



**NATIONAL INSTITUTES OF HEALTH  
CONSENSUS DEVELOPMENT CONFERENCE STATEMENT**

NIH Consensus Development Conference:  
Lactose Intolerance and Health  
February 22–24, 2010

*National Institutes of Health (NIH) consensus and state-of-the-science statements are prepared by independent panels of health professionals and public representatives on the basis of (1) the results of a systematic evidence review prepared under contract with the Agency for Healthcare Research and Quality (AHRQ), (2) presentations by investigators working in areas relevant to the conference questions during a 2-day public session, (3) questions and statements from conference attendees during open discussion periods that are part of the public session, and (4) closed deliberations by the panel during the remainder of the second day and the morning of the third. This statement is an independent report of the panel and is not a policy statement of NIH or the Federal Government.*

*The statement reflects the panel's assessment of medical knowledge available at the time the statement was written. Thus, it provides a "snapshot in time" of the state of knowledge on the conference topic. When reading the statement, keep in mind that new knowledge is inevitably accumulating through medical research.*

**Introduction**

Lactose intolerance is the syndrome of diarrhea, abdominal pain, flatulence, and/or bloating occurring after lactose ingestion. These symptoms—produced by malabsorption of lactose, a sugar found in milk and other dairy products—often result in avoidance of dairy products by afflicted individuals. Lactose malabsorption occurs because of a decreased ability to digest lactose, due to a deficiency in the levels of the enzyme lactase. Lactase breaks lactose down into two simpler sugars, glucose and galactose, which are readily absorbed into the bloodstream. This enzyme is produced by expression of the lactase-phlorizin hydrolase gene in the cells lining the small intestine.

Infants of every racial and ethnic group worldwide produce lactase and successfully digest lactose provided by human milk or by infant formulas. However, sometime after weaning, in the majority of the world's children, there is a genetically programmed decrease in lactase (lactase nonpersisters). Lactase nonpersistence affects a high but variable proportion of diverse populations in the United States, including Asian Americans, African Americans, Hispanic Americans, Native Americans, Alaska Natives, and Pacific Islanders.

The symptoms of lactose intolerance result from bacterial fermentation of undigested lactose in the colon. Lactose malabsorption can be diagnosed by having individuals ingest a standard dose of lactose after fasting and finding elevated levels of breath hydrogen, which is produced by bacterial fermentation of undigested lactose in the colon. Other diagnostic tools include measuring the lactase activity in an intestinal biopsy sample or genetic testing for the common

polymorphism that is linked to lactase nonpersistence. The demonstration of lactose malabsorption does not necessarily indicate that an individual will be symptomatic. Many variables determine whether a person who malabsorbs lactose develops symptoms, including the dose of lactose ingested, the residual intestinal lactase activity, the ingestion of food along with lactose, the ability of the colonic flora to ferment lactose, and individual sensitivity to the products of lactose fermentation.

Current management often relies on reducing lactose exposure by avoiding milk and milk-containing products or by drinking milk in which the lactose has been prehydrolyzed with lactase enzyme. Alternatively, lactase nonpersisters may tolerate moderate amounts of dairy products ingested with other foods. Many individuals, however, mistakenly ascribe symptoms of a variety of intestinal disorders to lactose intolerance without undergoing testing. This misconception becomes intergenerational when parents with self-diagnosed lactose intolerance place their children on lactose-restricted diets (even in the absence of symptoms) in the mistaken belief that they will develop symptoms if given lactose.

The public health burden from deficiencies attributable to lactose intolerance has not been established. Many adults and children who avoid dairy products—which constitute a readily accessible source of calcium, vitamin D, and other nutrients—are not ingesting adequate amounts of these essential nutrients. For example, most African American adolescents consume inadequate amounts of calcium and vitamin D because they avoid dairy products. Deficient intakes of calcium and vitamin D are risk factors for decreased bone mineral density. This may increase the risk of fracture throughout the life cycle, especially in postmenopausal women. Very low intake of vitamin D can lead to the development of rickets, especially in children of African descent and other highly pigmented individuals. Although reduced-lactose dairy and nondairy alternative products are typically fortified with calcium, vitamin D, and other nutrients, they may be more expensive and less widely available than conventional dairy products. The bioequivalence of these and other calcium supplements is uncertain.

To examine this important topic more closely, the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development and the Office of Medical Applications of Research of the National Institutes of Health convened a Consensus Development Conference to assess the available scientific evidence related to the following questions:

1. What is the prevalence of lactose intolerance, and how does this prevalence differ by race, ethnicity, and age?
2. What are the health outcomes of dairy exclusion diets?
3. What amount of daily lactose intake is tolerable in subjects with diagnosed lactose intolerance?
4. What strategies are effective in managing individuals with diagnosed lactose intolerance?
5. What are the future research needs for understanding and managing lactose intolerance?

