



116th

ABBOTT NUTRITION **RESEARCH CONFERENCE**

**THE MATERNAL MICROBIOME
& PERINATAL COLONIZATION**



CONFERENCE HIGHLIGHTS

- Dysbiosis of the gut microbiota is associated with allergies, asthma, autism, colon cancer, Crohn's disease, COPD, diabetes, obesity, multiple sclerosis, rheumatoid arthritis, and ulcerative colitis. (p 5)
- During development, a fetus can be "misinformed" by maternal pharmaceutical exposure, malnutrition, inflammatory diseases, and dysglycemia. (p 6)
- The gut microbiome may regulate metabolic and cardiovascular health in pregnancy. (p 7)
- Prenatal exposure to stress leads to alterations in the gut microbiome in a preclinical model. (p 7)
- Although milk produced by healthy moms was once thought to be sterile, emerging research suggests that it contains a diverse and live microbial community. (p 8)
- Bifidobacteria support infant health by: inhibiting pathogens, regulating metabolic function, and promoting SCFA production. (p 9)
- The microbiome of full-term vaginally born, exclusively human milk-fed infants with no previous exposure to antibiotics can be considered the "gold standard." (p 9)
- Up to one-third of preterm births may follow microbial invasion of the amniotic cavity. (p 10)
- Human milk partially corrects gut dysbiosis and decreases the risk of NEC in preterm infants. (p 11)



An attentive audience learned the latest research on the maternal and infant microbiomes at the 116th Abbott Nutrition Research Conference.

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New research continues to stress the importance of gut bacteria across the lifespan. The first 1,000 days of life—conception to two years—is a critical time in human development, in part because of the lasting effects of these tens of trillions of symbiotic and sometimes pathogenic microorganisms. Recognizing the emerging science and vital implications for maternal and infant health, Abbott Nutrition Health Institute (ANHI) convened an expert interdisciplinary group of scientists from nutrition, molecular genetics, neonatology, and neurology to present their scientific contributions on April 19 and 20, 2017, at Ross Park, headquarters of Abbott Nutrition Research & Development in Columbus, Ohio, USA.

Topics included the maternal microbiome's possible influences on an infant's immune response, metabolism, neurodevelopment, birth weight, and gestation time. Researchers also discussed the biochemistry of human milk, benefits of human milk in infant development, and the role of the microbiome in nutritional status. Although many studies presented associations and not causation, agreement among the scientific community exists that additional maternal and pediatric microbiome research is necessary to improve human health, with implications for lowering asthma and allergies, reducing obesity and diabetes, and preventing preterm births and infections.

Abbott Nutrition Research & Development's **Sean M. Garvey, PhD**, Manager Area Scientific Affairs, **Larry W. Williams, MD**, Global Medical Affairs, Senior Medical Director, and **Snigdha Mishra, PhD**, Divisional Vice President, Scientific & Medical Affairs, welcomed distinguished researchers and attendees to the 116th Abbott Nutrition Research Conference on The Maternal Microbiome and Perinatal Colonization. The goal of the *Highlights* newsletter is to provide an overview of the presentations, key points, and research opportunities. The health implications of the human gut microbiome are seemingly boundless, and the journey of intervention discovery is accelerating with anticipation of practical application. Whether a dietitian, nutrition educator, researcher, physician, nurse, or clinician, *Highlights* presents the most recent global maternal and infant gut microbiome research that may benefit clinical practice to improve health, prevent chronic diseases, and reduce the impact of acute illnesses.

A BRIEF HISTORY OF MICROBIOLOGY



Speakers and attendees discuss their research during breaks.

More than 125 years ago, Robert Koch and Louis Pasteur identified germs as the cause of infectious diseases and since then, humans have been at war with microbes. The discovery and application of antibiotics and vaccines have saved millions of lives; however, has the hygiene battle to eradicate microbes throughout the past century caused unintended consequences in human health that we are just now unearthing?

WHAT IS THE GUT MICROBIOME?

