Letters to the Editor

Recommended Health-based Limits in Occupational Exposure

Sir,

Drs Ali and Al-Swailem\(^1\) raised a very important subject for industrial and occupational health in this journal. The exposure limit for the maximum allowable concentration for the occupational work environment of every substance is urgently needed in Saudi Arabia and the relevant authorities should indeed compose local RHBL tables allowing for different environmental and physical variations.\(^1\)

Our comments will concentrate on the recommended health-based limit table. The table as presented suffers from an easily corrected deficiency, namely that much of the data is out of date. Most of the limits as they appear in the table have been revised. We will comment here on the radiation limits only. While we believe that Drs Ali and Al-Swailem have performed a valuable service in starting the process of setting limits for hazardous substances and agents for the Kingdom, it would be unfortunate if the limits receiving official approval were not the most recent standards set by the appropriate international bodies and for this reason required modification almost from the time they were officially adopted.

The authors indicated that the radiation (gamma) limit is 0.3 Roentgens/week (0.3 R/W), which is equivalent to 15 R/year. This limit is out of date being abandoned in 1958 by the International Commission on Radiological Protection (ICRP). The National Committee on Radiation Protection and Measurement (NCRP) had abandoned this limit a year before that, in 1957.\(^2\) The unit (Roentgen) is a measure of exposure; it is not used in the current radiation protection limit, rather, a dose equivalent unit is used which is the absorbed dose \(\times\) a weighing factor of biological effect for each type of radiation (quality factor). The SI unit for dose equivalent is J/kg with the SI derived unit of Sievert (Sv) (Sv = 100 rem).

The current international dose equivalent limit recommended by the ICRP and NCRP is 50 mSv (5 rem) per year for uniform whole body irradiation, as of April 1990.\(^3\) The ICRP is expected to update and revise its limit by the first quarter of 1991.

Where the workers receive radiation from external sources and from intake of radioactive material together, the effective dose equivalent from the external exposure, the dose equivalent from total intake during the year by ingestion must be added to the external dose and the total kept below the limit noted above.

To control the intake of radioactive material, the ICRP specifies an annual limit for intake (ALI) leading to derived limits of air concentration (DAC) for each radionuclide during any year.\(^4\)

The ICRP urges that the actual operational dose limit for any radiological activity be more restrictive than the maximum recommended dose limit which is defined as the ALARA (as low as reasonably achievable) concept. Economic and social factors must be considered in implementing the ALARA limit.

We hope that the process of setting limits for hazardous substances which Drs Ali and Al-Swailem have started will now be vigorously pursued by the appropriate authorities in the Kingdom.


References


Sir,

I thank Mr Al-Haj and Dr Calkins for their comments. We collected the data in the RHBL tables from various sources, some of which are not recent. This means that some of our data may have been changed, but mostly to a minor degree. However, the limits given are not exhaustive, nor exclusive, and the need for recent local figures is highly appreciated. The figures which Mr Al-Haj and Dr Calkins have been kind enough to supply are definitely more correct and more informative. Thus it is advisable to consider the data in the RHBL tables as rough guiding parameters. We will contact the International Labour Office for the most recent figures and will send them for publication as soon as possible.

We would again thank Mr Al-Haj and Dr Calkins for their interest and we are ready to participate with them in any specialized group entitled to study the RHBL tables for Saudi Arabia.

Professor Mohamed El-Shabrawy Ali PhD
Chairman, Department of Community & Family Medicine, King Saud University, PO Box 2925, Riyadh 11461, Saudi Arabia

Sir,

I read with interest the article entitled by Drs Ali and Al-Swalem (Saudi Med J 1990; 11(2): 152–156). It is reassuring to find that occupational medicine is getting an overdue attention in the general medical periodicals, especially in countries undergoing rapid industrialization.

However, it is remarkable to find that the recommended exposure limits included in the article are very much outdated. The latest figures adopted by most industrialized countries are much lower than those quoted in the article, in certain cases by a factor of 10–100.