At the conference, invited experts presented information relevant to these questions. A systematic evidence review, prepared under contract with the Agency for Healthcare Research and Quality, was summarized; the systematic evidence review (available at <http://www.ahrq.gov/clinic/tp/lactinttp.htm>) emphasizes randomized controlled trials with health outcomes as their endpoints. Conference participants also provided oral and written comments in response to the conference questions, and the panel considered all of this evidence when preparing the consensus statement.

**1. What is the prevalence of lactose intolerance, and how does this prevalence differ by race, ethnicity, and age?**

The prevalence of lactose intolerance is difficult to discern because studies have varied in their interpretation of what constitutes this condition. To estimate accurately the prevalence of lactose intolerance, one first must define lactose intolerance to permit the identification of those individuals with the condition and the exclusion of those without the condition. By applying this definition to a representative population sample, one can then estimate the prevalence in the general population and assess how this prevalence differs by age and race/ethnicity. We define lactose intolerance as the onset of gastrointestinal symptoms following a blinded, single-dose challenge of ingested lactose by an individual with lactose malabsorption, which are not observed when the person ingests an indistinguishable placebo. Although lactose malabsorption and lactase nonpersistence can be easily identified, they are not equivalent to lactose intolerance.

The prevalence of lactose intolerance in the United States cannot be estimated based on available data. None of the potentially relevant studies identified in the systematic review used an adequate definition of lactose intolerance or evaluated a representative sample of the U.S. population. Studies that assessed self-reported lactose intolerance provided limited insight because the self-diagnoses were not confirmed by testing for lactose malabsorption, and the symptoms seen in true lactose intolerance may result from several other conditions such as irritable bowel syndrome. Some studies evaluated only the genetic predisposition to lower than expected levels of lactase in adults (lactase nonpersistence) without assessing lactose malabsorption or intolerance directly.

Although these studies shed some light on the epidemiology of lactose intolerance (discussed below), they cannot be used to estimate the prevalence of lactose intolerance. Many individuals who have the biologic underpinnings for lactose malabsorption (low lactase levels or a genetic profile associated with low lactase) or who have demonstrated lactose malabsorption do not experience the onset of or an increase in the severity of gastrointestinal symptoms following a blinded lactose challenge. Complicating this further, evidence demonstrates that many who self-report lactose intolerance show no evidence of lactose malabsorption. Thus, the cause of their gastrointestinal symptoms is unlikely to be related to lactose.

Despite the limitations in the available studies, there were several noteworthy observations. First, lactose intolerance determined by self-report or nonblinded lactose challenge is less frequent across all ethnic groups than is lactose malabsorption determined by breath hydrogen tests or lactase nonpersistence determined by biopsy or genetic testing. Second, lactose

intolerance, lactose malabsorption, and lactase nonpersistence vary across racial and ethnic groups with the lowest reported occurrence in European Americans and higher although variable occurrence in African Americans, Hispanic Americans, Asian Americans, and Native Americans. The systematic evidence review notes that the racial and ethnic variability in lactose intolerance following nonblinded lactose challenge was not as extreme as that reported in lactose malabsorption and lactase nonpersistence. Third, lactose intolerance with nonblinded lactose challenge and lactose malabsorption was low in young children, but increased with age. In children younger than 6 years, lactose malabsorption was low in all the studies and peaked between ages 10 and 16 years. Little evidence suggests that lactose intolerance increases in older persons. These trends need to be verified by representative population studies using the case definition of lactose intolerance.

## **2. What are the health outcomes of dairy exclusion diets?**

The health outcomes of dairy exclusion diets depend on whether other sources of nutrients, such as calcium and vitamin D, occur in the diet in sufficient quantities to replace dairy products as a source of these nutrients, and to what extent other components of milk are beneficial.

Calcium is necessary for normal growth and bone development as well as subsequent maintenance of bone density. The strongest argument for promotion of dairy ingestion is the beneficial effect of calcium (and fortified vitamin D in milk) on growth and development of the skeleton. Calcium is necessary for adequate bone accretion and optimal peak bone mass, which is a likely determinant of risk for osteoporosis and fragility fractures later in adult life. Evidence would suggest that certain age groups, such as children and teenagers, may be at increased risk for deficient bone acquisition if their diets are deficient in calcium or vitamin D. There is weak evidence that children with diets deficient in calcium have increased fracture rates. The maximal accumulation of bone mineral, and therefore the maximal calcium requirement, occurs during puberty. Although studies indicate that young children who drink milk are likely to meet or exceed the adequate intake for calcium, teenagers, as a group, tend not to take in enough calcium to meet recommended needs. This is exacerbated by dairy avoidance in individuals who consider themselves lactose intolerant, regardless of whether they have undergone objective testing for lactose intolerance.

