Maturation of Visual Acuity Is Accelerated in Breast-Fed Term Infants Fed Baby Food Containing DHA-Enriched Egg Yolk

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ABSTRACT Between 6 and 12 mo of age, blood levels of the (n-3) long-chain PUFA, docosahexaenoic acid (DHA), in breast-fed infants typically decrease due to diminished maternal DHA stores and the introduction of DHA-poor solid foods displacing human milk as the primary source of nutrition. Thus, we utilized a randomized, clinical trial format to evaluate the effect of supplemental DHA in solid foods on visual development of breast-fed infants with the primary outcome, sweep visual-evoked potential (VEP) acuity, as an index for maturation of the retina and visual cortex. At 6 mo of age, breast-fed infants were randomly assigned to receive 1 jar (113 g/d) of baby food containing egg yolk enriched with DHA (115 mg DHA/100 g food; n = 25) or control baby food (0 mg DHA; n = 26). Gravimetric measures were used to estimate the supplemental DHA intake which was 83 mg DHA/d in the supplemented group and 0 mg/d in controls. Although many infants in both groups continued to breast-feed for a mean of 9 mo, RBC DHA levels decreased significantly between 6 and 12 mo (from 3.8 to 3.0 g/100 g total fatty acids) in control infants, whereas RBC DHA levels increased by 34% from 4.1 to 5.5 g/100 g by 12 mo in supplemented infants. VEP acuity at 6 mo was 0.49 logMAR (minimum angle of resolution) and improved to 0.29 logMAR by 12 mo in controls. In DHA-supplemented infants, VEP acuity was 0.48 logMAR at 6 mo and matured to 0.14 logMAR at 12 mo (1.5 lines on the eye chart better than controls). At 12 mo, the difference corresponded to 1.5 lines on the eye chart. RBC DHA levels and VEP acuity at 12 mo were correlated (r = 0.50; P = 0.0002), supporting the need of an adequate dietary supply of DHA throughout 1 y of life for neural development. J. Nutr. 134: 2307–2313, 2004.

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Long-chain PUFA (LCPUFA)⁶ have an important role in visual development during infancy. Compared with infants fed commercial formulas lacking LCPUFA, breast-fed infants have more advanced electroretinographic function as early as 6 wk of age (1) and more mature visual acuity by 4 mo of age (2). In longitudinal assessment of the effect of maternal and infant dietary factors in infant visual development, Williams et al. (3) reported that the variable most associated with stereovisual acuity at 3.5 y of age was breast-feeding. Children who had nursed for even short periods during infancy had more mature visual stereovisual acuity than children who had never received human milk. The (n-3) LCPUFA, docosahexaenoic acid [DHA; 22:6(n-3)], which is present in human milk but absent in unsupplemented infant formulas, may be the major factor responsible for this benefit.

Direct evidence for a role of DHA in visual development is that term infants fed formula with adequate amounts of DHA and a balanced amount of the 20-carbon (n-6) LCPUFA, arachidonic acid [ARA; 20:4(n-6)], have improved visual and mental development with no adverse effect on growth (4–6). Furthermore, DHA in infant formula was associated with shorter-look duration to novel stimuli on the Fagan test (7) and with improved visual acuity in a multistudy meta-analysis (8). Critical reviews of this literature were published recently (9–11).

A woman producing milk for her infant derives a major portion of milk LCPUFA from her endogenous stores (12). Human milk can vary considerably in its LCPUFA content depending on the diet of the mother and the amount of

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LCPUFA mobilized from her tissues over the course of the current and any preceding pregnancies and/or lactations (13). The concentration of DHA in human milk varies from as little as 0.1% of total fatty acids in women consuming Western diets to as much as 1.4% in Inuit women in North America and 2.78% in Chinese women from a fishing village, both consuming large amounts of marine animal foods (14–16).

