A model for the dynamics of human weight cycling

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The resolution to lose weight by cognitive restraint of nutritional intake often leads to repeated bouts of weight loss and regain, a phenomenon known as weight cycling or "yo-yo dieting". A simple mathematical model for weight cycling is presented. The model is based on a feedback of psychological nature by which a subject decides to reduce dietary intake once a threshold weight is exceeded. The analysis of the model indicates that sustained oscillations in body weight occur in a parameter range bounded by critical values. Only outside this range can body weight reach a stable steady state. The model provides a theoretical framework that captures key facets of weight cycling and suggests ways to control the phenomenon. The view that weight cycling represents self-sustained oscillations has indeed specific implications. In dynamical terms, to bring weight cycling to an end, parameter values should change in such a way as to induce the transition of body weight from sustained oscillations around an unstable steady state to a stable steady state. Maintaining weight under a critical value should prevent weight cycling and allow body weight to stabilize below the oscillatory range.

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1. Introduction

Obesity has reached the proportions of a global epidemic that needs to be addressed in its physiological and psychological aspects (Fairburn and Brownell 2002; Wadden *et al* 2002). Overweight is associated with increased risk of developing a number of diseases (Must *et al* 1999). When seeking to control overweight dieting is the most commonly used approach. In the US, where the prevalence of overweight continues to increase (Kuczmarski *et al* 1994; Mokdad *et al* 1999), as many as 25% of men and nearly 50% of women in 1985 reported trying to lose weight. Dieting, however, is often accompanied by repeated bouts of weight loss and regain, a phenomenon known as weight cycling (Blackburn *et al* 1989; Brownell 1989; Brownell and Rodin 1994). Among those on diet, most initially lose weight but will eventually regain it and, often, will even exceed their original weight after failing to sustain their efforts to control dietary intake. This pattern of alternating phases of dieting and relapse is also known as "yo-yo dieting".

Weight cycling is a process of clinical importance, since a number of studies in humans and rodents suggest that increased risks of morbidity (notably, cardiovascular disease) and mortality may be associated with fluctuations in body weight (Ernsberger *et al* 1996; Jeffery 1996). The clinical implications of the phenomenon were assessed by a Task Force assembled by the NIH (National Task Force on the Prevention and Treatment of Obesity 1994).

Mathematical modelling has been used for long to clarify the conditions in which oscillations occur in biological systems (Goldbeter 1996, 2002). The purpose of this paper is to explore the mechanism of weight cycling by means of a theoretical model and to relate the phenomenon to other rhythmic processes in biology.

2. The *PQR* model for weight cycling

The *PQR* model for weight cycling, named after its three variables, is based on a feedback of psychological nature, by which the subject decides to reduce dietary intake once a certain weight is exceeded. The three variables considered

