increased and some being decreased and associated via statistical method to disease risk 3 to 5 years later.

SUMMARY

[0004] Creating a healthy intestinal environment is important for the overall health of the mammal. The inventors have discovered a means of providing or removing key metabolites and/or their precursors in the intestine in amounts sufficient to change the overall intestinal metabolome. The abundance of key metabolites can act in nutritive, absorptive, metabolic and immunological functions to promote the overall health of the mammal. These metabolites can also be administered in a therapeutic capacity to restore homoeostasis in conditions of altered metabolic (*i.e.,* obesity, Type 2 diabetes) and cognitive function (*i.e.*, cognitive development, learning, depression).

[0005] These metabolites may be increased or decreased alone or in combination to modulate the physiology and biochemistry of the infant gut. The present disclosure provides for compositions, methods and protocols to provide adequate levels of these compounds to restore and promote nutritional and metabolic health of the intestine, and health of other key organs including the liver and central nervous system. Monitoring the status of the some or all of the metabolites may be used to identify persons at risk of developing diseases in the future. Individuals identified as being at risk for a particular disease or condition may be clinically monitored at a future date to demonstrate the absence or reduction in symptoms associated with said disease or condition.

[0006] Compositions and methods described in this application provide the means of altering the metabolome to prevent gut dysfunction. Unhealthy levels of certain metabolites (insufficient or excess metabolites) in the gut can also be described as fecal dysmetabolosis or a dysmetabolic state. The inventors discovered that in developed countries, such as the US, presumed healthy newborn infants are unexpectedly deficient in important gut metabolites and/or their precursors that are important for reducing oxidative stress, for metabolic regulation of food intake (satiety), for brain growth and development including cognition, and as facilitators of detoxification of certain polyphenols like benzoic acid, among other things. These sub-clinical findings may be important predictors of long-term health and chronic deficiencies and may lead to an increased risk for inapprorprite gut development or maturation leading to conditions such as, but not limited to, metabolic disorders, including Type 2 diabetes and/or obesity, and/or Type 1 diabetes, allergy, atopy, asthma.

[0007] In particular, this disclosure provides methods of monitoring the health of mammals. The health of the mammals can be monitored by (a) obtaining a fecal sample from the mammal; (b) determining the amount of metabolites in the sample; and/or (c) identifying a healthy state versus a dysmetabolic state in the mammal based on the abundance or deficiency of the metabolites in the sample. The metabolites can be some of the metabolites listed in column 2 of Table 1. In some examples, the method of monitoring the health of a mammal can include treating a dysmetabolic mammal by administering bacteria, mammalian milk oligosaccharides (MMO) or both. In some examples, a method of monitoring health can include treating a dysmetabolic mammal by administering bacteria, selective oligosaccharides (OS) or both. Selective oligosaccharides are typically DP3 to DP20, selective oligosaccharides are more preferably DP3 to DP10 and may be from any source: chemical synthesis; plant; algae; yeast; bacteria; or mammals. The oligosaccharides administered are typically functional equivalents to Mammalian milk oligosaccharides (MMO). In some examples OS and MMO are structurally equivalent. The bacteria typically comprise bacteria capable of colonization in the mammalian colon. The bacteria and/or the OS can be administered in respective amounts to change the abundance of the one or more metabolites in the feces of the mammal to a non-dysmetabolic level.

[0008] The disclosure also provides methods of maintaining the health of a mammal by administering bacteria and/or OS. Additionally, a fecal sample can be obtained from the mammal, and the level of metabolite(s) in the sample can be determined. A metabolic state in the mammal can be identified based on the concentration and/or level and/or content of the metabolite(s) in the sample, and the bacteria and/or the OS can be administered in response to the identified dysmetabolic state.

[0009] In some examples, the health of a mammal can be maintained by administering bacteria or OS in an amount sufficient to change the level of metabolite(s). The amount, periodicity, and/or duration of the bacteria and/or OS that is administered can be different than the amount of the bacteria and/or OS administered in response to an identified dysmetabolic state. The bacteria can be capable of colonization of the colon.

