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Differences in Fertility Associated with Caffeinated Beverage Consumption

Bette Caan, DrPH, Charles P. Quesenberry, Jr, PhD, and Ashley O. Coates, MPH

ABSTRACT

Objectives. The effect of caffeine consumption on fertility was examined prospectively in 210 women.

Methods. Women reported on caffeinated beverage consumption and pregnancy status monthly. Odds ratios for becoming pregnant were calculated for both high and moderate vs low consumption.

Results. No significant association was found for any of the caffeinated beverages except tea. Drinking one-half cup or more of tea daily approximately doubled the odds of conception per cycle.

Conclusions. These data suggest that caffeine may not be the responsible agent for variation in fertility associated with consumption of the beverages examined. (*Am J Public Health.* 1998;88:270–274)

Introduction

An effect of caffeine on human fertility was first reported in a prospective study by Wilcox et al.,[1] who reported a 50% reduction in the per cycle probability of conceiving with intakes equivalent to 1 cup of coffee per day. This was followed by 7 additional reports using retrospectively collected data and an additional prospective study. Five of the retrospective studies[2–6] reported a decrease in fertility due to caffeine, 1 in smokers only[4] and 1 in nonsmokers only.[6] Two of the retrospective studies[7,8] and the other prospective study[9] found no relationship. The potential for reporting biases in retrospective studies and the inconsistency in reported findings prompted us to conduct this study.

Methods

Two hundred ten volunteer members of the Kaiser Permanente Medical Program who were trying to conceive were followed for 12 months or until the month after they became aware they were pregnant, whichever came first. Only those women who had been trying to conceive for 3 months or less were eligible to participate. If a women had been trying to conceive for 1, 2, or 3 months prior to entering the study, her first cycle of entry into the study was not categorized as cycle 1; rather, it was respectively categorized as cycle 2, 3, or 4. Based on that schema, not all 210 women started in cycle 1. Women could start anywhere from cycle 1 to cycle 4, depending on how many months prior to entry they were trying to conceive. The number of women at risk in

The authors are with the Division of Research, Kaiser Permanente Medical Care Program of Northern California, Oakland.

Requests for reprints should be sent to Bette Caan, DrPH, Kaiser Permanente Medical Care Program of Northern California, Division of Research, 3505 Broadway, Oakland CA 94611.

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TABLE 1—Selected Characteristics of 187 Women Enrolled in a Northern California Health Maintenance Organization Who Were Trying to Conceive between 1990 and 1992

	No.	Mean (SD)	Percentile			Consuming None, %
			25th	50th	75th	
Total caffeine, mg/wk	187	601.0 (724)	49	289	939	8.0
Regular coffee, servings/wk	187	4.2 (6.5)	0.0	0.5	7.0	41.7
Decaffeinated coffee, servings/wk	187	1.5 (3.0)	0.0	0.0	1.0	52.0
Tea, servings/wk	186	1.6 (3.5)	0.0	0.0	1.0	53.8
Decaffeinated tea, servings/wk	187	3.0 (6.1)	0.0	0.5	2.8	37.4
Caffeinated sodas, cans/wk	186	2.1 (4.0)	0.0	0.5	2.8	30.6
Alcoholic beverages	187	2.2 (4.4)	0.0	0.9	2.4	28.9
Smoking (yes/no)	186	...	...	...	...	93.0
Weight, lb	187	136.0 (24.7)	119	132	150	...
Age, y	187	31.8 (4.2)	29	32	35	...
Intercourse, times/wk	185	2.5 (1.6)	1.5	2.0	3.0	...

each cycle was the number of women trying to conceive minus the number of women without caffeine data available for that cycle. (For the majority of women, caffeine data were unavailable only when subjects conceived during their first cycle in the study and exposure data from the previous month were necessary because they ovulated early in the month.) Women who became pregnant were excluded from future cycles. Women who were breast-feeding and couples with diagnosed fertility problems were considered ineligible. When recruited into the study, women were told we were examining the relationship of diet to fertility.