A few examples of these differences are:

<table>
<thead>
<tr>
<th>Substance</th>
<th>Old Limit</th>
<th>New Limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroform</td>
<td>10 ppm</td>
<td>100 ppm</td>
</tr>
<tr>
<td>Iodine</td>
<td>0.1 ppm</td>
<td>1 ppm</td>
</tr>
<tr>
<td>Nickel carbonyl</td>
<td>0.05 ppm</td>
<td>1 ppm</td>
</tr>
<tr>
<td>Ethylene oxide</td>
<td>1 ppm</td>
<td>100 ppm</td>
</tr>
<tr>
<td>Phosgene</td>
<td>0.1 ppm</td>
<td>5 ppm</td>
</tr>
<tr>
<td>Vinyl chloride</td>
<td>5 ppm</td>
<td>500 ppm</td>
</tr>
</tbody>
</table>

Nevertheless, the principle of including articles on occupational health in your journal is most welcome.

DR M. W. DAoud
Medical Advisor, Kuwait Oil Tanker Company,
PO Box 810, Safat, 13009 Kuwait

Sir,

Dr Daoud’s comments are correct. Our figures were taken from various sources, some of which are outdated. I am very thankful to Dr Daoud for his interest.

PROFESSOR MOHAMED EL-SHABRAWY ALI PHD
Chairman, Department of Community & Family Medicine,
King Saud University, PO Box 2925,
Riyadh 11461, Saudi Arabia

Alopecia and Typhoid Fever

Sir,

This letter records two cases of alopecia following typhoid fever. Alopecia may have been one in the long list of complications of typhoid not seen today and rare before the advent of chloramphenicol.1

The two cases, a 21-year-old Jordanian male and 29-year-old Pakistani male, represent 0.2% of enteric fever patients documented in Kuwait for the period 1982–1988. They were remarkably similar and shared the following features: systemic illness; initial therapy with chloramphenicol, gentamicin and metronidazole; blood culture growth of Salmonella typhi; continuance of chloramphenicol to 14 days; gradual improvement and complete recovery with no relapse. Both received metronidazole for 5 days; the Jordanian gentamicin for 7 days and the Pakistani, gentamicin for 10 days.

The Jordanian sustained a 3-week fever prior to admission and presented at follow-up, a total of 7 weeks after initial illness, with alopecia marked over the frontal area. He was referred to a dermatologist who observed spontaneous return of normal hair growth. The initial illness in the Pakistani lasted 2 weeks and alopecia was noted a total 5 weeks later, with generalized thinning and loss of scalp hair. The dermatologist again reported spontaneous improvement although the patient was lost to final follow-up. In both cases, evident hair loss was confined to the scalp with no apparent cause, except for the known association with a preceding febrile illness.

Hair loss following systemic illness, shock or a physiological state such as pregnancy is known as telogen effluvium.2 Here, the anagen phase of hair growth is abruptly followed by the catagen end-stage (particularly of older hairs), manifest by shedding a few weeks later. The resting telogen phase then prevails until hair growth resumes within a few months. Cytotoxic agents are another well-known cause of hair loss. Drug-induced telogen effluvium has been noted with many drugs—hemin, carbamazepine, lithium, indomethacin, allopurinol, levodopa and propranolol—to name but a few as listed in a range of dermatology texts. Gentamicin is also listed but it would be difficult to incriminate in our patients, and especially as it is commonly used in severe infections.

We would not normally administer three drugs in suspected typhoid, but such was the severity of infection and initial diagnostic doubt that we felt obliged to do so in these patients. Emerging resistance patterns will dictate that other drugs, including the aminoglycosides, will be increasingly used for parenteral therapy in severe typhoid. More drug resistant cases with prolonged fever can be expected in this region. Telogen effluvium is still likely to remain a rare complication, but one can confidently reassure the patient of its temporary nature.

SHIHAB AL-SHIBRAWY, MOHAMED AL-SHABRAWY DH DTMAH
Professor, Department of Community & Family Medicine,
Infectious Diseases Hospital, PO Box 4710, 13048 Safat, Kuwait

References

Poor Response to Chloroquine Therapy in a Case of Sudanese Falciparum Malaria

Sir,

The recent article on this subject by Drs Malik et al. (Saudi Med J 1990; 11(2): 143–144) raises some interesting points. I would comment that taking prophylactic chloroquine 1 day before entering an endemic area, although acceptable in the last resort, is less preferable than starting prophylaxis 1 week prior to entry. It would appear that the patient took nine weekly doses, a total of 2.7 g chloroquine base, although it is possible that he may have missed taking consecutive weekly prophylaxis.
It is still believed that chloroquine can be used as the first line of treatment in chloroquine-resistant falciparum infections (CRPF). An examination of the Table 1 in the article reveals that the patient received a total of 6.6 g chloroquine base over a period of 45 days. Unfortunately, after the first course of treatment his blood was not examined for malaria parasites so it is possible that the parasitaemia persisted until his admission to hospital on 14 October 1988. The subsequent clinical progress indicates the presence of R1 chloroquine resistance. However, there is a possibility that initially this may have been an example of chloroquine failure as opposed to chloroquine resistance, and that unless the patient had been closely supervised by the prescribing physician it is also possible that he did not, in fact, ingest a full oral course of chloroquine.