[0009a] In one aspect, the present disclosure refers to use of a bacterial composition comprising bacteria capable of colonization of the intestine for the preparation of a medicament for increasing the amount of one or more metabolites in the intestine of a mammal, wherein a) the bacterial composition is to be administered; and b) oligosaccharide (OS) is to be administered; wherein the bacteria and the OS are to be administered in respective amounts sufficient to increase the amount of the one or more metabolites in the intestine of said mammal.

[0009b] In one aspect, the present disclosure refers to use of a bacterial composition comprising bacteria capable of colonization of the intestine for the preparation of a medicament for decreasing the amount one or more metabolites in the intestine of a mammal, wherein a) the bacterial composition is to be administered; and b) OS is to be administered; wherein the bacteria and the OS are to be administered in respective amounts sufficient to decrease the amount of one or more metabolites in the intestine of said mammal.

[0010] In some examples, the disclosure provides a method of decreasing and/or maintaining a low level of metabolite(s) in the colon of a mammal by (a) administering a bacteria; and (b) administering OS; where the bacteria and the OS are administered in respective amounts sufficient to maintain a level of one or more metabolites in the feces of said mammal. In some

examples, this method includes one or more metabolites selected from the compounds listed in column 2 of Table 1. In another example, the levels of the bacteria and/or the metabolite can be modulated by altering the level of OS in the diet

[0011] The disclosure also provides methods of establishing or altering an infant, non-infant or specific mammal's gut metabolome by administering particular bacteria and/or OS and monitoring the mammal. The mammal can be monitored by obtaining a fecal or systemic sample and determining the increase or decrease in metabolites, such as but not limited to, serotonin metabolites, tryptophan metabolites, and/or lactate conjugates such as 3-4 hydroxyphenyl lactate, indole lactate and/or phenyllactate. Evaluating the alteration of metabolite relative to a healthy state will allow for the administration of bacteria and/or OS in response to the identified dysmetabolic state.

[0012] In any of the above examples, the metabolite(s) concentrations that are monitored or altered include those such as, but not limited to γ-glutamyl-containing di- or tri-peptides, pipecolic acid, hippurate, serotonin, tryptophan, and/or lactate conjugates such as 3-4 hydroxyphenyl lactate, indole lactate and/or phenyllactate. In some examples, the abundance of γ-glutamyl-cysteine is at least 20-fold greater than the γ -glutamyl-cysteine abundance in the colon of a human infant, which is not colonized by the administered bacteria. In a preferred example, the level of pipecolic acid or salt thereof is at least 10-fold greater than the pipecolic acid level in the colon of a human infant, which is not colonized by the bacteria. In a preferred example, the level of γ-glutamyl-containing di- or tri-peptides is at least 10-fold greater than the level in the colon of a human infant which is not colonized by the bacteria. Using the above examples, oxidative stress of an infant can be reduced. Additionally, bacterial hippurate degraders can be reduced. Jaundice can be prevented or ameliorated.

[0013] In another example, one or more metabolites comprises pipecolic acid or a salt thereof. In another example, the level of pipecolic acid or salt thereof is at least 10-fold greater than the pipecolic acid level in the colon of a human infant which is not colonized by said bacteria.

[0014] Any of the methods described herein can reduce the risk of a mammal developing metabolic disorders such as, but not limited to Juvenile Diabetes (Type I), obesity, asthma, atopy, Celiac's Disease, food allergies, autism, as compared to a dysmetabolic mammal. The risk can be reduced by 20%, 30%, 40%, 50%, 60%, 70%, 80%, or 90%.

[0014a] In one aspect, the present disclosure refers to use of a bacterial composition comprising at least one species capable of consuming MMO by the internalization of that MMO within the bacterial cell for the preparation of a medicament for reducing the risk of Juvenile Diabetes (Type I), wherein a) the bacterial composition is to be administered, and; b) OS is to be administered at a dose sufficient to maintain colonization of the colon of the mammal by bacteria in the bacterial composition of step (a), wherein the risk is reduced as compared to a dysbiotic infant;

wherein the bacterial composition and the OS are to be administered in respective amounts sufficient to increase or decrease the amount of one or more metabolites in the intestine of said mammal;

