WIDER
WORKING PAPERS

Adapting to Undernourishment
The Clinical Evidence and Its Implications

by Partha Dasgupta and
Debraj Ray

WP 10 April 1987

World Institute for Development Economics Research
United Nations University

ADAPTING TO UNDERNOURISHMENT

THE CLINICAL EVIDENCE AND ITS IMPLICATIONS

by

Partha Dasgupta

(University of Cambridge)

and

Debraj Ray

(Indian Statistical Institute)

(First Version)
June 1986

(Revised Version)
October 1986

This article was prepared for the WIDER conference on Poverty and Hunger: the poorest billion, held in Helsinki, July 21-25, 1986. We have benefited greatly from discussions with Devaki Bhaya, A. Das Gupta, Barbara Harriss, Michael Lipton, S Osmani, and Manoj Panda.

1. INTRODUCTION

In estimating the prevalence of undernourishment in a region or country it has been common practice to choose a benchmark - or, as some would say, a critical limit - which reflects nutrition requirements and then to calculate the percentage of the population falling below the benchmark. (See e.g. Dandekar and Rath, 1971; Reutlinger and Selowsky, 1976; and FAO, 1978.) The logic underlying the choice of the benchmark has varied across studies. But a common driving hypothesis has been that a person's long run nutrition requirements are more or less fixed and that the variation in requirements often observed across otherwise similar people is to be explained largely by differences in their innate physiological characteristics. In other words, it has been assumed that interpersonal variations in nutrition requirements dwarf intrapersonal variations. This common hypothesis has been given its sharpest articulation in a study by Reutlinger and Alderman (1980) who, in estimating the extent of world-wide undernourishment, have dispensed with the exclusive use of overall regional benchmarks and have worked directly with a statistical distribution of individual intakes and requirements.

This common underlying hypothesis has recently come under sharp attack, (Sukhatme and Margen, 1978, 1982; Sukhatme, 1981. The attack is easy to describe, but its validity is far from simple to assess. It consists of the claim that the nutrition requirement of any given individual varies in the long run over a wide range, and that this variation is achieved through an auto-regulatory process of adjustment of body metabolism. In other words, variations in nutrition intake within this range do not involve any significant alteration in the persons' weight, or in his body composition, or indeed in his physical and mental capabilities. Or, to put it in yet another way, the claim is that within a wide range a reduced nutrient intake triggers an auto-regulatory mechanism which permits the individual to adjust - to adapt -to the

reduction in a costless manner. The claim is, then, that observed variations in nutrition intakes among otherwise similar persons are not to be explained by interpersonal differences in requirements, but rather by intrapersonal variations occasioned by the autoregulatory mechanism. It follows from this that existing nutrient benchmarks, or norms, such as population average requirements, as used for example by Dandekar and Rath [1971] and Reutlinger and Selowsky [1976], and person-specific requirements, as in Reutlinger and Alderman [1980], overstate greatly the extent of undernourishment and must therefore be reduced so as to encompass the fact of autoregulation.[1] This claim, and the question of its incorporation in the measurement of the extent of undernourishment has caused such a furious debate in the development literature that the disentangling of rational arguments from polemics and submerged value judgements is a difficult task. Nevertheless, our concern in this article is to try and assess the claim.

The measurement of poverty and undernourishment is not the only issue at stake. If a regulatory mechanism exists, it has important implications for the positive economic theory of labour markets and involuntary unemployment in resource-poor economies. They have to date been largely unexplored. (For a preliminary analysis of these issues see Dasgupta and Ray, 1986, 1987.) A second task of this article is to study some of these implications.

One might well ask, given that there has been so much debate over these matter, what all the fuss is about. We all know that quantitative estimates are fuzzy, if for no other reason than the serious limitations imposed by the

---

1 Reutlinger and Alderman [1980], using the 1978 FAO estimates of average energy requirements in various regions and assuming a normal distribution of requirements with a coefficient of variation of 15%, calculated that in the mid-1970s the total number of undernourished people in the world was about 800 million. For a comparison of the methods used by FAO [1978], Reutlinger and Selowsky [1976] and Reutlinger and Alderman [1980] in estimating the extent of global undernourishment see Beaton [1981]. See also Beaton [1983].

raw data. Why bother with an exact choice of minimum nutrition requirements? Precise quantitative estimates are unnecessary: what matters are qualitative changes, such as those occurring over time.

There are several reasons why quantitative estimates matter. First, one must note that a limited data set is no excuse for failing to construct a comprehensive system of measurement. Second, the choice of nutrition requirements (or more generally, a poverty line) is not only theoretically capable of altering dynamic trends, it has done so in practice,. Finally - and this is a most serious consideration - even if different nutrition requirements, or poverty lines, agree over time, the question of which line to use is of crucial importance. Government planners, policymakers, business groups, social activists and the man in the street use these figures to discuss the economy, to press their own cases, and to allocate funds. To argue, therefore that quantitative estimates do not matter is to misrepresent reality at a most basic level. The controversy that we shall assess has had a tremendous impact on individuals and institutions. It is no mere academic debate.

The organization of this article is as follows. In Section 2 we will discuss the statistical and measurement issues associated with autoregulation. Our central concern will be with the arguments advanced by Dr P V Sukhatme, a major proponent of the autoregulation hypothesis. Our conclusion will be that there appears to be no statistical evidence for long term adaptation to a reduced nutrient intake, although there is evidence of the existence of short term adjustment mechanisms. Accordingly in Section 3 we will turn to an examination of the clinical evidence concerning autoregulation. Both adjustment and adaptation mechanisms will be discussed there. We will show that the evidence does point to certain areas of the human system where adaptation is a distinct possibility. It also points to other areas where the

system may move in the opposite direction. Furthermore, there is also evidence that if adaptation exists, it is purchased at a cost. The cost involves, among other things, a reduction in the capacity for sustained physical and mental activities, and a greater susceptibility to infection and disease.

These conclusions imply that if one wishes to adopt the approach of using a cutoff line (followed by a headcount) to measure undernourishment, then there is little merit in the suggestion that the nutrition norm be reduced substantially from population-average requirements. For the suggestion either involves a refusal to call an individual undernourished unless the evidence for doing so is overwhelmingly strong in a probabilistic sense, or exhibits a high tolerance for the risks and dangers of adaptation.

Of course, substantial interpersonal variations in requirements and some knowledge of the joint distribution of intake and requirement may require the use of modified headcount measures that are different from those implied by population-average cutoffs (see Section 2.1, equation (2)). And there are ways of incorporating (potentially costly) autoregulatory behaviour into measures of undernourishment. We will discuss these matters in Section 2.5.

Section 3 also raises the issue of short-term adjustment. The tentative conclusion we will reach is that most adjustment takes place through the action of the storage mechanism, depositing or running down energy in body stores in the form of fat or protein. In particular, short term changes in the efficiency of energy metabolism do not appear to be significant. (See also WHO, 1985, pp 13, 50.)The implications of this finding are discussed, most especially in the context of its effect on long term requirements.

In Section 4 we will look at the implications of regulation for the positive economic theory of labour markets. We note that while the existence of regulation does not compel us to change our normative notions of poverty measurement, it can nevertheless have a tremendous impact on the way in which

labour markets function in resource-poor economies. Both adjustment and adaptation are introduced into the existing nutrition-based theories of labour markets and involuntary unemployment in developing countries. These modifications appear to yield insights not obtainable from the current theory.

2. REGULATION: STATISTICAL AND MEASUREMENT ISSUES

2.1 The Debate

The measurement of undernourishment and poverty has recently been the subject of controversy.[2] Our objective in this section is to provide a survey of the statistical aspects of regulation, and to examine their implications for the measurement of undernourishment. To clear the route, we avoid one aspect of the debate, for the reason that it is irrelevant for our purposes: that concerning the distinction between undernourishment and poverty. Calorie-based poverty lines are common in India, as they are elsewhere. Whenever the actual energy intake of an individual unit (or group) is being compared to the calorie requirements of the poverty line, and head-counts below the line are being computed in this fashion, we shall say that we are dealing with the measurement of undernourishment. When the income (or expenditure) of the unit is being so compared, we shall call it the measurement of poverty.[3]

---

2 Much of this debate may be found in Sukhatme [1978,1981a,b,1982a], Dandekar [1981,1982], Dasgupta [1984], Zurbrigg [1983], Mehta [1982], Krishnaji [1981], Rao [1981a,b], Gopalan [1983a], Chakravorti and Panda [1981], Paranjpe [1981], Seckler [1982], Payne and Cutler [1984], Chafkin [1985], and Vaidyanathan [1985]. The Bulletin of the Nutrition Foundation of India has published articles relating to the controversy. Important examples are Gopalan [1982, 1983b] and Rand and Scrimshaw [1984]. Other journals which have published much in this field are clinical journals such as the American Journal of Clinical Nutrition, and the Ecology of Food and Nutrition. Quite a few of our more important references are articles published in these journals.

3 A failure to distinguish the two has led to added debate. See, for example, Rao [1977,1981a,b] and Dandekar [1981].

We will begin by covering well-known ground, with a brief discussion of the calorie-based methods that have been used for the construction of poverty lines. In the main we will restrict ourselves to the head count measure, as the debate has largely centered on head counts. There is, however, a link (which we will discuss briefly below) between our discussions and the considerations that have led to newer measures of poverty.[4]

The primitives for a nutrition-based poverty line must always be nutrients, though the requirements may occasionally be stated not in terms of calories, protein, and so forth, but in terms of foods of various kinds.[5] We will restrict ourselves to nutrient specifications. In fact we simplify further and concentrate on calorie requirements.[6] Now, the neglect of protein requirements is not a serious offence for the Indian case, and indeed for many developing countries.[7] More serious is the neglect of other nutrient requirements, in particular those of vitamin A, iron and the B-group vitamins.[8] Their automatic supply is not ensured even if energy needs are met. Our only

---

4 See, for example, Watts [1968], Sen [1977] and Foster, Greer and Thorbecke [1984]. For a survey of these new measures and their axiomatic basis, see Foster [1984].

5 In India (our main setting), the Indian Council for Medical Research (ICMR) provides one such example. They publish balanced-diet lists in addition to nutrient specifications.

6 Later, however, in our analysis of the clinical evidence, we shall consider protein requirements and the possibilities of regulation there.

7 The writings of Sukhatme in this context have been justly influential. See Sukhatme [1970,1972,1974]. For a more general discussion of the protein problem, see the references in Gopalan [1983b]). The reason protein deficiencies can be overlooked for countries such as India is not because proteins are unimportant, but because the nature of the diets in these countries is such that protein requirements are almost always met when calorie needs are met. This statement needs to be qualified for diets which draw heavily on tubers, such as cassava and yam, where the fulfillment of calorie requirements says little about protein. This is why kwashiorkor is important in some African countries whereas the main childhood manifestation of undernourishment in India is marasmus.

8 See Gopalan [1983b] for a useful of discussion of these points.

excuse for isolating calorie requirements and ignoring these other requirements is that the main debate on regulation and adaptation has focussed on energy requirements.

The Food and Agricultural Organisation (FAO), in a series of publications, has systematically revised calorie requirements downwards (see FAO, 1957,1963,1973,1978). Their 1973 estimate for the reference man stood at 2,600 Kcal for maintenance and 400 Kcal for 'moderate' activity, yielding a total of 3,000 Kcal per day. The FAO reference man was an adult male aged 20-39, weighing 65 kg., and living in a mean ambient temperature of 10°C. It is due largely to these specifications of the reference man that the FAO's more recent estimates of requirements in tropical regions have been much lower. (Their 1978 estimate for Asia was 2,210 Kcal per day. But see the joint report from WHO, FAO and UNU,- WHO, 1985 - for a most recent set of estimates.)

The Indian estimates have varied considerably, but the more recent ones are not too different from the FAO figure. The Indian reference man (if one can at all point to one in a situation where nature and nurture intertwine closely to determine average weights and sizes[9]) weighs less than the FAO [1973] reference man and lives in an ambient climate with a mean temperature of around 25°C. Both these factors lower the energy requirement, the former by reducing energy needs to maintain body frame, and the latter by reducing the basal metabolic rate.[10] These observations should be qualified by the remark that unskilled labourers in India perform tasks that are at the very least on the hard side of 'moderate', indeed often those that are extremely strenuous.

---

9 See, for example, Gopalan and Narasinga Rao [1974] and the discussion in Bliss and Stern [1978b].

10 We define these terms and discuss the issues involved in more detail in Section 3.

These adjustments are the immediate ones that come to mind, and there are a great many more that are worth considering.[11] For this reason the variations in estimates of calorie requirement in India have been large. The Indian Council for Medical Research (ICMR), for example, recommends 2,800 Kcal per day (Gopalan and Narasinga Rao, 1974), but even their recommendations have varied, (see Rao, 1981a for a description of how the ICMR criteria based on food baskets have themselves changed over a decade). Dandekar and Rath [1971] in their pioneering study on poverty used 2,250 Kcal per day as the per capita requirement for India, though this corresponds to a higher requirement when converted into a figure for the reference man, (see below, Section 2.2). Bardhan's work on poverty in India (Bardhan, 1973) described a food basket whose calorie value is estimated (in Bliss and Stern, 1978b) to be 2,386 Kcal. The Planning Commission employs separate criteria for rural and urban sectors, placing these at 2,300 Kcal and 2,100 Kcal respectively. And there are many others,[12] but a complete enumeration of these figures is not central to our purpose here. What needs to be emphasized is that despite wide variations, these estimates share the common assumption that individuals have fixed requirements (barring interpersonal differences due to different genotypes). To understand the furore following Sukhatme's statement of his position, this point must be appreciated. It is not so much the lower requirements espoused by Sukhatme, rather, it is the idea of adaptation (to a history of low intakes) that goes with it, which has been the source of so much controversy.[13]

---

11 Bliss and Stern [1978b] provide an example of the possible margins of disagreement that might occur, by constructing suitable modifications of the early FAO figures.

12 See, for example Dasgupta [1984] and a survey of measures in Rao and Vivekanand [1982].

13 As we have already observed, different estimates for requirements have coexisted relatively peacefully.

To establish Sukhatme's position clearly, we will quote extensively from his own writings. To many, he is the originator of the adaptation hypothesis, and certainly it is he who has been most active in expounding the implications of the hypothesis for the measurement of poverty. We will therefore study his argument in detail.[14] But in order to do that it is as well to introduce some notation and restate what we referred to as the "common hypothesis" in the Introduction.

Let x denote a persons' actual calorie intake and y his requirement. (We are thus assuming for the moment that people have fixed requirements.) Of course, requirement is not directly measurable, and so needs to be estimated from desirable energy expenditure. By the qualification 'desirable' we mean the energy expenditure of a person whose body weight and physical activity have been chosen at desirable levels and, in the case of a child, whose growth rate has been targeted. For such a person, energy balance would imply an equality of intake with expenditure. Thus for such a person requirement, y, equals his desirable intake. And this can differ from his actual intake, x.Let F(x,y) be the (probability) density of (x,y) pairs in the population under study. Now consider the level of calorie requirement of the 'reference man', as described in any one of the studies mentioned above. (Sukhatme focuses on Dandekar and Rath, 1971). Call this β. This reference man is typically interpreted to be the average man. (See, e.g. Who, 1985, p. 15.) Thus

---

14 Sukhatme's position is developed in a number of papers and lectures, especially in the course of the Economic and Political Weekly discussions referred to earlier (above, Footnote 1, p.6).

$$\beta = \int_{x} \int_{y} y\, F(x,y)\, dx\, dy \quad [15]$$

As there must be genotypic variations within the population the standard deviation of y is positive. Let σ_m denote the standard deviation of requirements. Since individuals are assumed to have fixed requirements σ_m reflects interpersonal variations exclusively.

A crude index, I_1, of the head count of the undernourished, (see Dandekar and Rath, 1971; Reutlinger and Selowsky, 1976) is

$$I_1 \equiv \int_{x<\beta} \int_{y} F(x,y)\, dx\, dy. \qquad (1)$$

This yields the percentage of the population with an intake less than β. The deficiencies of this index are well known.

For this model of nutrition the correct head-count index is I_2, where

$$I_2 \equiv \int_{x} \int_{y>x} F(x,y)\, dx\, dy \qquad (2)$$

This yields the proportion of the population whose intakes are less than their requirements.[16] In practice, of course, we would have a sample of n observations on intakes, $x_1, \ldots x_n$. To apply a measure of the form (2), we would require some knowledge of the conditional distributions $F(y|x_i)$ for each i. The appropriate (sample) head-count index corresponding to (2) is then given by

---

[15] By his reference to Arthur Bowley's work, where apparently half of England was dubbed undernourished because they were consuming below the average (see Sukhatme, 1978), Sukhatme appears to imply that β is the average intake for the population. This implication is clearly unwarranted, as the calorie figures used by Dandekar-Rath and others are explicitly recognised to be (average) calorie requirements, not average calorie consumption.

[16] In (1) and (2), we are assuming a continuous probability density only for the purposes of exposition. Clearly more general distributions can be accommodated.

$$\hat{r}_2 \equiv \sum_{i=1}^{n} \left[\int_{y>x_i} F(y|x_i)\, dy \right] \qquad (2')$$

See, for example, Reutlinger and Alderman [1980].