Keywords. Model; oscillations; rhythms; weight cycling

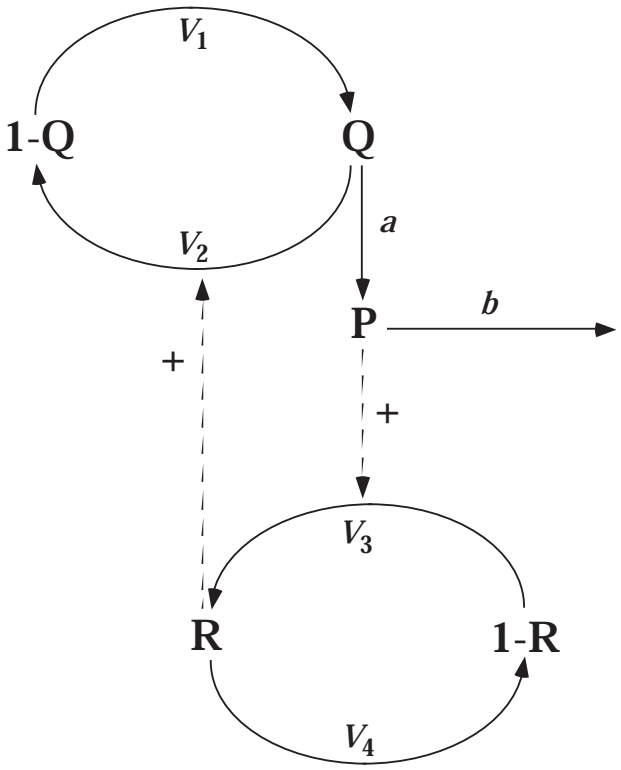

Figure 1. *PQR* model for weight cycling. Weight P (in excess of a basal reference value) increases with dietary intake Q at a rate aQ, and decreases due to energy dissipation at a rate that saturates at a maximum value b. An increase in weight P causes the degree of resolution to lose weight, or cognitive restraint (R) to increase at a maximum rate V_3P. Habituation causes R to wane at a maximum rate V_4. Dietary intake tends to increase at a maximum rate V_1 and to decrease at a maximum rate V_2R. Dashed arrows indicate the regulations exerted by P on R and by R on Q, respectively. The phenomenological eqs (1b) and (1c) ensure that at steady state R and Q exhibit a threshold dependence on P and R, respectively (see figure 2). In an appropriate range of parameter values the *PQR* model gives rise to sustained oscillations of the limit cycle type corresponding to weight cycling.

(see figure 1) are body weight (P), dietary intake (Q), and the degree of resolution measuring cognitive restraint (R) by which the subject decides to reduce the amount of dietary intake Q once weight P becomes too large. Both Q and R are normalized to vary between 0 and 1. Weight P and dietary intake Q are expressed as amounts in excess of a basal, reference value. The healthy weight (Garn 1996; Meisler and St Jeor 1996) reached around the age of 21 when growth is completed corresponds to a certain value $P > 0$ that is specific to each individual. Built into the model equations are threshold functions for the dependence of R on P, and of Q on R, which reflect the fact that the decision to lose weight by reducing dietary intake is generally of a sudden, all-or-none nature. Thus, the degree of cognitive restraint R increases abruptly once weight P passes a threshold value, whereas dietary intake Q undergoes a sharp decrease once R exceeds a threshold level. Such thresholds, naturally associated with time delays, prove to be crucial for the occurrence of sustained oscillations.

2.1 *Evolution equations*

The time evolution of the three variables is governed by the following differential equations:

$$\frac{dP}{dt} = aQ - b\frac{P}{K+P} \tag{1a}$$

$$\frac{dQ}{dt} = V_1\frac{(1-Q)}{K_1+(1-Q)} - V_2R\frac{Q}{K_2+Q} \tag{1b}$$

$$\frac{dR}{dt} = PV_3\frac{(1-R)}{K_3+(1-R)} - V_4\frac{R}{K_4+R}. \tag{1c}$$

The first equation indicates that the increase in weight P above the reference value is linked to the excess dietary intake Q, with a proportionality constant a measuring metabolic efficiency, and that P decreases, owing to metabolic energy dissipation, at a rate characterized by a function of the Michaelis-Menten type encountered in enzyme kinetics where the reaction rate initially rises with the level of substrate and reaches a maximum value when the substrate level becomes large. Here the dissipation rate reaches a maximum value, b, when excess weight P is much larger than constant K, which measures the value of P yielding half-maximum rate. Similar results are obtained when the sink for P is of a linear form ($-bP$).

The second and third equations express the way dietary intake and the degree of cognitive restraint are controlled and vary in the course of time. Each of these equations contains a positive and a negative term measuring, respectively, the rate at which Q or R increases or decreases. Thus eqs (1b) and (1c) indicate that both Q and R vary in a reciprocal manner between two reservoirs, the constant sum of which remains equal to unity. Equation (1b) indicates that Q tends to increase at a maximum rate V_1 multiplied by a Michaelian function in which the quantity (1-Q) plays the role of substrate: the smaller Q – i.e. the stronger the restriction –, the stronger the 'craving' to increase dietary intake. The tendency to increase food intake diminishes as Q tends to its maximum value equal to unity. At the same time Q tends to decrease at a maximum rate V_2R proportional to cognitive restraint, multiplied by a Michaelian function in which Q plays the role of substrate.

The structure of eq. (1b) reflects the existence of opposing factors acting on the quantity of nutritional intake. These factors mediate the neurohormonal control of appetite

