Feeding Method, Nicotine Exposure, and Growth during Infancy

Edmond D. Shenassa, ScD, MA, FACE[1,2,3], Edoardo Botteri, PhD[4,5], and Hanne Stensheim, MD[4]

Objective To answer 3 questions: (1) Are infants breastfed by smokers at risk of rapid weight and length gain? (2) Is rapid growth during infancy partially attributable to ingestion of smokers' breastmilk? (3) If so, what are the implications for breastfeeding by smokers?

Study design Using data from the Norwegian Mother, Father and Child Cohort Study and Medical Birth Registry of Norway (n = 54 522), we examined changes in weight, length, weight-for-length z-score (WFLZ) during infancy in the context of maternal smoking (0, 1-10, or >10 cigarettes/day) and feeding method during the first 6 months (breastfed, formula fed, mixed fed). We fit generalized linear models, adding a smoking by feeding method interaction term to evaluate the effect of ingesting smokers' breastmilk.

Results Breastfed infants of both light and heavy smokers experienced WFLZ gains of 0.05 (95% CI, 0.01-0.09) and 0.13 (95% CI, 0.07-0.18), respectively. Among mixed-fed infants, only heavy maternal smoking predicted WFLZ gain (0.10; 95% CI, 0.05-0.16). Among exclusively formula-fed infants, maternal smoking did not predict rapid growth. Interaction models suggest that infants ever breastfed (ie, breastfed and mixed-fed groups combined) by heavy smokers gained weight (100 g; 95% CI, 30-231) and length (2.8 mm; 95% CI, 0.1-5.6), attributable to ingesting smoker's breastmilk.

Conclusions Infants breastfed by smokers experience rapid growth; some of these gains are attributable to ingesting smokers' breastmilk. Among infants breasted by light smokers, these gains are within the range of normative growth patterns for healthy, breastfed infants. *(J Pediatr 2024;14:200127)*.

Evidence on whether breastmilk can potentially promote optimal growth among infants exposed to nicotine can inform clinical guidelines for breastfeeding by smokers.[1-4] Postnatal exposure to nicotine and its metabolites is associated with rapid weight and length gain during infancy,[5,6] with potential long-term sequalae throughout the lifespan, including an increased risk of overweight[7-10] and cardiovascular disease and its related morbidities, as indicated by studies of developmental origins of health and disease.[11-14] Given that breastfeeding during infancy promotes optimal growth, it is reasonable to ask whether benefit of breastfeeding extends to infants breastfed by smokers.[15-18] Answering this question, however, is complicated by possible obesogenic properties of smokers' breastmilk. Nicotine and its metabolites are readily available in smokers' breastmilk[6,19] and smoking alters breastmilk's protein[20,21] and lipid[21,22] concentrations. These properties of smokers' breastmilk may be obesogenic. Thus, 3 interrelated questions emerge: (1) Are infants breastfed by smokers at risk of rapid weight and length gain? (2) If so, is rapid growth among infants breastfed by smokers attributable to ingestion of smokers' breastmilk? (3) If so, what are the implications for breastfeeding by smokers?

Approximately 2 decades ago, the American Academy of Pediatrics, despite strongly discouraging smoking,[4] removed smoking as a contraindication for breastfeeding mothers.[23] During this time, only 3 epidemiological studies[24-26] have examined the synergy between feeding methods, nicotine exposure, and growth during infancy. We address this shortcoming by analyzing data from the Norwegian Mother, Father and Child Cohort Study (MoBa) to examine the synergy between feeding method, nicotine exposure, and growth during infancy. We contribute to the literature by considering the feeding method, nicotine exposure, and growth in greater detail than before, along with a more extensive set of confounding variables than has been possible to date. The secondary aim of the study is to determine possible existence of sex differences in any observed effects.

MoBa	The Norwegian Mother, Father and Child Cohort Study
WFLZ	Weight-for-length *z*-score
WHO	World Health Organization

From the [1]Department of Epidemiology and Biostatistics, Maternal & Child Health Program, FMSC, University of Maryland, College Park, MD; [2]Department of Epidemiology and Biostatistics, School of Public Health, Brown University, Providence, RI; [3]Department of Epidemiology and Biostatistics, School of Medicine, University of Maryland, Baltimore, MD; [4]Department of Research, Cancer Registry of Norway, Oslo, Norway; and the [5]Section for Colorectal Cancer Screening, Cancer Registry of Norway, Oslo, Norway

Funding for this study was provided to Shenassa by the University of Maryland.

Ethics Approval: The establishment of MoBa and initial data collection was based on a license from the Norwegian Data Protection Agency and approval from The Regional Committees for Medical and Health Research Ethics. The MoBa cohort is now based on regulations related to the Norwegian Health Registry Act. The current study was approved by The Regional Committees for Medical and Health Research Ethics 2018/425. The work for this manuscript was considered exempt by the Institutional Review Board at the University of Maryland.

Data Availability: Data used for this study are available upon request from thye Norwegian Institute of Public Health (https://www.fhi.no/en/studies/moba/) contingent on approval from the Institute and payment of a fee.

https://doi.org/10.1016/j.jpedcp.2024.200127

Methods

MoBa is a population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health.[27] Between 1999 and 2008, all pregnant women in Norway in 17th and 18th weeks of pregnancy were mailed an invitation to participate in the study. Forty-one percent of pregnant women from throughout Norway consented and were enrolled in MoBa. Some women participated in the MoBa during multiple pregnancies. Participants received questionnaires during pregnancy and until the child was 8 years of age. Currently, MoBa includes 103 434 births where the mother answered a questionnaire. The present study is based on version 12 of quality-assured data files released in January 2019. We used data from five questionnaires administered approximately during 17th, 22nd, and 30th weeks of pregnancy and at 6 and 18 months of age. All questionnaires are available on the website of the Norwegian Institute of Public Health (https://www.fhi.no/en/studies/moba/).

Data from MoBa and the national Medical Birth Registry of Norway were linked via unique identification numbers given to all residents of Norway. For this study, the Medical Birth Registry of Norway provided data on reproductive history, gestational age (based on last menstrual cycle and, since 1999, ultrasound estimation), birth outcomes, and anthropometric measurements.

Infants were excluded from the analytic sample owing to withdrawal of consent (n = 226), multiple pregnancies (n = 1834), miscarriages or stillbirth (n = 934), missing or unlikely anthropometric measures (n = 3608), length at birth <45 cm (n = 1379, World Health Organization (WHO) criteria for being born at term), missing data on feeding method at 6 months (n = 20 114), anthropometric measurements at age 12 months (n = 18 983) or smoking habits after birth (n = 1819), yielding an analytic sample of 54 522 (**Figure**).

