HUNGER MINIMIZED JUICE FASTING SYSTEM

Applicants: Sudarshan Narasimhan, Flemington, NJ (US); Dave Narasimhan, Flemington, NJ (US)

Inventors: Sudarshan Narasimhan, Flemington, NJ (US); Dave Narasimhan, Flemington, NJ (US)

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ABSTRACT
The hunger minimized fasting system relies on providing a blood glucose level at all times in the range of 5 to 10 mM, satisfying glucose needs of the brain and metabolizing blood glucose through anaerobic glycolysis to release ATP at the extramitochondrial portion of the cell. Consuming only solid free nutrient liquids eliminates brain hunger response. Liquids consumed during fasting include solid filtered vegetable soup and clear fruit juices of 8 to 12 ounces taken every 2 to 4 hours having calorie deficiency of 600 to 1400 as compared to minimal daily calorie requirements for an adult. Fat released from storage is metabolized by ATP produced by glycolysis at the extramitochondrial location and enters the interior of the mitochondria, enabling the TCA cycle. Weight loss observed during fasting is about one half to one pound per day.
Fig. 1

Features of the Present Invention

- Fasting person consumes vitamins and minerals daily, juices and vegetable soup every 2-4 hours
- Blood glucose increases with low total calories consumed
- Hunger pangs are minimised during fasting
- Blood glucose -> Glycolysis forms Pyruvic acid & ATP at cell extramitochondria
- Pyruvic acid + CoA + ATP -> Acetyl CoA entering inner membrane of mitochondria producing ATP in TCA cycle
- Fat molecules released from fat storage due to low calorie intake & delivered to cell extramitochondria
- ATP at extramitochondria from glycolysis reacting with fat and CoA forming Acyl CoA entering mitochondria with the help of Carnitine
- Acyl CoA -- Acetyl CoA in mitochondria producing ATP in TCA cycle
Fig. 2A-1

**Stages of Glycolysis Preparatory Phase**

**Step 1** Glucose \((C_6H_{12}O_6)\) + Hexokinase + ATP  \(\rightarrow\) Glucose-6-phosphate \((C_6H_{11}O_6P)\)
+ ADP

**Step 2** Glucose-6-phosphate \((C_6H_{11}O_6P)\) + Phosphoglucoisomerase  \(\rightarrow\) Fructose-6-phosphate \((C_6H_{11}O_6P)\)
Fig. 2A-2

Step 3 Fructose 6-phosphate (C₆H₁₂O₅P₁) + phosphofructokinase + ATP $\rightarrow$ Fructose 1,6-bisphosphate (C₆H₁₀O₆P₁) + ADP

Step 4 Fructose 1,6-bisphosphate (C₆H₁₀O₆P₁) + aldolase $\rightarrow$ Dihydroxyacetone phosphate (C₃H₆O₅P₁) + Glyceraldehyde phosphate (C₃H₇O₅P₁)

Step 5 Dihydroxyacetone phosphate (C₃H₆O₅P₁) $\rightarrow$ Glyceraldehyde phosphate (C₃H₇O₅P₁)
Fig. 2B-1

**Stages of Glycolysis Pay-off Phase**

**Step 6** two Glyceraldehyde phosphate (C₃H₅O₇P₂) + Triose phosphate dehydrogenase + 2H⁺ + 2P + 2NAD⁺ --> two 1,3-bisphosphoglycerate (C₃H₅O₇P₂) + 2NADH + 2H⁺

**Prior Art**

![Diagram showing the reaction involving glyceraldehyde 3 phosphate dehydrogenase.

**Step 7** two molecules of 1,3 bisphosphoglycerate (C₃H₅O₇P₂) + phosphoglycerokinase + two ADP --> two molecules of 3-phosphoglycerate (C₃H₅O₇P₁) + two ATP

**Prior Art**

![Diagram showing the reaction involving phosphoglycerokinase.

**Step 8** two molecules of 3-phosphoglycerate (C₃H₅O₇P₁) + phosphoglyceromutase --> two molecules of 2-Phosphoglycerate (C₃H₅O₇P₁)

**Prior Art**

![Diagram showing the reaction involving phosphoglyceromutase.]
Fig. 2B-2

Step 9 two molecules of 2-phosphoglycerate (C₃H₅O₄P₁) + enolase → two molecules of phosphoenolpyruvic acid (PEP) (C₃H₅O₄P₁) + H₂O

PRIOR ART

\[
\begin{align*}
\text{H} - \text{C} - \text{OH} - \text{P} & \xrightarrow{\text{Enolase}} \text{H} - \text{C} - \text{O} - \text{P} \\
\text{CH₂OH} & \quad \quad \quad \quad \quad \quad \quad 2\text{H₂O} \\
\end{align*}
\]

Two 2-phosphoglycerate

Two 2-phosphoenolpyruvate

Step 10 two molecules of PEP (C₃H₅O₄P₁) + pyruvate kinase + 2 ADP → 2 molecules of pyruvic acid (C₃H₄O₃) + 2 ATP

PRIOR ART

\[
\begin{align*}
\text{C} - \text{O} - \text{P} & \xrightarrow{\text{Pyruvate kinase}} \text{H} - \text{C} = \text{O} \\
\text{CH₂} & \quad \quad \quad \quad \quad \quad \quad 2\text{ADP} \quad 2\text{ATP} \\
\end{align*}
\]

Two phosphoenolpyruvate

Two Pyruvate
Fig. 4

Prior Art

Pyrivic acid 3C
2H⁺

CO₂

Acetyl Co-A 2C

Coenzyme A

Citric acid 6C

H₂O

H₂O

Cis-aconitic acid 6C

H₂O

Isocitric acid 6C

Qxalosuccinic acid and 6C

CO₂

H₂O

α-ketoglutaric acid and 5C

2H⁺
**HUNGER MINIMIZED JUICE Fasting SYSTEM**

1. FIELD OF THE INVENTION

[0001] This method relates diet management; and, more particularly, to a fasting method that utilizes juices and vegetable soup to stave off hunger pangs by providing a high sugar and carbohydrate content that satisfies the brain glucose-glycogen need.

2. DESCRIPTION OF THE PRIOR ART

[0002] There is a global epidemic of obesity taking place, which leads to several obesity related diseases. There is a strong desire to effectively lose excess body weight. The present invention relates to a fasting method designed to lose excess body weight and body fat, and improve the functionality of critical body organs without creating hunger pangs as the duration of fasting effort progresses.

