hypothesis

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Milder forms of obesity may be a good evolutionary adaptation: 'Fitness First' hypothesis

T. P. Rakesh[1*], T. P. Syam[2]

[1]Assistant Professor in Medicine, Government Medical College, Thrissur, Kerala, India

[2]MS Scholar, Department of Electrical Engineering, Indian Institute of Technology, Madras, India 600036

*Correspondence:
rakeshtp@yahoo.com

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ABSTRACT The prevalence of obesity is steadily increasing and is considered maladaptive, as it is a risk factor for diabetes, hypertension, cardiovascular illnesses and cancer. However, contrary to popular belief and expectations, recent studies have shown that people with milder grades of adiposity survive better (obesity paradox), both in normal and adverse conditions. Several new observations have been made on how insulin resistance accompanying obesity may be beneficial in selected situations. Insulin resistance operates at the post receptor level and selectively involves the phosphatidylinositol 3-kinase pathway controlling glucose metabolism while leaving the mitogen-activated protein kinase pathways intact, which promotes somatic growth. In insulin-resistant states, glucose is shunted away from the glycolytic pathways to the pentose phosphate pathway generating more nicotinamide adenine dinucleotide phosphate (NADPH) for antioxidant enzymes for combating stress. Mild obesity improves survival probability but at the same time decreases fertility. Anthropological evidence shows that humans produce fewer children in resource-rich environments, leading to improved biological fitness of progeny. This article examines the situation of the obesity epidemic from a fresh evolutionary point of view, discusses and integrates the evidence from medicine, molecular biology, evolution and anthropology, and hypothesizes that milder forms of adiposity may be an evolutionary adaptation of humans to a resource-rich environment – a mechanism improving survival and promoting investment in fewer offspring, thereby improving the biological fitness of the race. However, this article does not recommend that readers maintain a bulging waistline.

Illustration by Eloïse Kremer

INTRODUCTION Obesity is a state in which excess fat is deposited at various sites in the body, gradually leading to a variety of health-related problems. The incidence of obesity is increasing worldwide; it is estimated that there are more than 1.4 billion people in the world who are overweight, of whom 500 million are obese[1]. In the USA, in 2012, it was estimated that 67.3% of people were obese or overweight[2].

As obesity became a symbol of richness and plenty, hypotheses regarding why humans become obese primarily revolved around the environmental changes gifted by modern society. Thus, surplus calorie intake and sedentary lifestyle were considered modifiable environmental risk factors leading to obesity in the background of genetic predisposition. The evolutionary explanations of obesity include the thrifty gene hypothesis (TGH), which states that the organism senses the adverse initial conditions of malnutrition in the foetal stage and, as an adaptive response, eats more and collects resources for future survival. Several other theories incorporate the change in the lifestyle of humans from the hunter-gatherer to sedentary lifestyle, a classical example being the Pima Indians[3].

The obesity paradox was noted in several studies when it was found that people with lesser degrees of adiposity (overweight and mild obesity) survived better during several critical illnesses and chronic debilitating states. It was believed that the extra amount of fat somehow gives considerable advantage during the periods of prolonged hardship. Fat cells are now viewed as a large active endocrine organ. Recent findings also suggest that during insulin-resistant states, an altered metabolic state promotes growth and produces more nucleic acid elements and nicotinamide adenine dinucleotide phosphate (NADPH) for maintaining repair and antioxidant function, respectively, all at a cost of hyperglycemia.

Principles of evolution dictate that organisms always try to improve their fitness (*ability of an individual or population to both survive and reproduce in a particular environment*), which enables them to propagate their genes into the future. When resources become plentiful, the strategy of producing fewer offspring in

order to improve fitness would be appropriate for an organism with a long life history like humans, especially when mortality rates are low. As obesity, a marker of rich resources, reduces fertility, changes at the molecular and individual level may also be operating in parallel with this principle.

This article examines the increasing trend of obesity in humans from a fresh evolutionary point of view in light of several new pieces of evidence available from various fields of science.

