Chronic Nonspecific Diarrhea of Childhood

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Introduction

Over the past half century, the entity known as chronic nonspecific diarrhea of childhood or toddler's diarrhea, has followed a path from case descriptions to disease and finally, within the past 10 years, to a defined functional disorder. Chronic nonspecific diarrhea of childhood was originally thought to be part of the ‘celiac syndrome’. As Davidson and Wasserman [1] noted in their seminal paper published in the Journal of Pediatrics in December of 1966, the pioneering pediatric gastroenterologists of the time, had defined a number of specific disorders within what was then called the ‘celiac syndrome’, including gluten-induced enteropathy, disaccharidase deficiencies, lymphangiectasia and abetalipoproteinemia. Chronic nonspecific diarrhea was the general term within that syndrome given to those children with persistent diarrhea where no cause could be identified. Anderson [2] felt that chronic nonspecific diarrhea was a result of starch intolerance and advocated a high-protein, low-fat, low-starch diet. Davidson and Bauer [3] and Prugh and Shwachman [4] showed that starch ingestion was not the culprit and in 1956 Cohlan [5] first used the term chronic nonspecific diarrhea.

Ten years later Davidson and Wasserman [1], in a retrospective examination of the case histories of 186 children, provided a description of this entity, which remains accepted even today, 50 years later. While a strict definition of diarrhea was not provided in this paper, these children were selected because of persistent or recurring episodes of loose stools without an identifiable cause, including cystic fibrosis, identifiable pathogens, celiac disease and other enteropathies. In more than 75% of the children diarrhea began between 6 and 20 months of age, although in 12% the onset was between...
birth and 5 months of age. Eighty-eight percent had cleared the diarrhea by 39 months of age, with another 10% clearing it by 48 months of age. The character of the stool was fairly consistent for the majority of children in that the first stool of the day was large and partly formed. Later bowel movements during the day were smaller and looser. Mucous in the stool was reported in 87% of the patients. When blood was present it was always associated with fissures or excoriation of the perianal skin. Every patient was growing and gaining weight normally according to reference standards of the time and functional bowel disorders, i.e. ‘irritable bowel syndrome’, were very common among the parents and families of these patients. Five to forty-seven percent of these patients who were treated with specific agents such as kaolin/pectin, bismuth and various antibiotics had a clear, positive response to these interventions. Perhaps most importantly, 80% improved when a full and normal diet for age was instituted.

In 1999 a working team published a set of definitions for childhood functional gastrointestinal disorders following on the Rome criteria published for adults [6]. Functional diarrhea, which the team recognized was also known as toddler’s diarrhea, chronic nonspecific diarrhea and irritable colon of childhood, was defined by daily painless passage of 3 or more large unformed stools for more than 4 weeks, with an onset of between 6 and 36 months of age, and passage of stools during waking hours in children who were thriving on an adequate calorie intake. The working team emphasized the importance of avoiding restrictive diets, the fact that children recover spontaneously and that the most effective treatment is reassurance for the parents.