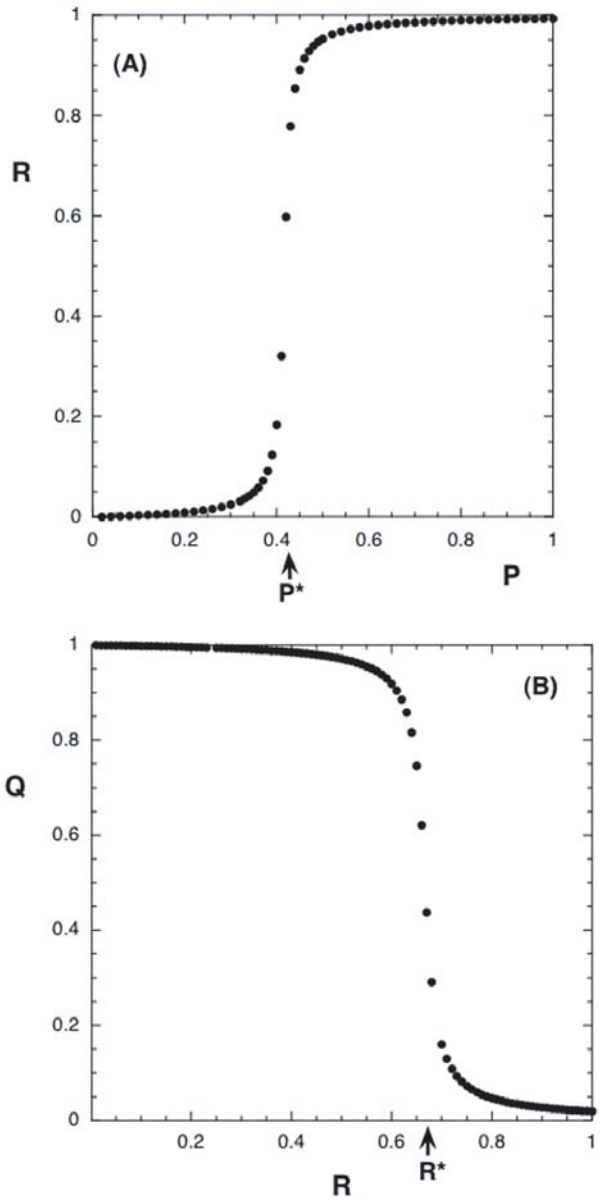

Figure 2. Threshold dependence of R on P and of Q on R. In **(A)**, the degree of cognitive restraint R is shown to rise abruptly when weight P exceeds the threshold value P^*. Conversely, R decreases sharply when P drops below P^*. In **(B)** dietary intake Q drops abruptly when R exceeds the threshold R^*, and increases when R passes below this value. The curves are generated by determining the steady states of R as a function of P and of Q as a function of R through numerical integration of eqs (1c) and (1b), respectively. The thresholds predicted by eqs (2a,b) correspond to the values P^*=0.418 and R^*=0.667. Parameter values are (per unit time): a=b=0.1, V_1=1, V_2=1.5, V_3=6, V_4=2.5; moreover, K=0.2 (per unit weight), and K_i=0.01 (i=1,...4). Both R and Q are expressed as fractions varying between zero and unity. One unit of excess weight P corresponds, typically, to 25 and 75 kg for cases of mild and severe overweight, respectively.

(Wynne *et al* 2005). Thus, a variety of neuropeptides synthesized in the gut regulate food intake. The first term in eq. (1b) reflects the orexigenic influence of one of these neuropeptides, ghrelin, which enhances appetite and increases food intake in humans (Wren *et al* 2001). The second term in eq. (1b) –besides reflecting negative regulation by cognitive restraint R– expresses the effect of other gastrointestinal peptides such as cholecystokinin and glucagon-like peptide-1, which promote satiety and reduce food intake in humans (Flint *et al* 1998; Woods 2004). On a longer time scale, insulin and leptin, secreted respectively by the pancreas and by adipocytes, act as anorexigenic signals and decrease food intake. Ghrelin may also be a long-term regulator of energy intake since plasma ghrelin levels are correlated with body mass (Wynne *et al* 2005). The action of all these peripheral signals is integrated at the level of the hypothalamus, primarily in the arcuate nucleus where neuronal circuits using specific neuropeptides act to stimulate or reduce food intake (Schwarz *et al* 2000; Wynne *et al* 2005).

The structure of eq. (1b) therefore takes into account the opposing influences of orexigenic and anorexigenic signals involved in the neuro-hormonal control of appetite, as well as the role of cognitive restraint R, which tends to decrease food intake. Cognitive restraint is the psychological variable responsible for weight cycling. In the absence of coupling to cognitive restraint, P and Q would settle to a stable steady state for a given set of parameter values.

