E C O N O M I C S B U L L E T I N

The effect of food intake on longevity

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Abstract

We investigate the startling but real possibility that a reduction in calories can lead to a increase in longevity. In perhaps the simplest model, it may be evolutionarily optimal for a permanent reduction in the food supply to cause such an increase in longevity. However, it is impossible to account for all the data if fertility is a function of the food input alone. We propose, therefore, a more flexible model, incorporating both metabolic by-products and infectious disease. Where there is little infectious disease, the only effect of decreased food is to decrease the production of metabolic by-products, so individuals have increased life expectancy. On the other hand, where infectious disease is prevalent, decreased food intake increases mortality because the resulting reduction in immune function increases the impact of the disease. These predictions are consistent with an empirical model based on income per capita and calorie intake per capita across modern nations.

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1 INTRODUCTION

Empirical research on a wide range of animals has demonstrated a startling possibility that has attracted much recent attention: That less food may lead to an increase in longevity. (See Heilbronn and Ravussin (2003) for a recent review.) Although the evidence concerning the effect on humans of substantial calorie reduction is incomplete, since the longevity of humans makes investigating this experimentally difficult—to say the least, preliminary data concerning individuals in richer societies is consistent with such an increase in longevity. These data concern a favorable effect of calorie reduction on serum cholesterol, for example. (See Walford *et al* (1992), and Martin *et al* (1986).) However, from a wider empirical point of view, these experimental data are rather puzzling. That is, average life expectancy is surely highly positively correlated with food consumption per capita, across a large sample of modern nations with widely varying levels of economic development. How can these apparently conflicting sets of observations be reconciled? What might be the underlying causes of the effect of food consumption on longevity? Answering such questions not only has important implications for the description of national economic development and of individual health status, but also for the interpretation of archaeological data.

How might evolutionary models help explain these phenomena? To this end, we adopt the hypothesis that even modern behavior is evolutionarily appropriate, with the sole crucial exception that the last link from behavior and circumstances to fertility has been weakened or broken, as a result of effective contraception, in particular. Although we accept that theoretical predictions concerning fertility might not be empirically borne out, predictions concerning mortality are intended to be taken at face value.

The present approach should be contrasted with the hypothesis that modern life expectancy is reduced by overeating. Under this hypothesis, our current overindulgence in rich and sweet foods arises from tastes that evolved in severely calorie-restricted circumstances when such tastes were appropriate. It is becoming increasing clear, however, that late hunter-gatherers were surprisingly well-nourished. For example, although it is well-known that that there is an ongoing increase in stature in recent history, it is less well-known that this increase has so far merely roughly restored the stature humans had during hunting and gathering. (See Angel (1975, Table 1).) Furthermore, hunter-gatherers seemed to suffer from relatively little infectious disease, presumably due to the low population densities at which they lived. (See Cohen and Armelagos, (1984), and Steckel and Rose, (2002).) Since the circumstances faced by hunter-gatherers were then surprisingly similar to our current circumstances, it is worth considering how the modern relationship between food intake and longevity could have arisen as a biologically optimal adaptation.

The first model we consider here—Model I—is perhaps the simplest possible. It is related to the previous biological literature, and captures the fundamental biological trade-off between energy expenditure on current reproduction and energy expenditure to reduce mortality. (The Appendix shows that Model I illu-

minates some of the discussion in this previous biological literature to Model I.) If increasing the food supply increases mortality, then expenditure on mortality reduction is essentially an inferior good.

An apparently quite distinct intuition concerning the effect of food on longevity runs as follows. Suppose that individuals are subject to stochastic fluctuations in the food supply. If there is a shortfall in the current food supply, it seems that such an individual might find it advantageous to dramatically reduce reproduction in the current period, shifting energy resources instead into survival, with the aim of holding out until the food supply improves. We show that Model I can be directly reinterpreted in this light.

For either of these effects to apply in Model I, fertility must be a convex function of energy input, over the relevant range. If people in modern richer societies are subject to these effects, this convexity arises at high energy input levels, which seems implausible *a priori* given the presence of other factors of production involved in reproduction.