**Etiology**

Dietary fat intake was shown to play a role in a significant number of children with chronic nonspecific diarrhea. In 1979 Cohen et al. [7] reported 5 patients with the onset of chronic nonspecific diarrhea that coincided with efforts to restrict fat in the children’s diets in an attempt to protect against the occurrence of coronary vascular disease in later life. By and large these patients had been placed on skim milk and the diets contained ≤27% of total calories derived from fat. In addition, several of these children were ingesting large amounts of fruit juice in the diet. When the fat in the diet was increased to between 35 and 50% of total calories, the diarrhea symptoms resolved in all 5 of these children. Cohen et al. [8] extended these observations in a subsequent report later in that same year in which they showed that increasing the fat content of the diet resulted in the resolution of diarrhea in 82% of another group of children with chronic nonspecific diarrhea. They could not account for the lack of response to this dietary change in the remaining 18% of patients in this retrospective study but felt that the carbohydrate, fiber and calorie content of the diet did not play as important a role as fat intake.
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Stool analysis has not been particularly helpful in determining the etiology of chronic nonspecific diarrhea of childhood. Davidson and Wasserman [1] observed the presence of vegetable fibers, starch and fat droplets upon microscopic stool examination in a high percentage of their reported patients. Subsequently Jonas and Diver-Haber [9] suggested that the extractable water phase of stools was appreciably increased in patients with chronic nonspecific diarrhea and that this water phase contained 50% of the total stool bile acids. While their 7 study subjects had normal stool weights when compared to controls (between 5 and 10 g/kg/24 h), stool electrolyte and bile acid concentrations were moderately increased. These findings were similar to a third group of patients studied with bacterial overgrowth syndrome. The authors suggested that children with chronic nonspecific diarrhea have an induced secretory state in the large bowel as a result of bile acids entering the colon, leading to the production of loose watery stools. The validity of these findings has not been confirmed. It should be noted that those patients in the group with chronic nonspecific diarrhea were between 18 months and 12 years of age and, of the 7 children, 5 had persistent abdominal pain. Furthermore the control subjects were between 8 and 15 years of age. Neither of these groups, therefore, may truly represent the typical infant or child with or without chronic nonspecific diarrhea.

Disordered intestinal motility has also been suggested as the basis of toddler's diarrhea. Intravenous dextrose infusion failed to disrupt the migrating motor complex in any of 8 children studied with toddler's diarrhea [10]. However children with chronic nonspecific diarrhea do not malabsorb, as is often the case for those with severe motility disorders, and the ages of the children studied were between 3.7 and 11.5 years, an age range much more typical for irritable colon than for toddler's diarrhea.

A number of authors have suggested that the excessive consumption of beverages containing high concentrations of various sugars contributes to or causes chronic nonspecific diarrhea. By the early 1990s it was reported that children younger than 12 years of age consume 28% of all juice and juice drinks in the United States, in spite of constituting only 18% of the population. National surveys in the United States have shown that almost 90% of infants consume juice and 10% of children 2–3 years old drink more than 355 ml/day [11]. The principle carbohydrates of fruit juices include sucrose, fructose, glucose and sorbitol. Maximum absorption of the sugars, glucose and fructose, occurs when they are present in equimolar concentrations [12]. When the fructose concentration in a beverage exceeds the concentration of glucose, as it does in apple and pear juice compared to white grape juice, then more malabsorption occurs [13]. Most importantly, however, the sugars from all of these juices are equally well absorbed if an excessive amount of juice is not consumed (i.e. ≤10 ml/kg body weight) [14]. The consequence of carbohydrate malabsorption is the production of hydrogen, carbon dioxide, methane and the short-chain fatty acids, acetic,
propionic and butyric acid in the colon. Salvage of these gasses and fatty acids occurs in the colon thereby scavenging a portion of the malabsorbed carbohydrate. The carbohydrates and fatty acids that remain can induce fluid secretion as well as present an osmotic load to the colon leading to chronic diarrhea.

Breath hydrogen testing has been used to further examine the roles of the various carbohydrates contained in fruit juice in chronic nonspecific diarrhea. Kneepkens et al. [15] demonstrated a significant increase in breath hydrogen into the abnormal range following the ingestion of 250 ml of apple juice in both children with chronic nonspecific diarrhea as well as a group without gastrointestinal symptoms. Daily apple juice consumption did not differ significantly between the 2 groups (between 236.5 and 443.6 ml juice/day on average). When glucose was added to the apple juice the breath hydrogen concentrations decreased significantly. Thus it was felt that fructose was accounting for approximately 80% of the incomplete carbohydrate absorption and sorbitol for 20%. When apple juice was eliminated from the diets of those with chronic nonspecific diarrhea, normalization of the frequency and consistency of the stools occurred.