The human gastrointestinal (GI) tract is divided into sections, allowing for the digestion and absorption of nutrients, but also the compartmentalization of commensal microorganisms. The gut is home to trillions of microbes, collectively called the microbiota or, when referring to methods that capture the microbiota's mixed genome, microbiome. Gut microbes are at the metabolic mercy of their human host for sustenance and fuel. Any foods that remain undigested and unabsorbed after passing through the upper GI tract may be metabolized by microbes and absorbed in the colon. Two-thirds of the gut microbiota is unique to that individual.¹ Factors contributing to heterogeneity among individuals include: food intake, genetics, age, medications, and other environmental factors. Researchers continue to unravel how the pre- and perinatal gut microbiomes influence infant health throughout the lifespan. Research also has identified associations with many of the chronic conditions that are increasing throughout the world. Dysbiosis, microbial imbalance or maladaptation on or inside the body, has been associated with allergies and asthma, autism, colon cancer, Crohn's disease, chronic obstructive pulmonary disease (COPD), diabetes, obesity, multiple sclerosis, rheumatoid arthritis, and ulcerative colitis.²

THE GUT MICROBIOME'S INFLUENCE ON ASTHMA

Higher asthma rates globally are disproportionately associated with higher income countries. Today, the United Kingdom has the highest asthma rate at 18.4%, with the United States (10.9%) and Canada (14.1%) closely behind, yet the rate of Mexico is 3.3%.³

B. Brett Finlay, PhD, described a study from his laboratory that used a murine asthma model to show that the gut microbiome has a profound influence on asthma.⁴ Further research indicated that an early life period was critical for later asthma susceptibility.⁵ This result seemed to be mediated by altered immune development, including T helper 17 cells and regulatory T cells. The research identified a *critical window* early in life where dysbiosis is most influential in this experimental model of asthma.

More recently, Dr Finlay and his coauthors compared the gut microbiome of 319 subjects enrolled in the Canadian Healthy Infant Longitudinal Development (CHILD) Study and showed that infants at risk for asthma exhibited transient gut microbial dysbiosis during the first 100 days of life.⁶ The relative abundances of the bacterial genera *Lachnospira*, *Veillonella*, *Faecalibacterium*, and *Rothia* were significantly decreased in children at risk for asthma.



Dr Brett Finlay discussed how asthma is associated with a particular gut microbiome signature at 3 months of age.

GUT MICROBIOME AND CHRONIC MALNUTRITION

Environmental enteropathy (EE) is an understudied chronic inflammatory disease of the small intestine and is a contributor to global childhood malnutrition. In addition to inflammation, small intestines from patients with EE also show villous blunting and increased intestinal permeability, thus interfering with nutrient uptake and optimal growth and development. Until recently, no preclinical models were available to enable a clearer understanding of the pathophysiology of EE and to test potential therapies.⁷ In 2015, Dr Finlay and his collaborators established the first animal model of EE by dual administration of a moderately malnourished diet low in protein and fat and a commensal bacterial composition of Bacteroidales species and *Escherichia coli* to mice. The result was increased intestinal inflammation, villous blunting, and 35% reduction in body weight, compared to malnourished diet alone. Fascinatingly, neither the Bacteroidales species alone nor the *Escherichia coli* alone, in combination with the malnourished diet, could replicate the growth stunting observed with the combination of genera. Beyond the intestinal pathology common to human EE, this mouse model also showed remarkable changes in the intestinal gut microbiome and intestinal metabolome, along with increased susceptibility to enteric infection. These findings provide evidence indicating that both the microbiome and diet interact to contribute to the etiology of EE.

THE INFLUENCES OF THE GUT MICROBIOME DURING PREGNANCY

BARKER HYPOTHESIS:

Adverse environmental stimuli that occur during prenatal life induce developmental adaptations that later result in the increased risk of what we otherwise assumed were chronic diseases.