wherein the one or more metabolites is selected from the compounds listed in column 2 of Table 1, or the one or more metabolites comprises a metabolite selected from the group consisting of γ-glutamyl-containing di- or tri-peptides, γ-glutamyl-cysteine, pipecolic acid, hippurate, 3-hydroxyhippurate, 4-hydroxyhippurate, benzoic acid, cholate, cholate sulfate, chenodeoxycholate, choline, creatine, palmitic acid-9-hydroxy-stearic acid (PAHSA 16:0/OH-18:0), and Oleic Acid-Hydroxy Stearic Acid (OAHSA 18:1/OH-18:0), nonadeconate, arachinate, eiocosenoate, stearate, 15-methylplamitate, 17-methylpalmitate, behenate, margarate, palmitate, myristate, citrate, succinate, serotonin, tryptophan, lactate conjugates, 3-4 hydroxyphenyl lactate, indole lactate, and phenyllactate;

optionally wherein the concentration of γ-glutamyl-containing di- or tri-peptides is at least 10-fold greater than the level in the colon of a mammal which is not colonized by the bacteria;

optionally wherein creatine is greater than the creatine abundance in the colon of a mammal which is not colonized by the administered bacteria;

optionally wherein the concentration of pipecolic acid or salt thereof is at least 10-fold greater than the pipecolic acid level in the colon of a mammal which is not colonized by the bacteria;

optionally wherein hippurate level is determined, said level of hippurate indicating detoxification of the intestine of said mammal; or wherein the amount of bacterial hippurate degraders are reduced in the microbiome, optionally wherein the bacterial hippurate degrader comprises Group B Streptococci, *Camplyobacter jejuni,* or *Listeria monocytogenes*; or wherein the level of hippurate is further compared to level of benzoic acid or other polyphenols; and optionally further comprising adding supplemental glycine to the diet if the mammal is in need of benzoic acid or polyphenol detoxification;

optionally wherein the concentration of cholate, chenodeoxycholate, cholate sulphate is greater than cholate, chenodeoxycholate, cholate sulphate abundance in the colon of a mammal which is not colonized by the administered bacteria;

optionally wherein long chain fatty acid metabolites are increased compared to levels in a mammal which is not colonized by the bacteria, said fatty acid metabolites preferably including palmitic acid-9-hydroxy-stearic acid (PAHSA 16:0/0H-18:0), Oleic Acid-Hydroxy Stearic Acid (OAHSA 18:1/0H-18:0), nonadeconate, arachinate, eiocosenoate, stearate, 15-methylplamitate, 17-methylpalmitate, behenate, margarate, palmitate, myristate, or a combination thereof;

optionally wherein the one or more metabolites comprise a TCA cycle metabolite, said TCA cycle metbolite preferably comprising citrate or succinate; and

optionally wherein tryptophan metabolites are increased compared to levels in a mammal which is not colonized by the bacteria, said tryptophan metabolites preferably including serotonin, tryptophan, lactate conjugates, 3-4 hydroxyphenyl lactate, indole lactate, and/or phenyllactate.

[0015] In any of the above examples, the oligosaccharides can include the carbohydrate polymers found in mammalian milk, which are not metabolized by any combination of digestive enzymes expressed by mammalian genes. The selective oligosaccharides composition can include one or more of lacto-N-biose (LNB), N-acetyl lactosamine, lacto-N-triose, lacto-N-tetraose (LNT), lacto-N-neotetraose (LNnT), fucosyllactose (FL), lacto-N-fucopentaose (LNFP), lactodifucotetraose, (LDFT) sialyllactose (SL), disialyllacto-N-tetraose (DSLNT), 2'-fucosyllactose (2FL), 3'-sialyllactosamine (3SLN), 3'-fucosyllactose (3FL), 3'-sialyl-3-fucosyllactose(3S3FL), 3'-sialyllactose (3SL), 6'-sialyllactosamine (6SLN), 6'-sialyllactose (6SL), difucosyllactose (DFL), lacto-N-fucopentaose I (LNFPI), lacto-N-fucopentaose II (LNFPII), lacto-N-fucopentaose III (LNFPIII), lacto-N-fucopentaose V (LNFPV), sialyllacto-N-tetraose (SLNT), their derivatives, or combinations thereof. The OS can include: (a) a Type II oligosaccharide core where representative species include LnNT; (b) one or more oligosaccharides containing the Type II core and GOS in 1:5 to 5:1; (c) one or more oligosaccharides containing the Type II core and FL 1:5 to 5:1; or (d) one or more oligosaccharides containing the Type II core and SL 1:5 to 5:1; or (e) a combination of (a)- (d). The OS can include: (a) a Type I oligosaccharide core where representative species include LNT; (b) one or more oligosaccharides containing the Type I core and GOS in 1:5 to 5:1; (c) one or more oligosaccharides containing the Type I core and FL 1:5 to 5:1; (d) one or more oligosaccharides containing the Type I core and SL 1:5 to 5:1; or (e) a combination of (a)- (d). In some examples, Type I and Type II oligosaccharides in combination with any of GOS, FL, or SL. Type I or type II may be isomers of each other. Other type II cores include but are not limited to trifucosyllacto-N-hexaose (TFLNH), LnNH, lacto-N-hexaose (LNH), lacto-N-fucopentaose III (LNFPIII), monofucosylated lacto-N-Hexose III (MFLNHIII), Monofucosylmonosialyllacto-N-hexose (MFMSLNH).