Hoekstra et al. [16] extended these observations and showed that other substances in fruit juice in addition to fructose and sorbitol may be implicated in causing the diarrheal stools. They compared processed apple juice, which had been treated enzymatically to produce a clear fluid with freshly pressed and unprocessed juice. When both types of juice were provided at 10 ml/kg, breath hydrogen increased to ≥20 ppm in 8 of the 10 individuals consuming clear juice compared to 5 of 10 consuming cloudy unprocessed juice. The mean breath hydrogen concentration was higher in those who consumed clear apple juice. These juices were then provided in a 4-week crossover clinical trial to 12 children. Clear apple juice significantly promoted diarrhea, and the authors suggested that nonabsorbable mono- and disaccharides present as a result of the enzymatic processing of apple pulp were an important factor in apple juice-induced chronic nonspecific diarrhea.

More recently Lebenthal-Bendor et al. [17] showed that acetylated distarch phosphate, a modified starch used in some baby foods, led to elevated breath hydrogen in 2 of 21 toddlers who consumed a formula containing 8% of the modified starch. None of these toddlers had loose stools unless they consumed a formula that contained 2% sorbitol and 5% fructose in addition to the modified starch. However, sorbitol added to the formula containing native starch, which was used as the control formula, also led to loose stools in 2 of the study subjects. Thus it is not clear that modified starch by itself plays any significant role in chronic nonspecific diarrhea of infancy in the absence of other known factors such as consumption of sorbitol and fructose.

Greene and Ghishan [18] documented that almost one fifth of the 85 patients they reported on with chronic nonspecific diarrhea consumed more
than 2.5 times their daily fluid requirement in addition to their usual diet. Most of the fluids consumed by their patients with chronic nonspecific diarrhea were hypertonic because of high concentrations of carbohydrate, although 3 of these patients were consuming large volumes of water alone. Thus it is clear that fluid intake in excess of the capacity of the intestinal tract to absorb it, and in many cases combined with a high osmotic load, is an important factor in the development of chronic nonspecific diarrhea in many children.

**Treatment**

In the past 50 years many agents have been used to treat chronic nonspecific diarrhea. Davidson and Wasserman [1] reported significant success with a number of different agents and approaches. Cohlan [5], who, as noted, coined the term chronic nonspecific diarrhea, reported on the successful use of Diodoquin in his description of this entity. Hamdi and Dodge [19] suggested that aspirin and loperamide are effective in the treatment of chronic nonspecific diarrhea, in part as a result of an increase in plasma prog-staglandins, particularly PGF$_2\alpha$, that they observed in their study subjects with chronic nonspecific diarrhea [20]. The validity of the observation regarding plasma prostaglandins and its relationship to chronic nonspecific diarrhea remains to be established.

Nevertheless, treatment with both aspirin and loperamide carries significant risks, particularly when used for a condition that by and large can be treated by dietary modifications and in any event has no significant health implications. In one report, 3 children between the ages of 23 and 34 months with toddler's diarrhea unresponsive to dietary changes were treated with loperamide. All 3 became drowsy with irritability and personality changes within 3–5 days of starting treatment [21]. Loperamide has opiate-like toxic effects and may clearly cause severe central nervous system depression along with its effects on the gastrointestinal tract. Aspirin has been linked with Reyes syndrome and when used indiscriminately may result in acute or chronic salicylism. Thus both loperamide and aspirin are unacceptable treatments for toddler's diarrhea. The use of antibiotics is also inappropriate for the treatment of toddler's diarrhea. Diodoquin, for example (used by Cohlan [5]), can cause an irreversible neuropathy. Microbial resistance to antibiotics is a growing public health issue and there is no place for antibiotic use in the treatment of chronic nonspecific diarrhea.

In addition to normalizing and optimizing the diet of children with chronic nonspecific diarrhea, added dietary fiber may be of some benefit. Smalley et al. [22] reported that 7 of 23 children with chronic nonspecific diarrhea responded to an unrestricted diet alone. When psyllium was added to the diets of these 23 children, 87% responded to the combination of unrestricted
diet and bulking agent. As the authors point out, this was not a controlled study and they emphasize that a normal unrestricted diet should be the first approach to the treatment of this condition.