2.2 The Sukhatme Thesis

It is this formulation of the nutrition problem which has been challenged by Sukhatme. The challenge has its starting point in the experimental observation (see Edholm et al, 1970; Sukhatme and Margen, 1978, and the references in Section 3 below) that

> **"Intake does not balance expenditure, even when averaged over a week. This is tantamount to undermining the whole basis of investigating the energy balance by simultaneous measurement of energy intake and expenditure."** (Sukhatme, 1978).

Sukhatme rejects the view that such large fluctuations can be the result of measurement error, and argues, that this intertemporal **"intra-individual variance is the dominant part of the total variation"**, dwarfing inter-individual variation of the kind that we have allowed for above. This leads to the second stage of Sukhatme's argument, which deals with the presence of substantial autocorrelation in intra-individual variation. This observation is first made for the Edholm et al [1970] data set on energy balances, which, even though it is **"limited to three weeks with breaks after each week, [is] consistent with AR [autoregressive] series of order one with serial correlation of the first order"** (Sukhatme, 1978). The observation is then made for the nitrogen (N) balance study reported by Sukhatme and Margen [1978].[17]

> "Statistical analysis of the series shows that N balance on successive days is correlated all along the series ... In particular, the autocorrelation of the first order is found to have a fairly high value". (Sukhatme 1978).

---

17 One must be careful here, as we shall point out in Section 3. Nitrogen balance studies deal with protein intakes and expenditures. Extensions of the results obtained here to energy balance may simply not be valid.

Sukhatme then turns to the implications of autocorrelation. According to him, the major implication of serial correlation is the existence of a regulatory mechanism controlling the efficiency of energy-use within the body. Rather than measurement error,

> "the more possible explanation in our view would appear to lie in the stochastic stationary nature of the physiological mechanism generating energy balance in a man maintaining body weight ... the conclusion [from the experiments] is that a man is in balance in a probabilistic (homeostatic) sense in that his balance on any day is regulated by the balance on the preceding day and it varies between fixed limits independent of [the time period]." (Sukhatme, 1978.)

A similar implication is also drawn in Sukhatme and Margen [1978], where it is stated that serial correlation in the N balance "implies that the daily N balance, like energy balance, is regulated" (emphasis ours).[18] Furthermore, Sukhatme [1982a] argues that

> "the only inference [he] can draw is that energy intake is used with variable efficiency by means of some homeostatic mechanism working for the good of the whole body and controlling body weight in the process ... the body has reversible mechanisms to bring about for itself a change it needs over time for maintaining health and activity by slowing down or speeding up rates of metabolism to preserve homéostasis".

To sum up, autoregulation, in Sukhatme's view, appears to be the "**modification of requirements without detriment**" (Rand and Scrimshaw, 1984), and it is in his view a logical implication of serial correlation in energy balance.

The clinical basis for the existence of regulation will be examined in a later section. Assuming for now that regulation of this sort is indeed implied, what are the implications for measurement? Again, it is as well to have Sukhatme's own words on the subject:

---

18 It is interesting that Sukhatme-Margen [1978] draws an analogy with the 'regulation' of energy balance, while the Sukhatme [1978] paper, postulating energy balance regulation, leans on the Sukhatme-Margen work for empirical support!

> **"When the observed intake for any day or period is therefore less than the average requirement, worked out from the FAO/WHO scale, it cannot be taken to imply that a man is undernourished, as Dandekar and Rath do, unless his intake is so low as to be below the lower limit of the confidence interval for the chosen level of significance ... it follows that in any observed intake distribution on nutrition unit basis with a nutrition unit having the same daily requirement as the reference adult, namely β, the proportion of the population below $\beta - 2\sigma_w$ will determine the incidence of** undernourishment and poverty." (Sukhatme, 1978).

In the foregoing quotation, σ_w is the standard deviation of intrapersonal variation for the reference man. Sukhatme assumes a normal distribution of intakes and, as mentioned earlier, regards interpersonal variations (σ_m earlier) to be negligible. If it is in fact not negligible and if inter- and intrapersonal deviations are uncorrelated then the overall standard deviation, σ, would satisfy the relation,

$$\sigma^2 = \sigma_w^2 + \sigma_m^2. \qquad (3)$$

We have now at hand the key ingredients of the debate. The common hypothesis, mentioned in the Introduction, is that σ_m is far in excess of σ_w. The "adaptationist" thesis is that σ_m is negligible when compared to σ_w. Thus, in choosing $\beta-2\sigma_w$ as the benchmark - the cut-off level of intake in the measurement of undernourishment - Sukhatme is choosing, roughly speaking, the bottom of the range of intakes which in his view, any individual can safely adapt to.

Sukhatme's application of the undernourishment line so constructed leads to dramatic results.

First, he considers the requirement of 2,750 Kcal per consumer unit, which was adopted by the National Sample Survey (NSS) of the Government of India (NSS, 26th round) at the instance of FAO.[19] This estimate corresponds closely to that of Dandekar and Rath [1971], whose figure of 2,250 Kcal is stated per capita, and not for the reference man or consumer unit (see, e.g. Dandekar, 1981). To transform this into a requirement per consumer unit (in the Indian case), we divide by 0.8 (see footnote 19) to obtain approximately 2,800 Kcal per consumer unit.

Second, Sukhatme suggests a coefficient of variation of 15% to capture the sum of intra- and inter-individual variation in consumer units. That is, σ is 15% of β, in our terminology. As the relevant data are given at the household level, and as there are about 4 consumer units to a household (footnote 19), the 'mean minus two sigma' rule entails subtracting $2(\sigma/\sqrt{4})$ from β. Sukhatme does so, and arrives at his cut-off line of 2,300 Kcal per consumer unit.[20]

---

19 A consumer unit, which corresponds close to our notion of the reference man, is used to transform household data into 'adult equivalent' terms in the National Sample Survey. To illustrate, the National Sample Survey (NSS) (26th round, 1971-2), used the following equivalences between consumer units and persons in the households surveyed: 4.29 consumer units for 5.39 persons per rural household, 3.81 consumer units for 4.72 persons per urban household, which worked out to an all-India average of 4.19 consumer units for 5.26 persons per household. Therefore, if one wishes to translate a per capita requirement into a per-consumer-unit requirement, the former must be divided by the ratio 4.19/5.26, or approximately 0.8. In the text, we do precisely this for the Dandekar-Rath requirement of 2,250 Kcal per capita, to avoid confusion. It corresponds to a higher figure for the consumer unit.

20 Sukhatme's arithmetic is incorrect. The implied coefficient of variation needed to obtain 2,300 Kcal is 16.4%. We ignore this relatively minor slip, even though poverty calculations are known to be extremely sensitive to the line. Sukhatme's statement that the coefficient of variation is 15% is also open to a charge of inconsistency. Dandekar [1981] reports four different figures stated by Sukhatme in different papers, ranging from 300 to 400 Kcal, (as estimates of intra-individual variation).

Applying this figure, urban Maharashtra's undernourishment head count takes a plunge from 63% to 33%, Punjab's headcount 'falls' from 20% to 10%, and the all-India head count now stands at the refreshingly 'low' figure of 25% (urban) and 40% (rural).

It is important to evaluate the argument. In the remainder of this section we will be concerned with the validity of Sukhatme's statistical reasoning. We will also discuss the implications that regulations, (or adaptation), in Sukhatme's sense have for the measurement of undernourishment. An examination of the clinical basis for Sukhatme's argument is deferred to Section 3.

2.3 A Simplified Model of Energy Requirement

We begin by considering a simplified model of energy balance, which we will extend later (Sections 3 and 4). Divide time into discrete periods $t = 0,1,2 \ldots$, and consider an individual with initial requirement β_0. This requirement is conditional on a prespecified level of activity.[21] Let us suppose that a constant amount q of energy is to be devoted to this activity, and that an amount r_t (at time t) is to be expended for basic metabolism and the maintenance of the frame of the body. We shall refer to r_t as the resting metabolic rate (RMR).[22] Let s_t be the energy released from (or stored in) the body, say, in the form of fat. Finally, let x_t denote the intake of the individual at time t. Ignoring waste for simplicity, we have:

$$x_t = r_t + q + s_t, \quad t \geq 0. \tag{4}$$

---

21 We take it that this level of activity is determined by the demands of the individual's environment. After all, the question is one of living on lower intakes while "maintaining body weight and [engaging] in similar activity from day to day" (Sukhatme, 1981a).

22 In Section 3, we take up the concept of RMR in detail when discussing the clinical evidence concerning adaptation.

Let S be an exogenously given constant that stipulates the outer limit of 'borrowing' from the body; i.e. we postulate that for continuing health of the individual, it must be the case that

$$\sum_{t=0}^{T} s_t \geq -S, \qquad T \geq 0 \tag{5}$$

If this does not hold for some T, we say that the individual is malnourished.

We reiterate at this stage that the structure we are using is simplified for the purpose of exposition. The threshold S, for instance, has been chosen to be 'history-independent'. Moreover there is in reality no sharp threshold; what we have is a 'probability of breakdown' which is an increasing function of S, the extent of 'borrowing' from the body. However, these extensions, while complicating the analysis, adds little to our understanding of the basic issues at this stage.[23] Furthermore, we have not introduced a term to capture explicitly the efficiency of energy metabolism, though we take this up in Section 3. Finally, we have assumed that energy is stored as efficiently as it is run down; (this assumption is implicitly embedded in the summation of s_t in (5)). Again, in Section 3 we shall consider the implications of relaxing this assumption.

The individual lives in an environment which gives (or denies) him access to food. The relevant scenario is that of an economic environment where the individual earns an income (perhaps in every period), which we suppose for

---

23 Sukhatme, too, makes a similar assumption as is evident from the excerpts above. Of course, he too is aware of the simplifications involved. He mentions that by the use of the term "**threshold**, [he does] **not imply that there is any sharp discontinuity in the distribution. All that is meant is that the risk of undernutrition remains about the same over a wide, though limited, range of intakes.**" We are therefore meeting Sukhatme on his own analytical ground.

simplicity to be all spent on food. We represent the income opportunities measured in 'energy units' as an exogenously given stochastic process $\langle z_t \rangle$. One could think of a number of examples in which the process $\langle z_t \rangle$ takes different forms. Under long-term employment, for instance, z_t is a fixed number (perhaps with a time trend). On the other hand, an individual who is a casual labourer might be unemployed each day (or week) with some probability. The corresponding random process of labour incomes is then represented by $\langle z_t \rangle$.

Given intakes and incomes, we define the sequence $\langle K_t \rangle$ by the condition

$$K_T = \sum_{t=0}^{T-1} (z_t - x_t) + K_0, \quad T \geq 1 \tag{6}$$

where $K_0 \geq 0$ is exogenously given. We impose the feasibility condition that

$$K_T \geq 0, \quad T \geq 0 \tag{7}$$

Equation (7) states that the current food budget plus past savings cannot be overstepped at any date. Finally, to close the system, we specify how r_t, the RMR, might vary over time. At the moment, we keep things general by noting that r_t is a function (possibly a degenerate one) of the history of past intakes (activity levels are constant). Writing $h_t = (x_0, \ldots, x_{t-1})$, for $t>0$ we have,

$$\left.\begin{aligned} r_t &= r_t(h_t), \quad t \geq 1 \\ r_0 &\text{ given.}^{24} \end{aligned}\right\} \tag{8}$$

Now, a few definitions within the context of this simple model. The body will be said to be capable of adjustment if $S>0$. The body is capable of adaptation if $r_t(.)$ is a nondegenerate function. Positive adaptation will be said to exist if $r_t(h_t)$ is increasing in the components of h_t. And finally, we

---

24 The functions $\langle r_t(h_t) \rangle$ can be taken to be random without adding anything to the analysis.

define regulation to be the entire complex of adjustment and positive adaptation.[25]

Some comments on these definitions will be useful. Intuitively, it would seem reasonable to say that a body can adjust if it can vary its intake around the "going" requirement (for period 0 in this model it is $\beta_0 = q + r_0$), at least for a few periods. For the purpose of our analysis the downside variation is clearly important, and in our model this is possible if and only if S>0.

Adaptation is a different matter altogether. This requires a change in the requirement itself as a function of past intakes. It is naturally captured in the nondegeneracy of the $r_t(.)$ functions.[26] Adaptation is then positive if a history of low intake is "absorbed" by the body by a lowering of its requirements. This is precisely how we have defined it above.

Nevertheless, we should reitérate that both adaptation and adjustment go deeper than what is suggested by these definitions. In Section 3 we will discuss possible sources of regulation in much greater detail. Speaking broadly, all regulatory features that permit the body to cushion itself against short-term fluctuations in intake will be labelled adjustment. Similarly, all regulatory features that allow the body to accommodate itself to a permanently lower intake will be termed adaptation.

---

25 A second look at the excerpts from Sukhatme's writings (above) will reveal that all the components that we have subsumed under the term 'regulation' have been described by him.

26 This is not to suggest unbounded adaptation. None of the protagonists in the debate have suggested this, and needless to say, there is no clinical support for it.

2.4 Analysis of the Sukhatme Thesis

We now consider Sukhatme's argument, step by step.

(A) Intakes vary while expenditures are relatively stable: While Sukhatme's use of the term "expenditure" is unfortunate and has even lured some into thinking that he might be unaware of the First Law of Thermodynamics,[27] his intention is quite clear from the context. He is referring to expenditure on physical activity, and is suggesting that variations are absorbed by RMR, the 'storage term' s, and changes in the efficiency of energy metabolism.[28]

There can be no doubt from the evidence to be discussed later, that Sukhatme's suggestion is valid. And it appears that most of the protagonists in the debate accept this.[29] Moreover, this evidence implies a capacity for adjustment, in the sense that we have defined it. But adjustment to what extent? Sukhatme reports that this "intra-individual variation" does not vanish when averaged over a week or two, a statement largely based on Edholm et al., [1970]. But does this variation persist without substantial loss of body weight when intakes are averaged over a month or more? The reason this question would appear to be important is that the National Sample Survey data on expenditures and food consumption are monthly averages, and this is the data base for the Dandekar-Rath study, and many other studies, including Sukhatme's own calculations. In terms of our model, this can be illustrated simply. Suppose that there is no adaptation, so that $r_t = r$ for all t, and define $\beta = q + r$. Then on using equations (4) and (5) a little algebra reveals that for all $T \geq 0$.

---

27 See, for example, Mehta [1982].

28 This last term is not considered explicitly here but we will do so below in Section 3.

29 For instance, Dandekar [1981] states that he has "**no difficulty admitting such variation**".

$$[(\sum_{t=0}^{T} x_t)/T] - \beta \geq -S/(T+1) \qquad (9)$$

This places a bound on how far the time-averaged intake can fall below the requirement, as a function of the time-period of averaging: the longer the period over which the averaging is done, the smaller is the permitted deviation between average intakes and requirements. Whether intra-individual variation persists for a month or more would therefore depend on the size of S, and on the quantitative significance of short term changes in the efficiency of energy metabolism. We will argue below (Section 3) that there is little or no evidence that intra-individual variation of this order exists.

(B) Autocorrelation implies regulation: As Scrimshaw and Young [1984] observe, "Sukhatme never defines precisely what he means by regulation other than to state that it is a consequence of autocorrelation". Nevertheless, there are enough indicators in Sukhatme's writings to suggest that he has in mind both the phenomena of adjustment and adaptation in the sense we are using the term here. For instance, he draws from the existence of autocorrelation in intra-individual variation the following moral:

> "The test of health is regulation of energy balance and maintenance of body weight and level of desired activity and not the level of intake only, as long as the latter is above the threshold value" (Sukhatme, 1981a).

Here, the concept of "threshold value" is made operational by Sukhatme in the statistical manner that we have described above, (that is, mean minus two standard deviations). This prescription clearly corresponds to adjustment, as defined here. Moreover, the existence of autocorrelation allows Sukhatme to conclude that

> **"fortunately for most of us, unless the intake is too low, the basal metabolic rate is found to decrease as the intake decreases, and in consequence the efficiency of energy utilisation is improved"** (Sukhatme, 1978).

This corresponds to adaptation.[30] To be sure, Sukhatme refers also to the biological evidence (to be considered later). But the presence of autocorrelation is, to him, a crucial indicator of regulation.[31]

The presence of autocorrelation is an important signal. It is important because it tells us that the (large) observed fluctuations in energy balance (intake minus dissipated output) are unlikely to be due to noise or measurement error. This must be qualified by the possibility that autocorrelation may simply be the result of long term physiological trends within the body (see Section 3). However, we find it impossible to understand how the presence of serial correlation in intake (with relatively stable expenditure on physical activity) implies anything over and above the easily admitted phenomenon of (short-run) adjustment, which is already a consequence of point (A). To see why autocorrelation implies nothing more, consider for instance, the stylized example of a farmer. A farmer's physical activity is fairly stable through the year, certainly more so than his output of food, which is realised only at certain points in the year and that too with some randomness. Given the limitations of the informal credit market and inadequate storage facilities, his 'income' at each date will be a stochastic process (our sequence $\langle z_t \rangle$) with cyclical properties. This process will also display substantial autocorrelation, for 'incomes' will be low for some periods before harvest, and high for some periods after harvest. Given the feasibility

---

30 However, there is a difference between basal and resting metabolism. See Section 3 for details.

31 See, for example, Sukhatme [1978,1981a,1982] and Sukhatme and Margen [1978,1982].

requirement (7) on intakes, the stochastic process $\langle x_t \rangle$ and the calorie balance process so generated will also have autocorrelation. But this is an autocorrelation imposed by the vagaries and inequities of the farmer's environment, providing him with an unstable income and demanding a steady expenditure of energy. Of course, the fluctuation in intakes implies some degree of forced physiological adjustment. But the autocorrelation? It implies nothing further about the biology of nutrition.[32]

To use the phenomenon of autocorrelation to suggest that regulation (especially in its costless adaptive form) exists is not only misleading; it has serious implications for the measurement of undernourishment that may well be wrong.