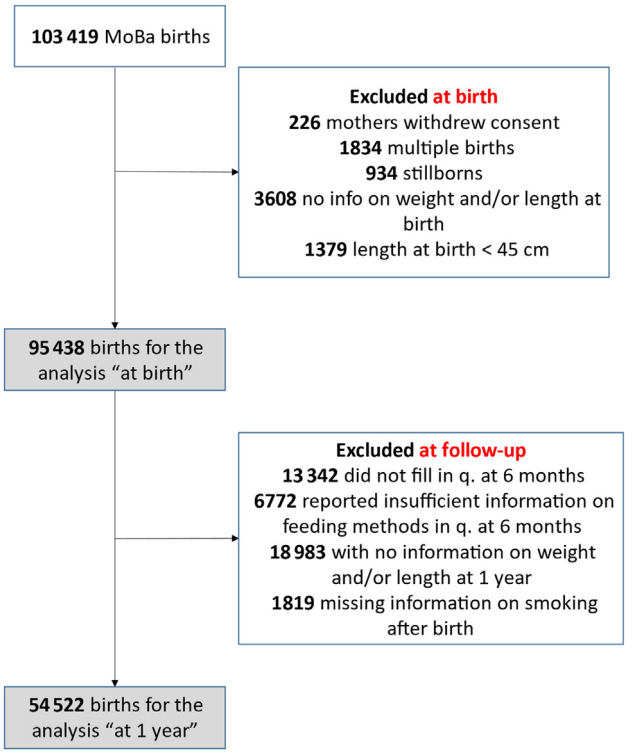

Figure. Flow diagram of the MoBa analytic sample.

Study Variables

We parameterized feeding methods during the first 6 months of infancy with 3 indicator variables: Exclusive breastfeeding, mixed feeding (breastfeeding and formula), and exclusive formula-feeding based on monthly reports during the first 6 months of infancy. Infants who were breastfed only one out of the first 6 months were assigned to the formula group, and likewise, infants who were formula-fed only one out of 6 months were assigned to the breastfeeding group. Infants who were either breastfed or formula-fed ≥2 months were assigned to the mixed-feeding group. To maximize the number of breastfed infants we also created an *ever-breastfed* category by combining exclusively breastfed and mixed-fed categories into one group. When (a) data for feeding method was missing for only one of the 6 months and (b) the same feeding method was reported for the months immediately preceding and following the month for which data was missing, we imputed a missing value with the value from the adjacent months. Time of introduction of solid food was parameterized with 3 indicator variables: <4, 4-5 or ≥6 months.

Postpartum smoking is based on reported 'current' number of cigarettes smoked daily or weekly and was parameterized with 3 indicator variables: 0, 1-10 (light smoking), >10 (heavy smoking) cigarettes/day. Respondents who reported to have 'never smoked', had a value of zero imputed if they had missing values for their current number of cigarettes smoked. Reported number of cigarettes smoked *during* pregnancy was parameterized with 3 indicator variables according to the same procedure used to code postpartum smoking. The cut-offs for the 2 smoking categories are based on empirical distribution of self-reported postnatal smoking; designations of 'light' and 'heavy' smoking refer only to the relative amount of postnatal smoking among this population. Any intake of other tobacco products (eg, snuff or patches) was parametrized with a dichotomous (yes/no) variable. Anthropometric measures (ie, weight and length) at 0, 8 and 12 months were obtained from Medical Birth Registry of Norway, we estimated weight-for-length *z*-scores (WFLZ) using WHO standards.[28] WHO's standards are based on a longitudinal (1997–2003) population-based study of healthy, singleton, breast-fed infants born to non-smokers in Brazil, Ghana, India, Norway, Oman and the United States (n = ~8500). As such, these standards are interpreted as normative growth patterns for healthy infants raised in healthy environments who were breastfed according to recommended guidelines. A Z-score of 0 indicates that the observed value is identical to normative standards.

All regression models included the following maternal covariates: age at delivery (continuous), marital status (married/cohabiting or single/divorced/widowed), maternal education (≤ high school, 1-4 or ≥ 5 years of higher education), place of birth (Norway/abroad), family income (continuous), parity (0, 1 or ≥ 2), gestational diabetes and/or hypertension (yes/no diagnosed?), maternal BMI at conception (continuous). Any intake of other tobacco products, average daily caffeine consumption during pregnancy based on self-reported Food Frequency Questionnaire completed approximately during 22nd week of gestation.[29] Regression models also include covariates for infants' sex, Apgar score at 5 minutes (≤7 vs > 7), gestational age at birth (weeks).

Statistical Methods

We tested the association between continuous variables and postpartum smoking with linear regression models. We used ANOVA to test the association between continuous variables and feeding method. To test the association between categorical variables and postpartum smoking or feeding method, we used Chi-square or Chi-square for trend, as appropriate. We used multivariable generalized linear models to identify independent correlates of 1-year *changes* in weight, length, and WFLZ. Assessing changes between birth and age 1 implicitly accounts for values of these anthropometric measures at birth.

To evaluate whether ingesting smokers' breastmilk can be potentially obesogenic we used the 'difference in differences' method, this is because high collinearity between the 3 sources of nicotine exposure (in utero, breastmilk, secondhand smoke) precludes use of separate covariates to estimate the effect attributable specifically to ingesting smokers' breastmilk. Difference in differences method allows estimation of growth attributable *specifically* to ingesting smokers' breastmilk by contrasting growth associated with any nicotine exposure among breastfed and among formula-fed infants. Breastfed infants can be exposed to nicotine in utero, by ingesting breastmilk and via inhaling secondhand smoke. Formula-fed infants can be exposed to nicotine only in utero and via secondhand smoke. The difference in growth between these 2 groups of infants (ie, difference in differences) is logically attributable to the only source of exposure that is *not* common between these 2 groups, ie, breastmilk. It can be shown that this difference in differences can be estimated simply by fitting an interaction term for smoking status and feeding method (Please see **Appendix 1**, online; available at www.jpeds.com).[30] Such an interaction term was included in all the regression models. Finally, for our secondary aim, we fit separate interaction terms among boys and girls to determine existence of sex-specific effects.

To account for multiple births for the same pregnancy, we used generalized estimating equations with mothers as the clustering variable in all models, and a compound-symmetry covariance structure. All analyses were performed using SAS version 9.4, SAS Institute, Cary, NC. All tests were 2-sided.