At the end of each calendar month, each woman in the study completed a mailed food-frequency questionnaire[10] covering the past month's intake of common foods, including the frequency and serving size of regular and decaffeinated coffees, teas, and sodas. Each medium beverage serving was assigned the following caffeine values in milligrams: regular coffee, 104; decaffeinated coffee, 2; regular tea, 36; decaffeinated tea, 0; regular caffeinated sodas, 40; and diet caffeinated sodas, 52.[11] Small serving sizes were assigned half the medium amount, and large serving sizes were assigned 1.5 times the medium amount. Information was also obtained on smoking, exercise, frequency of intercourse, stress, and use of alcohol, vitamins, and nonprescription drugs.

Because data were collected by calendar month but menstrual cycles started at any point during the month, each cycle needed data assigned from the corresponding calendar month. For caffeine and smoking data, if estimated ovulation date (date of menses − 14 or reported conception date) was later than the 14th, the current month's data were assigned; otherwise, the previous month's data were assigned. For intercourse data, if the estimated ovulation date was the 1st or 2nd of the month, the previous month's data were assigned; otherwise, data from the current month were assigned. Cycles in which there was no intercourse were excluded from the analysis. In 1 case, a woman was excluded for 3 cycles because she reported using birth control pills during that time and was assumed not to be at risk of conception for those cycles.

Fertility in relation to total caffeine and each of the individual beverages was examined by estimation of odds ratios (the odds of pregnancy for the exposed group and the reference group expressed as a ratio). We compared women with moderate vs low and high vs low intakes for each of 3 groups of cycles (1 through 3, 4 through 6, 7 through 12). We stratified by these 3 groups of cycles to examine possible differences in effects in early vs late cycles. Other studies have reported an effect only

TABLE 2—Lifestyle Characteristics, by Level of Caffeine or Tea Intake, of 187 Women Enrolled in a Northern California Health Maintenance Organization Who Were Trying to Conceive between 1990 and 1992

	Caffeine, mg/day			Tea, servings/day		
	≤10.4	10.5–106.8	>106.8	0	≤0.5	>0.5
Weight, lb, mean (SD)	134.8 (19.4)	136.1 (26.3)	138.6 (27.4)	136.2 (23.4)	133.5 (22.6)	145.5 (33.9)
Cigarette smokers, no. (%)	2 (3.5)	5 (7.4)	6 (10.0)	6 (6.1)	6 (9.5)	1 (4.4)
No. Alcoholic beverages/wk, mean (SD)	0.76 (1.28)[a]	2.57 (6.07)	3.03 (3.88)	1.37 (2.22)[b]	3.26 (6.54)	2.60 (4.09)
Physical activity (2–3 times/wk), no. (%)	32 (55.2)	37 (55.2)	32 (53.3)	51 (52.0)	34 (54.0)	15 (65.2)
Use vitamins regularly, no. (%)	35 (60.3)	28 (40.6)	28 (46.7)	49 (49.0)	30 (47.6)	11 (47.8)
Stress score ≥5, no. ($)	20 (34.5)	17 (25.0)	24 (40.0)	30 (30.3)	23 (36.5)	8 (34.8)
No. previous pregnancies, mean (SD)	1.28 (1.47)	1.06 (1.21)[c]	1.80 (1.58)	1.33 (1.33)	1.33 (1.66)	1.57 (1.38)
Energy intake, kcal, mean (SD)	1468 (603)	1511 (513)	1603 (525)	1465 (532)[ac]	1479 (499)	1849 (536)
Fat intake, g, mean (SD)	52.3 (20.6)	58.6 (25.5)	63.0 (25.7)	55.4 (22.9)[ac]	55.7 (21.0)	75.5 (32.2)
Fat, % kcal, mean (SD)	32.8 (6.20)	34.7 (6.12)	34.8 (6.39)	33.9 (6.36)	33.9 (5.58)	36.3 (6.58)
Fruit and vegetables, servings/day, mean (SD)	2.49 (1.65)	1.96 (1.40)[c]	2.66 (1.63)	2.37 (1.53)	2.34 (1.74)	2.22 (1.34)

Note. Chi-square may not be valid for smoking as a result of small expected cell counts.
[a]Overall F test $P < .05$, level 1 significantly different from level 3.
[b]Overall F test $P < .05$, level 1 significantly different from level 2.
[c]Overall F test $P < .05$, level 2 significantly different from level 3.