Finally in an area with no transmission, and if the patient did not intend travelling into an endemic area for a further 3 months the treatment of gametocytaemia was not necessary: however, if gametocytaemia was to be treated, a single dose of primaquine 45 mg, was all that was required.

IAN F. M. SAINT-YVES
Al Hada Hospital, PO Box 1347, Taif, Saudi Arabia

Sir,

The points raised by Dr Saint-Yves are well taken. I agree with him that chloroquine prophylaxis is better taken a week before travelling. Chloroquine is still used as the first line of treatment even in some areas with proved chloroquine resistance, since sensitive strains of the parasite could be encountered. However, we have to be alerted to chloroquine resistance since many documented reports are accumulating from Africa and the Far East. Our patient was a microbiology technician working in the College of Medicine. The first two treatments with chloroquine were given before the patient came under our care. Unfortunately, we do not have a record of his blood smears at that stage. After admission to the hospital we were supervising his treatment and blood smears closely and there was no doubt about compliance. I agree that in areas with no transmission it is not necessary to give primaquine, but although there is no documented transmission, Abha City is only 20–30 km from the lowland where transmission occurs.

DR G. M. MAULIK
Assistant Professor of Medicine,
King Saud University, Abha Branch,
College of Medicine, PO Box 641,
Abha, Saudi Arabia
Saudi Medical Journal 1991; 12(2): 159

The Role of Vascular Surgery in Cerebrovascular Disease

Sir,

I totally disagree with the author’s pessimistic view regarding the role of vascular surgery, namely carotid endarterectomy in the prevention of stroke. Carotid endarterectomy has a definitive role in the prevention of stroke in symptomatic stenotic or ulcerated extracranial carotid artery disease. However, its place has not yet been defined in the asymptomatic group. Indeed, few medical issues are more controversial than the management of the latter group, as the natural history is not completely understood. I feel that Dr Bwala has overestimated this by quoting the higher reported figures of stroke and ignoring those rates around 2%.1 It is difficult to state that vascular surgery has not been demonstrated to be convincingly more successful than medical therapy alone, as Dr Bwala concluded. We will have to wait for the results of prospective, randomized clinical trials, such as those by the Veteran Administration (VA) and the National Institutes of Health which have been on-going in the USA since 1983 and 1987 respectively.2,3 It will be another 2 years before conclusive data are reported from the VA trial and perhaps an additional period of time before the NIH trial results are available. Meanwhile, as a vascular surgeon, I recommend prophylactic carotid endarterectomy for patients with high-grade stenosis, i.e. more than 75% reduction in the artery’s cross-sectional diameter, as detected by non-invasive techniques. Those patients with asymptomatic carotid artery stenosis should be observed by non-invasive techniques to allow early detection of cases progressing to high grade stenosis, who are therefore at a high risk of immediate occlusion, which is associated with an unacceptable incidence of immediate neurological sequelae (25%).4

I personally feel that aggressive follow-up is indicated for patients who have established contralateral occlusion and asymptomatic ipsilateral carotid artery stenosis. In certain cases, carotid endarterectomy may be performed at an earlier stage (<75% stenosis).

Finally, I would like to see this subject reviewed again in two to three years’ time after the completion of the on-going studies. Moreover, at that time, the subject of cerebrovascular disease might be looked at from a different angle, i.e. the point of vascular surgery.

