Correspondence

Correspondents are asked to be brief.

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Bacteriological Checks on Food Handlers

SIR,—Bacteriologists are sometimes asked to make routine examinations of the faeces of food handlers with the object of detecting carriers of such intestinal pathogens as typhoid or paratyphoid bacilli, food-poisoning salmonellas, and dysentery bacilli. I am often asked for advice about the value of the proposed routine examinations and your readers may care to know my views.¹

Examination of the faeces of individual food handlers should be carried out whenever there is any question of their having suffered an intestinal illness or been put at risk by close contact with a known infected patient—for example by eating food prepared by such a patient. Likewise, if any circumstances suggest that food handlers have been involved in an outbreak of food-poisoning or dysentery, such examinations will normally be carried out as part of the investigation at the request of the local health department.

But if purely routine laboratory checks on the faeces of food handlers are asked for, I think that this work should not be undertaken. The potential benefit to the public health is not likely to be proportional to the effort and resources that would be required, even if these were available. Pre-employment clinical assessment, on the other hand, is extremely important. Employees who have recently been abroad or come from abroad should also have their medical

history very carefully checked before beginning work or returning to work. Any with a history suggestive of intestinal disease should have their faeces carefully examined; but this is a different matter from undertaking regular routine laboratory examinations. The public health must be protected, of course, in every practicable way, but strict application of the rules of hygiene in the handling and preparation of food will do much more to that end than will routine examinations of faeces.

When laboratory examinations are found to be necessary, their timing and planning should always be discussed with the bacteriologist concerned. Such consultation is particularly desirable when any considerable number of specimens must be examined, but it should always be undertaken because it is the best means of ensuring a wise selection of cases, use of the best methods for collection and transmission of specimens, and the right interpretation of reports. Also by ensuring a discriminating use of the laboratory resources available, the consultation is likely to guarantee increased efficiency in the carrying out of any tests agreed upon as necessary.—I am, etc.,

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Routine Laboratory Screening

SIR,—Dr. M. H. B. Carmalt and his colleagues have demonstrated, as have many others, that routine laboratory screening with a multiple analyzer may occasionally bring to light certain previously undiagnosed diseases and a variety of haematological and biochemical abnormalities (7 March, p. 620). What they have not done is to show that prognosis of any of the conditions diagnosed has been in any way influenced by their efforts. To detect a biochemical abnormality is one thing; to change the course of the disease is another.

Although the World Health Organization has arbitrarily defined a haemoglobin level of below 12 g./100 ml. as abnormal, the evidence for saying that a woman who has a value of 11 g./100 ml. is anaemic is flimsy in the extreme. What is needed is a

correlation between haemoglobin level and a realistic index of disability. Cochrane and Elwood¹ have clearly pointed that this is not available. Moreover, in a community study they were unable to show an improvement in symptoms following iron therapy, though the haemoglobin level subsequently reached a standard approved by the W.H.O. In short the administration of iron under these circumstances might well be described as relieving symptoms in an asymptomatic patient. The example cited by the authors of the usefulness of routine normal tests is that of an anaemic patient who showed a normal blood urea, thereby excluding uraemia as the cause of his anaemia. I cannot but recall doing just this some years back when I was Sir Edward Wayne's house physician. He good-naturedly pointed

to the results of the routine urine analysis that I had done myself and asked me how often I had renal failure of sufficient severity to cause anaemia without there being albuminuria. I am looking yet. As for the detection of early diabetes, no one has yet shown that its ultimate prognosis has been influenced one jot or tittle by early diagnosis. The same applies to most of the other entities likely to be brought to light by multiphasic screening.