Studies show that the presence of lactose does not necessarily affect the efficiency of calcium absorption across the intestine, and that lactase nonpersisters do not have significant impairment in calcium absorption. Thus, the limiting factor in achieving optimal peak bone mass in young individuals is the intake of calcium. Similarly, in older individuals, low calcium intake rather than deficient absorption probably is a major factor contributing to loss of bone mass. Replacement of calcium using supplements or dairy products slows the rate of bone loss in older people, possibly as a result of an overall decrease in bone turnover. Across the age spectrum, the factor limiting adequate calcium accrual in many individuals probably is dairy avoidance.

Dairy exclusion diets may exacerbate the risk for osteoporosis for those already at greatest risk. These include women throughout the life cycle and certain racial/ethnic groups. Low intake of dairy products may lead to deficiencies of necessary nutrients for bone health such as vitamin D,

in addition to low calcium intake. Individuals with diseases that result in decreased calcium absorption due to intestinal inflammation (inflammatory bowel disease) or that require the use of corticosteroids (which in themselves directly reduce bone mass) have increased risk of osteoporosis.

Dairy exclusion diets may decrease gastrointestinal symptoms (bloating, cramps, flatus, and diarrhea) in symptomatic individuals who have lactose malabsorption or lactose intolerance. The degree of relief is likely related to the level of expression of lactase and the quantity of lactose ingested. People who remain symptomatic on a dairy exclusion diet may have other causes for their gastrointestinal symptoms, such as irritable bowel syndrome, celiac disease, inflammatory bowel disease, or small bowel bacterial overgrowth.

Dairy exclusion diets may affect other health outcomes. In several studies, individuals taking calcium supplements or increased dairy intake have decreased blood pressure. Calcium supplementation has been suggested to improve cardiac and vascular smooth muscle contractility; however, additional research is needed to clarify whether this has a significant impact on cardiovascular risk. Calcium ingestion has been associated with decreased risk of development of adenomatous colon polyps; it is not known whether this translates into decreased rates of colon cancer. One area of recent interest is the effect of lactose ingestion on colonic bacterial populations, as this may increase production of fatty acids such as butyrate, which may promote mucosal growth and reduce inflammation.

### **3. What amount of daily lactose intake is tolerable in subjects with diagnosed lactose intolerance?**

Among individuals appropriately diagnosed with lactose intolerance, differences in a variety of factors—including lactase activity, gastric emptying rates, fecal bacterial metabolites, colonic mucosal absorptive capacity, and intestinal transit time—can greatly influence susceptibility to develop intolerance symptoms following the ingestion of foods and beverages containing lactose. Individuals differ in the intensity of symptoms of lactose intolerance due to differences in abdominal pain perception and psychological impact of pain and social discomfort. Determining the amounts of lactose that can be tolerated is necessary to develop evidence-based dietary recommendations that meet the needs of the individual.

There was limited high-quality evidence to address the above question. Pertinent studies used different definitions of lactose intolerance, study population selection criteria, lactose administration procedures, and assessment and follow-up methods. The majority of studies used a single dose of lactose without food and evaluated short-term responses. Efforts often were not made to mask the taste difference between lactose-free milk and milk containing lactose. Only a handful of studies tested the participants in a double-blinded fashion with increasing amounts of lactose administered throughout the day to determine the daily tolerable load of lactose. The majority of studies examined small numbers of participants, and either few or no studies focused exclusively on children, pregnant women, or lactating women.

In the majority of available studies, participants were classified as malabsorbers or absorbers based on breath hydrogen measurements or a blood glucose test, and symptoms of lactose intolerance were not always required for study entry. A blinded control was rarely employed to define lactose intolerance at study entry; thus it is probable that some individuals would have reported symptoms following ingestion of lactose-free solutions. The majority of studies investigated individuals with proven lactose malabsorption, not diagnosed lactose intolerance. As a result, only recommendations for individuals with proven lactose malabsorption and perceived lactose intolerance can be made with reasonable assurance.

The available evidence suggests that adults and adolescents who have been diagnosed with lactose malabsorption could ingest at least 12 grams of lactose when administered in a single dose (equivalent to the lactose content found in 1 cup of milk) with no or minor symptoms. Individuals with lactose malabsorption can tolerate larger amounts of lactose if ingested with meals and distributed throughout the day. However, 50 grams of lactose (equivalent to the lactose content found in 1 quart of milk) usually induces symptoms in those adults with lactose malabsorption when administered as a single dose without meals. For women with lactose malabsorption, tolerance to dietary lactose may improve during pregnancy but then worsen after delivery. Some data suggest that the routine ingestion of lactose increases the amount of lactose that is tolerable in both adults and adolescents. There is no scientific evidence to identify the tolerable dose of lactose for children with lactose malabsorption.

We stress the importance of additional scientific investigations to provide evidence-based and culturally sensitive recommendations about the amount of daily lactose intake that can be tolerated by lactose-intolerant individuals, with special emphasis on pediatric and adolescent populations and pregnant and lactating women.