TABLE 3—Relative Odds of Becoming Pregnant Associated with Daily Intake of Caffeine, Overall and by Cycle Group, in 187 Members of a Northern California Health Maintenance Organization Who Were Trying to Conceive between 1990 and 1992

	Cycles 1–3			Cycles 4–6		
	No. Pregnant Cycles	No. Non-Pregnant Cycles	OR (95% CI)	No. Pregnant Cycles	No. Non-Pregnant Cycles	OR (95% CI)
Caffeine, mg/day						
≤10.4	14	70	1.00 . . .	12	65	1.00 . . .
10.5–106.8	24	85	1.45 (0.69, 3.04)	12	78	0.83 (0.35, 1.98)
>106.8	20	67	1.55 (0.72, 3.34)	11	82	0.73 (0.30, 1.77)
Regular coffee, servings/day						
0	28	90	1.00 . . .	14	88	1.00 . . .
0.1–1	16	86	0.59 (0.29, 1.17)	16	79	1.26 (0.57, 2.74)
>1	14	46	0.97 (0.46, 2.04)	5	58	0.54 (0.19, 1.59)
Decaffeinated coffee, servings/day						
0	27	121	1.00 . . .	21	128	1.00 . . .
0.1–1	30	86	1.60 (0.88, 2.89)	12	82	0.89 (0.42, 1.91)
>1	1	15	0.33(0.04, 2.59)	2	15	0.79 (0.17, 3.72)
Regular tea, servings/day						
0	19	130	1.00 . . .	25	134	1.00 . . .
0.1–0.5	23	73	2.22 (1.12, 4.37)	8	71	0.59 (0.25, 1.37)
>0.5	16	15	7.25 (3.06, 17.17)	2	20	0.54 (0.12, 2.47)
Caffeinated soda, servings/day						
0	21	64	1.00 . . .	10	71	1.00 . . .
0.1–0.5	31	123	0.76 (0.40, 1.44)	20	112	1.25 (0.55, 2.83)
>0.5	6	34	0.55 (0.20, 1.52)	5	38	0.92 (0.29, 2.89)

Note. OR = odds ratio; CI = confidence intervals.

Continued

after 2 cycles of trying.[1] If not strongly related, risk factors may not be apparent until later cycles, when the most fertile women are eliminated from the cohort as a result of conception.

We also calculated the overall odds ratios, which included all months, comparing separately the high and moderate intake group with the low intake group. We used Cox's discrete failure time regression model to obtain estimates of the overall odds ratios, which allowed for dropouts and control of confounding, time-dependent covariates, and late entry into the study.[12] Covariates were included in the model if there was a priori knowledge of an association or if the regression coefficient associated with exposure changed appreciably when the covariate was added to the model.

Results

Of the 210 women entering the study, 187 contributed to the analysis because they were at risk of conception for some duration of the study and dietary data were available for their time at risk. In these 187 women, 118 conceptions occurred during the course of the study. An additional 48 women dropped out at some point before they became pregnant or completed their 12 months (predominantly because they changed their minds about becoming pregnant), and 21 women remained who were still not pregnant at the completion of the study.

Mean total caffeine intake was 610 mg weekly, the approximate equivalent of 6 cups of coffee per week. Only 8% of women consumed no caffeine from any source (Table 1).

Caffeine drinkers with the highest intakes had a significantly higher number of previous pregnancies and fruit and vegetable servings than those with moderate intakes. Persons who consumed the most tea had significantly higher energy and fat intakes than the two lower consumption groups (Table 2).

There was no significant decrease in fertility associated with total caffeine, coffee, decaffeinated coffee, or caffeinated soda for either moderate or high consumption. However, in later cycles (4 through 6, 7 through 12), there was a suggestion that both total caffeine and coffee were associated with a nonsignificant reduction in fertility. Tea consumption at the highest level was significantly associated with an increase in fertility. This relationship was observed only in cycles 1 through 3 (Table 3). There were no statistically significant interactions between cycle of trying and any of the exposure measures (data not shown).

Discussion

Our point estimates do not demonstrate a significant association between regular coffee or total caffeine consumption and a reduction in fertility; based on our very wide confidence intervals, however, we cannot rule out the possibility of such an association. In fact, we did observe a nonsignificant decrease in fertility with both total caffeine and coffee consumption in later cycles.

Lack of sufficient statistical power is not an explanation of our finding of no association with either coffee or total caffeine. Based on the observed cohort of 187, this study had 80% power to detect an odds ratio of 0.5 (2-sided, significance level = .05) among those with caffeine consumption equivalent to 1 cup or more per day relative to those consuming the equivalent of less than 1 cup per day, estimates at exposure levels very similar to what Wilcox et al. found to be significant in their prospective study.