The choice of Michaelian functions for the two terms in eq. (1b) is largely phenomenological, and expresses in a simplified, compact form the complexity of neurohormonal control of food intake. The choice of this formalism can be justified as follows. First, as is often the case in biochemistry and physiology, these functions are saturable, i.e. they increase from zero up to a plateau corresponding to a maximum value, and therefore cannot grow in an explosive manner. Thus, the rate of decrease in Q triggered by anorexigenic signals rises with Q up to a maximum value, while the rate of increase in Q triggered by orexigenic signals diminishes from a plateau and goes to zero as Q increases from zero up to its maximum value. Second, a key property of eq. (1b) is that it naturally produces a threshold in the dependence of Q on R at steady state (see below, figure 2B). This result is based on prior work on biochemical systems controlled by protein covalent modification, where thresholds originate from similar equations involving two antagonistic enzyme reactions which obey Michaelis-Menten, saturable kinetics (see below).

The structure of eq. (1c) is similar to that of eq. (1b). It indicates that the degree of resolution R increases in a Michaelian manner with (1-R), at a maximum rate V_3P proportional to weight, while R decreases at a maximum rate V_4 multiplied by a Michaelian function in which R plays the role of substrate. The latter term reflects a loss of cognitive restraint in the course of time due to a process of habituation,

which is analogous to the desensitization encountered at the cellular level in the response to hormonal or sensory stimuli.

In the above equations, parameters a and b thus have a metabolic meaning as they measure how weight P increases with food intake Q or decreases autonomously due to energy dissipation. Parameters V_1 and V_2 respectively measure the tendency of Q to increase with orexigenic signals or to decrease with anorexigenic signals and with cognitive restraint R, while V_3 and V_4 have a psychological nature and measure the rate at which R increases with P or wanes in the course of time.

2.2 *Threshold values of* P *and* R

The point important for weight cycling is that the phenomenological eqs (1b) and (1c) can generate sharp thresholds in the steady-state dependence of R on P and of Q on R. The existence of sharp thresholds in equations of this sort has been extensively studied in the context of regulation of protein activity by reversible modification, e.g. phosphorylation-dephosphorylation, as a result of "zero-order ultrasensitivity" (Goldbeter and Koshland 1981). Equations very similar to eqs (1a)-(1c) involving thresholds have been used for modelling the biochemical clock that drives the early cell division cycles in amphibian embryos (Goldbeter 1991, 1996).

Provided that the constants K_i (i=1,...4) are much smaller than unity, eqs (1b) and (1c) ensure that R will undergo an abrupt increase as soon as P exceeds a threshold value P^* (figure 2A) and that Q will drop sharply when R passes a threshold value R^* (figure 2B). The threshold values P^* and R^* are given by the following analytical expressions (see Goldbeter and Koshland 1981) as a function of the maximum rates V_i and threshold constants K_i (i=1,...4):

$$P^* = \left(\frac{V_4}{V_3}\right)\left(\frac{1+2K_3}{1+2K_4}\right) \tag{2a}$$

$$R^* = \left(\frac{V_1}{V_2}\right)\left(\frac{1+2K_2}{1+2K_1}\right). \tag{2b}$$

For the parameter values considered in figure 2, P^*=0.418 and R^*=0.667 (R is a dimensionless quantity, while P is expressed in units which vary depending on whether the model applies to moderately or severely overweight persons; see below).

3. Dynamics of weight cycling

3.1 *Sustained oscillations*

The analysis of the model indicates that depending on parameter values, the system governed by eqs (1a-c) can either evolve towards a stable steady state or undergoes sustained oscillations around an unstable steady state. Eq. (1a) indicates, however, that a steady state only exists if energy dissipation is sufficiently large at a given metabolic efficiency, i.e. $b > aQ$. Given that Q varies between 0 and 1, this condition is satisfied when $b \geq a$. In the contrary case, when energy dissipation is not large enough, no steady state is reached and weight P will increase monotonously in the course of time.

A typical example of sustained oscillations produced by the model is shown in figure 3A. Oscillations in excess weight P are accompanied by periodic variations in both the degree of cognitive restraint R and excess dietary intake Q. The period of the oscillations in figure 3A is of the order of ten time units. A reasonable estimate would be obtained if time were expressed, for example, in units of 2 weeks to 1 month, which would correspond to a weight cycle of the order of half a year to one year (Wadden *et al* 1996). As to the magnitude of the changes in weight, cycles generally correspond to a loss (followed by a gain) of some 5 or 15 kg in moderately or more severely overweight persons (Wadden *et al* 1996). To account for such changes, excess weight P in figure 3A should be expressed in units of 25 kg and 75 kg, respectively. These estimates set the time and weight units in which parameter values listed in the legends to figures 2 and 3 should be expressed to match the magnitude and time course of weight cycling.

