Baylor College of Medicine

USDA/ARS CHILDREN'S NUTRITION RESEARCH CENTER

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Houston, Texas
Permit 2597

NUTRITION & YOUR CHILD
is published by Baylor College of Medicine as a research and information update for the general public and Center volunteers and supporters. Send comments or change of address information to

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The Children's Nutrition Research Center is operated by Baylor College of Medicine, in cooperation with Texas Children's Hospital, for the Agricultural Research Service of the United States Department of Agriculture.

JOIN A CNRC NUTRITION STUDY!

Houston-area residents are invited to participate in the following nutrition research projects designed to help CNRC scientists learn more about the nutritional needs of children. Free parking is provided. For most studies, financial compensation is provided.

For questions on becoming a CNRC research volunteer call Noemi Islam at 713-798-7002 or email nislam@bcm.edu.

Adult Volunteers with Diabetes Needed H-34291

The study will investigate whether type 2 diabetic patients make an important compound called arginine in different amounts. Eligible participants must be African American or Hispanic men, between the ages of 20 and 60 years, diagnosed with type 2 diabetes within the last 10 years, overweight and with no other chronic medical conditions. For more information, contact Adriana Cardenas at 713-798-7003 or adriana.cardenas@bcm.edu.

Teen Talk Study H-46202

Baylor College of Medicine is recruiting 14- to 17-year-olds living in rural communities and their parents to help researchers understand what affects their food and physical activity choices and body weight. For more information, contact Chishinga Callender at 713-798-0506 or Noemi Islam at 713-798-7002.

Teen Heart Health H-30665

12- to 21-year-olds and young adults (normal weight and overweight) with and without type 2 diabetes are needed for a research study investigating risk for heart disease in youth. Study involves body composition, scan and blood tests. If interested, call 713-798-7002

Healthy Pediatric Volunteers Needed H-43759

Healthy boys and girls between ages 3 to 18 years are needed for a study that compares the microbiome of children with primary sclerosing cholangitis (PSC) and ulcerative colitis (UC) to that of healthy children. Those interested must be declared healthy by their pediatrician, speak English fluently, and not be taking any medications, especially antibiotics and hormonal birth control, for at least 6 months prior to participation. Eligible volunteers will collect stool and saliva samples at home five times during one year and send completed samples using a pre-paid mailing package. They also will answer brief online and/or printed surveys coupled with each saliva and stool collection. If interested, contact Alison Shaw via email Alison.Shaw@bcm.edu or call 832-824-0977.

Baylor Infant Biomarker of Nutrition Study H-43692

Help researchers determine if a quick skin reading can tell them about what your infant eats. Researchers are seeking caregivers 18 or older and their infants, 4 months and younger, to participate in a study that involves three visits for skin readings, body measurements, infant food records and optional blood and breast milk sample. For more information, call Jocelyn Chang at 713-798-0517 or e-mail BIBStudy@bcm.edu.

A Pediatric Gastroparesis Registry H-41641

Researchers at Baylor College of Medicine and Texas Children's Hospital are conducting a research study to learn how slow stomach emptying (called gastroparesis) affects children and how to treat it. Children ages 5 to 17 years who have been diagnosed with gastroparesis or have a combination of pain, nausea, vomiting, early satiety or postprandial fullness may be eligible. The study requires visits to the CNRC. If you would like more information about this study, please contact study coordinator Heather Charron at 713-798-0381 or charron@bcm.edu.

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VOL 1 NO. 1 ▪ 2020

NUTRITION & YOUR CHILD

SHAPING YOUR CHILD'S EATING HABITS

Experts from the USDA/ARS Children's Nutrition Research Center at Baylor College of Medicine outline the importance of caregiver influence on children's eating habits in a detailed scientific statement written on behalf of the American Heart Association. CNRC faculty member Dr. Alexis Wood, assistant professor of pediatrics at Baylor, served as the lead author of the statement.

The manuscript focused on what caregivers can do for children, starting while they are still in the womb to age 5, to help them maintain a healthy body weight, in particular the role caregivers play in order to help children develop and maintain eating behaviors associated with a healthy body weight. The statement is unusual in that it focusses not on *what* children eat, but *how* they eat – that is, those behaviors that describe the where, when and how much children eat. Such eating behaviors, for example, eating in the absence of hunger (the amount children eat when they say they are full), have been linked to excess body weight across both child- and adulthood. In fact, the development of healthy eating behaviors may be more important than the actual foods consumed.

"We focused on what caregivers can do to help children develop and maintain eating behaviors that are associated with a healthier weight across the fast-changing developmental stages of the first five years of a child's life," Wood said.