LOW BIRTH WEIGHT IS ASSOCIATED WITH THE RISK OF:

- Glucose intolerance
- Insulin resistance
- Hypertension
- Obesity
- Early puberty
- Anxiety/increased stress responses
- Osteoporosis
- Cancer
- Allergy and asthma

Developmental programming describes the influence of adaptive prenatal interactions—between fetus and mom—on postnatal physiology. Adverse events occurring during these critical developmental windows shape and mold primordial cells and systems, which may increase risk of developing disease. Adversity in this context may include poor maternal or infant nutrition (under- and overnutrition or micronutrient deficiencies), stress, or exposure to maternal disease. This framework of prenatal disease risk programming, termed the *Developmental Origins of Health and Disease*, was founded from early epidemiological studies conducted by David Barker, PhD, who demonstrated an association between birth weight and mortality due to ischemic heart disease in adulthood.⁸ This initial observation led to the formation of the Barker hypothesis (Fig 1).⁹

“The womb may be more important than the home,” **Deborah Sloboda, PhD**, quoted during her presentation. She explained how the fetus can be “misinformed” because of pharmaceutical exposures and drugs, nutrition, inflammatory diseases, and maternal metabolism.

Recently, the emerging role of the gut microbiota in obesity¹⁰ has become a target of intense investigation. Obesity is associated with a shift in the microbiota, characterized by decreased abundance of Bacteroidetes (phylum that includes *Bacteroides*) and increased abundance of Firmicutes (phylum that includes *Lactobacillus*).¹¹⁻¹² The concept of an “obese gut microbiota” has begun to emerge, in which interventions including weight loss via caloric restriction or bariatric surgery have shown that a loss in adiposity appears to shift gut microbiota to a more favorable increase in the abundance of Bacteroidetes.¹³

Evidence continues to emerge on the relative impact of the prenatal environment on microbiota composition throughout life. Although not supported empirically, whether the relationship between commensal bacterial transmission and obesity risk is direct or indirect, emerging support regarding modulation of maternal systems, including nutrient uptake and utilization and/or inflammatory-mediated changes in gut function, have demonstrated plausible mechanisms by which obesity may be transferred between a mother and her children.^{14,15}

METABOLISM AND BLOOD PRESSURE IN PREGNANCY

During pregnancy, females experience metabolic and cardiovascular changes. The placenta releases many hormones and growth factors, which not only affect the fetus but also the mother. Placental release of human placental lactogen and growth hormone induces insulin resistance in the mother, which ensures adequate glucose supply to the developing fetus. Women with higher pre-pregnancy levels of insulin resistance, including women with high pre-pregnancy body mass index (BMI) and those who are genetically predisposed to insulin resistance, are at higher risk of developing gestational diabetes. Gestational diabetes is associated with adverse outcomes for mother and infant immediately, as well as in the future.¹⁶

The composition of the mother’s gut microbiome changes during gestation. During the third trimester, the microbiome has a higher proportion of bacteria belonging to the phylum Proteobacteria, which is associated with a pro-inflammatory state. Research indicated that the gut microbiome may contribute to the immune and metabolic changes observed in pregnancy.¹⁷

Fig 1. The Barker hypothesis.⁹



Dr Sloboda discussed evidence showing that a mother’s gut microbiota “educates” the fetal immune system during pregnancy.

The gut microbiota synthesize vitamins, such as vitamin K, and ferment indigestible carbohydrates into short-chain fatty acids (SCFA).¹⁸ In regards to cholesterol, probiotics may lower cholesterol uptake through: assimilation of cholesterol by growing cells, binding of cholesterol to cellular surface, incorporation of cholesterol into the cellular membrane, deconjugation of bile via bile salt hydrolase, coprecipitation of cholesterol with deconjugated bile, binding action of bile by fiber, and production of SCFA from oligosaccharides.¹⁹ The gut microbiota also regulate the immune system through altering the release of cytokines from the cells lining the intestinal wall. Lastly, the gut microbiota can alter the permeability of the intestinal wall²⁰—affecting leakage of bacterial products across the intestinal wall—leading to possible inflammation. Figure 2 illustrates the system-wide interactions among the immune system, nutrition, microbiome, metabolism, and genetic predisposition.²¹