[0016] The mammal can receive OS at a dose of over 25%, 40%, or 50% of the mammal's total dietary fiber. Over 10%, 25%, 40%, 50%, 60%, or 75% of the total oligosaccharide can be represented by one or more units of N-acetyllactosamine (Type II core). Additionally, the oligosaccharide composition can include 2'FL and/or GOS.

[0017] The OS can be administered prior to, contemporaneously with, within 2 hours or, or after the administration of the bacteria. The OS and/or bacteria can be administered for at least 1, 3, 10, at least 20, at least 30, at least 60, at least 90, at least 120, at least 150, or at least 180 days.

[0018] In any of the above examples, the bacteria can be capable of colonization in the colon. For example, the bacteria can be from the genus of *Bifidobacteria*, *Lactobacillus*, or *Pediococcus*, such as *B. adolescentis, B. animalis*, *B. animalis* subsp. *animalis, B. animalis* subsp. *lactis, B. bifidum, B. breve, B. catenulatum, B. longum*, *B. longum* subsp. *infantis, B.*

longum subsp. *longum, B. pseudocatanulatum, B. pseudolongum, L. acidophilus, L. antri, L. brevis, L. casei, L. coleohominis, L. crispatus, L. curvatus, L. fermentum, L. gasseri, L. johnsonii, L. mucosae, L. pentosus, L. plantarum, L. reuteri, L. rhamnosus, L. sakei, L. salivarius, P. acidilactici, P. argentinicus, P. claussenii, P. pentosaceus, P. stilesii L. paracasei, L. kisonensis., L. paralimentarius, L. perolens, L. apis, L. ghanensis, L. dextrinicus*, *L. shenzenensis, L. harbinensis, P. parvulus,* or *P. lolii*.

[0019] In any of the above examples, the bacteria can be Bifidobacterium longum subsp. infantis EVC001 as deposited under ATCC Accession No PTA-125180; cells were deposited with the American Type Culture Collection at 10801 University Blvd, Manassas, VA 20110 under the Budapest Treaty on the International Recognition of the Deposit of Microorganisms for the Purposes of Patent Procedure, the "Deposited Bacteria."

[0020] Additionally, "Deposited Bacteria," as used herein, refers to the isolated Bifidobacterium longum subsp. infantis EVC001, deposited with the ATCC and assigned Accession Number, and variants thereof, wherein said variants retain the phenotypic and genotypic characteristics of said bacteria and wherein said bacteria and variants thereof have LNT transport capability and comprise a functional H5 gene cluster comprising BLON2175, BLON2176, and BLON2177.

[0021] A "functional H5 cluster," refers to a cluster of genes in Bifidobacteria responsible for the uptake and metabolism of human milk oligosaccharides. A functional H5 cluster comprises Blon_2175, Blon_2176, and Blon_2177. The H5 cluster comprises the following genes: Blon_2171, Blon_2173, Blon_2174, Blon_2175, Blon_2176, Blon_2177, and galT.

[0022] In any of the above examples, the mammal can be a human, such as an infant, an adolescent, an adult, or a geriatric adult. The mammal can be an infant human that is dysbiotic.

