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The behaviour of a neutral model of weight regulated only by body mass

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Abstract

While there are many mechanisms that may be involved in the regulation of body mass in humans and other animals, it is not so clear how much regulation is needed beyond the negative feedback effect of body mass itself. Here we model weight changes as a stochastic process, and show that it behaves approximately as an autoregressive process. Using published estimates of the energy cost of weight gain, the effect of weight on resting metabolic rate and the daily variation in intake and activity, we show that fluctuations in weight will be small. The effect of excess intake is also examined, and the assumptions and limitations of the model are discussed.

1. Introduction.

There can be little doubt that humans and other animals have mechanisms to regulate their body weight. It can be observed that most people maintain a fairly steady weight over many years and decades, while a small imbalance between energy intake and expenditure will soon accumulate to produce weight change. There has been much investigation into what these regulation mechanisms are, and it seems fair to say that we are still some way from understanding them, and the relative importance of the different mechanisms involved.

What is also unclear is how strong the regulation is in the short to medium term. It has been pointed out (Speakman *et al*, 2002) that body mass itself can play a role in weight regulation. Our purpose in this paper is to examine the patterns of body mass fluctuations when this is the only source of regulation. Although not asserting that such a mechanism is the only one, or the main one, it should be of use in evaluating their effect to see how body weight behaves, compared to what would occur if *no* sophisticated regulation were taking place.

We can assert that some form of regulation must take place. Variations in energy intake and expenditure will produce fluctuations in weight. If these were equally likely to be positive or negative, regardless of current weight, then weight would follow a random walk without limit and with infinite variance, even though intake and expenditure remain stable and balanced. Simple random walks like this have no tendency to return to their starting, or any other, value. This is a property of random walks (Cox & Miller, 1977, §2). Regulation of weight will however result from the fact that energy expenditure is a function of weight. What we will study is how much regulatory effect results from assuming only this, and stable intake.

Our intention is to present a neutral model of weight regulation by body mass, and to describe its behaviour. We do not assume or assert that this the principal way that weight is regulated in humans or any other animals, but wish to determine how much of an effect it could have. The only assumptions made are that energy expenditure varies with body mass. The cases where energy intake remains stable independent of small weight fluctuations, and where it is proportional to weight are both examined.

2. Methods.

We first introduce some notation. We will index the steps of time which we model by i . These may be days (and we will assume this) but could be weeks or even months. We will denote body weight by W , energy intake and expenditure by I and E , resting energy expenditure by R , physical activity level by P and the energy cost of weight gain and loss by C . We assume the costs of loss and gain to be the same. This could readily be changed, just making the model a little more complex but without changing its behaviour. This is discussed later. We have that $E = PR$, as this is how P is defined. Weight will be recorded in kg and energy in MJ, and the units of the other quantities follow from this.

Resting energy expenditure depends on weight, and there are a variety of formulas which attempt to model this association. A majority are linear, and so we will assume that

$$R = \alpha + \beta W$$

Published formulas often include a dependence on height also, but this will not change longitudinally in an adult, and is included in the constant α . It will be convenient to write the linear formula in an alternative form $R = \beta(W + W_C)$, where the constant term is expressed via a weight W_C . It may well be that the association between R and W is not linear, as it has been pointed out that weight gain tends to be proportionally more of fat than lean body mass, and that the former is less metabolically active. This would lead to a reduction in the rate of increase in R at higher weights. However, we will retain the linearity assumption for simplicity. The model could easily be adapted if necessary. If W is recorded in kg, and R in MJ/d, then Schofield (1985) estimates that β is 15.1 for men and 14.8 for women aged 17-29, and less for those who are 30 or over

Energy expenditure is $P\beta(W + W_C)$, and if mean physical activity is μ_p , then mean energy expenditure will be $\mu_p\beta(W + W_C)$.