Results

Characteristics of 54 522 pregnancies of 47 956 mothers by maternal smoking status and feeding method during the first 6 months of infancy appear in **Table I**. The likelihood of smoking during pregnancy was lower among families with higher income and attained education. The mean gestational age was 39.7 weeks, 3% of the sample had a gestational age of <37 weeks. During the first sixth months of infancy, 66% of mothers breastfed exclusively and 12% smoked. Compared to infants born to non-smokers, infants born to light smokers (1-10 cigs/day) weighed 4 g more and were the same length, infants born to heavy smokers (>10 cig/day) weighed 99 g less and were 5 mm shorter. One-year changes in weight, length and WFLZ associated with smoking among each feeding group appear in **Table II**. Breastfed and mixed-fed infants but not formula-fed infants, experienced gains in weight and length during infancy that reflected the dose of maternal smoking (p for trend <.05).

In multivariable analysis (**Table III**, panel A), we estimated associations between feeding-type and maternal smoking with changes in weight, length and WFLZ during infancy. Compared to breastfed infants, mixed-fed infants experienced larger gains in weight (214 gr, 95% CI, 194, 234), length (3.9 mm, 95% CI, 3.4, 4.3), and WFLZ (0.15, 95% CI, 0.13, 0.17); formula-fed infants experienced still larger gains in weight (361 gr, 95% CI, 311, 412), length (6.4 mm, 95% CI, 5.3, 7.6), and WFLZ (0.26, 95% CI, 0.21, 0.31). Compared to infants of non-smokers, only infants of heavier smokers experienced statistically significant gains in weight (172 gr, 95% CI, 132, 211), length (1.7 mm, 95% CI,0.08, 2.6), and WFLZ (0.12 mm, 95% CI, 0.08, 0.15).

The joint effect of feeding methods and maternal smoking on 1-year change in weight, length and WFLZ for each feeding group appear in **Table III**, panel B. Both breastfed and mixed-fed infants experienced rapid weight and length gains in a manner reflecting the amount of maternal smoking. Compared to infants breastfed by non-smokers, infants breastfed by light and by heavy smokers gained more weight and length as reflected in WFLZ gains of 0.05 (95% CI, 0.01, 0.09), and 0.13 (95% CI, 0.07, 0.18) respectively. Among mixed-fed infants, only infants of heavy smokers gained more weight and length as reflected in WFLZ gain of 0.10 (95% CI, 0.05, 0.16). In contrast, among exclusively formula-fed infants, maternal smoking was not associated with growth.

Next, we considered evidence of interaction (ie, difference in differences[30]) between maternal smoking and feeding method to determine the extent to which these gains may be attributed to ingesting smokers' breastmilk (**Table III**, panel C). We found evidence of interaction only among infants who were *ever-breastfed* (ie, breastfed or mixed-fed), by a heavy smoker (>10 cigarettes/day). These interaction models suggest that during first year of life infants ever-breastfed by a heavy smoker, gain an average

Table I. Characteristics of 54 522 mother-infant pairs by maternal smoking status and feeding method during the first 6 months of infancy: MoBa

		Maternal smoking				Feeding method			
	Total sample n = 54 522	0 n = 47 793	1-10 cig/day n = 3427	>10 cig/day n = 3302	*P**	Exclusively Breastfed n = 36 008	Mixed-fed n = 16 686	Formula-fed n = 1828	*P**
Mother Characteristics									
Age at birth, parity = 0 (n = 25 532), mean (SD)	28.7 (4.3)	28.9 (4.2)	27.9 (4.3)	26.7 (5.1)	<.01	28.8 (4.1)	28.5 (4.5)	28.1 (5.0)	<.01
Age at birth, parity>0 (n = 28 990), mean (SD)	31.8 (4.0)	31.9 (4.0)	30.9 (4.0)	31.0 (4.6)	<.01	31.9 (3.9)	31.5 (4.2)	31.5 (4.3)	<.01
Marital status†									
Married‡ n (%)	52 539 (97.4)	46 324 (97.9)	3247 (96.0)	2968 (91.8)	<.01	34 895 (97.9)	15 929 (96.6)	1715 (95.2)	<.01
Education†									
Up to high school, n (%)	16 245 (31.4)	12 825 (28.3)	1305 (40.5)	2115 (68.5)	<.01	9332 (27.2)	5963 (38.0)	950 (55.2)	<.01
1-4 yrs university, n (%)	22 648 (43.8)	20 515 (45.2)	1355 (42.1)	778 (25.2)		15 474 (45.2)	6617 (42.2)	557 (32.3)	
≥5 yrs university, n (%)	12 786 (24.7)	12 031 (26.5)	559 (17.4)	196 (6.3)		9455 (27.6)	3116 (19.9)	215 (12.5)	
Cigarettes smoking during pregnancy†									
0 cig/day, n (%)	50 191 (92.8)	46 685 (98.5)	2 713 (80.2)	793 (24.1)	<.01	33 865 (94.9)	14 832 (89.4)	1494 (82.5)	<.01
1-10 cig/day, n (%)	1902 (3.5)	422 (0.9)	469 (13.9)	1011 (30.7)		971 (2.7)	797 (4.8)	134 (7.4)	
>10 cig/day, n (%)	1987 (3.7)	302 (0.6)	200 (5.9)	1485 (45.2)		837 (2.3)	966 (5.8)	184 (10.2)	
Infant Characteristics									
Weight at birth (gr), mean (SD)	3641 (501)	3647 (499)	3651 (508)	3548 (512)	<.01	3653 (490)	3617 (519)	3629 (533)	<.01
Length at birth (mm), mean (SD)	505 (20)	506 (20)	506 (21)	501 (21)	<.01	506 (20)	505 (21)	504 (21)	<.01
WFL z-score at birth, mean (SD)	0.45 (1.00)	0.45 (1.00)	0.47 (1.00)	0.45 (1.01)	.84	0.47 (0.99)	0.42 (1.03)	0.50 (1.04)	<.01
Introduction of solid food (month)	4.7 (0.9)	4.7 (0.9)	4.4 (0.9)	4.1 (0.9)	<.01	4.9 (0.9)	4.2 (0.8)	3.9 (0.7)	<.01
Gestational age (weeks)†, mean (SD)	39.7 (1.5)	39.7 (1.5)	39.7 (1.5)	39.6 (1.6)	.02	39.7 (1.4)	39.6 (1.5)	39.5 (1.6)	<.01
<37, n (%)	1665 (3.1)	1433 (3.0)	101 (3.0)	131 (4.0)	<.01	944 (2.6)	633 (3.8)	88 (4.8)	

§Exceeding the Norwegian recommendations.
**P* for trend from linear regression models for continuous factors and Chi-square test for trend for categorical factors.
†Numbers do not sum up to the total because of missing values.
‡Married or cohabitant.