Even more basically, it is impossible to account for all of the observations in terms of food intake alone. For example, the caloric intake for "Biosphere 2" was about 30% less than planned, as is similar to the cuts used in experiments on rats. If average US food intake in 2002 were cut by 30%, however, this yields a food intake near that for Gabon. Far from having a life expectancy 50% greater than in the US, where the figure is 77.1 years, Gabon's life expectancy is only about 56.7 years.

A more direct and flexible approach to this issue is suggested by recent research on the biochemical determinants of cellular aging. This research shows that metabolic activity in the cell produces various biochemical by-products, including, for example, reactive oxygen species, or ROS. These by-products are implicated in aging, with ROS's implicated in some serious neurological disease for example. (See Love and Jenner (1999).) Although it is possible to clean up such by-products, it seems very costly. It is not impossible to prevent aging, that is, but it may be too expensive. (McElwee *et al.* (2004).) In this setting, a reduction in food supply may increase longevity, simply because the reduction in metabolic activity entails fewer biochemical by-products being produced.

However, we also argue that it is relevant that the data concerning the longevity enhancement of rodents by calorie reduction were obtained under laboratory conditions, where the animals were largely sheltered from infectious diseases. Similarly, individuals who live in developed economies can, to some extent, protect themselves from exposure to infectious disease and can effectively treat it if it nevertheless occurs. On the other hand, humans living in less-developed nations are exposed to a variety of infectious diseases, and lack effective treatment. In these less favored circumstances, metabolic expenditure by the individual on the immune system must be crucial.

We therefore develop an alternative model—Model II—that incorporates the effects of both metabolic by-products and of the resistance to infectious disease by the immune system. At low levels of infectious disease, the production of metabolic by-products is the dominant effect, and an increase in food intake leads to a reduction in longevity, as is consistent with the data from rodents

in laboratories. (The increase in food supply is nevertheless evolutionarily advantageous, since the increase in fertility outweighs the decrease in longevity.) At high levels of infectious disease, as apply in poorer modern nations, for example, an increase in food intake leads to increased longevity because it leads to an increase in the energy used by the immune system, and the favorable effect of this outweighs the deleterious effect from the increase in metabolic by-products. Again, Model II bears direct reinterpretation in terms of the response of individuals exposed to a stochastically varying food supply.

The paper finally checks the predictions of Model II for human life expectancy as a function of food intake and income per capita, using recent national data. Income is intended as a proxy for public-health measures that serve to reduce the incidence of infectious diseases, and for the provision of health care that would treat such diseases if they did arise. Each of the explanatory variables food intake and income indeed have significant linear effects on longevity, when used in a regression to explain life expectancy. Furthermore, the coefficient of an interaction term between food intake and income is negative and statistically significant. Thus increased food intake has a positive effect on longevity at low income levels, but a negligible or negative effect at high income levels. The estimated per capita income at which the change in sign occurs corresponds to a nation near but not at the top of the world per capita income table. The estimated model thus suggests that increased consumption of food in richer modern nations actually decreases longevity.

2 MODEL I—REPRODUCTION VERSUS SURVIVAL

Consider an adult animal that, at age $t = 1, 2, ...$, has available constant energy "income" y. An amount s_t is devoted to improve survival to the next period, so this probability of survival is $p(s_t)$, where $p'(s) > 0$, $p''(s) < 0$, for all $s > 0$. For simplicity, it is assumed that $p'(0) = \infty$, which serves to rule out $s = 0$. Energy cannot be stored, so the remaining energy, $y - s_t \geq 0$, is used to produce expected offspring according to $f(y - s_t)$. It is assumed that $f'(c) > 0$, for all $c \geq 0$ and that $f(0) = 0$.

The demographic description of the population is then completed as follows. Newborns, with age $t = 0$, have no energy budget, and no fertility, but survive the first period of life with exogenous probability $\bar{p}$.

