Viewpoint

Rwanda: a case of demographic entrapment

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In 1990, *The Lancet* published the first of three articles by King about demographic entrapment.[1-3] He argued convincingly that many regions in the world are severely endangered by the growth of their own populations. Few will disagree with him or with others who describe the intolerable burden of an exploding population in an environment of scarce resources.[4] What was new was the agonising dilemma that King presented to public health policy-makers. Primary health care, according to King, should refrain from targeting child mortality, and, for example, should withhold highly effective oral rehydration therapy to the population at large, because more surviving children boost population growth and thereby close the demographic trap.

Rwanda: a case study

Rwanda, as King and Elliott noted in the third article,[3] is a sad example of the consequences of demographic entrapment. In 1993, the population density was 285/km^2, which is 50 more than in the UK; half of Rwanda's citizens were under 15 years of age. Less than 10% lived in cities; most were living "up the hill", in fragile ecosystems that were fast eroding due to deforestation and unsustaining agriculture. Nevertheless, 40% of the gross domestic product was generated by agriculture, there being virtually no other industries in the country.[5] In 1993, 5·2 children were born per 100 of the population; each woman is expected to have 8·5 children during her life time at the present birth rates.[6] While population growth rates remained well above 3% from 1980 onwards, economic growth stagnated at 0·5%, an indication of increasing empoverishment. The young and dynamic—peasants without land, shepherds without herds, workers without labour—chose to fight their way out of the trap, and clear the scarce farmlands of their competitors.[4]

Yet if Rwanda shows us the devastating power of the closing demographic trap, we have empirical data to study how it came that far. The table shows demographic data from Rwanda since 1970. We have no good figures of under 5 (child) mortality in 1970, but these must have been high—there was no organised primary health care, no immunisation programme covering the highly lethal measles, tetanus, or whooping cough, and no oral rehydration techniques. Between 1985 and 1993, child mortality declined from 22·3% to 19·8% (table). Extrapolation of that change backwards to 1970 yields a child mortality of 28%, a reasonable estimate for a sub-Saharan country in that period.[6]

With that background, calculation of the effect of decreasing child mortality on population growth is easy. The birth rate remained more or less constant in Rwanda at 5·1–5·2 per 100. In 1970, for every 5 children born, 3·5 would have survived their fifth birthday whereas in 1993 4 will—ie, an extra 0·5 will participate in the population growth. Over the observed period, population growth rate indeed increased by 0·5%; most of this increase is probably attributable to decreased child mortality.

What would have been the effect of maintaining a high child death rate, at the level of 1970? The growth rate would have remained at 2·9%, a population doubling time of 24·2 years. The actual doubling time between 1970 and 1993 was 22·1 years, a figure that accords with a gradual decline of child death rates from 30% to 20% and a concomitant increase in growth rate from 2·9% to 3·4%. Thus keeping child death rates constant at around 30% would have resulted in a gain of only 2 years.

Population growth is determined mainly by birth rates not by death rates

Once a child is born, whether 0·7 or 0·8 will reach their fifth birthday is inconsequential. The drama of Rwanda was not the decline of child mortality from 30% to 20% but the constant birth rates above 5%. A decline in fertility of only 10% would have offset that child mortality decline.

The argument that we have no time to lose, and that desperate times need desperate solutions, does not hold. The time we gain by maintaining high child death rates is trivial, and will not prevent the demographic trap from springing. As has often happened before, well-intentioned doctors have made the mistake of overrating the effects they have on population health.[7] The good news is that there is no agonising moral dilemma for doctors, public health, and primary health care—and there is and never was a reason to let the children die an easily preventable death. The bad news is that the international community stood by and watched Rwanda die from excess fertility—20 years ago mathematical models predicted its complete collapse.[8] There was no way that this small, poor, rural, ecologically fragile country could sustain such population density and increase.[8] Rwanda has a very Catholic population and a powerful Catholic church, with a long tradition of strong opposition to family planning.[9] Whereas highly educated Roman Catholic women in the West can easily dismiss the teachings of Rome, this is much harder for the illiterate poor of Rwanda, who find solace in religion. The Hutu extremists have been called possessed by devils by Western press. I hold that those

	Population ×1000	Birth per 100	Death per 100	Growth per 100	Under 5 mortality per 100*
1970	3790	5·1	2·2	2·9	28
1985	6274	5·2	1·9	3·3	22·3
1993	7790	5·2	1·8	3·4	19·8

World Health Statistics Annuals, 1970–1993; the under-5 mortality of 1970 is the author's estimate. *Probability that a liveborn child will die before his or her fifth birthday.