Living in a country where routine multiphasic screening is indeed routine, I would point out that it is nothing like as helpful as many would have us believe. In those hospitals where this service has been available for some time, it has been shown that around 40% of the abnormalities detected by routine screening are ignored by the physicians in attendance, despite the fact that various methods have been employed to designate the abnormal values—for example, denoting them with an asterisk. Routine screening becomes a form of "biochemical bingo" and inevitably leads to short cuts in history taking. To those who advocate the concept of routine biochemical and haematological screening, the monograph *Presymptomatic Detection and Early Diagnosis*² is recommended as an unbiased review of its limitations. Many more randomized controlled trials need to be done before it is going to be possible to say that the routine performance of a specific biochemical or haematological test (the W.R. excluded) is a worthwhile procedure.—I am, etc.,

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Epidemic Malaise

SIR,—The articles of Dr. C. P. McEvedy and A. W. Beard (3 January, pp. 7 and 11) are of considerable concern because of the authors' contention that benign myalgic encephalomyelitis (epidemic neuromyesthesia) is a psychosocial phenomenon related to mass

hysteria or to altered medical perception in the community. Their erroneous conclusions about this illness may impair future investigations of similar outbreaks.

It is apparent that the authors failed to do their homework, and demonstrated a surprising lack of information about the principles of epidemiology and of psychiatry. Had they reviewed the literature on the subject, they would have discovered that Albrecht, Oliver, and Poskanzer¹ investigated an outbreak of this illness in New York and pointed out that an easily recognized laboratory abnormality occurs in this illness. There is a considerable increase in creatinuria and an increase in the creatine/creatinine ratio, suggesting an abnormality of muscle. On recovery from the symptoms the creatinuria disappeared. Drs. McEvedy and Beard also failed to point out that the epidemic curve, in at least one outbreak,² was consistent with person to person spread, and that radial spread over time was demonstrated from the centre of the community to the more rural areas.

The question of mass hysteria has been considered by the authors of most papers relating to this disease and in each instance has been discarded for a number of reasons—namely, (1) cases occurring within the same household are varied in their features and course; (2) separate illnesses appear at random intervals instead of simultaneously; (3) epidemiologically, the consistency of course and similarity of symptoms despite the variety of people and communities that were affected make hysteria unlikely. The disease is consistent from outbreak to outbreak in different countries, different years and different peoples. (4) The mental symptoms of depression, emotional lability, impaired memory and difficulty concentrating are consistent with organic disease as compared with the shallowness and indifference of hysteria. (5) Muscle pain is a striking feature of most outbreaks. It is clear that sporadic cases of this disease cannot be readily identified. It is only in the epidemic form that the distinctive epidemiological features allow characterization.

Instead of ascribing benign myalgic encephalomyelitis to mass hysteria or psychoneurosis, may I suggest that the authors consider the possibility that all psychoneurosis is residual deficit from epidemic or sporadic cases of benign myalgic encephalomyelitis?—I am, etc.,

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Onions and Blood Fibrinolysis

SIR,—Recently my colleagues and I² demonstrated that raw, fried, or boiled onions increased the blood fibrinolytic activity but had no effect on serum cholesterol levels, re-calcified clotting times, thrombotests, and fibrinogen levels. The study reported below is a follow-up of our previous study, and was undertaken to discover if the property in the onion which causes

this increase in fibrinolysis was present in commercially available dried onions and in lyophilized onions.

The dried onions used in this study (supplied by Gentry Corporation, California, U.S.A.) are granulated, minced, and chopped and in weight one part is equivalent to ten parts of raw peeled onions. The dried onions were reconstituted before use by soaking in cold water in the ratio 1:3. A lyophilized sample of onion (supplied by Kabi Pharmaceuticals, Stockholm, Sweden) was prepared from 1 kg. of onions, which was homogenized, the mixture centrifuged and the precipitate discarded. The sample ready for use weighed 40g.