Define now $\bar{V} = \max_{s\in[0,y]} \left[f((y-s) + p(s)/\bar{p}\right]$. If r is defined by $1+r = \bar{p}\bar{V}$, then $\bar{V}$ solves $\bar{V} = \left(\max_{s\in[0,y]} \left[f(y-s) + \frac{p(s)}{1+r}\bar{V}\right]\right)$. That is, since it solves this Bellman equation, $\bar{V}$ is maximized expected offspring, discounted at rate $1 + r$, for an adult of any age $t = 1, 2, ...$

Suppose now that r^* is a feasible steady state growth rate, such that $r^* > r$. Suppose also that V^* is the associated expected offspring for an adult of age $t = 1$, discounted at rate r^*. In general, the Euler-Lotka equation in the present circumstances is $1 = \frac{\bar{p}f(y-s_1)}{1+r} + \frac{\bar{p}p(s_1)f(y-s_2)}{(1+r)^2} + ... = \frac{\bar{p}}{1+r}\left(f(y-s_1) + \frac{p(s_1)f(y-s_2)}{(1+r)} + ...\right)$.

In particular, then $1 + r^* = \bar{p}V^*$, so that $V^* > \bar{V}$. Without loss of generality, r^* and V^* are generated by an age-independent policy, $s(\cdot)$, say, so $V^* = [f((y - s(y)) + p(s(y))V^*/(1 + r^*)] = (f((y - s(y)) + p(s(y))/\bar{p})$, contradicting the definition of $\bar{V}$. Hence r is the maximum possible steady state growth rate for this population.

It follows that the optimal choice of s maximizes $1 + r = \bar{p}f(y - s) + p(s) = \bar{p}\left(f(y - s) + p(s)/\bar{p}\right)$. If this optimum value of s is interior, then the first-order condition is $-f'(y-s)+\frac{p'(s)}{\bar{p}} = 0$,with $f''(y-s)+\frac{p''(s)}{\bar{p}} < 0$ as the second-order condition. For simplicity, this concavity condition is assumed to hold globally. Now, $\frac{d(y-s)}{dy} = \frac{\frac{p''(s)}{\bar{p}}}{f''(y-s)+\frac{p''(s)}{\bar{p}}} > 0$, but $\frac{ds}{dy} = \frac{f''(y-s)}{f''(y-s)+\frac{p''(s)}{\bar{p}}} < 0$ iff $f''(y - s) > 0$. That is, an increase in energy income always leads to an increase in expected offspring. Such an increase in energy income leads to a decrease in longevity if and only if the fertility function f is (locally) convex.

2.1 A Stochastic Reinterpretation

Consider instead how the individuals modelled above would respond to transitory shocks in stochastic energy income. Suppose then that the y_t are identically distributed random variables, independent across individuals, age, and time, and that the population is large. Each individual can condition current behavior on the current realization of y_t, but can condition only on the distribution of future incomes. Proceeding in an analogous fashion to the nonstochastic case, define now $\bar{V} = E_{y_t}\left(\max_{s_t\in[0,y_t]}\left[f(y_t - s_t) + \frac{p(s_t)}{\bar{p}}\right]\right)$. If r is defined by $1 + r = \bar{p}\bar{V}$, then $\bar{V}$ obviously solves $\bar{V} = E_{y_t}\left(\max_{s_t\in[0,y_t]}\left[f(y_t - s_t) + \frac{\bar{V}p(s_t)}{1+r}\right]\right)$. But, since it solves this Bellman equation, $\bar{V}$ is again maximized expected offspring, discounted at rate $1+r$, for an adult of any age $t = 1, 2, ...$ Since the population is large, the law of large numbers implies that $\bar{V}$, and hence r, are realized exactly. It is not hard to show that r is the maximum possible steady state growth rate for this population.

Thus the optimal choice of s_t maximizes $\bar{p}f(y_t-s_t)+p(s_t) = \bar{p}\left(f(y_t - s_t) + p(s_t)/\bar{p}\right)$. Remarkably, this is precisely the same criterion as that describing the response to non-stochastic income. Hence the short-run individual response to a transitory variation in stochastic income is precisely the same as the long-run aggregate response to a permanent change in non-stochastic income. It follows that a short run decline in stochastic energy income leads to an increase in survival only if the fertility function f is locally convex.

The following is an intuition for the close connection between the results in the two scenarios. Under the assumptions here, producing an offspring that survives to the next period has precisely the same evolutionary payoff as having the parent survive to the next period. The optimal strategy for both the short-run stochastic case and the long-run non-stochastic case is then to equate the marginal benefit of energy spent to enhance these two options. That is: $p'(s_t) = \bar{p}f'(y_t - s_t)$. Since this optimal choice depends only on the individual's realized

income y_t, whatever the basis for this, the optimal choice is necessarily the same in the two cases.