Research suggests that many children are born with the ability to self-regulate their food intake by eating in response to hunger cues and stopping eating when full. During the first five years of a child's life, caregivers face many challenges in allowing children to self-regulate their eating, and these may vary according to a child's developmental trajectory. In infancy, for example, it can be difficult to tell if an infant is crying out of hunger or from distress. Giving a crying baby a bottle to soothe them when they are in distress not related to hunger may lead a baby to eat when not hungry. Researchers theorize that this, in turn, may lead to a general tendency for children to eat when not hungry that persists into adult life. One way to help prevent establishing this tendency may be to educate caregivers and enable them to recognize crying in response to hunger versus distress.

In late infancy, most children develop the ability to communicate their hunger and distress more distinctly, but this stage can bring new challenges. During toddlerhood, it is developmentally normal for children to experience food fussiness and food neophobia (an initial fear of new foods). This can be a challenging time for caregivers who want their child to enjoy a wide variety of foods. Some parental strategies for achieving the goal of encouraging consumption of new foods can have the unintended effect of reducing a child's ability to stop eating in response to their own cues of hunger and fullness, fostering instead a shift by which children eat in response to caregiver cues.

According to Wood, using strategies that are coercive to young children, such as offering rewards or using punishments to get children to try new foods, can alter their ability to self-regulate. Alternative strategies include repeatedly offering the food, eating the food enthusiastically in front of the child, and/ or serving the new food alongside a preferred food, such as serving vegetables with ranch dressing.

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STUDY HIGHLIGHTS IMPORTANCE OF PRE- AND POSTNATAL NUTRITION

A collaborative study in mice by researchers from USDA/ARS Children's Nutrition Research Center at Baylor College of Medicine emphasizes why good nutrition of the pregnant mother and good postnatal nutrition are critical for a healthy life.

The study builds on previous studies that showed that mice that do not get enough to eat during a critical but relatively short period immediately after birth grow more slowly, including slower muscle growth. In the current study, researchers explored how this affects their capacity for physical activity or exercise as adults. The study was published in the *Journal of Physiology*.

"Exercise capacity depends not only on the muscles, but also on the heart's ability to pump blood to the body in response to exercise. So if there is an effect on exercise capacity, it is essential to determine if this is due to the muscles, the heart, or something else," explained Dr. Marta Fiorotto, associate professor of pediatrics – nutrition at the CNRC.

In the study, healthy newborn mice (pups) were nursed by mouse moms that consumed a diet containing insufficient protein. This reduces the amount of milk the moms produce, and as a result their pups grow more slowly than pups nursed by moms fed a normal diet. Once the pups could eat solid food, they were all fed a good diet that supports rapid growth.

When the mice were adults, researchers performed a number of tests similar to those done in humans to assess their heart function. This included a treadmill stress test, echocardiogram and ultrasound measurements of their heart size and function, followed by studies of how their heart cells functioned.

"We found that the mice that had been poorly nourished during this critical postnatal time had a smaller lean (muscle) mass than their well-fed counterparts, and that their hearts were smaller," Fiorotto said. "To our surprise, we found that in males, the smaller heart was in proportion to their smaller lean mass, but in the females, their heart was more severely affected. The results of the exercise test showed that the females' exercise capacity was compromised by their postnatal nutrition."

From the echocardiogram and ultrasound measurements, researchers confirmed that the left ventricle (the engine of the heart) was smaller and its ability to pump blood was lower in the females who had been poorly nourished as infants. When not exercising, the females could maintain normal function by increasing their heart rate. But they were limited in how much they could increase their heart's ability to pump when they needed to work harder, particularly when exercising. In addition to the smaller heart, there were other functional problems with the females' hearts. These findings would help explain why the exercise capacity of the female mice was less than normal.

"These results emphasize how, beginning in utero and through infancy, nutrition and growth can have life-long effects on the health of an individual."

"In addition, we now need to determine what is preventing the compromised growth of the heart in comparison to the rest of the body, and why female mice are especially at risk," she said. "With this information we can determine if there are interventions that could be used to reduce the problem and its lifelong consequences."

Others who contributed to this study include David Ferguson at Michigan State University and a postdoctoral fellow at the CNRC when the study was conducted, Tanner Monroe (now at Duke University), Celia Pena Heredia, Ryan Fleishmann, George Rodney and George Taffet, all of Baylor College of Medicine.

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"If you focus on manipulating the feeding environment, such as the foods presented and how other people interact with them, the child can still stop eating when full and is less likely to look for external cues for when to eat or stop eating. Further, research suggests that such strategies are the most effective way of increasing the likelihood that children will eventually accept a variety of foods," Wood said. "Conversely, if you focus on the child's behavior directly, such as through the use of punishment, threats or even rewards to achieve the eating behavior you want, your approach may, in the moment, prevent the child from self-regulating, because the child isn't eating in response to his or her own cues, but instead is eating in response to caregiver's cues. The tendency to eat in response to the environment, not hunger, is a strong predictor of weight status."