The model is developed specifically for humans. Animal weight regulation will be subject to many features which do not occur in humans, such as the seasonal availability of food and the influence of weight on ability to procure it. We will make the assumption that a person eats as much or as little as they wish, and that the amount thereby eaten each day fluctuates at random about an overall mean which is stable. What autocorrelations and compensations there are between consecutive days have been shown to be weak (de Castro, 1999).

Then

$$I \sim N(\mu_I, \sigma_I^2)$$

so that intake is Normally distributed with a mean μ_I and variance σ_I^2 . It may be that the true distribution is not Normal, but that will not affect the nature of the model, and it can be expected by the Central Limit Theorem that over time the behaviour of a model with some other distribution would be the same.

Regarding energy expenditure, we will suppose that

$$E \sim N(\mu_p\beta(W + W_C), \sigma_E^2)$$

We also assume independence of I and E . (This is discussed later.)

We see that for a specified mean intake μ_I , the weight given by

$$W = \frac{\mu_I}{\mu_p\beta} - W_C$$

is one at which mean intake and expenditure balance, and so is one to which body weight will tend to return. This follows since if W falls below this, E will decline, the expectation of $I - E$ will be positive and so weight will drift upwards, towards the value $\mu_I / \mu_p\beta - W_C$. Similarly, if weight increases, the same mechanism will tend to reduce

it again. This therefore fulfils the role of what has been called (not without some controversy) a “weight set point” (Cabanac, 1991). We shall denote this as W_0 . Note that it is not a physiological characteristic which determines intake: it is a mathematical consequence of a stable intake. The long term behaviour then is that as long as μ_I and μ_P do not change, weight will tend to fluctuate about the set point. In the next two sections we ask two questions:

- (i) what do the fluctuations about W_0 look like? and
- (ii) what does the weight pattern look like if μ_I or μ_P does change?

Fluctuations in weight

If W_i is the body weight on day i , the weight the next day will be

$$W_{i+1} = W_i + C(I_i - E_i) \quad (1)$$

This leads to a stochastic process for W_i . It will be convenient to write $I = \mu_I + \varepsilon_I$, where $\varepsilon_I \sim N(0, \sigma_I^2)$. Likewise, $E = \mu_P \beta(W + W_C) + \varepsilon_E$. Then (1) becomes

$$W_{i+1} = W_i + C(\mu_I + \varepsilon_I - \mu_P \beta(W + W_C) - \varepsilon_E),$$

which with some rearrangement leads to

$$W_{i+1} = W_i - C\mu_P \beta(W_i - W_0) + C(\varepsilon_I - \varepsilon_P)$$

and can also be written in terms of deviations from W_0 :

$$(W_{i+1} - W_0) = (1 - C\mu_P \beta)(W_i - W_0) + C(\varepsilon_I - \varepsilon_P) \quad (2)$$

This is an autoregressive process of order 1 (Cox & Miller, 1977, §7.2). The first term on the right contains the tendency for weight to move back towards W_0 , while the second pair of terms are the purely stochastic part. From equation (2), it is possible to derive the long term behaviour of W :

- (i)** It will have a Normal distribution, as it is the sum of a series of terms each with a Normal distribution.
- (ii)** It will have a mean of W_0 . This follows from taking the expectation of both sides of equation (2), and since $E(\varepsilon_I - \varepsilon_P) = 0$, we must have that $E(W_i - W_0) = 0$.
- (iii)** The variance of W_i , which is also the variance of $W_i - W_0$, follows from equating the variance of both sides of (2). It leads to the standard result for the variance of an order 1 autoregressive process, and

$$\text{Var}(W) = \frac{C^2(\sigma_I^2 + \sigma_E^2)}{1 - (1 - C\mu_P\beta)^2}.$$

This can be expressed in terms of the coefficients of variation (CV). If we write these as $\gamma_I = \sigma_I / \mu_I$ and $\gamma_E = \sigma_E / \mu_P\beta(W + W_C)$, then we can rearrange the above expression for $\text{Var}(W)$ to give

$$\frac{\text{Var}(W)}{W_0^2} = \frac{Cb}{2 - Cb} \left(\frac{W_0 + W_C}{W_0} \right)^2 (\gamma_I^2 + \gamma_E^2). \quad (3)$$

Alternatively, we could make the approximation of replacing γ_E with γ_P , the coefficient of variation of P. Thus the CV of weight is proportional (after all are squared) to the sum of the CVs of intake and activity.