Conclusion

Approximately 50 years of observation and investigation have defined chronic nonspecific diarrhea of childhood or toddler’s diarrhea as a functional bowel disorder with no consequences for growth, development or long-term health. The incidence and prevalence of this functional disorder is unknown, although it is clearly a common condition seen frequently by primary care physicians and pediatric gastroenterologists. Laboratory investigations are of no use or benefit when the criteria for chronic nonspecific diarrhea in infancy are met. Breath hydrogen testing in infants with chronic nonspecific diarrhea, including the fructose breath hydrogen test, is of no use [23].

The most useful approach to this disorder is to reassure the parents, to normalize the diet within current guidelines for carbohydrate, protein, fat and fluid intake, and to observe the child. It has also been noted that the symptoms of chronic nonspecific diarrhea may decrease significantly simply by asking parents to create a diet diary and record stool frequency and consistency for a 1-week period before any specific dietary interventions are initiated [24]. Thus raising the amount of fat in the diet above that recommended in current guidelines is not required. This disorder inevitably resolves by the time the child starts school. There is overlap between this disorder and irritable colon of childhood and some of those children with a diagnosis of chronic nonspecific diarrhea who have intermittent periods of diarrhea after starting school may in fact have irritable bowel syndrome.

References

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Discussion

Dr. H. Hoekstra: The symposium will end with educational recommendations. Do you have more recommendations in this group of children?

Dr. Kleinman: I think education is the best approach to this disorder. Primary care providers, as well as gastroenterologists need to be educated that this is a problem and not a disease. There are two target audiences, the health care providers and the parents. This can be accomplished with printed materials, or electronic formats, materials that are handed out in the physicians office and informational articles in lay magazines, which is where parents today get most of their medical information. I think that is the effective way of dealing with this issue.

Dr. H. Hoekstra: I think it is important to make a recommendation to reinstall the meal in-between. We should advise against the practice of toddlers walking with a bottle between the 3 main courses, and we should recommend the restoration of the coffee and tea break with sitting down and eating an apple instead of drinking apple juice.

Dr. Kleinman: Parents equate fruit juice with fruit and feel that if they can provide the juice then they are providing the nutritional benefit of the fruit. Thus some part of that reeducation has to be to get parents to understand that fruit is a very appropriate part of the child's diet and the juice really at the very least needs to be quite limited and could probably be eliminated from the diet with no harmful effect at all.
Dr. Hernell: I have the impression that, at least in Sweden, 10–15 years ago nonspecific diarrhea was a rather common diagnosis and then it just disappeared. Now inflammatory bowel syndrome in childhood has become a much more common diagnosis. Do you think that it is just semantics or what the explanation?

Dr. Kleinman: Anecdotally we have had the same experience. I think there is enough overlap between chronic nonspecific diarrhea (CNSD) and irritable colon, so that if the prevalence of irritable colon is increasing, as I think it is, in our own minds we are probably blending the two right now and diagnosing irritable colon more readily than we did before. But I do have the overall impression that I don’t see as much CNSD as I did 20 or 30 years ago or even 10 years ago.

Dr. Sinaasapel: I remember that we had the same situation in the 1980s. We had a large number of children with this disorder and when we wanted to start a study with this group it just disappeared, and thereafter we hardly ever saw it again. I can’t remember exactly, but I have the impression that at that time we did not have as many problems with constipation as we have now. So I have the impression that we changed the problem from diarrhea to constipation at that time, but I am probably wrong. One small comment, it is not only the juice in boxes but sometimes the habits of the parents that must be questioned. When they say that they just give 1 orange/day or that they give one orange juice/day, they must also be asked how many oranges they use. Sometimes in my practice I hear that the parents use 8–10 oranges/day for a small child. So when simply asking about fruit, it is also important to ask what fruit is given and how much.

