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NONNUTRITIVE SWEETENERS IN BREAST MILK

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Abstract

Nonnutritive sweeteners (NNS), including saccharin, sucralose, aspartame, and acesulfame-potassium, are commonly consumed in the general population, and all except for saccharin are considered safe for use during pregnancy and lactation. Sucralose (Splenda) currently holds the majority of the NNS market share and is often combined with acesulfame-potassium in a wide variety of foods and beverages. To date, saccharin is the only NNS reported to be found in human breast milk after maternal consumption, while there is no apparent information on the other NNS. Breast milk samples were collected from 20 lactating volunteers, irrespective of their habitual NNS intake. Saccharin, sucralose, and acesulfame-potassium were present in 65% of participants' milk samples, whereas aspartame was not detected. These data indicate that NNS are frequently ingested by nursing infants, and thus prospective clinical studies are necessary to determine whether early NNS exposure via breast milk may have clinical implications.

The numerous benefits of breastfeeding are well established (LaKind and Berlin, 2002; Wang and Needham, 2007). In fact, the American Academy of Pediatrics recommends exclusive breastfeeding for 6 mo, followed by continued breastfeeding while complementary foods are introduced, with continuation of breastfeeding for 1 yr or longer (American Academy of Pediatrics, 2012). However, lactating women are frequently concerned about the influence of foods, beverages, and medications on their babies' health (Groer et al., 2002). This also applies to the use of nonnutritive sweeteners (NNS). Typical recommendations are vague and range from "their consumption is generally believed to be

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safe while breastfeeding" (Nice et al., 2000) to "sweeteners should be avoided as long as possible" (La Leche League International, 2008).

LactMed, a database supported by the National Library of Medicine, is an excellent source of information regarding drugs and chemicals that may have possible adverse effects for nursing infants. Because human data for rebaudioside A and acesulfame-potassium are not available, LactMed states that the risk to the breastfed infant appears to be low, but an alternate artificial sweetener with more data available may be preferred. Aspartame is described as not detectable in breast milk, yet mothers are encouraged to be prudent in avoiding aspartame when nursing an infant with phenylketonuria due to its metabolism into aspartic acid and phenylalanine. Sucralose is reportedly "poorly absorbed after oral ingestion" (Schiffman and Rother, 2013) and thus "not likely to reach the bloodstream of the infant or cause any adverse effects in breastfed infants." Only saccharin levels after ingestion of a saccharin-containing beverage were previously measured in human breast milk (Egan et al., 1984). Since NNS consumption is common and may be used for maternal postpartum weight loss, and because data on NNS in breast milk are not available or sparse, the aim of this study was to determine the presence and concentrations of sucralose, acesulfame-potassium, and saccharin and confirm the absence of aspartame in human breast milk.

METHODS

Twenty lactating women were recruited irrespective of NNS use. Volunteers anonymously donated a sample of their breast milk and completed a brief questionnaire to determine which, if any, NNS-containing items were consumed within 24 h prior to sample collection. Information regarding infant age their infant's age, exclusive breastfeeding, time of sample collection, and time of previous breastfeeding/pumping session was collected. No remuneration was provided.

Samples and questionnaires were handled by an independent coordinator not involved in this project. The study was approved by the National Institutes of Health Office of Human Subject Recruitment and Protection and informed consent was deemed not necessary. Saccharin, sucralose, acesulfame-potassium, and aspartame were measured using liquid chromatography–mass spectrometry. Analyses were performed with an Acquity I-Class UPLC (Waters Corp., Milford, MA) and an Acquity UPLC BEH C-18 column (2.1 mm × 50 mm, 1.7 μm) coupled with a Q-Exactive MS (Thermo Scientific, Waltham, MA) with an HESI-II electrospray source.

RESULTS

Infants were 6.8 ± 4.2 mo old and 26% were exclusively breastfed. The majority of maternal NNS intake occurred via sweetener packets or diet beverages. All but one participant reported 0–2 cans of diet soda per day (ID 12 reported 7 cans per day). Saccharin, sucralose, and acesulfame-potassium were present in breast milk of 13 women, while aspartame was not detected (Table 1). Acesulfame-potassium was also found in breast milk from participants who reported no NNS consumption ($n = 4$).