Table: **Selected demographic figures for Rwanda**

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who believe in devils should forget about studying the state of mind of the desperate Rwandese and concentrate on the devastating consequences of denying effective family planning to the poor of this world, who are smothering each other through the sheer growth of their numbers.

References

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BOOKSHELF

Hand eczema

Edited by Torkil Menné and Howard I Maibach. Boca Raton: CRC Press. 1994. Pp 334. $103. ISBN 0-849373557.

Remarkably this is the first focused reference source on the very common, often vexing, chronic, and even disabling problem of hand eczema. Up to 10% of dermatological practice and as much as 30% of occupational and industrial medical practice centre on this disorder which also features prominently in medical litigation, workmen's compensation, and disability issues. This new volume of skilfully assembled contributions, mostly from Scandinavian and northern European experts and investigators, now fills the void for such a basic reference text.

The book reflects current widespread confusion about the definition of two key concepts—namely eczema and atopy, as highlighted in a masterful introductory chapter by Wilkinson and in four later chapters by Schwanitz, Veien and Menné, Rystedt, and Diepgen and Fartasch in which the atopic state in relation to various aspects of hand eczema is extensively considered. Although different views about definition and clarification of these concepts and their historical backgrounds are of value to specialists, generalists are likely to sense mainly the confusion. For them, eczema as a large subset of dermatitis can be viewed simply as a very common superficial cutaneous inflammatory reaction pattern resulting from keratinocyte injury, brought about either by directly injurious physical or chemical irritants and/or by cytokines associated with cell-mediated delayed-type allergy. This reaction pattern is focused in the outermost couple of tenths of a mm of skin; its histopathological hallmark is spongiosis (intercellular oedema between keratinocytes) probably as a result of increased extracellular protein osmotic forces caused by leakage of protein from autolysing injured cells. Subsequent reparative epidermopoietic stimulation may overcompensate, and in subacute and chronic phases of the reaction result in parakeratotic hyperkeratosis and epidermal thickening (acanthosis). Erythema and surface change (vesiculation and/or serous oozing in acute, scaling in subacute, and lichenification in chronic phases) are the resultant key clinical morphological manifestations of the eczematous pattern while itching and itch hyperexcitability are its symptomatic hallmarks.

From the clinician's perspective, once the eczematous pattern is recognised, the important issue is to analyse the specific factors in any particular case that have caused or are aggravating the epidermal injury and to determine how to eliminate or counter them. The management of eczema or superficial skin failure is analogous to the management of heart failure: clinical recognition, understanding of pathophysiology, analysis of specific aetiological factors in the particular case, and management by elimination of underlying causes where possible and otherwise by empirically useful measures. This book on hand eczema provides a great wealth of detailed information to assist clinical recognition and analysis for aetiological factors particularly in the occupational setting. Special chapters are devoted to the problem in hairdressers, fish industry workers, farmers, construction workers, dental personnel, metal workers, hospital workers, caterers, and those using rubber gloves.

The extensive attention given to the atopic state and nickel contact allergy in this book will interest specialists especially. The atopic state can simply be considered a constitutional (genetically determined) classification category that includes about 20% of the population. The categorisation is based on weal and erythema-type immediate immunoglobulin-based cutaneous allergies to common environmental allergens. Over the years, this defining immunological characteristic has been found to cluster with various phenotypic traits as reviewed in chapters by Rystedt and by Diepgen and Fartasch. Only when chronic eczema occurs in this constitutional setting should it be designated atopic eczema, which often has special clinical aspects and may require special management strategies.

In a multiauthored sharply focused book such as this there is considerable redundancy but this overlap provides useful reinforcement and better illuminates the subject. Thestrup-Pedersen succinctly reviews the basic science of skin inflammation. The fascinating roles of adhesion molecules, inflammatory cell trafficking, major histocompatibility antigens, and cytokines are considered and speculated about, but alas this basic information has yet to be sufficiently integrated into the clinical setting to be practically useful. Missing from consideration is discussion of the possible role of cell-mediated autoimmune sensitisation to epidermal injury antigens as a potential mechanism for the stubborn chronicity, poor prognosis, easy recurrence, and tendency to spread of many hand eczemas. This omission is especially puzzling in view of the fact that the English dermatologist, Whitfield, in 1921 on this basis introduced the idea of autoimmunity as a pathogenic mechanism into medical thinking. Major therapies for hand eczemas are