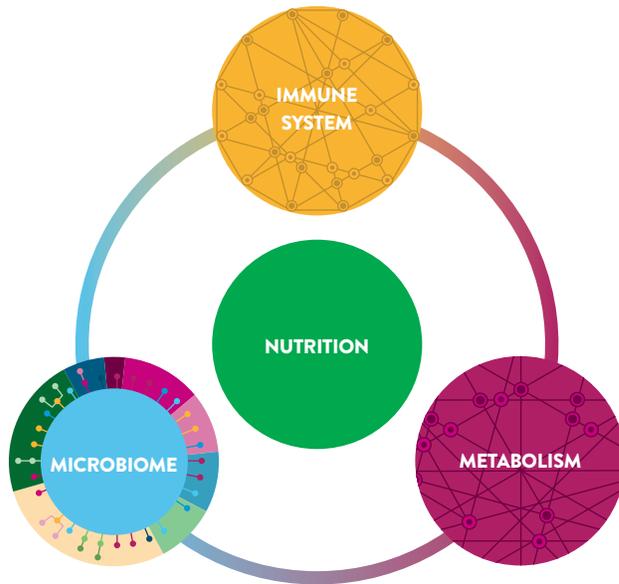


Fig 2. Nutrition, microbiome, metabolism, genetic predisposition, and the immune system interact to influence health and disease.

Marloes Dekker Nitert, PhD, concluded that the microbiota is a regulator of metabolic and cardiovascular health in pregnancy and manipulation of the composition of the gut microbiota could therefore be a new target for the prevention of pregnancy complications. Candidate strategies for altering gut microbiota composition include altering dietary fiber intake as well as supplementing with pre- and probiotics, with the goal of increasing the abundance of microbes that produce SCFA in the gut.

PRENATAL STRESS

Although genetics plays a role in the transmission of mental illnesses, maternal stress and mental illness during pregnancy also influence the developing fetal brain with long term cognitive and behavioral implications, such as an increased risk of altered stress response, anxiety disorders, and depression in adulthood.^{22,23} Intrauterine growth factors, hormones, and mom’s immune system all contribute to healthy fetal development, along with communication between developing gut and brain, also called the gut-brain axis. Postnatally, microbes are an essential part of the gut-brain axis and evidence exists that the gut microbiome affects the gut-brain axis and influences brain development, function, and behavior.

Tamar Gur, MD, PhD, presented her research regarding the effects of prenatal stress on the maternal microbiome, inflammation, and changes in growth factors *in utero* and into adulthood.²² In a mouse model, findings suggested that prenatal exposure to stress leads to alterations in the microbiome, cytokines, and brain derived neurotrophic factor—important in synapse formation and neurodevelopment—in *in utero*. These alterations continue into adulthood and are accompanied by increased anxiety-like behavior and changes in cognition in female offspring.²⁴



Dr Dekker Nitert described how butyrate exerts multiple physiological effects and appears to play a key role in controlling blood pressure and inflammation during pregnancy.



Dr Gur described the effects of prenatal stress on offspring behavior in a mouse model.

THE HUMAN MILK MICROBIOME

Although milk produced by healthy women was once thought to be sterile, emerging research suggests that it contains a diverse microbial community.²⁵⁻²⁸ This microbiome, consisting predominantly of lactobacilli, streptococci, and enterococci,²⁹ undergoes major shifts during the first month postpartum,³⁰ but otherwise remains relatively stable over time.²⁷ In addition, the composition of the milk microbiome is influenced by geographical location and by mode of delivery. For instance, milk produced by Spanish mothers contained a greater abundance of Bacteroidetes, while milk from Chinese mothers was more abundant in Actinobacteria.³¹ There are limited data and mixed findings with regard to the effect of Cesarean section (C-section) on the milk microbiome, but some studies indicate that vaginal delivery was associated with higher microbial diversity and richness, as compared to C-section delivery.^{28,30-32} Interestingly, milk samples from mothers who underwent elective C-sections expressed a different microbiome from those who delivered vaginally, but this difference disappeared if the C-sections were non-elective.^{28,29,33}

“Human milk is the only food designed by Mother Nature intended solely for the nourishment of humans.”