Table II. Infant anthropometric measures at birth and 1-year of age by maternal feeding method and smoking status - MoBa

Infant anthropometric measures	Total sample	Exclusive breast-feeding n = 36,008				Mixed-feeding n = 16,886				Exclusive formula-feeding n = 1,828			
		No smoking n = 33,018	1-10 cig/day n = 1,707	>10 cig/day n = 1,283	P*	No smoking n = 13,473	>10 cig/day n = 1,564	>10 cig/day n = 1,649	P*	No smoking n = 1,302	Smoking n = 156	>10 cig/day n = 370	P*
Weight at birth (gr)	3641 (501)	3656 (489)	3674 (497)	3566 (495)	<.01	3626 (519)	3624 (517)	3537 (516)	<.01	3651 (524)	3668 (536)	3536 (552)	<.01
Weight at 1 year (gr)	9960 (1116)	9853 (1083)	9947 (1134)	9966 (1189)	<.01	10 127 (1136)	10 096 (1118)	10 213 (1185)	.03	10 303 (1144)	10 382 (1236)	10 334 (1214)	.56
Weight difference (gr)	6318 (1040)	6197 (1004)	6273 (1034)	6401 (1119)	<.01	6500 (1054)	6473 (1023)	6676 (1112)	<.01	6652 (1069)	6715 (1223)	6798 (1162)	.02
Length at birth (mm)	505 (20)	506 (20)	506 (20)	502 (20)	<.01	505 (21)	505 (21)	501 (21)	<.01	505 (21)	505 (21)	500 (21)	<.01
Length at 1 year (mm)	767 (27)	764 (27)	766 (28)	763 (28)	.94	770 (27)	769 (28)	769 (28)	.12	773 (27)	774 (27)	770 (27)	.03
Length difference (cm)	261 (25)	258 (25)	260 (25)	262 (26)	<.01	265 (25)	265 (25)	269 (27)	<.01	268 (25)	269 (27)	270 (24)	.30
WFL z-score at birth	0.45 (1.00)	0.46 (0.99)	0.50 (0.99)	0.48 (1.00)	.26	0.42 (1.03)	0.43 (1.01)	0.42 (1.02)	.96	0.50 (1.04)	0.54 (1.05)	0.47 (1.02)	.72
WFL z-score at 1 year	0.46 (0.99)	0.24 (0.92)	0.30 (0.93)	0.39 (0.99)	<.01	0.40 (0.95)	0.40 (0.95)	0.54 (0.97)	<.01	0.53 (0.98)	0.63 (1.01)	0.67 (0.98)	.01
WFL z-score difference	0.49 (1.00)	−0.22 (1.24)	−0.20 (1.23)	−0.09 (1.31)	<.01	−0.02 (1.26)	−0.03 (1.25)	0.11 (1.28)	<.01	0.03 (1.33)	0.09 (1.42)	0.19 (1.29)	.04

*P for trend from linear regression models.

of 100 g (95% CI, −30, 231) of weight and 2.8 mm (95% CI,0.1, 5.6) of height that is specifically attributable to ingesting smoker's breastmilk.

Next, we fit separate interaction terms among boys and girls to determine whether the observed effects may be sex-specific (Table IV). Evidence of interaction was apparent only among girls breastfed or ever-breastfed by a heavy smoker. Most notably, ever-breastfed girls gained an average of 251 g (95% CI, 66, 437) and 5.9 mm (95% CI, 2.0, 9.7) attributable to ingesting smoker's breastmilk. No such gains were observed among boys.

We conducted 2 sensitivity analyses. First, we fit interaction terms for parameters of interest at eight months of age. Wherever there was evidence of an interaction, this evidence was stronger at eight rather than 12 months of age (estimates not shown). Second, we fit interaction terms in sample that excluded SGA infants (n = 53 255) and found similar estimates to those found in the overall sample. Finally, among a sample limited to SGA infants only (n = 1 267), we found no evidence of an interaction between maternal smoking and feeding method (estimates not shown).

Discussion

We addressed a gap in the literature on the synergy between feeding methods, nicotine exposure, and growth during infancy by answering 3 interrelated questions: First, are infants breastfed by smokers at risk of rapid weight and length gain? Second, if so, is rapid growth among infants breastfed by smokers attributable to ingestion of smokers' breastmilk? 3) Finally, if so, what are the implications for breastfeeding by smokers?

Among this large, population-based sample, we observed an independent dose-dependent association between maternal smoking and rapid weight and length gain among both breastfed and mixed-fed but not formula-fed infants (Table II). These dose-dependent associations were most pronounced among breastfed infants, and less pronounced among mixed-fed infants. This evidence of rapid growth among infants breastfed by smokers agrees with earlier observations among members of a Health Maintenance Organization (HMO)[24] and contrasts with null findings among Dutch birth cohorts.[25] We suspect that positive findings among the HMO members, in part, reflect the much higher prevalence of smokers among that urban sample than the nationally representative Dutch sample (41% vs 8%). Moreover, the Dutch sample likely included lighter smokers. As such, the Dutch study's null finding likely reflects the protective effect of breastfeeding among infants exposed to light and moderate maternal smoking. Extant evidence suggests that infants breastfed by heavy smokers are at risk of rapid weight and length gain.

Next, we found limited evidence that rapid gains in length, but not weight, among infants breastfed by heavy smokers may be partially attributable to ingesting smokers'

Table III. Multivariable analysis of change in weight, length, and WFLZ during infancy as a function of feeding method and postpartum cigarette smoking*