DISCUSSION

Saccharin, sucralose, and acesulfame-potassium were present in breast milk of 65% of participants, most of whom reported NNS intake during the day prior to donating a breast milk sample. Interestingly, NNS were also detected in samples of women who did not report NNS consumption, likely due to poor recognition of NNS-containing products (Sylvetsky et al., 2014). The absence of measurable aspartame was not surprising as aspartame is rapidly metabolized into aspartic acid and phenylalanine following ingestion. Changes in aspartate and phenylalanine concentrations following aspartame exposure were previously documented, yet not considered to be clinically relevant (Stegink et al., 1979). However, this assessment is controversial, since animal studies suggested adverse consequences on metabolic programing through exposure to aspartame during pregnancy (Araujo et al., 2014).

Other potential effects of early NNS exposure are the influence on the gut microbiome (Suez et al., 2014; Abou-Donia et al., 2008; Schiffman and Rother, 2013) and on future food choices and dietary patterns, since taste preferences are shaped early in life (Schwartz et al., 2013). Rodent studies demonstrated that animals exhibited heightened preferences for both caloric sweeteners (sucrose) and NNS (acesulfame-potassium) in adulthood, when they were exposed to acesulfame-potassium either in utero or through breastfeeding (Zhang et al., 2011). Other investigators reported that NNS exposure during lactation may promote development of metabolic abnormalities (von Poser Toigo et al., 2015) and obesity (Araujo et al., 2014), yet similar studies do not exist in humans.

Despite the limitations of our study including small sample size, imprecise information on timing of NNS ingestion and breast milk sampling, self-reporting of NNS consumption, and lack of rebaudioside A data, our results indicate that (1) breastfed infants are frequently exposed to NNS, and (2) avoiding NNS is challenging due to their omnipresence in the food supply and hygiene/cosmetic products. Whether NNS at concentrations found in breast milk exert biological effects requires further study. However, sucralose at concentrations several orders of magnitude lower than those in our samples was shown to induce oxidative stress and feeding and behavioral abnormalities in a model species of ecotoxicology (Eriksson Wiklund et al., 2014).

In contrast to certain medications contraindicated for breastfeeding, but essential to maternal health and well-being, NNS use during lactation is a choice. Our findings suggest that women may wish to limit NNS consumption while breastfeeding, given the lack of information regarding short- and long-term consequences of NNS exposure during infancy. Our findings support the need for further pharmacokinetic and clinical studies and raise important research questions, including whether NNS affect infant gut microbiome, alter their sweet taste preference, and contribute to future metabolic abnormalities and obesity.

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TABLE 1

Concentrations of NNS in Human Breast Milk Based on Reported NNS Consumption

	Acesulfame-K		Saccharin		Sucralose		Aspartame	
ID	**Reported**	**μg/ml**	**Reported**	**μg/ml**	**Reported**	**μg/ml**	**Reported**	**μg/ml**
Participants reporting no NNS intake								
2	N	0	N	< LOQ	N	0	N	< LOQ
4	N	0	N	0	N	0	N	< LOQ
13	N	0.02	N	< LOQ	N	0	N	< LOQ
15	N	0.04	N	< LOQ	N	< LOQ	N	< LOQ
16	N	0.07	N	< LOQ	N	< LOQ	N	< LOQ
19	N	0.09	N	< LOQ	N	< LOQ	N	< LOQ
Participants reporting intake of ≥ 1 NNS								
1	Y	2.22	N	0.02	Y	0.01	Y	< LOQ
3	Y	1.22	N	< LOQ	N	0	Y	< LOQ
5	Y	1.83	N	0	Y	0	N	< LOQ
6	Y	0.13	N	< LOQ	Y	0.01	N	< LOQ
7	Y	0.03	N	0	N	0	Y	< LOQ
8	N	0.21	N	0.01	Y	0	N	< LOQ
9	Y	1.00	N	0.01	Y	0.04	N	< LOQ
10	N	0	N	< LOQ	Y	< LOQ	N	0
11	Y	0	N	0	N	0	Y	0
12	N	0.01	Y	1.42	N	0	Y	0
14	N	0	N	0	Y	< LOQ	N	0
17	N	< LOQ	N	< LOQ	Y	< LOQ	N	< LOQ
18	Y	< LOQ	N	< LOQ	N	< LOQ	Y	NF
20	Y	1.047	N	< LOQ	N	< LOQ	Y	< LOQ

Note. < LOQ, below limit of quantitation. N, no consumption reported; Y, reported consumption.

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