The rationale for this study was that at ~6 mo of age, infants are beginning to be fed semisolids foods and thus are likely to have a reduction in dietary DHA as reflected in decreased blood DHA levels (4,6). This reduction in the infant’s DHA intake may be due to a concomitant reduction in consumption of human milk (17) combined with increasing intake of DHA-poor weaning foods (18).

The objective of this randomized clinical trial was to determine whether DHA-enriched baby food provided as a supplemental source of DHA to breast-fed infants in the second 6 mo of life altered blood lipid fatty acid profiles and modified visual development. In addition, to assess whether the long-chain fatty acids affected infant metabolism, we evaluated total antioxidant capacity, blood chemistry, and hematology. Supplementary DHA was provided in the form of ready-to-feed baby foods made with DHA-enriched egg yolk providing DHA and ARA.

**SUBJECTS AND METHODS**

**Subjects.** Healthy term infants receiving human milk born at either Presbyterian Hospital of Dallas or Medical City Dallas Hospital were enrolled in the study. Additional infants were recruited through advertisements. Inclusion criteria were a gestational age at birth >37 wk, a birthweight >2800 g, exclusive breastfeeding in hospital and for the first 4 mo of life with a maternal intention to continue breastfeeding, a good possibility of long-term follow-up, and informed consent to the protocol. Exclusion criteria were any underlying disease or congenital malformation that was judged likely to interfere with the evaluation of the study material, any abnormal maternal dietary patterns, and any evidence of maternal metabolic disease.

Informed consent was obtained from one or both parents before the infant’s participation. This research protocol observed the tenets of the Declaration of Helsinki and was approved by the Institutional Review Boards of the University of Texas Southwestern Medical Center (Dallas), Presbyterian Hospital of Dallas, and Medical City Dallas Hospital.

Computer-generated randomization codes with variable-length blocks of 8–12 were used to assign 55 infants to 1 of 2 groups at 6 mo of age. Both groups received study baby foods and were directed to feed the baby 1 jar of study food per day. As incentive, all parents received store coupons for purchasing commercial baby foods at their local store. One group received control baby foods, and the other group received baby foods containing DHA-enriched egg yolk.

Fifty-one infants completed the study: 26 in the group receiving the control baby food, with 1 drop-out (due to viral infection), and 25 in the group receiving the baby foods made with DHA-enriched egg yolk with 3 drop-outs (2 due to constipation and 1 refusal to eat solid foods at 6 mo of age), yielding a 93% completion rate.

**Baby foods.** DHA-enriched eggs were obtained from hens receiving a diet essentially free of LCPUFA but containing flaxseed and soybean meal as sources of the DHA precursor, α-linolenic acid (α-LNA) (19). The egg yolks were separated, pasteurized, and spray-dried. The dried egg yolks at 120 g/kg food (12%) were used to prepare semisolids, ready-to-feed DHA-enriched baby foods as described by Theuer et al. (20,21); all foods were packaged in hermetically sealed jars containing 113 g food. The control baby foods were devoid of egg yolk but otherwise contained the same ingredients as the DHA-enriched foods. The dried egg yolk and the baby foods were analyzed for fatty acids by Medallion Laboratories (22) (see Table 1). The dried egg yolk contained ~2% of fatty acids as DHA; the DHA-enriched baby foods contained ~115 mg DHA/100 g food (i.e., ~130 mg DHA/113-g baby food jar). The fat content of the different flavors of DHA-enriched baby foods (5.8–8.1 g/100 g) was 5- to 6-fold higher than that of control foods (0.1–2.3 g/100 g), resulting in a higher energy density of the DHA-enriched baby foods (greater by ~230 kJ/100 g).