THE HYPOTHESIS Obesity, a marker of a resource-rich state, affects the two fundamental processes of human life history – survival and reproduction. Therefore, based on the observations and evidence as discussed further, it is proposed that *overweight* and *mild obesity* may be an evolutionary norm, increasing the biological fitness of humans by simultaneously increasing survival probability and reducing fertility in a resource-rich environment, thereby promoting more investment in fewer offspring.

SUPPORTING ARGUMENTS Obesity among animals is a rarity and of all the mammals, humans are one of the fattest[4]. Fat content of mammals varies from 1% to 45% and some species are even able to survive and reproduce with <1% total fat[5]. Few species deposit fat indefinitely, even if palatable foods are freely available, and Pond[5] has suggested that human obesity cannot be explained entirely in terms of a common famine-tackling adaptation alone.

PATHOPHYSIOLOGY AND CURRENT THEORIES OF OBESITY Like several other conditions where the causative mechanism cannot be pinpointed, obesity is explained by the combined influence of genes and environmental factors. Sedentary lifestyle and surplus food intake are believed to be the main environmental risk factors responsible for the obesity epidemic, making it a common 'life style disease'[6]. The main evolutionary explanation for the phenomenon of obesity is the thrifty phenotype hypothesis (TPH), which postulates that fetal programming occurring during an adverse condition like low birth weight makes the person go for a thrifty state where more food is ingested throughout life, leading to obesity[7]. Though initially received with great scepticism, the theory later found support in several human and animal studies, and its non-genetic aspects were most convincing when it was found that only two of the 45 known type 2 diabetes-prone genes are associated with low birth weight[8].

The TGH says the famine-selected genes over years are likely responsible for efficient fat deposition. Proponents of the TGH note that our species has probably undergone intense selection for thriftiness within the past 5 million years, in relation to seasonality, enlargement of the brain and alteration of its own environment[9]. Mathematical models and other evolutionary principles are against this hypothesis and, hence, it is losing appeal as there could be hundreds or thousands of other candidate genes that are also responsible[10]. The 'carnivore connection' theory postulates that a change in diet from the high protein diet of ancestral conditions to a high carbohydrate diet, especially with a high glycemic index, is important in the evolution of insulin resistance (IR)[11].

All of these hypotheses primarily address the phenomenon of obesity as *maladaptive* with accompanying illnesses increasing the mortality and morbidity of humans.

Adipocytes, the storage cells of fat, were believed to function merely as depot sites of extra fuel, but recent findings prove that adipocytes constitute a highly active endocrine organ, secretions of which profoundly affect a variety of metabolic reactions involving glucose regulation, hypothalamic function, blood pressure, immunity and even reproduction[12]. Adipocytes secrete a number of inflammatory mediators like interleukins and transforming growth factor α, which play a major role in producing IR. Adipocytes also secrete hormones like leptin[13] and adeponectin[14], which regulate satiety and energy expenditure and thus body composure of total fat. Adipocytes were even proposed as a newer member of the immune system, considering their secretions of inflammatory mediators, expression of C1qTNF-related protein super family (part of innate immune system) and granulocyte-regulating function of leptin, adiponectin and resistin[15].

CLINICAL MEASURES OF OBESITY In humans, clinical measures of obesity include body mass index (BMI), waist-to-hip ratio (WHR) and skinfold thickness. BMI, defined as weight in kilograms divided by the square of height in meters, is widely used in clinical practice as it is very easy to measure. In general, normal BMI is taken as 18.5-24.9 kg/m^2, above which comes overweight and obesity[16] (Table 1).

The accumulated evidence suggests that all of the main complications of obesity – osteoarthritis, various cancers, gall bladder illnesses, sleep apnea[16], diabetes, hypertension and dyslipidemia – are more pronounced in *higher* grades of obesity,

Table 1 | Classification of overweight and obesity by BMI

Category	BMI (kg/m2)
Underweight	<18.5
Normal	18.5-24.9
Overweight	25-29.9
Obesity	≥30
Grade 1	30-34.9
Grade 2	35-39.9
Grade 3	≥40

and survival benefits (obesity paradox) lie within the overweight and grade 1 obesity groups.

