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NONADHERENCE TO DIETARY RESTRICTION, SELF PROTECTION, AND THE RISK OF MYOCARDIAL INFARCTION

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ABSTRACT

Despite bearing increased risk of suffering a heart attack, failure to adhere to a low-fat dietary regimen is wide-spread among high-risk individuals. The present paper applies a rational expected-utility-maximization model to inquire into the individual's decision to deviate from a prescribed low-fat diet, offering an economic explanation for excess high-fat consumption. The implications of self-protection against the risk of dying from an attack, through subscribing to emergency services that reduce delay in diagnosis and treatment, are further examined. The analysis reveals, counter-intuitively, that self-protection and dietary adherence can be complements, suggesting that public health policy may help reduce both the risk of an attack and the risk of dying from an attack through providing incentives for subscribing to delay-reducing services.

KeyWords: High-Fat Consumption, Diet Prescription, Nonadherence, Hazard Rate, Heart Attack, Self-Protection

JEL Classification: I12, I18, D81.

PsycINFO Classification: 3360, 3361.

I. Introduction

Coronary heart disease (CHD), caused by the narrowing of the coronary arteries that supply blood to the heart, is the number one killer in the US, accounting for 40 percent of all deaths.[1] It is considered, in part, to be a disease of lifestyle, associated with a number of poor health habits, such as a high-fat, cholesterol-rich diet, smoking and a low activity level. A primary clinical manifestation of CHD is myocardial infarction (MI), otherwise known as a heart attack, which is the death of the heart muscle due to the loss of blood supply.[2] Approximately one million Americans suffer a heart attack annually, four hundred thousand die as a result. Consequently, modification of CHD risk-related behavior to reduce the incidence of CHD morbidity and mortality obtain high priority in public health intervention efforts. However, getting people to develop good health habits and give up the risky behaviors that damage their health is not an easy task. In particular, despite convincing evidence that adherence to low-fat diet can significantly lower blood cholesterol levels,[3] failure to adhere to dietary recommendations is wide-spread.

Why is it then that high-risk individuals might not adhere to prescribed dietary regimens? Health psychology suggests a number of explanations:[4] preferences for meat and fat dairy products are well established and hard to alter; dietary recommendations might necessitate drastic changes in meal planning, cooking methods and eating habits; they may be too restrictive, monotonous, and associated with a lower quality of life. In addition, a low sense of self-efficacy (i.e., disbelief in one's ability the change one's diet) or a low level of health consciousness may attenuate adherence. The present paper addresses this question from a rational decision-making standpoint, proposing a rigorous, economic-oriented model to

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[1] See Taylor (1995, Ch. 14).

[2] A heart attack is caused by the formation of a blood clot on a cholesterol plaque located on the inner wall of an artery to the heart. Cholesterol plaque is a hard, thick substance on the artery walls, caused by deposits of cholesterol, a fatty chemical, which is part of the outer lining of cells in the body. Over time, the accumulation of cholesterol plaque may cause substantial thickening of the artery walls and significant narrowing of the arteries. Occasionally, the surface of the cholesterol plaque becomes sticky, causing blood clotting which completely block the artery (American Heart Association, 1993).

[3] See Carmody et al. (1987) for a review of the literature.

[4] See Taylor (1995, Ch. 5) for a detailed discussion of the psychological explanations and related literature.

explain individuals' deviation from a prescribed low-fat diet. Most importantly, while psychological explanations seem to focus on current inconveniences and discomfort as deterrents to adherence, the economic model takes explicit account of the risk involved, confronting future costs with present benefits of nonadherence. The analysis reveals that the risk of undergoing a heart attack, which is assumed to increase with the level of excess fat consumption, is not just a deterrent to nonadherence. It also plays the role of a discount factor of future costs, reducing their value further the greater the deviation from the recommended diet. This drives the individual to behave less respectfully towards the future, offering an economic motivation for a seemingly irrational behavior. Another interesting result is that an increase in the level of external risk factors (e.g., high blood pressure, diabetes, stress, family history), might actually *increase* high-fat consumption, rationalizing the psychological notion that if one knows that his time is short, he will seek increased enjoyment from the time still left.

The present paper belongs, on the one hand, to as yet a small literature which applies a rational economic approach to the analysis of health/clinical psychology phenomena, such as addictive behavior [Becker and Murphy (1988), Chaloupka (1991)], job burnout [Yaniv (1995)], suicide threat [Rosenthal (1993), Yaniv (1997a)], diagnosis delay [Yaniv (1997b)], or phobic disorder [Yaniv (1998)]. On the other hand, the paper is related to the broader literature of behavioral health economics, in particular to that dealing with individual health decision-making under uncertainty, such as Chang (1996), Francis (1997), Liljas (1998), or Picone and Wilson (1998). The paper begins (Section II) with modeling the individual's decision to consume high-fat products in excess of a prescribed low-fat diet, risking the occurrence of a heart attack at some uncertain time in the future, the distribution of which is affected by excess fat consumption. It then proceeds (Section III) by examining the implications of self-protection against the risk of dying from an attack, through subscribing to emergency services that reduce delay in diagnosing an attack or in obtaining emergency treatment. The analysis reveals, counter-intuitively, that self-protection and dietary adherence can be complements, suggesting that public health policy may help reduce both the risk of an attack and the risk of dying from an attack through providing incentives for subscribing to delay-reducing services. The paper concludes (Section IV) with a summary of the main results and some related remarks.