	Change during infancy		
	Weight	Length	WFL z-score
	gr (95% CI)	mm (95% CI)	Change (95% CI)
A – Main effects - Full Sample			
Feeding method			
Breastfed	reference	reference	reference
Mixed-fed	214 (194, 234)	3.9 (3.4, 4.3)	0.15 (0.13, 0.17)
Formula-fed	361 (311, 412)	6.4 (5.3, 7.6)	0.26 (0.21, 0.31)
Cigarettes Smoked/day			
0	reference	reference	reference
1-10	22 (−13, 57)	0.5 (−0.3, 1.3)	0.02 (−0.01, 0.05)
>10	172 (132, 211)	1.7 (0.8, 2.6)	0.12 (0.08, 0.15)
B – Main effects – Stratified Samples†			
Breastfed			
Cigarettes Smoked/day			
0	reference	reference	reference
1-10	59 (12, 107)	1.2 (0.1, 2.4)	0.05 (0.01, 0.09)
>10	176 (115, 238)	1.2 (0.1, 2.6)	0.13 (0.07, 0.18)
Mixed-fed			
Cigarettes Smoked/day			
0	reference	reference	reference-
1-10	−25 (−78, 27)	−0.6 (−1.8, 0.6)	0.01 (−0,06, 0.04)
>10	167 (110, 223)	2.2 (0.9, 3.5)	0.10 (0.05, 0.16)
Formula-fed			
Cigarettes Smoked/day			
0	reference	reference	reference
1-10	115 (−75, 305)	1.8 (−2.0, 5.6)	0.08 (−0.08, 0.25)
>10	119 (−15, 253)	−0.9 (−3.7, 1.9)	0.11 (−0.01, 0.24)
C – Interaction – Full Sample‡			
Ever breastfed*smoking§			
Cigarettes Smoked/day			
0	reference	reference	reference
1-10	56 (−250, 138)	−1.1 (−4.9, 2.8)	−0.03 (−0.20, 0.15)
>10	100 (−30, 231)	2.8 (0.1, 5.6)	0.04 (−0.08, 0.16)

Mother–infant pairs, MoBa (n = 54,522).
*All models included the following covariates: age at birth, parity, place of birth, marital status, education, family income (Norwegian Kroner), BMI at conception, other nicotine intakes during pregnancy (eg snuff, patches), gestational diabetes, gestational hypertensions, sex of infant, gestational age, apgar score at 5 minutes, introduction of solid foods.
†Separate models fit among each feeding group.
‡The interaction term estimates the difference between differences: (ever breastfed, 1-10 cig/day vs 0 cig/day) vs (Formula, 1-10 cig/day vs 0 cig/day). See the Methods section for details.
§Ever-breastfed group includes both breastfed and mixed-fed groups.

breastmilk. This evidence was most pronounced among infants who were *ever breastfed* by heavy smokers. This observation agrees with evidence from the only other study of the association between ingesting smokers' breastmilk and growth during infancy.[26] Together findings from these 2 studies suggests that salubrious effects of breastmilk on growth during infancy may be limited by the amount of maternal smoking. However, within the broad range of smoking in this large study, rapid gains in weights and lengths attributable to ingesting smokers' breastmilk did not meaningfully vary from population norms (as indicated by low values for WFLZ: an index of deviation from normative growth patterns for healthy, breastfed infants raised in healthy environments).[31] In sum, evidence implicates importance of the amounts of both maternal smoking *and* ingested breastmilk. However, rapid growth among infants breastfed by light smokers does not exceed normative growth patterns at 1-year.

This epidemiological evidence is supported by laboratory evidence that infants can metabolize relatively small amounts of ingested nicotine to cotinine and then clear the cotinine.[32] Based on our findings, and other evidence that breastfeeding can mitigate harmful effects of secondhand smoke exposure,[33-35] we conclude that in the short-term obesogenic properties of smokers' breastmilk is outweighed by the many benefits of breastfeeding, particularly among light smokers.

Our sex-specific analyses revealed effects of heavy smoking on 1-year weight and length gains among breastfed and ever-breastfed girls, but not boys. Animal models suggest that nicotine exposure during periods of plasticity, such as lactation, can have sexually dimorphic effects on hormones which influence infants' developmental trajectory.[36,37] For example, nicotine can program production and function of hormones involved in the development of white adipose tissue (eg, corticosterone and insulin)[38,39] in a sexually dimorphic manner.[37,39] The extent to which these laboratory findings may be further replicated among human populations requires detailed consideration of various mediating and moderating factors including maternal morphology and lived experiences (eg, stress, education, parity) is beyond the scope of the present study. Evidence of sexual

Table IV. Sex-specific multivariable interaction terms for change in weight, length, and WFLZ during infancy as a function of feeding method, postpartum cigarette smoking, and infants' sex*,†

	1-year weight change (gr)	1-year length change (mm)	1-year WFL z-score change
Girls			
Breastfed			
Cigarettes Smoked/day			
0	Ref.	Ref.	Ref.
1-10	−4 (−258, 249)	−1.8 (−7.2, 3.6)	0.04 (−0.19, 0.27)
>10	209 (12, 405)	5.3 (1.2, 9.4)	0.10 (−0.08, 0.27)
Mixed fed			
Cigarettes Smoked/day			
0	Ref.	Ref.	Ref.
1-10	−10 (−265, 246)	−3.6 (−9.1, 1.8)	0.08 (−0.15, 0.31)
>10	168 (−22, 360)	4.0 (0.0, 8.1)	0.08 (−0.09, 0.25)
Ever Breastfed‡			
Cigarettes Smoked/day			
0	Ref.	Ref.	Ref.
1-10	36 (−214, 286)	−1.8 (−7.1, 3.5)	0.08 (−0.14, 0.31)
>10	251 (66, 437)	5.9 (2.0, 9.7)	0.13 (−0.03, 0.30)
Boys			
Breastfed			
Cigarettes Smoked/day			
0	Ref.	Ref.	Ref.
1-10	−69 (−380, 243)	1.1 (−4.7, 7.0)	−0.09 (−0.36, 0.19)
>10	−99 (−295, 97)	−2.3 (−6.4, 1.8)	−0.07 (−0.24, 0.12)
Mixed-fed			
Cigarettes Smoked/day			
0	Ref.	Ref.	Ref.
1-10	−225 (−537, 86)	−1.0 (−6.9, 4.9)	−0.23 (−0.51, 0.05)
>10	−37 (−228, 155)	1.2 (−2.9, 5.2)	−0.06 (−0.23, 0.11)
Ever Breastfed			
Cigarettes Smoked/day			
0	Ref.	Ref.	Ref.
1-10	−116 (−425, 192)	−0.5 (−5.2, 6.3)	−0.14 (−0.41, 0.14)
>10	−30 (−215, 155)	0.2 (−3.7, 4.0)	−0.04 (−0.21, 0.13)

Mother–infant pairs, MoBa (n = 54,522).
*All models included the following covariates: age at birth, parity, place of birth, marital status, education, family income (Norwegian Kroner), BMI at conception, other nicotine intakes during pregnancy (eg snuff, patches), gestational diabetes, gestational hypertensions, sex of infant, gestational age, apgar score at 5 minutes, introduction of solid foods.
†Interaction terms represent the difference in differences between formula-fed infants and the stated exposure (ie, breastfed, mixed-fed, ever breastfed): (eg, Breastfed, 1-10 cig/day vs 0 cig/day) vs (Formula, 1-10 cig/day vs 0 cig/day). See the Methods section for details.
‡Ever-breastfed group includes both breastfed and mixed-fed groups.

dimorphism presented here should be considered as hypothesis generating for future human studies rather than confirmation of laboratory evidence.