NEW EVOLUTIONARY APPROACH TO OBESITY In evolutionary science, the life history theory (LHT) interprets events like growth, survival and reproductive success, the key factors deciding the fitness of organisms[17]. The LHT incorporates trade-offs and energy allocation by the organism that occur during attempts of acquiring growth, taking measures to reduce mortality and making decisions on current versus future reproduction. Time and calories (collectively called 'investment') in the juvenile period are distributed towards growth, learning and reducing instantaneous mortality rate,

which affects future energy production and reproduction. Humans, with a long life history, postpone reproduction for a considerable period of time for education and acquiring resources that give an advantage in future reproductive efforts – an initial investment called embodied capital.

The accumulated mass of adipocytes – a highly active endocrine organ – may be considered a form of embodied capital, which, as per recent evidence, might give an advantage during natural selection at a cost of hyperglycemia, as discussed further.

Obesity reduces relative risk of death (obesity paradox) Intuitively, conditions that are the risk factors for diabetes, hypertension and dyslipidemia should increase the relative risk of death. Earlier studies on obese individuals were consistent with this and showed increased mortality, especially in *higher* grades of obesity[18]. The obesity paradox was noted when several observational studies found that although obese individuals are at higher risk of developing cardiovascular diseases, they survive better during periods of acute and chronic illnesses compared with non-obese individuals. In patients admitted to surgical intensive care units with a critical illness, it was found that the survival rate was better if they were obese[19]. With diseases like chronic renal failure[20] and chronic obstructive airway disease[21], where the periods of hardship can run over years, obese individuals survive better. In an observational study of 108,927 individuals with acute heart failure, the outcome was better among overweight and obese people compared with non-obese people[22]. In chronic heart failure, higher BMI was associated with lower risk of death[23]. Better survival was also observed in patients suffering from coronary heart disease[24], as well as during surgery for this condition[25].

Recent evidence shows that even for people without any prior illness, mild obesity (grade 1) may be beneficial. In a study of 21,925 men, aged 30-83 years, obesity did not appear to increase all-cause mortality risk, provided that cardiorespiratory fitness (CRF) was good[26]. A large meta-analysis of 97 studies with total sample size of more than 2.88 million individuals found that overweight and grade 1 obesity was associated with lower all-cause mortality – interestingly, the latter gave the best advantage, while other higher grades of obesity were harmful[27].

Studies that specifically addressed the mystery of the obesity paradox found that in ischemic heart disease, lean body mass is also a predictor of mortality along with body fat in an inverse fashion[28]. Cardiorespiratory fitness was found to be another good prognostic predictor and attenuated the effect of obesity; however, among patients with low CRF, the obesity paradox was noted[29].

The precise explanation of this paradox is not yet known and major blame for this strange observation is made towards the various biases and overlooked factors of the studies, such as inadequacies of BMI as a measure of obesity, CRF of study participants, better medical attention enjoyed by obese individuals and even the protection from a fall by the cushioning effect of fat[30].

Molecular mechanisms showing advantages of insulin resistance Once insulin binds with its receptor at the cell membrane, the effects are mediated through two pathways. The phosphatidylinositol 3-kinase (PI 3-kinase) pathway decides the metabolic actions of insulin and the mitogen-activated protein kinase pathways (MAP kinase) through Grb2/Sos regulate the anabolic actions like cell growth. It has been observed that IR selectively involves these post receptor pathways, mainly affecting the former[31] and leaving the mitogenic actions like growth and repair intact. This will probably be applicable only during mild IR and hyperglycemia, as higher degrees of hyperglycemia and accompanying deposition of advanced glycation end products can severely compromise the cell function.

Inside the cell, glucose may be catabolized via the tricarboxylic acid (TCA) cycle into acetyl Co-A or may be channelled through the pentose phosphate pathway (PPP) generating ribose-5-phosphate, used for nucleotide synthesis, generating NADPH. NADPH maintains the redox potential of glutathione and plays an important role in the killing of pathogens by white blood corpuscles. In IR during stress situations like a critical illness, more glucose is shunted through the PPP, generating NADPH and nucleic acid elements, as demanded by the situation[32]. Less glucose goes directly through the TCA cycle and, instead, pyruvate generated from anaerobic glycolysis enters it, generating more TCA cycle intermediates that enter the pathways of gluconeogenesis, lipogenesis, purines and pyrimidines[33]. Thus, IR seen during stress helps the cell to combat oxidative stress and repair processes. It is worth noting that measures to control the accompanying hyperglycemia during a critical illness by giving insulin were associated with increased mortality[34].