Five varieties of study foods were shipped directly to each participant’s home; a minimum of 216 jars was provided for the 6-mo feeding period with a goal of providing 1 jar of study food/d. Vegetable, cereal with fruit, and fruit and custard varieties were included. Food intake was estimated for the first 2 mo of the trial by collecting and weighing the opened jars to determine the unused portion. Potential discrepancies between food intake and food disappearance (amount missing from each jar) were minimized by instructing the parent to spoon out only a small amount of food at a time and then to spoon out additional food as needed. The food disappearance data served to estimate compliance and the overall food and DHA consumption of study infants.

**General protocol.** Informed consent was obtained and randomization occurred at the 6-mo visit. The assigned foods were shipped within 3–7 d. Visual function (sweep VEP acuity and stereoacuity) and growth were assessed at 6, 9, and 12 mo, and blood samples were taken at 6 and 12 mo.

**Sweep VEP acuity.** VEP acuity was the primary outcome measure and was assessed according to the sweep parameter protocol developed by Norcia and colleagues (23,24) with the use of vertical gratings phase reversing at 6 Hz. Details of the protocol were described previously (4). Sweep VEP acuities were expressed in logMAR (minimum angle of resolution; e.g., the Snellen equivalents of 20/20 correspond to a MAR of 1 min arc and logMAR of 0.0 whereas 20/200 corresponds to an MAR of 10 min arc and logMAR of 1.0).

**Stereoacuity.** Random dot stereocuity was assessed with the use of forced-choice preferential looking and the Infant Randot Stereotests (25) as described previously (26). Random dot stereocuity was chosen as an outcome measure because it reflects cortical processing; detection of the disparate stimulus depends on the cortical combination of monocular images that lack any form information. Stere-
acuity was expressed in log arc s (log of the minimum detectable binocular disparity; e.g., a 40 arc s disparity corresponds to 1.60 log arc s).

**Growth.** Weight, length, head circumference, and triceps and subscapular skinfold thickness measures were described previously (4) and were obtained at 6, 9, and 12 mo. Growth data were normalized by expression as Z-scores derived for term infants of appropriate age and sex by comparison with published normative data by the Department of Health and Human Services as part of the National Health and Nutrition Examination Survey III (27).

Blood samples (2.0 mL) were collected at 6 and 12 mo by heel stick aided by infant heel warming packs into tubes containing EDTA. Plasma and RBC were separated by centrifugation at 3000 × g for 10 min at 4°C; lipids were extracted and transmethylated with boron trifluoride:methanol, and methyl esters were analyzed by capillary column GC with flame ionization detection [see (6) for details]. The fatty acid level was reported as mass concentration for baby foods and both the relative percentage of total fatty acids and mass concentrations [μmol/L plasma (data not presented) or packed RBC (basis of the addition of an internal standard (23,0)]

**Total antioxidant capacity.** Total antioxidant capacity of plasma was measured using an enhanced chemiluminescence modification of the total peroxyl radical trapping parameter [TRAP assay (28)]. The antioxidant capacity of an aliquot (20 μL) of citrate anticoagulated plasma diluted 1:10 with isotonic sodium chloride solution was established by its ability to quench a horseradish peroxidase-catalyzed reaction generated by a chemiluminescence kit (cat. # RPN 190; Ortho-Clinical Diagnostics). Quenching capacity was assayed on a luminometer (Turner Designs) with subsequent quantification by comparison to a standard curve (10⁻¹⁰ μmol/L) of the synthetic vitamin E derivative, Trolox (6-hydroxy-2,5,7,8-tetramethylchloro- man-2-carboxylic acid; Aldrich). Antioxidant activity was expressed as μmol/L equivalents of this vitamin E derivative.

**Blood hematology and chemistry.** At 6 and 12 mo, aliquots of whole EDTA-anticoagulated blood were sent to a central clinical laboratory (LabCorp) for hematological assay. The analysis included platelet count, red and white blood cell counts, hemoglobin and hematocrit determination, mean corpuscular volume, and hemoglobin quantitation (Coulter LH750 Hematology Analyzer; Beckman Coulter). At 12 mo, blood chemistry was analyzed in serum samples sent to the same laboratory. The analysis included the following: determination of glucose, blood urea nitrogen, creatinine, blood urea nitrogen:creatinine ratio, sodium, potassium, chloride, carbon dioxide, calcium, total protein, albumin, globulin, albumin:globulin ratio, total bilirubin, and activities of alkaline phosphatase, aspartate aminotransferase, and alanine aminotransferase (Modular Hitachi, Roche).

