Neural Tube Defects in Jamaica following Hurricane Gilbert

E. Marilyn Watson Duff, MScNutr, BScN, and Edward S. Cooper, MB, BS, FRCP

A B S T R A C T

An increased incidence of obvious live-birth neural tube defects (i.e., spina bifida cystica and encephalocele) occurred in Jamaica 11 to 18 months after Hurricane Gilbert. The conceptions of the affected babies coincided with a rise in megaloblastic change in sickle cell patients, suggesting a widespread drop in dietary folate intake. A detailed history was taken from each of the 17 affected mothers (case subjects) and 51 unaffected mothers (matched control subjects). The case subjects reported a significantly lower mean intake of dietary folate in the periconceptional period (154 μg/day) than did the control subjects (254 μg/day). The temporary increase in neural tube defects was associated with a diet comparatively low in folate in the periconceptional period, suggesting the dietary level of folate that fails to protect against neural tube defects under natural conditions. (*Am J Public Health.* 1994;84: 473–476)

Introduction

In 1991 the Medical Research Council Vitamin Study showed that recurrence of neural tube defects could be prevented by daily supplementation of the diets of high-risk mothers with 4 mg of folate around the time of conception.[1]

After the publication of this report, there was controversy over the dosage of folate supplementation and particularly the possible toxic effects of circulating unmetabolized free folate on the developing embryo.[2–6] In addition, differences of opinion existed worldwide as to what may be considered a folate-replete diet.[6–9]

In Jamaica, the incidence of obvious live-birth neural tube defects (spina bifida cystica and encephalocele) is low, averaging 1.4 per 10 000 live births over 10 years (an average of 2.0 referrals per quarter). In the aftermath of Hurricane Gilbert (September 12, 1988) there was an increase in live-birth neural tube defects with a peak of 5.0 per 10 000 (7 referrals) in one quarter. This increase, which has been reported elsewhere,[10] was significant (Poisson model; $P < .005$) and coincided in periconceptional time with an overt, significant increase in folic acid deficiency in a vulnerable group: homozygous sickle cell patients.[11]

The overwhelming, all-island devastation in the aftermath of Hurricane Gilbert greatly enhanced recall of events around that time. The results presented here, despite the retrospective nature of the information and the small numbers, shed light on the dosage controversy and the range of daily dietary folate that is inadequate to prevent neural tube defects.

Methods

The cases were the 17 mothers of babies with obvious live-birth neural tube defects (spina bifida cystica and encephalocele) born in the third and fourth quarters of 1989 and the first quarter of 1990 who were referred for treatment within the Jamaican hospital system. All cases of neural tube defects were clinically confirmed in tertiary institutions. Anencephaly was excluded because such cases are not usually referred and reports of this diagnosis could not be confirmed. No record of an anencephalic live birth or stillbirth was available from the main hospitals nor from the Ministry of Health. The recording of such birth defects is known to be deficient.[12]

Each case subject was matched with three control subjects, mothers of babies with a birthweight of >2.5 kg and without any apparent physical or developmental defects. Case and control subjects were matched for maternal age, month and year of conception, periconceptional geographic location (Figure 1), parity, and educational level. All of the subjects were of similar socioeconomic and ethnic background (African descent).

A 56-item questionnaire was used in a structured personal interview of each of the case and control subjects by the sole investigator (E.M.W.D.). Blinding of the identities of the case and control subjects was not feasible. Each interview was completed within 11–18 months of the subject baby's birth. The questionnaire was developed, pretested, and precoded to explore 32 health, environmental, and dietary variables in the postdisaster periconceptional period of each subject's pregnancy (see Appendix). Question 55 consisted of a "food folate content scale" that listed 34 common food items and their estimated folate content, designed to give an approximate indication of the subject's daily intake of free folate (in micrograms) in that period for comparison between case and control subjects. This scale was developed from food composition tables[13] and prorated for local dietary servings.

The authors are with the Tropical Metabolism Research Unit, University of the West Indies, Jamaica, West Indies.

Requests for reprints should be sent to E. Marilyn Watson Duff, MScNutr, BScN, Tropical Metabolism Research Unit, University of the West Indies, Mona, Kingston 7, Jamaica, West Indies.