There is evidence that IR accompanying obesity also promotes more glucose disposal through the PPP – activity in the PPP was stimulated more by serum from obese than from normal weight males[35]. Antioxidant function is vital when fighting pathogens and may be the reason for better survival and lesser degrees of immunological deterioration in overweight and obese patients with HIV compared with normal or lean patients[36].

The forkhead box 'O' (FOXO) family of forkhead transcription factors are members of forkhead proteins and their expression controls the genes regulating the cell cycle, reactive oxygen species (ROS) detoxification, apoptosis, glucose metabolism and probably lifespan[37,38]. In mammals, there are four groups of these factors – FOXO1, FOXO3, FOXO4 and FOXO6. Inhibitors of transcription factors of the FOXO class include signal transduction through the PI 3-kinase pathway[39]. During fasting states (e.g., in famine), when signalling through the PI 3-kinase pathway is low, these transcription factors are up-regulated and enhance cell survival by inducing cell-cycle arrest and quiescence[37]. FOXOs also induce antioxidant enzymes like catalase[40] and manganese superoxide dismutase (MnSOD)[38]. Considering the up-regulation of FOXOs produced by the reduced signalling through PI 3-kinase pathway seen in IR, it has been

suggested that IR may be an evolved adaptation to combat stress[41].

The intact somatic growth, shunting of glucose through PPP generating more NADPH and nucleic acid components, and increased expression of FOXOs – all seen as a result of IR – may well give obese individuals an evolutionary advantage in maintaining growth, combating stress and maintaining low mortality, probably within a narrow range of adiposity and blood sugar.

Obesity, fertility and resources at large scale from the LHT perspective The LHT observes that, as reproductive efforts are costly and can compromise growth and survival function, organisms regulate the number of offspring to sustain race depending upon environmental variables such as available resources and mortality rates. Species with a shorter lifespan tend to produce more offspring and those with a long lifespan, like humans, tend to produce fewer offspring. Such trade-offs are central to the principle of LHT and the resulting fitness is subjected to natural selection. 'Fitness' in evolutionary terms means the probabilistic function representing the ability of the race to sustain the copies of a gene in the long term and should not be confused with physical fitness[42]. Fitness can be calculated as the product of the survival probability of offspring to their number. Stable strategies that evolve improve the fitness with several trade-offs, a classical one being between quantity and quality.

In resource-rich environments, it is not necessary that humans produce more offspring to improve fitness. Parameters of fitness were better for parents who invest in fewer children, as observed among Shuar hunter-horticulturists of South America[43]. As the environmental conditions improve, instead of producing more offspring, organisms like humans prefer to invest more resources in fewer offspring[43]. In addition, primates and human societies with higher fertility rates have smaller offspring[44] and, hence, less probability of survival and ability to control resources. On the other hand, in adverse situations, the race is sustained with a higher rate of reproduction – the highest fertility rates per woman are seen in countries with the lower gross national income per capita and adverse environmental conditions like political instability[45].

Obesity reduces fertility It is a well-known fact that obesity decreases fertility in several ways, in both sexes. In males[46] as well as in females[47], the risk of infertility was positively correlated with BMI. In males, obese individuals showed lower sperm quality[48,49] and testosterone levels[50]. In one study, obese men reported fewer sexual partners and more erectile dysfunction[51].

It seems that females are more affected by obesity than males with respect to reproductive issues, which affect all stages of female reproduction, resulting in lower fertility. Obesity reduces fertility, spontaneous conception and chance of live birth – the last due to a higher risk of miscarriages, along with obesity-related complications of pregnancy[52]. Furthermore, obesity leads to anovulation, menstrual irregularities and less success infertility treatment[53]. Polycystic ovary syndrome (PCOS), one of the main causes of infertility in females, is characterized by multiple cysts in the ovaries, anovulation and hyperandrogenism, and 50% of women with PCOS are obese and show features of IR[54].