(C) The "mean minus two standard deviations" rule for poverty measurement: As we have already observed, Sukhatme proceeds to suggest that the FAO/WHO estimates should be reduced by two standard deviations (encompassing both intra- and inter-individual variation) and that this should be used as the cutoff line. In fact, Sukhatme argues that one can place "physiological meaning" on this new threshold, which has been established using the "chosen level" of significance.

---

32 The example above is set in a somewhat extended time scale. To argue the same point within a shorter time scale, consider the example where an individual receives a steady income. Suppose that he eats well, though with restraint, on weekdays, but indulges himself on weekends. Moreover, he is a hard worker during the week and rests on weekends. The derived sequence of calorie balances will, no doubt, display excellent autocorrelation! But what does this tell us about adjustment (over and above the considerations suggested by (A)), or about adaptation?

One can certainly extend or modify this straightforward rule in a number of ways,[33] but a crucial question needs to be faced. What is this 'chosen level' of significance and how is it established? The level $\beta-2\sigma$ is a statistical cutoff. Sukhatme puts it forward as a physiological threshold. Now, a necessary condition for such a proposition to be valid is that costless regulation exists and intrapersonal variation (σ_w) is greatly in excess of interpersonal variation (σ_m). But no evidence is produced to show that σ_m is indeed negligible relative to σ_w (Points A and B above). This necessary condition continues to remain a hypothesis, which we explore further in Section 3.

In any event, the condition is necessary and by no means sufficient. For these is no explanation advanced as to why the extent of admissible regulation coincides so neatly with two standard deviations below the mean! Sukhatme is taking not only a qualitative position, he is also taking a quantitative one.

Now consider the social costs involved if the hypothesis happens to be wrong. Suppose, for example, that it is σ_w which is negligible as compared to σ_m. Then the "mean minus two standard deviations" rule (over intakes) would carry with it the implicit null hypothesis that an individual is not undernourished. In this case the rule would demand Sukhatme's "type one error" - classifying a person as undernourished when he is not - to be made with a probability of only 2.275%. As Dandekar puts it,

---

[33] Krishnaji [1981], for example modifies the Sukhatme rule by recognising that different genotypes will have different intra-individual variations and so one cannot simply add the two variances to arrive at a measure of total variance. But there is an implicit acceptance of "the chosen level of significance". Chakravorti and Panda [1981] are well aware of the significance of the 'chosen level', but as part of their exercise they experiment with different intake-distributions, such as the beta.

> **"It means that we will not accept the existence of undernourishment unless the evidence is overwhelming. If we meet a household whose energy intake is below the average requirement, we shall suppose that its particular requirement must also be below the average or in fact below its actual intake; it eats less because it needs less. We take cognisance of its low intake as possible evidence of undernourishment only if the intake is so low that the probability of the requirement being lower still is very low."** (Dandekar, 1981).

In response to Dandekar's comment, Sukhatme's answer **"is that the level of significance should indeed, be left to be chosen by 'God' ... So far I am concerned, 'God' resides in human values, not in the theory of statistical inference"**. Comment is superfluous.[34]

To summarise:

(A): Fluctuation in intakes with a relative stability in expenditure appears to be an experimentally verified occurrence. We have no difficulty in admitting that this implies adjustment but it still remains to be seen how persistent the feature of adjustment is. This is especially relevant when working with NSS household expenditure data which are effectively monthly averages.

(B): Autocorrelation in intakes implies nothing over and above (A), barring the provision of evidence that the fluctuations described in (A) are not due to experimental error. And finally;

(C): Granted that persistent adjustment is possible, the mean-minus-two-standard-deviations formula advocated by Sukhatme carries an extremely strong bias against the existence of malnutrition. In a less-developed country where

---

34 The ideological undercurrents in the debate have been much discussed. See, for example, Gopalan [1982], Dasgupta [1985], Payne and Cutler [1984], Banerji [1981], Charkavorti and Panda [1981] and Zurbrigg [1983].

equity and poverty alleviation are presumably primary aims, this is unwarranted, and potentially very costly in terms of human welfare.[35]

2.5 A New Measure of Undernourishment

We conclude this section with a brief discussion of the potential merits of Sukhatme's proposal. To do so, we ignore adaptation for the moment and concentrate on the phenomenon of adjustment. Suppose, in fact, that adjustment is significant relative to the time period of measurement.[36] Simplify further and assume that individuals all have similar genotypes as regards nutrition; that is, everyone is the reference man. Finally, assume that we have an estimate (in the form of a distribution function) of the extent of intraindividual variation.

Now consider a sample of individuals, and the corresponding sample of intakes for the time period under measurement. We specify - explicitly - the probability with which we are permitted to commit the error of classifying a person as well-nourished when in fact he is not. (This probability will, of course, reflect our values concerning the importance that we attach to the problem of malnutrition and poverty.) A simple statistical calculation will then yield a level x^* below which we will call a person undernourished.

---

35 Sukhatme (1982a) reports that "**already [their] work has aroused widespread interest among economists, statisticians, social scientists, biochemists, nutritionists and medical doctors. It has also aroused interest in the Planning Commission, the Indian Council for Medical Research and in the Department of Science and Technology**". Sukhatme's arguments also appear to have convinced such development economists as Srinivasan (Srinivasan, 1981) and Lipton (Lipton, 1983).

36 In the context of NSS data, this would mean that adjustment is persistent on the time scale of a month or more.

What we learn from Sukhatme's argument (although he does not explicitly say so) is that the level x^* in no way reflects the calorie requirement estimated by nutritionists. That line is given by β, the requirement of the reference man, (see e.g. WHO, 1985, Chapter 2). It is important that the level x^*, which is a statistical threshold, be kept distinct from the nutritional norm given by β. Failure to do so has led to an unnecessary debate.[37] The level x^* not only reflects β, it attempts also to capture variations around that line and the value judgements of the social scientist that are embedded in the significance level of the hypothesis test. Its level can be higher or lower than that of β.[38]

These observations suggest a departure from the standard practice of headcount measurement. We motivate a new measure of undernourishment by noting first that in the foregoing discussion the observer has an implicit 'model' of undernourishment. What is this model? It is this. There are, first of all, two states: breakdown and good health. Given the observation (or a set of observations) on food intake, the observer assigns a value 1 (for breakdown) or 0 (for good health). It is the choice of the set of observations to which the observer should assign the value 1 that is at the heart of the debate. Given that this set has been chosen, the observer take the average of all the one's and zero's in the population. This is the measure of undernourishment.

---

37 A number of economists and nutritionists have accused Sukhatme of suggesting that a person living continually at the lower threshold can survive in a healthy way. And indeed his writings appear to imply such conclusions (see also Seckler, 1982, 1984b). There is nothing in the statistical findings to support this form of adaptation. Moreover, we are arguing that it is unnecessary to invest what is a statistical threshold with a different meaning, namely, physiological requirement. One can call it a poverty line if one wishes, but it does not mean that the reference man living on a steady intake equal to this threshold can live a healthy life.

38 Note, for example, the implicit value judgement in the statement of Rand and Scrimshaw [1984], "If an individual's requirement varies over time, would he not need to be assured the highest level that he required rather than the lowest?" See also Chakravorti and Panda [1981].

One can argue that it is precisely this insistence on assigning either a 'zero' or a 'one' which is responsible for a large chunk of the controversy. A more detailed statistical model, coupled with a better knowledge of, and appreciation for, the clinical literature would permit us to attach probabilities of breakdown to every observation. Suppose that this is expressed as a function $p(x)$, where x is the observed intake. One possible measure of malnutrition is then

$$M_1 = [\sum_{i=1}^{n} p(x_i)]/n, \qquad (10)$$

where x_i is the intake of person i in a sample population of size n.

Observe that this measure subsumes as a special case the one in which there are pure interpersonal variations. To see this simply interpret $p(x_i)$ as the probability that requirement exceeds the observed intake x_i, that is,

$$p(x_i) = \int_{y>x_i} F(y|x_i)dy$$

in the language of Section 2.1, and now compare (10) with (2'). The simplest headcount measure using cutoffs, such as those used by Dandekar and Rath [1971] and Sukhatme [1978] are also special cases. (For any cutoff β, set $p(x) = 1$, for $x<\beta$ and $p(x) = 0$ for $x \geq \beta$.)

Our proposed measure also incorporates the possibility of (costly) autoregulation. To see this, assume for convenience that there are no interpersonal variation. Suppose, further, that the time-period underlying the observation x_i is given. (For NSS, data x_i represents a monthly average). Then $p(x_i)$ will stand for the probability that the body will break down (due to stress, infection or disease), conditional on the observation that x_i has

been the observed intake. A satisfactory quantification of p(.) will require that available physiological data be put together in a consistent statistical way. (See Section 3.) This measure can therefore be viewed from another angle, in that it is a way of seeing the basic disagreements which have fuelled the controversy.

Finally, one may be interested in a related measure. Suppose that there is some x such that p(x) = 0. Consider the minimum such x, call it x*. A discriminating measure of undernourishment might be constructed to highlight further the risk $p(x_i)$ associated with some observation x_i. This is done by weighting the risk by the (proportionate) nutrition gap $(x^* - x_i)/x^*$. In doing so, we capture not only the extent of the risk faced by the individual but the difficulty of its alleviation. Our second measure is therefore

$$M_2 = [\sum_{i=1}^{n} p(x_i)(x_i - x^*)]/nx^* \qquad -(11)$$

There is a close formal similarity between M_2 and the new measures of poverty. If the observations are incomes and x* is interpreted as a poverty income value, then $(x^* - x_i)/x^*$ is precisely the ith persons's proportionate income gap. The function p(x) may be thought of as a weighting scheme, analogous to the way in which absolute deprivation would be captured in a poverty measurement.[39]

---

39 In measures of relative deprivation one must use a set of weights where each weight is in some way sensitive to the positions of others. Sen [1976] uses the ranks of the poor as weights but this is only one possible weighting system, and there are other, equally persuasive, systems where the weights are dependent only on the absolute shortfall. See Kakwani [1980] or Foster [1984].

3. REGULATION: THE CLINICAL EVIDENCE

The heated discussions in the pages of the Economic and Political Weekly (and elsewhere) might create the impression that Sukhatme (and Seckler) were the initiators of the regulation hypothesis.[40] This is not true. Adaptation to a reduced or infrequent supply of calorie has been the subject of study since the beginning of this century, dating back at least to the laboratory-controlled experiments of Benedict (Benedict, 1907,1915). One of the classic studies of the physiology of human adaptation to continuing calorie deficiency is that which was undertaken in 1950 by Keys and his colleagues (Keys et al, 1950). And there is a wealth of data on the effects of calorie deprivation in experimental animals (see Fabry, 1969, for a detailed discussion). The survey by Grande [1964] summarises some of this earlier literature. Other studies on calorie deprivation or on fluctuating calorie intake, such as Edmundson [1977,1979] or Edholm et al. [1970] have received a more explicit treatment in the current debate. We will examine these presently.

Our discussion of the existing clinical literature is constrained in several respects. First, we are ourselves students in this area, and we cannot pretend that we understand, in detail, the many intricacies of the biological processes underlying regulation. Secondly, there is doubt that any satisfactory answer exists in the available literature. Not only does a great deal more empirical work needs to be done to understand the implications of undernourishment for economic behaviour, there is much that is not understood at the more basic physiological level. Finally, there is an intriguing feature of

---

40 Sukhatme himself suggests that his work, along with that of Margen and Seckler, "**has caused an upheaval in the [current] philosophical and scientific concepts [that] will almost certainly imply a change in the nature and direction of future research.**" (Sukhatme, 1981a).

the clinical studies that has crucial implications for the measurement of undernourishment, though not for the positive economic theory that one might construct from this.[41] This is the use of adaptation as a term to describe the change in the equilibrium of an organism (man, in our example) relative to the external environment (calorie intake history in the present context) as this environment is exogenously altered. This corresponds roughly to our usage so far. However, the use of the term permits a variety of 'adaptive' mechanisms to come under its rubric. This can cause a problem. For, when it is the undernourishment of man we wish to study and estimate, many of these mechanisms can legitimately be regarded as socially undesirable. For example, Fabry writes, referring to the Keys et al [1950] study:

> **"The final effect of adaptation [to low calorie intake] is a reduction of the total energy output ... The greatest ratio of the 'actively' saved energy is accounted for by the reduced physical activity. Spontaneous reduction of movement, which in undernourished subjects are part of the complex of marked behavioural differences ... thus play a very substantial role in the adaptation to a reduced food intake"** (Fabry [1969, 20-22]).

'Adaptation' at the price of physical debilitation, manifested for instance in the 'spontaneous reduction of movements' carries very different ethical connotations from those the word normally suggests. This is certainly not to suggest that biologists are harsh, unfeeling creatures. But they have a well-defined clinical view of adaptation, which must not be taken out of context. This also makes our task more difficult. Which features of adaptation should be socially acceptable? Which features should be rejected? These are difficult matters, but they must not be evaded. In other words, we

---

41 See Section 4 for a discussion of the implications of adaptation for the economic theory of labour markets and unemployment in resource-poor economies.

are arguing that there are types of adaptation which a defendable measurment theory should ignore. An act of measurement (of undernourishment) is thus a normative act.

Important, for instance, is the question: 'adapted' or not, does the person feel hungry? Does he experience suffering as a consequence of his low or fluctuating intake of food? However difficult these questions may be to translate into practice, they cannot be lost sight of. Of course, the 'adaptationists' in our debate recognise these points; (not fully, though, as we shall argue). But even the concern with regulatory mechanisms which do not impair the ability of the individual to do productive work does not go far enough. For example, many Indian labourers perform back-breaking tasks in unhealthy, hostile environments.[42] There can be no question that they work hard relative to their low levels of energy intake. Even were they capable of adapting, could it be suggested that they do not go hungry? Banerji [1981] makes this point forcefully and clearly. His empirical work is perhaps the only real attempt there has been to quantify the extent of hunger in this basic sense. While registering our basic sympathy with this approach, we recognise that there are other dimensions to the problem, and it is to these we now turn.

We are interested in the existence of regulation and its positive and normative implications. To narrow down the vast area of coverage that is implied, we concentrate on the following specific issues:

(i) What is the biological evidence for adjustment to a low or fluctuating intake of nutrients, especially of calories?

(ii) What is the biological evidence for adaptation to a low or fluctuating intake, in the sense that the ability to perform tasks remains unimpaired?

---

42 Durnin and Passmore [1967] note that ricksha-pulling in Calcutta is among the most arduous tasks in the world in terms of energy requirement.

(iii) What implications do (i) and (ii) have for the measurement of undernourishment and poverty?

(iv) What implications do (i) and (ii) have for economic behaviour, in particular, for theories of the labour market in resource-poor economies?

Issues (i), (ii) and (iii) will be the subject of study of this section. The study of (iv) will be left to Section 4.

3.2 Components for the Energy Balance Equation

We begin by extending the basic structure of Section 2. Recall equation (4) (with time subscripts dropped):

$$x = r + q + s \qquad (11)$$

The components of this equation need to be studied in greater detail, and we proceed to do this.

1. **The Atwater factors and metabolisable energy:** Consider first the energy intake x, which represents the metabolisable energy available to the individual. Now, this does not equal the energy value of food ingested, v, say. From v, we must subtract losses of energy due to digestion (fecal losses). What is left may be called digestable energy. But this, too, contains further losses, due largely to incomplete oxidation of protein and other nitrogenous materials in the body. The resulting excretion of urea, creatinine and uric acid contains energy. These uriary losses are deducted from digestible energy to yield x, the metabolisable energy.

It transpires that these losses, divided by the gross energy content of food intake, appear to be quite stable across individuals, though they depend, of course, on the type of food ingested. The percentage of retained energy

varies between 90 and 95%, and these (food-specific) coefficients are known as the Atwater factors.[43] The Atwater factors are used widely to provide information on the calorie value of various types of food.

2. **The resting metabolic rate and its components:** Consider, next, the term r in (11). We have defined this to be the resting metabolic rate (RMR). It has two significant components . The first is the basal matabolic rate (BMR). This is defined as the energy expenditure of an individual who is at rest in a thermoneutral environment and who has fasted for a period of fourteen hours.[44] It is the energy required to maintain body temperature, to sustain heart and respiratory action, to supply the minimum energy requirement of resting tissues and to support ionic gradients across cell membranes. FAO (1973, p.107) suggest a BMR of roughly 1,700 Kcal per day for its reference man. The BMR has certain broad properties (see Heim, 1985). For instance, it is roughly a constant fraction of body surface area. Furthermore, expressed as a fraction of body area, it varies inversely with age and body weight. And finally, there are important links between BMR and undernourishment. We will explore this below.

The second component of RMR is the increased metabolic rate resulting from the ingestion of food. Food ingestion causes a great deal of heat

---

43 See, e.g. Atwater and Benedict [1903]. However, even in discussions on nutritionally healthy populations there are disagreements regarding the reliability of the Atwater factors, for example, when applied to foods containing large amounts of indigestible carbohydrates (dietary fibre). There are suggestions that the variability of retention may be higher, between 80 and 95% (see, e.g. Heim 1985).