**Sample size.** Sample sizes were estimated using the method described by Rosner (29) for α = 0.05 and 1 − β = 0.90. With the use of standard deviations for sweep VEP [0.1 μgMAR corresponds to 1 line on an eye chart (4)] from our present and past studies of term infants, the final sample size per group at 12 mo required to detect a 1-SD difference between groups was 21 infants. This sample size was also sufficient to detect a 1-SD difference between groups in random dot stereoaucuity [0.2 log arc s; e.g., 40 arc s compared with 60 arc s; (25)] and a <1% difference in the DHA or ARA fatty acid composition of RBC lipids (3). Measurements of antioxidant capacity, hematological and blood chemistry variables were not obtained in our previous studies of infant nutrition; thus, the experimental data required to estimate potential dietary effects of these secondary analyses were not available. Anticipating a 10% loss to follow-up over 12 mo, we planned to recruit 25 infants for each diet group. In actuality, 55 infants were enrolled and 51 completing the study, for a 93% completion rate.

**Statistical analyses.** All data were handled in a coded manner. The data were analyzed with two-way repeated-measures ANOVA after verifying that they met normality criteria. Planned comparisons were carried out to compare the means of the 2 diet groups at each age point. Because 4 pairwise comparisons were conducted for each of the 5 vision outcome variables (acuity and stereoaucusity), only planned comparisons with P < 0.01 were considered significant (Bonferroni adjustment of 0.05/4 or 0.0125). Because linear regression was simi-

**RESULTS**

Despite the expressed intention to continue breast-feeding their infants, mothers in the control and DHA-enriched groups breast-fed for 9.7 mo and 8.8 mo, respectively (see Table 2); 65% of the infants in the control group and 80% of infants in the supplemented group were weaned from human milk to formula before 12 mo of age. The study was largely completed before the commercial availability of infant formula fortified with LCPUFA in the United States. Only 1 infant in the control group and 3 in the DHA-enriched group were weaned from human milk to infant formula containing DHA and ARA during the 6-mo trial interval. Removal of these infants from data analysis did not affect the results of the statistical analyses.

Dietary sources of DHA for infants in the study included both human milk and enriched baby foods. The estimated intakes of DHA from human milk during the trial were 37 mg/d in control infants and 28 mg/d in the supplemented group. Consumption of baby food by the control group was 84 ± 23 g food/d (−0.75 jar/d; Table 2). Based on gravimetric measures, control infants consumed 0.3 mg supplemental DHA and ARA/d, respectively, from baby food during the 6-mo study. Infants in the DHA-enriched group consumed 72 ± 31 g baby food/d (about 0.66 jar/d); this was different from controls (P = 0.12). Infants fed DHA-enriched baby foods were estimated to have consumed 83 mg supplementary DHA/d and 56 mg supplementary ARA/d during the 6-mo trial.

The fatty acid content of RBC lipids did not differ between groups at the start of the trial (6 mo; Table 3); however, by 12 mo, the groups differed in RBC lipid DHA, docosapentaenoic acid [DPA; 22:5(n-6)], and total (n-3) LCPUFA (P < 0.002). RBC DHA levels decreased in the control group from 3.8% at 6 mo to 3.0% at 12 mo (P = 0.012). In contrast, RBC DHA levels increased (P < 0.002) in the DHA-enriched group from 4.1% at 6 mo to 5.5% at 12 mo. RBC DHA levels expressed...
as mass concentration showed similar changes (Table 3, P < 0.002).