In contrast, optimal body weight enhances fertility in females in several ways. Non-obese females have optimal sex hormone profiles[55] and lower endocervical pH, the latter promoting sperm penetration[56]. They also have fewer irregular menstrual cycles[57] and even ovulate more frequently[58]. Finally, when it comes to appearance, women with optimal WHR are most attractive to other males[59,60].

Obesity increases with age and each reproductive effort In the USA, during 2009-10, the overall prevalence of obesity in the age groups 20-39, 40-59 and above 60 years were 32.6%, 36.6% and 39.7%, respectively, with a statistically significant increasing linear trend by age[61]. In 2010, the number of births per 1,000 women was 108.3, 96.5 and 45.9, respectively, in the age groups 25-29, 30-34 and 35-39 years[62]. A study among Iranian couples showed that reproduction appeared to be a risk factor for developing obesity as the number of offspring is positively associated with obesity in both men and women[63].

The best available explanation of the higher birth rates in the younger age groups would be the high fecundity of the younger age and the advantages of early reproduction. Although estimating how much obesity contributes to the decrement in fertility requires multilogistic regression analysis, it is worth noting that obesity – a condition that leads to reduced fertility – is at a minimum during the peak reproductive time and steadily increases as age and reproductive attempts increase. From an evolutionary point of view, this may be viewed as an attempt to simultaneously down-regulate fertility and improve survival probability by becoming obese – an LHT event enhancing biological fitness.

Obesity and lifespan Oxidative damage is one of the initial and accepted theories on the mechanism of aging[64]. Calorie restriction is the only modality that has been shown to increase lifespan in mice, primates and several other species. There is evidence that signalling through the insulin receptor pathway may play a role in regulating lifespan. Situations leading to reduced signalling, either directly or due to reduced levels of insulin, as in starvation, are correlated with increased lifespan, and are evolutionarily preserved in several species[65]. The mechanism operating may be the reduced activation of PI 3-kinase pathway during low calorie intake, which up-regulates the FOXOs, enabling better antioxidant capabilities and survival.

As IR produces less signalling through the PI 3-kinase pathway, it will be interesting to examine the data on the lifespan of obese or overweight individuals. Unfortunately, the reliability of such studies is plagued by methodological flaws and inconsistencies[66], and earlier studies have found an increase in all-cause mortality with increasing obesity[67].

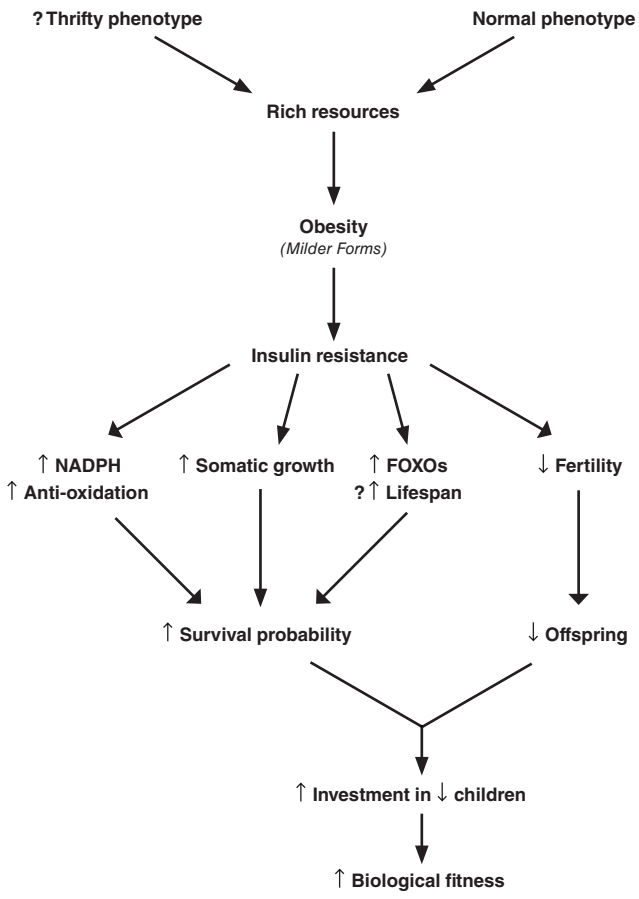

Figure 1 | 'Fitness First' hypothesis. Milder forms of obesity increasing survival probability and decreasing fertility in a resource-rich environment, promoting investment in fewer children, ultimately improving biological fitness.

