Section of Epidemiology and Preventive Medicine

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[*November* 19, 1954]

DISCUSSION ON THE ABO BLOOD GROUPS AND DISEASE

(*continued* from February *Proceedings*, p. 139)

Dr. R. B. McConnell gave preliminary data concerning the blood groups of 815 diabetics from the diabetic clinic, David Lewis Northern Hospital, Liverpool.

For analysis the patients were divided into the two main clinical types which are thought to be inherited in different ways, type I by a recessive gene, and type II by a dominant. 30 patients were not classifiable.

In 425 type I cases there were 5% more of group A at the expense of group O than in the control group, but this difference is not significant. In the 131 type I cases in whom the onset had been before the age of 30, there were 12% more of group A than in the controls, and this difference is statistically significant. ($\chi^2 = 8{\cdot}21$). This difference is particularly marked in the males.

In the 360 type II diabetics the blood group distribution is completely normal. The type II diabetics are mainly middle-aged obese women who do not often develop duodenal ulcer even when they are not diabetic. In the younger diabetics the lack of group O may provide the basis for the low incidence of duodenal ulcer.

Dr. C. A. Clarke gave preliminary data concerning the ABO blood group distribution of 1,488 peptic ulcers from three Liverpool hospitals.

In the duodenal ulcers (1,028 cases) 59·6% were group O, and 27·9% group A. In the gastric ulcers (460 cases) 49% were group O, and 40·2% group A.

The control figures taken from 1,000 blood donors from the Liverpool Regional Board Transfusion Service gave 48·10% group O, and 40·60% group A.

These findings, which are in general agreement with Professor Aird and his colleagues (1954, *Brit. med. J.*, ii, 315), suggest that the association between group O and peptic ulcer is much more marked in duodenal than in gastric ulcer.

[*December* 13, 1954]

Environmental Factors in Anæmia and Infection in Pakistan

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EPIDEMIOLOGY is an old science. Unlike some other old sciences, however, it has always had one great consistency, the concept of environmental causation of disease. In this paper I shall present a summary of my studies on anæmia and infection, two types of illness which are common throughout Asia. My purpose is to see how far the occurrence of such illness can be related to the known dominant environmental factors and to reveal, possibly by default, how much remains unknown and unexplored.

These studies were carried out in Karachi in Pakistan, as part of a WHO teaching assignment, and everything I say represents the work of a team[2] which it was my privilege to lead.

Our methods were simple. We estimated the hæmoglobin levels, and made certain other tests in a group of over 1,000 women. We examined fæces microscopically from another group of over 1,000 men and women. Hospital in-patients, and people who were manifestly ill, were excluded from each group. All the individuals examined were interviewed to obtain factual data about housing, size of family, occupation, wages and so forth. To some extent, our groups were selected, in that we tried to include reasonable minimum numbers

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[2]Acknowledgment is made to the following members, who participated in these studies: Professor T. Husain, Dr. S. S. Reza, Dr. A. S. Khan, Dr. S. H. Bokhari, Dr. A. N. Moten, Dr. M. A. Alvi, Dr. S. Bashir Khan, Miss Ruqiya; and to Lt.-Col. S. M. K. Mallick, M.D., F.R.C.P., Principal of Dow Medical College, for his interest and support.

of each age group, income group, &c., so that our results should be as far as possible a fair cross-section of the population.

Anæmia Survey

As might be expected, there was no doubt at all that the hæmoglobin levels in our cross-section of the women of Karachi were very definitely related to their social status. Fig. 1

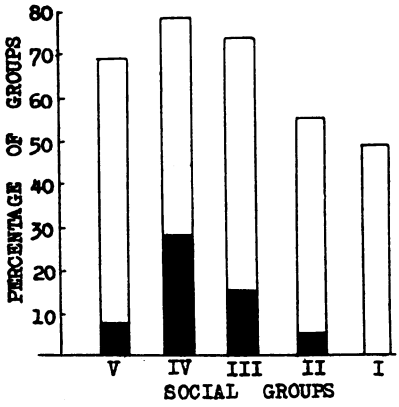

FIG. 1.—Anæmia survey in Karachi. Female subjects of child-bearing age. Relationship between social group and Hb level.

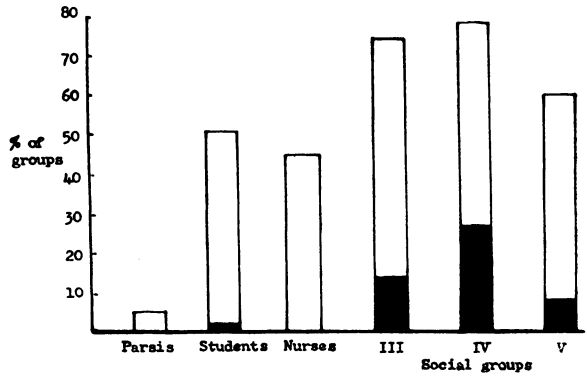

FIG. 2.—Anæmia survey in Karachi. Hæmoglobin levels in different groups of women.