#### **4. What strategies are effective in managing individuals with diagnosed lactose intolerance?**

Available studies about effects of interventions, such as reduced-lactose dairy products, probiotics (a live microbial food component that benefits the recipient through improved intestinal microbial balance), and colonic adaptation, have important limitations that preclude definitive recommendations. There is a need for well-designed, controlled studies on potential therapeutic interventions with well-defined populations, blinding of observers and subjects, adequate control populations, an adequate duration of symptom observation, and sufficient power for outcomes of interest.

Regardless, it is important to distinguish lactose intolerance from other etiologies of gastrointestinal symptoms. Targeting the specific underlying condition likely will optimize outcomes and help avoid unnecessary food group restriction. Whether individuals who have diagnosed themselves as lactose intolerant will accept interventions that ask them to consume a food they believe leads to side effects is unknown. Education regarding lactose intolerance and appropriate evaluation of gastrointestinal symptoms may be the most productive therapeutic approach in these individuals.

Even in persons with lactose intolerance, small amounts of milk, yogurt, hard cheeses, and reduced-lactose foods may be effective management approaches. As noted above, individuals with lactose malabsorption probably can ingest 12 grams of lactose (the equivalent of 1 cup of milk) without significant symptoms, particularly if ingested with other foods. Lactase-treated products may be tolerated better than nontreated products, but more research is needed.

Whether individuals with lactose intolerance have important nutritional deficiencies or long-term clinical sequelae is unknown, but skeletal health is a concern. Although dairy foods are an excellent source of calcium, protein, magnesium, potassium, riboflavin, other nutrients, and, when fortified, vitamin D, these individual nutrients are available in other foods and supplements. Data are lacking on the effects of interventions designed to increase dairy intake versus counseling affected individuals on ways to meet nutrient requirements from other sources. An overall nutritional eating plan should be emphasized, focusing on nutrients potentially reduced by a dairy-free diet while maintaining appropriate caloric intake. An excellent source of overall nutritional guidance as well as nondairy dietary sources of calcium—such as calcium-fortified soy or rice drinks, fruit juices, soy products, dried beans, and leafy greens—can be found at [www.mypyramid.gov](http://www.mypyramid.gov). The following table is an example for how individuals who wish to meet the daily requirements of calcium could do so by using selected dairy products.

**Table 1. Daily Requirements of Calcium by Age and Comparative Serving Equivalents of Common Dairy Sources**

		Low-fat milk	Low-fat plain yogurt	Low-fat hard cheeses (cheddar, provolone, mozzarella, etc.)
		Per cup	Per cup	Per 1.5 oz
Energy (kcal)		102	148	93
Lactose (g)		11–13	11–17*	0.3–1
Calcium (mg)		305	332	301
Calcium/lactose ratio (mg/g)		23–28	20–30	301–1,003
Age (yr)	Calcium Needed (AI; ** mg/d)	Amount Needed To Provide AI for Calcium		
1–3	500	1.6 cups	1.5 cups	2.5 oz
4–8	800	2.5 cups	2.4 cups	4.0 oz
9–18	1,300	4.3 cups	3.9 cups	6.5 oz
19–50	1,000	3.3 cups	3.0 cups	5.0 oz
51+	1,200	3.9 cups	3.5 cups	6.0 oz

\* Despite the high lactose content, low-fat plain yogurt is generally much better tolerated than low-fat milk by individuals with lactose malabsorption.

\*\*The adequate intake (AI) for calcium is based on 1997 Institute of Medicine Dietary Reference Intakes (DRIs).

Note 1: AI for pregnancy and lactation remains the same.

Note 2: Alternative nondairy sources for calcium may be found at [www.mypyramid.gov](http://www.mypyramid.gov).

Some strategies, such as colonic adaptation, where lactose intake is gradually increased over time, do have intriguing preliminary data and may be helpful in some individuals. Although researchers continue to investigate the various treatment strategies, individual treatment approaches can be developed both for lactose-intolerant individuals and for those who avoid dairy foods for other reasons. Individualized strategies could combine inclusion of small amounts of dairy foods and lactase-treated products and could provide suggestions for alternate nutrient sources, emphasizing the approaches and food items that are acceptable to and accessible to each individual. The goals of treatment should be to ensure adequate intake of nutrients important for skeletal health and other clinical outcomes. There are likely stages of the life cycle when meeting these goals is particularly critical for bone accrual and maintenance, such as during adolescence, pregnancy and lactation, and older age.