(iv) The autocorrelation ρ between weights d days apart is given by

$$\rho(d) = (1 - C\mu_P\beta)^d.$$

Change in intake or activity.

In our simplified model of weight regulation, weight will fluctuate only a little around a set point which is determined by the mean intake and activity, $W_0 = \mu_I / \mu_P\beta - W_C$. While an individual may be reasonably weight stable in the short or medium term, it is well known that weight can change in the long term. One of the main ways in which this happens is the longitudinal trend towards increased weight with age.

Weight will change if any one of the three parameters μ_I , μ_P or β change, and no other one changes to compensate for it. So if mean intake increases by 10%, with no change in activity or in β , then the weight set point W_0 will increase. This limits the effect of an excess of intake over expenditure: it will not lead to an unlimited increase in weight, since expenditure will increase to match it.

A change in activity level μ_P will have an effect which is reciprocal to but otherwise exactly the same as an intake increase. We may be less inclined to think that β might change, although some of the equations for predicting RMR (the Schofield equations for example) are age dependent, with β decreasing with increasing age. It is not unreasonable to suggest that this might explain a significant part of the age related increase in weight, since a change in β , with no change in intake or activity, will increase W_0 .

It is possible to describe how weight will change following a substantial change in any of the three parameters, such as might occur if an individual makes a diet or lifestyle

change in order to attempt weight reduction. If current weight is substantially different from W_0 , more than the fluctuations we have already described, then each step change from i to $i+1$ will be small, since $C\mu_p\beta$ is small. From (2), the expectation of weight after d days will be given by

$$E(W_{i+d} - W_0) = (1 - C\mu_p\beta)^d (W_i - W_0) \quad (4)$$

Weight will asymptotically approach W_0 . In reality, the fluctuations which we have omitted will be present, and will ensure that the weight will reach W_0 at some point, and thereafter fluctuate about it.

Energy imbalance.

We have considered what happens when energy intake remains stable, and shown that body mass will adjust so that it is in balance with it on average, and weight will then fluctuate about the set point defined by this intake. In this section we look at what happens if intake exceeds requirements on average. The previous section shows that if this excess intake remains stable, it simply defines a new set point, which body mass will tend towards. Another model of excess intake is to suppose that on average it exceeds current requirements by a small proportion, and that this excess therefore changes in response to weight changes. We will denote this proportion by u , and suppose it to be a few percent.

Biologically, we might suppose various reasons why such a tendency to excess might exist. The cause might be external, through what is referred to as the obesogenic environment (Lake *et al*, 2010), where highly palatable energy-dense food items are readily and inexpensively available, and heavily advertised. Or the cause might be internal, where what is referred to as the “thrifty phenotype” (Hales and Barker, 2001) inclines many individuals towards gaining weight when food is easily available, as a buffer against potential future food shortages. Whatever the cause, the effect will be the same: average intake will exceed average requirements, and weight will tend to be gained.

We have as before that $W_{i+1} = W_i + C(I_i - E_i)$, but now suppose that

$$\mu_I = (1+u)\mu_p\beta(W+W_C) \quad (5)$$

so that

$$W_{i+1} = W_i + uC\mu_p\beta(W_i + W_C) + C(\varepsilon_I - \varepsilon_E),$$

which may be rearranged

$$W_{i+1} = (1 + uC\mu_p\beta)W_i + uC\mu_p\beta W_C + C(\varepsilon_I - \varepsilon_E) \quad (6)$$

This stochastic process has the form that the next weight is an affine function of the previous weight, where the coefficient is slightly greater than 1, plus a random term.