% of groups with Hb less than 80%
% of groups with Hb less than 60%

shows the proportion of women who were anæmic in each of five social groups, ranging from the wives or daughters of wealthy merchants and potentates (group I) at one extreme, to the refugee and beggar class at the other (group V). In the communities amidst which we worked, these various social groups were virtually self-defined, and there was very little overlap between them. Anæmia has been depicted in two grades, the milder type with hæmoglobin values between 60–80% (100%=14·8 grams) and the more severe forms with hæmoglobin values of less than 60%. It must be remembered that all these women were walking and attending to their usual tasks, or, shall we say, carrying their burdens. Yet we find that, in groups III, IV and V, more often than not they were anæmic, sometimes seriously so. In the more fortunately placed groups (I and II), the incidence of anæmia, though hardly negligible, was significantly less.

There was one interesting anomaly which kept reappearing. As we pass from social groups I to IV, the incidence of anæmia increases sharply. But in group V—composed of the wives and daughters of the unemployed, the refugees and the beggars—there was less anæmia than in group IV.

I think there may be a simple explanation of this, but meanwhile let us glance at the results from other environmental angles.

Social status is partly, but seldom entirely, influenced by income. When we analyse our results crudely in terms of income groups we find therefore more anæmia where there is less money. If, however, we refine this analysis (Fig. 2) to the extent of separating out a number of women into communities, irrespective of income, we find that money did not of itself confer—though it may be allied with—what the nutritionists would call Fe value. Our students (Fig. 2) were not necessarily wealthy, and our nurses were not well paid, but they were better placed than their sisters in social groups III–V, even if they did not quite measure up to the irreproachable hæmatological state of the exclusive Parsi community.

If we make allowance for age (Table I) we find that anæmia was most prevalent during the critical period between 15 and 30 years, and showed a significant decline in incidence thereafter. In the physiologically active period, then—and in Asia, women may marry and bear several children before the age of 18—anæmia, often severe, is present in a sizable majority of those women. This prevalence diminishes as we climb the social scale and, incidentally, I might mention that the anomaly of group V, already mentioned, emerges afresh in this closer analysis: it seems that in Karachi, you are wealthier in hæmoglobin at least, if you are the wife of a beggar in group V than if you are the wife of a craftsman in regular employment in groups III and IV. According to local rumour, you may be wealthier in other ways as well.

TABLE I.—ANÆMIA SURVEY IN KARACHI

Mean Hæmoglobin % Levels of Women According to Age and Social Group

	Weighted Means Social group			
Age groups	V and IV	III	II and I	Mean
15–20 years	67%	68%	72%	69%
21–25 „	65%	67%	76%	69%
26–30 „	65%	66%	76%	69%
31–51 „	71%	73%	73%	72%
Over 51	76%	76%	84%	79%
No. of cases	417	296	355	
Means	69%	70%	76%	

I cannot spend time in demonstrating all the factors which we investigated in this survey. But, briefly, pregnancy was found to increase the vulnerability to anæmia at all ages though not necessarily in all communities: some women are only properly cared for when they become pregnant. Among other factors which may be mentioned are Purdah-observance, which was associated with a higher anæmia rate this time in the more privileged, and not in the lowly, social classes. Migration, though significant historically, did not seem to affect Hb values but, oddly enough, the degree of literacy did: at least, literate women in all the social groups were less anæmic than the illiterate. The number of children borne did not seem to matter, but parous women on the whole had lower Hb levels than the nulliparous.

Now it is often said that there is something mysterious and macrocytic about anæmia in the Orient. Our results did not bear this out. Indeed, most of our anæmia was of simple nutritional type and, in the residue, chronic infections such as malaria almost certainly played a large part. True macrocytic anæmia was quite rare, accounting for less than 3% of severe anæmias, and, of course, none of it was Addisonian.

INFECTION

So much for anæmia. Our second survey was designed to estimate the incidence and environmental factors associated with another group of prevalent diseases, intestinal parasitic infections.

The crude carrier rate for various infections is shown in Table II. These estimates were made from single samples of fæces, each sample being subjected to two wet-film examinations and, if these were negative, to concentration by the flotation method. This means that, high as they are, the observed figures must fall short of the actual carrier-rate of each organism. If for example, we take infection with *E. histolytica*, the observed figure was 18·4%. To this can be added, in all probability, the majority of those showing, on one examination, indefinite cysts or exudate, so that the total becomes about 30%. If we had examined more specimens, or if we impose the mathematical correction suggested by Dobell (1916) and others (Stewart *et al.*, 1948), this figure would increase to about 40%. A proportionate increase can be assumed for the other infections listed, so it is obvious that we were dealing with a fair volume of infection.