Ideally clinicians should help prevent smoking initiation and onset of tobacco addiction. Given that smoking addiction generally precedes family planning and pregnancy, we emphasize the critical importance of also helping breastfeeding mothers to quit smoking.[4,40] However, abating nicotine addiction is particularly difficult during this inherently stressful transitional period.[41,42] Post-partum quit rates are notoriously low, especially among people who smoked during pregnancy.[42,43] Postpartum smokers would benefit when routine postpartum care is complemented with counseling-based cessation programs[44,45] that counsel abstinence over the *longer-term*.[40] Such programs can also effectively include a breastfeeding promotion component.[46] A longer-term approach to abstinence is more likely to be successful and evidence presented here suggests that this approach will not adversely influence growth trajectory of infants breastfed by light smokers.

Although this conclusion is in accord with recommendations from health agencies indicating that breastfeeding by smokers is preferable over formula-feeding,[47] we emphasize the many negative consequences of exposure to nicotine and secondhand smoke. Nicotine exposure during infancy can program metabolic function in a manner that increases adiposity and elevate risk of obesity and other metabolic dysfunctions as early as childhood and continuing through adulthood.[36,48-50] Moreover, while the bulk of evidence regarding pathogenic consequences of nicotine exposure through breastmilk over the lifecourse comes from laboratory studies in which animals were administered doses of nicotine that correspond to heavy smoking among humans, exposure to lower doses of nicotine should not be considered safe.[51,52] Increasingly discrepant growth trajectories throughout the lifecourse remain a possibility. Clinicians should caution breastfeeding mothers of the possible pathogenic consequences of even light smoking on the long-term growth and development of their infant.[51,52]

We note the study's weaknesses. The analytic sample for this study is subjected to self-selection. During MoBa's initial recruitment period, ∼45% of all mothers who gave birth in Norway consented to participate. Compared to the general population, mothers who reported healthy behaviors are overrepresented among MoBa participants. However, an investigation of bias arising from self-selection in MoBa's sample did

not find estimated associations to be biased.[53] Nevertheless, the higher prevalence of maternal smoking, and the lower birthweight and height of excluded infants suggests a higher prevalence of SGA infants among the excluded group than in the analytic sample (**Supplementary Table 1**; available at www.jpeds.com). Our relatively small sample of SGA infants did not allow us to examine SGA infants potentially diverging growth trajectory,[54] our findings may not generalize to infants who were born growth restricted. We lacked data on the actual amount of breastmilk intake. Because maternal smoking can inhibit breastmilk intake via reduced milk production, early weaning, and other possible complications,[19,32,55-57] breastfed and mixed-fed infants of smokers may have ingested less breastmilk than those fed by non-smokers. Consequently, more pronounced weight gain among these infants may reflect lower breastmilk intake rather than obesogenic properties of smoker's breastmilk. Since we lack detailed data on infants' overall nutrient intake and potentially other relevant variables,[57] our findings might be subject to a degree of unmeasured confounding. Finaly, because mother-infant dyads were followed only during infancy, possible longer term obesogenic sequalae of nicotine exposure via breastmilk remain unknown.

In this largest and most detailed study of feeding method, nicotine exposure, and growth during infancy, we observed rapid growth among infants breastfed by smokers; and limited evidence that some of these gains can be attributed to ingesting smokers' breastmilk. The sum of available evidence suggests that salubrious effects of breastmilk on growth during infancy may be limited by the amount of maternal smoking. However, among average-for-gestational-age infants of light smokers, these gains are within the range of normative growth patterns for healthy, breastfed infants. Clinicians caring for light smokers can continue to promote breastfeeding while referring patients to programs that counsel smoking cessation over the longer term. Among light smokers, benefits of breastfeeding far outweigh short-term obesogenic consequences. Possible long-term obesogenic consequences remain unknown. ■

CRediT authorship contribution statement

Edmond D. Shenassa: Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Edoardo Botteri:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis. **Hanne Stensheim:** Writing – review & editing, Data curation.

Declaration of Competing Interest

The authors declare no conflicts of interest.

Submitted for publication Mar 22, 2024; last revision received Sep 10, 2024; accepted Sep 30, 2024.

Reprint requests: Edmond D. Shenassa, ScD, MA, FACE, Department of Epidemiology and Biostatistics, School of Public Health, 4254 Stadium Dr, College Park, MD 20742. E-mail: Shenassa@UMD.edu