[0003] Many patents and prior art documents relate to fasting methods. These methods aim towards reducing the body weight of the fasting person by merely reducing the calorie intake by way of limiting the quantity of food consumed. These methods do not pay attention to body mechanisms that control brain function, the needs of muscle tissues, or the perception of hunger. Restriction of calorie intake generally results in the slowing down of the body metabolism and body quickly adapts to this calorie intake reduction and eliminating non-essential body functions in order to maintain core body functions. When a person exits from the fasting routine, body weight is quickly regained back to its original weight value or that is in excess of the original weight value. Prolonged deliberate fasting generally results in the fasting person feeling extremely tired and unable to move readily. In many cases, the muscle mass in the fasting person’s body is consumed to generate the required glucose or glycogen substitute for the brain function, and causes the loss of protein rich muscle tissue which is consumed to produce a glucose substitute that results in the loss of muscle mass, further weakening the fasting person’s body.

[0004] Generally people discontinue juice fasting routine pretty early in the process due to hunger pangs. They are deprived of energy, which is required for daily activities, or to support tasks that require muscular movement, or to have mental focus required for concentration, and this leads to a rapid failure of the juice fasting attempt. The method detailed herein addresses these issues and provides a unique solution.

[0005] U.S. Pat. No. 6,069,131 to Marsh discloses preoperative beverage composition and method of treatment. This specially formulated beverage composition is designed to be ingested by a pre-operative patient at least about 2 hours prior to administration of anesthesia. The beverage composition is a single-serving volume containing at least about 200 calories, which calories are primarily from a non-protein, non-fat source, such as one or more carbohydrates. The composition includes about 48 grams maltodextrin, about 6 grams fructose and about 6 grams glucose, in water with enough citric acid to provide a final solution pH of about 4.3. This beverage composition, when ingested during pre-operative fasting, at least about 2 hours prior to administration of anesthesia, encourages compliance with pre-operative fasting requirements; reduces the incidence of symptoms associated with prolonged fasting, such as feelings of hunger and thirst, lightheadedness, irritability and headache; and should reduce the risk of aspiration pneumonia by providing a residual gastric volume and gastric pH within generally accepted ranges. Also, contemplated herein is the method of using this beverage composition to increase compliance with pre-operative fasting guidelines and thereby decrease the risk of aspiration pneumonia in the anesthetized/sedated patient. This fasting beverage is designed only for a short time fast of typically two hours before administering an anesthetic and is not designed for a long time fast typically required for hunger free fasting that reduces body fat and improves body mass index.

[0006] U.S. Pat. No. 8,715,742 to Koide discloses method for reducing weight in a subject. This method is provided for reducing weight in a subject by administering an effective amount of a composition comprising omega-3 polyunsaturated fatty acid (PUFA), at least one of L-arginine, L-ornithine, an L-arginine precursor and an L-ornithine precursor, and at least one of nucleoside, a nucleoside and a nucleic acid. The method can also be used to treat obesity, hyperlipidemia, diabetes and/or hypertension and for improving diastasis, or treating adult disease or disposition to adult disease. This method does not provide adequate glucose or glycogen required for brain functioning and satiation.

[0007] U.S. Pat. No. 7,629,329 to Lee et al. discloses a method for increasing muscle mass and strength through administration of adenosine triphosphate. This method uses compositions that have an effective amount of Adenosine Triphosphate (“ATP”) sufficient to increase intracellular and extracellular concentrations of ATP in a mammal to improve anaerobic exercise capacity by increasing muscle size and/or strength. Preferably, a gastric acid secretory, inhibitory coating is applied to the effective amount of ATP in a manner that protects the ATP from degradation by gastric juices. ATP compositions may be administered in nutraceutical or functional food dosage forms, including oral and non-oral delivery forms. In addition, the effective amount of ATP may be combined with amino acids, botanicals, functional foods, herbs, nucleotides, nutraceuticals, nutrients, pharmaceuticals, proteins, and/or vitamins in an effort to enhance the targeted activity of the composition. In spite of the coating that protects the ATP from being destroyed by the gastric juices, the ATP levels are not increased for at least about 60 minutes from the time at which the ATP containing composition is consumed as indicated in the figures. The composition does not provide sustained or increased muscle activity after the consumption of the ATP containing composition, rather kicks in only after an hour later.

[0008] U.S. Pat. No. 7,825,084 to Harris, et al. discloses methods and compositions for increasing the anaerobic working capacity in tissues. The composition comprises a beta-alanylhistidine dipeptide and a glycine, an insulin, an insulin mimetic, or an insulin-action modifier and administering the composition to the tissue increases beta-alanylhistidine dipeptide synthesis in the tissue, thereby increasing the anaerobic working capacity in the tissue. The cause an increase in the blood plasma concentrations of beta-alanine and/or creatine. The composition contains artificial chemicals and does not contain natural ingredients or compounds indicated in the present invention disclosure.

[0009] U.S. Pat. No. 7,897,169 to Ueda, et al. discloses ubiquinol-enriched fat-containing foods. The process for producing a ubiquinol-enriched oil-fat-containing food product for human ingestion comprises dissolving ubiquinol in oil/fat under heating first followed by cooling to obtain homoge-
neous solution with a melting point of not lower than 20° C. the cooling action solidifying the homogenous composition. The solidified composition is kneaded to form oil-in-water emulsion. The composition formed is not a gel or paste like substance and is not contained in a ready to use individually packed pouches. The composition is oil based, not water based and does not have other nutrients than ubiquinol.

[0010] US Patent application 20110123653 to McKever et al. discloses compositions and methods for optimizing exercise recovery. The method decreases post-exercise recovery time in a subject using compositions that contain one or more polymethoxylated flavones (PMFs). The composition is an orange peel extract. The post-exercise recovery time is the time for a subject’s post-exercise oxygen consumption VO₂ level to return to a pre-exercise VO₂ level. PMF composition is selected from PMFs, which are selected from the group consisting of 5,6,7,3',4'-pentamethoxyflavone (sinenestin); 5,6,7,8,3',4'-hexamethoxyflavone (noebeatin); 5,6,7,8,4'-pentamethoxyflavone (tangeretin); 5-hydroxy-6,7,8,3',4'-pentamethoxyflavone (arranetin); 5-hydroxy-7,8,3',4'-methoxyflavone; 5,7-dihydroxy-6,8,3',4'-tetramethoxyflavone; 5,7,8,3',4'-pentamethoxyflavone; 5,7,8,4'-tetramethoxyflavone; 5,5',6,7,8,3',4'-heptamethoxyflavone; 5-hydroxy-3,6,7,8,3',4'-hexamethoxyflavone; 5-hydroxy-6,7,8,4'-tetramethoxyflavone; 5,6,7,4'-tetramethoxyflavone; 7-hydroxy-3,5,6,8,3',4'-hexamethoxyflavone; and 7-hydroxy-3,5,6,3',4'-pentamethoxyflavone. This composition merely reduces recovery time and does not increase muscle energy output during exercise. It is not a fasting routine and does not control hunger pangs.