HASAN ALI AL ZAHRAI FRCSE(Edinburgh) FICA
Assistant Professor of Vascular Surgery,
King Abdulaziz University, College of Medicine,
Department of Surgery, PO Box 6615, Jeddah 21452, Saudi Arabia
Saudi Medical Journal 1991; 12(2): 159

References
SIR,

I note the points raised by Dr Al Zahraini which centre on carotid endarterectomy. I agree with him that the management of cerebrovascular disease is one of the most controversial topics in neurology and neurosurgery. As such I have taken care to be as fair and objective as possible. That is why I have cited multicentre reviews rather than single hospital studies. A recent up-to-date review of the subject testifies to the controversies and uncertainties. In this, like in any controversial subject, there is a spectrum of views and practice. It is true that some centres have low morbidities and mortalities but at the same time it is important to mention centres with poor results to enable both patient and doctor to choose the safest and most effective treatment. The article published did not condemn carotid endarterectomy in its entirety. Rather, in view of the uncertainty of its superiority over medical treatment alone, it should be restricted to high risk groups and to centres which have low morbidities and mortalities to justify their practice until the matter is fully elucidated. This should not be interpreted as pessimism but as advice which is in the patient's interest. In fact, there has been a dramatic decline in the use of carotid endarterectomy in the USA in the light of the ongoing controversy. I think this is a prudent development safeguarding the patient whilst we keenly await the results of ongoing trials. A similar trend obtains in extracranial–intracranial bypass surgery in the aftermath of the disappointing multicentre trials.

I share Dr Al Zahraini's view that the subject in question will need to be updated as results of trials reach their conclusions. Future developments may be influenced by the prediction of patients likely to benefit from surgery by the use of positron emission tomography scan (PET-Scan) or single photon emission tomography (SPECT). However, the expense and complexity of PET-Scan limits its use to research units. SPECT being cheaper and easier to operate lends itself to clinical use for the determination of the vasomotor reserve of the central vessels using carbon dioxide. Alternatives to endarterectomy might develop along the line of percutaneous transluminal angioplasty and laser thrombendarterectomy. The procedures are still in their infancy and face many obstacles but may prove to be useful in selected groups of patients.

In conclusion, my article was written to highlight the uncertainties that obtain in the management of cerebrovascular disease. Rather than pessimism, it is cautious optimism that should prevail to safeguard patients' interests until the safest and most effective treatment is determined.

S. A. BWAJ, MRCPI(UK), FMCPS
Consultant Physician, King Khalid Military City Hospital, PO Box 807, Hafr Al-Batin, 31991, Saudi Arabia

References

Sir,

The article by Dr S. Bwala (Saudi Med J 1990; 11(2): 83–86) was very interesting. Although the asymptomatic carotid bruit remains a controversial issue, I agree with many points the author raised, but he goes on to say that 'Surgery had no effect compared with medical therapy alone in preventing cerebral ischemic events'. In the next paragraph, he went further to say that ' . . . the disturbing fact is that the complications are unacceptably high . . . more likely to sustain stroke as complication'.

Firstly, it appears, by lumping them together that the author ignored the fact that various types of lesions involving the carotid artery have different outcomes. If the pathology is an ulcer, and if it is small, then the use of antiplatelet therapy may be enough. For an excavating, or compound ulcer, or for an occluding atheroma, or subtotal haemorrhage, however, antiplatelet drugs, regardless of the dose, or duration of administration have no effect. For these cases at the present time, surgical intervention is the answer.

Secondly, the author should have given more consideration to the natural history of these lesions. Less than 70% narrowing of the carotid lumen, has a low probability of being associated with stroke, while 75% occlusion or more has a higher chance of leading to stroke.

The third issue is the bilaterality of the disease. In other words, when both sides are involved, the chances of a stroke occurring are greater.

All these dictate appropriate but definitive treatment rather than the use of antiplatelet drugs alone.

Finally, in special units for cerebrovascular diseases, the surgical complications (including stroke), can be kept well below 3%.

S. MAJED AL-AMANI MD
Unit Thoracic & Cardiovascular Surgery, Department of Surgery, King Fahd Hospital of the University, PO Box 2931, Al-Khobar 31525, Saudi Arabia

References

Sir,

I am pleased to read Dr Al-Awami’s letter and note the points he raised. The population of patients with carotid vascular disease is not a homogenous one pathologically. Certainly there is a subpopulation of these patients with the type of lesions enumerated by him. This was the reason why I pointed out that the number of patients subjected to the procedure should be restricted. For the sake of brevity I did not expand on this subpopulation. I agree that there are good centres with low complication rates for carotid endarterectomy. The procedure should be restricted to such centres in general in view of the current controversy and dare I say medicolegal climate in the USA. In fact, on the latter point, guidelines and audit procedures have been proposed in the patients’ interests pending the clarification of the controversy. Caution is the watchword as testified by the recent death rate from the number of carotid endarterectomies performed. The search continues.