Dr. Kleinman: In terms of fruit, unfortunately in the United States we almost never have to ask how many oranges parents feed their child each day because the answer generally is zero. Fruits and vegetables are really under-consumed, certainly in the United States and in other developed countries.

Dr. Branski: I would like to ask you to comment on anecdotal papers on the role of prostaglandins in causing CNSD. Also reported in these articles was that inhibition of prostaglandin synthesis via the cyclooxygenase pathway by aspirin and indomethacin (non-steroidal anti-inflammatory drugs) was helpful [1, 2].

Dr. Kleinman: There have been several papers written on prostaglandin use for CNSD. I didn’t discuss them in my presentation because they are so anecdotal and the study populations so poorly defined that I didn’t think it was worthwhile. As you said indomethacin was used in the past for some of these children and the high risk of an adverse effect of that or any other prostaglandin-inhibiting agent make it almost incomprehensible that anyone would use these drugs for CNSD today.

Mr. Benjacob: Is gut permeability altered in these children?

Dr. Kleinman: I don’t know of any studies that examined gut permeability in children with CNSD. I would imagine, because these are otherwise completely healthy children with healthy guts, that if one examines gut permeability it will be no different from an age-matched healthy child.

Dr. El-Din Amry: I would like to ask about the concept of a diarrheagenic diet and constipating diet. In every culture it is well known that some foods cause constipation and other foods can cause mild diarrhea, and pediatricians sometimes use this concept to help to treat their patients: with simple gastroenteritis they give them a diet that causes constipation and in the case of constipation they give them a diet that causes looser stools. What do you think of this concept?

Dr. Kleinman: I guess I would take issue with the concept that there are foods that are likely to cause diarrhea and other foods that are likely to cause constipation. I think that most of the literature studying the question shows in fact that the stool character depends upon the amount of a particular food, the age at which it is consumed, the concentration of nutrients in that food and other factors. So the idea that
any particular food is constipating or likely to cause diarrhea I think is a notion that we probably need to move away from and instead consider the entire diet of the child and whether it is appropriate for age.

Dr. H. Hoekstra: Perhaps I can comment on this point as well. Another way to look at the stool problems in toddler's diarrhea is by looking at the water-holding properties of the stool. This aspect has been intensively studied by Wenzl et al. [3]. Diarrhea can be defined as a failure of the water-holding properties, and this may well be the case in CNSD.

Dr. Bueno: You mainly emphasized the possibility that food is involved in such a disease which is close to irritable bowel syndrome in children. Do we have any data suggesting that stressful events may also play a role? Do we now have retrospective studies suggesting that children suffering from CNSD may have a prevalence for irritable bowel syndrome in adult life?

Dr. Kleinman: In terms of stress, anecdotally virtually all of these are healthy happy little toddlers, so probably stress does not play a huge role. But as Davidson pointed out, there is often a strong family history of irritable bowel syndrome. It is very difficult in a 2-year-old to tell whether they have an irritable bowel syndrome or CNSD. Most of the time we end up making the diagnosis of irritable bowel syndrome when the condition persists beyond 4 or 5 years.

Dr. Schmitz: You said that this is a condition that occurs between 6 months and 3–4 years of age, which I think is our experience, meaning that it occurs during a window in the life of the children. As you also said, it can be corrected by a modification of the diet or induced by the diet. So my question is, what correlation do you make between the fact that it occurs at a given age and the fact that this is a time of diversification and introduction of new foods?

Dr. Kleinman: That is an excellent point. It is a period of time when the diet of the young child actually tends to be quite monotonous, and while they are adding new foods, neophobia, fear of new foods, is also very strong at that age, and so there are lots of opportunities for these young children to be on relatively restricted diets. So I think it is a very unique opportunity for us to provide not only nutritional counseling but behavioral counseling to parents, and we ought to capitalize on that and help parents to understand how to change behavior in a positive way in the young child.