Sustained oscillations generated by the PQR model correspond to the evolution toward a closed curve in the phase plane formed when one of the three variables, e.g. P, is plotted as a function of either Q or R (figure 3B). This closed curve is known as a limit cycle since it can be reached from inside or outside, regardless of initial conditions, as shown in figure 3B. The time taken to travel once along the cycle corresponds to the period of oscillations. Limit cycle oscillations are particularly stable as they are characterized by a unique period and amplitude for a given set of parameter values. Most biological rhythms represent sustained oscillations of the limit cycle type (Goldbeter 1996, 2002).

3.2 *Mechanism of oscillations*

The mechanism underlying weight cycling in figure 3A can best be followed by turning to the steady-state curves of figure 2. Let us start at a value of P smaller than the threshold P^*. Because P is relatively small, the degree of restraint R is also reduced and less than R^*. As the value of Q is large because Q is assumed to decrease due to cognitive restraint only when $R > R^*$, P will slowly increase in the course of time. Consequently P moves to the right on the curve of figure 2A, until it passes the threshold value P^*. This leads to an abrupt rise in the degree of restraint R : the passage beyond the critical weight P^* triggers in the subject the

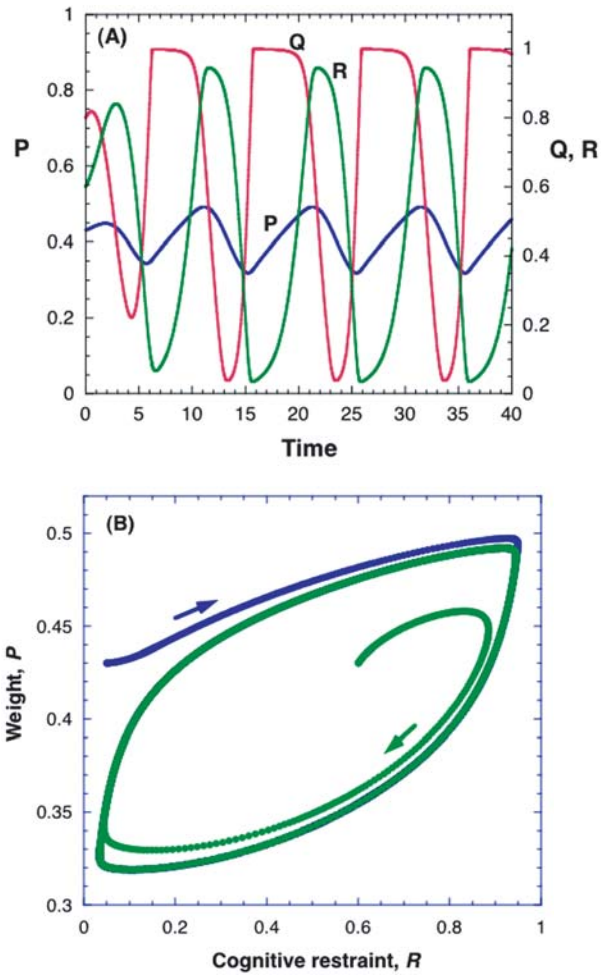

Figure 3. Sustained oscillations generated by the model for weight cycling. (**A**) Time evolution of excess weight P (in blue), degree R of cognitive restraint (in green), and fraction Q of maximum dietary intake in excess of a basal value (in red). (**B**) The oscillations correspond to the evolution towards a limit cycle shown here in the (P, R) plane as a projection of the trajectory followed by the 3-variable system governed by eqs (1a-c). The same closed curve, known as a limit cycle, is reached from two distinct initial conditions: P=0.43, Q=0.9, R=0.6 (green trajectory), and P=0.43, Q=0.8, R=0.05 (blue trajectory, which merges with the green one), corresponding to points located inside and outside the limit cycle, respectively. To match typical durations of weight cycles, time in (**A**) should be expressed in units of the order of one week to one month. The curves are obtained by numerical integration of eqs (1a-c), using the Berkeley Madonna program. Parameter values are as in figure 2.

decision to reduce dietary intake. The increase in R beyond the threshold R^* causes Q to drop sharply, as shown by the curve in figure 2B. As a result of this drop in dietary intake, after a lag P will begin to decrease. Eventually, in conjunction with habituation, the passage of P below the threshold P^* in figure 2A triggers a decline in the degree of restraint R. When R drops below the threshold R^* in figure 2B, again after a lag Q will start to increase. This relapse will cause P to resume its rise: a new round of weight cycling begins.