*FOXO: f*orkhead box 'O'; *NADPH:* nicotinamide adenine dinucleotide phosphate.

In the Health and Retirement Survey (HRS) – a prospective longitudinal study to estimate the burden of mortality – the highest life expectancy at age 55 was found in overweight (BMI 25-29.9 kg/m^2), highly educated non-smokers[68].

In another observational study including 359,387 Europeans, *overall* BMI remained significantly associated with the risk of death when waist circumference or WHR were also taken into consideration[69]. The analysis was carried out according to the standard obesity grading, but a closer look shows that the relative risk of death was lowest in the group with BMI between 25 and 28 kg/m^2, and was more apparent in males. In particular, relative risk of death due to respiratory-related illnesses were lowest in this group and relative risk of death due to neoplasms was low or equal to the persons with normal BMI. Mild degrees of obesity even seemed to provide protection from current smoking as the relative risk of death among smokers was lowest in overweight group.

As prevalence of obesity among children is also increasing, by intuition the hypothesis should predict that it should be complementary to adult obesity. Available evidence suggests that the prevalence of obesity in childhood is half of that of adults and although childhood obesity is a risk factor for obesity in adulthood[70], it remains controversial as to whether it leads to increased mortality in later life[71,72]. However, the fact that some studies found it not predictive of adult mortality point towards the possibility that the mechanisms to improve survival – to live with extra fat – may be operating from childhood.

CONCLUSION The amount of fat that people carry is steadily increasing and has reached 'pandemic' scales. The adverse effects of obesity are more pronounced in higher grades of obesity and studies addressing the same show increased mortality when the *entire* spectrum of obesity is taken into account. Since the accumulating evidence favors the survival advantages of lesser degrees of adiposity and its detrimental effect on fertility, the bulging waistline of humans may be explained by this 'fitness first' hypothesis, which states that adiposity – overweight and grade 1 obesity – may be an evolutionary adaptation, ultimately aiming for better biological fitness (Fig. 1). The decreased fertility associated with adiposity may not be acceptable to an *individual*, but considering the true definition of fitness – which is a property of the *race* rather than its individual member – the ability of the gene pool to persevere for a very long time with appropriate trade-offs remains the prime consideration.

When mathematical models were taken into account, among the adult US population, one study found that life expectancy at birth would be higher by 0.21 to 0.93 year if obesity did not exist[73]. As the evidence for the influence of mild obesity on lifespan remains conflicting, from an evolutionary point of view it may be argued that a small loss of lifespan would be negligible and is unlikely to influence the life history variables, as shortening would most likely occur during advanced age where reproductive prospects are very low. Moreover, at these age groups, any support for the progeny in the form of grandparenting or reciprocal altruism becomes negligible due to the small time frame. Also, if mild degrees of obesity have given advantage during the periods of reproduction, parenting and early grandparenting, then, from an evolutionary point of view, diseases and mortality due to later complications may be immaterial.

A mathematical representation of the hypothesis build on already existing models on quantity–quality trade off and energy allocation strategies to life history variables is included in the supplementary material of this manuscript. The range of adiposity between which the metabolic alterations give the best result needs to be further modelled mathematically,

considering dynamic interactions of variables like the age of onset of obesity, reproductive behaviour, varying body weight, survival benefits, mortality, fertility rates and parameters of fitness, given the fact that higher degrees of obesity can be deleterious to survival. Larger observational studies specifically looking at the benefits of obesity will be needed to generate high-quality evidence before any clinical recommendations can be made and, as such, this article does not recommend that readers maintain a bulging waistline. In addition, it needs to be verified whether the sedentary lifestyle – a risk factor for obesity – is perceived by the body as a signal implying less struggle for food, allowing the organism to concentrate more on the reproductive process.

