TABLE II.—*Methaemoglobin Formation from Nitrates and Nitrites in Water*

Date	Subject	Total Hb % of Normal (Haden) (100%= 15·6 g./100 mL)	Methaemoglobin as % of Total Hb	Drinking-water Supply
4/7/53	Baby (cyanosed)	93	56·8	Well
8/12/53	,, (cured)	80	Nil	Town
,,	Child (aged 2)	75	16·7	Well
,,	,, (,, 3)	70	9·5	,,
,,	Mother	77	9·5	,,
11/12/53	Father	90	Nil	Well-water at night; meals out during the day

described is applicable to the Evelyn–Rubicon photometer, whereas the instrument used here is similar to that described by Salt (1950), fitted with a Tinsley galvanometer. An instrument-correction factor was therefore worked out by means of known solutions of methaemoglobin, and has been applied to the values quoted.

Comment

It can be seen that those members of the family whose water supply was derived entirely from the well all showed levels of methaemoglobin far above the upper limit of the normal range for this method of estimation. The baby, who had been receiving town water for a matter of four months, and the father, who fed out at canteens during the day, both gave negative results.

This short study emphasizes that where a family are relying on a water supply of high nitrate or nitrite content a young baby is more prone to show the effects of methaemoglobinaemia than its elders, as its daily intake of the water, on a weight-for-weight basis, is much larger. Symptoms may be precipitated by the onset of a pyrexial illness, increasing its oxygen demand. Also, if a baby from a rural area presents with a cyanosis unexplained by physical examination, investigations of the water supply may prove to be time well spent.

We are grateful to Mr. M. M. Love, county analyst, for kindly carrying out the water analyses for us.

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Changes in the law governing the production and supply of milk in Scotland come into force on October 1. The changes represent further steps towards securing a safe and clean milk supply. Bottles of pasteurized milk will have to be fitted with caps which seal the lip of the bottle, and bottling will have to be done on the premises where pasteurizing takes place. This is to prevent milk being handled after it has been pasteurized. The special designation "standard" will not be recognized after September 30. In "specified areas," which include all cities and large burghs and many small burghs and landward areas, the sale by retail of standard milk will be forbidden. The only grades of milk allowed to be sold in these areas will be certified, tuberculin tested, pasteurized, and sterilized. A producer's licence to use the special designations certified or tuberculin tested will be granted from October 1 only if the herd from which the milk comes is attested or is in a tuberculosis eradication area of the Ministry of Agriculture and Fisheries. The effect of this is that certified and tuberculin tested milk will in future come from farms where all the cattle, and not only the milk cattle, are certified to be free from bovine tuberculosis.

STAPHYLOCOCCAL FOOD-POISONING DUE TO INFECTED COW'S MILK

BY

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Although *Staph. pyogenes* is commonly found in cow's milk, outbreaks of food-poisoning arising from such a source are relatively very rare. This fact also makes a definite diagnosis difficult, since the isolation of these bacteria from milk or milk products during the investigation of an outbreak of food-poisoning does not imply that they are the cause of it.

This account of two small related outbreaks of food-poisoning is not without interest in that the infective agent, clotted cream, was prepared from milk from a cow that was found to be persistently excreting *Staph. pyogenes* in her milk. Furthermore, the staphylococcus was of a type rarely found in bovine infection, that type being predominantly a human pathogen.

The Outbreaks

On January 9, 1954, notification was received that a small outbreak of food-poisoning had occurred on the previous evening. Seven persons in one family were involved (Table I), and the suspected food was home-made clotted cream. The cream was eaten at 4.30 p.m. and was followed in six cases, in three to three and a half hours, by the abrupt onset of nausea, abdominal pain, and vomiting followed by diarrhoea. In the seventh case symptoms were confined to nausea and general malaise. No case required admission to hospital, but all except the seventh patient were incapacitated for a period varying from six to forty-eight hours.

TABLE I.—*Persons Involved in Outbreak on January 8, 1954*

	Age	Onset	Duration	Nausea	Vomiting	Diarrhoea
Mr. S. ..	67?	4 hours	3 hours	+	−	−
Mrs. S. ..	48	3 ,,	6 ,,	+	+	+
Mrs. B. ..	45	3–3½ ,,	48 ,,	+	+	+
John B. ..	12	3½ ,,	12 ,,	+	+	+
Mrs. G. ..	73	3½ ,,	24 ,,	+	+	+
Miss H. ..	74	3½ ,,	24 ,,	+	+	+
Miss C. ..	66	3½ ,,	20 ,,	+	+	+

Further investigations revealed that clotted cream prepared from the milk of the same cow was eaten without ill effect on December 26 but that a similar incident had occurred on January 2 following consumption of the cream by another family living on the same premises (Table II). The cream was prepared on all occasions in a similar manner, on January 2 by Mrs. S. and on January 8 by Mr. S.

