cross-reactivity with an arthropod-borne virus as the presence of GBV-C RNA had the same association with possible arthropod exposure.

Our findings suggest a possible vector-borne transmission of GBV-C.

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Folate deficiency, neural tube defects, and cardiac disease in UK Indians and Pakistanis

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In maternity units in the North Thames (West) Region, UK, there were some 47 500 births plus terminations for fetal abnormality in 1996. Data from the Congenital Malformation Register suggested to us that there was a higher rate of pregnancies with neural-tube defects in women of Indian or Pakistani origin. It is unlikely that this disparity is entirely the result of reporting bias as similar trends have been reported elsewhere.[1]

Folate deficiency in women of childbearing age increases their risk of having a pregnancy affected by a neural-tube defect.[2,3] We measured red-cell folate in successive blood samples from 61 healthy Indian or Pakistani women and 131 other women of child-bearing age (16–50 years) in North Thames (West) region in 1996 (immunoassay, Abbott IMX, normal range 150–645 ng/mL). Indian and Pakistani women had significantly lower concentrations of red-cell folate (mean levels and range of 246 [SD 87] *vs* 286 [160] ng/mL, $p<0.02$ with independent samples *t* test, as both data sets were normally distributed).

Coronary heart disease (CHD) is a common cause of death in those of Indian or Pakistani origin aged over 30 years in this region. In a study aimed at testing the association of hyperhomocysteinaemia with premature CHD in a randomly selected healthy male cohort, we found—with the same assay—that red-cell folate was significantly lower in 200 Indian and Pakistani men compared with 200 age-matched other men, aged between 35 and 60 years (mean 363 [128] *vs* 409 [147] ng/mL, $p<0.001$; both data sets normally distributed).

Red-cell folate is determined by genetic and nutritional factors: a twin study has suggested that the genetic contribution to the phenotypic variance in red-cell folates was approximately 46%.[4] Population cohort studies show that 5–15% of Europeans have a genetic predisposition to lower concentrations of folate by virtue of mutations in their folate-pathway genes.[5] Such genetic predispositions increase the risk of both neural-tube defects and cardiovascular disease. The frequency of a common mutation (677 C→T) in the methylenetetrahydrofolate reductase (MTHFR) gene was lower in Gujerati women compared with women of UK or Irish origin in a preliminary local study. It is therefore unlikely that this mutation accounts for the low folate concentrations we describe. By contrast, dietary deficiency in Indian and Pakistani populations in the UK has been well documented.[2,5]

Urgent work is required to determine whether the trends identified in North West Thames are the result of a higher prevalence of mutations in genes which modulate folate metabolism, or of a deficient folate intake, or of a combination of these. There is an additional duty to educate Indian and Pakistani women of childbearing age about the need for folate supplementation, and to investigate the merit of folate supplementation as a cardioprotective measure.

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Cerebrospinal-fluid τ protein and aspartate aminotransferase in Parkinson's disease

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There is still no diagnostic test or biological marker for Parkinson's disease (PD) based on biochemical analysis of blood or cerebrospinal-fluid (CSF). Riemenschneider et al[1] studied τ protein concentration and aspartate aminotransferase (AST) in the CSF of patients with neurodegenerative diseases, and suggested that the combination of increased CSF τ and AST activity is a specific biological marker for Alzheimer's disease (AD). Mental impairment is a frequent complication of PD. Alzheimer-type neuropathology (ATP) is increased in the brains of people with PD, and the frequency of clinical dementia has been estimated by Jendroska et al to be 46%.[2]

We studied CSF τ and AST in 115 patients (40 women) with PD, mean age 62 (SD 8·6 years). All had a lumbar

Marker	Controls (n=16)	Parkinson's disease	
		With dementia (n=48)	Without dementia (n=67)
τ (ng/L)	155 (8·6)	204 (21·4)	199 (24·0)
AST (U/L)	4·9 (0·6)	4·9 (0·4)	4·8 (0·3)

All values are mean (SE).

τ protein concentration and AST activity in patients with Parkinson's disease and controls

puncture. 48 had a Mini Mental State Exam (MMSE) score of 25 or less at the time of sampling. The control group was 15 patients (seven women; mean age 58 [SD 10·2] years) who had a lumbar puncture because of other suspected diseases but who did not have dementia. Routine CSF analysis was normal in patients and controls. The concentration of phosphorylated and unphosphorylated τ protein was measured by ELISA (Innogenetics, Zwijndrecht, Belgium), and AST activity was determined according to International Federation of Clinical Chemistry recommendations at 30°C with a Hitachi 717 analyser (Boehringer Mannheim, Mannheim, Germany) (table). We found no difference between patients with PD who had dementia and those who did not (table). CSF τ and AST activity did not correlate with MMSE scores, duration of symptoms of PD, duration of cognitive decline, age, or sex.

