International Journal of Mathematical Archive-8(1), 2017, 194-199
Available online through www.ijma.info ISSN 2229 – 5046

CUMULATIVE DAMAGE MODEL FOR LEPTIN RESPONSE TO CARBOHYDRATE

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(Received On: 03-12-16; Revised & Accepted On: 30-12-17)

ABSTRACT

Cumulative damage model is applied to test whether the macronutrient content of the meal could influence postprandial leptin response. A unit suffers two kinds of damage which occurs either by shocks or increases with time. It fails only when the total amount of damage exceeds a failure level K. For the purpose of preventing failure, the damage is checked when each shock occurs and a unit is replaced before failure when the total damage exceeds a threshold level k. The expected cost rate is obtained, and an optimal level k which minimizes it is derived, when shocks occur in a Poisson process.*

Key Words: *Cumulative damage model, Non-homogeneous Poisson process. hunger and satiety*

INTRODUCTION

Hunger or satiety was related to leptin response, both were measured every hour after food intake, with a visual analogical scale. This method yields data with important inter individual variability that could reduce the statistical power to uncover subtle difference among meals. Despite this variability, two-way ANOVA showed a statistically significant interaction between test and postprandial interval (T0-T9 h) for hunger and satiety ratings. In women, between 1 and 9 h postprandially, both high-fat and high-carbohydrate meals were associated with significantly greater ratings of satiety and lower ratings of hunger than during the fasting experiment. There was no evidence of a statistically significant difference in hunger and satiety between carbohydrate and fat meals in women. In men, both carbohydrate and fat meals were associated immediately after food intake with higher and lower ratings of satiety and hunger, respectively, than during the fasting experiment. However, from the third and fourth hours to the end, hunger and satiety scores were similar in the carbohydrate meal and fasting experiment. Whereas satiety ratings were higher 3 and 4 h after the fat meal than after the carbohydrate meal, hunger ratings were lower 6 and 7h after the high-fat meal than after the carbohydrate meal (Fig).