44 See, e.g. Heim [1985]. By a thermoneutral environment we refer to the range of ambient temperatures within which the individual is in thermal balance, at a constant body temperature, with no net heat loss or production.

production and loss; this extra production will be called diet-induced thermogenesis (DIT).[45] The magnitude of DIT is certainly not negligible - probably around 600 Kcal per day.[46] It has been suggested that DIT mechanisms play an important role in regulation. These are controversial matters, we will nevertheless look into them briefly.

3. **Physical Activity:** The term q in (11) represents energy requirements for physical activity. We now study this more closely. Let α denote a type of activity (carrying loads, harvesting, working on a conveyor belt and so on) and λ its level (kilogram-miles per day, bushels per day, bolts tightened per hour and so forth). The activity and its level will demand a certain amount of energy. In a given experimental situation, the energy cost of each activity may be directly measured [47] or obtained from published tables.[48] As a rough indicator, FAO [1973] suggested a requirement of 400 Kcal per day for moderate activity for their reference man, but activities requiring in excess of 1,000 Kcal per day would by no means be uncommon.

The energy requirements in physical activity depend on the genotype of the individual and his history. (An example of an important indicator is the body weight of the individual.) But the manner in which these requirements are affected is a complicated issue, and we will postpone its discussion for the

---

45 The phenomenon is called by various names: specific dynamic action of food, thermic effect of food, postprandial increase in heat production, and so on. See Heim [1985].

46 FAO (1973) suggests 1.5 times the BMR as a reasonable approximation for energy required for pure maintenance. For its reference man this being 2,600 Kcal, one may infer that it regards the DIT of such a person to be 866 Kcal per day.

47 The measurement of oxygen consumed and carbon dioxide produced (and preferably also urinary nitrogen output) can provide a reliable indicator of energy expended on various activities. The Weir equation (Weir, 1949) provides one link between these variables, and can be used for the estimation.

48 See, for example, Durnin and Passmore [1967], Bannerji and Saha [1970], and WHO [1985].

moment. But we note that for a given activity α, its level λ and a given history h_t for the individual at time t, the energy requirement q_t is given by the function

$$q_t = e_t(\alpha,\lambda,h_t) \qquad t\geq 0 \tag{12}$$

4. Storage: The additions to (or running down of) body stores of energy are reflected in the final term of (11), which is s. At the risk of some simplification,[49] we postulate that given the weight, w_t, in period t and the magnitude of s_t (positive or negative), the weight in period t+1 is determined by the relation

$$s_t = s(w_{t+1}\ w_t) \tag{13}$$

It should be noted that the function s(.,.), (like our other functions) depends on the individual's genotype. In particular, the form in which energy is stored in the body may differ significantly among individuals. A large part of the storage is in the form of fat, but there are individuals who store significant amounts in the form of protein.[50] These factors will have a role to play in our later discussions.

We believe that if there are any mechanisms for adjustment or adaptation, their impact must be on one or more of the four categories described above. It may help the reader to know that most of the pleas for regulation come under items 2 and 4, (see, for example, our analysis above of the Sukhatme hypothesis). But we will consider each category in turn.

---

49 The simplification essentially arises from the fact that we are ruling out feedback effects of history on the form of the function s(.,.). This does not appear to be important, as far as we can tell from the existing literature.

50 Dugdale and Payne [1977] construct an interesting model where people are classified as metabolically fat or metabolically lean, depending on their genetic predispostion to store energy in the form of fat or protein respectively. This form of 'leaness' or 'fatness' has important implications for adjustment to fluctuating intake, (see below).

3.3 Metabolisable Energy

How reliable are the Atwater factors in giving us estimates of the metabolisable energy of various types of food? In undernourished populations, the answer appears to be: not very. In fact, the evidence is strong that undernourishment (especially coupled with episodes of acute diarrhoea [51]) leads to a significantly impaired ability to digest nutrients. The Atwater factors, which predict a nutrient digestibility of 95% plus (barring protein) may be completely off the mark in these situations.

For the populations in less-developed countries, Atwater factors generally lead to overestimates of digestibility. Uauy [1985] observes that the mucosa of the small intestines of people living in these countries display changes that are characteristic of subclinical nutrient malabsorption, and that digestibility rarely exceeds 90% as a result. In addition, undernourishment or recent episodes of acute diarrhoea, can bring down nutrient absorption to below 80%, (Molla, 1984).

But that is not all. Food intakes that have a high percentage of dietary fibre (crude fibre exceeding 10 g per day) possess digestibility coefficients that are overestimated by the Atwater factors.[52] Fibre intakes exceeding 50 g per day are not unusual in rural tropical populations or among vegetarians, (Uauy, 1975). This may decrease digestibility by a further 10%, (Uauy, 1985; Prynne and Southgate, 1979).

All this is bad news for the 'adaptationists'. But the objection may be raised that this 'negative adaptation' is not significant quantitatively. So it is as well to see what order of magnitude is involved. Suppose an

---

51 The connections between undernourishment and illnesses such as diarrhoea are discussed below.

52 This is because foods containing large amounts of indigestible carbohydrates (dietary fibre) have detrimental effects on the digestibility of other nutrients, thereby reducing the metabolisable energy content of proteins and fats, (Hein, 1985).

individual's daily requirement of metabolisable energy is 2,500 Kcal. Multiplication by an average Atwater factor of 95% leads to a 'gross' requirement of approximately 2,600 Kcal per day. But now consider a rescaling of the digestibility coefficient along the lines suggested above. This would be anywhere between 70% and 95%, the lower figure being more near the mark for undernourished people in tropical countries. Take a figure of 75% and rescale the metabolisable energy requirement. The new gross requirement is approximately 3,300 Kcal per day, which is 700 Kcal per day higher.[53] It is clear then that the numbers involved are far from insignificant. (As a measure of relative magnitude, it might help to recall the FAO [1973] prescription of 400 Kcal per day needed solely for moderate activity of the reference man.)

3.4 Resting Metabolism

It is here the issue of regulation and its normative implications acquires its greatest complexity. One is concerned not only with the existence of adaptive mechanisms, one is concerned also with their normative interpretation. Rather than linger over these issues in the abstract, we go directly to the evidence.

Our starting point is the classic Minnesota experiment of Keys and his colleagues on the biology of human starvation.[54] The analysis of Taylor and

---

53 We are not suggesstting that 75% is in fact the appropriate coefficient. Much work needs to be done on this problem before anything substantially precise can be said. Nevertheless, the orders of magnitude predicted by the preliminary evidence that we have quoted are striking, and deserve much more attention than they have received. The proponents of adaptation do not mention these issues, to the best of our knowledge.

54 The basic reference is Keys et al. [1950]. See also Taylor and Keys [1950], and the article by Young and Scrimshaw [1971] for an insightful account of the Minnesota experiment and related considerations.

Keys [1950] with data drawn from the experiment is particularly instructive. Thirty two subjects were investigated.[55] In the course of the control period they were in energy balance with an average daily intake of 3,492 Kcal. Over an experimental period of six months, the food intake was lowered to 1,570 Kcal per day.[56] The effects were dramatic:

> "The subjects suffered a marked loss of strength and endurance as the starvation period progressed. The men commented that they felt as if they were rapidly growing old. They felt weak and they tired easily. They moved cautiously, climbing stairs one step at a time, and obviously reduced unnecessary movements to a minimum." (Taylor and Keys, 1950).

Body weight fell, but stabilised at the end of the six-month period, when a new energy balance had more or less been established. The average loss of body weight was 24%. An average of 1,922 Kcal per day had therefore been 'saved' by the body. How was this saving accomplished? To see this, consider the following table:

---

55 The subjects were resident in the Laboratory of Physical Hygiene for one year, where the experiments were carried out.

56 This "starvation diet" consisted of potatoes, cabbage, turnips and cereals, with only a few grams of animal protein each week. The protein content of the diet was 54.5 g per day, and adequate allowances of minerals and vitamins were provided (except for riboflavin and vitamin A).

Table 1: Energy saved by subjects after 6 months of calorie undernourishment

Source of saving	Energy saved (Kcal per day)	% of total calories saved
Basal metabolism	614	31.9[a]
DIT	192	10.0
Energy expended on physical activity	1,116	58.1[b]
Total saved	1,922	100.0

[a] 65% (i.e., 20.7% of total calories saved) for decrease of metabolising tissue mass, and 35% (i.e. 11.2% of total calories saved) on account of lower tissue metabolism.

[b] 60% (i.e., 34.9% of total calories saved) on account of reduced physical activity, and 40% (i.e., 23.2% of total calories saved) on expenditure for work due to reduced body weight.

Source: Data from Taylor and Keys [1950], taken from Fabry [1969].

In comparison with the corresponding values in the control period, the basal metabolic rate (BMR) declined by 19%, the energy expended on work fell by 71% and the specific dynamic effect of food (DIT) fell proportionately to the reduced intake, by about 50%. In terms of calories saved, reduced physical activity was the largest contributor (58.1% of total calories saved) with BMR a not-too-close second (31.9%)

Now, the Minnesota experiment was not designed to answer the questions that we have been asking here, in particular, those concerning adaptation with unchanging physical activity levels. But it does raise the following question: what if the experiment were redone with the subjects being required to maintain a specified activity level? This experimental situation would mirror more closely the actual experience of low-intake labourers who are forced to work at specified levels in order to earn a living. After all, the reduced

'starvation' diet of 1,570 Kcal per day is close to the average energy intake of Kerala (approximately 1,600 Kcal per day), and is certainly as large as the nutrition intake of large sections of the Indian population.[57]

It may be argued that comparison with India is improper. Indians have over generations of low intake and 'adaptations' have taken place on a corresponding time scale. The Minnesota experiment, using North Americans with generations of comfortably high food intake, obviously do not mirror this situation. But one must be careful. A Lamarckian notion of intergenerational improvement, in the sense that the child acquires adaptation from the parent, is simply not tenable, though it may be granted that genotypes with lower needs have had advantages in the selection process. What is at issue in the present debate is the question of phenotypic adaptation. The underlying premise is that the genotypes of people in less-developed countries (determining body size and weight) are not be very different from their counterparts in developed economies.[58] The Minnesota experiment is, therefore, relevant to our inquiry, and the modified version, requiring unchanging activity, would have been especially so.

While it is difficult to speculate on the consequences of such a modification, it is worth noting that Taylor and Keys are themselves quite cautious in drawing implications from the experiment for 'positive' or

---

57 The Kerala figure is NSS data (see National Sample Survey, Sarvekshana, January 1979), though it may be on the low side (see Kumar, 1986). As for Indian nutrition-intake data, there is a good deal of information. See, e.g., Rao [1977] and Dasgupta [1984].

58 For example, Gopalan and Narasinga Rao [1974] write that "**it has been shown... that the growth potential of Indian children is not different from that of European or American children**". Then again, we have in WHO [1985, p.36] that; "**A number of studies have attempted to assess the possibility of ethnic differences in BMR but these have failed to identify any differences that could not be related to the nutritional state or possibly to climatic conditions.**" The increasing body sizes and weights of people in Japan and China also appear to support this assertion in a general way.

'beneficial' adaptation. They observe that much of the adaptation is obtained **"by the rather desperate expedient of reducing the mass activity of the organism. This mechanism ... is entirely passive and produces major limitations and stresses of its own."** [59] Nevertheless, the experiment provides a key insight into one possible mechanism for positive adaptation: that occurring via a lowering of the basal metabolic rate. In the Minnesota experiment this fell by 19%, and accounted for 31.9% of the total calories saved.[60] A lower BMR has been observed in other experiments dealing with reduced calorie intake.[61] One influential study (Edmundson, 1977, 1979)[62] identifies the basal metabolic rate as the fundamental adaptive mechanism under calorie deficiency. The study has received attention in the current debate[63] and merits discussion in some detail.

The subjects were 54 East Javanese farmers. Each subject was measured six times at two month intervals (for one year) for a total of 324 man days of data. Food intake was measured and its metabolisable energy content determined. Activities were recorded for each individual, and mean energy outputs were determined for ten basic activities. Work outputs for each individual were then calculated by multiplying the mean energy output per minute by the number of minutes each subject engaged in that activity.

---

59 Taylor and Keys [1950].

60 This observation needs to be qualified, however, by the fact that the greater part of the reduction was accounted for by the reduced amount of metabolising tissue (see footnote (a) of Table 1). When calculated as a fraction of lean body mass, the BMR was only 15% lower (Taylor and Keys, 1950).

61 See, e.g., Grande, Anderson and Keys [1958] and the survey by Grande [1964]. These experiments suggest that a lower BMR per unit of lean body mass participates in the improved energy balance, indicating a reduction of metabolic processes in tissues.

62 A summary of these two papers may be found in Edmundson [1980].

63 See, e.g., Sukhatme [1982a].

The results were striking. While the mean energy intake (2,430 Kcal per day) corresponded closely to mean energy output (2,443 Kcal per day), there was no association between average intake and output for individuals. The ratio of energy intake to output (averaged over the six observations for each subject) ranged from a low of 0.59 to a high of 1.63. Edmundson concluded:

> **"This implies that trained observers watched 54 villagers very closely for 324 days, with the specific purpose of carefully determining how hard these men were working, [and] could detect no discernable differences in the observed work output of men with high and low energy intakes."** (Edmundson, 1977).

Edmundson's second study, 1979, is a continuation of the first. Eleven of the earlier 54 subjects were chosen for further testing. Most of them had exhibited unusually high or unusually low intakes in the earlier study, and the rest served as controls.[64] The subjects were so selected that average heights (and weights) in the high and low intake groups were similar. Basal metabolic rates of these subjects were measured, and a six-day average of energy intakes was constructed for each individual.

The BMR of the high-energy-intake group was found to be twice as high as that of the low-energy-intake group. Subjects enjoying high energy intake also expended greater energy in performing standard tasks, though the difference here was not as significant. Edmundson concluded that there is an adaptive increase in metabolic efficiency, in response to a prolonged period of low intake.

This study is certainly closer in spirit to the sort of issues we have in mind, and so it is worth discussion in a bit more detail. Consider the first experiment. While calorie intakes were measured individually, energy

---

64 Six of these had exhibited intakes below 2,000 Kcal per day in the early study, three had exhibited intakes greater than 3,000 Kcal per day, and two had intakes of about 2,500 Kcal per day.

outputs were calculated for each subject by multiplying the number of minutes spent on a particular activity by the (group) averaged energy expenditure per minute on that activity. Now, this is a peculiar thing to do, for there is then no way to judge the work output of each individual per unit of time. Suppose you are ill-fed and hungry, and work at a lethargic pace for an hour. Your well-fed counterpart works for an hour, too, but gets a lot more done. Edmundson's technique of measuring energy outputs would give both of you the same number! It is no wonder, then, that the 'most efficient' low-intake subject in that experiment released 1.63 times the energy that he ingested.

In response to this one might invoke Edmundson's second experiment. Here, subjects with high energy intake expended more energy per minute for performing standard tasks. It might be argued, therefore, that this greater efficiency permitted low-intake subjects to work just as hard while using less energy. But this argument fails on two counts. First, of the two tasks, the lighter one did not exhibit differences in efficiency (between the two groups) that were significant at the 1% level, though the heavier one did. In any case the differences are not high enough to allow one to argue that all subjects were working equally in the first experiment. Second, the tasks measure an efficiency rate. They do not measure the stamina, or the ability, to perform at a certain rate for prolonged periods of time.[65]

The Edmundson experiment therefore retreats into the same realm as the Minnesota experiment. While it is stated that there were "no discernable differences" in physical activity, the measurement techniques used simply do not permit such an assertion to be made.

---

65 It will help to disclose at this stage what these 'standard work tasks' were. The light task involved pedalling a bicycle ergometer at a rate of 50 watts per minute, while the heavy task involved pedalling to produce a work output of 100 watts per minute. Energy intakes in this process do give an index of efficiency, and with lower resting metabolism it is not surprising that low-intake subjects are more efficient. But this exercise reveals nothing about their capacity for doing sustained work.

Now, one may ask: if our basic concern is with a possible adaptive mechanism in the BMR, why worry whether work output stays constant? After all, the BMR was undoubtedly lower in the low-intake subjects, and to the extent that this was due to a lower oxygen utilisation of tissue [66], it is indicative of some degree of adaptation, even though one needs to be careful in drawing any conclusions about the direction of causality involved.[67]

The answer is that we do not know whether a fall in the BMR as a response to undernourishment is at all separable from a decline in physical stamina and activity. Taylor and Keys [1950], for instance, warned that a great part of the decline in BMR may due to the wastage of tissue. In particular, the heart may undergo considerable loss of muscle mass during periods of prolonged low intake. Young and Scrimshaw [1971] also observe that the heart size shrinks in these situations, and the effect of such a phenomenon on physical activity, especially on work that requires stamina, is not difficult to predict. And, at a more general level, Gopalan [1983] argues that for an adult, adaptation to a low intake "may not pose serious health hazards", but only if "the inadequacy can be totally compensated by reduction in activity." [68]

It is important to remember that an exhibition of a decline in the basal metabolic rate as a response to low intake is not an indicator of acceptable adaptation. For instance, children with marasmus or kwashiorkor are

66 Recall that in the Minnesota experiment, a great deal of the fall in BMR was due to wastage of active tissue.

67 Edmundson is careful. He recognises that "**it is difficult to say whether the low-intake subjects eat less simply because their needs are less as a function of normal physiologic variability or whether their low BMR's represent a combination of short and long term adaptations to low energy intake**". (Edmundson, 1979, p. 193).