– MICHELLE (SHELLEY) MCGUIRE, PhD

MATERNAL DIET AND THE HUMAN MILK MICROBIOME

Michelle (Shelley) McGuire, PhD, explained that although optimal maternal nutrition is imperative for offspring development, much more research is needed to define the relationships between maternal diet, milk nutrient content, and the milk microbial community structure. New research from her laboratory³⁴ showed that maternal energy, lipid (especially the fatty acids docosahexaenoic acid [DHA] and eicosapentaenoic acid [EPA]), carbohydrate, and fiber intake were positively correlated to the relative abundance of Firmicutes. Intake of various micronutrients were negatively correlated to the relative abundance of Firmicutes. The study also uncovered additional links between maternal diet and milk microbiome, including numerous relationships between the intake of various amino acids and the relative abundance of other phyla.³⁴

An additional example of maternal diet affecting the milk microbiome involved maternal intake of lactic acid bacteria from fermented foods and the subsequent transfer of these same species to infants through breastfeeding.³⁵ Strikingly, the lactic acid bacteria: *Lactobacillus plantarum*, *L. fermentum*, *Pediococcus pentosaceus*, and *L. brevis* have been found in food, maternal feces, human milk, and infant feces.³⁵ In fact, a member of one of these lactic acid bacteria, a specific strain of *L. fermentum*, has been clinically shown to treat mastitis.³⁶



Dr McGuire discussed how characterizing human milk composition is critical to understanding optimal nutrition during infancy.

HUMAN MILK INTERACTIONS AND THE DEVELOPING INFANT MICROBIOME

Human milk is composed of hundreds and thousands of distinct bioactive molecules. These bioactive molecules make their way to the lower GI tract of infants and impact their gut microbiome (Fig 3). Oftentimes, infants fed human milk have a predictable microbial community assembly, explained **David Sela, PhD**. An important consequence of breastfeeding is higher numbers of *Bifidobacterium* in infants' feces.³⁷

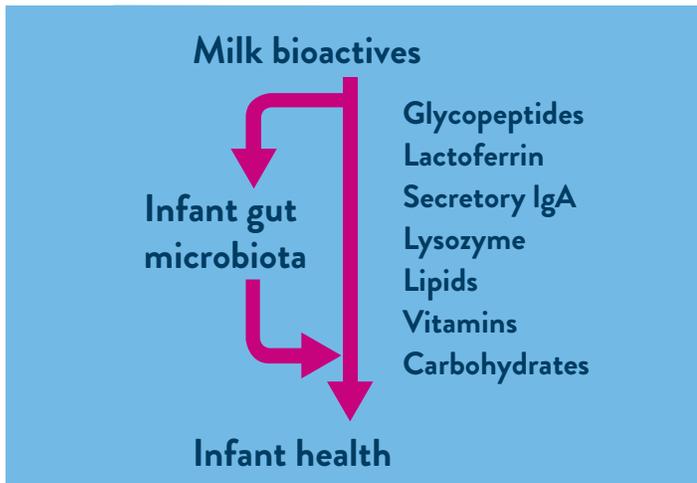


Fig 3. Impact of milk bioactives.

Bifidobacteria are gram-positive microbes that are regarded as beneficial for the host.³⁸ Bifidobacteria dominate the intestinal microbiome of human milk-fed infants during the first years of life. In contrast, the microbiome of formula-fed infants is quite different in composition and is not dominated by bifidobacteria, emphasizing how human milk bioactives may shape the infant gut microbiome (Fig 3). Bifidobacteria benefit the developing infant through the inhibition of pathogenic bacterial colonization, optimization of metabolic function, and production of SCFA.

Human milk oligosaccharides (HMOs), the third most abundant solid component in human milk, are bioactive sugars that are minimally digested by infants. HMOs range in length and complexity, from short chain trisaccharides such as 2'-fucosyllactose, to long chain sugars with tens of saccharide units.



Dr Sela answers questions about HMOs in human milk.

Approximately 200 different types of HMOs have been detected among samples of human milk.³⁹ Although infants themselves minimally digest HMOs, these unique prebiotic molecules support the growth of specific bifidobacteria and especially *Bifidobacterium longum* subsp. *infantis*, a type of *Bifidobacterium* that is found exclusively in the infant GI tract.⁴⁰ Not surprisingly, bifidobacteria harbor a suite of genes—not found in the human genome—dedicated to the expression of proteins that facilitate the uptake and metabolism of HMOs. Commensalism is thus well portrayed by the complementing gut microbiome and human genome. Dr Sela's current research looks at how bioactive compounds, such as HMOs, interact with the gut microbiome.