TABLE II.—*Persons Involved in Outbreak on January 2, 1954*

	Age	Onset	Duration	Nausea	Vomiting	Diarrhoea
Mr. W. ..	55	3 hours	30 hours	+	+	+
Mrs. W. ..	57	2 ,,	30 ,,	+	+	+
Mrs. H. ..	59	3 ,,	30 ,,	+	+	+
Mr. A.* ..	21	—	—	−	−	−

* Mr. A. ate only a very small amount of cream, "Less than a teaspoonful."

The cow, a single-house cow, was always milked by Mr. W. It had calved on December 12, 1953, and had not shown any signs of mastitis. Clinical examination revealed the udder to be normal, and the milk from each quarter

was quite unchanged, macroscopically or microscopically. The milking routine was satisfactory, and examination of food-handlers revealed nothing of any significance—for example, head colds, small septic-finger lesions, etc.

Bacteriological Examination

A specimen of the clotted cream received on January 9 yielded a profuse growth of *Staph. pyogenes* on blood agar. This organism was also isolated from a composite milk sample taken with sterile precautions from the four quarters of the cow's udder.

On January 19 nasal and throat swabs from Mr. and Mrs. S. and Mr. W., and milk samples from each quarter of the cow were examined. Large numbers of *Staph. pyogenes* were present in the left nostril of Mr. W. and smaller numbers in the left fore-quarter of the cow. Staphylococci were not grown from any of the other specimens. On January 29 the above examination was repeated with similar results. On this occasion a swab was also taken from the back of Mr. W.'s right hand; *Staph. pyogenes* was recovered from it on culture.

The cultures referred to above showed the characteristics usually associated with *Staph. pyogenes*, but two of their properties merit special reference. Firstly, when tested for haemolysin production by the method of Elek and Levy (1950) they were all found to produce α and δ toxin but no β toxin. Secondly, they produced opacity in egg yolk when tested by the method of Gillespie and Adler (1952). They were phage-typed by Dr. R. E. O. Williams, of the Staphylococcal Reference Laboratory, Colindale, who reported that they all had the same phage pattern, 53+.

On January 29 clotted cream was made by exactly the same methods that had been used on previous occasions, bacterial counts being carried out at appropriate intervals by the method of Miles and Misra (1938). The results are shown in Table III. No other bacteria were found to be

TABLE III

Time and Technique	*Staph. pyogenes* × 10^4 per ml.	
	Cream	Milk
January 29, 8 a.m. Cow milked; milk then placed in china jug in pantry and left for cream to rise.		
9 a.m.	—	1·25
January 30, 10 a.m.	2·5	0·02
Jug placed on plate of Aga range.		
6 p.m.	2,000	50
Left in pantry overnight.		
January 31, 10 a.m.	7,500	45

present. No detailed temperature recordings were made while the cream was on the range, although a temperature of 38° C. was recorded on one occasion.

Discussion

The available evidence indicates that *Staph. pyogenes* was responsible for the outbreak of food-poisoning. The origin of the strain of that organism is of some interest. Dr. R. E. O. Williams reported that strains of *Staph. pyogenes* having a phage pattern identical with or closely similar to our strain of *Staph. pyogenes* are fairly commonly isolated from human beings and have been incriminated in outbreaks of food-poisoning. He points out, however, that this type of staphylococcus has been isolated from materials from cows. Macdonald (1946) and Smith (1948) found that most staphylococci isolated from milk belonged to phage-type 42D. Phage 53 was not in use when they carried out their work, but since the number of untypable strains they encountered was small it can be concluded that phage-type 53 is, at least, not commonly found in milk. A consideration of the other properties of our strain is probably of greater interest, as it did not produce β toxin, a toxin which is characteristic of strains isolated from cattle—for example, Minett (1936), Smith (1947), and Elek and Levy (1950). Again, it is noted that our strain produced opacity in egg yolk. Adler, Gillespie, and Herdan (1953) found that 84.1% of strains of human origin but only 12.9% of strains isolated from cow's milk or from cases of bovine mastitis possessed this property. It is therefore probable that the strain of *Staph. pyogenes* responsible for this outbreak of food-poisoning was of a type that is usually associated with human beings and that the origin of the infection in the cow's quarter was a focus in the nose of Mr. W., the milker. If this was the case it is noteworthy that it did not prevent Mr. W. becoming a victim of the food-poisoning.

