



Because there is no reason to suppose that I is correlated with P, except in so far as the trends in them might by chance coincide, on taking logarithms and expanding

$$\begin{array}{rl} \log_{10} \ L \ = \ \log_{10} \ (1 - \mathbf{k}\bar{P}) \ - \ \log_{10} \ (1 - \bar{P}) \ + \ 0.43429 \\ [\mathbf{k}/(1 - \mathbf{k}\bar{P}) \ - \ 1/(1 - \bar{P})] \ (\bar{P} - \bar{P}) \ + \ T \ + \ e \end{array}$$

where \overline{P} is the mean value of P; T, trend in $\log_{10} I$ and can be represented by a low order polynomial in time; e, variation about trend in $\log_{10} I$ and is independent of P.

So, in the multiple regression of $\log_{10} L$ on P and low order powers of time, the coefficient of P is 0.43429 $[k/(1-k\bar{P})-1/(1-\bar{P})]$ and this may be estimated by the usual multiple regression techniques, hence k can be found.

No allowance was made for the fact that the older children had had a slightly longer time in which to die from pneumonia while incubating leukaemia than the younger children. But, even ignoring this factor, the leukaemia blocking effects of pneumonia deaths were clearly much more marked for the older than for the younger children, for the estimated pneumonia death rates for preleukaemic children were thirty-six times higher than normal for children who would otherwise have died from leukaemia between 2 and 4 years of age, and only four times higher than normal for children who would otherwise have died from leukaemia before the age of 2 years. Hence pneumonia alone probably accounts for about half of the observed increase in leukaemic mortality.

Because preleukaemia and leukaemia must be the result of a continuous process with important side effects on haemopoietic tissues, we tentatively suggest that other side effects should be expected if any haemopoietic neoplasm is in competition not only with the temporary effects of bacterial infections but also with the more lasting effects of parasitic infections. Evidence that spleen and lymph node blocking effects are incurred as a result of malaria or kalazar is purely circumstantial, but it includes (a) the rarity of lymphatic leukaemias in China, Japan, Malaya and India⁹⁻¹¹; (b) the anatomical distribution of the tumours found in cases of African lymphoma (that is, jaws, eyes, ovaries, and so $on)^{12}$; (c) the geographical distributions of these cases and their tendency to "drift"'13; (d) the fact that the commonest type of juvenile neoplasm is lymphoma in the tropics¹⁴ and leukaemia in Europe and North America.

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Lactase Deficiency

Bolin and Davis¹ have reported a lactase deficiency in non-Caucasians. There is an increasing interest in lactase and it could be important to have our baselines correct. Western communities now live on comparatively high protein diets. We tend to think that we are meat eaters, but in fact the figures published by the Food and Agriculture Organization of the United Nations in 1966 illustrate that a large proportion of our animal protein comes from milk (Table 1). Although some African minority groups use milk as adults, most non-Caucasians use insignificant amounts, relying largely on cereals or fish. They will, however, drink their mothers' milk for perhaps the first 9-24 months of life and presumably have adequate lactase during this period.

Table	1.	EXAMP	LES	OF	MEAT	AND	MILK	PR	OTEIN	AVAII	LABLE	\mathbf{IN}	SOME
CAUCAS	IAN	AND	NON	-CAU	ICASIA	N CO	UNTRIF	s	FROM	FAO	REPOR	\mathbf{r}	(1966)

	Before	e 1939	1964		
	Meat	Milk	Meat	Milk	
United Kingdom United States Australia	$10\ 6\ 14\ 4\ 24\ 0$	$5.1 \\ 7.2 \\ 5.0$	$11.8 \\ 20.4 \\ 22.0$	$7.6 \\ 8.6 \\ 7.2$	
Philippines Japan China (Taiwan)	3·0 0·6 3·7		$\frac{2.7}{2.0}$ 3.7	1.1	

Bolin and Davis make the interesting suggestion that the "defect" reflects a low level of milk consumption. Perhaps one should go further: if lactase is analogous to the inducible enzymes it might well be proper for it to disappear on weaning. Our curious habit of lifelong suckling by remote control could perpetuate the enzyme. It may be more useful to think of Caucasians as being odd. The idea that non-Caucasians have a "deficiency' of lactase would imply a genetic or nutritional disturbance as opposed to a simple "food dependence mechanism". This view could be misleading. Looking at this matter the other way round seems to add interest to the question of Caucasian food habits. It might be quite valuable to know if the enzyme can be regenerated in non-Caucasian adults.

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Selective Passage of Prostaglandins across the Lung

STIMULATION of the splenic nerves releases prostaglandins from the spleen to give concentrations of as much as $0.2 \ \mu g/ml.$ in splenic venous blood¹. The release of prostaglandins might influence organs remote from the