For Model I to generate a reduction in longevity from an increase in food intake, the production function for offspring must be at least locally convex. Such convexity, however, seems only plausible at relatively low levels of food intake.

3 MODEL II—METABOLIC BY-PRODUCTS, INFECTIOUS DISEASE AND MORTALITY

Even more importantly, recall that it is simply impossible to account for all the experimental and national aggregate data using a model in which fertility depends on food intake alone. A more realistic model then requires at least one more explanatory variable.

Also recall that recent research has established the production of metabolic by-products as an underlying cause of aging. Model II then builds in an appropriate effect along these lines. Suppose then that each adult has deterministic and constant energy income $y \in [0, \bar{y}]$, for some bound $\bar{y} > 0$. The production function for offspring from energy devoted to this, c, is again f where $f'(c) > 0$, for all $c \in [0, \bar{y}]$; $f(0) = 0$ but f is now concave, so $f''(c) < 0$, for all $c \in [0, \bar{y}]$. The probability of survival is now $P(y, \alpha, s)$. Here, $y \in [0, \bar{y}]$ is energy income from the previous period. Its inclusion in the survival function represents the deleterious effect of metabolic by-products, so $P_y < 0$. The parameter α represents the incidence of infectious disease, so that $P_\alpha < 0$. As in Model I, $s \in [0, \bar{y}]$ represents energy expenditure on survival. In Model II, this expenditure is more specifically on the immune system, to counteract infectious disease, so that $P_s > 0$, $P_{ss} \leq 0$, and $P_{\alpha s} > 0$, whenever $\alpha > 0$. In the absence of infectious disease, when $\alpha = 0$, however, investment in the immune system has no benefit, so that $P_s(y, 0, s) = 0$.

Reasoning as in Model I, define now $\bar{V} = \max_{s \in [0, \bar{y}]} \left[f((y - s) + P(y, \alpha, s)/\bar{p} \right]$. If r is defined by $1 + r = \bar{p}\bar{V}$, then $\bar{V}$ solves $\bar{V} = \left(\max_{s \in [0, y]} \left[f(y - s) + \frac{P(y, \alpha, s)}{1 + r} \bar{V} \right] \right)$. That is, since it solves this Bellman equation, $\bar{V}$ is maximized expected offspring, discounted at rate $1 + r$, for an adult of any age $t = 1, 2, ...$ It follows, furthermore, that r is the maximum possible steady state growth rate for this population.

Hence the expression $\bar{p} f(y - s) + P(y, \alpha, s) = 1 + r$ is to be maximized over s. If $\alpha = 0$, the optimal $s = 0$, as seems relevant to a laboratory rat, or to an individual in a prosperous nation, both benefiting from a low disease environment. In this case, where there is no choice to be made, an increase in the food supply enhances current fertility, but reduces survival.

However, when $\alpha > 0$, it may be optimal to choose $s > 0$. The first-order condition for this is $\bar{p} f'(y - s) = P_s(y, \alpha, s)$. It follows that $\frac{ds}{dy} = \frac{\bar{p} f'' - P_{sy}}{\bar{p} f'' + P_{ss}}$. If $P_{sy} \geq 0$, it follows that $\frac{ds}{dy} > 0$, so an increase in y leads to an increase in s.

Under the present assumptions, in particular the concavity of f, if y did not appear in P, an increase in y would increase s. The assumption that $P_{sy} \geq 0$ assures that the appearance of y in P reinforces this effect.

It is a reasonably weak assumption that there exist $\ell, m, n > 0$ such that $P_{\alpha s} \geq \ell$, $\frac{dv}{dy} = \frac{\bar{p}f'' - P_{sy}}{\bar{p}f'' + P_{ss}} \geq m$, and $P_y \geq -n$, for all $\alpha > \bar{\alpha}$, where $\bar{\alpha} > 0$ is any lower bound, and for all $s, y \in [0, \bar{y}]$. It then follows that $\frac{dP(y,\alpha,s)}{dy} = P_y + P_s \frac{ds}{dy} \geq -n + \alpha \ell m > 0$, for $\alpha > \max\left[\frac{n}{\ell m}, \bar{\alpha}\right]$. Thus, the net effect of an increase in food intake, y, is to increase survival, whenever infectious disease is a sufficiently important factor, as is presumably true for animals in the wild, and for humans in less-developed modern nations.