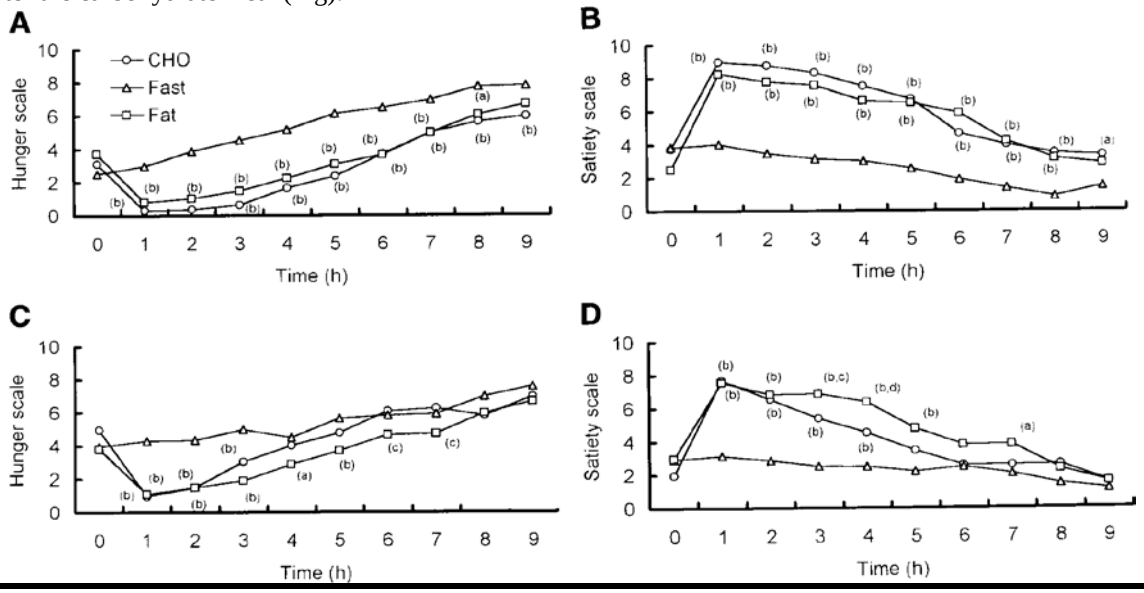

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Fig. Mean ratings of hunger (*A* and *C*) and satiety (*B* and *D*) according to time during fasting experiment and after food intake in women (*A* and *B*) and men (*C* and *D*). Carbohydrate, fat, and fasting experiments are represented by circles, squares, and triangles, respectively. ANOVA with repeated measures indicates a statistically significant interaction between test meal (carbohydrate/fat/fast) and postprandial intervals (T0-T9) for insulin and leptin in both men and women. Post hoc analysis: different from corresponding point in time during fast, *P*, 0.01 (a) and *P*, 0.001 (b); different from corresponding point in time after carbohydrate meal, *P*, 0.01 (c) and *P*, 0.001 (d).

That a carbohydrate meal induces a greater postprandial leptin response than an isoenergetic fat meal, that this response is correlated to the physiological postprandial insulin response, and there is no evidence of an association among postprandial leptin response, postprandial satiety or hunger, and subsequent food intake.

In women, postprandial leptin levels were higher after the fat meal than during the fasting experiment. This effect reached statistical significance in women but not in men. This gender difference may be the consequence of the relatively higher energy content of a meal in women compared with men (30%). On the other hand, the higher levels of circulating leptin in females may facilitate the discrimination between fast and a fat meal. Because fat ingestion had no significant impact on insulin secretion, these results suggest that factors other than insulin explain the difference in postprandial leptin variation between fat meal and fasting. In this respect, the energy load of the fat meal could play an independent role in the regulation of leptin secretion. It has been suggested that leptin acts as a sensor of energy balance. An energy deficit induced either by fasting or an increased energy expenditure, such as a marathon run acutely contributes to decreased leptin levels, independently of changes in the fat mass. Finally, Wang *et al.* demonstrated that leptin concentration is influenced by a nutrient-sensing mechanism; they found in vitro that the leptin gene expression was related to the activity of the hexosamine biosynthetic pathway, a cellular sensor of energy availability The impact of leptin levels on spontaneous food intake has not been explored in humans. Nine hours after the meal, food intake was not significantly different after the carbohydrate or fat meal nor was leptin concentration inversely correlated to food intake, suggesting that in humans leptin has no impact on the short-term regulation of food intake. These results are consistent with the effects of leptin on food intake in animals. In rodents, food intake was reduced after 4 h of an intraperitoneal infusion of leptin and in monkeys the central administration of recombinant leptin had no acute effect but reduced food intake the following day. These studies suggest that enhanced leptin secretion is more closely related to longterm (days) than to short-term (h) food consumption.

MATHEMATICAL MODEL

Suppose that shocks occur according to a nonhomogeneous Poisson process with intensity function $\lambda(t)$ and mean value function R(t), i.e., $R(t) \equiv \int_0^t \lambda(u)du$. Let X_i (i=12)be the i^{th} arrival between successive shocks and let

$S_j \equiv \sum_{i=1}^{j} X_i$ where S_0=0.The probability that at least j shocks occur during (0, t] is given by

$$H_j(t) \equiv P_r\{S_j \le t\} = \sum_{i=j}^{\infty} \frac{[R(t)]^i}{i!} e^{-R(t)},\ j = 0,1,2...$$

Each shock a random amount of damage to a unit, and the damage is additive. Random variables Y_i(i=1,2,....)denote the amount of damage produced by the i^{th} shock, and are Nonnegative, independent, and identically distributed. Each Y_i has a general distribution G(x), i.e., G(x) = $P_r\{Y_i \le x\}$ (i=1,2....).The total amount of damage is $Z_j \equiv \sum_{i=1}^{j} Y_i$ after j shocks, with $Z_0 \equiv 0$ and with the distribution function.