The process is not stationary, and we no longer have a set point, or a stable long term average. The expectation of the weight will increase with time, and by recursive application of (6), setting a starting weight of W_s , and using that the sum of a geometric series is given by $\sum_{k=0}^{n-1} a^k = \frac{a^n - 1}{a - 1}$, we obtain

$$E(W_{s+d}) = (1 + uC\mu_p\beta)^d W_s + \frac{(1 + uC\mu_p\beta)^d - 1}{uC\mu_p\beta} uC\mu_p\beta W_C$$

and if we note that since $uC\mu_p\beta$ will be small, and using the approximation that $(1 + a)^n = 1 + na$ for small a , we have the simpler form

$$E(W_{s+d}) = W_s + duC\mu_p\beta(W_s + W_C)$$

which is linear in d , the number of days from the arbitrary starting point. Weight will increase at a rate which is proportional to the product of the duration of excess, the amount of excess, and the weight that this amount of excess is converted to. A similar calculation leads to the variance of the stochastic process

$$\text{Var}(W_{s+d}) = dC^2(\sigma_I^2 + \sigma_E^2).$$

This is the variance about the previous expression for the expectation, rather than about a constant fixed point. As with the mean, the variance is not constant either, and also increases linearly with time. Weight at later time points then becomes increasingly unpredictable. This is the case even when $u = 0$, as in this model intake will track weight. Since the variance cannot in reality increase without limit, we can conclude that some additional regulatory mechanism must exist. Many extensions to the above model could be proposed for this. One fairly natural one would be to require mean intake to remain above some lower limit, even if weight fluctuations caused W to dip to a point which would take it below this limit. The model could be modified in this and many other ways. Analytical results for the expectation and variance would no longer be readily obtainable, but the model behaviour could be explored by simulation.

3. Results.

The main outcome of our modelling is an estimate of how variable weight will be when it is regulated only by the effect of body mass. This is given by equation (3).

Its value will depend on the coefficients of variation of intake, γ_I , and activity level, γ_P ,

and the terms $C\mu_p\beta$ and $\left(\frac{W_0 + W_C}{W_0}\right)$. We take these in turn.

Variation in intake (γ_I).

This is not a physiological constant which will be the same for everyone. Some will be more regular in their intake and some more variable. In addition to this, within individual variation will contribute to apparent between individual variation in any short term study. This was confirmed by Tarasuk & Beaton (1992), who found the coefficient of variation to range from 19% to 45%, with a mean of 27%. Bingham (1987) examined 17 separate studies and obtained a mean value of 23%. We thus propose to use 25% as a typical value for intake variation.

Variation in activity (γ_P).

The effect of daily variation in activity level is similar to that of intake, although it appears to be less often studied. Goldberg *et al* (1991) use a value of 12.5%, about half that of intake variation.

The term $C\mu_P\beta$.

This term is small. It is the ratio of the daily energy cost of maintaining 1 kg of body mass, $\mu_P\beta$, to the energy cost of forming it in the first place, which is C^{-1} . The denominator of the multiplier in equation (3) is thus approximately 2. We need estimates for the three factors C , μ_P and β . Since our claim will be that the variability of weight regulated only by body mass is lower than might be expected, we shall prefer to use the higher values among those that are available.

C is the energy cost of weight gain, expressed in reciprocal form as the amount of weight that is gained or lost for a positive or negative energy balance of 1 MJ. One source of estimates for this is FAO (1991), who quote a range of values. In adults recovering from anorexia nervosa, the estimate is 0.037 kg/MJ, and for intentional overfeeding of adults it is 0.029 kg/MJ. Other estimates can be found. For example, Vortuba *et al* (2002) quote 0.018 kg/MJ in obese women and Forbes *et al* (1986) found 0.030 kg/MJ in normal weight adults. The energy used in weight loss may differ from weight gain (and is discussed further below). Hall (2008) considers a value of $C = 0.031$ kg/MJ and discusses the way that this is likely to differ depending on the body composition changes that different individuals losing weight would experience.