Dr. Schmitz: That is a good interpretation, but there is another one which finds that this period of age is the one in which the child begins to eat fibers because he/she has never eaten fibers before that. So it could be said that chronic diarrhea is also a kind of situation of non-adaptation to a new food, which would be fibers. Then since fibers are not digested by the gut, by the small bowel, it could be viewed as a phase of adaptation of the colonic motility or flora or both, I don't know, to this new food. This would explain why CNSD is typically a situation of weaning and of food diversification. What is your opinion on that?

Dr. Kleinman: I think it is a good idea. In the US the consumption of fiber is low, particularly during those years, and I am not sure how many children that would pertain to. I think it is an excellent idea and certainly one that we could look at pretty easily.

Dr. H. Hoekstra: So it fits well with the concept of Dr. Kneepkens of inadequate fermentation.

Dr. Schmitz: Exactly, and it would be nice to have Dr. Salminen's opinion about the possibilities of bacteria in this situation. What happens to the fibers? What do you know about that?

Dr. Salminen: One thing we know is that the bacteria wonderfully adapt to different situations, so I am actually trying to reflect back to the chronic. I rather see problems in short-term diarrhea caused by bacteria because the adaptation is not always
immediate, but if you work on the basis of days and weeks, they adapt to utilizing most carbohydrate substrates. So there is perhaps an added extra to what you said.

Dr. Hernell: I think it is quite clear that this can be only part of the explanation for this type of diarrhea because we saw children with a well defined diet and just made them increase the amount of fat in the diet and many of them were cured. So this seems to be a symptom and the reasons behind it are most likely multiple.

Dr. Kleinman: And don't forget, in all of the studies anywhere from 15 to 20% of the subjects don't respond to those dietary manipulations. So either it is a heterogeneous group of children, some of whom have irritable bowel syndrome and perhaps other issues, and in addition there may be some other mechanisms that remain to be understood like the fiber issue.

Dr. H. Hoekstra: I have a question for Dr. Salminen. In a situation of very fast intestinal transit, is it possible to temporally flush out a normal flora?

Dr. Salminen: I think it is extremely difficult to flush it out totally. It will come back on the mucosal level in the long run; it can be disturbed for a long while but I would say in most of the cases it will return. You can of course disturb this by different means but it is very difficult to, unless it is in the very young infant.

Dr. Sinaasappel: With regard to your recommendations, and I think this is a disorder or a complaint from developed countries. But developing countries are imitating our habits and always walk a little bit behind us, so I think that although the problem can be solved quite easily, it might be a problem for developing countries in particular. They also have a load of infections in those areas which might increase the problem or might harm the child, although it is just a preventable cause. Although we agree on the cause and how to treat it or to prevent it, I think for developing countries it is a different matter.

Dr. Kleinman: Developing countries are going to be very busy dealing with obesity and at the same time trying to figure out how to treat hunger and starvation, so they may not have time to pay attention to this issue. But you are absolutely right, the kind of things that we have seen in the industrialized world tend to appear in the developing world 20 years afterwards. So it is something to keep in an eye on.

Dr. Keller: I have a question regarding improving toddler's diarrhea with fats. This is true in part for this nonspecific diarrhea of childhood. You told us about some overlap to irritable bowel syndrome, but there are some, at least adolescents or schoolchildren or adults, with irritable bowel syndrome whose symptoms will worsen by adding fat.

Dr. Kleinman: Yes, and in fact when those papers were published in the late 1970s it seemed intuitive. It is very clear that many of the adults with irritable bowel syndrome have symptoms that are much worse if they are eating a high-fat diet. So one of the objections to increasing the fat in the diet of children with CNSD was that it was going to make it worse, but the fact is it doesn't. For those children who have CNSD and not irritable bowel syndrome, the fat does seem to improve it. Nobody today advocates a very high-fat diet for these children. What most recommend is a diet that has about 30% of total calories from fat.

Dr. Leathwood: There is a complex communication problem to be solved. The parents are worried and upset. They are getting advice, sometimes contradictory, from many sources and they no longer know who to believe. To them, your recommendations even seem to go against what they thought was 'healthy eating'. So it should not be surprising that you have difficulties to convince them.