68 Gopalan goes on to state that in the case of a child, not surprisingly, a total reduction of activity in fact hampers its physical and intellectual growth and development. See the references in Gopalan [1983]. See also WHO [1985.]

known to 'adapt' by lowering significantly their basal metabolic rates (Jaya Rao and Khan, 1974).[69] This is not a normal state but a strategic response to a life-threatening environment.

How much of a change in the basal metabolic rate (following low calorie intake) can be treated as acceptable physiological adaptation, as opposed to a pathological response? This is a difficult question, and its satisfactory resolution requires careful study of a number of aspects. We have emphasised that the fall in BMR could be due to a wastage of tissue and not to a depressed oxygen utilisation of active tissue.[70] The two situations are quite different. We can thus do no more than to insist that there are basic uncertainties in interpretation which cannot be ignored. Whether the BMR adapts significantly to low intake in an acceptable way is still very much an open question. WHO, 1985, p.50 go further,

> **"The documented changes in metabolism when energy intake is altered suggest, therefore, that with the present state of knowledge the range of metabolic adptation must be considered to be small."**

We conclude with diet-induced thermogenesis (DIT) as a possible adaptive mechanism.[71] The most well known experiments that have been conducted have addressed the effects of overfeeding, and thus inducing obesity. The aim here has been to study energy balance in subjects.[72] It has been conjectured

---

69 One can argue, in fact, that the marasmic infant adapts better. For in the case of marasmus, the child discards its muscles in order to protect the more important organs (liver, pancreas and intestines), while these organs are damaged in the child with Kwashiorkor, (see Gopalan, 1983).

70 As Jaya Rao and Khan [1974] observe (but do not carry out), expressing BMR as a fraction of lean body mass may help to isolate a little better the degree of adaptation involved.

71 These observations are largely taken from Rand, Scrimshaw and Young [1985].

72 See, e.g. Sims [1976], Jung et al. [1979] and Schutz et al. [1984].

that DIT mechanisms might play a part in energy balance regulation, and that obesity might be traced to a defect in these mechanisms (when DIT does not increase following high intake).[73] But all this is still controversial.

3.5 Efficiency of Energy Metabolism

A related issue is the possibility of adaptation through changes in the efficiency of energy metabolism. Sukhatme and Margen [1982] advance this as a hypothesis. But the assertion is based on little or no cited evidence (the details we relegate to a footnote, [74]). Rand, Scrimshaw and Young [1985] test the Sukhatme-Margen hypothesis with the original data from five sets of long-term metabolic studies. There were 42 subjects in all, who were examined for periods of 63-90 days.[75] In each study, crude estimates of energy intake required to maintain body weight for each subject were arrived at. During the study, the amounts of energy estimated were supplied at an unchanged level. The subjects were asked to maintain their usual level of physical activity in order to achieve a relatively constant energy expenditure in this sphere.

---

[73] See, Jung et al. [1979], Danforth [1981], Miller [1979] and Jequier [1983]. It has been suggested that thermogenesis in brown adipose tissue might be the principal energy buffer (Himms-Hagen, 1984), but this area is controversial, as the high metabolic rate of this tissue may not be significant for the organism as a whole (Uauy, 1985).

[74] The body does not take energy directly from food. For example, glucose is converted into ATP, which is then broken down to supply the body's energy needs. In the process of ATP formation, a large fraction of energy is dissipated as heat. This fraction is at least as large as 45-50%, but in many individuals it is higher. Sukhatme and Margen (1982) argue that high-intake individuals are relatively inefficient in this conversion. For example, they state that an individual with an intake of 1,900 Kcal per day will have an efficiency of 50%, whereas in an individual consuming 3,200 Kcal per day the efficiency is 30%. For an intermediate intake of 2,550 Kcal per day the efficiency factor is 37%. These numbers are plucked from the air and do not seem to have any experimental basis. It is worth noting, too, that when these intakes are multiplied by the respective conversion factors, they all give the same figure - about 950 Kcal! This "backcalculation" also appears to suggest (unintentionally, perhaps) a belief that all adaptive changes are due to changes in energy metabolism.

[75] The five studies dealt with dietary nitrogen and energy intake for 16, 8, 6, 6, and 6 subjects for 63, 84, 82, 90 and 82 days respectively. See the references in Rand, Scrimshaw and Young [1985].

These studies attempt to minimise daily variations in energy intake and physical activity. Therefore, if there is a regulatory aspect to energy metabolism which adjusts to short-term fluctuations in body needs (or intakes) while maintaining body weight, this would presumably manifest itself in the observation that most subjects quickly settle down to a constant body weight.

This did not happen. In 19 out of 42 subjects there were significant increases in body weight, and another 14 displayed significant declines in weight. (16 of these 32 individuals had body weight increasing or decreasing throughout the length of the study.) Of the remainder, 8 subjects showed fluctuating body weight with no apparent trend, and only 2 maintained a stable body weight. It was therefore clear that for the great majority of the subjects, "the apparently modest imbalance between energy intake and expenditure was not effectively buffered by adaptive thermogenesis" [76], or by changes in the efficiency of energy metabolism.

3.6 Physical Activity

We now turn to our third category of energy use: physical activity. Recall that one of the requirements of acceptable' adaptation in the studies we have been commenting upon is that physical activity at the ongoing level should not be lowered. To capture this, fix the activity (α) and its level (λ). The question of adaptation in this sphere may now be rephrased thus: do low-intake subjects require less energy to perform the given activity at the given level, relative to their high-intake counterparts?

Now, there are a number of ways in which an individual's nutrition history might affect current work-efficiencies, but we will simplify and focus on two particular aspects of a low-intake history: small body size and low body weight. There is much evidence that both these factors are consequences

---

76 Rand, Scrimshaw and Young [1985].

of a history of low intake, the former more so in the case of childhood undernourishment.[77] Our question then simplifies to whether people of small stature and weight are more sparing in their energy use, when asked to perform the task (α,λ). This is also connected to the debate on the "small but healthy" hypothesis, a revealing title fashioned by Seckler.[78]

The evidence on this issue is limited. We have observed that Edmundson's [1979] study yielded a better efficiency rate (measured as the ratio of work output to energy consumption) for people with low intake. But there are studies that indicate no such efficiency difference. For instance, Spurr et al. [1984] studied the effect of malnourishment on treadmill walking, using a sample of school-aged Colombian boys. They concluded that there was no evidence to indicate that shorter, lighter people are mechanically more efficient than their larger counterparts. This is not surprising. There is no doubt that ceteris paribus, a heavier or bigger person uses more energy simply to 'carry' his larger dimensions along. This would tend to raise work efficiency of smaller people. On the other hand, there are many tasks where physical strength is an asset. Activities such as stone-cutting, sugarcane harvesting and carrying loads come easily to mind. There are many others. A heavier person may well get more work accomplished on the treadmill, though he may also use more energy per unit of time. But there is no reason to expect a significant difference in the ratio of work to energy, though Seckler [1984], contrary to Spurr and his colleagues, feels "the limited evidence indicates that, down to a low limit of size, small people are more efficient workers".

---

77 Familiar scales for grading protein-calorie malnutrition (PCM) are based on these observations. The well-known Gomez scale (Gomez et al., 1956) uses weight-for-age as a percentage of the Harvard standard (for normal, healthy individuals). The Waterlow scale (Waterlow, 1976) uses height-for-age (as a percentage of the Harvard standard) for grading chronic PCM. See also Downs [1964] and Vaidyanathan [1985].

78 See, e.g., Seckler [1980, 1982, 1984], Payne and Cutler [1984], and Chafkin [1985].

In any event, one may ask: why should work efficiency be the sole criterion? Comparisons of efficiency do not answer our basic question, which is couched in terms of a fixed activity and a given level of that activity. Work capacity is equally relevant. We therefore turn to a brief discussion of studies dealing with this issue.

Areskog et al. [1969] used as their subjects six groups of Ethiopian males of varying ages. Anthropometric and other laboratory data were taken. The capacity to do work was then measured on a bicycle ergometer, with the work-load being increased stepwise at 6 minute intervls.. They physical work performed at a heart rate of 170 per minute was taken as an index of work capacity.[79] A high positive correlation between body weight and work capacity was obtained.[80]

Other studies on malnutrition and reduced work capacity agree with this finding. Noteworthy is a study undertaken by Desai et al. [1984] on marginal malnutrition, body size and work capacity.[81] The subjects were migrant adolescent males in outhern Brazil. A local group of well-to-do adolescent boys of the same age served as controls. Dietary data revealed that energy and nutrient intakes were marginally lower in the migrants, and that they had lower reserves of fat and muscle mass relative to the controls. Work load experiments were undertaken in a manner similar to that in Areskog et al. [1969]. The results were that at submaximal work loads, the migrants, exhibited oxygen consumption and work efficiency similar to that of the

---

79 An ECG was done at regular intervals and the respiration rate and blood pressure were also measured. The work done at a heart rate of 170/min. was obtained by linear interpolation from the data. Sjostrand [1947] and Wahlund [1948] discuss principles for the measurement of work capacity.

80 Similar results were obtained by Satyanarayana et al. [1977], who found that work capacity was correlated with weight, height and habitual physical activity. Weight accounted for 64% of the variation in work capacity.

81 Dutra de Oliveira et al. [1985] provides a concise summary of this and related literature on body size and work capacity.

controls but achieved this at a higher fraction of their work capacity. This manifested itself in heart rates that were significantly higher. Physical work capacity (measured as work at a heart rate of 170/min.) was one-third lower in the migrants.[82]

Dutra de Oliveira et al. [1985] summarise some of this literature. They conclude by observing that

> **"The implications of the association between small body size and impaired productivity are now becoming evident. Our studies in Brazil, and those of others in India, Colombia and Guatemala clearly suggest that small body size due to inadequate dietary intake and the resulting decreased productivity in the lower socioeconomic classes could perpetuate their poverty through a vicious cycle..."**[83]

Again, while nothing definite can be said, the thrust of these studies is all too clear. A history of low intake that manifests itself in small body size and/or low weight is unlikely to create adaptive advantages for the individual in the sphere of physical activity. While the results on work efficiency are somewhat ambiguous, those on work capacity are quite sharp, suggesting that there is a reduction of work capacity in conditions of undernourishment.

These issues concerning physical activity can be depicted in a simple manner, (see Figure 1). We consider two individuals, one with a history of low intake, the other, high. We hold the weight of each individual as a constant, and so, for each individual equation (4) reduces to

$$x_i = r_i + q_i \ , \qquad (i=1,2), \qquad (14)$$

where i is the label of the person. Now, the level of activity, λ_i, depends upon q_i, and it is an increasing function of q_i <u>for a given person</u>. Write this as $\lambda_i(q_i)$. It is, by definition, zero when q_i is nil, and remains zero if x_i

---

82 Satyanarayana et al. [1979] found a similar reduction in the work capacity of Indian boys with a history of undernourishment during childhood.

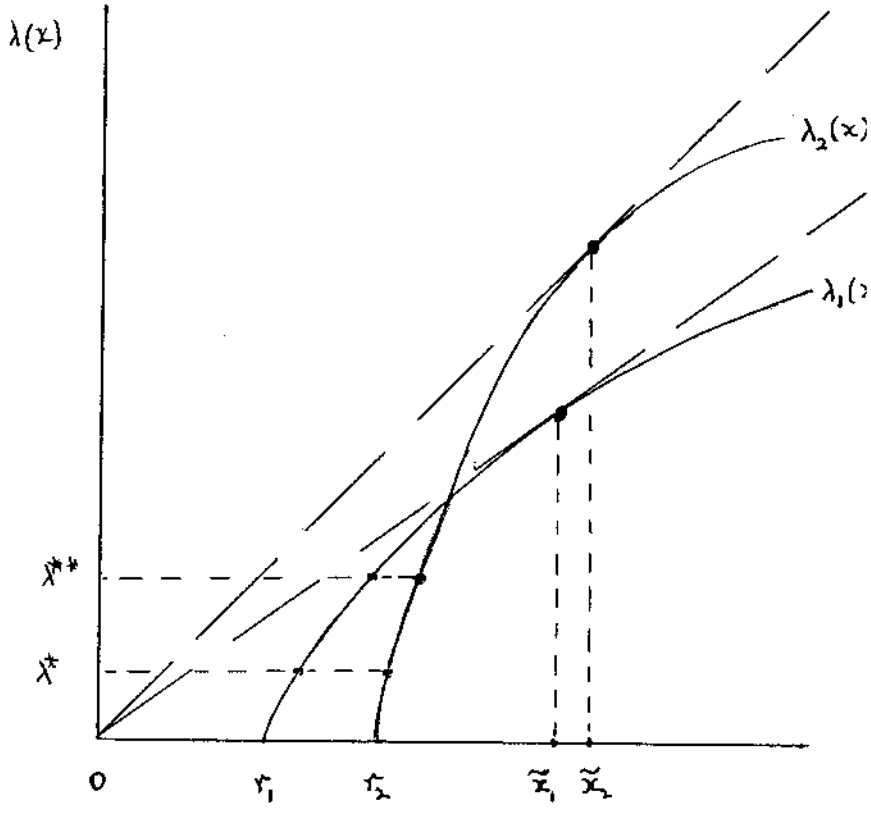
83 Refererences to these studies may be found in Dutra de Oliveira et al. [1985].

falls short of r_i. Using (14) then we may construct the function $\lambda_i(x_i)$ and in Figure 1 we present two such functions, one for each of the persons under review. In Figure 1 we have assumed that the RMR (r_1) of person 1 – the one with a history of lower intakes – is lower than the RMR (r_2) of person 2. λ^* and λ^{**} are two levels of physical activity. As drawn in Figure 1 the two functions relating work output to calorie intake, $\lambda_1(x)$ and $\lambda_2(x)$, are consistent with what is often inferred from Edmundson's experiments: the ratio of work output to energy consumption is higher for the person with the lower intake at each of the two levels of work output, λ^* and λ^{**}. But it is not at all clear why we should jubilate over this. For note that person 2 has a greater capacity to work. At high enough intakes he can do more work per unit of time than his rival. Of greater significance, person 2 is intrinsically more efficient; in that, the maximum ratio of work output to energy consumption that 2 can offer (at intake level $\bar{x}_2$) exceeds the maximum ratio that 1 can offer (at intake level x_1). The implications of this for the operations of the labour market, and thus of employment opportunities, are explored in a preliminary way in Section 4. (See also Dasgupta and Ray, 1986.)

To be sure, there are other possible configurations of such curves. It depends on the activity in question. But before we think of other configurations we should remind ourselves that the poor in less-developed countries are often engaged in strenuous activities, not sedentary. It is a cruel play upon words which labels them the "weaker" members of society, when what they are forced to do in order to earn a livelihood is often back-breaking work.

Over and above this is a more general observation that needs to be made. The issues that are raised by Figure 1 are very much the ones that need to be raised when debating economic policy. We need to have some idea of the

Figure 1



shape of the curves in Figure 1 over a wide range of energy consumption. Even if it were true that the person with a history of low intakes is more efficient at certain specified activity levels, it would not, on its own, be a matter of significance. As we noted earlier, biologists use the term 'adaptation' in a certain manner. This usage does not carry with it the connotation that adaptation is necessarily "costless".

3.7 Storage

This brings us to our last source of regulation: that occuring through adjustment. In a sense we have come full circle, for in Section 2 it was the issue of adjustment and its implications for measurement that was our principal concern. Here, we will limit ourselves to some observations on adjustment through storage, and to some remarks on the possible effects of fluctuating intake on long-term requirements.

We begin with a discussion of the experiment performed by Edholm and his colleagues (Edholm et al., 1970). This study has received much attention in the debate on regulation.[84] The subjects were 64 British infantry recruits observed at 6 centres for 3 weeks. Energy intake was measured every day for each recruit, and daily energy expediture was recorded for 35 of these men. The mean daily intake was 3,850 Kcal and the mean daily expenditure was 3,750 Kcal.

While there was a significant relationship between (time) average intake and expenditure for individual subjects, there was no relationship between food intake and energy expenditure on the same day (nor with one day lags).[85] Moreover, the variability of daily expenditures was considerably

---

84 See above, Section 2.

85 However, Edholm et al. report a "slight tendency" for a day of high intake to be followed by a day or two of low intake, and the same was true of expenditure, though to a les extent.

smaller than that of intake in all the 6 centres. Daily weight and daily calorie balance were positively and significantly related, and the correlation was enhanced when these variables were averaged over five to seven days.

These observations suggest some degree of adjustment on a day-to-day basis, with a significant part of the adjustment being effected by body weight changes. Sukhatme's assertion (see Sukhatme, 1978) that there exists a physiological regulatory mechanism "maintaining body weight" is therefore not unambiguously borne out by the Edholm et al. [1970] data, even though Sukhatme leans heavily on this data for his arguments. Moreover, we have earlier discussed the study of Rand, Scrimshaw and Young [1985] which suggests strongly that there is no adjustment mechanism over and above the storage mechanism, and this necessitates changes in body weight.

A related experiment by Sukhatme and Margen [1978] deals with protein intakes and outputs. This may appear anomalous at first blush; for, there is no apparent connection between energy regulation and 'regulation' in the case of protein use. Nevertheless, this study has been used as a basis by Sukhatme for his postulate of energy regulation, and so merits attention.