μ_P is the mean activity level. A value between 1.5 and 2 is most often assumed for this, although it can certainly be higher in those who participate a lot in sport or have other active occupations. Goldberg *et al* (1991) compare various studies and find a range of 1.46 to 1.80. We shall use the upper limit of this range, 1.8, in our calculations.

β is the linear dependence of RMR to body weight, and we use units of MJ/kg. The Schofield equations give a range of values for this coefficient depending on age and gender. The highest is 0.0634 MJ/kg for men aged 18-29. Many other prediction equations have been developed, and there is a considerable literature discussing and comparing them. Most prediction equations include a term for height, in recognition

that at a fixed weight, height is associated with body composition, and so we cannot use them directly. Height will not change during weight loss or gain in adults, but it means that β depends on it.

The term $(W_0 + W_C) / W_0$ depends on W_0 and W_C . It is natural to use the value of W_C that is associated with Schofield equation from which β is derived. In that case, we have that the constant is 2.9MJ and thus $W_C = 2.9 / 0.0634 = 45.8\text{kg}$. If we take a typical adult male weight to be 75kg, then $(W_0 + W_C) / W_0$ is 1.61.

Estimation of variability.

If we combine the values we have selected above, so that $\gamma_I = 0.25$, $\gamma_P = 0.125$, $C = 0.037 \text{ kg/MJ}$, $\mu_p = 1.8$, $\beta = 0.0634 \text{ MJ/kg}$, $W_0 = 75\text{kg}$ and $W_C = 45.8\text{kg}$ then from equation (3) we find that the coefficient of variation of weight is 2.1%. For a typical adult weighing 60-90kg, this is a standard deviation of only 1.6 kg.

It is more difficult to visualise the appearance of the autocorrelation, which for the above parameters is 0.996. It can be illustrated by some simulations of the model, which are shown in Fig 1, which shows changes in weight over a hypothetical five year period, where the set point W_0 is 75kg. The high autocorrelation means that the fluctuations have the appearance of transient trends and periods of slightly higher and lower weight. However, these all result from completely independent day-to-day fluctuations in intake and in activity, about a mean which does not change. Any interpretation of the temporary upward and downward trends in terms of spells of weight gain or loss caused by action or failure of regulatory mechanisms is unnecessary.

Weight changes.

We have seen in (4) how weight will change if there is a large mismatch between intake and starting weight. The set point weight is approached exponentially, at a rate determined by $1 - C\mu_p\beta$. For the values chosen above, this has a value of 0.0042. Thus the weight difference from the set point will decrease with a ‘half-life’ of $\ln 2 / 0.0042 = 165$ days, and 90% of the weight change will have been achieved after $\ln 10 / 0.0042 = 548$ days. These calculations do not depend on the starting or final set point weight, although in practice (4) is only relevant when the difference between these two are far apart. Fig 2 illustrates the behaviour of equation (4) and also shows the similar behaviour of weights which are subject to random fluctuations as in equation (1).

Excess intake.

The main result of the model suggested in (5) and (6) is that a small percentage excess of mean intake over requirements will lead to an increase in expected weight, but that this increase will not be steady, but increasingly unpredictable. Fig 3 shows some realisations of the model with values of model parameters as described above. The erratic behaviour relative to a smooth steady increase is clear. Thus although a persistent and steady small tendency towards excess will produce a longitudinal increase in weight in an individual, this increase is not at all expected to be smooth and

monotonic. Erratic fluctuations will be superimposed on top of it. While in many individuals such fluctuations would be interpreted as the result of conscious attempts to intervene in the pattern of weight change, the model we propose demonstrates that other explanations are possible, as the simple interaction between body mass and random variation in intake and expenditure can also produce such substantial fluctuations.

4. Discussion.

Regular long-term weight data in healthy humans is difficult to find. We can speculate that this is due to the difficulty of finding volunteers willing to be weighed scientifically at least once a week for a number of years, the sort of data that would be needed to compare with what our model predicts. Certainly some people do weigh themselves regularly, and quite likely some will keep a written record, but this does not find its way into the scientific literature. We might also expect that those who monitor their own weight regularly mostly do so as part of an effort to reduce it or at least to keep it from increasing, and so are likely to be using conscious behavioural mechanisms to regulate their weight. This is something explicitly excluded from our assumptions.