The sufficiency indices for (n-3) fatty acids ([(n-6)/(n-3)] LCPUFA ratio), DHA [DHA/DPA(n-6)], and essential fatty acids (Mead acid [20:3(n-9)]/ARA) improved in the DHA-supplemented group. The unsaturation index was significantly elevated in RBC of supplemented infants; this summation of double bonds is reflective of an increase in the fluidity of RBC membranes. Trenn et al. (30) reported that an increase in the unsaturation index of 10 units (based on the percentage of total fatty acids) in cultured retinoblastoma cells increased the lateral mobility in the membrane bilayer (i.e., an ~30% increase in pyrene eximer formation) and increased transport of choline across the cell membrane by 12%.

In control infants, VEP acuity at 6 mo was 0.49 ± 0.13 logMAR; it improved to 0.45 ± 0.14 logMAR at 9 mo and to 0.29 ± 0.11 logMAR at 12 mo (Fig. 1). In the DHA-supplemented group, VEP acuity was 0.48 ± 0.10 logMAR at 6 mo and improved to 0.31 ± 0.13 log MAR at 9 mo and to 0.13 ± 0.1 logMAR at 12 mo. Compared with controls, infants in the DHA-supplemented group had improved visual acuity by 0.14 and 0.16 logMAR at 9 and 12 mo, respectively, (P < 0.002), equivalent to ~1.5 lines on an eye chart.

VEP acuity at 12 mo was correlated with RBC DHA levels at 12 mo (r = −0.50; P = 0.0002) (Fig. 2) such that infants with high DHA levels had lower logMAR values, i.e., more mature acuity. In addition, the estimated dietary intake of DHA from human milk and baby food on an individual basis was correlated with VEP acuity (r = −0.49, P < 0.0002) as well as with RBC DHA levels (r = 0.57, P < 0.0002).

Stereocuity at 6 mo was 2.54 ± 0.54 log arc s and improved to 2.25 ± 0.47 log arc s at 12 mo in the control infants. In the supplemented infants, stereocuity was 2.37 ± 0.34 log arc s at 6 mo and improved to 2.22 ± 0.35 log arc s at 12 mo. However, stereocuity at 12 mo did not differ between the 2 diet groups (P = 0.8).

Despite differences in energy and fat content of the study baby foods, the groups did not differ in weight, length, head circumference, or skin-fold thicknesses at 6, 9, and 12 mo (P > 0.3 for all measures; data not shown).

Total plasma antioxidant capacity did not differ between the 2 diet groups at the onset of the trial (345 ± 78 μmol/L for controls and 328 ± 107 μmol/L Trolox equivalents for the supplemented group; P = 0.53) or at 12 mo (335 ± 63 vs. 321 ± 109 μmol/L Trolox equivalents; P = 0.58, respectively).

Protocol compliance was excellent and the DHA-enriched foods were well tolerated. There were 3 adverse events recorded for controls: 2 were not diet-related (neuroblastoma and occluded tear duct, both requiring surgery), and 1 infant had a 3-fold elevation in aspartate aminotransferase at 12 mo.
In the supplemented group, there were 3 adverse events: 2 were not diet-related (genetically associated elevation in alkaline phosphatase and eczema since birth) and 1 infant had a 5-fold elevation in alkaline phosphatase at 12 mo. All events were reported to the Institutional Review Boards and to patients’ pediatricians. The groups did not differ in hematological measures at either 6 or 12 mo of age (P > 0.1). Similarly, their blood chemistries did not differ at 12 mo (P > 0.15). Upon termination of the study, neither group had mean hematological results that were outside of the normal range, although both groups had levels of creatinine (for control and supplemented groups, 3.2 and 3.0 mg/dL, respectively) and carbon dioxide (18.2 and 18.5 mEq/L) that were marginally below normal and albumin levels (43.6 and 42.8 g/L) that were slightly higher than normal; none were of clinical importance.

