2003 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada. *Can J Diabetes* 2003;27(suppl 2).
22. Haffner SM: Management of dyslipidemia in adults with diabetes. *Diabetes Care* 2003;26(suppl 1):S83-S86.

Case Examples/Product Application

ICU Scenario — Enhancing Patient Care

In many hospital critical care units, continuous insulin delivery via insulin drip is utilized in patients with hyperglycemia/diabetes to maintain tight glycemic control because tight glycemic control is highly correlated with better patient outcomes. At the same time, these patients are also receiving a standard tube-feeding formula.

Oftentimes, the patient may remain on this standard formula while transitioning out of the ICU to the general hospital floor and then when after discharged either to home care or long-term care. This transition becomes particularly problematic when managing insulin needs and blood glucose levels. As a result, patients are more likely to experience hyperglycemia because they are no longer receiving intensive insulin therapy and the insulin delivery schedule has not been adequately established. The use of a standard formula further exacerbates the issue. As a result, patient quality of life may suffer and the overall scenario can contribute to higher healthcare costs and poor outcomes.

Introducing a diabetes-specific formula in the intensive care unit and continuing to use the diabetes-specific formula can help ease this transition in terms of enabling better blood glucose control while providing the benefits of nutrition specifically tailored to meet patient needs.

Acute Care/Long-Term Care

A 74-year-old male, weight 195 lb (6'0", usual body weight of 190 lb), with diabetes, heart disease, and congestive heart failure is admitted to the hospital from an assisted-living facility. Patient is admitted to the hospital after suffering a stroke. Patient also suffers from elevated blood glucose (average fasting 180-210 mg/dL for 2 months, A1C = 9), and a stage 1 decubitus ulcer on his ankle. A PEG tube is eventually placed (type of tube for longer-term feeding) because swallowing function is compromised as a result of the stroke. Patient was managed with oral diabetes medication and changed to insulin due to inability to eat by mouth. Due to increased needs for healing of the ulcer and restricted fluids/volume secondary to congestive heart failure and aspiration risk, a more calorically dense formula is needed. Furthermore, a diabetes-specific formula to help manage blood glucose levels is key to help promote proper healing and blood glucose control. This patient remains in the hospital for 4 weeks during which his ulcer resolves, blood glucose levels are improved (average fasting 140 mg/dL), congestive heart failure is controlled, and swallowing function improves.

Calorie needs: 2580-2750 Cal/day

Protein needs: 103-129 g/day

Needs best met with Glucerna 1.2 Cal @ 90 mL/hr x 24 hr to provide 2592 Calories and 129 grams of protein/day

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After 4 weeks, this patient returns to a long-term skilled-care facility for an increased level of care. To make sure nutritional needs are met, the PEG tube for tube feeding will remain and patient will receive oral supplementation with thickened liquids and pureed foods. Anticipated intake from thickened liquids and pureed foods is approximately 500 kcal. The remaining needs will be met with Glucerna 1.2 Cal as protein needs and calorie needs are still elevated, less volume is still desirable secondary to congestive heart failure, and blood glucose management is still a priority. The American Heart Association recommends that patients with diabetes with heart disease consume 1 gram EPA + DHA/day, which is delivered via Glucerna 1.2 Cal. Less volume via a calorically dense formula is also desired because the remaining nutritional needs will be over 15 hours rather than 24 hours. Shortened feeding time will allow for the patient to more easily attend activities and speech therapy during the day.

Weight: 190 lb

Calorie needs: 2235-2580 Cal/day

Protein needs: 90-100 g/day

Needs best met with Glucerna 1.2 Cal @ 100 mL/hr x 15 hr to provide 1800 Calories and 90 grams of protein/day plus 500 Cal from thickened liquids and pureed foods

Glucerna

Glucerna products are for use under medical supervision as a part of a diabetes management plan.

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