[0011] The publication “Brain Glucose Sensing, Counter-regulation, and Energy Homeostasis” by Nell Marty, et al. published at PHYSIOLOGY 22: 241-251, 2007 available at web page at http://physiologyonline.physiology.org/content/22/4/241. This publication discloses methods by which the human brain monitors closely the blood glucose as a signal to control feeding behavior and energy expenditure. The glucose sensing neurons are highly represented in hypothalamic nuclei and the brain stem, regions involved in the control of energy homeostasis and food intake. Brain has very little or no storage of glucose or glycogen and does not have reserve ATP and requires continuous feed of blood glucose for brain operation. Stomach and intestine sense presence of food through Glucose-incretin secretion K-Cells (GIP) L-Cells (GLP-1) of the enteric nervous system.

[0012] The Lipolysis and the Oxidation of Fatty Acids by Michael W King is available at http://themedicalbiochemistry.org/fatty-acid-oxidation.html and describes a mechanism by which fatty acid is removed from storage and oxidized for liberation of energy at muscles, as well as body cells.

[0013] The web page at http://en.wikipedia.org/wiki/Fatty_acid Metabolism discloses that when blood sugar is low, decreasing insulin levels signal the adipocytes to activate hormone-sensitive lipase, and to convert triglycerides into free fatty acids. These free fatty acids have very low solubility in the blood, typically about 1 μM. However, the most abundant protein in blood, serum albumin, binds free fatty acids, increasing their effective solubility to ~1 mM. Thus, serum albumin transports fatty acids to organs such as muscle and liver for oxidation when blood sugar is low.

[0014] The web page at http://en.wikipedia.org/wiki/Beta Oxidation states that free fatty acids cannot penetrate the plasma membrane due to their negative charge. Once in the cytosol, activation of the fatty acid is catalyzed by long fatty acyl CoA synthetase. A fatty acid reacts with ATP to give a fatty acyl anhydride, plus inorganic pyrophosphate, which then reacts with free coenzyme A to give a fatty acyl-CoA ester plus AMP. If the fatty acyl-CoA has a long chain (10 or more carbons) then it is reacted with carnitine to form acyl carnitine, which is transported across the inner mitochondrial membrane by a Carnitine-acylcarnitine translocase. If the fatty acyl-CoA contains a short chain (less than 10 carbons) it can simply diffuse through the inner mitochondrial membrane.

[0015] A number of advertisements relate to fasting, reduction in body weight as well as improvement of high blood pressure, diabetes and other illnesses. These programs do not disclose their methodology or scientific basis for the methods used and expected results as a function of time.

[0016] There remains a need in the art for a safe and effective fasting system wherein the person undergoing the fasting procedure does not suffer excessive hunger or loss of muscle tissue without using synthetic medications and can effectively decrease body fat while maintaining full body energy.

SUMMARY OF THE INVENTION

[0017] The hunger minimized fasting system of the present invention utilizes a number of natural body processes to allow hunger free fasting without the feeling of hunger pangs for fasting periods lasting from 10 to 90 days. Prior art methods do not allow such prolonged fasts without the feeling of hunger. More importantly, with such prior art methods, the progressive fast decreases body energy available and a person becomes very weak and is generally unable to move. By way of contrast, the system of subject invention maintains blood sugar level in the range of 5 to 10 mM (90-180 mg/dL), which satisfies the brain’s requirement for a continuous supply of glucose, since the brain does not store much glycogen or ATP. The brain also has sensors in the gut, which sense the presence of solids in the digestive organs and invoke digestive juices, again creating hunger pains. The absence of solids in the juices or soups used during fasting, which include fruit juices of various fruits and vegetable soup that is filtered of all solids, suppresses the brain generated hunger sensation and maintains satiety during prolonged fasting. Entering the fasting phase requires a pre-fast phase of 3 to 5 days wherein only fruits are consumed in the morning followed by raw vegetables in the evening, again restricting overall calorie intake. The body quickly learns to extract nutrients from this diet and is now ready for entering the fasting stage.

[0018] The fasting stage comprises the inclusion of liquefied vitamins and minerals essential for producing all the enzymes and hormones needed to assimilate blood glucose created from the consumption of sugar or carbohydrate rich juices and soups. Since the calorie intake is deficient by about 600 to 1400 calories as compared to the required daily calorie requirement, fat reserves are used to generate sufficient calories for the daily functioning of the body with the help of hormone sensitive lipase.

[0019] The fasting step involves consumption of 8 to 12 ounces of various clear fruit juices and solid free prepared vegetable soups every 2 to 4 hours representing a caloric intake of 800 to 1200 calories only, which is deficient by about 600 to 1400 calories on a daily basis. The consumption of the juices results in a steady blood glucose level of 5 to 10 mM (90-180 mg/dL) satisfying the brain’s glucose need; the absence of solid material in the digestive track does not
invoke the brain hunger response; and the fasting person remains satisfied throughout the fasting period. The brain does not demand release of glycogen stored in the muscles and liver, a step that generally results in the weakening of the fasting person. If blood glucose is not available, the brain will demand the liver to attack muscle tissue that is in contact with blood to convert the muscle to glucose simulating releasing nitrogen rich waste. Eating protein or meat does not solve this problem since the eaten food is not in contact with blood. The technology of the subject invention avoids all these problems by brain demanding extraction of glycogen from muscles and liver or the degradation of muscle tissue. The system of the present invention maintains a consistent blood glucose level.

[0020] The blood glucose from the consumption of juices and soups is converted to pyruvic acid and two ATP molecules in the extramitochondrial portion of the cell by the glycolysis process as detailed below. This process is anaerobic and does not care if oxygen is present or not. The pyruvic acid reacts with coenzyme A in the presence of pyruvic acid dehydrogenase enzyme using a molecule of ATP creating Acetyl Co A that enters the inner barrier of the mitochondria undergoing TCA cycle, which produces many molecules of ATP. TCA cycle occurs aerobically within the mitochondria, which has all the enzymes needed for TCA cycle except succinate dehydrogenase.