$$\Pr\{Z_j \le x\} \equiv G^{(j)}(x), \qquad j = 0,1,2...$$

Where $G^{(j)}(x)$ is the j-fold Stieltjes convolution of G(x)with itself, and $G^{(0)}(x) \equiv 1$ for x ≥ 0 and 0 for x<0

Further, suppose that a unit suffers another kind of damage continuously over time. For instance, a solar cell is a typical example. A solar cell is exposed to several galaxy cosmic rays and its generating ability depends on the area of the cell's surface. These rays damage the surface and weaken the ability continuously. That is the damage to a solar cell would be increasing with time at a constant rate a, independent of shocks. Hence, the total damage at the j^{th} shock is given by aS_j+Z_j. A unit fails if the total damage exceeds a failure level K.

It is difficult to monitor the state of a unit continuously. In our model, we check the total damage after a shock occurs. A preventive replacement is done before failure if the total damage exceeds a threshold level k ($0 \le k \le k$) at checking . Similarly, a failure replacement is done only when the total damage at the end of shock is greater than K.

The probability that a unit is replaced at failure, i.e., the total damage has exceeded a failure level K after a shock occurs, is

$$\sum_{j=0}^{\infty}\Pr\{Z_j + aS_j \le k, Z_{j+1} + aS_{j+1} > K\}$$

$$= \sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{k-at}\Pr\{Y_{j+1} + aX_{j+1} > K - at - z \mid S_j = t\}dG^{(j)}(z)dH_j(t)$$

$$= \sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{k-at}\left\{1 - \int_0^{K-z-at/a} G[K - z - a(t+y)]dH_{j+1}(y,t)\right\}dG^{(j)}(z)dH_j(t),$$

Where $H_{j+1}(y,t)=\Pr\{X_{j+1} \le y \mid S_j=t\}$, and we put that $x/a = \infty$ for any $x > 0$ when $a=0$.Further,the probability that a unit is replaced before failure, i.e. the total damage has exceeded a threshold level k and is less than K, after a shock occurs, is

$$\sum_{j=0}^{\infty}\Pr\{Z_j + aS_j \le k, k < Z_{j+1} + aS_{j+1} \le K\}$$

$$= \int_0^{k/a}\int_0^{k-at}\Pr\{k - z - at < Y_{j+1} + aX_{j+1}$$

$$\le K - z - a(t+y) \mid S_j = t\}dG^{(j)}(z)dH_j(t)$$

$$= \sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{k-at}\left\{\int_0^{(K-z-at)/a} G[K - z - a(t+y)]dH_{j+1}(y,t)\right.$$

$$\left. - \int_0^{(k-z-at)/a} G[k - a - a(t+y)]dH_{j+1}(y+t)\right\}dG^{(j)}(z)dH_j(t)$$

it is evident that (3)+(4)=1. Thus, the mean time to replacement is

$$\sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{k-at}\int_0^{\infty}(t+y)dH_{j+1}(y,t)dG^{(j)}(z)dH_j(t)$$

$$- \sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{k-at}\int_0^{(k-z-at)/a}(t+y)G[k - z - a(t+y)]dH_{j+1}(y+t)dG^{(j)}(z)dH_j(t)$$

$$= \sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{\infty} ydH_{j+1}(y,t)G^{(j)}(k - at)dH_j(t)$$

Let c_1 be the preventive replacement cost of a unit and $c_2(>c_1)$ be the cost for a failure replacement. Then, the expected cost rate, is from (3)-(5),

$$C(k) = \frac{1}{D(k)}\left\{c_1 + (c_2 - c_1)\sum_{j=0}^{\infty}\left[\int_0^{k/a} G^{(j)}(k - at)dH_j(t)\right.\right.$$

$$\left.\left. - \int_0^{k/a}\int_0^{(K-z-at)/a} G[K - z - a(t+y)]dH_{j+1}(y,t)dG^{(j)}(z)dH_j(t)\right]\right\}$$