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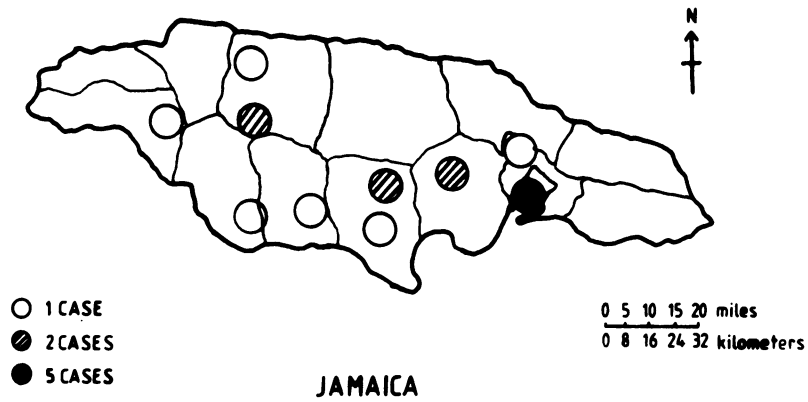
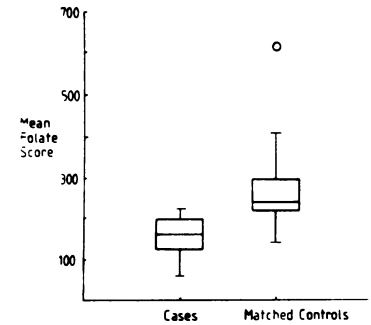
Editor's Note. See related annotation by Beresford (p 348) in this issue.



Note: Each circle has a diameter of approximately 8 kilometers.

FIGURE 1—The periconceptional geographic locations in Jamaica of the case subjects and their matched control subjects.



Note. The high outlying score was that of the only subject (control) who took a daily supplement.

FIGURE 2—The mean folate intake scores (micrograms/day) for the two groups, the upper and lower limits, and the upper and lower quartiles of the two distributions.

TABLE 1—Mean Daily Folate Intake Reported in the Periconceptional (Post-Hurricane) Periods of Neural Tube Defect Case Subjects and Control Subjects

Group	No.	Mean ± SD Daily Folate Intake (μg)	95% CI
Case subjects	17	153.6 ± 49.3	123, 185
Control subjects	50[a]	253.9 ± 64.3	233, 275

Note. $t = -5.86$; $df = 65$; $P < .0001$.
[a]One high outlying score was omitted from analysis.

The periconceptional period was defined as an 18-week period starting 12 weeks before conception and ending 6 weeks after conception.

Information on postdisaster food availability and relief supplies was obtained from the Salvation Army, the main relief agency.

The mean folate scores of group 1 (case subjects) and group 2 (control subjects) were compared by using the *t* test for independent samples. Fisher's Exact Test and the corrected chi-square test were also used where applicable. Possible confounding variables such as minor trauma and oral contraceptive use were controlled for with multiple logistic regression analysis.

Results

The mean daily dietary intake of folate was significantly lower ($P < .0001$) in group 1 (case subjects) (154 μg/day, 95% confidence interval [CI] = 123, 185) than in group 2 (control subjects) (254 μg/day, 95% CI = 233, 275) (Table 1, Figure 2). The range for group 1 was 60–220 μg/day. The range for group 2 was 140–410 μg/day with a high outlying intake of 616 μg/day, that of the only mother (control subject) who in addition to a dietary intake of 445 μg/day supplemented herself with a multivitamin "liquid food" preparation containing folic acid (approximately 170 μg/day). For each case subject the mean folate intake of the matched control subjects was higher (Figure 3).

There was no association with maternal smoking; alcohol, marijuana, or other substance abuse; pesticide exposure; intake of previously suspected teratogenic foods (i.e., ackee,[14] potato,[15] cassava, Jamaican white yam[16]); bush teas; pica; sources of drinking water; clinic attendance; environmental or psychosocial stress; hyperemesis; hypertension; spacing of pregnancies; incestuous parenting; or previous birth defects.