3.3 *Parameter range yielding oscillations*

While the above explanation clarifies the dynamics of weight cycling, it may convey the wrong impression that oscillations necessarily occur once the feedback loop based on cognitive restraint operates. A key insight from the model, however, is that sustained oscillations only occur in precise conditions, in a domain bounded by critical values of each of the control parameters. This property is illustrated in figure 4 as a function of parameter V_4, which measures the rate at which the cognitive restraint that controls the reduction of dietary intake wanes over time. This parameter largely governs the tendency to relapse. The family of curves in figure 4 shows the time evolution of excess weight for ten values of V_4 increasing from 0.1 to 10 by steps of 1.1.

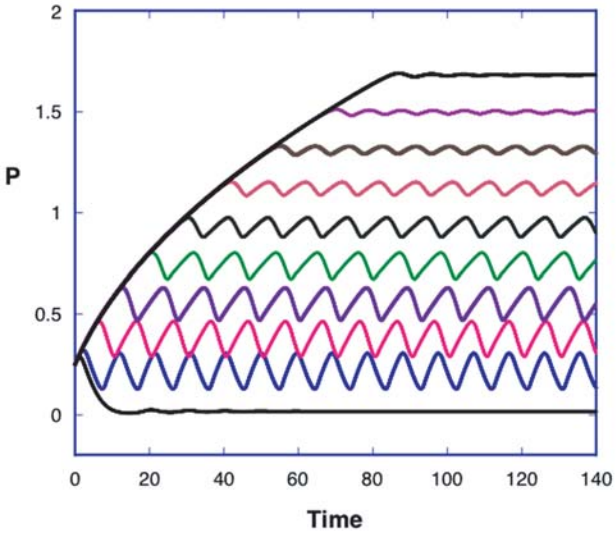

Figure 4. Domain of occurrence of weight cycling as a function of parameter V_4, which measures the rate of waning of the resolution to reduce dietary intake by cognitive restraint. Shown is the family of curves that represent the time evolution of excess weight P for V_4 increasing from 0.1 (lowest curve) to 10 (upper curve) by 9 steps of 1.1. For V_4=0.1 (first curve from the bottom) a stable steady state corresponding to a low value of P is reached. For V_4 > 9.4, excess weight stabilizes at a relatively larger value. In the interval between these two critical values of V_4, sustained oscillations in P corresponding to weight cycling occur. Both the latency (i.e. the time to the first peak in P) and the mean level of the oscillations increase with parameter V_4. The curves are obtained by numerical integration of eqs (1a-c), using the parameter values of figures 2 and 3. Initial conditions are P=0.25, Q=0.9, R=0.02.

For the parameter values considered in figure 4, sustained oscillations are observed in the range $0.12 < V_4 < 9.28$. For V_4 less than 0.12, P reaches a stable, low steady-state value (lower curve). When V_4 exceeds this critical value, sustained oscillations occur. The latency (i.e. the time to the first peak in P) and mean level of P during oscillations rise progressively as the value of V_4 increases. At the same time, the amplitude of the oscillations in P progressively diminishes, until the system reaches a stable steady state corresponding to a relatively large value of P when V_4 exceeds the second critical value close to 9.3 (upper curve). Sustained oscillations therefore occur in a range bounded by two critical values of V_4. These critical parameter values correspond to a Hopf bifurcation, i.e. to the onset of limit cycle oscillations when the steady state becomes unstable.

Equations (2a) and (2b) suggest that the ratios (V_1/V_2) and (V_3/V_4) are of crucial importance for oscillations, rather than the absolute values of the individual rates. Numerical simulations confirm that sustained oscillations only occur when each of these ratios lies within a range bounded by two critical values. The domain in which weight cycling occurs is shown in figure 5. Outside this closed domain, weight P stabilizes at a stable steady state that tends to be low as (V_1/V_2) decreases and (V_3/V_4) increases, or high in the opposite case. The amplitude of weight cycling can be very low near the boundary of the oscillatory domain, and increases as the system enters the domain of oscillatory behaviour in figure 5 (see figure 4).