## **5. What are the future research needs for understanding and managing lactose intolerance?**

Reliable estimates of the U.S. prevalence of lactose intolerance and lactose malabsorption are not available in a representative population of diverse ages and races/ethnicities. Most of the available research assessed subjective symptoms in an unblinded fashion in selected groups of subjects or in individuals unable to fully absorb lactose irrespective of symptoms of lactase nonpersistence. Therefore, we recommend that a study be conducted to determine the prevalence of lactose intolerance in the U.S. population and the differences across age and racial/ethnic groups. The study should examine a representative sample of the U.S. population and determine the following:

- The prevalence of self-reported baseline symptoms
- The prevalence of lactose malabsorption with or without symptoms following a blinded lactose challenge
- The relationship between self-reported symptoms and the presence of lactose malabsorption
- The prevalence of lactose intolerance in those individuals with lactose malabsorption based on the blinded challenge.

The best approach to minimize placebo effects is to conduct blinded challenges using a standardized, taste-masked dose with and without lactose and to define symptoms using a well-validated scoring system. Studies on what constitutes an optimal challenge dose of lactose also should be conducted. Dietary history regarding lactose consumption and symptoms associated with polymorphisms affecting lactase gene expression potentially could obviate the need for taste-masked, blinded oral challenges with lactose and placebo. An opportunity exists to use the infrastructure of the ongoing National Health and Nutrition Examination Survey or other ongoing nationally representative studies, which already are collecting dietary intake data and would allow additional and potentially informative evaluation of the intake of lactose-



containing foods in those with rigorously determined lactose malabsorption with or without symptoms.

Despite the widespread belief that decreased vitamin D and calcium intake associated with restricted intake of dairy products will lead to poor health outcomes, particularly related to bone mineral density and risk for fractures, few data are available on bone health in individuals with lactose intolerance and dairy avoidance. Future studies should investigate the association between dietary calcium intake and outcomes in people with lactose intolerance on low-lactose diets. A diverse population should be evaluated including children, the elderly, males and females, members of ethnic/racial subgroups, and those with susceptible genetic polymorphisms. The latter genetic alterations should include potential modifying genes. Also, the efficacy of dietary calcium intake from nondairy products and from nutritional supplements should be examined in relation to bone health and as to whether other foods influence calcium absorption from these sources.

Although puberty is the period of most rapid accrual of bone mineral, studies are needed to determine whether calcium intake during this period will affect the subsequent risk to develop osteoporosis. Other health outcomes including obesity, diabetes, cardiovascular disease, and cancer also should be assessed in individuals with treated and untreated lactose intolerance and in other individuals avoiding milk products because of perceived lactose intolerance in comparison with the general population. Additional issues of importance need to be addressed in children with lactose intolerance through long-term observational studies and randomized controlled clinical trials of various treatment strategies. These issues include the incidence of infection, allergic disease, and standard measures of growth and development.

Data are lacking as to whether individuals of different races/ethnicities, ages, and genders who have lactose malabsorption have differing tolerance to lactose. Blinded, randomized controlled trials are needed to determine if the quantity of lactose that can be tolerated by lactose-intolerant individuals varies by race, ethnicity, age or gender. Symptoms should be reported in a standardized, validated format so that clinically important differences can be appreciated.

The lack of uniformity in study design and methodology hampers a rational, evidence-based approach to management of lactose intolerance. Defining the tolerable dose of lactose in those with lactose malabsorption is critical to determining the clinical importance of lactose malabsorption and the prevalence of lactose intolerance, and it may provide critical information for management. A stepwise approach should be developed to define the specific amount of dairy foods to introduce to the individual with lactose intolerance (i.e., the greatest amount of lactose that is not associated with symptoms). Studies also should be conducted to confirm whether lactose is better tolerated if distributed throughout the day or given with meals. Some individuals have reported moderate value in reducing symptoms by using lactase or lactose-hydrolyzed milk; however, sample sizes and the reporting of symptoms were so variable in reported studies that making firm recommendations is difficult. The use of prebiotics (a nondigestible food component, usually a carbohydrate, which benefits the recipient by promoting intestinal colonization by beneficial bacteria) and probiotics in dietary supplements and foods including yogurt is a popular intervention for individuals with lactose intolerance, but further studies are needed to document the efficacy of such products in reducing symptoms. Calcium

intake from low-lactose dairy products, nondairy products, and nutritional supplements is an alternative management strategy in individuals with lactose intolerance, but few data are available on the effect of such interventions on individual outcomes, including bone mineral content and fractures.

It will be important to determine whether testing for lactose malabsorption will change the behavior of individuals who avoid dairy products, many of whom may not have lactose intolerance. Future research should employ standardized interventions, blinded controls, and reporting of improvement of symptoms in a consistent, validated fashion to compare the efficacy of these dietary management strategies in obtaining clinically meaningful health outcomes.

Once effective interventions have been identified, behavioral and culturally sensitive approaches to convince people to adopt recommended dietary changes should be developed and tested. Clearly, the perception of symptoms in individuals with lactose intolerance may be highly subjective and very susceptible to a number of psychological and cultural factors. Thus, various strategies may result in very different behavioral changes, and their effectiveness should be compared rigorously.