SUPPLEMENTAL

A mathematical representation for 'Fitness First' hypothesis We would like to propose a mathematical representation for the 'Fitness First' hypothesis, which states that milder forms of obesity may be a good evolutionary strategy for promoting more investment in fewer children by simultaneously decreasing fertility and increasing survival probability[1] — the latter observed phenomenon called the obesity paradox. Here we select and combine already existing models on the offspring quantity-quality trade-off in humans and energy investment decisions for growth, development and mortality reduction, that decide fitness and attempt to show that accumulated fat may be beneficial in improving biological fitness.

Model

a) Fertility reduction and biological fitness in humans There is ample evidence to show that humans tend to produce fewer children when resources become plenty. Also, this quantity-quality trade-off is supported by several bio-economical mathematical models. Kaplan suggested that biological fitness can be represented by the product of the number of offspring, their survival probability and their income, and higher income parents invest more per child than their financially poorer counterparts[2]. Becker and Lewis[3] suggested a model proving the benefits of the quantity-quality trade off from economical point of view. This bio-economical model was refined later with a stronger proof — the parental decisions on the quantity of children and quality, q, can be shown in such a way that the percentage decrease in quantity n is larger than that in quality q[4].

$$\text{1. i.e } \frac{\Delta n}{n} > \frac{\Delta q}{q}$$

b) Obesity increasing parental investment For this, we select a model based on life history theory by Kaplan et al. for natural selection on age at first reproduction and investments in mortality reduction[5]. The original model considers a juvenile phase lasting for a time period, t, during which energy is invested for two purposes — as embodied capital for growth and learning, which determines future energy production, P, and for reducing mortality rate, μ. During the reproductive period, growth stops and all further energy is allocated to reproduction. The energy production grows at some constant rate, g, due to the effects of initial capital. If P_a is the energy production of an adult at the end of the juvenile period, adult production of energy P_x, at time, x, after the juvenile period, t, would be:

$$P_x = P_a e^{g(x-t)}$$

If λ is the amount spent for mortality (μ) reduction and 1 - λ is the amount spent for growth and development, the adult production of energy channeled for reproduction at age, x, would be:

$$\text{2. } P_{r,x} = (1-\lambda) P_a e^{g(x-t)}$$

Now assume that milder forms of obesity play some role in the decisions on energy allocation. Let β be the amount of energy spent on becoming obese and maintaining this state. This energy is spent for growth and development but paradoxically (obesity paradox) it reduces instantaneous mortality rate[6] μ; i.e.,

$$\frac{d\beta}{d\mu} > 0;\ m < \beta < n$$

where m and n define the energy limits which optimally decide the level of obesity – to be in the overweight and grade 1 obesity range – as higher grades of obesity may be deleterious.

As obesity reduces instantaneous mortality rate, smaller amounts of energy, λ, are needed for investing in mortality reduction. Hence from equation (2), the new production of energy, $P'_{r,x}$, available for reproduction at age x influenced by the obesity factor will be:

$$\text{3. } P'_{r,x} > P_{r,x}$$

suggesting that the adult production of energy reserved for reproduction or parental investment is better if milder forms of obesity exist, reducing instantaneous mortality rate.

The equations (1) and (3) can be taken to represent the final two limbs of the hypothesis, and, when combined, represent improved biological fitness.

The adverse effects of obesity are more pronounced in higher grades of obesity and the milder forms may improve the biological fitness of the species. The above mentioned representation is simple, built on previous models for fitness and may be considered to support the same. More complex models are required to find the exact interactions between the varying levels of fatness on fitness.**H**

CONFLICTS OF INTEREST Authors declare no conflict of interest.

ABOUT THE AUTHORS Dr. Rakesh is a specialist in Internal Medicine, working at the Govt. Medical College, Thrissur (a tertiary level teaching institute), Kerala, India, as Assistant Professor in Medicine. His other areas of interest include evolution, evolutionary medicine, anthropology and mathematics. Mr. Syam T P is a research scholar from Indian Institute of Technology, Madras, Chennai, India. His areas of interest includes control theory, mathematical modelling of linear systems and evolutionary biology.

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