[0021] Due to the combination of a reduced caloric intake and the brain having sufficient blood glucose levels, the only way the body can get adequate calories is by reaching out to consume stored fat tissues. Hormone sensitive lipase extracts fat from storage with the help of adrenaline hormone and is bound to blood serum albumin proteins and transported to cells and arrives at the extramitochondrial portion of the cell. Due to their negative charge these fats cannot enter the inner mitochondrial barrier. Once in the cytosol of the cell, activation of the fatty acid is catalyzed by long fatty acyl CoA synthetase. A fatty acid reacts with ATP to give a fatty acyl adenylate, plus inorganic pyrophosphate, which then reacts with free coenzyme A to give a fatty acyl-CoA ester plus adenosine monophosphate (AMP), precursor to ATP. If the fatty acyl-CoA has a long chain (10 or more carbons) then it is reacted with carnitine to form acylcarnitine, which is transported across the inner mitochondrial membrane by a Carnitine-acylcarnitine translocase. The acyl-CoA and adenosine monophosphate (AMP), precursor to ATP undergo TCA cycle producing large number of ATP molecules.

[0022] Thus the technology of the subject invention uses body’s metabolic processes to force the extraction of stored fat and conversion to ATP energizing body cells. The key feature is the presence and accumulation of ATP formed by the glycolysis process in the extramitochondrial portion of the cell. From blood glucose, the glycolysis process produces ATP and pyruvic acid, which is converted to Acetyl CoA that enters the inner mitochondria barrier as stated above. The fat molecules from fat storage are released into the blood stream by hormone sensitive lipase with the help of adrenaline hormone and transported bound to serum albumin arriving at the extramitochondrial portion of the cell, the very place where glycolysis produces ATP and is readily available. Once in the cytosol, activation of the fatty acid is catalyzed by long fatty acyl CoA synthetase. The fatty acid reacts with ATP to give a fatty acyl adenylate, plus inorganic pyrophosphate, which then reacts with free coenzyme A to produce a fatty acyl-CoA ester plus adenosine monophosphate (AMP), precursor to ATP. If the fatty acyl-CoA has a long chain (10 or more carbons) then it is reacted with carnitine to form acylcarnitine, which is transported across the inner mitochondrial membrane by a Carnitine-acylcarnitine translocase. During beta oxidation within mitochondria, acyl-CoA ester is produced and undergoes TCA cycle creating many molecules of ATP. If ATP is absent, the fat in the form of triglycerides and monoglycerides cannot enter the mitochondrial inner barrier and are returned back to storage. Thus the presence of ATP produced by glycolysis of sugar and carbohydrate rich juices and soups is essential for the metabolism of fats in the mitochondria. This is the mechanism by which both sugar and fat are converted to ATP in the TCA cycle producing large number of ATP molecules. This is the central feature of the invention.
and liver enabling the muscles to work without being tired. Due to the presence of stable blood glucose levels, the brain does not order the muscles to be degraded by liver enzymes to release sugar like products into the blood stream saving the muscle mass. Since the fasting person does not perceive hunger, the fasting can be continued for long durations like 90 days easily without feeling of hunger, deprivation or feelings of muscle weakness and tiredness. The hunger free fasting may be conducted for a number of days at the fasting person’s home or in a fasting center or a hospital.

[0026] Significant advantages are realized by practice of the present invention. In a preferred embodiment, the hunger minimized fasting system of the present invention comprises:

[0027] 1) preparing fasting person for 3 to 5 days prior entering fasting phase;

[0028] 2) providing liquefied vitamin and mineral supplement on a daily basis enabling production of numerous enzymes for use in glycolysis, the process of conversion blood glucose to pyruvic acid and adenosine triphosphate (ATP) as well as beta oxidation of fats in the TCA (Kreb) cycle;

[0029] 3) consuming on a daily basis clear solid free vegetable soup of 8 to 12 ounces in the morning and before bed time and consuming juices every 2 to 4 hours during waking hours representing a caloric intake of only 800 to 1200 calories representing a deficit of 600 to 1400 calories maintain blood glucose levels in the range of 5 to 10 mM (90 to 180 mg/dl) preventing onset of hunger sensing mechanism present the brain hypothalamus and brain stem preventing or minimizing hunger sensation of the fasting person and the blood glucose entering anerobic glycolysis process synthesizing pyruvic acid and two ATP molecules in the extramitochondrial portion of the cell;

[0030] 4) absence of solid material in the digestive organs including stomach and intestine reducing or eliminating response from brain hunger sensing mechanism of gut sensors and hormones by K-cells and L-cells;

[0031] 5) said reduction in caloric intake with blood sugar levels maintained in the satiety region causes fat molecules from fat storage to be released into the blood stream by hormone sensitive lipose with the help of adrenaline hormone and transported bound to serum albumin arriving at the extramitochondrial portion of the cell the very place where glycolysis produces ATP and is readily available, fatty acid reacts with ATP to give a fatty acyl adenylate, plus inorganic pyrophosphate, which then reacts with free coenzyme A to produce a fatty acyl-CoA ester plus adenosine monophosphate (AMP), precursor to ATP which enters the inner barrier of the mitochondria with the help of carnitine to form acylcarnitine during beta oxidation to produce acyl-CoA ester that undergoes TCA cycle with the mitochondria and producing many molecules of ATP, the essential constituent for all cell operations; and

[0032] 6) exiting the fast follows the same procedure as the fast entry phase, but adding additional insoluble fibers such as whole flax seeds, chia seeds or bran flakes as well as adding pro-biotic microbres to adjust the digestive system into re-accepting solids;

[0033] whereby said brain hunger sensing mechanism is minimized or prevented and glycogen is not depleted from muscles and liver allowing movement and exercise of the fasting person while all the muscle tissue are preserved from any muscle degradation.