There have been studies which have looked at longer term weight variation in humans, generally obtained at intervals of at least a year and usually longer. The main interest here tends to be in changes that occur over periods of decades, particularly in whether or not there have been increases. The pattern of random changes, which we have modelled, is not addressed. Some work which does give some indications of this is that of Lee et al (2001), who in addition to examining 10 year BMI trends as regression slopes report that the variation around the subject specific slopes of 2%. Assuming height doesn't vary, the value for body weight will be the same. Yatsuya et al (2003) estimate fluctuations in the same way, and report a mean of 1.22kg (mean subject weight not reported). These estimates are encouragingly similar in magnitude to those obtained from our modelling.

We have described a model for weight regulation which is based only on the body mass and a stable average energy intake. It is not our intention to assert that this realistically models weight regulation in humans. What we wish to offer is a reference against which the effects of more sophisticated mechanisms can be compared. What we have shown is that the effect of body mass alone can keep weight to within a few percentage points of an overall set point which depends on mean energy intake and activity levels.

Our description of the effects of body mass, weight change, intake and activity makes several assumptions which are simpler than the reality. We need to consider how the model would be affected were these issues accounted for.

Linearity of weight / RMR association

We have assumed that RMR is linearly dependent on weight. There are certainly reasons to suppose this false in a population, but we are only concerned longitudinally within an individual. The linearity is likely not to be accurate for larger weight changes, due to differing proportions of lean and fat mass, with the former being more

metabolically active. However, linearity will be a good approximation for weight changes of a few kg, and we have seen that the effect of regulation by body mass will keep changes within such a range.

Energy costs of weight gain and loss

We have supposed that the energy costs of weight gain and loss to be the same, i.e. that dietary energy is stored and recovered from body mass with 100% efficiency. Realistically, more energy will be needed to form a kg of lean or fat tissue than needs to be expended to lose it. This will not greatly affect the patterns of weight change. If daily intake varies little, then not much body mass will be created or lost. If the intake variation is greater, then the body mass fluctuations thereby induced will take place with less than 100% efficient use of energy, and can be seen as reducing the weight set point.

Energy is not just stored in lean and fat tissue, as there is shorter term storage in the body's glucose and glycogen reserves, in the blood and liver. These also have a physical weight, but the corresponding energy cost factor (the equivalent of C in our model) may be different. A more complex model with energy stored in different compartments (and we could add the digestive system for example) could be envisaged, but the overall behaviour would be similar.

Growth

The model was developed for adults who are no longer growing. Clearly things must be different in children and adolescents, where the long term weight trend is increasing. In the shorter term, the same weight fluctuations must occur, although scaled down proportionally as mean energy intake and expenditure in absolute terms are lower. Patterns such as those in Fig 3 would be expected. We might speculate that in very young children and babies, where intake and activity are determined mainly by parents and carers, that day to day variation is less than in adults, and fluctuations will be correspondingly reduced. We also recognise that for children, who must increase in weight, it is vitally important that mean intake exceeds expenditure. It does not need to do so every day or week, but some mechanism to ensure that it does at a time scale of months or years must exist. This mechanism must also respond to the gradual increase in expenditure as weight increases during growth.

Body water content.

Weight fluctuations can also arise from the effect of variations in body water, which is increased by intake and reduced by perspiration, urination etc. However, there is no long term water storage, and so these fluctuations will not have any cumulative effect.

