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Correction

Effect on lipoprotein profile of replacing butter with margarine in a low fat diet: randomised crossover study with hypercholesterolaemic subjects

Two errors occurred in table 4 of this paper by Alexandra Chisolm *et al* (13 April, pp 931-4). The mean percentage fatty acid composition of C20:4n-6 on the margarine diet should be 0.80 [not 0.08 as published], and C16:0 on the margarine diet needs to have an asterisk to indicate it is significantly different from C16:0 on the butter diet. The authors would also like to express their thanks to Sarah Bethell and Steve Tiszavari for their excellent technical help.

Population mixing and the incidence of childhood leukaemias: retrospective comparison in rural areas of New Zealand

John D Dockerty, Brian Cox, Barry Borman, Katrina Sharples

In 1988 Kinlen hypothesised that childhood leukaemia occurs as a rare response to a common infection and that population influxes into rural areas with higher proportions of susceptible individuals favour epidemics of the infection and increases in the incidence of leukaemia.¹ Most ecological studies of childhood leukaemia in relation to this issue have supported the hypothesis.² Our study was conducted to test whether population influxes into rural areas of New Zealand were associated with increases in the incidence of childhood leukaemias.

Subjects, methods, and results

Forestry developments, including the building of a pulp and paper mill, occurred in the adjacent Rotorua, Matamata, and Whakatane areas of New Zealand's North Island in the 1950s. These developments led to population growth and the establishment of the new towns of Kawerau and Murupara. In this study boundaries were defined for the Rotorua, Matamata, and Whakatane areas (study areas) based on local government divisions. Care was taken to ensure that the areas remained as equivalent as possible over the years. The population of the combined study areas increased greatly at the time of the forestry developments compared with a smaller increase in the rest of New Zealand (fig 1). Similar increases occurred in each individual area (Rotorua, Matamata, and Whakatane).

Childhood leukaemia registrations for New Zealand residents (1949-83, ages 0-14) were obtained from the

National Cancer Registry. On the basis of the street address at diagnosis (or occasionally the domicile code) each child was classified as living in one of the study areas or in the rest of New Zealand. Quinquennial age specific incidence rates were calculated for childhood leukaemias in the study areas (combined) and in the rest of New Zealand. Age adjusted incidence rate ratios (Mantel-Haenszel method³) with 95% confidence intervals were also calculated. To assess differences among the trends in the age specific rates we used the likelihood ratio test for heterogeneity.³

Fig 1—Rate ratios for childhood leukaemia (ages 0-14, study areas versus the rest of New Zealand). Middle years of the time periods were census years, and population increases are average annual increases for all ages since previous census

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During 1949-83 there were 1134 registrations for childhood leukaemias. The age adjusted rate ratios (study areas versus the rest of New Zealand) were not significantly raised during or after the greatest influx (1951-6; fig 1). For ages 0-4, 5-9, and 10-14 the rate ratios were under 1.8, and none was significantly raised. There was no significant heterogeneity in the rate ratios between time periods for any age group, but for ages 5-9 and 10-14 there were few registrations in the study areas. Subsequently we ran analyses for the 15 years 1949-63. The age adjusted rate ratio was 0.84 (95% confidence interval 0.50 to 1.44). The age specific rate ratios were: 0.92 (0.45 to 1.87) for ages 0-4 years, 0.97 (0.40 to 2.37) for ages 5-9, and 0.37 (0.06 to 2.43) for ages 10-14.

The study areas were very rural in the early 1950s, but prior population mixing would have occurred in Rotorua through tourism. Nevertheless, the age adjusted rate ratio (1949-63) was not raised in any of the three areas (including Matamata and Whakatane, where tourism was negligible).