These preliminary findings suggest that τ and AST concentrations are not sensitive markers in PD.

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Motion sickness, migraine, and menstruation in mariners

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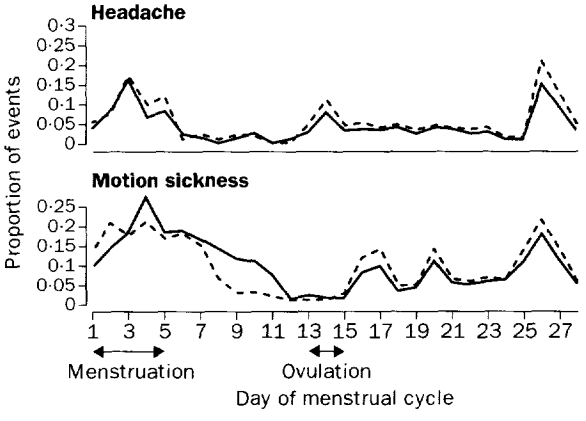
Women have a higher incidence of migraine than do men and their attacks are often related to the menstrual cycle. Women are also slightly more prone to motion sickness[1] and it has been suggested that their susceptibility may follow endocrine rhythms.[2] Regardless of sex, evidence suggests a higher incidence of motion sickness amongst migraineurs,[3] implicating a common mechanism, but the issue is clouded by similarities in their symptoms. Occasional individuals are triggered into attacks of headache and vomiting, a day or more in length, by limited exposure to motion such as short car rides; this is more than simple motion sickness. Studies of motion sickness have overlooked the problem of diagnosis when it is provoked by vehicle motion, particularly in those at risk from migraine. We sought to clarify these issues by studying symptoms in the crew of the 1997 British Telecom "Global Challenge". The race has six "legs" varying from 8 to 45 days and, despite partial habituation, many sailors are sick at intervals throughout because of exposure to conditions of extreme motion sickness provocation. The prolonged repetitive challenge to motion sickness susceptibility permits the study of interrelations between headache, motion sickness, and hormonal cycles.



Headache and motion sickness through the menstrual cycle
The results are presented for 17 women who had either motion sickness or migraine during the race (solid line) and for 12 women who had both motion sickness and migraine during the race (dotted line). A calculation was made of the number of days, for each day of her menstrual cycle, that the woman had been aboard the yacht throughout the race. The data were examined to identify on how many of these days she had had either a headache or motion sickness. For each day of the cycle the number of days where events occurred was divided by the total number of days observed. The results were averaged to give the proportion of events shown in the figure.

In the pre-race week 111 sailors completed the Reason Motion Sickness Questionnaire[4] and a headache incidence and classification questionnaire based on the International Headache Society Diagnostic Guidelines.[5] The 34 women also indicated the regularity of menstruation, its relation to headache and use of oral contraceptives. During the race, sailors were requested to enter headache, motion sickness symptoms, and menstruation in daily logs; 25 men and 27 women complied.

In pre-race questionnaires, headache was reported by 97% of women (45% classified as migrainous) and 69% of men (23% migrainous) without motion sickness being an important trigger. Female (40·9) and male (48·1) motion sickness susceptibility scores were not significantly different and were unrelated to headache.

At sea, 12 sailors (nine women) had migraine, averaging five attacks each. Only eight of these had reported migraine before sailing. A further six fulfilled the migraine criteria on their pre-race questionnaire but did not experience migraine during the race. Those who had migraine during the race had more frequent motion sickness (averaging 13 events compared with six for non-migraineurs; p=0·0098) and non-migrainous headache (7·5 headaches compared with three; p=0·0039). Pre-race reports of migraine did not predict motion sickness but were highly correlated with all types of headache at sea. A women with incapacitating headaches related to rough seas was treated effectively with sumatriptan by the race doctor (CP).

Female crew were most susceptible to motion sickness from 3 days before menstruation to day 5 (figure). Frequency of headache of all kinds was also greatest during this phase of the cycle. Motion sickness susceptibility was least around the time of ovulation in contrast to headache when a further peak was observed (figure). Motion sickness susceptibility in males appeared to follow a 4–5 day cycle but more data would be needed to confirm this trend. Their headaches were not apparently cyclical.

The study has demonstrated that the occurrence of motion sickness and headache in most sailors were independent although exposure to rough seas could be a migraine trigger in certain individuals who did not otherwise have attacks. When motion sickness or headache occurred in female sailors they were related to the menstrual cycle.

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