**DISCUSSION**

In the current randomized clinical trial, breast-fed infants receiving LCPUFA-enriched baby foods during mo 6–12 of life had an 83% elevation in RBC DHA levels (Table 3) resulting from an approximately 2-fold greater intake of DHA compared with unsupplemented infants (Table 2). In addition, DHA-supplemented infants had more mature VEP acuity than control infants at 9 and 12 mo of age (by 0.14 and 0.16 logMAR, i.e., ~1.5 lines on an eye chart; Fig. 1). Furthermore, the blood lipid level of DHA was significantly correlated with VEP acuity such that infants with higher levels of RBC DHA had better visual acuity (Fig. 2). Metabolic measures were equivalent in both groups with no major diet-related adverse events.

No benefit to stereoscopic visual acuity attributable to DHA-enriched baby food was evident in the current trial; this is consistent with a previous trial using LCPUFA-enriched infant formula (31). In both of these studies, infants received human milk for the first 4–6 mo of life, which may have provided sufficient nutrition for optimal development of stereovision. However, in a separate trial in which infants were randomized to receive control formula or LCPUFA supplemented formula beginning at 6 wk of life, this environmental influence was evident at a 4-mo time point but not later (26). Thus, a “critical period of sensitivity” appears to occur up to 6 mo of age in the maturation of stereoscopic visual acuity to environmental influences (e.g., dietary factors).

The biochemical and functional results from this study are consistent with an earlier randomized clinical trial of breast-fed infants weaned between 4 and 6 mo of age to receive either DHA + ARA-enriched or nonenriched infant formula (31). At 12 mo, infants fed a nonenriched diet had a 50% reduction in RBC-DHA concentration compared with weaning levels. In contrast, infants fed the LCPUFA-enriched formula had a 24% higher RBC-DHA content compared with weaning levels and at 12 mo had a 1.5-fold higher DHA level than that in the nonenriched infant group. In this formula trial, we estimated that the supply of DHA was ~0.2–0.4 g DHA/6 mo in the control group (primarily due to endogenous DHA synthesis from α-LNAs) compared with a dietary intake of ~22 g DHA/6 mo in LCPUFA-supplemented infants. The 1-yr-old supplemented infants had improved VEP acuity by 0.103 log MAR (1 line on the eye chart) compared with the nonsupplemented group.

The average amount of human milk consumed each day between 6 and 9 mo of age decreases from ~750 mL to about 625 mL (17). Because the average fat content of human milk is ~37 g/L (17), the daily intake of human milk fat over this period would be ~25 g. With an average DHA content of human milk fat in the United States of ~0.2 g/100 g total milk fatty acids (27,32), the DHA intake of exclusively breast-fed older infants in the United States would be ~50 mg/d. Between 6 and 9 mo, the average dietary DHA intake of infants fed the baby foods made with DHA-enriched egg yolks was estimated to be 133 mg/d from both human milk (50 mg/d) and solid food sources (83 mg/d), whereas between 9 and 12 mo, the majority of infants were weaned and solid foods alone contributed DHA at an average of 83 mg/d. Thus, for the entire 6-mo trial period, the supplemented infants received an average of 108 mg DHA/d compared with 38 mg DHA/d in control infants who received only human milk until 9.7 mo of age. This corresponded to an approximate 2-fold increase in DHA intake by the supplemented group (7 vs. 20 g/d; Table 2).

Body weight over the 6- to 12-mo period averaged 8.4 kg; thus, the mean intake of DHA for the DHA-supplemented infants was 13 mg/(kg·d). However, intake for these infants from 6 to 9 mo while still breast-feeding was 17 mg/(kg·d) but dropped to ~9 mg DHA/(kg·d) from 9 to 12 mo when the only source of DHA was from enriched baby foods. These amounts are ~15 and 55% lower than the 20 mg DHA/(kg·d)
recommended by the FAO/WHO Joint Expert Consultation (33). By comparison, the mean DHA intake for the control group during the 6-mo study was only 4.5 mg/(kg·d). Human milk and supplemented infant formula are among the few foods available to infants in the United States that contain a nutritionally relevant amount of DHA + ARA. Infant formula is the logical choice as a vehicle for providing DHA and ARA to younger infants who are not breast-fed. During weaning to solid foods, the North American infant receives very little DHA from the diversified mixture of ordinary foods customarily included in the weaning diet. This assumption was validated in Australia (18) and in Finland (34).