TABLE II.—CARRIER-RATE OF INTESTINAL PARASITIC INFECTIONS IN MISCELLANEOUS HOSPITAL PATIENTS AND STAFF EXCLUDING CASES OF ACUTE BACILLARY DYSENTERY

Organism	Number of cases	% of total
Entamœba histolytica ..	248	18·4
Indef. cyst and/or exudate	177	13·1
Giardia lamblia	109	8·1
Ascaris	47	3·5
Trichuris	25	1·9
Ankylostoma	22	1·6
Tænia nana	36	2·7
Tænia saginata	6	0·4
Others	293	21·7
Total cases	1,348	

To examine the influence of environmental factors, I have grouped together the total observed figures for all these infections, with a correction to allow for multiple infections. This gives an "infected" and a "non-infected" group, which is useful for the purpose of this paper, though it does not reveal differences which might exist between the various types of infection.

An analysis of the income-groups of the subjects (Table III) shows that infection was

TABLE III.—INCIDENCE OF PARASITIC INTESTINAL INFECTIONS ACCORDING TO INCOME-GROUPS OF SUBJECTS

Rs/month	Less than 40	41–80	81–120	121–200	201–440	Over 440	Total cases
Infected ..	44	279	138	74	19	6	560
	(62·9%)	(59·6%)	(53%)	(63·8%)	(55·9%)	(37·5%)	
Not infected	26	189	122	42	15	10	404
	(37·1%)	(40·4%)	(47%)	(36·2%)	(44·1%)	(62·5%)	
Total cases	70	468	260	116	34	16	964

widespread at all levels, and that there was no significant difference in the incidence of infection between the lower and middle income groups at least. Freedom from want did not apparently confer freedom from amœbiasis.

TABLE IV.—INCIDENCE OF PARASITIC INTESTINAL INFECTIONS ACCORDING TO HOUSING CONDITIONS OF SUBJECTS

	Refugee huts	Urban slums	Middle-class and residential	Total cases
Infected ..	121 (56%)	195 (58%)	78 (59%)	394
Not infected	97 (44%)	139 (42%)	55 (41%)	291
Total cases	218	334	133	685

In this part of our survey, we were for various reasons unable to obtain the same amount of social and environmental data about our cases as in the anæmia surgery. In the remaining figures, therefore, the data have had to be simplified, and the numbers are smaller but still, I think, sufficient for comparisons to be made. For instance, we imagined that infection might be related to housing conditions so, instead of dividing our cases into their various social grades, we divided them according to the types of dwellings they inhabited (Table IV). It appeared that the incidence of infection was remarkably uniform, that you were as likely to find intestinal parasites in the residential suburbs as in the slums.

TABLE V.—INCIDENCE OF PARASITIC INTESTINAL INFECTIONS ACCORDING TO NUMBER OF INDIVIDUALS PER ROOM

No. per room	6 or more	3–5	2	1 or less	Totals
Infected subjects:					
Number:	23	107	36	6	172
% of infected group	13%	62%	21%	4%	
% of housing group	59%	59%	61%	55%	
Not infected:					
Number:	16	74	23	5	118
% of group	14%	63%	19%	4%	
% of housing group	41%	41%	39%	45%	
Totals	39	181	59	11	290

To investigate this aspect in closer detail and, necessarily, with much smaller numbers, we then classified our cases in terms of crowding in their homes (Table V). Again we met with a striking and quite unexpected uniformity in the incidence of infection.

These then are the facts of our study and, as such, I trust they are unambiguous. If by anæmia we mean a hæmoglobin value of less than 80% (11·8 grams %), then anæmia was very prevalent indeed in a cross-section of the female population of Karachi in that it affected nearly half of the women in the wealthy class and 75% of those in the artisan class. I am fairly confident about these figures but I have to confess that I am less confident about the word anæmia. For instance, the hæmoglobin value of 100% (14·8 grams %) is based upon the average value of male subjects in the Western world. With Asiatic males in Karachi, we found that more than half had hæmoglobin levels of less than 100%, the median value being only 92%. Among European females, we found that nearly three-quarters had values just below 80%. All of these estimations were made upon apparently healthy, active subjects, and I do not pretend to know what is the explanation; but it is possible that climate, hydration, locality and other factors peculiar to the latitude—and perhaps the longitude—were somehow involved.

