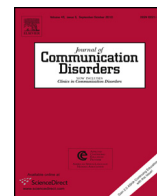




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Breastfeeding may protect against persistent stuttering

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ABSTRACT

Purpose: This study investigated the hypothesis that breastfeeding in infancy might protect against persistent stuttering in children.**Method:** We collected new data from the mothers of current and past participants in the Illinois Stuttering Research Program on their children's feeding history during infancy. We obtained 47 usable responses, for 17 children with persistent stuttering and 30 children who recovered naturally after a period of stuttering.**Results:** A chi-squared test for linear trend revealed a significant relationship between breastfeeding duration and the likelihood of natural recovery for the boys in the sample. Mothers of children in the persistent group were no more likely to report early feeding difficulties which might have suggested an underlying oral motor deficit in children predisposed toward persistent stuttering.**Conclusions:** Our results offer preliminary support for the idea that breastfeeding may confer a measure of protection against persistent stuttering. The fatty acid profile of human milk, with its potential to affect both gene expression and the composition of neural tissue, may explain this association. Further research is called for.**Learning outcomes:** The reader will be able to discuss at least one reason why human milk may make a difference in neurodevelopment generally and with regard to stuttering outcomes specifically. Additionally, the reader will be able to describe the relationship between breastfeeding duration and stuttering recovery observed in this sample.

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1. Introduction

Approximately 3–5% of preschool-aged children will begin stuttering. In some cases the onset of stuttering is dramatic: a previously fluent child will wake up one morning unable to utter a fluent sentence. An accompanying puzzle is the phenomenon of natural recovery: that same child may stutter severely for weeks or months and then gradually return, without intervention, to normally fluent speech.

Preschool-aged children who begin to stutter will recover naturally approximately three-quarters of the time (Yairi & Ambrose, 1999), but predictor variables and causal factors are poorly understood. This study was driven by the hypothesis that breastfeeding could confer a measure of dose-related protection against persistent stuttering. The following sections will describe the rationale for this hypothesis, summarize the existing literature on breastfeeding and speech-language development, and briefly review the etiology of stuttering.

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1.1. Human milk and neurodevelopment

Fluent speech, with its 140,000 neuromuscular events per second (Darley, Aronson, & Brown, 1975), depends on normal neurodevelopment, and neurodevelopment can be influenced by diet in early infancy. It is widely recognized that the neurological system changes rapidly during the first two years of life; the question less often considered is what, precisely, is required to build a brain. A newborn's brain weighs, on average, 350 g; a year later it will weigh 1100 g (Lawrence & Lawrence, 2005). More than half the solid weight of that newly built tissue will be lipid, and dietary fat intake exerts a significant influence on its composition (Farquharson, Jamieson, Logan, Cockburn, & Ainslie Patrick, 1992). The rationale for this study is that the differing fatty acid profiles of human milk and infant formula have the potential to affect children predisposed to stuttering via two different mechanisms: first, by subtly altering the composition and thus the function of the brain itself, and second, by influencing gene expression (Jump, 2004; Oddy, 2006).

Human milk contains two fatty acids that have been identified as particularly important for early neurodevelopment: the omega-3 (subsequently abbreviated as n-3) fatty acid docosahexaenoic acid (DHA) and the omega-6 (n-6) fatty acid arachidonic acid (AA). DHA is the fatty acid most prevalent in the mammalian brain; DHA levels within the brain are determined by dietary levels (Innis, 2007). DHA and AA are present in both gray and white matter, including myelin (Nettleton, 1995). Across most of the lifespan the body can synthesize DHA and AA, as well as other long-chain fatty acids, from shorter-chain fatty acids. In early infancy, however, this synthesis process is not adequate for optimal brain development; research shows that the rate at which DHA is incorporated into brain tissue outstrips the rate at which it can be synthesized. In infants who lack a dietary source of DHA, other fatty acids are incorporated into the brain to compensate for the dearth of DHA (Farquharson et al., 1992). Thus differences in early diet can have long-lasting effects. In her 2006 study of the long-term effects of breastfeeding among Australian children, Oddy (2006) states, "Dietary alterations of n-3 and n-6 [fatty acids] can trigger dramatic alterations in brain lipid composition associated with changes in physical properties of membranes, alterations in enzyme activities, receptors, carrier mediated transport and cellular interactions" (p. 181). Differences in the fatty acid composition of brain tissue could contribute to differences in cell-to-cell communication, alterations in the function of synaptic membranes, and subtle impairments in nerve conductance and neurotransmission (Lauritzen, Hansen, Jørgensen, & Michaelsen, 2001; Oddy, 2006).

In contrast to human milk, most varieties of infant formula do not contain DHA/AA. Supplementation of infant formula with DHA/AA began in 2001 in the US, but the increased cost of EFA-fortified formula means that many children continue to receive the unsupplemented version. Furthermore, the impact of formula supplementation on neurodevelopment is controversial. Studies of long-term neurodevelopmental effects have reported no clear benefit to supplemented formula, suggesting that modified fatty acid profiles are not the sole determinant of neurodevelopmental outcomes (de Jong, Kikkert, Fidler, & Hadders-Algra, 2010; Smithers et al., 2010; see also a 2003 meta-analysis by Koo). Indeed, it would be reductive to argue that fatty acids are exclusively responsible for the differences observed in populations of breastfed and formula-fed children. Readers are referred to Riordan (2005) for further information on the properties of human milk that may influence neurodevelopment, and are encouraged to keep in mind that these observed differences may well be a synergistic effect of multiple nutrients, or may be related to as-yet-identified human milk constituents.

Though the mechanism is incompletely understood, research does show that diet affects the composition of neural tissue. Farquharson et al. (1992) obtained necropsy gray matter samples from infants who died of SIDS, comparing exclusively breastfed babies with exclusively formula-fed babies. All the formula-fed babies had significantly less DHA in their cerebrocortical tissue than their breastfed counterparts; those fed formula were observed to have a corresponding increase in n-6 series fatty acids.

Because of the ethical concerns raised by manipulation of the nutrient content of an infant's diet, animal studies can provide some additional information on this topic. Rodent studies are particularly useful for this purpose because of similarities between rodent pups' prenatal/postnatal brain growth patterns and those of human infants. Lim, Hoshiba, and Salem (2005) compared adult rats given varying types of milk during infancy. In findings that paralleled those of Farquharson and colleagues in their study of human infants (1992), they reported that early diets high in n-6 fatty acids were clearly associated with brain tissue high in n-6 fatty acids. In the rat pups, this difference in neural tissue composition was associated with significant performance deficits, a finding that raises questions about the potential long-term ramifications of early diets high in n-6 fatty acids in human populations.

Neurotransmission can be affected by fatty acid profiles; in addition, the fatty acid composition of a cell membrane can have significant effects on the expression of genes within that cell. Oddy reports: "n-6 and n-3 fatty acids directly govern the transcription rate of specific genes. . . . This means that the n-6 and n-3 in our cell membranes exert a significant influence on the way a given genetic profile is expressed" (p. 180). For researchers studying genetically mediated phenomena, including certain forms of speech-language impairment, this is an observation of critical import.

Research into the impact of human milk on speech-language development has examined children from infancy onward. A 1999 study by Vestergaard et al. points to a connection between breastfeeding and early pre-speech development. In this prospective cohort study, babies who were exclusively breastfed for a longer period produced variegated babbling at earlier ages. The effect persisted after control for multiple potential confounding variables, including social class, maternal education, prenatal smoking, birthweight, gestational age, and number of prior illnesses.

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