Where

$$\mathrm{D(k)} = \sum_{j=0}^{\infty}\int_0^{k/a}\int_0^{\infty} ydH_{j+1}(y,t)G^{(j)}(k - at)dH_j(t)$$

This equation agrees with equation (5) in (2) when a = o. In particular, if a unit is always replaced at first shock, i.e.,k=o,the expected cost rate is

$$C(0) = \frac{c_1 + (c_2 - c_1)\left\{1 - \int_0^{k/a} G(K - ay)dH_1(y,0)\right\}}{\int_0^{\infty} ydH_1(y,0)}$$

When a unit is replaced only at failure, i.e. k=K, the expected cost rate is

$$C(K) = \frac{c_2}{\sum_{j=0}^{\infty}\int_0^{K/a}\int_0^{\infty} ydH_{j+1}(y,t)G^{(j)}(K - at)dH_j(t)}$$

Suppose that G(x) has a density g(x) and shocks occur in a Poisson process with rate ⋋, i.e. $\frac{dG(x)}{dx} = g(x)$ and ⋋(t)= ⋋. Then, we have

$$H_j(t) = \sum_{i=j}^{\infty} \frac{(\lambda t)^i}{i!} e^{-\lambda t}, \quad j = 0,1,2...,$$

$$H_{j+1}(y,t) = H_1(y) = 1 - e^{-\lambda y}, \quad j = 0,1,2....,$$

We seek an optimal level k* which minizes C(k) in (6).Differentiating C(k) with respect to k and setting it equal to zero, we have.

$$\sum_{j=0}^{\infty} \int_0^{k/a} \int_0^{k-at} \int_0^{(K-z-at)/a} G[K - z - a(t+y)] dH_1(y) dG^{(j)}(z) dH_j(t)$$

$$- \int_0^{(K-k)/a} G(K - k - ay) dH_1(y) \sum_{j=0}^{\infty} \int_0^{k/a} G^{(j)}(k - at) dH_j(t) = \frac{c_1}{c_2 - c_1}$$

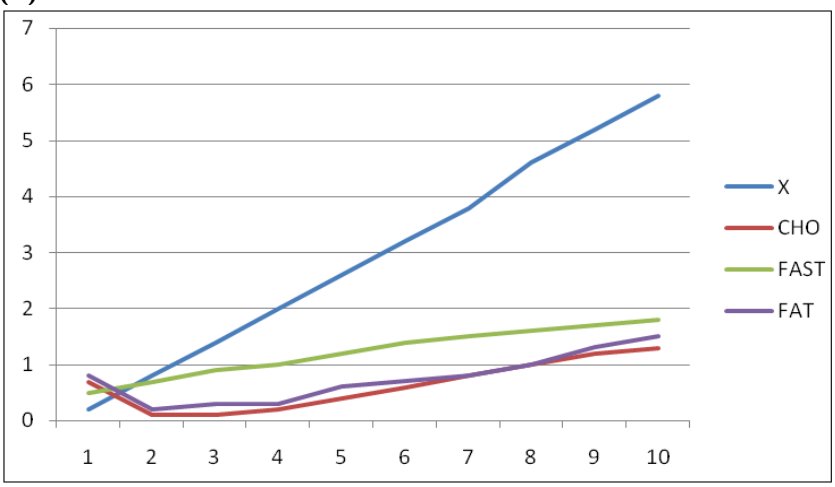
Denote the left-hand side of (11) by L(k|a). It is evident that L(0|a)=0,