Timescale for energy to weight conversion

The model presented in eqn (1) is based on the day as the unit of time. It is assumed that weight (as it affects E) remains constant for that day and any excess or deficit is then completely converted to a weight change overnight. This is a simplification, and although the day seems the most natural unit for the modelling, it is not the only possibility. However, a different choice would not substantially affect the conclusions. The variance derived in eqn (3) would change little. If we chose the week as the unit of time, for example, the term $C\mu_p\beta$ would increase by a factor of 7, since β would

change, but the term $(\gamma_I^2 + \gamma_P^2)$ would decrease by a factor of 7. The only change would be in the divisor $2 - C\mu_p\beta$, which would be minor since $C\mu_p\beta$ is small.

Independence of daily intake and activity.

We assumed intake and activity to be independent. There is little evidence that any substantial association exists in sedentary individuals, the majority in many populations. We might imagine an association to exist in those with higher activity. The effect of a positive association between intake and activity will be to reduce the variance of the fluctuations in weight. This follows since these fluctuations are driven by the random term $I - E = I - PR$. A positive correlation between the two terms in the difference will reduce its variance from what would be found when they are independent.

Conclusion

The conclusion of our modelling of weight variation is this: Body weight will remain within a few percent of a fixed point as long as intake and activity maintain a stable mean. All that regulatory mechanisms need to ensure is that these two conditions are maintained. It is easier to see that activity levels will maintain a natural stability, since they have a lower bound however sedentary one becomes, and substantial increases generally require conscious determination to achieve.

Intake might be imagined to be affected by weight. We could envisage that increased weight for example, might induce an individual to eat extra in order to maintain that extra weight. Such a scenario could lead to unlimited weight gain, or loss, should reduced weight prompt reduced intake. What we claim our model shows is that what weight regulatory mechanisms need only do is to ensure that these 'runaway' positive feedback effects do not happen. They need only ensure that intake and activity remain stable, and then the negative feedback effect of body mass alone will do the rest.

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Figure Captions.

Fig 1. Three examples of simulated weight fluctuations. Initial weight is 75kg, $C\mu_p\beta = 0.0042$, intake variability is 25% and activity variability is 13%.

Fig 2. Comparison between continuous approximation to the weight change model (equation 4) and a stochastic realisation with the same parameters. ($W' = 95\text{kg}$, $W_0 = 75\text{kg}$, $C\mu_p\beta = 0.0042$)

Fig 3. Illustration of 5 realisations of the excess intake model (equations 5 & 6). The change in expected weight is also shown as the smooth dark line. ($W_s = 75\text{kg}$, $C\mu_p\beta = 0.0042$). Mean intake exceeds requirements by 3%.

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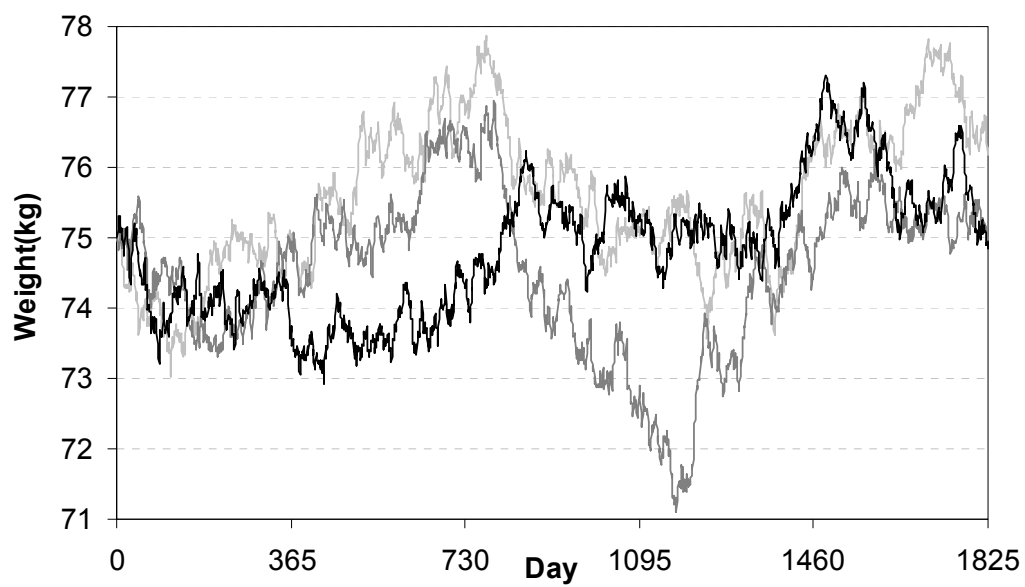


Fig 1. Three examples of simulated weight fluctuations (shown in different shades of grey). Initial weight is 75kg, $C\mu_p\beta = 0.0042$, intake variability is 25% and activity variability is 13%.