S. A. Bwala MBChB(uk) FMCP
Consultant Physician, King Khalid Military City Hospital, Northern Area Armed Forces Hospital, PO Box 807, Hafir Al-Batin 31991, Saudi Arabia

References

Foods Precipitating Myocardial Infarction

Sir,

'Some foods scald the heart, others scald the brain'. Imhotep

The original concept that myocardial infarction had triggering events that preceded it by minutes or hours

was later overshadowed by the rather authoritative statement of Master in 1960 that 'coronary occlusion takes place irrespective of the physical activity being performed or the type of rest taken.' This erroneous assumption of Master prevailed until recently despite the abundance of anecdotal coupling chronologically of unaccustomed physical activity, panic, sudden grief, financial losses, sexual intercourse and even an overdose of happiness with myocardial infarction and cerebrovascular accidents.

The discovery in 1985 of a definite circadian pattern in the frequency of myocardial infarction and its confirmation later by other large studies and the documentation of changes in the blood pressure, platelet aggregability, tissue type plasminogen activity, plasma cortisol, plasma epinephrine and that all correlated well with this circadian pattern finally toppled Master's view on this subject.

The role of diet however has not been updated in this context and continues to be investigated with respect to its long-term effects on myocardial infarction and strokes. The adverse consequences of a heavy meal are assumed to be only due to the increased haemodynamic load on the susceptible heart during digestion as in exercise, thus totally disregarding any effect the end products of digested food could have on the coagulability, viscosity, tissue type plasminogen activity of blood and on the calibre of the coronary arteries during the hours following ingestion.

So many drugs are now known to have either salutary or adverse effects on these parameters. Is it conceivable that among the thousands of ordinary and exotic food items none could have similar effects? So far only anecdotal evidence exists chronologically linking the consumption of cheese and wine to myocardial infarction and the consumption of yogurt and fish (Arab folklore) to cerebrovascular accidents.

In view of this embarrassing deficiency on the subject in the medical literature, I envisage a simple prospective study that could be easily performed in any hospital that has a coronary care unit or a neurology unit.

A detailed questionnaire concerning the food items ingested by the patient during the 24 hours preceding the documented myocardial infarction or stroke should be designed. The questions would be directed to the relatives of the patients, or even better to patients themselves after they have stabilized.

After several hundred or thousand questionnaires are completed and analysed by computers, certain patterns might emerge that would unfortunately have us add yet another list of food items to be avoided (or others to be more often consumed) by atherosclerotic patients and the general population prophylactically.

Bochloe L. Artigaan MD FMCP
281 Oak Avenue, Riveredge N.J. 07661, USA

References
2Master AM. The role of effort and occupation (including physicians) in coronary occlusion. JAMA 1960; 174: 942-948.
Sir,

I wish to thank Dr Maghribi for his careful assessment of our article. My colleagues and I were led to write it in the belief that the social aspects of illness and disease need closer attention from clinicians in the Middle East. We are delighted that our article sparked the interest and critical attention of Dr Maghribi.

The brunt of Dr Maghribi’s critique falls upon Table 1: Health status indices in underdeveloped African nations, developing Arab nations, and developed European and North American nations. He argues that our category of ‘developing Arab nations’ is so heterogeneous that it cannot constitute a single category.

We accept the particular facts that Dr Maghribi states. We wholly endorse his strong call for analysis based on clearly defined, internally homogeneous categories. We do not, however, see in his remarks the elements of a better strategy for analysis, nor are we persuaded that the course we have followed is vague or misleading.

At its simplest our argument holds that the three groups of nations — Group A, Group B, and Group C — stand at different levels on an independent (causative in a broad sense) scale that we call ‘development’. (We do not claim that we have precisely defined this variable, nor do we regard a precise definition as essential for the purpose of our analysis.) We then proceed to set forth several health consequences or correlates that we regard as dependent variables.

What of Category B, the Arab nations? Setting aside for the moment our focus on health variables, we can say that the Arab nations are a relatively homogeneous group in their language (Arabic), their culture (Arab), and their religious tradition (Muslim). We say ‘relatively’ homogeneous in recognition of the fact that among the Category B nations there exist identifiable groups of non-Arabic speakers and persons who are non-Muslim in religious profession. Nonetheless, ‘Arab nations’ is a meaningful category or interpretive unit of analysis.

Now we return to our examination of health variables. It so happens — in an empirical sense, the contemporary world is thus constructed — that the Arab nations form a meaningful category in terms of health processes. For the health variables we studied — life expectancy, infant mortality, and daily calorie supply — the Arab nations have a set of distinctive values, overlapping only to a small degree the values of Categories A and C.