BRIEF DESCRIPTION OF THE DRAWINGS

[0034] The invention will be more fully understood and further advantages will become apparent when reference is had to the following detailed description of the preferred embodiments of the invention and the accompanying drawings in which:

[0035] FIG. 1 illustrates the key features of the present invention wherein ATP is available at the extramitochondrial portion of the cell due to glycolysis of sugars and carbohydrates consumed and the fats also arrive at the same location allowing decomposed products of glycolysis and fat enter the mitochondria due to the presence of ATP;

[0036] FIGS. 2A-1 and 2A-2 illustrate the steps of the Preparatory Phase of Glycolysis that occurs in the extramitochondrial portion of the cell showing loss of 2 ATP molecules;

[0037] FIGS. 2B-1 and 2B-2 illustrate the steps of the Pay-off Phase of Glycolysis that occurs in the extramitochondrial portion of the cell showing gain of 4 ATP molecules;

[0038] FIG. 3 illustrates the beta oxidation of fat brought onto a cell that occurs within the mitochondrial portion of the cell; and

[0039] FIG. 4 illustrates the TCA cycle that occurs within the mitochondrial portion of the cell.

DETAILED DESCRIPTION OF THE INVENTION

[0040] This invention relates to a system for fasting during a prolonged period without hunger pangs and without becoming very tired or unable to move and exercise. The system also prevents the loss of muscle tissue while losing a significant amount of body weight which includes fat and water, on a daily basis, typically in the range of half to one pound.

[0041] The present invention employs a unique approach during juice fasting that provides benefits to the person undergoing the procedure. It was surprisingly found that the degree of hunger perceived during juice fasting is dependent on numerous brain hunger sensing functions. The brain tissue has very little storage of glucose or glycogen or adenosine triphosphate (ATP) molecules and requires a continuous supply of glucose for functioning of the brain at about 5 mM or 90 mg/dl. As a primary sensing mechanism, the hypothalamus and brain stem monitors very closely the blood glucose or glycogen levels and provides strong hunger sensations when the blood glucose or glycogen levels are low, creating a strong urge to eat. If the glucose or glycogen levels are not replenished, the brain commands the muscles and liver to release stored glycogen into the blood stream. If this stored glycogen is unavailable the brain commands the liver to degrade muscles to produce glucose simulant at the expense of normally unused muscle tissue. Again, when the blood glucose or glycogen levels are high the insulin release is triggered converting the glucose or glycogen in the blood to be combined to form triglycerides which are transferred for storage as adipose tissue. In addition, the fat release mechanism is immediately stopped when blood glucose concentration is too high. Thus the key to hunger free fasting is to provide an adequate amount of blood glucose or glycogen levels to satisfy the brain's need for glucose without exceeding glucose levels that trigger a release of insulin resulting in the formation of triglycerides. Also, when high blood glucose levels are present, the release mechanism from fat storage is halted. The

[0042] Thus the strategies for hunger free fasting involve drinking cooked vegetable soup that is filtered of all solid material and 8 to 12 ounce of juice every 2 to 4 waking hours. Breakfast and pre-bedtime meal is essentially the vegetable filtered soup. The overall carbohydrates contained in the juice and vegetable soup is only in the range of 800 to 1200 calories leaving a calorie deficit of about 600 to 1400 calories for a person normally consuming 1800-2400 calories per day.

[0043] The body requires these calories for the functioning of essential body functions including breathing, supporting the liver, heart and lung function as well as maintaining body temperature. This calorie deficiency has to be made up by use of body fat since fat is the primary fuel used by the body for all aerobic muscles, which contain fat burning brown muscle fibers intermixed with capillaries supplying blood oxygen rich blood. All cells and muscles performing steady work such as heart muscles and muscles that provide body stability are all aerobic muscles of this type. The next aspect of the present invention is to facilitate the release of fat in the form of tri-glycerides or mono-glycerides from lipid storage.

[0044] The food consumed, whether in the form of carbohydrates or sugars, converts to glucose in the blood stream either rapidly or slowly depending on the glycemic index of the food consumed. Proteins are more slowly digested and also convert to glucose in the blood stream. Fats take a very long time to digest and enter the intestinal walls after enzyme action that breaks down fat into tri or mono glycerides. Fats do not convert to glucose in the blood stream.

[0045] Since the amount of food in the stomach and intestine is reduced the ability to eliminate waste is minimized. Enemas or suppositories may be used to improve evacuation.

[0046] The utilization and conversion of glucose and fats into adenosine triphosphate (ATP) through different initial pathways as detailed in FIGS. 1A-1, 1A-2, 1B-1 and 1B-2 are shown. The TCA cycle is illustrated in FIG. 2 which produces ATP in aerobic conditions.

[0047] FIG. 1 illustrates a block diagram of the features of the subject invention. When the fasting person consumes a sugar rich and or carbohydrate rich juice or soup, the brain detection of hunger is satiated. This blood glucose is processed by glycolysis, which produces two pyruvic acid molecules and two ATP molecules per molecule of glucose. The pyruvic acid reacts with coenzyme A aided by ATP to produce acetyl CoA, which enters the mitochondria and undergoes TCA cycle producing a number of ATP molecules powering the cell operation. Since the amount of calories consumed by this juice and soup diet is smaller than that is required for the sustenance of the body, the deficient calories are obtained from fats. Hormone sensitive lipase is released at the fat reserves as mono and triglycerides and carried by the blood bound to serum albumen and delivered at the extramitochondrial portion of the cell. The released fat in the extramitochondrial region of the cell reacts with coenzyme A assisted by energy rich ATP molecule to form acyl CoA which enters the inner barrier of mitochondria assisted by carnitine. Within the mitochondria the acyl CoA converts to acetyl CoA, by the process known as betu oxidation, which is passed on to the TCA cycle producing a large number of ATP molecules. The key feature of the invention is making ATP available at all times due to consumption of sugar and carbohydrate rich juices and or soups every two to four hours while at the same time limiting the total calorie intake so as to force the body to release fat from storage. The fat arrives at the extramitochondrial portion of the cell and is again processed due to the presence of ATP at this location. The fasting person does not feel hungry even after fasting for a number of days and the fat is drawn from fat reserves and used as calories, improving the weight and BMI of the person. The muscles of the fasting person are not degraded during fasting.

[0048] FIGS. 2A-1 and 2A-2 illustrates the preparatory phase of glycolysis where two ATP molecules are consumed as shown in Steps 1 and 3. A large number of enzymes are involved in Steps 1 through 5 of the preparatory phase each performing a specific function.

[0049] FIGS. 2B-1 and 2B-2 illustrates the pay-off phase of glycolysis where four ATP molecules are produced as shown in Steps 7 and 10. Thus the glycolysis, which is a combination of steps 1 through 10 produce two excess molecules of ATP. A large number of enzymes are involved both in the preparatory and payoff phases each performing a specific function.