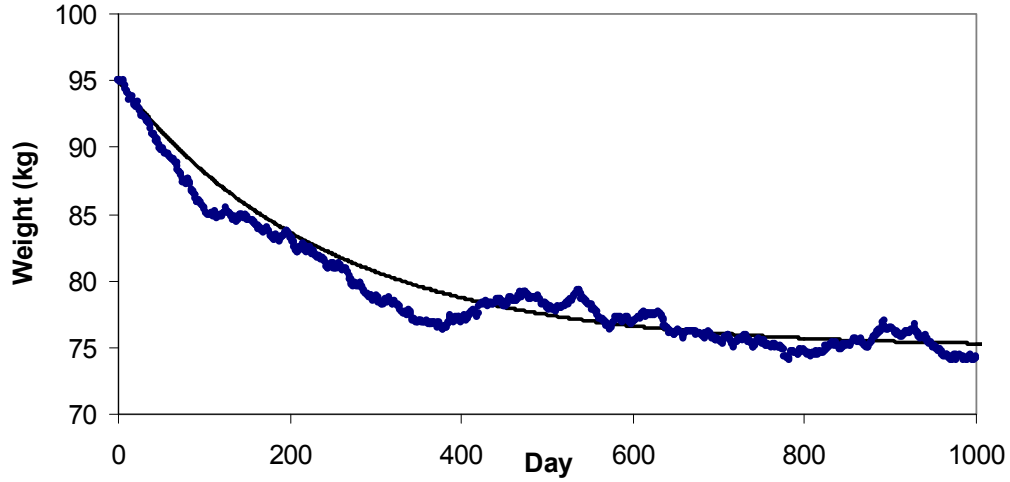


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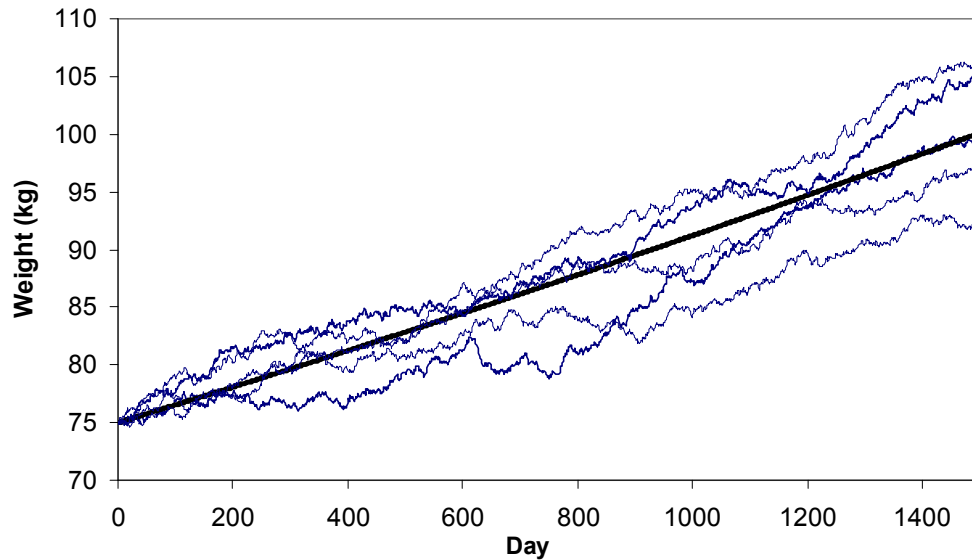


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