Be that as it may, we can drop our Western ideas about 100% hæmoglobin quite considerably, and still have to admit, from the figures submitted, that anæmia in Pakistan is a real problem, for there would seem to be millions walking, working and bearing children with hæmoglobin values of less than 50% (7·4 grams %). Since most of this anæmia is nutritional in type, it is not surprising to find that its incidence runs parallel to the major social factors which I have mentioned—poverty, child-bearing, the low status of women in certain communities. Even so, the extent to which the already high incidence of anæmia increases in the physiologically active age period (15–30 years) and in pregnancy, especially in its third trimester, seems to me quite startling and not confined in its effect to this generation only. Paradoxically, the incidence of anæmia lessens significantly in the lowest social strata, among the wives of beggars and unemployed refugees, indicating possibly that some subtle urban stresses, absent in these people, may contribute to the higher incidence in the artisan classes above. This, however, was the only important exception which we encountered. In other respects, anæmia appeared to be a fairly exact mirror of environmental conditions.

In complete contrast, we found that parasitic intestinal infection, also a widespread disease, was unrelated in incidence to differences in income, community or type of home. It seems therefore that infection breaks through the social structure of the country, whereas anæmia is caused by it.

Now it is tempting to think that a little sensible social medicine could explain these differences and rectify these wrongs. And, indeed, there are obviously a number of simple

solutions, which, aimed though they are from a distance of 7,000 miles cannot be altogether wide of the mark. For example, it would not be difficult to define, here and now, the groups most vulnerable to anæmia. In fact, one might say that the answer to nutritional anæmia lies largely in the pockets of those unaffected by it. Or, with regard to infection, one could justifiably suggest that when the volume of infection reaches a certain level, environmental barriers to its spread begin to break down; that a massive influx of refugees (there are over half a million in Karachi) may so strain housing and sanitary resources that isolated foci of infection become confluent, and common to all; that the rich are, after all, served at table by the poor, in Asia if not in England. All these are, I repeat, simple answers which cannot be seriously wrong. But it would be an injustice to the complexity of life in the countries concerned to suggest that they are the proper answers. At the risk of sounding quite unscientific, I am going to say that you cannot give health to Asia by a touch of Western epidemiology here and there. I do not think that we can cure the malnutrition in Indo-Pakistan by powdered milk from the U.S.A. or by iron supplements from England.

I began this address by implying that there might be contradictions to the important rule that preventable environmental factors induce community diseases. The problems and paradoxes which I have mentioned are not such as to dislodge such a rule, but they do perhaps qualify its applicability here and there. Translated from figures and charts into human and economic terms, they are the kind of challenge that, in the small world of to-day, one cannot readily ignore.

REFERENCES

DOBELL, C. (1916) *Spec. Rep. Ser. med. Res. Comm.*, No. 4.
STEWART, G. T., O'MEARA, P. J., and KERSHAW, W. E. (1948) *J. R. N. med. Serv.*, **34**, 2.

Dr. S. S. Reza: The result of the survey leaves little doubt that the severe degree of anæmia is connected with the economic state of the masses.

The widespread mild anæmia according to the accepted hæmoglobin standard may be peculiar to that part of the world for socio-economic reasons or the accepted hæmoglobin standard may be too high for the population, since they manage to live a normal life for all practical purposes with about 11·8 grams % hæmoglobin.

Further investigation is required to assess the normal hæmoglobin value for that part of the world.

The Measurement of Malaria Transmission

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Two full malaria surveys have been made by my colleagues Davidson and Draper (1953) and Davidson (1955) on principles derived from a theoretical analysis of the mathematical nature of transmission prepared by myself (Macdonald, 1950, 1952 *a*, *b*). The work was carried out in two parts of East Africa, in Tanganyika and Uganda, where malaria transmission is perennial and holo-endemic, or of an extreme and continuous intensity which has been much studied in near-by localities (Wilson, 1936, 1939; Garnham, 1929, 1949; Wilson *et al.*, 1950, &c.). The predominant parasite is *Plasmodium falciparum*, the incidence of infection by ages is shown in Table I, and the type of endemicity is particularly characterized by its stimulation of an immunity manifested by a marked decrease in parasitic density from the age of about 2 years onwards, and a slower progressive reduction in the parasite rate which can be seen in Table I.

TABLE I.—SPLEEN AND PARASITE RATES, TANGANYIKA AND UGANDA

	Tanganyika			*Uganda*		
Age group	No. exam'd.	Spleen rate %	Crude parasite rate %	No. exam'd.	Spleen rate %	Crude parasite rate %
2 weeks to 2 months	109	18	21	31	34	61
3 to 5 months ..	57	69	75	72	75	81
6 to 11 months ..	74	87	88	98	89	92
12 to 23 months ..	62	89	87	150	94	99
2 to 4 years ..	89	89	96	266	88	98
5 to 10 years ..	192	73	85	211	68	95
11 to 14 years ..	78	53	74	36	31	81
15 years and over ..	83	26	42	57	20	33