The internal variations that Dr Maghribi points out do not contradict this conclusion. It really does not matter that some of the Arab nations have long seacoasts and others do not; nor that Libya lacks a metropolis while Morocco, Egypt, and Iraq each possesses one or more cities with a population of two million or more.

We do not, however, dismiss with the same ease the marked per-capita-income variations within Category B, to which Dr Maghribi alludes. Income and wealth do not directly procure or guarantee favourable health status, but they can at least provide the material base for health care resources and for living conditions — food and shelter — conducive to health. Even so, there are compelling reasons why we do not believe that this source of variation defeats the thrust of our argument. Per-capita-income is merely an average figure, which may well conceal huge socioeconomic differentials within a nation.

Medical Sociology Outlined and Applied to the Arab Middle East

Sir,

The aim of this letter is to draw your attention to one aspect of the valuable study on this subject published in your journal (Saudi Med J 1989; 10: 441–445); this is the non-homogeneity of the group of countries referred to as Arab (Group B) in Table 1. This stands for a highly non-homogeneous group from different aspects relevant to the study. Here are examples of the non-homogeneity:

(a) geographical distribution: e.g. the countries of the Mediterranean basin were mixed with countries such as Saudi Arabia (desert and Asian) and Iraq (non-Mediterranean and Asian — with a very small coastal margin). Such discrepancies were apparently not present in Group A for example.

(b) Population concentration: high contrast between densely populated areas such as Cairo, Egypt, and Libya which has a low population concentration. Such a difference does not equally exist between UK and France.

(c) Variability of ethnic groups: e.g. Kurds of north Iraq and Bar-Bar of Algeria.

(d) Unemployment rate: this is as high as 25% in Egypt and is virtually nil in Saudi Arabia. Such a difference cannot be detected between the unemployment rates in France and West Germany.

(e) Gross national product per capita: this is the main defect in the argument for homogeneity. The study quoted mixed the highly rich OPEC countries such as Saudi Arabia and Libya with such countries as Egypt and Algeria which have the highest foreign debt and the largest foreign debt per capita respectively. Such a fact cannot be detected equally in Groups A and B.

These facts are relevant to medical sociology as they affect many aspects of the health of society as e.g. disease distribution and medical resources.

With the Arabic group itself recognizing its own potential and actual variations, such facts should have been recognized. I cannot (being a non-specialist) measure the actual and full impact of these speculations on the published studies.

Ahmed H. Maghribi
Al Barsha Building (Room 7)
Al Awkef Street, Saida, Lebanon
On the other hand, a relatively poor nation might with wise distribution and deployment of its limited resources stand high on health status indicators. The health experience of several Latin nations suggests a threshold effect, whereby a modest investment yields large dividends.

EUGENE BENNETT GALLAGHER PhD, Professor
Hotel Intercontinental, Room 721,
PO Box 163, Al-Ain, United Arab Emirates

Age at Menarche of Schoolgirls in the Asir Region, Saudi Arabia

Sir,

An article on this subject appeared in a recent issue of the journal (Saudi Med J 1990; 11(1): 59-63).

Although the presentation of the paper is fairly clear, one feels that there are several omissions and inaccurate statements, which, in my view, make its appreciation rather difficult. The authors start by saying that there are great variations between the age at menarche in different populations in many countries. A cursory look at Table 4 clearly indicates that this is not so. There are differences, but it is doubtful whether these are statistically significant, especially in the absence of any indication of the variation in the data (i.e. SD). Also data presented in Table 4 were obtained at different times (for example some in 1966 and others in 1983).

The mean age at menarche for the small families was 13.07 ± 0.16, and 13.81 ± 0.15 for the large families. The difference may be statistically significant by the method employed by the authors. Whether this difference is biologically, medically or socially significant is unclear. This point should have been elaborated in the Discussion.

The authors state that age at menarche was found to be independent of size at maturity. This, if anything, would detract from the importance of determining age at menarche.

The authors think that with improved health care and family planning, young Saudi women would be as early in maturing as girls in Northern or Central Europe. Yet, another look at Table 4 shows that the menarche in London, for example, was 13.0 years and in Sweden 13.1 years. Is this significantly different from the menarche of Saudi girls reported in this study? Incidentally, in Havana (Cuba) the menarche was found to be 12.8 years. Surely the socioeconomic conditions in the latter country are worse than in Saudi Arabia.