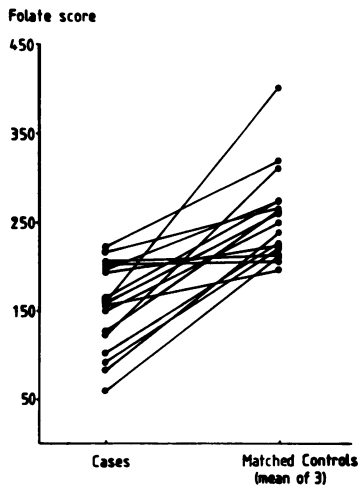
There was no apparent relationship with maternal medication use, but oral contraceptive use[17] was higher among the case subjects, and this difference approached statistical significance (odds ratio [OR] = 3.8, 95% CI = 0.95, 15.44, $P = .058$).

No previous birth defects were reported by any of the subjects in either group. Stillbirths were reported to have occurred in previous pregnancies of two of the case subjects and none of the control subjects. Trauma (minor trauma from blows and falls) was reported only by subjects in group 1 (OR = 5.3, 95% CI = 3.16, 8.73, $P < .0005$). There was no apparent explanation for this.

Population fertility (live births) increased by 4.3% over the same months of the previous year (Statistical Institute of Jamaica, 1988–1990, unpublished data). Although there was no general shortage of food reported by the subjects, there was a change in eating patterns at that time, towards the use of prepared and packaged foods (relief supplies consisting of rice, flour, sugar, cornmeal, tinned corned beef, sausages, sardines, and mackerel) with a considerably reduced intake of fresh produce.

Discussion

We recognize the limitations of retrospective data and a study in which blinding of case and control identities was not practicable, but the special opportunity presented by this experiment of nature mandated the investigation.



Note. In each instance the mean folate intake of the three matched control subjects was higher than that of the case subject.

FIGURE 3—The dietary folate intake scores (micrograms/day) of each case subject compared with the mean dietary folate intake scores of the three matched control subjects.

Within these limitations, the results show that this post-Hurricane Gilbert increase in neural tube defects was associated with a diet comparatively low in folate in the periconceptional period.

The range of reported daily dietary folate intakes among the case subjects (60–220 μg/day) and among the control subjects (140–410 μg/day) gives an approximate indication of the folate content (i.e., micrograms of free folate daily) of the individual diets of the mothers in their post-hurricane periconceptional periods. From this result it may be suggested that a dietary intake of <220 μg of folate per day in the periconceptional period may be inadequate to prevent neural tube defects in the babies of Jamaican mothers. No estimate of the converse, the intake that is protective, can come from a case–control study such as this one.

The MRC Vitamin Study Research Group suggested that the United Kingdom may have a lower folic acid intake than other countries.[1] The usual daily mean intake of folate for British women is 209 μg/day.[8]

The U.S. recommended daily allowance for folate is 180 μg/day for nonpregnant women and 400 μg/day during pregnancy. In the 1976–1980 US Health and Nutrition Survey, median folate intakes were 166 μg/day and 146 μg/day for White and Black women, respectively.[6]

Scott et al.[8] and Leeming[4] emphasized the danger of exposure of the developing embryo to circulating unmetabolized folic acid arising from high-dose supplementation. They recommend that a woman contemplating pregnancy could avoid neural tube defects in her baby by either taking a low-level folic acid preparation or eating an improved or even fortified diet high in folic acid. Strieff[18] suggested that orange juice was a good dietary supplement for folic acid. This suggestion was endorsed by Lucock et al.,[3] who demonstrated in their experiments an enhanced absorption and metabolism of folic acid after ingestion of 500 mL of pure orange juice.

In view of possible adverse reactions to high-dose folic acid supplementation and the recognized interrelation of nutrients in a varied diet adequate in folic acid and related nutrients (such as ascorbate, pyridoxine, and cobalamin), it has been suggested that dietary sources of folate may be preferable to high-dose supplementation.[7]

The Jamaican diet usually includes a wide variety of tropical fruits and vegetables throughout the year. Many of these (e.g., ripe bananas and oranges) contain appreciable amounts of folate and ascorbate. Chicken and beef liver and kidney, eggs, callaloo (a local vegetable similar to spinach), avocados, and steamed rice are also popular in the Jamaican diet. That may account for the usually low incidence of neural tube defects in the Jamaican population.