BRIEF DESCRIPTION OF THE FIGURES

[0023] Figure 1. Amount (CFU/g) of B. longum subsp. infantis (B. infantis) in fecal samples as measured by qPCR during the intervention period and a follow-up period in both vaginally- and C-section-delivered human infants. The black line and dots represent all infants who were supplemented with B. infantis for 21 days starting at 7 days of life. All infants receiving the standard of care (no probiotic) are depicted with the grey line and dots. The band around each line represents a 95% confidence interval around the line. The end of supplementation occurred at day 28 and samples were collected until day 60 of life.

CLAIMS

1. Use of a bacterial composition comprising bacteria capable of colonization of the intestine for the preparation of a medicament for increasing the amount of one or more metabolites in the intestine of a mammal, wherein
   a) the bacterial composition is to be administered; and
   b) oligosaccharide (OS) is to be administered;
   wherein the bacteria and the OS are to be administered in respective amounts sufficient to increase the amount of the one or more metabolites in the intestine of said mammal.

2. Use of a bacterial composition comprising bacteria capable of colonization of the intestine for the preparation of a medicament for decreasing the amount one or more metabolites in the intestine of a mammal, wherein
   a) the bacterial composition is to be administered; and
   b) OS is to be administered;
   wherein the bacteria and the OS are to be administered in respective amounts sufficient to decrease the amount of one or more metabolites in the intestine of said mammal.
3. The use according to any one of claims 1 or 2, wherein the one or more metabolites is selected from the compounds listed in column 2 of Table 1.
4. The use according to any one of claims 1 or 2, wherein the one or more metabolites comprises a metabolite selected from the group consisting of γ-glutamyl-containing di- or tri-peptides, γ-glutamyl-cysteine, pipecolic acid, hippurate, 3-hydroxyhippurate, 4-hydroxyhippurate, benzoic acid, cholate, cholate sulfate, chenodeoxycholate, choline, creatine, palmitic acid-9-hydroxy-stearic acid (PAHSA 16:0/OH-18:0), and Oleic Acid-Hydroxy Stearic Acid (OAHSA 18:1/OH-18:0), nonadeconate, arachinate, eiocosenoate, stearate, 15-methylplamitate, 17-methylpalmitate, behenate, margarate, palmitate, myristate, citrate, succinate, serotonin, tryptophan, lactate conjugates, 3-4 hydroxyphenyl lactate, indole lactate, and phenyllactate.
5. The use according to claim 4, wherein the concentration of γ-glutamyl-containing di- or tri-peptides is at least 10-fold greater than the level in the colon of a mammal which is not colonized by the bacteria, or wherein the concentration of γ-glutamyl-cysteine is at least 20-fold greater than the γ-glutamyl-cysteine abundance in the colon of a mammal which is not colonized by the administered bacteria.

6. The use according to any one of claims 4-5 wherein creatine is greater than the creatine abundance in the colon of a mammal which is not colonized by the administered bacteria.
7. The use according to any one of claims 4-6, wherein the concentration of pipecolic acid or salt thereof is at least 10-fold greater than the pipecolic acid level in the colon of a mammal which is not colonized by the bacteria.
8. The use according to any one of claims 4-7, wherein hippurate level is determined, said level of hippurate indicating detoxification of the intestine of said mammal; or wherein the amount of bacterial hippurate degraders are reduced in the microbiome, optionally wherein the bacterial hippurate degraders comprise one or more of Group B Streptococci, *Camplyobacter jejuni,* or *Listeria monocytogenes*; or wherein the level of hippurate is further compared to level of benzoic acid or other polyphenols; and optionally further comprising adding supplemental glycine to the diet if the mammal is in need of benzoic acid or polyphenol detoxification.
9. The use according to any one of claims 4-8, wherein the concentration of cholate, chenodeoxycholate, cholate sulphate is greater than cholate, chenodeoxycholate, cholate sulphate abundance in the colon of a mammal which is not colonized by the administered bacteria.
10. The use according to any one of claims 4-9, wherein long chain fatty acid metabolites are increased compared to levels in a mammal which is not colonized by the bacteria, said fatty acid metabolites preferably including palmitic acid-9-hydroxy-stearic acid (PAHSA 16:0/OH-18:0), Oleic Acid-Hydroxy Stearic Acid (OAHSA 18:1/OH-18:0), nonadeconate, arachinate, eiocosenoate, stearate, 15-methylplamitate, 17-methylpalmitate, behenate, margarate, palmitate, myristate, or a combination thereof.
11. The use according to any one of claims 4-10, wherein the one or more metabolites comprise a TCA cycle metabolite, said TCA cycle metbolite preferably comprising citrate or succinate.
12. The use according to any one of claims 4-11 wherein tryptophan metabolites are increased compared to levels in a mammal which is not colonized by the bacteria, said tryptophan metabolites preferably including serotonin, tryptophan, lactate conjugates, 3-4 hydroxyphenyl lactate, indole lactate, and/or phenyllactate.
13. The use according to any one of claims 1-12 wherein said use reduces the risk of said mammal developing a metabolic disorder, said metabolic disorder being preferably selected