In the absence of any detailed information about the character of staphylococci isolated from milk or milk products that have been incriminated in outbreaks of food-poisoning it is tempting to speculate why so few outbreaks have been caused by the consumption of these foods despite the fact that they often contain *Staph. pyogenes*. One explanation that received support from the observations made during the investigation of the present outbreak is that the staphylococci that usually infect the cow's udder do not produce enterotoxin. It is realized that other explanations are also quite possible.

Summary

Two small outbreaks of staphylococcal food-poisoning caused by clotted cream prepared on two different occasions from the milk of the same cow are described.

Staph. pyogenes was found to be present in the milk from one of the quarters of the cow's udder on several occasions. The same type of staphylococcus was also isolated from the nose of the milker. The characters of these strains of staphylococci more closely resembled strains of human than bovine origin.

ADDENDUM

After these two outbreaks of staphylococcal food-poisoning steps were taken to deal with the two staphylococcal carriers—Mr. W., who was responsible for the milking, and the cow concerned.

In the case of Mr. W. the condition apparently cleared up after treatment with sulphonamide and penicillin, negative nasopharyngeal swabs being obtained. Negative bacteriological results were also obtained from culture of samples of milk from the cow on two occasions, following treatment with repeated intramammary injections of procaine penicillin cereate.

The household then recommenced to drink milk and cream in its raw state and with no ill effects until May 14—which happened to be a freak weather period with maximum day temperatures approaching 80° F. (28.7° C.). On this occasion skimmed cream was the offending vehicle of infection, and was eaten by Mr. W., Mrs. W., Mrs. B., and Miss H., only a very small quantity being eaten by Mr. W., who was unaffected; but the three latter were taken acutely ill with nausea, diarrhoea, and vomiting about three hours later.

Specimens were taken for bacteriological examination as follows: (1) Samples of cream remaining from the meal were found to contain approximately 10^7 viable staphylococci (type 53+) per ml. (2) A sample of milk from the same quarter as was affected in the earlier outbreak was found once more to be excreting 10^5 staphylococci (type 53+) per ml. (3) A nasal swab from Mr. W. gave a profuse growth of staphylococci (type 53+). (4) Staphylococci (type 53+) were cultured from faeces of Mr. and Mrs. W. On this occasion faeces were also examined for organisms of the salmonellae–shigellae group by culture on desoxycholate–citrate–agar medium with and without previous enrichment in selenite-f, with completely negative results.

The cream was prepared by skimming milk which had been allowed to stand overnight in a pantry from milking-time, 4 p.m., on the previous day. The cream thus obtained was placed in a refrigerator from 10 a.m. until eaten at 7 p.m.

This last outbreak in this series of three related outbreaks is noteworthy in that it is a clear demonstration that a recent attack of staphylococcal food-poisoning confers no immunity.

We are grateful to Dr. R. E. O. Williams for help and advice.

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Medical Memoranda

Smoking and Lung Cancer

For purposes of comment the results recorded by Dr. R. Doll and Professor Bradford Hill in Table III on page 1273 of the *British Medical Journal* for December 13, 1952, have been graphed. The degree of cigarette-smoking is specified by them in the following way: 1 Cig.–, 5 Cigs.–, 15 Cigs.–, and so on. It is perhaps least open to objection to plot, for any particular range, the average value, and this has been done in the graphs of Fig. 1. We are not entitled

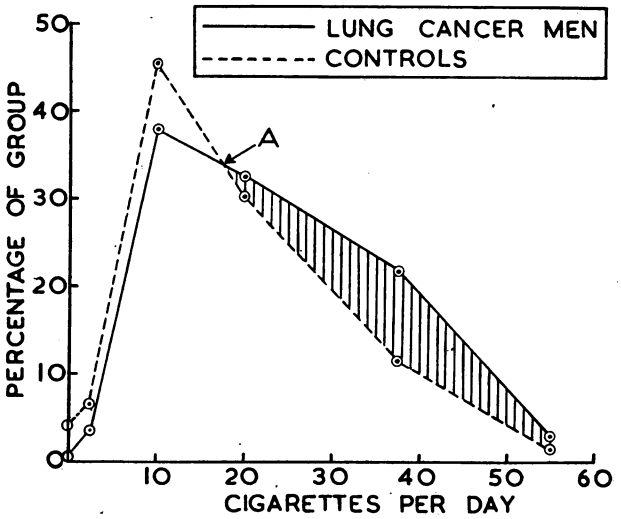

FIG. 1

to conclude from this that heavy cigarette-smoking is the cause of lung cancer; a strictly impartial comment would be that "it will be seen that there is a higher percentage of heavy smokers among the cancer men than among their controls."