Nitrogen inputs and outputs are a proxy for protein use, and the term nitrogen balance is employed to describe the underlying "protein balance".[86] Sukhatme and Margen report nitrogen-balance time series for 6 subjects.[87] Nitrogen intake was controlled by the experimenters, and was held fixed for various periods. It was observed that nitrogen output fluctuated from day to day, with no apparent trend. The authors argue against the possibility that

---

86 See, e.g., Sukhatme and Margen [1978], Rand, Scrimshaw and Young [1979, 1985] and many other protein-use experiments. Protein-use in the body is signalled by nitrogen excretion in the urine and faeces; the former is quantitatively dominant and it is this that is usually employed as a measure of total nitrogen output. See Torun [1985] for an illuminating introduction to the chemistry and metabolism of proteins. See also WHO [1985].

87 For subjects 1 and 2, the series was 84 days long, and for the remainder they were between 30-40 days.

these fluctuations are simply the result of measurement error, or represent uncorrelated noise. In particular, nitrogen balances on adjacent days were found to be correlated, with the extent of serial correlation diminishing as pairs of values farther separated in time were examined. This intraindividual variation persisted even when output was averaged over two weeks. The authors concluded that the protein requirements of an individual maintaining his body weight[88] will vary "from week to week" with "stationary variance". The resulting chain of statistical implications that is drawn from this and the Edholm et al. experiment has already been examined in Section 2.

Let us suppose, for the sake of argument, that experiments dealing with protein-use can be extrapolated to energy balances, although there are problems with such a drastic supposition.[89] Here, then, is indirect evidence supporting short-term adjustments without the need for body weight changes. It is necessary, though, to look at the phenomenon of autocorrelation once again. Recall from Section 2 the use of autocorrelation as an identifying device for regulation, and our criticism of it in the context of the Edholm et al. [1970] experiment. That criticism no longer applies here, for intakes in the Sukhatme-Margen experiment were controlled by the experimenters.

However, suppose that there are long-term trends in body requirements of energy and protein that are not being corrected for. These trends may arise, for instance, from a change in body composition over time (Rand,

---

88 The subjects were given a calorie diet that was chosen to maintain body weight.

89 Rand and Scrimshaw [1984], among others, express strong reservations about such an analogy. One reason for its inappropriateness is the fact that there is a tremendous recycling of old proteins occuring continously in the body. The fresh dietary intake accounts for only a small fraction of the aminoacids that are being synthesised at any one point of time. There is therefore more scope here for variation in the balance, a scope not shared by energy balances (see, e.g., Torun, 1985).

Scrimshaw and Young, 1979). Energy or nitrogen outputs would then mirror this trend and induce autocorrelation in the balance. But such serial correlation is clearly not symptomatic of any form of regulation.

Rand, Scrimshaw and Young [1979, 1985] examine this line of reasoning. Their [1979] experiment used 21 subjects, who were given controlled diet with fixed nitrogen intake.[90] The daily urinary nitrogen excretion was measured and corrected for a linear trend, to minimise possible effects of alteration in body composition. After this was done, only two out of 21 subjects showed significant serial correlation in daily nitrogen balance. Therefore the residual variation (after the trend is removed) has no direct implications.

Rand, Scrimshaw and Young [1985] redo these calculations for five sets of long-term metabolic studies (see details above) with 42 subjects in all. The subjects were supplied with unchanged amounts of protein in each study, and their daily (urinary) nitrogen excretion was measured. Uncorrected nitrogen excretion data exhibited autocorrelation in 19 out of 42 individuals. The data was then "corrected". The major correction was for trend, which was done by fitting various polynomials to the data. The resulting data displayed daily variation, but no autocorrelation except in only four subjects. The authors concluded that for most individuals daily variations in nitrogen excretion are random and not indicative of any form of adjustment.

While no definite conclusions are possible, the available evidence appears to indicate that Rand, Scrimshaw and Young are correct. That adjustment possibilities exist cannot be doubted; the human body can smooth out short-term fluctuations. But it can do so largely through changes in body

---

90 The subjects were divided into two groups. Group 1 (resp. 2) consisted of 16 (resp. 5) subjects and was examined for 8 (resp. 11) weeks. See Rand, Scrimshaw and Young [1979] for more details.

weight, that is, by using up stores of fat or other sources of energy. And it is plain that an adjustment mechanism cannot go on working permanently "on the down side". For that, one has to invoke adaptation.

3.8 The Energy Costs of Fluctuating Intakes

We conclude with some remarks on the possible impact of a fluctuating calorie intake on long-term average requirements. We will assume here that fluctuations are mediated by changes in the body stores of energy, (since this is the tentative conclusion from the available data).

Storage mechanisms add to the energy content of the body when there is surplus intake, and draw on the body stores when there is a deficit. The major energy store is in the form of fat, followed by energy stored in the form of protein.[91] The proportion stored in each form appears to be a genotypic characteristic of the individual, with the fat stores accounting for perhaps 85% of the total, on average.[92]

Now, storage is not free, though the cost of running the stores down is low. Data on the cost of building up stores is hard to come by; nevertheless one can get some information from studies of children and adolescents recovering from malnutrition.[93] Heim [1985] states that to deposit a gram of protein requires 8.7 Kcal of energy, while a gram of fat costs 12 Kcal. Drawing down a gram of protein releases 4 Kcal, while a gram of fat releases 9 Kcal. These differences have an obvious implication: a person with a

---

91 See, e.g., Young and Scrimshaw [1971]. There is also a very small supply of energy (a few hyndred calories) in the form of glycogen in the liver and muscle. The store of glycogen in the liver is in fact the first source of energy release when there is a drop in food intake, or a large gap between meals.

92 See, e.g., Dugdale and Payne [1977], who place the modal value of the protein store (as a fraction of the total) at 0.05 - 0.10, but admit that the distribution has a "long tail". Their definitions of metabolically fat and lean people are based on this ratio. Young and Scrimshaw's data puts the average at 0.15.

93 See the references in Heim [1985].

fluctuating energy intake will need, on average, more than a person with a fixed intake. But this qualitative statement needs to be buttresed by some notion of its quantitative significance. To provide some idea of the magnitudes involved, we use our algebraic framework, together with the knowledge gleaned from the available evidence. Assume, for simplicity, that the resting metabolic rate is a fixed number (r) and that the energy required for physical activity is fixed at q. We may then write the following special case of equations (4) and (13):

$$x_t = r + q + A(w_{t+1} - w_t) \quad , \quad \text{if } w_{t+1} < w_t \qquad (15)$$

$$= r + q + B(w_{t+1} - w_t) \quad , \quad \text{if } w_{t+1} \geq w_t$$

with B>A>0. These inequalities reflect the storage cost.

If intakes are fixed for all time, requirement is simply r + q. Now consider a fluctuating intake; specifically, one that fluctuates between a low value (x^1) and a high value (x^2), with $x^1 < r + q < x^2$. The long-term average intake is $x^* = (x^1 + x^2)/2$. A little algebra shows that for body weight on average to remain constant

$$x^* = (r + q) + [(B-A)(x^2-x^1)]/2(A+B) \qquad (16)$$

so that x^* is clearly greater than r+q. Now we take an example from Sukhatme and Margen [1982]. Using an average requirement of 2,550 Kcal - (r + q) - for the reference Indian male, Sukhatme and Margen calculate the lower "threshold" to be 1,900 Kcal per day (x^1). What does this imply for the value of the larger intake and for the average? The numbers above for storage yield values

of 8.25 for A and 11.5 for B.[94] Using these values in (16), x^2 and x^* turn out to be 3,456 Kcal and 2,678 Kcal respectively, implying an average intake 130 Kcal per day higher than that resulting from a fixed intake. This is no small number. It is about a third of the amount that the FAO (1973) prescribed for moderate activity (see Section 2.1).[95]

3.9 Summary of the Clinical Evidence

We summarise these findings in the context of our original question: what are the implications for the measurement of undernourishment and poverty?

Take the phenomenon of adjustment first. We have argued in Section 2 that even if adjustment is shown to exist, its impact on the statistical cutoff line for determining poverty is ambiguous. But does adjustment which can proceed by "maintaining body weight" exist at all? It appears that the answer is 'no'. Granted, there is a mechanism which is capable of adjustment by regulating the energy stores of the body. But such a mechanism cannot justify a downward revision of human requirements. In fact, it can quite conclusively be established that long-term requirements will increase as a result of fluctuating intake (Subsection 3.8).

We are then left with adaptation. In summary, the clinical literature indicates the following observations. First, undernourishment, especially among rural populations in tropical countries, is likely to lower the digestibility of food. Conversion coefficients such as the Atwater factors are not terribly relevant in these contexts. The effect of lowered digestibility

---

94 These numbers are Kcal per gram. We have assumed that energy is released from protein and fat in the same ratio as it is deposited.

95 There is another possible effect of fluctuating intake on long-term requirements. Fabry [1969] argues that intermittent feeding has very different effects from those of a sustained low intake. His experiments with rats suggest that the basal metabolic rate increases in situations of fluctuating intake (see esp. pages 84-89).

is an increse in the energy needs of individuals, as measured by the energy content of food that they consume. The quantitative magnitude of the revision may be quite large (Subsection 3.3).

Second, there is almost certainly a reduction in the basal metabolic rate under conditions of sustained low intake. The magnitude of this reduction is uncertain, and so is the extent to which the reduction is achieved by better oxygen utilisation of tissue (rather than loss of tissue). It is also unclear whether and to what extent this reduction interferes with physical activity. It may be tentatively concluded that while there is some adaptation here, it is achieved at the cost of some compromise, involving a greater stress on the body. Other related forms of adaptation include diet-induced thermogenesis and changes in the efficiency of energy metabolism. The former is not too well-understood, while the latter does not appear to be particularly significant (Subsections 3.4 and 3.5).

Finally, low intakes may lead to reduced body weight and body size. The impact of this on work efficiency is ambiguous, with the results appearing to depend on the type of activity. However, the impact on work capacity is fairly unambiguous. This is lowered in situations of undernutition (Subsection 3.7).

What implications do these findings have for the measurement of poverty? The existence of adaptation is far from clear. Indeed, where some form of adaptation can be shown to be present (such as in the BMR), it finds a countervailing form of "negative" adaptation, such as reduced work capacity. But the point we wish to make here is different. Let us grant that the foregoing evidence can be construed to weigh in favour of adaptation. Does this imply that we should reduce the figures for nutritional requirements?

This problem brings us back to the centrepiece of Section 2, the logic of statistical inference. It is necessary to describe explicitly the risks that we are willing to turn a blind eye to when measuring undernourishment. It is evident even from our cursory examination of the evidence that adaptation is not purchased free of charge. Besides, we have not even considered the vast literature on the well-established links between low intake and illness. There is not only no question that the "malnutrition-infection complex" exists (Keusch, 1980), but every indication points to the fact that the relationship is a synergistic one.[96] Undernourishment increases susceptibility to infection, just as surely as a history of illness aggravates the symptoms of undernourishment.

Undernourishment precipitates infection. The most common mechanism is the impairing of defence systems. Such impairment may well be a price that has to be paid for adapatation to a reduced intake. The body is weakened in its capacity to form antibodies against an infection. The result is a fatality rate for common diseases and respiratory infections that is unbelievably high.[97] The incidence and severity of illnesses such as diarrhoea also increase with malnutrition.

These connections are reinforced by observed statistical correlations between morbidity and calorie intake. For the case of India, Kumar's careful study of the village of Vembayan in the Trivandrum district of Kerala reveals **"that the frequency of illness lessens as incomes pick up"**, (Kumar, 1985). Noting, too, the predominance of respiratory and gastric ailments, he concludes

---

96 See Latham [1975] for an introduction to the issues involved. There is also the important connection between malnutrition (especially in children) and subsequent risk of mortality. See, e.g. Chen et al. [1980].

97 Latham [1975] reports that the fatality rate from measles in many poor countries is over 15%. In Mexico, the fatality rate is 180 times the U.S rate. The corresponding ratios for Guatemala and Ecuador are even higher - 268 and 480 respectively.

that there is **"evidence of stress on the system - and amongst those engaged in heavy manual work - an indication that a combination of physical exertion and inadequate nutrition are taking their toll"**. The pattern is similar even if one considers very general interstate figures on per capita intake and morbidity in India. Vaidyanathan [1985] does precisely this and obtains an inverse relationship between the two variables.

Illness aggravates undernourishment. Apart from the obvious costs in terms of inability to work during periods of illness, there are serious metabolic costs associated with fever that do not disappear overnight.[98] Bacterial infections are known to lead to an increased loss of nitrogen from the body, and there is a general depletion of body protein, especially from muscles. This leads to a marked reduction in the ability to work. Infections with fever are known to lead to anorexia, characterised by a loss in appetite which leads to a reduced food intake. Intestinal parasites play a central role in the feedback from infection to malnourishment. For example, hookworms cause intestinal blood loss, leading to a considerable loss of iron and thus to iron-deficiency anaemia. And that most common of all parasites, the roundworm, has huge metabolic needs of its own and takes them from the host. Gastrointestinal diseases, especially diarrhoea, are important in precipitating both kwashiorkor and marasmus in children.

The evidence that we have taken some pains to describe is there for all to see. The issue is this: is it justifiable to undertake a reduction in the calorie-based poverty line, Sukhatme-style, on the basis of the available literature? For us, the answer is "no". Nevertheless, one might decide that Sukhatme was "correct", after all, and might thus proceed to revise requirements downwards. In doing so, he must not run away from the explicit

---

98 Each degree centigrade rise in temperature is assoicated with a 13% increase in BMR, and the figure is probably higher for children (Heim, 1985).

value judgements that are involved, in the manner described in this and the preceding section. These judgements are bound up in turn with the risks that the observer deems tolerable for millions of people living and working from day to day on a low intake of food. In particular, the larger the downward revision, the greater is the implied tolerance for these risks. It is easy, but dangerous.

In terms of the value judgements that we are making, we find these risks unacceptable. The statistical logic that is involved in Sukhatme's measurement rule is now clear and certainty lowers the line for measuring undernourishment. But, in the words of Gopalan [1983b],

> **"Those interested in building a strong vigorous nation, of healthy productive adults, and of active children who can run, play and bounce about, grow and develop into healthy adults, may however not be prepared to buy such a prescription."**

4. IMPLICATIONS FOR THE THEORY OF LABOUR MARKETS

4.1 Malnutrition, Unemployment and the Distribution of Assets

The connection between nutrition and work productivity (as in Figure 1 above) leads to a very different economic theory of labour markets.[99] Leibenstein [1957] was probably the first economist to explore this connection in a formal way, and subsequent contributions were made, largely in a partial equilibrium setting by Mirrlees [1975], Stiglitz [1976], Bliss and Stern [1978a] and others. The studies here have mainly been of two types. There is the question of allocation of food among members of a poor utilitarian family farm when nutrition affects productivity (Mirrlees, 1975; Stiglitz, 1976), and the problem of labour employment by a monopsonistic firm (Leibenstein, 1957, Stiglitz, 1976; Bliss and Stern, 1978a). In both these problems, the

---

99 It is not our purpose to survey the literature here, so we shall only indicate the main references.

nutrition-productivity relation has a fundamental effect. In the former, the allocation problem under the utilitarian criterion necessitates unequal division amongst ex-ante identical people. In the latter situation, the result is involuntary unemployment, with employed workers receiving a higher wage than their reservation wage.

In our earlier work (Dasgupta and Ray, 1986, 1987), we developed the implications of the nutrition-productivity relation in a competitive general equilibrium setting. Involuntary unemployment was shown to be linked to the incidence of malnutrition, and these to be related in turn to the production and distribution of income and thus ultimately to the distribution of assets.[1] It was estabilished there that despite the presence of involuntary unemployment, market equilibrium in the theoretical economy is Pareto-efficient. It follows that short-run programmes of employment generation must necessarily involve the redistribution of assets, or food transfers.

These are strong results. But they are based on the assumption that there is a link between current nutrition and current productivity. As we have seen from our survey, this assumption is not correct, unless a person is already weakened from inadequate intakes in the past. Adjustment and adaptation may act to mediate a fluctuating or low intake. It is therefore important to examine how these results are affected when the nutrition-productivity relationship is modified to incorporate regulation.

This is not the place to rework the general equilibrium theory that we have described above, although this can be done. We will instead concentrate on the central feature that is common to all these models - the labour market

---

100 It may be worth noting that there is a strong empirical relationship between the extent of malnutrition and the ownership of land assets. See, e.g., Valverde et al. [1977], or Bairagi [1983].

and involuntary unemployment. Readers familiar with the literature mentioned here will be able to extend the modifications implied for labour market models to the more general theories.

Notice first that our attitude is seemingly contradictory. In Sections 2 and 3 we have been wary of drawing the conclusion that acceptable adjustment or adaptation exists. And now we are proposing to conduct an exercise on the modifications necessary for an understanding of decentralised resource allocation mechanisms occasioned by the phenomenon of regulation. In fact there is no contradiction. In a market with profit-maximising firms, an emloyer could not care less whether the adjustment or adaptation that he is exploiting is socially acceptable. Consequently, while regulation of the sort examined here might not affect our theory of poverty measurement, it may well cause us to rethink the positive economic theory of labour markets.