Additional work needs to be done to improve the management of patients with irritable bowel syndrome and a hypersensitive colon who also may have lactose intolerance.

## **Conclusions**

- Lactose intolerance is a real and important clinical syndrome, but its true prevalence is not known.
- The majority of people with lactose malabsorption do not have clinical lactose intolerance. Many individuals who think they are lactose intolerant are not lactose malabsorbers.
- Many individuals with real or perceived lactose intolerance avoid dairy and ingest inadequate amounts of calcium and vitamin D, which may predispose them to decreased bone accrual, osteoporosis, and other adverse health outcomes. In most cases, individuals do not need to eliminate dairy consumption completely.
- Evidence-based dietary approaches with and without dairy foods and supplementation strategies are needed to ensure appropriate consumption of calcium and other nutrients in lactose-intolerant individuals.
- Educational programs and behavioral approaches for individuals and their healthcare providers should be developed and validated to improve the nutrition and symptoms of individuals with lactose intolerance and dairy avoidance.

## **Consensus Development Panel**

### **Frederick J. Suchy, M.D.**

Panel and Conference Chairperson  
Professor of Pediatrics  
Chief of Pediatric Hepatology  
Jack and Lucy Clark Department of Pediatrics  
Mount Sinai School of Medicine of New  
York University  
Mount Sinai Kravis Children's Hospital  
New York, New York

### **Patsy M. Brannon, Ph.D., R.D.**

Professor  
Division of Nutritional Sciences  
Cornell University  
Ithaca, New York

### **Thomas O. Carpenter, M.D.**

Professor  
Department of Pediatrics  
Director  
Yale Center for X-linked Hypophosphatemia  
Yale School of Medicine  
New Haven, Connecticut

### **Jose R. Fernandez, Ph.D.**

Associate Professor  
Department of Nutrition Sciences  
The University of Alabama at Birmingham  
Birmingham, Alabama

### **Vicente Gilsanz, M.D., Ph.D.**

Director  
Childrens Imaging Research Program  
Childrens Hospital Los Angeles  
Professor of Radiology  
Pediatrics and Orthopaedic Surgery  
Keck School of Medicine  
University of Southern California  
Los Angeles, California

### **Jeffrey B. Gould, M.D., M.P.H.**

Robert L. Hess Professor in  
Pediatrics-Neonatology  
Stanford University School of Medicine  
Stanford, California

### **Karen Hall, M.D., Ph.D.**

Clinical Associate Professor  
Department of Internal Medicine  
University of Michigan Medical School  
Research Scientist  
Geriatric Research, Education, and Clinical  
Center (GRECC)  
Veterans Affairs Ann Arbor Healthcare System  
Ann Arbor, Michigan

### **Siu L. Hui, Ph.D.**

Professor  
Department of Medicine  
Division of Biostatistics  
Senior Biostatistician  
Center for Aging Research  
Indiana University School of Medicine  
Senior Scientist  
Regenstrief Institute  
Indianapolis, Indiana

### **Joanne Lupton, Ph.D.**

Distinguished Professor  
William W. Allen Endowed Chair in  
Human Nutrition  
Department of Nutrition and Food Science  
Texas A&M University  
College Station, Texas

### **Julie Mennella, Ph.D.**

Member  
Monell Chemical Senses Center  
Philadelphia, Pennsylvania

### **Natalie J. Miller**

Graduate Student  
Combined Veterinary Medicine (VMD)  
and Ph.D. Program  
School of Veterinary Medicine  
University of Pennsylvania  
Co-founder  
Cares4Pets  
Philadelphia, Pennsylvania

**Stavroula Kalis Osganian, M.D.,  
Sc.D., M.P.H.**

Assistant Professor  
Harvard University  
Director  
Clinical Research Program  
Children's Hospital Boston  
Boston, Massachusetts

**Deborah E. Sellmeyer, M.D.**

Associate Professor of Medicine  
Director  
Metabolic Bone Center  
Johns Hopkins Bayview Medical Center  
Baltimore, Maryland

**Speakers**

**Lin Chang, M.D.**

Codirector  
Center for Neurobiology of Stress  
Professor of Medicine  
David Geffen School of Medicine  
University of California, Los Angeles  
Los Angeles, California

**Catherine M. Gordon, M.D., M.Sc.**

Director  
Children's Hospital Bone Health Program  
Adolescent/Young Adult Medicine  
and Endocrinology  
Children's Hospital Boston  
Associate Professor of Pediatrics  
Harvard Medical School  
Boston, Massachusetts

**Richard J. Grand, M.D.**

Professor of Pediatrics  
Harvard Medical School  
Program Director  
Clinical and Translational Study Unit  
Director  
Center for Inflammatory Bowel Disease  
Children's Hospital Boston  
Boston, Massachusetts