[0050] Glycolysis is an anaerobic metabolic pathway that has a sequence of 10 steps all of which are enzyme catalyzed. Accordingly, the sequence of these reactions converts glucose into pyruvate, producing two additional ATP molecules from each glucose molecule. This metabolic process produces high-energy compounds of ATP (Adenosine triphosphate) and NADH (nicotinamide adenine dinucleotide). This process is an anaerobic reaction; the presence or absence of oxygen does not alter the reaction. The process of glycolysis happens in the extramitochondrial portion of the cell, often referred to as the EMP pathway. Glucose undergoes partial oxidation to produce two molecules of pyruvic acid, which is the starting point of the tri-carboxylic acid cycle (TCA cycle), which is also known as the citric acid cycle or Kreb cycle. The TCA cycle takes place aerobically within the mitochondria portion of the cell. All these processes of glycolysis and the TCA cycle require a large number of enzymes which are all synthesized by the cells and liver.

[0051] FIG. 3 illustrates the beta oxidation of fatty acids taken from slide number 29 of web page http://www.authorstream.com/Presentation/sGuest38680-330425-beta-oxidation-lipids-education-ppt-powerpoint. Beta oxidation of fatty acids is shown in this figure. Long chain acyl-CoA is cycled through reactions 2 through 5. Acyl-CoA is split off by thiolase as shown at reaction 5. ATP is needed for the first step of the beta oxidation process outside the inner mitochondrial membrane as shown. Carnitine is needed for the entry of acyl-CoA into the inner mitochondrial membrane. The fatty acid is degraded to acetyl CoA and enters the citric acid cycle as shown. All the enzymes used outside the inner mitochondrial membrane have to be manufactured by liver and the cell.

[0052] FIG. 4 illustrates the TCA cycle. This TCA cycle is detailed at the web page http://biology.tutorvista.com/cell/glycolysis.html. The citric acid cycle is a sequence of enzyme-catalyzed chemical reactions, which are used by all
the aerobic organisms to produce energy. Energy is generated through the oxidation of acetate that is derived from carbohydrates, fats and proteins into carbon dioxide. Pyruvate molecules are created from glycolysis. In the presence of oxygen, pyruvate produces acetyl-CoA by reaction with coenzyme A consuming one ATP. The fats are also degraded to acetyl-CoA and brought into the mitochondria. In the presence of oxygen, the acetyl-CoA produced by glycolysis or fat degradation enters the citric acid cycle inside the matrix of the mitochondria and it gets oxidized to CO₂, and also at the same time reduces NAD⁺ to NADH. H₂O and CO₂ are the waste products created during this cycle. The cycle consists of eight steps, which are catalyzed by eight different enzymes. The steps are detailed below.

[0053] Step 1: Synthesis of Citric Acid. This step of the Krebs cycle is an aldol condensation reaction and it is an irreversible reaction. Oxaloacetic acid and the acetyl CoA condense to form citric acid in the presence of the enzyme citrate synthase. The net effect of this reaction is to join a two-carbon with a four-carbon molecule, which yields a six-carbon molecule which is the citric acid. This is called the synthesis of citric acid.

[0054] Step 2: Dehydration of citrate. It is a reversible reaction. Under the action of the enzyme citrate, citrate is isomerized to form isocitrate.

[0055] Step 3: Oxidation and Decarboxylation of isocitrate. This reaction is catalyzed by the enzyme isocitrate dehydrogenase. This is an irreversible reaction where isocitrate undergoes oxidative decarboxylation yielding three NADH molecules. These are the first NADH molecules produced in the cycle and also CO₂.

[0056] Step 4: Oxidative, decarboxylation of α-ketoglutarate. The enzyme α-ketoglutarate dehydrogenase complex catalyzes the conversion of α-ketoglutarate to succinyl CoA. This reaction produces the second CO₂ and also the second NADH of the cycle. The coenzymes that are required in the reaction are thiamine pyrophosphate, lipoic acid, FAD, NAD⁺ and CoA.

[0057] Step 5: Substrate level phosphorylation. This reaction is catalyzed by the enzyme succinyl-CoA synthetase. This reaction is exothermic and is GTP molecule, which is equivalent to ATP is generated in this reaction. The product of this reaction is succinicate and GTP.

[0058] Step 6: Oxidation. This reaction is catalyzed by the enzyme succinate dehydrogenase, in this reaction the final electron acceptor is the FAD coenzyme. This reaction yields two ATP molecules from the electron transport chain.

[0059] Step 7: Hydration. The hydration reaction is catalyzed by the enzyme fumarase. The fumarate is hydrated to form L-Malate.

[0060] Step 8: Oxidation. This is reversible reaction, catalyzed by the enzyme malate dehydrogenase. The malate is oxidized to form oxaloacetic acid. This is the final point of entry to the electron transport chain. This reaction generates the NADH and oxaloacetate.

[0061] Accordingly one pyruvic acid molecule yields one ATP molecule and one GTP molecule, which is equivalent to ATP. Also, NADH is an energetic molecule capable of producing ATP.

[0062] As indicated in http://en.wikipedia.org/wiki/Beta_oxidation a fat molecule produces a large number of ATP molecules. The ATP yield for every oxidation cycle is theoretically at maximum yield of 17, as NADH produces 3 ATP, FADH₂ produces 2 and a full rotation of the Citric Acid Cycle produces 12. In practice it’s closer to 14 ATP for a full oxidation cycle as in practice the theoretical yield isn’t attained, it’s generally closer to 2.5 ATP per NADH molecule produced, 1.5 for each FADH₂ molecule produced and this equates to 10 ATP molecules per cycle of the TCA (according to the P/O ratio).

[0063] As detailed at http://en.wikipedia.org/wiki/Beta_oxidation, beta-oxidation is the process by which fatty acid molecules are broken down in the mitochondria to generate acetyl-CoA, which enters the citric acid cycle, and NADH and FADH₂, which are used by the electron transport chain. Fatty Acid Catabolism involves three stages. The first stage of fatty acid catabolism is Beta-Oxidation. The second stage is acetyl CoA oxidation to carbon dioxide. The third stage is electron transfer from electron carriers to the electron transfer chain. Priming the fatty acid for oxidation is the ‘Carnitine Shuttle’. First Acyl-CoA is transferred to the hydroxyl group of carnitine by carnitine palmitoyltransf erase 1 (palmitoyltransferase) located on the outer mitochondrial membrane. Acyl-carnitine is shuttle inside by a carnitine-acylcarnitine translocase. Acrlycarnitine is converted back to acyl-CoA by carnitine acyltransferase (palmitoyltransferase) located on the inner mitochondrial membrane. The liberated carnitine returns to the cytosol for further transport of fatty acid.