Despite the direct effect of increasing y in lowering survival, the overall fitness effect of greater food is assumed to be always positive, whether the optimal value of s is zero or not. A sufficient condition for this is $\bar{p}f'(y) + P_y(y, \alpha, s) > 0$, for all $s, y \in [0, \bar{y}]$. Under this condition, it would never pay for the organism to consume only some of the available food.

3.1 A Stochastic Reinterpretation

It is worth noting that Model II can also be reinterpreted as an optimal individual response to transitory shocks in energy income. Suppose then that the y_t are identically distributed random variables, independent across individuals, age, and time, and that the population is large. Proceeding in an analogous fashion to the nonstochastic case, define now $\bar{V} = E_{y_t}\left(\max_{s_t \in [0, y_t]}\left[f(y_t - s_t) + \frac{P(y_t, \alpha, s_t)}{\bar{p}}\right]\right)$. If r is defined by $1 + r = \bar{p}\bar{V}$, then $\bar{V}$ is again maximized expected offspring, discounted at rate $1 + r$, for an adult of any age $t = 1, 2, ...$ It follows, again, moreover, that r is the maximum possible steady state growth rate for this population.

Since the expression $\bar{p}f(y_t - s_t) + P(y_t, \alpha, s_t)$ is to be maximized over s_t, and the short run individual response to a transitory variation in stochastic income is precisely the same as the long run aggregate response to a permanent change in non-stochastic income. That is, in the range where a fall in the permanent energy income leads to a decrease in mortality, individuals who experience idiosyncratic energy shortfalls will also have lower mortality. The intuition for this result is the same as for Model I.

3.2 What do National Data Reveal about Food Intake and Longevity?

We test the implications of our structural Model II by analyzing data on calorie intake by humans. A randomized trial concerning the longevity of humans with several levels of calorie intake and several levels of exposure to infectious diseases would clearly be unethical and would take a very long time, given the longevity of humans. In any case, the relationship that exists in the real world between these variables may differ from that arising in a controlled laboratory environment, and is of independent interest. We therefore use recent

national data. In particular, as a negatively correlated proxy for exposure to infectious disease (and the lack of health care) we use gross national income per capita (GNI). (Gross national income per capita, at purchasing power parity, for 2002, from the World Bank at http://devdata.worldbank.org/data-query/.) The model of the last section predicts a negative cross-partial between calorie intake and income. We estimate the following model.

$$\begin{aligned}\text{Life expectancy} = \quad & \beta_1 * (\text{Calorie}) + \beta_2 * (\text{GNI per capita}) + \beta_3 * (\text{GNI per capita})^2 \\ & + \beta_4 * (\text{Calorie*GNI per capita}) + \beta_5 + \text{ error term},\end{aligned}$$

where the error term is uncorrelated with any of the explanatory variables. The variable "Calorie" refers to the average daily calorie intake in a particular country. (Calorie data for 2002 the Food and Agriculture Organization at http://faostat.fao.org/faostat/. Life expectancy data for 2002 from the Worldbank, http://worldbank.org/data/.) Using ordinary least squares, we find the following.

Table 1 Regression of Life Expectancy on Calorie Intake and Income

Variable	Coefficient	Standard error	t-statistic	Probability
Calorie	0.012877	0.002447	5.262832	0.0000
GNI per capita	0.003077	0.000632	4.869533	0.0000
GNI per capita squared	-1.90E-08	1.22E-08	-1.550202	0.1232
Calorie $*$ GNI per capita	-6.23E-07	2.47E-07	-2.520368	0.0128
Constant	23.69238	5.592733	4.236280	0.0000

$R^2 = 0.641361$

Thus, the coefficients of the linear terms in calorie intake per capita and GNI are positive and significant, as anticipated. Moreover, the estimate of the cross partial derivative of life expectancy with respect to to the product of calorie intake and GNI is negative and statistically significant at the 95% confidence level. Thus, the empirical results using data on humans confirm a key implication of our structural model—that the relationship between calorie intake and longevity depends on the prevalence of infectious diseases (and the lack of good health care).