GUT MICROBIOME IN DEVELOPING NEONATES

Is there a placental microbiome? According to **Catherine Stanton, DSc, PhD**, there is. The placental microbiome is similar to that of the mother's oral microbiome⁴¹ and not of the human gut. And the placental microbiome is not as metabolically rich. However, a newborn infant's bacterial population resembles that of the mother's vaginal microbiome if born vaginally, but if born by C-section, the bacteria resembles the maternal skin.⁴² Additional variables reported to influence the development of an infant's gut microbiome include: gestational age, host genetics, feeding regimen, and perinatal antibiotic usage.⁴³ Thus, the microbiome of a full-term vaginally born, exclusively human milk-fed infant, with no previous exposure to antibiotics, can be considered the "gold standard" of microbiome in early life. An early life gut microbiome usually stabilizes by 2 or 3 years of age, at which time it resembles that of an adult.

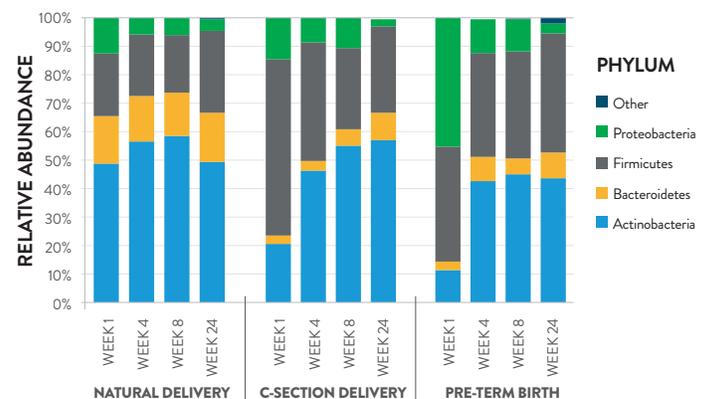


Fig 4. Infant gut microbiome composition to week 24 (INFANTMET Study).⁴⁴

A recent cohort study (INFANTMET) of both term and preterm human milk-fed infants who were born through spontaneous vaginal delivery or via C-section confirmed that mode of delivery and gestational age at birth have significant effects on early neonatal microbiome development.⁴⁴ However, by 24 weeks of age, all infants regardless of birth mode had similar though distinct gut microbiome (Fig 4). Bacteria belonging to the Actinobacteria

phylum that includes members of the *Bifidobacterium* genus were found to be a major component of the infant gut throughout this period and represented up to 50% of entire fecal microbiome. Interestingly, human-milk feeding for four months or more was found to impact the gut microbiome of infants born via C-section, but not of those delivered vaginally. A set of dichorionic triplets (monozygotic twins and a fraternal sibling) demonstrated their microbiomes were similar to each other compared to other infants, supporting the influence of host genetics and environment on early microbiome composition.⁴⁵

INFECTION AND INFLAMMATION:

POSSIBLE INFLUENCES IN PRETERM LABOR, NECROTIZING ENTEROCOLITIS, AND NEONATAL SEPSIS



Dr Romero explained that one-third of preterm births are because of microbial invasion of the amniotic cavity.

Preterm birth is a major health problem across the globe. In 2005, approximately 13 million preterm births worldwide occurred, which is 9.6% of all births. Of those, 11 million (85%) were in Africa and Asia. The highest rates occurred in Africa (11.9%) and North America (10.6%).⁴⁶ Although preterm labor is one syndrome, **Roberto Romero, MD, DMedSci**, explained the many causes.⁴⁷ Conceptually, a single test will not predict, nor will a single intervention prevent, preterm births.

The role of the maternal microbiome is of interest to researchers. Recently a study⁴⁸ found 15% of women in spontaneous preterm labor with intact membranes tested positive for microbes in amniotic fluid, thus suggesting a causal relationship. Yet, one in three preterm deliveries comes from a mother with microbial invasion of the amniotic cavity.⁴⁹ If infection is a predominant cause, can preterm labor be reduced with antibiotic intervention? Would the unintended consequences of maternal antibiotic interventions be worth the possible maternal microbiome dysbiosis, which then may affect the developing neonate and/or human milk?