[0064] Once the fatty acid is inside the mitochondrial matrix, Beta Oxidation can begin. It has 4 steps. Step 1 of Beta-Oxidation: Long chain fatty acid is dehydrogenated to create a trans double bond between C2 and C3. This is catalyzed by the fatty acyl-CoA dehydrogenase to produce trans-delta 2-enoyl-CoA. It uses FAD as an electron acceptor and it is reduced to FADH₂. Step 2 of Beta-Oxidation: Trans-delta 2-enoyl-CoA is hydrated at the double bond to produce L-B-hydroxyacyl-CoA. This is catalyzed by enoyl-CoA hydratase. Step 3 of Beta-Oxidation: L-B-hydroxyacyl-CoA is dehydrogenated again to create L-ketoacetyl CoA by B-hydroxyacyl-CoA dehydrogenase. This enzyme uses NAD as an electron acceptor. Step 4 of Beta-Oxidation: Thiolysis occurs between C2 and C3 (alpha and beta carbons) of L-ket acyl CoA. Thiolase enzyme catalyzes the reaction when a new molecule of coenzyme A breaks the bond by nucleophilic attack on C3. This releases the first two carbon units, as acetyl-CoA, and a fatty acyl-CoA minus two carbons. The process continues until all of the carbons in the fatty acid are turned into acetyl-CoA. Acetyl-CoA is the starting point for the TCA cycle as shown above.

[0065] The following examples are presented to provide a more complete understanding of the invention. The specific techniques, conditions, materials, proportions and reported data set forth to illustrate the principles and practice of the invention are exemplary and should not be construed as limiting the scope of the invention.

[0066] The fasting program of the subject invention can be practiced for a long time without hunger pangs, loss of muscle as detailed in actual test case results.

**EXAMPLE 1**

[0067] The first test subject is male 40 years old who conducted a fasting study by fasting for 11 weeks followed by monitoring the body for 12 additional weeks following the fast. The following table 1 details the results. The fast was started on Jun. 8, 2013 and terminated on Aug. 17, 2013 representing a weight loss of (68-48) or 16 kilograms or 35 pounds. The measured fat percentile, as measured at a professional gym, decreased from 18% at the start of the fast to
5% at the end of the 70-day fast. Having changed the eating habits due to this prolonged fast, the weight and body fat content remained stable as shown in the table. The percent of muscle during fast did not decrease, but increased as shown due to loss of body weight and remained stable for the 12 weeks after ending the fast.

**TABLE 1**

<table>
<thead>
<tr>
<th>Date</th>
<th>Begin Fast</th>
<th>Week</th>
<th>Kg</th>
<th>Lbs</th>
<th>%</th>
<th>Body fat</th>
<th>%</th>
<th>Body muscle</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jun. 8, 2013</td>
<td>1</td>
<td>84</td>
<td>184.8</td>
<td>15.1</td>
<td>18%</td>
<td>39.4</td>
<td>47%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jun. 15, 2013</td>
<td>2</td>
<td>81</td>
<td>178.2</td>
<td>12.2</td>
<td>15%</td>
<td>39.3</td>
<td>49%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jun. 22, 2013</td>
<td>3</td>
<td>77.1</td>
<td>169.6</td>
<td>8.3</td>
<td>11%</td>
<td>39.3</td>
<td>51%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jun. 29, 2013</td>
<td>4</td>
<td>76</td>
<td>167.2</td>
<td>7.5</td>
<td>10%</td>
<td>39.1</td>
<td>51%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jul. 6, 2013</td>
<td>5</td>
<td>74.1</td>
<td>163.0</td>
<td>6.9</td>
<td>9%</td>
<td>38.2</td>
<td>52%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jul. 13, 2013</td>
<td>6</td>
<td>72.6</td>
<td>159.7</td>
<td>4.8</td>
<td>7%</td>
<td>38.5</td>
<td>53%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jul. 20, 2013</td>
<td>7</td>
<td>71.5</td>
<td>157.3</td>
<td>4.3</td>
<td>6%</td>
<td>38</td>
<td>53%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jul. 27, 2013</td>
<td>8</td>
<td>69.7</td>
<td>153.3</td>
<td>4.9</td>
<td>7%</td>
<td>36.8</td>
<td>53%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aug. 3, 2013</td>
<td>9</td>
<td>69.5</td>
<td>152.9</td>
<td>4.1</td>
<td>6%</td>
<td>37.1</td>
<td>53%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aug. 10, 2013</td>
<td>10</td>
<td>67.7</td>
<td>148.9</td>
<td>3.3</td>
<td>5%</td>
<td>36.5</td>
<td>54%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aug. 17, 2013</td>
<td>11</td>
<td>68</td>
<td>149.6</td>
<td>3.5</td>
<td>5%</td>
<td>36.3</td>
<td>53%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**EXAMPLE 2**

A second subject is a 70-year-old male that conducted a fasting study for 17 days. The daily weight data is shown in Table 2. The first three days represent the pre-fast period and fasting is done for 8 days. The next three days were spent readjusting to a normal diet. Even this short fast resulted in a weight loss of (171-158.8) or 12.2 pounds. During fasting it was clearly apparent that exercise could be done.

**TABLE 2**

<table>
<thead>
<tr>
<th>Date</th>
<th>Pre-Fast</th>
<th>day #</th>
<th>Pounds</th>
<th>Jog/walk distance (mi)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jun. 11, 2014</td>
<td>1</td>
<td>171</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>Jun. 12, 2014</td>
<td>2</td>
<td>169</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>Jun. 13, 2014</td>
<td>3</td>
<td>168</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

**End Fast**

<table>
<thead>
<tr>
<th>Date</th>
<th>Jun. 14, 2014</th>
<th>4</th>
<th>167</th>
<th>2.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jun. 15, 2014</td>
<td>5</td>
<td>166</td>
<td>8.0</td>
<td></td>
</tr>
<tr>
<td>Jun. 16, 2014</td>
<td>6</td>
<td>165.2</td>
<td>2.3</td>
<td></td>
</tr>
<tr>
<td>Jun. 17, 2014</td>
<td>7</td>
<td>163.8</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>Jun. 18, 2014</td>
<td>8</td>
<td>163.2</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>Jun. 19, 2014</td>
<td>9</td>
<td>161.2</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>Jun. 20, 2014</td>
<td>10</td>
<td>160.4</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>Jun. 21, 2014</td>
<td>11</td>
<td>158.8</td>
<td>5.2</td>
<td></td>
</tr>
</tbody>
</table>

**[0069]** Having thus described the invention in rather full detail, it will be understood that such detail need not be strictly adhered to, but that additional changes and modifications may suggest themselves to one skilled in the art, all falling within the scope of the invention as defined by the subjoined claims.