Note next that many of the models mentioned above employ two sets of assumptions to put the theory to work. The first, as we have already observed, postulates a relationship between current intake and productivity. The second postulates that employers are actually aware of and exploit this relationship. There is an empirical literature exploring the validity of the second assumption, but the evidence is mixed.[101] However, in a competitive environment this second assumption is unnecessary as long as piece rates are paid for tasks and as long as the employer can observe, or monitor, the number of tasks performed. Our work uses this alternative construction. (See Dasgupta and Ray, 1986, for a more detailed discussion.) To simplify our analysis, we will

---

101 See, for example, Rodgers [1975], Bliss and Stern [1978b] and Bliss and Stern [1982]. For short-term contracts, the situation is unclear, though the predominance of partial payments in terms of meals at work may be taken as indirect evidence that the employer attempts to shift the intra-family distribution of food in favour of the worker. For long-term contracts (slavery being the best example) the evidence is stronger and favours the postulate.

consider separately the phenomena of adjustment and adaptation. Our arguments will be expository and heuristic in parts; a formal model can easily be constructed along the lines that we suggest.

4.2 Adjustment and the Labour Market

First consider adjustment. We restrict ourselves to short-term labour contracts here. It is clear that in the case of long-term contracts (say those lasting for a period of months), adjustment makes little or no difference to the theory developed in Dasgupta and Ray [1986].

We suppose that the resting metabolic rate of the individual is fixed at the level r, and the energy required for a given activity level is history-independent, (no adaptation). Assuming a single activity and combining equations 4, 11 and 13 we have:

$$x = r + e(\lambda) + s(w',w), \qquad (17)$$

where w is the initial weight of the individual, w' the weight at the end of the period and $e(\lambda)$ the energy expenditure associated with the activity level λ. Here, weight is to b e viewed as a store of energy for the individual, and as an index of general well-being. Remember that we are concerned with situations where obesity is not at all relevant.[102]

Consider first the situation where there is no work requirement and no food intake for the entire period under consideration. In this case, next period's weight w' is given as a function of initial weight w - call this f(w) - where f(w) solves.[103]

---

102 It is, of course, not being suggested that a small body weight is an unambiguous sign of ill-health. People may be genetically small and healthy. We are saying that given the genotype of the individual, a smaller weight is indicative of greater debility. It goes without saying that these remarks are not valid in the range of obesity.

103 We are assuming for simplicity that such a weight can be defined; i.e. that the worker is not already on the borderline of starvation.

$$s(f(w),w) + r = 0 \qquad (17)$$

Presumably, if the individual fails to find a job that earns him income and demands physical activity from him, he will have some source of energy intake (denoted by x^0) to fall back upon. We assume that to obtain this intake, the individual does not require to supply any significant amount of physical work.[104] If unemployed his end-of-period weight is given by a function k(w) which is the solution to

$$x^0 - s(k(w),w) = r. \qquad (19)$$

Clearly, as long as $x^0 > 0, k(w) > f(w)$.

Given the ranges of weight, the activity and the economic group we are studying, the continued well-being of the individual (at whatever level) is of primary importance to him.[105] So it is reasonable to postulate that the individual will not accept a job which drags his end-of-period weight w' below the level k(w), which is what he can guarantee himself. Once that is guaranteed, he prefers a larger income to a smaller one. So our postulate is that the individual's "preferences" are lexicographic. He first attempts to guarantee a certain level of well-being (k(w)). Once that is done, he tries to maximise income.[106]

---

104 Strictly speaking, this assumption is unnecessary. All that is required is that the individual earns this "reservation intake" from an activity which has relatively low energy demands (begging, odd jobs, living off friends and relative).

105 We are abstracting here from questions of family size and food distribution within the family. These add very little to the analysis (see Dasgupta and Ray, 1986).

106 Given our definition of well-being (not to be confused with the individual's "utility"), increasing well-being and increasing income are not the same thing.

The level of activity λ in employment determines the total income of the individual (which is assumed to be equal to his food intake).[107] In other words, we are supposing that there is a piece wage rate μ which is paid for each unit of the activity (often called an eficiency unit) that is supplied. Assume that firms (or employers) are perfectly competitive. Then standard methods yield the existence of a downward sloping derived demand curve for "efficiency units of labour", E, as a function of the piece rate. Call this $D(\mu)$.

Return to the individual labourer. We impose some structure on the energy function for physical activity in equation (17) by describing its inverse function (call it $\lambda(e)$).[108] Specifically, we assume that $\lambda(e) = 0$ for $0 \leq e \leq e^0$,[109] and it is increasing, continuous and bounded for $e > e^0$.[110]

Now suppose that the individual faces a piece rate of μ. He checks first whether he is able to supply a positive amount of work λ and attain an end-of-period weight of at least $k(w)$. This is equivalent to checking that the equation

$$\mu\lambda = r + e(\lambda) + s(k(w),w) \qquad (20)$$

has a solution with $\lambda > 0$. If it does not, then his labour supply at that wage rate is zero. If it does, then the individual chooses the maximum λ consistent with the satisfaction of (20). This yields the "supply curve" of efficiency units at the individual level. Call it $S(\mu)$.

---

107 These assumptions can be relaxed with no difficulty, (see Dasgupta and Ray, 1986).

108 This (together with a translation to include r) is precisely the function used in the models we have described, and in Figure 1.

109 The number e^0 may be equal to zero. All we require for our analysis is either $k(w) > f(w)$ or $e^0 > 0$. (See Figure 1 above.)

110 These correspond to the assumptions made in Bliss and Stern [1978a] and Dasgupta and Ray [1986, 1987].

For simplicity, consider a continuum of identical individuals, each indexed by a point on the interval [0,m]. The aggregate supply curve is then given by $mS(\mu)$. We will now establish the following result:

If m is large enough, there will be a subset of individuals denied access to the labour market, though they are able and willing to work at the going piece rate. There is involuntary unemployment in the sense that those who find jobs are better off than those who do not. Despite this, the piece rate does not fall.

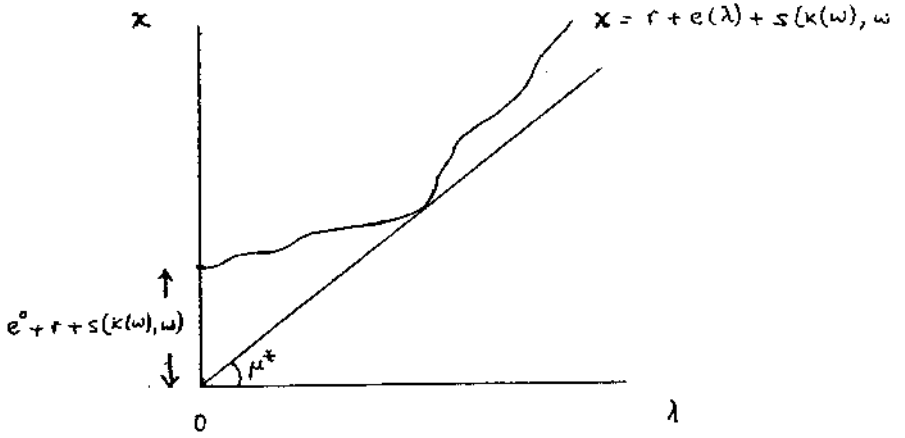
The argument that drives this result is easy to describe verbally. Consider the individual's decision at each piece rate μ. Suppose that (20) has a solution with positive λ. Then $S(\mu)$ is positive. Now lower μ. There will come a point - call it μ^* - when $S(\mu^*)$ is positive, but for any μ less than μ^* (20) does not have a solution with positive λ; and so $S(\mu) = 0$ (see Figure 2). At this threshold $S(\mu^*)$ is positive. This follows from the postulate that either $k(w) > f(w)$ or $e^0 > 0$, (any one of these conditions will do). Denote $S(\mu^*)$ by S^* (Figure 2). We may conclude that the aggregate supply curve has the property that $mS(\mu) \geq mS^*$ if $\mu \geq \mu^*$, and $mS(\mu) = 0$ if $\mu < \mu^*$. Thus the aggregate supply curve of "efficiency units" - that is, the supply of aggregate activity level -, is discontinuous at μ^*. (See Figure 3).

It is now easy to see that if m is large enough - so that mS^* is large enough - the equilibrium piece rate will be μ^*, since the market demand curve, $D(\mu)$, for labour activity will pass through the gap in the supply function at μ^*. At this piece rate everyone is capable of working, but not everyone will get a job. The fraction of employed people will be $D(\mu^*)/mS^*$.[111] The piece rate cannot fall because at a rate lower than μ^* workers cannot supply any effort.

---

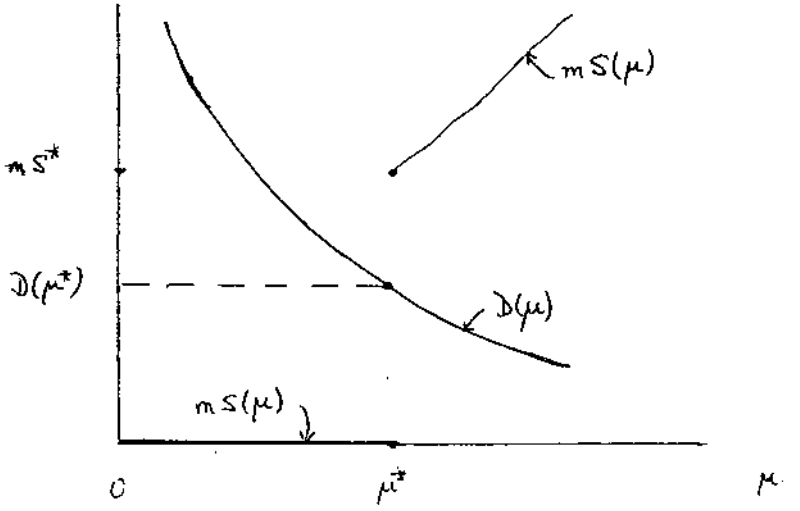
[111] The critical size of the labour force is $D(\mu^*)/S^*$. If m exceeds this there is involuntary unemployment.

Figures 2 and 3



determination of lowest viable piece rate, μ^*.

Figure 2



Involuntary Unemployment arising from fear-productivity link.
$[mS^* - D(\mu^*)] / mS^*$ is % of workforce unemployed

Figure 3

The unemployment is involuntary. People who find employment consume more than people who are unemployed and are therefore distinctly better off.

The reader can easily extend this analysis to the case where there is a heterogeneous labour group, or to the case of a monopsonistic employer. But what is clear from all this is that the standard analysis (as in Dasgupta and Ray, 1986) is robust against the phenomenon of adjustment. There are, of course, new issues arising from the explicit inclusion of adjustment. For instance, the lower is the "reservation weight", k(w), the quicker will be the deterioration of people who are subject to the whims of a causal labour market. Adjustment is a curse in disguise if the labour market is short-term and its workings are influenced by adjustment in the way we have just described. To the extent that people are driven to lower and lower values of k(w) by the lack of alternative opportunities, their ability to adjust in the short-term drives them to work for a reduced piece rate. The combination of adjustment, high labour supply and a casual labour market can be disastrous in the long run.

3.3 Adaptation and the Labour Market

We turn to the implications of adaptation. We analyse this by neglecting adjustment. However, r will now be explicitly a function of history.

Consider an individual at period t. Let $A_1 = (x_0 + x_1 + \ldots + x_{t-1})/t$ denote the average intake of the individual. We will write r as an increasing function $r_t(A_t)$ of the history of intakes. This corresponds to positive adaptation. Moreover, we allow for the possibility that the current energy-requirement function for physical activity also depends on average intake, though we do not specify its direction yet. For an individual at time t, we write

$$x_t = r_t(A_t) + e(\lambda, A_t) \qquad (21)$$

Now consider two types of individuals, 1 and 2, with 1 having a history of lower intakes than 2. Suppose that there is a large number in each group. At time t, suppose that both these groups are trying to sell their services on the casual labour market, where a going piece-rate has been announced. What will be the nature of the market equilibrium?

First suppose that history does not affect the energy requirements for physical activity: i.e., that A_t does not affect $e(.,.)$.[112] Then it is easy to establish the following result:

If the number of people in each group is large, equilibrium in the casual labour market involves the complete absence of group 2 workers. A fraction of group 1 finds employment and the remaining fraction is involuntarily unemployed.

The reasoning behind this claim is simple. Persons in group 1 - those with a history of low intakes - have a lower resting metabolic rate. But we have assumed that a person's nutrition history does not affect the energy expended at any given level of activity; that is, e_t is independent of A_t. Figure 4 presents for a person in each of the two groups the maximum level of activity he is capable of achieving as a function of his calorie intake. It is immediate from the figure that every piece rate which permits a person in group 2 to provide a positive level of activity is also a viable piece rate for a person in group 1. It is also immediate that there exist viable piece rates for persons in group 1 that are not viable for the others. These features are translated to Figure 5, which presents the supply functions of the two groups. As in Figure 3, each supply function has a discontinuity, at μ_1^* and μ_2^* respectively, but the discontinuity for group 2 occurs at the larger piece rate, μ_2^*. If the number of persons in each group is large the market demand

---

112 Recall that in Figure 1 we assumed that A_t does affect $e(.,.)$.

Figures 4 and 5

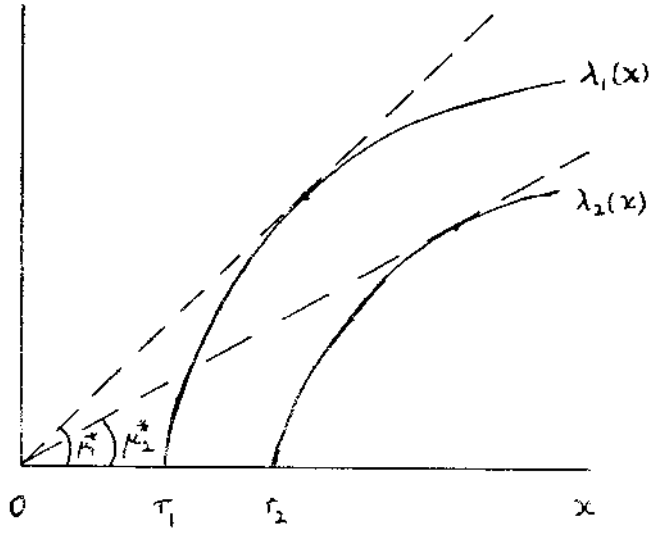


Figure 4

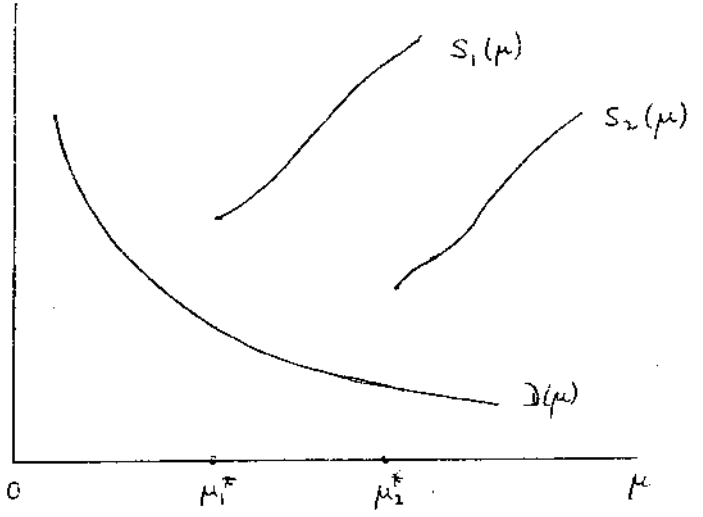


Figure 5

curve for labour activity will pass through both the discontinuities, and no group 2 person will find employment in this labour market. A fraction of group 1 will.

Now this is seemingly paradoxical, the conclusion that it is those with a better nutrition history who are entirely excluded from the labour market in question, while those have the worse history have at least some chance of being employed.[113] It suggests that there are future advantages to being ill-fed now!

In fact there is no paradox here. What the result indicates is that if there is significant adaptation, (never mind its "social acceptability"), and if there are no changes in the energy function for physical activity, then low-intake people may well find a temporary buffer in the short-term, casual labour market.

Moreover, we cannot conclude from this that the casual labour wage rate will settle at a level that permits continued well-being. At the lowered rate of metabolism these labourers may have drastically increased susceptibility to infection and disease. But the casual labour market is insensitive to all this. By its very nature, it is a myopic market.

These statements must be qualified by the possibility that the energy requirements for physical activity may be affected by history, (as in Figure 1). If the activity is such that a history of low intakes reduces efficiency, then there is a trade-off involved and the end result is ambiguous. In addition, if work capacity is considered, the results described here might be reversed as would be if the curves are as in Figure 1. One might tentatively conclude that the results of our example here will apply to activities where a reduced weight is not a huge disadvantage.

---

[113] For vividness one might imagine that a lottery is used to determine which people in group 1 are employed.

What of longer-term contracts? Here, the considerations are very different and are similar to the ones offered in Dasgupta and Ray [1986]. Any such model must include the possibility that a person with a history of low intakes is more easily prey to illness. One such bout of illness imposes a cost on the long-term employer, which he cannot afford to ignore. It seems very likely such considerations will dominate. We conjecture that the relative desirability of the two groups from the point of view of employers is likely to be the complete reverse when we consider long-term contracts. Here, the well-nourished person is at a premium. Moreover, the wage rates in the two markets will reflect this premium. Casual labour wage rates will be lower on two counts. First, due to the adaptation that results in a lowering of the piece rate, and second, the intrinsic myopia of short-term contracts, created by the fact that an illness of the worker imposes no cost on the employer. (See the remarks in Dasgupta and Ray [1986], Section V.)

The theory modified to include adaptation is richer, although more complicated. A full account of this theory is not available. But the considerations that we have mentioned permit us to draw a few broad conclusions, with which we end.