NECROTIZING ENTEROCOLITIS AND NEONATAL SEPSIS

The skin, GI tract, and the innate and adaptive immune systems of preterm infants are immature and function only marginally, often resulting in prolonged hospital stays and exposure to a variety of medications, medical surfaces, and instruments. As a result, preterm infants develop intestinal dysbiosis, which is associated with the risk of necrotizing enterocolitis (NEC) and neonatal sepsis.

NEC is the leading cause of death in very premature infants from 2 to 8 weeks of age. Yet, NEC is less common when human milk-fed, and is rare in full-term infants. In the United States alone, healthcare costs of NEC are estimated at up to \$1 billion annually.⁵⁰ NEC is an intestinal disease that predominantly affects premature infants; symptoms include a sudden onset of abdominal distention, bloody stools, abnormal abdominal radiographs, and sometimes intestinal perforation, leading to long term morbidity (Fig 5).⁵⁰⁻⁵²

LONG-TERM MORBIDITY OF NEC

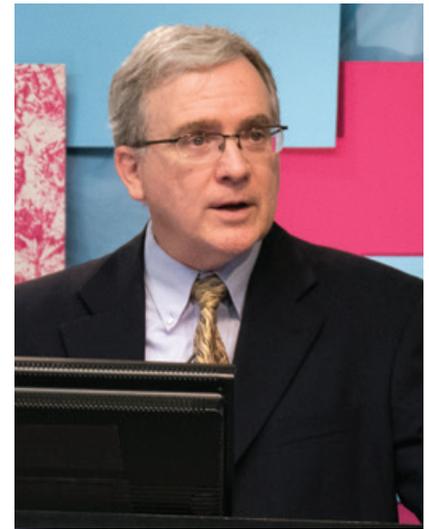
- Strictures
- Short gut
 - Malabsorption
 - Poor growth
 - Prolonged total parenteral nutrition
- Neurodevelopmental delays
 - Decreased brainstem audio-evoked responses
 - Decreased psychomotor development index
- Increased cerebral palsy and microcephaly

Fig 5. Long-term morbidity of NEC.⁵⁰⁻⁵²

Mark Underwood, MD, MAS, provided the following evidence linking premature neonatal dysbiosis to NEC.

- Animal models of NEC demonstrate dysbiosis and the central role of bacteria recognition by the infant, triggering a poorly modulated inflammatory response.⁵³
- Medications commonly administered to premature infants such as antibiotics and acid-blockers cause intestinal dysbiosis and increase the risk of NEC.⁵⁴⁻⁵⁶
- Human milk partially corrects intestinal dysbiosis and decreases the risk of NEC.⁵⁷
- Probiotics partially correct dysbiosis and decrease the risk of NEC in human trials and animal models.⁵⁸
- Human milk components, including lactoferrin, epidermal growth factor, and HMOs, decrease the risk of NEC in human studies and/or the incidence of NEC in animal studies.⁵⁹

Neonatal sepsis occurs when pathogenic microbes invade the tissue or blood causing symptoms of infection. The bacteria identified in late onset sepsis ([LOS] greater than 48-72 hours of age) often originate in the gut. Funisitis—inflammation of the connective tissue of the umbilical cord—predisposes preterm infants to neonatal gut dysbiosis, increasing the risk for LOS. In very low birth weight infants (VLBW), for every increase of human milk intake by 10 mL/kg/d, the probability of sepsis decreased by 19%, and neonatal intensive care unit costs were lowest for VLBW infants who received the highest amount of human milk during the first 28 days of life.⁶⁰

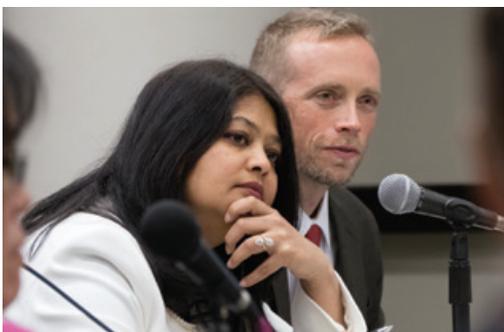


Dr Underwood discussed the pro-inflammatory nature of the infant gut microbiome of some preterm infants.