$$L(K \mid a) = \sum_{j=1}^{\infty} \int_0^{K/a} G^{(j)}(K - at) dH_j(t),$$

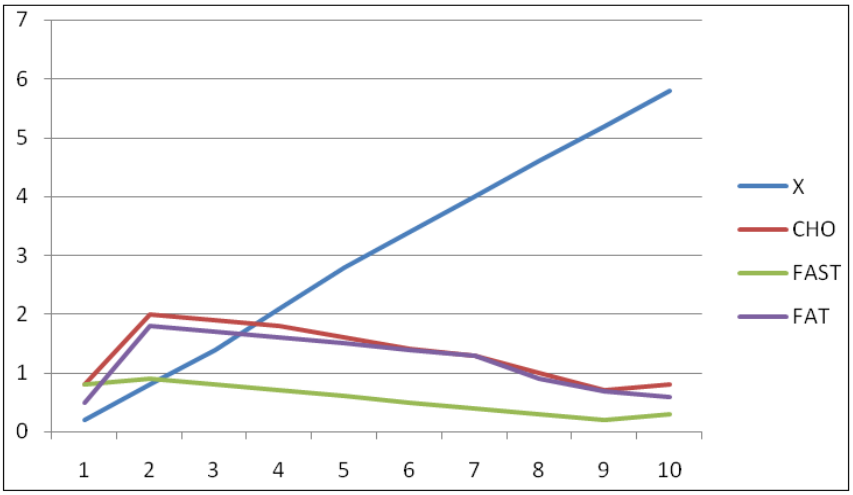
$$\frac{\partial L(k \mid a)}{\partial k} = \int_0^{(K-k)/a} g(K - k - ay) dH_1(y) \sum_{j=0}^{\infty} \int_0^{k/a} G^{(j)}(k - at) dH_j(t) > 0$$

Thus, L(k | a) is strictly increasing from 0 to L(K | a). Therefore, we have the following optimal replacement policy.

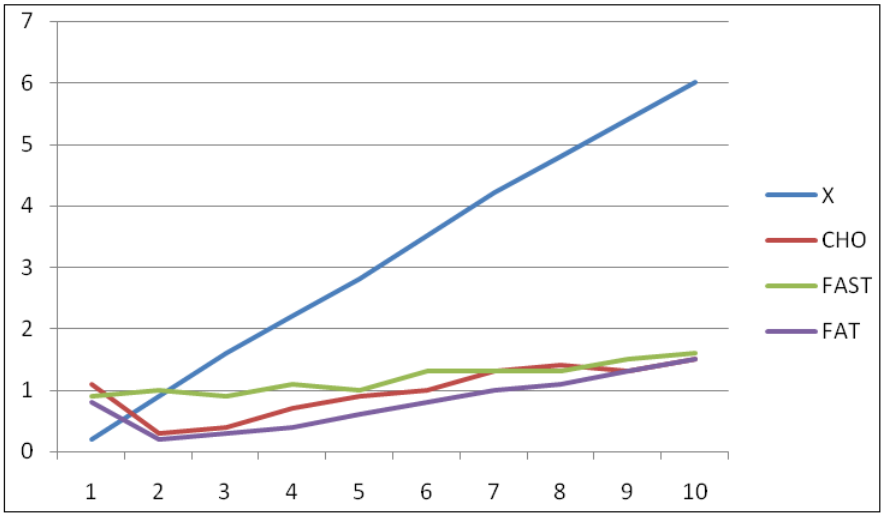
HUNGER SCALE (A)



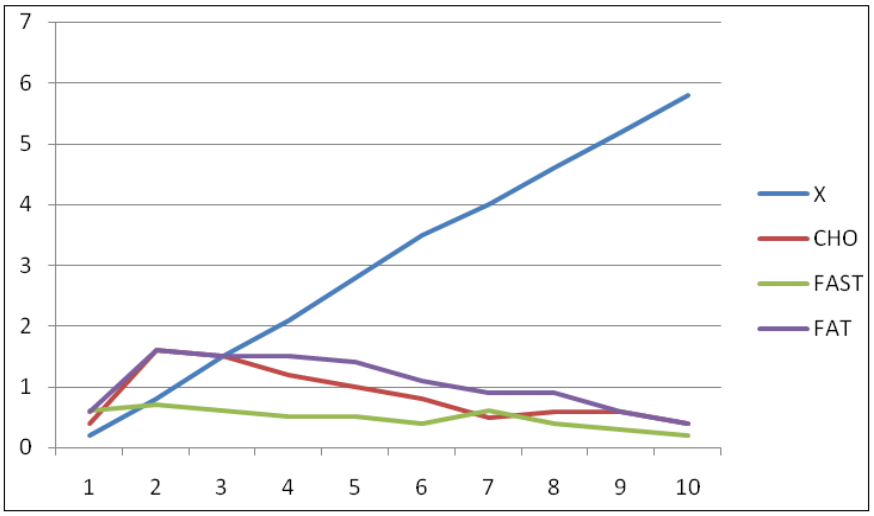
SATIETY SCALE (B)