What is claimed is:

1. A hunger minimized fasting system, comprising:
   a) preparing a fasting person for 3 to 5 days prior entering a fasting phase;
   b) providing liquefied vitamin and mineral supplement on a daily basis enabling production of numerous enzymes for use in glycolysis, the process of conversion blood glucose to pyruvic acid and adenosine triphosphate (ATP) as well as beta oxidation of fats in the TCA (Krebs) cycle;
   c) providing on a daily basis juices, clear solid free vegetable soup of 8 to 12 ounces every 2 to 4 hours during waking hours representing a caloric intake of only 800 to 1200 calories representing a deficit of 600 to 1400 calories, maintaining blood glucose levels in the range of 5 to 10 mM (90 to 180 mg/dL), preventing the onset of hunger sensation mechanism present in the brain hypothalamus and brain stem preventing or minimizing the hunger sensation of the fasting person;
   d) said clear solid free vegetable soup formed by lightly cooking vegetables in water at a temperature of about 80° C. to 95° C. to extract vitamins and minerals and micro nutrients and filtering out all solid material;
   e) the blood glucose entering anaerobic glycolysis process synthesizing two pyruvic acid molecules and two ATP molecules per glucose molecule in the extramitochondrial portion of the cell;
   f) absence of solid material in the digestive organs including stomach and intestine reducing or eliminating brain hunger response from gut sensors and hormones by K-cells and L-cells;
   g) said reduction in caloric intake with blood sugar levels maintained in the satiety region causing fat molecules from fat storage to be released into the blood stream by hormone sensitive lipase with the help of adrenaline hormone and transported bound to serum albumin arriving at the extramitochondrial portion of the cell the very place where glycolysis produces ATP and is readily available, fatty acid reacts with ATP to give a fatty acyl adenylate, plus inorganic pyrophosphate, which then reacts with free coenzyme A to produce a fatty acyl-CoA ester plus adenosine monophosphate (AMP), precursor to ATP which enters the inner barrier of the mitochondria with the help of carnitine to form acylcarnitine during beta oxidation to produce acyl-CoA ester and that undergoes the TCA cycle within the mitochondria, and producing many molecules of ATP, the essential constituent for all cell operations; and...
h) exiting the fast following the same procedure as the fast entry phase, but adding additional insoluble fibers such as whole flax seeds, chia seeds or bran flakes to adjust the digestive system for accepting solids; whereby the overall calories of juices and vegetable soup consumed on a daily basis fall well below daily minimum calorie requirements of the body by about 600 to 1400 calories necessitating use of stored body fat or lipids to enable the functioning of body tissues and muscles; whereby said brain hunger sensing mechanism is minimized or prevented and glycogen is not depleted from said fasting persons's muscles and liver, thereby allowing movement and exercise of the fasting person while all muscle tissues are preserved from any muscle degradation.

2) The hunger minimized fasting system as recited by claim 1, wherein said juices are clear solid free juices including orange juice, apple juice, pineapple juice, grape juice, pomegranate juice, coconut water, watermelon juice, cantaloupe juice, carrot juice, beetroot juice, celery juice, or combinations thereof.

3) The hunger minimized fasting system as recited by claim 1, wherein said clear solid free vegetable soup contains carrots, broccoli, beets, celery, potatoes, sweet potatoes and other vegetables.

4) The hunger minimized fasting system as recited by claim 1, wherein said fasting stage is in the range of 10 to 90 days.

5) The hunger minimized fasting system as recited by claim 1, wherein said fasting stage is extended to 120 days.

6) The hunger minimized fasting system as recited by claim 1, wherein said fasting stage cannot be restarted after exiting phase without going through the preparing phase which is offset from the exiting stage by at least 4 weeks.

7) The hunger minimized fasting system as recited by claim 1, wherein said exit phase of fasting includes the addition of higher levels of calories.

8) The hunger minimized fasting system as recited by claim 1, wherein said clear solid free vegetable soup formed by lightly cooking vegetables in water at 80° C. to 95° C. for 20 to 30 minutes to extract vitamins and minerals and micro nutrients and filtering out all solid material and cooling the filtered soup.

9) The hunger minimized fasting system as recited by claim 1, the body weight of the fasting person decreases by about 0.5 to 1 pound per day of fast, the decrease being largest in the initial stage of the fast and decreases gradually.

10) The hunger minimized fasting system as recited by claim 1, the body weight increases by about 5 to 10 percent as the fasting person gradually resumes normal diet.

11) The hunger minimized fasting system as recited by claim 8, wherein the soup is prepared by cooking the vegetables in water at a temperature of 80° C. to 95° C. for 30 minutes.

12) The hunger minimized fasting system as recited by claim 1, wherein evacuation is facilitated by use of enemas or suppositories.

13) The hunger minimized fasting system as recited by claim 1, wherein the fast is conducted at the home of the fasting person.

14) The hunger minimized fasting system as recited by claim 1, wherein the fast is conducted at a fasting facility or hospital.

15) A method for hunger minimized fasting, comprising:

a) preparing a person's body for fasting consuming only fruits in the morning, mid day and switching over to raw vegetables in the evening and prior to bed time for three to five days;

b) starting each day of fast by consuming 8 to 12 ounce of solid free vegetable soup with solid free liquefied vitamin and mineral supplement;

c) consuming 8 to 12 ounces of fruit juices every 2 to 4 hours throughout waking hours;

d) consuming 8 to 12 ounces of solid free vegetable soup with solid free liquefied vitamin and mineral supplement prior to bed time;

whereby hunger pangs are minimized even when steps b), c) and d) are followed every day throughout the fasting period which is less than 90 days.

* * * * *