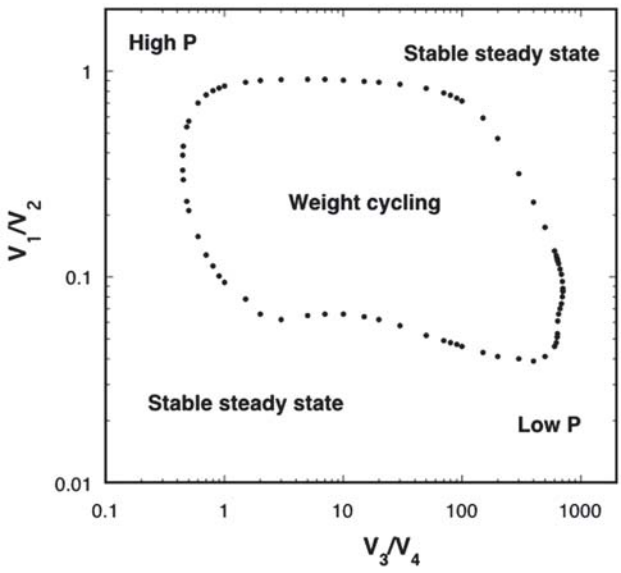

Figure 5. Domain of weight cycling as a function of (V_1/V_2) and (V_3/V_4). Upon varying these parameter ratios, V_2 and V_4 are held at their basal values of 1.5 and 2.5, respectively. The boundary of the domain of sustained oscillations (weight cycling) was determined by numerical integration of eqs (1a-c), for the parameter values of figures 2 and 3. Outside the domain of sustained oscillations, weight P stabilizes at a stable steady state. The steady-state value of P tends to increase with (V_1/V_2), and to decrease when (V_3/V_4) rises. The boundary of the oscillatory domain is the set of Hopf bifurcation points corresponding to the onset of limit cycle oscillations.

4. Discussion

Biological rhythms occur over a wide range of periods in a variety of systems including neural and cardiac rhythms, oscillatory enzyme reactions, pulsatile hormone secretion, and circadian rhythms. These oscillations originate from feedback processes that control the expression of genes or the activity of enzymes, receptors, or ion channels (Goldbeter 1996, 2002). The present study indicates that the phenomenon of weight cycling shares common properties with oscillatory phenomena observed at the cellular level in biological systems, even if the feedback process that drives the oscillations is of a psychological rather than genetic, biochemical or physico-chemical nature.

In relating weight cycling to biological rhythms of the limit cycle type, several caveats should be raised. The regularity found for oscillations of this sort in biological systems should not be expected for oscillations driven by a feedback of psychological nature, since the latter does not obey strict physico-chemical laws as do chemical or cellular systems. Moreover, beyond their stochastic variation, some parameters of the model are likely to change in the course of time; this may affect quantitatively, and even qualitatively, dynamic behaviour. Thus, after a first round of weight loss, biochemical parameters may evolve in such a way that regain will occur more readily, as a result of increased metabolic efficiency (Brownell *et al* 1986). Such biochemical changes could be reflected in the model by an increase in parameter a, which measures the rate at which weight increases with a given level of dietary intake. The rate of attenuation of cognitive restraint, measured by V_4, may also rise during successive weight cycles. When a progressive increase in a and V_4 in the course of time is incorporated into the model oscillations are still observed, but the mean level of P around which they occur rises (see figure 4). The upward drift in the oscillations corresponds to the clinical observation that the peak weight observed during successive cycles often tends to increase. If the rise in the two parameters is maintained, however, oscillations may eventually stop, and the weight will continue to increase monotonously. Incorporating changes in parameters such as a and V_4 may thus account for a progressive increase in weight with age.

Body weight and appetite are controlled by an array of peripheral signals which are integrated in the hypothalamus to stimulate or limit food intake (Schwarz *et al* 2000;

Wynne *et al* 2005). The model takes into account these opposing regulatory influences as well as the effect of cognitive restraint in setting the level of the nutritional variable Q. Weight cycling originates in the model from the negative feedback exerted by cognitive restraint on weight. The question arises as to whether the model might also pertain to a disorder such as anorexia, which involves severe nutritional restraint associated with unknown changes in endocrine and neural modulators of food intake. Although such extension goes somewhat beyond the framework of the present model we may note that because it is associated with excessive restraint, anorexia should correspond in the model to a situation in which P reaches a value well below that corresponding to the healthy weight. In the stability diagram drawn as a function of the ratios of parameters (V_1/V_2) and (V_3/V_4), this region would be located well under the oscillatory range, near the bottom, in the right corner in figure 5.