Only 3 foods common in the U.S. diet contain significant amounts of DHA, i.e., egg yolks, chicken, and oily fish. Both regular egg yolks and those from chickens fed special diets to increase the (n-3) fatty acid content contain measurable quantities of DHA. A large egg yolk contains 25 and 140 mg DHA and lecithin, respectively, even though oily fish such as salmon and tuna have been recognized as a safe food for babies and were used in various ancient cultures as a first solid food (35). Egg yolk was recommended ≥40 y ago to be started between 4 and 6 mo of age unless there was allergy in the family (36). More recent suggested guidelines for infants during the first 6 mo of life include the introduction of egg yolk at 5–6 mo (37). Most recently, Gibson et al. (38) and Makrides et al. (39) reported the effects of feeding normal and DHA-enriched egg yolks to formula-fed and breast-fed infants in the second 6 mo of life. Consuming 4 DHA-enriched egg yolks weekly significantly increased RBC DHA levels at 12 mo in breast-fed infants. Blood cholesterol levels were no higher than those of breast-fed infants. Gibson et al. (38) also found that infants fed egg yolk had improved iron status, as measured by higher serum iron levels and higher transferrin saturation. Based on estimates of food intake (Table 1) and the content of yolk in baby foods (12%), the consumption of yolk (~67 g/wk), and thus, cholesterol and iron, in the current study was nearly equivalent to that in the Gibson study (38,39). Egg yolk is also a rich source of choline-rich lecithin; choline is a vital constituent of membrane phospholipids and was shown to be an essential nutrient for brain development (40).

Chicken meat contains only a small amount of DHA. Pureed chicken with broth intended for infant use contains ~7 mg of DHA (and 43 mg of ARA) in the 55-g Recommended Amount Customarily Consumed Per Eating Occasion (41). Although chicken is an important source of DHA for adults (42), the low concentration of DHA in chicken makes this a poor source of DHA for infants.

In the United States, no commercial foods intended for infants contain fish. Fish is perceived to be highly allergenic by U.S. parents through oil fish such as salmon and tuna have reduced allergenicity if canned commercially (43). A more difficult issue nutritionally is that oily fish contain substantial amounts of DHA and eicosapentaenoic acid (EPA) but very little ARA. Pureed baby foods available in Europe made with trout and nasello (hake: white) supply, per 100 g, 100 to 200 mg of DHA and 25 to 70 mg of EPA but only 3 to 6 mg of ARA. Human milk contains some EPA if the maternal diet contains an EPA source (e.g., fish); thus, normal infant growth and development can occur in the presence of small amounts of EPA. However, providing supplemental EPA to infants without sufficient ARA is problematic. EPA inhibits the elongation of LA to ARA (44). A DHA-enriched (0.31% DHA) infant formula made with a low-EPA fish oil and containing relatively little EPA (0.08% of total fatty acids) but even less ARA (0.03%) significantly depressed RBC phospholipid ARA levels at 4 mo of age (45). Thus, the level of EPA in the infant diet should be limited (46). Infant formulas containing fish oils with a substantial EPA content were shown not to support (46) and to support (47) normal growth in preterm infants.

This trial demonstrates that the visual maturation of healthy infants is improved by continued supplies of DHA from both human milk and DHA-enriched baby foods well into 1 y of life. Modifications later in childhood to visual function and other neural processes by this DHA supplementation in baby foods are currently under investigation.

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