CONCLUSION AND RESEARCH OPPORTUNITIES

Despite the depth of information presented at the 116th Abbott Nutrition Research Conference on the Maternal Microbiome and Perinatal Colonization, many research opportunities still exist (Fig 6). As Dr Finlay described in his presentation, both epidemiology and molecular biology are now just uncovering the valuable role the gut microbes play in human health.

The maternal gut microbiome is an ever-evolving and highly complex system. Agreement among the scientific community exists that additional maternal and child microbiome research is necessary to improve human health to prevent preterm births and infections and noncommunicable diseases throughout life.



The interactive panel discussion highlighted key outstanding questions and future opportunities in maternal-infant microbiome research.



Fig 6. Maternal microbiome and perinatal colonization research opportunities presented during conference.

116TH ABBOTT NUTRITION RESEARCH CONFERENCE **FACULTY AND SCIENTIFIC STEERING COMMITTEE**



Left to right, front: Dr Francisco Rosales (Abbott), Dr Catherine Stanton, Dr David Sela, Dr Marloes Dekker Nitert, Dr Tamar Gur, Dr Deborah Sloboda, Dr Maureen Geraghty (Abbott)

Left to right, second: Dr Larry Williams (Abbott), Dr Penni Hicks (Abbott), Dr Michelle (Shelley) McGuire, Dr Mark Underwood, Karen Goehring (Abbott), Dr Saradhadevi Varadharaj (Abbott), Dr B. Brett Finlay, Dr Sean Garvey (Abbott)

Left to right, third: GERALYN DUSKA-McEWEN (Abbott), Dr Barbara Marriage (Abbott), Dr Enrique Vazquez (Abbott), Dr JoMay Chow (Abbott), Dr Marlene Borschel (Abbott)

Not pictured: Dr Roberto Romero

FACULTY AGENDA OF THE 116TH **ABBOTT NUTRITION RESEARCH CONFERENCE**

WEDNESDAY, APRIL 19

KEYNOTE ADDRESS

THE ROLE OF INFECTION AND INFLAMMATION IN PRETERM LABOR AND DELIVERY

Roberto Romero, MD, DMedSci
Intramural Division, NICHD, NIH, DHHS
Wayne State University
Detroit, Michigan, USA

THE ROLE OF THE INTESTINAL MICROBIOTA IN NECROTIZING ENTEROCOLITIS & NEONATAL SEPSIS

Mark Underwood, MD, MAS
The University of California Davis School of Medicine
Sacramento, California, USA

THURSDAY, APRIL 20

KEYNOTE ADDRESS

LET THEM EAT DIRT: RAISING CHILDREN WITH THEIR MICROBIOTA

B. Brett Finlay, PhD
University of British Columbia
Vancouver, BC, Canada

PERINATAL PROGRAMMING OF DISEASE RISK: MATERNAL, MICROBIAL AND METABOLIC INFLUENCES

Deborah Sloboda, PhD
McMaster University
Hamilton, Ontario, Canada

THE GUT MICROBIOME REGULATES METABOLISM & BLOOD PRESSURE IN PREGNANCY

Marloes Dekker Nitert, PhD
The University of Queensland
Brisbane, Australia

PRENATAL STRESS AND THE MICROBIOME: RELEVANCE TO NEURODEVELOPMENT

Tamar Gur, MD, PhD
The Ohio State University
Columbus, Ohio, USA

THE HUMAN MILK MICROBIOME—WHAT'S NORMAL, AND POSSIBLE FACTORS MEDIATING VARIABILITY

Michelle (Shelley) McGuire, PhD
Washington State University
Pullman, Washington, USA

HUMAN MILK INTERACTIONS WITH THE DEVELOPING INFANT MICROBIOME

David Sela, PhD
University of Massachusetts Amherst
Amherst, Massachusetts, USA

GUT MICROBIOTA IN DEVELOPING NEONATES

Catherine Stanton, DSc, PhD, MSc, BSc
APC Microbiome Institute & Teagasc
Moorepark Food Research Centre
Cork, Ireland

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