Dr. Kleinman: Yes, it goes well beyond what we can discuss here in the next few minutes, but clearly part of this is establishing credibility with the parents, and that generally doesn't happen the first time we meet them. The second is that they have to be convinced that what you are recommending is not going to harm their child, and that
generally doesn’t happen the first time that you meet them. Perhaps, at least in part, this helps explain why many of us still do tests on these children. We were talking about the breath test and the fact that the number of breath test that we do has decreased significantly but we still do breath test and one of the times that I recommend the breath test is in the parents who are so anxious that my words aren’t going to get through to them, they require some objective measure of their child’s health, and if you can offer them a relatively non invasive, not terribly expensive test then I think most of us go ahead and do that, but it does require significant efforts and it is not simply a matter of saying you child is well, go home and call me in 6 months. That is particularly true with the behavioral issue, you have to give them specific recommendations on how to modify behavior and that is not easy to do for many parents. I think that is a major difference between the 1950s and 60s and today. Many parents in the United States are very reluctant to provide significant guidance for their young children in the sense that in some way by limiting freedom you harm the child’s development and that is a very complex concept to work through. So I agree exactly with what you said and perhaps we will hear later in this conference some specific educational recommendations that will help us.

Dr. Steenhout: When you recommend stopping the consumption of fruit juice and installing treatment with increasing fat, how long does it take to see an improvement? My second question is, what sort of fat? Just an increased percentage of fat in the diet or have you some specific fat profile to recommend, balanced between n-3 and n-6?

Dr. Kleinman: The response happens very quickly. Within a day the character of the stool changes so it is one of those gratifying experiences where you make a recommendation and they actually see a cure in a sense, and it is a very good way to establish credibility with the patient. In terms of the fat in the diet, for most of the children who are on a diet that is severely restricted in fat, the parents have achieved that by either taking all dairy products out of the diet or shifting to non-fat dairy products. So the simplest thing to do is just to return the child to a full-fat diet or a diet containing a 1% fat dairy product. Most parents have little understanding of n-3 fats.

Dr. Steenhout: If the effect is practically immediate, in one day, by which mechanism can it be explained? Is it just by retrieving the sugar or the contaminant coming from the process of the fruit juice, or do you have an explanation by which mechanism the fat could act?

Dr. Kleinman: No one has experimentally defined the mechanism of fat in CNSD but the effect of fat on gut motility is well explored and it is the principle dietary nutrient that is responsible for the so-called duodenal break. The fat clearly slows motility. Most of these children who have a very low-fat diet are also consuming a lot of carbohydrates, a lot of juice, so it is almost always a mixed pattern. Thus the combination of adding some fat which slows motility and decreasing the amount of fluid and the amount of carbohydrate leads to a very rapid response. I don’t mean to make light of the importance of having children on an optimal blend of fats in the diet. The best way we can do that is by recommending a pattern of foods that the children should be eating and not so much getting into the science of different kinds of fat.

Dr. Moreno Villares: Going back to the idea of Dr. Schmitz concerning adaptation. In the last years, as more children from other countries come to our country in this range of age, we have seen that chronic diarrhea improves with time with no infectious disease. When they go back to their own country and then return to our country they again have these episodes of chronic diarrhea as if microflora could change because of the meal pattern in the different cultures. So we do not see classic toddler’s diarrhea, but this kind of episodic diarrhea in these people from foreign countries and traveling from one country to another.
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*Dr. Kleinman:* I would ask our colleague from Finland to comment on changes in stool flora or microbiota. When moving from country to country do you think that is the principal issue that is operating here or is it just that they change diet when they go from one country to another?

*Dr. Salminen:* I think it is probably both and we all know from adult situations that going from country to country can change our microbiota and have some consequences. But I think it is a combination of both because the diet can be totally different and the meal patterns are different, combined with the microbial changes.

**References**