Comment

This study does not provide support for Kinlen's hypothesis, and there are several possible explanations. There were few affected children in the study areas, so a small effect might have been missed. Bias may have been introduced by unmeasured confounders. Community size and population density are important in deter-

mining whether an infectious disease will maintain itself,⁴ and differences between the communities in this study and those studied in the United Kingdom may have produced differences in transmission of infection and different results. The relevant infectious agent may even have been absent from New Zealand in the 1950s. Alternatively, the reported relation between population mixing and childhood leukaemia could have non-infective explanations.⁵ Leaving speculation aside, the only firm conclusion of this study is that it does not support the population mixing hypothesis.

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Rupture of the uterus: a review of 32 cases in a general hospital in Zambia

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Rupture of the uterus is a major cause of maternal and perinatal deaths.¹ This study aimed to identify the risk factors associated with uterine rupture and to determine maternal and fetal outcomes.

Subjects, methods, and results

Between 1 July 1993 and 30 June 1994, 32 cases of uterine rupture were recorded at Mansa General Hospital, Zambia. This 412 bed secondary care centre receives referrals from hospitals, dispensaries, and health centres within Luapula province (population 160 000). During the study period there were 1858 deliveries, including 98 caesarean sections.

Table 1 shows the age and parity of women with uterine rupture. Age ranged from 15 to 46 years (mean 23 (SD 7) years), with a significantly higher proportion of teenagers than among all women giving birth (12/32 (38%) v 321/1858 (17%); $\chi^2=8.86$, df=1, $P<0.01$). Parity ranged from 1 to 11 (mean 2 (SD 2)). The study group contained a higher proportion of nulliparous women (14/32 (44%) v 501/1858 (27%); $\chi^2=4.4$, df=1, $P<0.05$) but not multiparous women (14/32 (44%) v 778/1858 (42%)) than overall.

Twenty patients with uterine rupture (63%) had not been booked for delivery in hospital. Rupture occurred during labour in 30 cases (94%). Rupture was associated with cephalopelvic disproportion in 24 cases (75%). Three women had previously had a caesarean section. Two ruptures occurring in hospital were due to oxytocic stimulation and assisted breech delivery with undiagnosed mild hydrocephalus. Postmortem examination of two patients who arrived in moribund condition showed a macerated fetus with its placenta outside the uterus in the abdominal cavity in one and a complete rupture in the lower uterine segment complicated with bladder injuries in the other.

Laparotomy (performed in 30 cases) showed complete uterine rupture in 21 patients (70%), located in the lower uterine segment in 16, and bladder injury in seven (23%). Procedures were carried out in 30 cases: subtotal hysterectomy in 14 cases (47%); caesarean section with uterine repair in eight (27%); uterine repair only in eight. Bladder injuries were repaired in five cases.

Postoperative mortality was high (14/32; 44%) and mainly due to sepsis (11 patients; 79%). Higher mortality was associated with nulliparity (79%), teen age (71%); and anaemia, sepsis, and shock on admission (64%). Sepsis and maternal mortality increased with time since the rupture and the distance between hospital and the patient's home or referring centre. Postoperative mortality was similar for patients booked or not (5/10 (50%) v 11/20 (55%)). One late complication, a vesicovaginal fistula, was due to bladder necrosis caused by prolonged fetal head compression before operation.

Mortality did not differ significantly in patients who underwent hysterectomy and those treated by uterine repair (5/14 (36%) v 11/16 (69%); $\chi^2=3.28$, df=1, $P>0.05$). Fetal mortality was 100%.

Comment

Because of inaccessibility of essential health information, lack of transportation, poor access to fixed health care services, and shortcomings in the supervision and monitoring of home deliveries, uterine rupture is common in rural and illiterate women.^{2,3} Those most

Table 1—Age and parity of women with uterine rupture, 1 July 1993 to 30 June 1994, Mansa General Hospital, Zambia

Age (years)	Parity										
	0	1	2	3	4	5	6	7	8	9	10
15-19	12										
20-24	1	3	3	2	1						
25-29	1	1	2	1							
30-34						1	1				
35-39										1	1
≥40							1				

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