References

1. American Academy of Pediatrics Committee on Drugs. The transfer of drugs and other chemicals into human breast milk. Pediatrics 1983;72:375-83. https://doi.org/10.1542/peds.72.3.375
2. Eidelman AI, Schanler RJ, Johnston M, Johnston M, Landers S, Noble L, et al. Breastfeeding and the use of human milk. Pediatrics 2012;129:e827-41. https://doi.org/10.1542/peds.2011-3552
3. American Academy of Pediatrics Committee on Drugs. The transfer of drugs and other chemicals into human milk. Pediatrics 2001;108:776-89. https://doi.org/10.1542/peds.108.3.776
4. Meek JY, Noble L. Technical report: breastfeeding and the use of human milk. Pediatrics 2022;150:e2022057989. https://doi.org/10.1542/peds.2022-057989
5. McLean C, Jun S, Kozyrskyj A. Impact of maternal smoking on the infant gut microbiota and its association with child overweight: a scoping review. World J Pediatr 2019;15:341-9. https://doi.org/10.1007/s12519-019-00278-8
6. Schulte-Hobein B, Schwartz-Bickenbach D, Abt S, Plum C, Nau H. Cigarette smoke exposure and development of infants throughout the first year of life: influence of passive smoking and nursing on cotinine levels in breast milk and infant's urine. Acta Paediatr 1992;81:550-7. https://doi.org/10.1111/j.1651-2227.1992.tb12293.x
7. Bruin JE, Gerstein HC, Holloway AC. Long-term consequences of fetal and neonatal nicotine exposure: a critical review. Toxicol Sci 2010;116:364-74. https://doi.org/10.1093/toxsci/kfq103
8. Gilman SE, Gardener H, Buka SL. Maternal smoking during pregnancy and children's cognitive and physical development: a causal risk factor? Am J Epidemiol 2008;168:522-31. https://doi.org/10.1093/aje/kwn175
9. Wen X, Shenassa ED, Paradis AD. Maternal smoking, breastfeeding, and risk of childhood overweight: findings from a national cohort. Matern Child Health J 2013;17:746-55. https://doi.org/10.1007/s10995-012-1059-y
10. Ong KKL, Ahmed ML, Emmett PM, Preece MA, Dunger DB. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. BMJ 2000;320:967-71. https://doi.org/10.1136/bmj.320.7240.967
11. Gonçalves R, Wiertsema CJ, Silva CCV, Monasso GS, Gaillard R, Steegers EAP, et al. Associations of fetal and infant growth patterns with early markers of arterial health in school-aged children. JAMA Netw Open 2022;5:e2219225. https://doi.org/10.1001/jamanetworkopen.2022.19225
12. Gillman MW, Ludwig DS. How early should obesity prevention start? N Engl J Med 2013;369:2173-5. https://doi.org/10.1056/NEJMp1310577
13. Gillman MW, Rifas-Shiman SL, Kleinman K, Oken E, Rich-Edwards JW, Taveras EM. Developmental origins of childhood overweight: potential public health impact. Obesity (Silver Spring) 2008;16:1651-6. https://doi.org/10.1038/oby.2008.260
14. Hu J, Aris IM, Lin PID, Rifas-Shiman SL, Perng W, Woo Baidal JA, et al. Longitudinal associations of modifiable risk factors in the first 1000 days with weight status and metabolic risk in early adolescence. Am J Clin Nutr 2021;113:113-22. https://doi.org/10.1093/ajcn/nqaa297
15. Kramer MS, Guo T, Platt RW, Vanilovich I, Sevkovskaya Z, Dzikovich I, et al. Feeding effects on growth during infancy. J Pediatr 2004;145:600-5. https://doi.org/10.1016/j.jpeds.2004.06.069
16. Baird J, Poole J, Robinson S, Marriott L, Godfrey K, Cooper C, et al. Milk feeding and dietary patterns predict weight and fat gains in infancy. Paediatr Perinat Epidemiol 2008;22:575-86. https://doi.org/10.1111/j.1365-3016.2008.00963.x
17. Mihrshahi S, Battistutta D, Magarey A, Daniels LA. Determinants of rapid weight gain during infancy: baseline results from the NOURISH randomised controlled trial. BMC Pediatr 2011;11:99. https://doi.org/10.1186/1471-2431-11-99