While the statement provides an up-to-date summary of the ways in which caregivers may have a powerful influence on children's eating behaviors, there are limitations. Eating behaviors in young children are very complex, and children differ in the extent to which they can self-regulate their food intake. Genetics is one factor that plays a role, but genetic contributions to food intake have not yet been well integrated into this research.

It is likely that future research will show that different children need different approaches, at least in part due to genetic influences. Similarly, due to factors not immediately apparent during the meal itself, caregivers also have differences in the extent to which they influence the feeding environment. Factors such as food access, financial pressures and mental health also can be powerful forces in shaping the feeding environment. Given these, and the role of genetics, how caregivers structure the feeding environment in the first five years of life is just one factor that can influence the development of eating behaviors.

"It is very important to recognize that just because caregivers have an important role in helping to shape eating behaviors, they do not bear the sole responsibility and in no way should this research be used to place blame on caregivers. Caregiver feeding and child eating is a didactic relationship which operates within a much wider socioeconomic context. It would go against the goal of creating a healthy feeding environment if this statement was used to cause stress on caregivers. Our goal was simply to highlight some of the many ways caregivers may be able help children self-regulate their eating during the first five years of life, and we desperately need research into how to harness other influencing factors," Wood said.

DNA COULD HELP DIAGNOSE BIRTH DEFECTS CAUSED BY MATERNAL DIABETES

Pregnant women with diabetes are at an increased risk of giving birth to a baby with birth defects. This uncommon disorder in newborns is known as diabetic embryopathy. The birth defects can include an improperly formed heart, brain, and skeleton. Researchers at the USDA/ARS Children's Nutrition Research Center at Baylor College of Medicine are studying how small marks on DNA, called methylation, might be used to better identify cases of diabetic embryopathy. Their work was published this year in the journal *Genetics in Medicine*.

Diabetic embryopathy is usually diagnosed through a process of elimination. A baby born with birth defects will be tested for other genetic syndromes. If all of those possibilities are ruled out, and the mother had diabetes during her pregnancy, doctors may diagnose the child with diabetic embryopathy. Dr. Neil Hanchard, assistant professor of molecular and human genetics at Baylor and a researcher at the CNRC, worked alongside Dr. John Belmont, adjunct professor of molecular and human genetics at Baylor, to find a more definitive way to identify this disorder.

"One of the things known about diabetes is that it affects how nutrients are processed down to a cellular level. It can disrupt how genes act," said Hanchard, the study co-author. "There's a hypothesis that maybe diabetes also impacts DNA methylation. Methylation fine-tunes how genes act to control processes in the body. There have been a couple of studies showing that children who had been exposed to diabetes in the womb have unusual DNA methylation patterns. But none of those studies related those patterns to birth defect outcomes."

This study included a control group of healthy newborns whose mothers did not have diabetes and a group of newborns with birth defects consistent with diabetic embryopathy. The researchers took a DNA sample from each child and examined the methylation sites of all their genes. Lead study author and then predoctoral fellow in molecular and human genetics at Baylor, Dr. Katharina Schulze, analyzed those results and found more than 200 regions in the DNA that were significantly different between the two groups. A third group of newborns was also evaluated who were healthy even though their mothers had diabetes; their DNA methylation pattern was somewhere in between that of the control group and the diabetic embryopathy group. According to Hanchard, this suggests the degree of methylation may impact the risk of birth defects.

As a result of identifying this association, the researchers wanted to find out whether the level of DNA methylation at these regions could be used as a test for diabetic embryopathy. Using the same newborns, the researchers randomly chose half of them to create a computer-assisted calculation for diabetic embryopathy and then tested these calculations on the other half.

They found the calculations could provide a fairly accurate classification of whether the child had diabetic embryopathy on the basis of only their genetic analysis (no clinical information). Furthermore, the methylation modifications found in this study were unique to diabetic embryopathy, as opposed to other causes of birth defects, according to Hanchard.

"When a mother has diabetes and her child has birth defects, there's often a lot of anxiety about what caused it," Hanchard said. "We're trying to get to a place where we can make that diagnosis, potentially make it earlier, and hopefully provide helpful information to parents during a really stressful time."

The next step in this research is to conduct DNA methylation analysis on a larger sample size to test for potential clinical uses. While the technique could help with clinical diagnosis, Schulze said their work does not pin down the underlying cause of diabetic embryopathy.

"We don't necessarily know if the changes we see are actually causing the defects," said Schulze, a current research associate in molecular and human genetics at Baylor. "That's why at this stage we are referring to this discovery as a 'biomarker.' We're hoping that we can use this test to improve the diagnosis of diabetic embryopathy. We can't yet say that if we were to change methylation at those specific locations in the DNA that it would change whether birth defects occurred."

"We're hopeful this is sort of an entry into this bigger idea that we can have more accurate diagnoses for this condition," Hanchard said. "Maybe this could be used for other things that can cause birth defects that we don't have good tests for."