Subjects	Day 1		Day 2		Day 3		Day 4		Day 5		Day 6	
	Breakfast only		Breakfast + dried granulated onions		Breakfast + dried minced onions		Breakfast + dried chopped onions		Breakfast + lyophilized onions		Breakfast + lyophilized onions	
	9 a.m.	11.30 a.m.	9 a.m.	11.30 a.m.	9 a.m.	11.30 a.m.	9 a.m.	11.30 a.m.	9 a.m.	11.30 a.m.	9 a.m.	11.30 a.m.
1	26.3	25.0	25.0	142.8	24.3	55.5	23.5	47.6	25.6	33.3	23.8	37.0
2	25.6	24.3	26.3	45.4	23.8	166.6	24.6	38.4	22.7	27.7	21.7	38.4
3	71.4	52.6	58.8	111.1	66.6	133.3	60.6	142.8	58.8	105.2	76.9	105.2
4	33.3	29.8	37.0	55.5	32.2	74.0	33.3	100.0	34.4	66.6	32.2	52.6
5	38.4	31.2	36.9	52.6	34.4	66.6	40.0	66.6	37.0	62.5	34.4	58.8
6	31.2	26.3	41.6	52.6	30.3	50.0	32.7	45.4	34.4	41.6	30.3	50.0

TABLE.—Euglobulin Lysis Times (in units) of Volunteers

Six volunteers were included in the study. On day one, after fasting, samples of blood were collected for the estimation of the euglobulin lysis time (E.L.T.) by the method described by von Kaulla,³ slightly modified and using an E.L.T. recorder.⁴ The lysis times obtained were expressed in units by multiplying the reciprocal of them in minutes by 10,000. After withdrawal of the blood samples mentioned the volunteers were given a breakfast at 9 a.m. containing a total of 39.8g. of fat and new samples of blood were collected after two and a half hours. On day two the procedure was repeated but this time 7½g. of granulated dry onions were added to the meal. On day three the method was almost identical with that outlined above, the only difference being that this time 7½g. of minced dry onions were added to the meal. On day four the meal was augmented by 7½g. of chopped dry onions. On days five and six, 3g. of lyophilized onion was added to the breakfast. The volunteers were made to rest throughout the experiment, since a previous study had

dies^{1,2} established that the factor is heat-stable and also not water-soluble.—I am, etc.,

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New Bronchodilator Aerosol Preparations

SIR,—Your readers will no doubt have seen in the *British Medical Journal* and elsewhere several advertisements for Medihaler-duo (Riker Laboratories), a combined aerosol of isoprenaline and phenylephrine supplied in pressurised dispensers for inhalation. A recent *B.M.J.* advertisement (14 March, p. XV) is headed "When it comes to safety, only one inhaler has been shown to improve the natural function of gas exchange in most patients—Medihaler-duo." Other advertising pamphlets contain statements such as "Medihaler-duo improves oxygenation of the blood . . . encourages elimination of carbon dioxide, and provides the physiological benefits associated with improved gas exchange," and "Medihaler-duo offers the prospect of greater safety in bronchodilator therapy." These statements carry the implication that the value of isoprenaline plus phenylephrine has been fully substantiated, but this is in fact not the case.

The only published studies I have so far been able to trace are reported in two short

letters to the *British Medical Journal* by Dr. T. T. Chapman (29 November 1969, p. 557) and Dr. K. M. Hume (17 January, p. 173). Both were clearly intended to be preliminary communications, and the authors' conclusions were couched in suitably cautious terms. Dr. Hume, for example, merely stated that his results supported Dr. Chapman's suggestion that "these findings may require to be considered in the future manufacture and prescription of bronchodilators." Nevertheless, some of his results, presented in tabular form, were reproduced in one of the advertisements for Medihaler-duo, and scientifically unwarranted conclusions, such as those quoted above, were drawn from them.

It is not the purpose of this letter to criticize the studies reported by Dr. Chapman and Dr. Hume, and the following comments are intended merely to draw attention to the way in which they have been exploited by the manufacturers of Medihaler-duo.

In Dr. Chapman's letter the pre-treatment levels of arterial oxygen tension (PaO₂) were