One study (Olsen[4]) has found that coffee drinking reduces fertility only in smokers. Only 13 women in our study smoked, too few to examine separately. However, excluding them from the analysis had no effect on our results.

Surprisingly, we found that women who drank more than one-half cup of tea per day had a significant increase in fertil-

TABLE 3—*Continued*

No.	Cycles 7–12			Overall[a]		
	No. Pregnant Cycles	No. Non-Pregnant Cycles	OR (95% CI)	No. Pregnant Cycles	No. Non-Pregnant Cycles	OR (95% CI)
Caffeine, mg/day						
≤10.4	9	46	1.00 . . .	35	176	1.00 . . .
10.5–106.8	6	62	0.51 (0.17, 1.54)	41	214	1.08 (0.65, 1.81)
>106.8	10	89	0.64 (0.24, 1.71)	39	232	1.09 (0.63, 1.89)
Regular coffee, servings/day						
0	12	62	1.00 . . .	53	230	1.00 . . .
0.1–1	6	71	0.49 (0.17, 1.40)	38	228	0.82 (0.51, 1.34)
>1	7	64	0.64 (0.23, 1.77)	24	164	0.77 (0.43, 1.37)
Decaffeinated coffee, servings/day						
0	16	107	1.00 . . .	61	338	1.00 . . .
0.1–1	7	75	0.60 (0.23, 1.55)	49	240	1.10 (0.71, 1.68)
>1	1	15	0.44 (0.05, 3.62)	4	44	0.49 (0.15, 1.38)
Regular tea, servings/day						
0	17	112	1.00 . . .	59	363	1.00 . . .
0.1–0.5	7	67	0.69 (0.27, 1.77)	38	204	1.17 (0.74, 1.85)
>0.5	1	18	0.34 (0.04, 2.79)	18	51	2.05 (1.09, 3.88)
Caffeinated soda, servings/day						
0	9	59	1.00 . . .	38	188	1.00 . . .
0.1–0.5	13	91	0.90 (0.36, 2.26)	63	313	1.09 (0.68, 1.73)
>0.5	3	46	0.45 (0.11, 1.78)	14	115	0.81 (0.41, 1.62)

[a]Controlled for age, body mass index, gravidity, frequency of intercourse, smoking, and alcoholic beverages.

ity. Most of this association was driven by the first 3 cycles, and there did not appear to be any relation in subsequent cycles. Three other human studies have examined tea independently of the other caffeinated beverages.[13–15] Only 1 of the 3 found, as we did, that tea significantly increased fertility. In a study of Danish women, Florack et al.[9] found that, relative to women drinking no tea, women drinking up to 5 cups of tea per day had an increase in fertility, with an odds ratio of 1.9 (95% confidence interval [CI] = 1.2,3.0), and women who drank more than 5 cups per day had an odds ratio of 3.2 (95% CI = 1.3,7.7). We were unable to test the effects of tea at that level, since very few women in our study had high consumption levels.

One possible explanation for the observed association between tea and increased fertility is that tea consumption may be associated with other lifestyle characteristics that would enhance fertility. In support of this hypothesis, we observed a similar magnitude of effect on fertility at levels of tea consumption much lower than those seen in the Dutch study. Also, Schwarz et al.,[15] in a study on lifestyle factors associated with both tea and coffee consumption, demonstrated that tea drinking is associated with a "preventive or healthier" lifestyle. They found that tea drinkers smoked less, ate less fat, and exercised more than coffee drinkers. We controlled for smoking, alcohol, and body weight. Other risk factors associated with a preventive lifestyle, such as exercise, vitamin supplementation, and stress, did not differ by tea intake in our data set.

Another possible explanation for the positive tea association is that a chemical component of tea other than caffeine may be responsible for the observed increase in fertility. The fact that there seemed to be a dose–response relationship, at least in the Dutch study,[9] and the fact that we did not find a relationship with noncaffeinated teas (which are mostly herbal) lends support to this explanation. Studies in vitro and in vivo have demonstrated that the polyphenolic compounds found in tea have the ability to inhibit chromosomal aberrations,[16] which could increase fertility by decreasing the number of nonviable embryos. In addition, hypoxanthine, one of the xanthines found in tea, may be the principal component of a follicular fluid that contributes to the maturation and, thus, fertilizability of oocytes.[17] However, some substances found in tea, such as tannic acid, have been associated with a decrease in fecundity.[18,19]

It is also possible that our tea finding could be spurious or due to chance alone, since a large number of hypotheses were tested and most of the increased risk of fertility was seen only in the early months.