from metabolic disorders consisting of Obesity and Type 2 Diabetes and combinations thereof, said risk being reduced as compared to a dysmetabolic mammal.

14. The use according to any one of claims 1-12, wherein said use reduces the risk of said mammal developing an autoimmune disease, said autoimmune disease preferably selected from the following auto-immune diseases: Juvenile Diabetes (Type I), asthma, atopy, Celiac's Disease, food allergies and combinations thereof, said risk being reduced as compared to a dysmetabolic mammal.
15. The use according to any one of claims 1-12, wherein said use reduces the risk of said mammal developing a cognitive disorder, said cognitive disorder being preferably selected from the following cognitive disorders: autism, depression, or learning disability, and combinations thereof, said risk being reduced as compared to a dysmetabolic mammal.
16. The use according to any one of claims 1-15, wherein the OS comprises mammalian milk oligosaccharide (MMO), and wherein the MMO comprises N-acetyl lactosamine, lacto N-tetrose, lacto-N-biose, N-acetyllactosamine, lacto-N-triose, lacto-N-neotriose, lacto-N-neotetrose, fucosyllactose, lacto-N-fucopentose, lactodifucotetrose, sialyllactose, disialyllactone-N-tetrose, 2'-fucosyllactose, 3'-sialyllactoseamin, 3'-fucosyllactose, 3'-sialyl-3-fucosyllactose, 3'-sialyllactose, 6'-sialyllactosamine, 6'-sialyllactose, difucosyllactose, lacto-N-fucosylpentose I, lacto-N-fucosylpentose II, lacto-N-fucosylpentose III, lacto-N-fucosylpentose V, sialyllacto-N-tetraose, their derivatives, or combinations thereof.
17. The use according to any one of claims 1-15, wherein the OS comprises MMO, and wherein the MMO consists of carbohydrate polymers found in mammalian milk which are not metabolized by any combination of digestive enzymes expressed by mammalian genes, or wherein the OS comprises a Type II oligosaccharide core where representative species include LNnT or LNFPIII and/or a Type I oligosaccharide core where representative species include LNT or LNFPI.
18. The use according to any one of claims 1-17, wherein the bacterial composition comprises bacteria of a genus selected from the group consisting of *Bifidobacteria*, *Lactobacillus*, and *Pediococcus*, which may be of species selected from *B. adolescentis, B. animalis, B. animalis* subsp. *animalis, B. animalis* subsp. *lactis, B. bifidum, B. breve, B. catenulatum, B. longum, B. longum* subsp. *infantis, B. longum* subsp. *longum, B. pseudocatanulatum, B. pseudolongum, L. acidophilus, L. antri, L. brevis, L. casei, L. coleohominis, L. crispatus, L.*

*curvatus, L. equi, L. fermentum, L. gasseri, L. johnsonii, L. mucosae, L. pentosus, L. plantarum, L. reuteri, L. rhamnosus, L. sakei, L. salivarius, P. acidilactici, P. argentinicus, P. claussenii, P. pentosaceus, P. stilesii L. paracasei, L. kisonensis., L. paralimentarius, L. perolens, L. apis, L. ghanensis, L. dextrinicus, L. shenzenensis, L. harbinensis, P. parvulus, and/*or *P. lolii*.