People with a history of low intake are progressively excluded from the more lucrative long-term labour contracts in the economy. The exclusion is progressive because of the wage gap that is likely to persist between casual and long-term markets, resulting in greater disparities over time. However, these excluded people will not lower the poverty rates in the economy by simply dying off. There are markets to absorb such people. These are the casual labour markets, offering a precarious source of living, where underfed, undernourished people by virtue of their "adaptation" actually find a place. But there are no inbuilt mechanisms in these markets that will reduce their undernourishment. Short-term contracts simply do not look ahead.

REFERENCES

Areskog, N. et al, (1969), Physical work capacity and nutritional status in Ethiopian male children and young adults, American Journal of Clinical Nutrition 22, 471-479.

Atwater, W. and F. Benedict, (1903), Experiments on the metabolism of energy and matter in the human body, Bulletin of the Office of Experimental Stations No. 136, Washington, DC.

Bairagi, R., (1983), Dynamics of child nutrition in rural Bangladesh, Ecology of Food and Nutrition 13 (1983), 173-178.

Banerjee, B. and N. Saha, (1970), Energy cost of some common daily activities of active tropical male and female subjects, Journal of Applied Physiology 29. 200-203.

Banerji, D., (1981), Measurement of poverty and undernourishment, Economics and Political Weekly 16, September 26, 1579-1582.

Bardhan, P., (1973), On the incidence of poverty in rural India of the sixties, Economic and Political Weekly, 8, February.

Beaton, G.H., (1981), Numerical Descriptors of the Nutrition Problem: An Analytical comparison of FAO and World Bank Approaches, mimeo. Faculty of Medicine, University of Toronto.

Beaton, G.H., (1983), Energy in Human Nutrition: Perspectives and Problems, (W.O. Atwater Memorial Lecture), mimeo. Faculty of Medicine, University of Toronto.

Benedict, F., Miles, W., Roth, P, and H. Smith, (1919), Human vitality and efficiency under prolonged restricted diet, Carnegie Institute Publication No. 280, Washington, DC.

Bliss, C. and N. Stern, (1978), Productivity, wages and nutrition, Part I: the theory, Journal of Development Economics 5, 331-362.

Bliss, C. and N. Stern, (1978), Productivity, wages and nutrition, Part II: some observations. Journal of Development Economics 5, 363-398.

Bliss, C. and N. Stern, (1978), Palanpur: studies in the economy of a North Indian village, Oxford University Press.

Chafkin, B., (1985), Bashing nutritionists: the "small but healthy?" hypothesis, Economic and Political Weekly 20, May 18, 896.

Chakravorti, S. and M. Panda, (1981), Measurement of incidence of undernourishment, Economic and Political Weekly, 16 August 1, 1275-1278.

Chen, L., Chowdhury, A. and S Huffman, (1980), Anthropometric assessment of energy-protein malnutrition and subsequent risk of mortality among preschool aged children, American Journal of Clinical Nutrition 33 (1980), 1836-1845.

Dandekar, V., (1981), On measurement of poverty, Economic and Political Weekly 25, July 25, 1241-1250.

Dandekar, V., (1982), On measurement of undernourishment, Economic and Political Weekly 17, February 6, 203-212.

Dandekar, V. and N. Rath, (1971), Poverty in India, Economic and Political Weekly 15, January 2-9.

Danforth, E., (1981), Diet-induced thermogenesis. Control of energy expenditure, Life Science 28, 1821-1827.

Dasgupta, P. and D. Ray, (1986) Inequality as a determinant of malnutrition and unemployment, I, Basic theory, Economic Journal, 96, 1011-1034.

Dasgupta, P. and D. Ray, (1986) Inequality as a determinant of malnutrition and unemployment, II, Policy options, forthcoming, Economic Journal, 97.

Dasgupta, R., (1985), Nutrition situation in India: a statistical analysis, Economic and Political Weekly 19, September 7, 1511-1512.

Desai, I., Waddell, Dutra, S., Dutra de Oliveira, S., Duarte., Robazzi, M., Romero L., Desai M., Vichi F., Bradfield R. and J. Dutra de Oliveira, (1984), Marginal malnutrition and reduced physical work capacity of migrant adolescent boys in Southern Brazil, American Journal of Clinical Nutrition 40, 135-145.

Downs, J., (1964), A Study in nutritional dwarfing, American Journal of Clinical Nutrition 15, 275-281.

Dugdale, A., and P. Payne, (1977), Patterns of lean and fat deposition in adults, Nature 266, March, 349-351.

Durnin, J. and R. Passmore, (1967), Energy, Work and Leisure, Heinnemann, London.

Dutra de Oliveira, J., Dos Santos J. and I. Desai, Commentary, (1985), Clinical nutrition of the young child, O. Brunser et al. (eds.), Raven Press, New York, 92-94.

Edholm, O., Adam, J., Healy, M., Wolf, H., Goldsmith, R. and T. Best, (1970), Food intake and energy expenditure of army recruits, British Journal of Nutrition 24, 1091-1107.

Edmundson, W., (1977), Individual variations in work output per unit energy intake in East Java, Ecology of Food and Nutrition, 6, 147-151.

Edmundson, W., (1979), Individual variations in basal metabolic rate and mechanical work efficiency in East Java, Ecology of Food and Nutrition 8, 189-195.

Edmundson, W., (1980), Adaptation to undernourishment: how much food does man need? Social Science and Medicine 14D , 119-126.

Pabry, P., (1969), Feeding Pattern and Nutritional Adaptations, Butterworths Academia.

FAO, (1957), Calorie requirements, Nutritional Studies 15, Rome.

FAO, (1962), Nutrition and working efficiency, Freedom from Hunger Campaign Basic Study 5, Rome.

FAO, (1963), Third world food survey, Freedom from Hunger Campaign Study Basic Study 11, Rome.

FAO, (1973), Energy and protein requirements, Nutrition meetings report series 52, Rome.

FAO, (1978), Fourth World Food Survey, Rome.

Foster, J., (1984), On economic poverty: a survey of aggregate measures, Advances in Econometrics, Basmann and Rhodes (eds.).

Foster, J., Greer, J. and E. Thorbecke, (1984), A class of decomposable poverty measures, Econometrica.

Gomez, F., Galvan, R., Frenk, S., Cravioto, J., Chavez, R, and J. Vasquez, (1956), Mortality in second and third degree malnutrition, Journal of Tropical Pediatrics 2, 77.

Gopalan, C., (1982), The nutrition policy of brinkmanship. Bulletin of the Nutrition Foundation of India, October.

Gopalan, C., (1983a), Small is healthy: for the poor not the rich, Economic and Political Weekly 18 (15), 591.

Gopalan, C., (1983b), Measurement of undernourishment: biological considerations, Bulletin of the Nutritional Foundation of India, July.

Gopalan, C., (1985),The mother and child in India, Economic and Political Weekly 20, January 26, 159-166.

Gopalan, C, and B Narasinga Rao (1974), Dietary Allowances for Indians, National Institute of Nutrition, ICMR, Special Report Series No 60.

Grande, F., (1964), Man under caloric deficiency, Handbook of physiology, Section 4: adaptation to the environment, in D. Dill et al (eds.), American Physiological Society, Washington DC, 911.

Grande, F., Anderson, J. and A. Keys, (1958), Changes in basal metabolic rate in man in semi-starvation and feeding, Journal of Applied Physiology 12, 230.

Heim, T., (1985), Energy metabolism,: theoretical and practical aspects, Clinical nutrition of the young child, O. Brunser et al (eds.), Raven Press, New York, 77-92.

Himms-Hagen, J., (1984), Thermogenesis in brown adipose tissue as an energy buffer. Implications for obesity, New England Journal of Medicine 311, 1549-1588.

Jequier, E., (1983), Does a thermogenic defect play a role in the pathogenesis of human obesity?, Clinical Physiology 3, 1-7.

Jung, R., Shetty, P., James, W., Barrand, M. and B Callingham, (1979), Reduced thermogenesis in obesity, Nature 279, 322-323.

Keusch, G., (1980), Homing in on interventions in the malnutrition-inflection complex, American Journal of Clinical Nutrition 33, 727-729.

Keys, A., Brozek, J., Henschel, A., Mickelson, O., Taylor, H., et al, (1950), The Biology of Human Starvation, Vols I and II, University of Minnesota Press, Minneapolis.

Leibenstein, H., (1957), Economic backwardness and economic growth, Wiley, New York.

Kakwain, N. (1980), Income Inequality and Poverty: Methods of Estimation and Policy Applications, Oxford University Press (New York).

Krishnaji, N., (1981), On measuring the extent of undernourishment: a note on Sukhatme's procedure, Economic and Political Weekly 16, May 30, 989-992.

Kumar, G., (1986), Malnutrition: an economic approach - with applications to India, mimeo. University of Oxford.

Latham, M., Nutrition and infection in economic development, (1975), Food: Politics, economics, nutrition and research,, P. Abelson (ed.), American Association for the Advancement of Science.

Lipton, M., (1983), Poverty, undernourishment and hunger, World Bank Staff Working Paper no. 597.

Mehta, J., (1982), Nutritional norms and measurement of malnutrition and poverty, Economic and Political Weekly 17, August 14, 1332-1340.

Miller, D., (1979), Prevalence of nutritional problems in the world, Proceedings of the Nutrition Society 38, 197-205.

Mirrlees, N., (1976), A pure theory of underdeveloped economies, Agriculture in Development Theory, L. Reynolds (ed.), Yale University Press, New Haven.

Molla, A., (1984), Absorption of macronutrients during the cause stage and after recovery from diarrhea of different aetiologies, Food and Nutrition Bulletin 10 (Suppl.), 289.

Paranjpe, H., (1981), How poor are we? Economic and Political Weekly 16, 1470.

Payne, P. and P. Cutler, (1984), Measuring malnutrition: technical problems and ideological perspectives, Economic and Political Weekly 19, August 25, 1485-1491.

Prynne, C. and D Southgate, (1979), The effects of a supplement of dietary fibre on faecal excretion by human subjects, British Journal of Nutrition 41, 495-503.

Rand, W. and N. Scrimshaw, (1984), Protein and energy requirements - insights from long-term studies, Bulletin of the Nutrition Foundation of India 521, October, 1-2.

Rand, W., Scrimshaw, N. and V Young, (1979), An analysis of temporal patterns in urinary nitrogen excretion of young adults receiving constant diets at two nitrogen intakes for 8 to 11 weeks, American Journal of Clinical Nutrition 32, 1408-1414.

Rand, W., Scrimshaw, N. and V Young, (1985), Retrospective analysis of data from five long-term metabolic balance studies: implications for understanding dietary nitrogen and energy utilization, American Journal of Clinical Nutrition 42, 1339-1350.

Rao, V.K.R.V., (1977), Nutritional norms by calorie intake and measurement of poverty, Bulletin of the International Statistical Institute 47(1), Proceedings of the 41st session invited papers, 645-654.

Rao, V.K.R.V., (1981a), Some nutritional puzzles: a note, Economic and Political Weekly, July 11-18, 1205-1208.

Rao, V.K.R.V., (1981b), Reply to Dandekar, Economic and Political Weekly.

Rao, V.M. and M. Vivekanand (1982), Calorie norm Controversy, Economic and Political Weekly, 17, February 13.

Reutlinger, S. and H. Alderman, (1980), the prevalence of calorie deficient diets in developing countries, World Development, 8, 399-411.

Reutlinger, S. and M. Selowsky, (1976), Malnutrition and Poverty: Magnitude and Policy Options, John Hopkins University Press, Baltimore.

Rodgers, G., (1975), Nutritionally based wage determination in the low-income labour market, Oxford Economic Papers, March.

Rothwell, M. and M. Stock, (1981), Regulation of energy balance, Ann.Rev.Nutr.1, 235-256.

Satyanarayana, K., Naidu, A., Chatterjee, B., and B. Narasinga Rao, (1977), Body size and work output, American Journal of Clinical Nutrition 30, 322-325.

Satyanarayana, K., Naidu, A., and B Narasinga Rao, (1979), Nutritional depreviation in boyhood and the body size, activity and physical work capacity of young boys, American Journal of Clinical Nutrition 40, 542-552.

Schutz, Y., Bessard, J. and E Jequier, (1984), Diet-induced thermogenesis measured over a whole day in obese and non-obese women, American Journal of Clinical Nutrition 40, 542-552.

Scrimshaw, N. and V. Young (1984)

Sen, A., (1976), Poverty: an ordinal approach to measurement, Econometrica 44, 219-231.

Seckler, D., (1982), Small but healthy: a basic hypothesis in the theory, measurement and policy of malnutrition, Newer concepts in Nutrition and their Implications for Policy, P. Sukhatme (ed.), Maharashtra Association for the Cultivation of Science, Pune.

Seckler, D., (1980), Malnutrition: an intellectual odyssey, Western Journal of Agricultural Economics 5, December, 219-227.

Seckler, D., (1984a), The "small but healthy?" hypothesis: a reply to critics, Economic and Political Weekly 19, November 3.

Seckler, D., (1984b), The "small but healthy?" hypothesis: an invitation to a refutation, Impact of P.V. Sukhatme on Agricultural Statistics and Nutrition, P. Narain (Ed.), Indian Society of Agricultural Statistics, New Delhi.

Sims, E., (1976), Experimental obesity, dietary induced thermogenesis and their clinical implications, Clin. Endo. Metab. 5, 377-395.

Sjostrand, T., (1947), Changes in the respiratory organs of workmen at an ore smelting works, Acta. Med. Scand 196 (suppl.), 687.

Spurr, G., Barac-Nieto, M., Reina, J. and R. Ramirez, (1984), Marginal malnutrition in school-aged Colombian boys: efficiency of treadmill walking in submaximal exercise, American Journal of Clinical Nutrition 39, 452-459.

Srinivasan, T., (1981), Malnutrition: some measurement and policy issues, World Bank Staff Working Paper no. 373, February.

Stiglitz, J., (1976), The efficiency wage hypothesis, surplus labour and the distribution of income in L.D.C.'s, Oxford Economic Papers 28, 185-207.

Sukhatme, P., (1961), The world's hunger and future needs in food supplies, Journal of the Royal Statistical Society 124, 463-525.

Sukhatme, P., (1970), Incidence of protein deficiency in relation to different diets in India, British Journal of Nutrition.

Sukhatme, P., (1972), Protein strategy and agricultural development, Presidential address, Indian Journal of Agricultural Economics, January-March, 2-3.

Sukhatme, P., (1974), The protein problem, its size and nature, Journal of the Royal Statistical Society Series A (general) 137, 166-199.

Sukhatme, P., (1978), Assessment of adequacy of diets at different income levels, Economic and Political Weekly 13, Special number, 1373-1384.

Sukhatme, P., (1981a), On the measurement of poverty, Economic and Political Weekly 16, August 8, 1318-1324.

Sukhatme, P., (1981b), On the measurement of undernourishment: a comment, Economic and Political Weekly 16, June 6.

Sukhatme, P., (1982a), Measurement of undernourishment, Economic and Political Weekly 17, December 11, 2000-2016.

Sukhatme, P., (1982b), Newer Concept in Nutrition and their Implications for Policy, Maharashtra Association for the Cultivation of Science, Pune.

Sukhatme, P., and S. Margen, (1978), Models of protein deficiency, American Journal of Clinical Nutrition, 31, 1237-1256.

Sukhatme, P., and S. Margen, (1982), Autoregulatory homeostatic nature of energy balance, American Journal of Clinical Nutrition 35, 355-365.

Taylor, H. and A. Keys, (1950), Adaptation to caloric restriction, Science 112, 215-218.

Torun, B., (1985), Proteins: chemistry, metabolism and nutritional requirements, Clinical nutrition of the young child, O. Brunser et al (eds.), (1985), Raven Press, New York, 99-118.

Uauy, R., (1985). Commentary, clinical nutrition of the young child, O. Brunser et al. (eds), Raven Press, New York, 96-98.

Vaidyanathan, A., (1985), Food consumption and size of people: some Indian evidence, Economic and Political Weekly 20, Review of Political Economy, July 27, PE-79 - PE-84.

Valverde, V., Martorel, R., Mejia-Pivaral, V., Delgado, H., Lechtig, A., Teller, C. and R Klein, (1977), Relationship between family land availability and nutritional status, Ecology of Food and Nutrition 6, 1-7.

Wahlund, H., (1948), Determination of the physical working capacity, Acta. Med. Scand. 215 (suppl.).

Waterlow, J., (1976), Classification and definition of protein-energy malnutrition, Nutrition in preventive medicine: the major deficiency syndromes G. Beaton and J. Bengoa (eds)., World Heath Organisation, Geneva.

Watts, H. (1968), An Economic Definition of Poverty, in D.P. Moynihan, ed., On Understanding Poverty, Basic Books (New York).

Weir, J., (1949), New methods for calculating metabolic rate with special reference to protein metabolism, Journal of Physiology 109, 1-9.

Widdowson, E., (1947), A study of individual children's diet, Special Report Series no. 257, Medical Research Council, London.

WHO, (1985), Energy and Protein Requirements: Report of a joint FAO/WHO/UNU Expert Consultation, Technical Report No. 724, Geneva.

Young, V. and N. Scrimshaw, (1971), The physiology of starvation, Scientific American 225, 14-21.

Zurbrigg, S., (1983), Ideology and the poverty line debate, Economic and Political Weekly 18, December 3, 2083-2084.