Our study improved on previous study designs in that we were able to examine beverage intake on a monthly basis prospectively, thereby obtaining data on caffeine exposure during the time period in which it might have an effect. We were also able to examine the effect of the individual caffeinated and noncaffeinated beverages separately, but, unfortunately, we were not fully able to distinguish decaffeinated teas from herbal teas. Future research should focus on separating out individual caffeinated and noncaffeinated beverages and obtaining specific measures of luteal and follicular phase exposures, both limitations of the current study. □

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Violence during Pregnancy: Measurement Issues

Terri J. Ballard, DrPH, Linda E. Saltzman, PhD, Julie A. Gazmararian, PhD, MPH, Alison M. Spitz, MPH, Suzanne Lazorick, MD, MPH, and James S. Marks, MD, MPH

ABSTRACT

Objectives. Standardized quantitative methods are needed to study occurrence and timing of violence in relation to pregnancy and to study the context in which pregnancy-related violence occurs.

Methods. Data from three published studies of prevalence of violence during pregnancy are used to illustrate ways to measure the association of violence in relation to pregnancy.

Results. Four patterns of violence in relation to pregnancy are identified, and related research issues are discussed. Also, 2 population-based surveys that address the suggestions presented here are discussed.

Conclusions. Better measurement of the association between violence and pregnancy will facilitate development of data-based prevention and intervention programs. (*Am J Public Health*. 1998;88:274–276)

Introduction

According to a recent review, the prevalence of women experiencing violence during pregnancy has been estimated to be between 0.9% and 20.1%, while the prevalence of violence at any time ranges from 9.7% to 29.7%.[1] Research has not yet confirmed whether pregnant women are at greater risk for violence initiated during pregnancy. Nor has it been confirmed, for women experiencing ongoing violence, whether the severity or frequency of violent incidents increases or decreases or whether violence ceases altogether during pregnancy. Further research is needed to understand the occurrence and timing of violence in relation to pregnancy and the context in which such pregnancy-related violence occurs. This knowledge will facilitate the development of data-based prevention and intervention programs addressing the specific needs of pregnant women who experience violence. In this paper, we provide several suggestions for improving investigation of the association of violence with pregnancy.

Measuring Frequency of Violence in Relationship to Pregnancy

Although many epidemiologic studies of violence during pregnancy report the prevalence of violence during the pregnancy under investigation as well as the prevalence of having a history of experiencing violence, few have specified a time period that excludes periods of pregnancy. For example, having a history of violence may mean ever experiencing violence, experiencing it during the year preceding a prenatal interview, or experiencing it during the 12 months preceding birth. In these examples, violence during the pregnancy under investigation is included as part of the definition of the history of violence.[1] To study whether violence occurring during pregnancy is specific to the pregnancy or is simply part of an ongoing pattern of violence, we suggest that the prevalence of violence be investigated during the following mutually exclusive time periods: violence during the pregnancy under study and violence during a specific time period before the pregnancy. Depending on whether violence occurred during each of those periods, 4 distinct patterns emerge: (1) no violence before pregnancy but vio-

At the time of this study, Terri J. Ballard was with the National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, Atlanta, Ga. She is now with the Instituto Superiore di Sanità, Rome, Italy. Linda E. Saltzman, Alison M. Spitz, and James S. Marks are with the Centers for Disease Control and Prevention. Linda E. Saltzman is also with the Division of Violence Prevention, National Center for Injury Prevention and Control; Alison M. Spitz and James S. Marks are also with the National Center for Chronic Disease Prevention and Health Promotion. Julie A. Gazmararian is with the Prudential Center for Health Care Research, Atlanta. Suzanne Lazorick is with the Department of Maternal and Child Health and School of Medicine, University of North Carolina at Chapel Hill School of Public Health, Chapel Hill, NC.

Requests for reprints should be sent to Linda E. Saltzman, PhD, National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, 4770 Buford Hwy, Mailstop K-60, Atlanta, GA 30341-3724.

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