Cause and effect are not always easily distinguishable. If an inquiry showed that men with cancer of the stomach took more alcohol than their controls it would be quite rational to look upon a little excess of alcohol as the effect and not the cause of the disease; it might be like this with smoking.

The two graphs intersect at the point A; here we find the two groups of men smoking cigarettes to the same degree. Is one entitled to conclude that those who have lung cancer contracted it *because* of their cigarette-smoking? It has to be remembered that for every lung-cancer man we are thinking of in connexion with smoking there is not only his opposite number serving as control in the statistical investigation by Doll and Hill, but there is quite a large number. At this stage it is convenient to bear in mind a useful quantity—namely, the number of men who have lung cancer at the present time.

The senior economist in the U.S. Public Health Service investigated the relation between the number of people ill from cancer and those dying from the disease in that country. From the information he obtained he was able to derive a ratio which, multiplied by the number of cancer deaths per year, gives approximately the number of people suffering from cancer at any moment within the period examined. Assuming that survival with cancer does not differ much in the two countries, and applying this same multiplying factor to our own present yearly total of deaths —for example, 75,000 in 1950—we derive the approximate figure of 150,000; the factor for all forms of cancer being about two. In the case of lung cancer the factor is almost certain to be less than two because the clinical course of the disease is shorter than the average for cancer; with a yearly mortality for cancer of the lung of 15,000 (10% being women) we shall be erring on the right side by putting the number of lung-cancer men at this moment as 30,000. There are about 12,000,000 men in England and Wales between the ages of 25 and 75, but their age distribution is not the same as that of lung-cancer men. Consequently we cannot say that there are available 400 controls to every cancer patient. If we halve this number and say 200, we shall probably be making a safe allowance for this difference in age distribution. Hence for any lung-cancer man in the Doll and Hill series there will be one man chosen as control out of an average of about 200; it will not always be 200, as we shall see.

If as an example we select the 10-cigarettes-per-day man, for every lung-cancer man there are about 200 similar smokers without cancer. Are we to conclude that if the one man in several hundred who gets lung cancer smokes 10 cigarettes a day as do the others, then smoking has caused the lung cancer? It is possibly true, but is not the long arm of causation being unduly stretched in maintaining the same position for all and every grade of cigarette-smoking?

Dr. Doll writes in the *British Journal of Cancer* of September, 1953: "Even very heavy smoking does not appear to be uniformly carcinogenic."

A man who smokes 40 cigarettes a day can perhaps be reckoned a very heavy smoker. A glance at Fig. 1 shows that of the group there were 19% lung-cancer men and 10% controls in this category, say 2:1. To get a proper perspective in this matter it must be remembered that the control non-cancer man is one out of *about* 100 who could serve as controls; so that it is one very heavy smoker who gets lung cancer as against 100 similar smokers who do not. Dr. Doll may be said to be indulging in a somewhat inverted meiosis in his statement.

In view of the fact that smoking is a widespread habit it would be of the greatest interest to compare the death rates from lung cancer in different countries with their smoking experiences. Unfortunately, there are very few countries which have the requisite data in appropriate form for any such comparison. With what data were available, however, Dr. Doll has drawn a graph in which death rates from lung cancer are plotted against the degree of cigarette-smoking. I have reproduced this as faithfully as I can in Fig. 2. Dr. Doll writes: "I doubt whether much significance should be attached to the result, *but it is not inconsistent with the existence of a relationship between lung cancer and cigarette-smoking.*" The italics are mine.

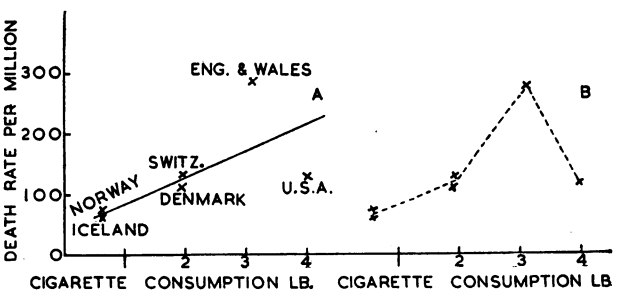

FIG. 2