In spite of its relative simplicity the *PQR* model provides a theoretical framework for weight cycling, which suggests ways to control the phenomenon. The view that weight cycling represents limit cycle oscillations, possibly modulated by a drift in parameter values, has indeed specific implications. The analysis of the model indicates that sustained oscillations only occur in a precise window bounded by critical values of a particular control parameter. Outside this range weight P reaches a stable steady state. The existence of a domain of oscillations bounded by critical values of a control parameter was illustrated in figure 4 as a function of parameter V_4, and in figure 5 as a function of the ratios (V_1/V_2) and (V_3/V_4). Similar results on the existence of critical values bounding a domain in which limit cycle oscillations occur can be obtained as a function of other parameters of the model such as V_1, a or b.

Thresholds play a key role in the mechanism of weight cycling. Thus, the oscillations in figure 3A disappear and the system settles at a stable steady state in which P is close to the relatively large value of 0.44 when the thresholds in the curves of figure 2 become less steep as parameters K_i (i = 1,...4) rise above the value of 0.05 (see figure 6). This result shows that weight cycling occurs only when the thresholds in the dependence of R on P and of Q on R are sufficiently sharp. Because oscillations require the passage of P and R back and forth from below to above their respective thresholds, another way to stop oscillations is to prevent the rise of P above the threshold P^* (by acting on a or b – the latter parameter measures energy dissipation and increases with physical exercise), or to ensure that cognitive restraint remains well above the threshold R^* (by increasing V_3 or diminishing V_4).

The requirement for threshold steepness and the existence of a critical range of parameter values in which oscillations occur are predictions of the model. These

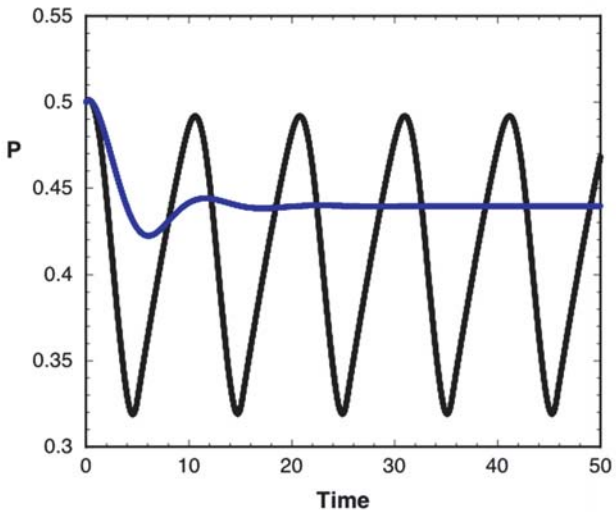

Figure 6. Weight cycling requires sharp thresholds in the dependence of R on P and of Q on R. Sustained oscillations obtained for K_i=0.01 (i=1,…4) – which values produce the thresholds shown in figure 2 – disappear when K_i=0.1 (blue curve). For this higher value of K_i the sigmoidal dependence of R on P and of Q on R is not as steep as that shown in figure 2. The curves are obtained for the parameter values of figures 2 and 3 by numerical integration of eqs (1a-c), starting from the initial values: P=0.5, Q=R=0.8.

results are missed in a simple verbal model, such as one that assumes that a subject diets until his/her weight falls below a certain value P_1 and resumes dieting when weight exceeds a higher value P_2. Rather than being based on a discontinuous, ad hoc description that always predicts oscillations of weight between two values imposed *à priori*, weight cycling was shown here to arise only in precise conditions, with an amplitude dependent on the parameters, in a model described by a set of three continuous differential equations.

According to the present model, weight cycling should appear or disappear abruptly when a critical value of a control parameter is passed. The implications of these results for the prevention of weight cycling lead to the concept of a therapeutic path in parameter space. In dynamic terms, the changes in parameter values associated with this path would induce the transition of body weight from sustained oscillations around an unstable steady state to a stable steady state. Maintaining weight under a critical value should bring weight cycling to an end and allow body weight to stabilize below the oscillatory range. This approach fits with the view (Stunkard *et al* 1979; Foster *et al* 1996) that the major issue in weight control is not so much one of losing weight as one of preventing relapse by adopting long-term lifestyle changes.

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