**Marshall A. Wolf, M.D.**

Professor of Medicine  
Harvard Medical School  
Brigham and Women's Hospital  
Boston, Massachusetts

**Alan E. Guttmacher, M.D.**

Acting Director  
*Eunice Kennedy Shriver* National Institute of  
Child Health and Human Development  
National Institutes of Health  
Bethesda, Maryland

**Robert P. Heaney, M.D., FACP, FACN**

John A. Creighton University Professor  
Osteoporosis Research Center  
Professor of Medicine  
School of Medicine  
Creighton University  
Omaha, Nebraska

**Susan L. Johnson, Ph.D.**

Associate Professor  
Department of Pediatrics  
Section of Nutrition  
University of Colorado Denver  
Anschutz Medical Center  
Denver, Colorado

**Jeanette N. Keith, M.D.**

Associate Professor  
Department of Nutrition Sciences  
Department of Medicine  
The University of Alabama-Birmingham  
Birmingham, Alabama

**Nancy F. Krebs, M.D., M.S.**  
Professor of Pediatrics and Head of Section  
of Nutrition  
Department of Pediatrics  
Health Sciences Center  
University of Colorado at Denver  
Aurora, Colorado

**Michael Levitt, M.D.**  
Professor  
Minneapolis Veterans Affairs Medical Center  
Division of Gastroenterology  
Department of Medicine  
University of Minnesota  
Minneapolis, Minnesota

**Josef Neu, M.D.**  
Professor  
Department of Pediatrics  
University of Florida College of Medicine  
Gainesville, Florida

**David S. Newburg, Ph.D.**  
Associate Professor of Pediatrics  
Harvard Medical School  
Director  
Program in Glycobiology, Pediatric  
Gastroenterology and Nutrition  
Massachusetts General Hospital  
Charlestown, Massachusetts

**Mary Ellen Sanders, Ph.D.**  
Consultant  
Dairy and Food Culture Technologies  
Executive Director  
International Scientific Association for  
Probiotics and Prebiotics  
Centennial, Colorado

**Dennis A. Savaiano, Ph.D.**  
Professor and Dean  
College of Consumer and Family Sciences  
Department of Foods and Nutrition  
Purdue University  
West Lafayette, Indiana

**Aasma Shaukat, M.D., M.P.H.**  
Investigator  
Minneapolis Veterans Affairs Medical Center  
Division of Gastroenterology  
Department of Medicine  
University of Minnesota  
Minneapolis, Minnesota

**Eric Sibley, M.D., Ph.D.**  
Associate Professor  
Division of Pediatrics-Gastroenterology  
Stanford University School of Medicine  
Stanford, California

**Andrew Szilagyi, M.D., FACN, FRCPC**  
Assistant Professor of Medicine  
McGill University School of Medicine  
Department of Medicine  
Division of Gastroenterology  
The Sir Mortimer B. Davis Jewish  
General Hospital  
Montreal, Quebec  
Canada

**Janet E. Taylor, M.D., M.P.H.**  
Psychiatrist  
Private Practice  
New York, New York

**Sarah A. Tishkoff, Ph.D.**  
David and Lyn Silfen University Associate  
Professor  
Departments of Genetics and Biology  
University of Pennsylvania  
Philadelphia, Pennsylvania

**Connie M. Weaver, Ph.D.**  
Distinct Professor and Head  
College of Consumer and Family Sciences  
Department of Foods and Nutrition  
Purdue University  
West Lafayette, Indiana

**Timothy J. Wilt, M.D., M.P.H.**

Codirector  
Minnesota Evidence-based Practice Center  
Core Investigator  
Minneapolis Veterans Affairs Center for  
Chronic Disease Outcomes Research  
Professor of Medicine  
University of Minnesota School of Medicine  
Minneapolis, Minnesota

**Richard J. Wood, Ph.D.**

Associate Professor  
Department of Nutrition  
School of Public Health & Health Sciences  
University of Massachusetts  
Amherst, Massachusetts

**Wilma J. Wooten, M.D., M.P.H.**

President  
San Diego Chapter  
National Medical Association  
San Diego County Health Officer  
San Diego, California

## **Planning Committee**

### **Gilman D. Grave, M.D.**

Chief  
Endocrinology, Nutrition, and Growth Branch  
Acting Director  
Center for Research for Mothers and Children  
*Eunice Kennedy Shriver* National Institute of  
Child Health and Human Development  
National Institutes of Health  
Bethesda, Maryland

### **Lisa Ahramjian, M.S.**

Communications Specialist  
Office of Medical Applications of Research  
Office of the Director  
National Institutes of Health  
Bethesda, Maryland

### **Stephanie Chang, M.D., M.P.H.**

Medical Officer  
Evidence-Based Practice Centers Program  
Center for Outcomes and Evidence  
Agency for Healthcare Research and Quality  
Rockville, Maryland