HUNGER SCALE (C)



SATIETY SCALE (D)



Mean rating of hunger(cho and fast)

A		C	
$H_j(x)$	$G_j(x)$	$H_j(x)$	$G_j(x)$
1.2820	1.3568	1.44570	0.5148

Mean rating of satiety(cho and fast)

B		D	
$H_j(x)$	$G_j(x)$	$H_j(x)$	$G_j(x)$
0.6916	1.6779	0.2567	1.1638

Mean rating of hunger(fast and fat)

A		C	
$H_j(x)$	$G_j(x)$	$H_j(x)$	$G_j(x)$
1.1567	1.6338	0.7833	1.7933

Mean rating of satiety(fast and fat)

B		D	
$H_j(x)$	$G_j(x)$	$H_j(x)$	$G_j(x)$
1.5966	1.5711	1.1828	1.5722

CONCLUSION

Cumulative damage model was applied to made carbohydrate meal induces a greater postprandial leptin response than an isoenergetic fat meal and that this response is correlated to the physiological postprandial insulin response. It has recently been demonstrated that weight loss was more strongly associated with change in percent energy from fat than with change in total energy intake while the fat-free mass was maintained. The relationship between leptin levels and carbohydrate intake suggests that leptin could contribute to the beneficial impact of carbohydrate-rich diets. Also find the stress effect of leptin in (0, t].

REFERENCES

1. Barrachina, M. D., V. Martinez, L. Wang, J. Y. Wei, and Y.Tache. Synergistic interaction between leptin and cholecystokininto reduce short-term food intake in lean mice. *Proc. Natl. Acad. Sci. USA* 94: 10455–10460, 1997.
2. Leal-Cerro, A., P. P. Garcia-Luna, R. Astorga, J. Parejo, R. Peino, C. Dieguez, and F. F. Casanueva. Serum leptin levels in male marathon athletes before and after the marathon run. *J. Clin. Endocrinol. Metab*. 83: 2376–2379, 1998.
3. Levine, A. S., and C. J. Billington. Do circulating leptin concentrations reflect body adiposity or energy flux? *Am. J. Clin. Nutr*. 68: 761–762, 1998.
4. R.M. Feldman, Optimal replacement with semi-Markov shock models, J. Applied Probability 13, 108-117, (1976).
5. Tang-Christensen, M., P. J. Havel, R. R. Jacobs, P. J.Larsen, and J. L. Cameron. Central administration of leptininhibits food intake and activates the sympathetic nervoussystem in *rhesus macaques*. *J. Clin. Endocrinol. Metab*. 84:711–717, 1999.
6. T. Stow and T. Nakagawa, Replacement policies for a shock model with two kinds of damage, *Stochastic Modelling in* Innovative *Manufacturing*, *Springer* Lecture *Notes* in Economics *and Mathematical Systems 445*, 18%195, (1997).
7. Wang, J., R. Liu, M. Hawkins, N. Barzilai, and L. Rossetti. A nutrient-sensing pathway regulates leptin gene expression in muscle and fat. *Nature* 393: 684–688, 1998.
8. Weigle, D. S., P. B. Duell, W. E. Connor, R. A. Steiner, M. R.Soules, and J. L. Kuijper. Effect of fasting, refeeding, and dietary fat restriction on plasma leptin levels. *J. Clin. Endocrinol. Metab*. 82: 561–565, 1997.

Source of support: Nil, Conflict of interest: None Declared.