19. Use of a bacterial composition comprising at least one species capable of consuming MMO by the internalization of that MMO within the bacterial cell for the preparation of a medicament for reducing the risk of Juvenile Diabetes (Type I), wherein

a) the bacterial composition is to be administered, and;

b) OS is to be administered at a dose sufficient to maintain colonization of the colon of the mammal by bacteria in the bacterial composition of step (a), wherein the risk is reduced as compared to a dysbiotic infant;

wherein the bacterial composition and the OS are to be administered in respective amounts sufficient to increase or decrease the amount of one or more metabolites in the intestine of said mammal;

wherein the one or more metabolites is selected from the compounds listed in column 2 of Table 1, or the one or more metabolites comprises a metabolite selected from the group consisting of γ-glutamyl-containing di- or tri-peptides, γ-glutamyl-cysteine, pipecolic acid, hippurate, 3-hydroxyhippurate, 4-hydroxyhippurate, benzoic acid, cholate, cholate sulfate, chenodeoxycholate, choline, creatine, palmitic acid-9-hydroxy-stearic acid (PAHSA 16:0/OH-18:0), and Oleic Acid-Hydroxy Stearic Acid (OAHSA 18:1/OH-18:0), nonadeconate, arachinate, eiocosenoate, stearate, 15-methylplamitate, 17-methylpalmitate, behenate, margarate, palmitate, myristate, citrate, succinate, serotonin, tryptophan, lactate conjugates, 3-4 hydroxyphenyl lactate, indole lactate, and phenyllactate;

optionally wherein the concentration of γ-glutamyl-containing di- or tri-peptides is at least 10-fold greater than the level in the colon of a mammal which is not colonized by the bacteria;

optionally wherein creatine is greater than the creatine abundance in the colon of a mammal which is not colonized by the administered bacteria;

optionally wherein the concentration of pipecolic acid or salt thereof is at least 10-fold greater than the pipecolic acid level in the colon of a mammal which is not colonized by the bacteria;

optionally wherein hippurate level is determined, said level of hippurate indicating detoxification of the intestine of said mammal; or wherein the amount of bacterial hippurate degraders are reduced in the microbiome, optionally wherein the bacterial hippurate degrader comprises Group B Streptococci, *Camplyobacter jejuni,* or *Listeria monocytogenes*; or wherein the level of hippurate is further compared to level of benzoic acid or other polyphenols; and optionally further comprising adding supplemental glycine to the diet if the mammal is in need of benzoic acid or polyphenol detoxification;

optionally wherein the concentration of cholate, chenodeoxycholate, cholate sulphate is greater than cholate, chenodeoxycholate, cholate sulphate abundance in the colon of a mammal which is not colonized by the administered bacteria;

optionally wherein long chain fatty acid metabolites are increased compared to levels in a mammal which is not colonized by the bacteria, said fatty acid metabolites preferably including palmitic acid-9-hydroxy-stearic acid (PAHSA 16:0/0H-18:0), Oleic Acid-Hydroxy Stearic Acid (OAHSA 18:1/0H-18:0), nonadeconate, arachinate, eiocosenoate, stearate, 15-methylplamitate, 17-methylpalmitate, behenate, margarate, palmitate, myristate, or a combination thereof;

optionally wherein the one or more metabolites comprise a TCA cycle metabolite, said TCA cycle metbolite preferably comprising citrate or succinate; and

optionally wherein tryptophan metabolites are increased compared to levels in a mammal which is not colonized by the bacteria, said tryptophan metabolites preferably including serotonin, tryptophan, lactate conjugates, 3-4 hydroxyphenyl lactate, indole lactate, and/or phenyllactate.

20. The use according to any of claims 1-18, further comprising:

c) determining the amount and/or concentration, of one or more metabolites in a fecal sample that has been obtained from the mammal;

d) identifying a dysmetabolic state in the mammal based on the amount and/or concentration of the one or more metabolites in the sample;

e) wherein optionally the bacterial composition and/or the OS is to be administered or re-administered in response to the identified dysmetabolic state;

f) optionally adjusting the amount, periodicity, and/or duration of the administration of the bacterial composition and/or the OS in response to an identified dysmetabolic state; and

g) optionally wherein the amount and/or type of the bacterial composition and/or the OS administered in step (a) is different than the amount and/or type of the bacterial composition and/or the OS administered in step (f).