antibody titre concentrations in the workforce were no different from those found in the general population. To study the ecology of *Legionella* in power stations the CEGB sampled water from 17 power stations every month through the summer of 1983. Four of these stations were sampled more intensively in 1984.

A total of 335 samples were taken from various parts of the cooling water system; 205 of these were taken from the condenser outlet (the warmest part of the system) and the cooling tower ponds. *Legionella* was isolated from eleven of the power stations sampled, the organism being present in low concentrations in 35% of the 205 samples of the circulating water. Six of the fourteen make-up sources of water tested were positive.

In all but 3 of the 205 samples the numbers of *Legionella* were very low. 49 of the positive samples contained fewer *Legionella* than found in the make-up water with the highest number, and most were at the limits of detection. The 3 samples with high numbers were obtained on isolated occasions from two stations and the numbers were still too low for the water to be regarded as possible sources of infection.

After the outbreak of legionnaires' disease in Stafford in April/May, 1985, suggestions were made in the press and on television that one or more of the CEGB's cooling towers could have been a source of the infection. The CEGB initiated a sampling programme of power stations countrywide, including some power stations which operate only rarely. Thirty-three power stations, all operating on inland rivers and using cooling towers were studied. *Legionella* was found in one river sample and in nine of the thirty-three stations sampled, 20% of the samples were positive; none of the samples from the other twenty-four stations was positive. On all occasions the concentrations were low and rarely above background levels, which confirm the findings in the 1983/84 programme. The results of these surveys, which will be published in full elsewhere, generally showed only an intermittent presence of *Legionella* in low numbers in power station waters. The numbers of organisms found cannot be considered a health risk and there is no requirement to modify current power station operational procedures.

Further work is in hand to follow the seasonal patterns of bacterial growth and to identify possible sites of multiplication of *Legionella* within a power station and to ensure that the ecology of the organism in the cooling water system is fully understood. Additionally, a national surveillance programme will be established to monitor continuously the situation countrywide.

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DOES ISOTRETINOIN CAUSE LIMB REDUCTION DEFECTS?

SIR,—Dr McBride (June 1, p 1276) describes a child with limb reduction defects whose mother took isotretinoin ('Accutane') during the pregnancy. Several important aspects of this case are missing from McBride's letter.

McBride examined the child in the capacity of a professional witness in litigation proceedings. When he saw the patient, corrective surgery had already been done on both hands, so his assessment of the affected hands was largely based on photographs. One of us (E. J. L.) examined the child before surgery. Both hands had complete syndactyly of all four fingers with absent middle and distal phalanges. McBride incorrectly stated that there were no fingers on the right. The syndactylous left fingertips had a ring-like circumferential constriction with four terminal bulbous tips grouped in a grape-like cluster distal to the constriction. The appearance was very suggestive of a circumferential constriction and amputation from an amniotic band.

McBride fails to mention that four expert witnesses concluded that the child's limb reductions were probably caused by amniotic bands. This conclusion was based on the following points: (1) one of the child's physicians reported that, during the newborn period, he had removed a band of tissue that was attached to one of the malformed limbs; (2) several of the malformed limbs had circumferential constrictions without complete amputation; and (3) the child's pattern of limb reductions did not resemble that produced when retinoic acid was given to laboratory animals at the comparable time in gestation. The child's limb reductions were all distal with normal proximal bones; in animals, retinoic acid usually causes more proximal limb deficiences when given before or during the initial appearance of the upper limb bud.

McBride speculated that isotretinoin may have caused limb reductions by damaging the apical ectodermal ridge of the developing limb bud. This is inconsistent with a large volume of experimental data showing that retinoic acid does not affect the cells of the apical ectodermal ridge.[1]

McBride incorrectly stated that the child's mother had taken isotretinoin for 14 days. She reported that she had taken it for only 7 days.

Isotretinoin is a human teratogen. It may cause limb reductions in human fetuses, but we do not think that isotretinoin caused the malformations in this case. The evidence that this child's limb reductions resulted from amniotic bands was very convincing, yet none of this information was provided in McBride's letter. McBride's report is misleading: he fails to provide the reader with the information necessary to judge whether a relationship was possibly causal. The following advice from the thalidomide era is still valuable, "Let us be on the alert by all means and report our observations, but we must be critical, because many pregnant women and their husbands are unnecessarily upset by uncritical accusations of drugs as being the cause of all congenital abnormalities."[2]

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1 Kochnar DM. Cellular basis of congenital limb deformity induced in mice by vitamin A. *Birth Defects* 1977; **13** (1). 111–54.
2. McBride WG Drugs and congenital abnormalities. *Lancet* 1962, ii· 1332

OESOPHAGEAL ULCERATION AND THEOPHYLLINE

SIR,—I wish to report a local rather than a systemic[1] complication of self-poisoning with theophylline.

A 31-year-old female who believed she was beginning to have one of her occasional bronchial asthma attacks as she was preparing for bed took a slow-release theophylline tablet ('Phyllocontin 350') while recumbent, without any accompanying fluid. She slept well that night. Since she had been entirely well before this episode, she had not been taking any other drugs. When she awoke the next morning she noticed a retrosternal burning pain, which became excruciating when she tried to swallow breakfast juice. After about 8 h she crushed one aspirin tablet in water, but because of the pain was able to swallow only about half the mixture. She was unable to take anything (including water and patent antacids) orally that day because of pain and vomiting. Her pain was felt behind the sternum in the right breast and in the back on the right side. She slept poorly. The next morning she consulted her family physician, and a barium meal revealed no abnormality. The pain continued to be exacerbated by swallowing but was partly controlled by a great deal of anaesthetic gel. Within a few days she noticed that she was losing weight and becoming dry.

When I saw her 9 days after the pain began, she was looking dehydrated and ill. At upper gastrointestinal endoscopy with a flexible fibreoptic endoscope the next day I found, at 34 cm from the incisor teeth, a 2 cm long oesophageal ulcer which spared only the right lateral side of the oesophagus. The ulcer was deep, nodular at the edges, and had some shaggy areas in its base. Distal to the ulcer the oesophagus was completely normal, as were the stomach and duodenum. Unusually, the patient experienced extreme pain when biopsy and cytology (brush) samples were being taken. The pain subsided slowly over the next week, during which the patient took large amounts of liquid antacids. Histology showed acute non-specific ulceration with granulation tissue. Re-endoscopy 20 days later, when the patient no longer had symptoms, showed normal oesophageal mucosa.