

SUBSPECIALIST CONSULT

The Child With Familial Hyperlipidemia

Start with the most important thing—education of the child and the family. This condition is familial, so relatives are more likely to have this form of hyperlipidemia as well.

Ask families about relevant history of early heart disease. “Early familial heart disease” is defined as a father or grandfather younger than age 55 years and/or mother or grandmother younger than 65 years with known heart disease.

We recommend screening all children by the age of 2 years for relevant family history. Studies now indicate lipid deposits can start as early as this age.

Clinical intervention often is more about prevention than treatment. Unless children are homozygous for one of the genetic defects associated with familial hyperlipidemia, they may not have signs or symptoms until they

reach their twenties or thirties.

It is appropriate for you to begin lifestyle recommendations with any overweight or obese child. Counsel the patient and family about better diet and exercise regimens. For example, instruct them to avoid fried foods and if they need to cook with oil, to use vegetable oil.

Recommend 60 minutes of moderate exercise daily. This does not have to be an hour all at once—it can be 20 minutes in the morning before the school bus comes, 20 minutes in the afternoon, and another 20 minutes in the evening. The physical activity does not have to be on the soccer field either. The patient can exercise by climbing the stairs or participating in a scavenger hunt at the mall.

The essential thing is getting the child off the couch and away from the computer. This is particularly important be-

cause many schools are cutting their physical education programs in this economy.

Emphasize to parents that familial hyperlipidemia is one of the preventable forms of heart disease. Parents have a choice if they want their children to lead long, healthy lives.

Monitor the child’s growth. If the child exceeds the 95th percentile on the growth chart, draw cholesterol levels. If the numbers are high, initiate at least a 6-month trial of diet and exercise. If, after this time, the cholesterol levels remain high, consider prescribing a low-dose statin. If medication fails to reduce high cholesterol after 2 months, I recommend these children see a subspecialist like myself.

For the most part, they come to me obese and/or with high cholesterol. I lecture them like you cannot believe, and their weight and cholesterol numbers improve. For this reason, I have very few patients for whom I have to start medication.

The cholesterol assay you do has to be a fasting lipid profile, not a random cho-

lesterol reading. A random test does not provide the most appropriate information. Use common sense regarding when to test kids. In other words, do not test cholesterol levels the day after their birthday, right after Halloween, or anytime between Thanksgiving and Christmas. Testing cholesterol at any time during spring and summer, if possible, is preferable.

You don’t need to refer most children with familial hyperlipidemia for cardiac stress testing. Stress testing is generally reserved for treatment-refractory patients with established high cholesterol. This provides useful baseline information for children we cannot control well. ■



BY ROSE CUMMINGS, D.O.

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Toxicology Panel Finds Soy Infant Formula Poses Minimal Risk for Adverse Developmental Effects

BY JEFF EVANS

ALEXANDRIA, VA. — Soy protein-based infant formula poses “minimal concern” for adverse developmental effects in infants, according to a 14-member panel from the National Toxicology Program who reviewed data from studies of animals and humans.

Their conclusion relied in part on new evidence derived from animal studies published since 2006, when the National Toxicology Program (NTP) last convened a panel to evaluate the safety of soy formulas. At that time, the panel did not complete an evaluation or issue a final opinion.

This time the panel determined that there was more than a “negligible concern” for adverse effects—the lowest of five possible levels of concern—primarily because several experimental animal studies and one study in humans reported adverse effects of soy isoflavones on the reproductive system.

Some of the new animal studies showed that rat pups fed pure genistein, a soy-derived isoflavone, had blood levels of the compound that were the same as those that have been reported in studies of infants who were fed soy formula. Genistein was associated with adverse effects on the rats’ reproductive system, such as uterine changes.

These findings prompted the expert panel to reexamine the evidence base for adverse developmental effects in humans, Dr. Gail McCarver, chair of the expert panel and a neonatologist and toxicologist at the Children’s Hospital of Wisconsin, Milwaukee, said in an interview.

The panel voted 10-2 in favor of minimal concern for adverse developmental effects, with one panel member voting for “some concern” and another voting for negligible concern. (The chair of the expert panel does not vote and one panelist was not present for the vote.)

The NTP is an interagency program headquartered at the Center for the Evaluation of Risks to Human Reproduction at the National Institute of Environmental Health Sciences. In 2010, the NTP, which has no regulatory authority, will prepare a final briefing from the report, public comments, and any new studies published.

In a summary of their conclusions, the panelists wrote that there was little evidence to support a higher level of concern because “studies of sufficient quality in humans have not been conducted to address the concerns raised from the experimental animal findings or to identify previously unrecognized end points.”

The only study that gave reason for a concern for adverse effects in humans showed that women who had been fed soy formula as infants had a significantly longer menstruation period (0.37 days) than did women who had been fed cow’s milk formula (JAMA 2001;286:807-14).

“The results today support the [American Academy of Pediatrics’] recent pol-

icy and what a lot of pediatricians are already doing: Saying that soy formula is best for vegetarian families who want to feed their kids in a vegetarian way or have a specific concern such as galactosemia or congenital lactase deficiency,” Dr. Alan Greene of Stanford (Calif.) University said in an interview.

The AAP’s 2008 guidance and review of the literature reported that “there is no conclusive evidence from animal,

adult human, or infant populations that dietary soy isoflavones may adversely affect human development, reproduction, or endocrine function” (Pediatrics 2008;121:1062-8).

Despite the gaps in research on the developmental effects of soy isoflavones in humans, Dr. Greene said he did not think isoflavones took priority over other compounds that may affect development. “I’m much more concerned about some of the estrogens that are in plastics and cosmetics—BPA and phthalates and endocrine-disrupting pesticides—than I am about the soy issue.” ■

DR. GREENE

The results support the AAP’s recent policy on dietary soy isoflavones, as well as current pediatric practice.

Disclosures: Dr. McCarver said she and the other panelists had no relevant disclosures to make. Dr. Greene was not involved with the drafting of the report. He said that he has performed consulting work for Silk, which manufactures soy milk, but has no relevant disclosures with manufacturers of soy infant formulas.

Understanding Acne Aids in Treatment

WASHINGTON — Understanding the pathogenesis of acne better equips physicians to manage the disorder and educate patients, Dr. Richard J. Antaya advised at the annual meeting of the American Academy of Pediatrics.

Microscopic precursor lesions called microcomedones evolve into two main types of lesions: open or closed comedones; or inflammatory lesions. The evolution involves several major factors: a defect in follicular keratinization, whereby the cells in the upper portion of the follicle become sticky and fail to shed; increased sebum production; and *Propionibacterium acnes* bacteria.

“How exactly the increased sebum plays into the follicular keratinization defect, or if it’s a separate defect altogether, is not really clear,” he explained. There is still more to learn about the role of hormone receptors, about how comedones form at the molecular level, and about why there is scarring in some cases and not in others.

“And we really don’t get why some patients go into remission,” said Dr. Antaya, director of pediatric dermatology at Yale University, New Haven, Conn. “If you look at sebum secretion rates in these patients, there’s really no difference—something happens ... in their immune system that stops the acne process.” The immune response to *P. acnes* is “where we may be seeing the true pathogenesis of inflammation.” Studies have shown that the severity of acne is proportional to *P. acnes* antibody titers.

—Christine Kilgore