As a conclusion the authors state that 'It will be very important to investigate the age at menarche from generation to generation at opportune intervals for the evaluation and monitoring of any national health programme.' What does this mean exactly?

BADREDIN HAMID ALI PhD
Department of Pharmacology, Al-Arab Medical University,
Faculty of Medicine, PO Box 18251, Benghazi, Libya

Sir,

I will respond to the points in Dr Ali's letter in the same sequence as they appear.

(1) Dr Ali starts by saying that 'One feels that there are several omissions and inaccurate statements ...', e.g. by reporting that there are great variations between the ages at menarche in different populations in many countries.

It is expected that the reader would have some idea about geographical polymorphism in somatic growth among different populations of mankind. Rate of maturation is influenced both by heredity and environment. Identical twin sisters growing up together in a good environment differ in age at menarche only by one or two months; sisters by an average of nearly one year. Environment exerts a powerful effect on the growing child. It either contributes positively to the fulfillment of a genetic plan or hinders it. Poorly off children, have a later menarche than better off children in all populations studied. Also, urban girls are earlier than rural girls in every rural-urban comparison so far reported. These rural-urban and social class differences were demonstrated clearly in Figure 4 of our article.

Although we reported the mean values for some countries without mentioning the variances or the standard errors of the mean to make it as simple as possible, the interested reader can refer to the references cited. In them will be found a vast collection of data, which supply the bibliographic background for the statements mentioned.

In poor rural areas in the southern USA, 30 undernourished Euro-American girls followed longitudinally had a mean menarcheal age of 14.4 years against 12.4 in the well-nourished controls. Malnutrition, disease and family size are the main environmental factors which could delay growth of children, and which we hope to eliminate or reduce by national health programmes. Indeed, one of the first effects of undernutrition is to slow down growth and postpone the ages of appearance of the various stages of physical maturity, e.g. secondary sexual characters and menarche.

(2) In our survey the mean age at menarche was 13.07 ± 0.16 for the small families; 13.81 ± 0.15 years for the large families and 13.48 ± 0.08 for the pooled data.

As we stated under Subjects, the two groups lived under more or less the same socioeconomic conditions, and differed only in family size. This point was elaborated in the Discussion section (p. 61), family size as a socioeconomic factor, represents a direct environmental effect and may reflect concealed poverty possibly acting on maturation through nutrition, parental attention and health care. Indeed, families spend less per head on food as the number of children in them increases. The effect of family size on growth and maturation is illustrated by data from the London County Council, and in the five West European longitudinal studies co-ordinated by the International Children's Centre (at London, Paris, Brussels, Zurich and Stockholm).

(3) In his penultimate paragraph, Dr Ali said 'The authors think that with improved health care and family planning, young Saudi women would be as early in maturing as girls in Northern or Central Europe ...'. As we stated in our study, more privileged girls with small
families and better educated parents have a mean age at menarche of 13.07 which is close to European values. On the other hand, girls from large families have a mean age of 13.81 years. This leaves no doubt that with improvement in health care and family planning, Saudi girls would be as early in their maturation as European girls. We disagree with Dr Ali that socioeconomic conditions in Havana (Cuba) are worse than in the semi-urban community we studied, although the economic conditions of Saudi Arabia are much better than in Cuba.

(4) As we stated clearly in the Introduction and in the Conclusions (p. 63), due to improvement of socioeconomic conditions over the last century, age at menarche in Europe, North America and even in some developing countries in Asia and Africa has shown a general downward trend. This tendency for children to mature earlier and to be bigger at all ages from generation to generation is known as the secular trend. This trend is coming to an end in the Western Hemisphere, but still operating in developing countries. In nineteenth century England menarche occurred on average around 15.0 years and in Norway at nearly 17.0 years.

In rapidly developing countries such as Saudi Arabia, improvement in the health and nutrition of children should lead to earlier menarche and a greater stature at each age. Therefore, the occurrence of a secular trend in a developing community is a useful index of progress and a good sign reflecting the success of the social and health welfare systems. Thus, comes the importance of revising growth standards including menarche from generation to generation.

Finally, I take this opportunity to clarify that the results reported by our study are based on a semi-urban community in the Asir region of Saudi Arabia and cannot be extrapolated to the whole Saudi population. A nationwide survey would be required to understand the situation throughout the Kingdom.