The massive destruction of crops and livestock wrought by Hurricane Gilbert had immediate impact on the diets of the people of Jamaica. Fresh produce was scarce and therefore expensive.[11] Many of the mothers reported returning to their usual diets 3 to 4 months after the hurricane. □

Acknowledgments

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(Appendix on next page)

APPENDIX—List of Variables Investigated by the Structured Personal Interview

1. *Personal information.* Subject's name, address, age, and educational level; occupations of subject and spouse; socioeconomic status (by employment skills rating); family structure; smoking; medication; substance use and abuse.
2. *Antenatal care.* Parity, previous stillbirths and/or birth defects, contraceptive use, spacing, general health, illnesses, accidents, results of diagnostic tests, medication, supplements, clinic attendance.
3. *Index baby.* Name; sex; date of birth; gestation; birth weight; month, year, and place of conception; place and type of delivery; health status at birth; admission(s) to hospital, diagnosis, treatment, and outcome.
4. *Hurricane experiences.* Subject's personal experiences in the aftermath of Hurricane Gilbert. Physical and psychosocial stress, damage, relocation, available food, drinking water.
5. *Periconceptional period.* Nutritional status (as described by the subject), supplements, medications, oral contraceptive use, illnesses, accidents, smoking, substance use and abuse, use of "bush teas," exposure to pesticides, consumption of previously suspected teratogenic foods (i.e., potato, Jamaican white yam, ackee, and/or cassava), pica, unusual stressful events. Exploration of periconceptional diet, determination of the "folate score."

The Use of Commercial Tanning Facilities by Suburban Minnesota Adolescents

Jennifer A. Oliphant, MPH, Jean L. Forster, PhD, MPH, and Colleen M. McBride, PhD

ABSTRACT

In 1991, 1008 suburban St. Paul, Minn, high school students were surveyed via self-administered questionnaire regarding use of commercial tanning facilities, injuries experienced from tanning, use of protective measures while tanning indoors, and knowledge of the risks of tanning. Overall, 34% of the respondents had used commercial tanning facilities. Fifty percent said they had not been warned by tanning facility operators about the risks of tanning indoors, 28% reported not being told to wear goggles, and 17% reported never wearing goggles. The results indicate that these adolescents use commercial tanning services at high rates, and often in ways that increase their risk for a variety of health problems. (*Am J Public Health.* 1994;84:476–478)

Introduction

Exposure to ultraviolet radiation, such as that from artificial tanning devices, is a risk factor for a variety of health problems including erythema, vesiculation, photosensitizing reactions, actinic elastosis, keratoses, basal cell and/or squamous cell carcinoma, and malignant melanoma.[1–3] Exposure to ultraviolet radiation can also result in compromised immune response, eye burns, and cataracts.

The risk of many health problems is higher when ultraviolet radiation exposure occurs prior to adulthood.[4] The damage begins with a child's first exposure and accumulates through his or her lifetime.[5,6] It is estimated that up to 78% of the risk from ultraviolet radiation exposure is completed by age 18.[7]

Indoor tanning is not safer than tanning in natural sunlight. Commercial tanning devices emit either mostly ultraviolet A or mostly ultraviolet B.[2,8,9] Although ultraviolet B injures the skin faster, ultraviolet A penetrates the skin more deeply.[9,10] Some commercial tanning beds may emit as much as 10 times more ultraviolet A than natural sunlight.[2]

Artificial tanning produces no known health benefits,[3] nor is it thought possible to tan safely.[6] Exposure to ultraviolet radiation from indoor tanning increases the chances of developing cancer from natural sunlight.[11,12] The sun protection factor from tanning indoors is only about 4, which offers no significant protection from sun exposure.[13] Moreover, severe ultraviolet radiation burns (those that cause pain for 48 hours or more[14]) increase the risk for malignant melanoma.[4,15]

Despite the serious risks associated with artificial tanning, fewer than half of the states currently regulate commercial tanning facilities. The US Food and Drug Administration (FDA) regulation requires only that the device be equipped with a calibrated timer, protective eyewear, and a small warning label.[16] A previous study found that 45% of all commercial tanning devices examined

Jennifer A. Oliphant is with the Johnson County Department of Public Health, Iowa City, Iowa. Jean L. Forster is with the University of Minnesota School of Public Health, Minneapolis. Colleen M. McBride is with the Center for Health Studies, Group Health Cooperative, Seattle, Wash.

Requests for reprints should be sent to Jean L. Forster, PhD, MPH, University of Minnesota School of Public Health, Division of Epidemiology, Suite 300, 1300 S 2nd St, Minneapolis, MN 55454-1015.

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