### **Richard J. Grand, M.D.**

Professor of Pediatrics  
Harvard Medical School  
Program Director  
Clinical and Translational Study Unit  
Director  
Center for Inflammatory Bowel Disease  
Children's Hospital Boston  
Boston, Massachusetts

### **Judy Hannah, Ph.D.**

Health Science Administrator  
Geriatrics and Clinical Gerontology Program  
National Institute on Aging  
National Institutes of Health  
Bethesda, Maryland

### **Van S. Hubbard, M.D., Ph.D.**

Rear Admiral, U.S. Public Health Service  
Director  
Division of Nutrition Research Coordination  
National Institute of Diabetes and Digestive  
and Kidney Diseases  
National Institutes of Health  
Bethesda, Maryland

### **Wendy L. Johnson-Askew, Ph.D., M.P.H.**

Public Health Nutrition and Health  
Policy Advisor  
Division of Nutrition and Research Coordination  
National Institutes of Health  
Bethesda, Maryland

### **Barnett S. Kramer, M.D., M.P.H.**

Associate Director for Disease Prevention  
Director  
Office of Medical Applications of Research  
Office of the Director  
National Institutes of Health  
Bethesda, Maryland

### **Kelli K. Marciel, M.A.**

Communications Director  
Office of Medical Applications of Research  
Office of the Director  
National Institutes of Health  
Bethesda, Maryland

### **Lata S. Nerurkar, Ph.D.**

Senior Advisor for the Consensus  
Development Program  
Office of Medical Applications of Research  
Office of the Director  
National Institutes of Health  
Bethesda, Maryland

Planning Committee members provided their input at a meeting held October 7–9, 2008.  
The information provided here was accurate at the time of that meeting.

**Josef Neu, M.D.**

Professor of Pediatrics  
Director of Neonatology Fellowship  
Training Program  
Department of Pediatrics  
Division of Neonatology  
University of Florida College of Medicine  
Gainesville, Florida

**Mary Frances Picciano, Ph.D.**

Senior Nutrition Research Scientist  
Office of Dietary Supplements  
National Institutes of Health  
Bethesda, Maryland

**Winston Price, M.D., FAAP**

Past President  
National Medical Association  
Executive Clinical Dean  
American University of Antigua College  
of Medicine  
Alpharetta, Georgia

**Susan C. Rossi, Ph.D., M.P.H.**

Deputy Director  
Office of Medical Applications of Research  
Office of the Director  
National Institutes of Health  
Bethesda, Maryland

**Mona Jaffe Rowe, M.C.P.**

Associate Director  
Office of Science Policy, Analysis,  
and Communications  
*Eunice Kennedy Shriver* National Institute  
of Child Health and Human Development  
National Institutes of Health  
Bethesda, Maryland

**Dennis A. Savaiano, Ph.D.**

Professor and Dean  
Department of Foods and Nutrition  
College of Consumer and Family Sciences  
Purdue University  
West Lafayette, Indiana

**Eric Sibley, M.D., Ph.D.**

Associate Professor  
Pediatrics–Gastroenterology  
Stanford School of Medicine  
Stanford, California

**Frederick J. Suchy, M.D.**

Panel and Conference Chairperson  
Herbert H. Lehman Professor and Chairperson  
The Jack and Lucy Clark Department  
of Pediatrics  
Mount Sinai School of Medicine  
New York University  
Pediatrician-in-Chief  
Mount Sinai Hospital  
New York, New York

**Connie M. Weaver, Ph.D.**

Distinct Professor and Head  
Department of Foods and Nutrition  
College of Consumer and Family Sciences  
Purdue University  
West Lafayette, Indiana

**Karen K. Winer, M.D.**

Medical Officer  
Endocrinology, Nutrition, and Growth Branch  
Center for Research for Mothers and Children  
*Eunice Kennedy Shriver* National Institute  
of Child Health and Human Development  
National Institutes of Health  
Bethesda, Maryland

Planning Committee members provided their input at a meeting held October 7–9, 2008.  
The information provided here was accurate at the time of that meeting.



## **Conference Sponsors**

### ***Eunice Kennedy Shriver* National Institute of Child Health and Human Development**

Alan E. Guttmacher, M.D.  
Acting Director

### **Office of Medical Applications of Research**

Jennifer M. Croswell, M.D., M.P.H.  
Acting Director

## **Conference Cosponsors**

### **National Institute of Diabetes and Digestive and Kidney Diseases**

Griffin P. Rodgers, M.D., M.A.C.P.  
Director

### **National Institute on Aging**

Richard J. Hodes, M.D.  
Director

### **Division of Nutrition Research Coordination, NIH**

Van S. Hubbard, M.D., Ph.D.  
Director

### **Office of Dietary Supplements**

Paul M. Coates, Ph.D.  
Director

## **Conference Partners**

### **U.S. Department of Agriculture**

Thomas Vilsack  
Secretary of Agriculture