Although this value is clearly rather imprecisely estimated, it is interesting that the level of GNI at which the marginal gain from increased food intake becomes zero is implied to be about $ 20,670 ('International Dollars'). This is just over the GNI of Spain ($ 20,460) which ranks 24th out of the sample of 154 countries for which all data were available. That is, the data suggest a small negative effect for higher food intake for the most prosperous 20 or so countries.

How large is the estimated effect of calorie restriction in rich modern societies? If the US food intake in 2002 of 3765.6 calories were cut by 30%, while maintaining the US 2002 GNI of $ 35,060, this generates approximately a 10 year increase in life expectancy, along the fitted curve. This represents much less than the 50% increase in life expectancy often seen in laboratory settings, but it is substantial nevertheless.

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APPENDIX—MODEL I AND THE PREVIOUS BIOLOGICAL LITERATURE

Shanley and Kirkwood (2000) consider the evolutionary effect of a stochastically variable food supply. A key feature of their model is a reproductive overhead, or fixed cost, for reproduction. They claim, on the basis of simulations that are not fully described, that it is optimal for a temporary shortfall in the food supply to induce a large reduction in resources used for current reproduction, thereby increasing resources devoted to survival. It is optimal, that is, for the animal to bide its time until the current temporary shortage is over. Why then does a mouse that has a fixed restricted food supply for its entire life live longer? The answer that Kirkwood (1999, Chapter 12, especially p. 181) gives can be paraphrased as follows: This response is favored by evolution in the short run. It is also optimal in the long run, he argues, just because what happens in the long run in response to such a permanent reduction in food is irrelevant. That is, such a mouse would not survive to reproduce at all. This answer is not convincing, since if there are animals that exhibit increased longevity in response to a mild permanent reduction in food, but still reproduce, this exact response is evolutionarily relevant.

The present Model I illuminates this issue, since reproduction there enjoys increasing returns to scale, as a generalization of reproductive overhead. On the other hand, this model is also otherwise simpler than Shanley and Kirkwood's in that it omits consideration of juvenile mortality, for example. Its properties can be obtained analytically, in contrast to the simulations used by Shanley and Kirkwood. In Model I, if a shortfall in individual stochastic energy income produces a decrease in mortality, a permanent reduction in the non-stochastic food supply also induces an increase in longevity. (This contrasts with the claim by Finch and Kirkwood 2000, p. 68, that the effect exemplifies the key role of chance in life history.) This effect does not arise because it is irrelevant what happens in the long run, but is the only appropriate evolutionary response. The claim of Shanley and Kirkwood (2000) is therefore more robust than asserted by Kirkwood (1999).

In order to further illuminate the Shanley and Kirkwood model, consider then the effect of a threshold, or "reproductive overhead" in Model I. Suppose this is at energy level $e > 0$, say, so that $f(c) = 0$, for all $c \leq e$. Suppose that the function f is increasing and concave beyond this point, so $f'(c) > 0$ and $f''(c) < 0$, for all $c > e$. The reproductive overhead thus bears the entire burden of generating a drop in longevity with an increase in income. Note that f must have a kink at $c = e$, but it is assumed that $\lim_{c \downarrow e} f'(e) < \infty$.

What is the general form of the functions s_t and c_t? There is an initial range of energy incomes within which $s_t = y_t$. At some critical energy income level, s_t falls discontinuously, and c_t rises discontinuously. For higher energy income levels, both s_t and c_t are strictly positive and increasing. Note that, once expenditure on current reproduction becomes positive, this expenditure strictly exceeds e. All reproduction can be deferred even when this is not forced by the existence of the overhead. Note finally that the functions s_t and c_t are well-

defined even when the energy income is less than the reproductive overhead: It is optimal in these cases to devote all energy income to survival.

At first, this might seem paradoxical, since there is then no reproduction. However, this choice makes the best of a bad situation. The growth rate of the population is $r = p(0) - 1 < 0$ when there is no energy income. Indeed, as long as energy income is less than the reproductive overhead, it is simply impossible to ever reproduce. However, enhancing survival does pay and so all energy should be devoted to this, even though growth rates must remain negative. Even when energy income is a little above the reproductive overhead, it will still pay to concentrate on survival.