References


Development of Nutritional Adequacy and Health Status in Saudi Arabia

Sir,

We congratulate Dr Almokhalalati on his excellent review (Saudi Med J 1990; 11(1): 18–24). Your readers may be interested in a study which we published after Dr Almokhalalati’s article was written in which we investigated the iron status and haematology of 9-month-old Saudi Bedouin infants living in Western Saudi Arabia. Hypochromic anaemia was common but only a minority of infants had serum ferritin levels indicative of iron deficiency. DNA studies showed that α-thalassaemia was probably a more important cause of hypochromic anaemia than had presumably been realized. The proportion of infants with serum ferritin levels below 10 µg/l (13/138–9%) was lower than that of both Caucasian and Asian infants of the same age in an urban area of the UK. Comparison of the Saudi infants we studied with those of the same age in the UK shows that the most striking difference in diet is not in the age of introduction of solid foods but in the earlier introduction of bottle feeding in the UK and the widespread use of fresh unmodified cows milk in UK infants during the second 6 months of life. Iron is well absorbed from breast milk and exclusively breast fed infants can maintain normal serum ferritin levels for the first 6–9 months of life. Fresh unmodified cows milk is not only a very poor source of iron but is known to cause significant blood loss from the gut in infancy and infants fed with formula milk have higher haemoglobin and serum ferritin levels than those fed with unmodified cows’ milk.

In our view it would be inadvisable for Saudi mothers to adopt the practice of giving their babies fresh unmodified cow’s milk in the first year of life. It is probable that iron deficiency anaemia could be avoided in almost all breast fed Saudi infants in the first year of life if adequate amounts of iron containing solid foods were introduced by 6 months of age. If breast feeding cannot be maintained for the first year of life, then iron supplemented infant formula feeds should be used rather than fresh unmodified cow’s milk.

References

Sir,

I would like to thank Drs Stevens and Ayoub for their valuable comments and for drawing attention to their interesting findings.

As the aim of my report was limited to providing information on the nature and magnitude of malnutrition in the Kingdom, I have not discussed in details the causes of microcytic hypochromic anaemia which are many; iron deficiency is only one cause and a number of factors such as malaria, hookworm infections and some red cells genetic disorders (e.g. thalassaemia and sickle cell diseases) may lead to such a condition. Studies carried out in the Kingdom demonstrate the occurrence of sickle cell, thalassaemia and glucose-6-phosphate deficiency genes at a level which is higher than other countries.¹⁻⁴

In one of these studies it was reported that sickle cell haemoglobin was detected in about 8–27% of the population (school children and adults living in villages in the western region of Saudi Arabia), the prevalence of G-6-P deficiency ranged between 8 and 15% in males in different villages, whilst α- and β-thalassaemia varied between 20–30% and 10–15% respectively. As hypochromic microcytic anaemia is associated with these inherited disorders in the absence of iron deficiency, it seems reasonable to suggest that hereditary disorders of haemoglobin could account for some of the anaemia thought to be due to iron deficiency in Saudi infants studied where these inherited diseases are common.

On these grounds, it is important to suggest that more studies on the relative role of iron deficiency and red cell genetic disorders in the pathogenesis of hypochromic anaemia are needed.

Regarding the view that iron deficiency anaemia could be avoided in the first year of life if adequate amounts of iron containing foods were introduced by 6 months of age, it certainly fits the current recommendation issued by the American Academy of Paediatrics (AAP, Committee on Nutrition) and the European Society for Paediatrics Gastroenterology and Nutrition (ESPGAN, Committee on Nutrition).²

A review of studies in the Arabian Gulf Countries³ indicated that raw cow's milk and non-iron fortified formulae were commonly used in many countries in the region. It was calculated that such food does not exceed 5.0 mg of iron/day while the recommended dietary allowances for infants during the second half of the first year was estimated to be between 10 to 15 mg/day.⁵,⁶ Therefore, to fulfil the iron requirement of an infant, from 6 to 12 months of age, consuming about 500 ml of milk/day, an iron concentration of not less than 1 mg of iron/100 available calories in a follow-up formula is needed.⁶,⁷ In these grounds the use of iron-fortified formula has increased steadily in recent years from 40% of the US infant formula sales in 1971 to about 80% in 1985.⁷ In brief, one could conclude that breast feeding or starting formula should not be replaced by unmodified cow's milk, but should be replaced by iron-rich follow-up formula and solid food.

References