18. Rybak TM, Goetz AR, Stark LJ. Examining patterns of postnatal feeding in relation to infant's weight during the first year. Appetite 2021;166:105473 https://doi.org/10.1016/j.appet.2021.105473
19. Shenassa ED. Maternal smoking during pregnancy and offspring weight gain: a consideration of competing explanations. Paediatr Perinat Epidemiol 2017;31:409-11. https://doi.org/10.1111/ppe.12405
20. Milnerowicz H, Chmarek M. Influence of smoking on metallothionein level and other proteins binding essential metals in human milk. Acta Paediatr 2005;94:402-6. https://doi.org/10.1111/j.1651-2227.2005.tb01908.x
21. Bachour P, Yafawi R, Jaber F, Choueiri E, Abdel-Razzak Z. Effects of smoking, mother's age, body mass index, and parity number on lipid, protein, and secretory immunoglobulin a concentrations of human milk. Breastfeed Med 2012;7:179-88. https://doi.org/10.1089/bfm.2011.0038
22. Szlagatys-Sidorkiewicz A, Martysiak-Żurowska D, Krzykowski G, Zagierski M, Kamińska B. Maternal smoking modulates fatty acid profile of breast milk. Acta Paediatr 2013;102:e353-9. https://doi.org/10.1111/apa.12276
23. Gartner LM, Morton J, Lawrence RA, Naylor AJ, O'Hare D, Schanler RJ, et al. Breastfeeding and the use of human milk. Pediatrics 2005;115:496-506. https://doi.org/10.1542/peds.2004-2491
24. Little RE, Lambert MD, Worthington-Roberts B, Ervin CH. Maternal smoking during lactation: relation to infant size at one year of age. Am J Epidemiol 1994;140:544-54. https://doi.org/10.1093/oxfordjournals.aje.a117281
25. Boshuizen HC, Verkerk PH, Reerink JD, Hemgreen WP, Zaadstra BM, Verloove-Vanhorick SP. Maternal smoking during lactation: relation to growth during the first year of life in a Dutch birth cohort. Am J Epidemiol 1998;147:117-26. https://doi.org/10.1093/oxfordjournals.aje.a009423
26. Shenassa ED, Wen X, Braid S. Exposure to Tobacco metabolites via breast milk and infant weight gain: a population-based study. J Hum Lact 2016;32:462-71. https://doi.org/10.1177/0890334415619154
27. Magnus P, Birke C, Vejrup K, Haugan A, Alsaker E, Daltveit AK, et al. Cohort profile update: the Norwegian mother and child cohort study (MoBa). Int J Epidemiol 2016;45:382-8. https://doi.org/10.1093/ije/dyw029
28. de Onis M, Garza C, Victora CG, Onyango AW, Frongillo EA, Martines J. The WHO Multicentre Growth Reference Study: planning, study design, and methodology. Food Nutr Bull 2004;25:S15-26. https://doi.org/10.1177/15648265040251S103
29. Sengpiel V, Elind E, Bacelis J, Nilsson S, Grove J, Myhre R, et al. Maternal caffeine intake during pregnancy is associated with birth weight but not with gestational length: results from a large prospective observational cohort study. BMC Med 2013;11:42. https://doi.org/10.1186/1741-7015-11-42
30. Tai-Seale M, Freund D, LoSasso A. Racial disparities in service use among medicaid beneficiaries after mandatory enrollment in managed care: a difference-in-differences approach. Inquiry 2001;38:49-59. https://doi.org/10.5034/inquiryjrnl_38.1.49
31. de Onis M, Onyango AW, Borghi E, Garza C, Yang H. Comparison of the World health Organization (WHO) child growth standards and the national center for health statistics/WHO international growth reference: implications for child health programmes. Public Health Nutr 2006;9:942-7. https://doi.org/10.1017/PHN20062005
32. Napierala M, Mazela J, Merritt TA, Florek E. Tobacco smoking and breastfeeding: effect on the lactation process, breast milk composition and infant development. A critical review. Environ Res 2016;151:321-38. https://doi.org/10.1016/j.envres.2016.08.002
33. Batstra L, Neeleman J, Hadders-Algra M. Can breast feeding modify the adverse effects of smoking during pregnancy on the child's cognitive development? J Epidemiol Community Health 2003;57:403-4. https://doi.org/10.1136/jech.57.6.403
34. Nafstad P, Jaakkola J, Hagen J, Botten G, Kongerud J. Breastfeeding, maternal smoking and lower respiratory tract infections. Eur Respir J 1996;9:2623-9. https://doi.org/10.1183/09031936.96.09122623
35. Ip S, Chung M, Raman G, Chew P, Magula N, DeVine D, et al. Breastfeeding and maternal and infant health outcomes in developed countries. Evid Rep Technol Assess 2007: 1-186.
36. Miranda RA, Gaspar de Moura E, Lisboa PC. Tobacco smoking during breastfeeding increases the risk of developing metabolic syndrome in adulthood: lessons from experimental models. Food Chem Toxicol 2020;144:111623. https://doi.org/10.1016/j.fct.2020.111623
37. Pinheiro C, Oliveira E, Trevenzoli I, Manhães AC, Santos-Silva AP, Younes-Rapozo V, et al. Developmental plasticity in adrenal function and leptin production primed by nicotine exposure during lactation: gender differences in rats. Horm Metab Res 2011;43:693-701. https://doi.org/10.1055/s-0031-1285909
38. Oliveira E, Moura EG, Santos-Silva AP, Fagundes AT, Rios AS, Abreu-Villaça Y, et al. Short- and long-term effects of maternal nicotine exposure during lactation on body adiposity, lipid profile, and thyroid function of rat offspring. J Endocrinol 2009;202:397-405. https://doi.org/10.1677/JOE-09-0020
39. Rodrigues VDST, Miranda RA, Soares PN, Peixoto TC, de Oliveira E, Manhães AC, et al. Neonatal nicotine exposure changes insulin status in fat depots: sex-related differences. J Dev Orig Health Dis 2022;13:252-62. https://doi.org/10.1017/S2040174421000131
40. Tobacco and nicotine cessation during pregnancy: ACOG Committee Opinion, Number 807. Obstet Gynecol 2020;135:e221-9. https://doi.org/10.1097/AOG.0000000000003822
41. Fahey JO, Shenassa E. Understanding and meeting the needs of women in the postpartum period: the perinatal maternal health promotion model. J Midwifery Womens Health 2013;58:613-21. https://doi.org/10.1111/jmwh.12139
42. Barber GA, Shenassa ED. Smoking status: a tacit screen for postpartum depression in primary care settings. J Affect Disord 2021;295:1243-50. https://doi.org/10.1016/j.jad.2021.09.033
43. Hajek P, Stead LF, West R, Jarvis M, Hartmann-Boyce J, Lancaster T. Relapse prevention interventions for smoking cessation. Cochrane Database Syst Rev 2013CD003999. https://doi.org/10.1002/14651858.CD003999.pub4
44. Chamberlain C, O'Mara-Eves A, Porter J, Coleman T, Perlen SM, Thomas J, et al. Psychosocial interventions for supporting women to stop smoking in pregnancy. Cochrane Database Syst Rev 2017;2020(3). https://doi.org/10.1002/14651858.CD001055.pub5
45. Diamanti A, Papadakis S, Schoretsaniti S, Rovina N, Vivilaki V, Gratziou C, et al. Smoking cessation in pregnancy: an update for maternity care practitioners. Tob Induc Dis 2019;17:57. https://doi.org/10.18332/tid/109906
46. Issany A, Hore M, Singh L, Israel J, Kocher MG, Wen X. Reciprocal associations between maternal smoking cessation and breastfeeding. Breastfeed Med 2022;17:226-32. https://doi.org/10.1089/bfm.2021.0199
47. Harris M, Schiff DM, Saia K, Muftu S, Standish KR, Wachman EM. Academy of breastfeeding medicine clinical protocol #21: breastfeeding in the setting of substance use and substance use disorder (revised 2023). Breastfeed Med 2023;18:715-33. https://doi.org/10.1089/bfm.2023.29256.abm
48. Fan J, Zhang WX, Rao YS, Xue JL, Wang FF, Zhang L, et al. Perinatal nicotine exposure increases obesity susceptibility in adult male rat offspring by altering early adipogenesis. Endocrinology 2016;157:4276-86. https://doi.org/10.1210/en.2016-1269
49. Desai M, Ross M. Fetal programming of adipose tissue: effects of intrauterine growth restriction and maternal obesity/high-fat diet. Semin Reprod Med 2011;29:237-45. https://doi.org/10.1055/s-0031-1275517
50. Behl M, Rao D, Aagaard K, Davidson TL, Levin ED, Slotkin TA, et al. Evaluation of the association between maternal smoking, childhood obesity, and metabolic disorders: a national toxicology program workshop review. Environ Health Perspect 2013;121:170-80. https://doi.org/10.1289/ehp.1205404
51. Shenassa ED, Rogers ML, Buka SL. Maternal smoking during pregnancy, offspring smoking, adverse childhood events, and risk of major depression: a sibling design study. Psychol Med 2023;53:206-16. https://doi.org/10.1017/S0033291721001392
52. Shenassa ED, Gleason JL, Hirabayashi K. Fetal exposure to tobacco metabolites and depression during adulthood: beyond binary measures.

Epidemiology 2024;35:602-9. https://doi.org/10.1097/EDE.0000000000001757

53. Nilsen RM, Vollset SE, Gjessing HK, Skjaerven R, Melve KK, Schreuder P, et al. Self-selection and bias in a large prospective pregnancy cohort in Norway. Paediatr Perinat Epidemiol 2009;23:597-608. https://doi.org/10.1111/j.1365-3016.2009.01062.x
54. Li X, Eiden RD, Epstein LH, Shenassa ED, Xie C, Wen X. Etiological subgroups of small-for-gestational-age: Differential neurodevelopmental outcomes. PLoS One 2016;11:e0160677 https://doi.org/10.1371/journal.pone.0160677
55. Amir LH. Maternal smoking and reduced duration of breastfeeding: a review of possible mechanisms. Early Hum Dev 2001;64:45-67. https://doi.org/10.1016/S0378-3782(01)00170-0
56. SM D, LH A. The relationship between maternal smoking and breastfeeding duration after adjustment for maternal infant feeding intention. Acta Paediatr 2004;93:1514-8. https://doi.org/10.1080/08035250410022125
57. Shenassa ED, Kinsey C, Moser Jones M, Fahey J. Gestational weight gain: historical evolution of a contested health outcome. Obstet Gynecol Surv 2017;72:445-53. https://doi.org/10.1097/OGX.0000000000000459