II. The Model

Consider an individual, who, during a given period of time, spends his or her disposable income, Y, on the consumption of high-fat, cholesterol-rich products, c, and low-fat, cholesterol-free products, h. Suppose that a blood test taken at the beginning of the period reveals above-normal values of cholesterol in his or her blood. Consequently, he or she is advised by a physician to stick to a low-fat diet under which high-fat consumption does not exceed the level of $\bar{c}$. Consuming high-fat products in excess of the recommended level bears an increased risk of undergoing a heart attack in the future. While the physician is unable, of course, to foresee the exact time that a heart attack will occur, he does have an opinion, which he shares with his patient, about the distribution of the time of an attack, $\dot{F}(t)$, where $F(t)$ represents the cumulative probability that an attack will occur by some time t in the future. In particular, the hazard rate, which is the probability of undergoing a heart attack at some time t in the future *given that a heart attack has not occurred prior to that time*, $\dot{F}(t) / [1 - F(t)]$, is assessed by the physician to be an increasing, convex function of high-fat consumption and of a number of external risk factors, denoted by S, such as high blood pressure, diabetes, cigarette smoking, obesity, stress, personality type, genetic predisposition, etc.[5] Denoting the hazard rate by $\lambda(c, S)$, we thus assume that $\lambda_c > 0$, $\lambda_S > 0$, $\lambda_{cc} > 0$, and $\lambda_{cS} \geq 0$.[6]

If suffering a heart attack at some period t in the future, the individual is assumed to either die or completely recover by the end of the period, where the probability of dying, γ, depends on the speed by which a heart attack is diagnosed and emergency treatment is provided. Suppose that treatment costs are fully covered by health insurance and loss of income during recovery is fully compensated by sick-pay benefits, so that the only major harm caused to the individual if he or she does not die is the psychological shock

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[5] See Criqui (1986) for a review of the literature on risk factors contributing to CHD.

[6] Picturesquely, the hazard rate may be viewed as the rate by which cholesterol deposits accumulate on the artery walls, $\dot{F}(t)$, as a ratio of the artery fraction still unblocked, $1 - F(t)$. That is, it is not narrowing per se that counts, but narrowing in relation to the effective artery opening.

accompanying the dreadful event and hospitalization in a coronary care unit, K.[7] Suppose, however, that the psychological shock is sufficiently intense to induce the individual to strictly adhere to the recommended diet, $\bar{c}$, thereafter. Adhering to the recommended diet ensures, by assumption, that a second attack will not occur in the future; hence $\lambda(c, S) = 0$ for $c \leq \bar{c}$.[8]

Suppose now that the individual must decide on whether or not to adhere to the prescribed diet prior to experiencing a heart attack, and if not - by how much to deviate from the physician's advice; that is, how much of high-fat products to consume in excess of the prescribed level. Suppose further that the individual decides on these questions through maximizing the present value of his or her expected lifetime utility stream from the consumption of high-fat and low-fat products, taking into account the adverse effect of high-fat consumption on the risk for future heart attack and its physical and psychological consequences. This may be viewed as a problem in optimal control, formulated as

$$Max \quad \int_0^{\infty} e^{-\delta t}\{[1-F(t)]U[c(t), h(t)] + F(t)(1-\gamma)\bar{U} - \dot{F}(t)K\}dt \qquad (1)$$

$$subject\ to: \quad \dot{F}(t) = [1-F(t)]\lambda[c(t), S] \qquad (2)$$

$$and: \quad h(t) = Y - c(t), \quad c \geq \bar{c}, \qquad (3)$$

where δ denotes the discount rate of future utility, and $U[c(t), h(t)]$ and $\bar{U} \equiv U(\bar{c}, Y-\bar{c})$ represent the individual's pre-attack and post-attack utility levels from consumption at time t, respectively, assumed to be an increasing, concave function of both products, thus satisfying $U_c > 0$, $U_h > 0$, $U_{cc} < 0$ $U_{hh} < 0$. For simplicity it is assumed that disposable income, Y, as well as product prices are constant over time.

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[7] Aside of having to face the fact that they have had a heart attack and the possibility of recurrence during the acute phase of hospitalization [Froese et al. (1974)], MI patients may experience cardiac arrest, having to be resuscitated through artificial means. This is likely to produce additional psychological problems such as nightmares, anxiety and depression [Druss and Kornfeld (1967)].

[8] Relaxing this assumption will add technical complexity to the model, without invalidating its results.

The integrand (1) discounts the expected stream of lifetime utility over an infinite time horizon. At any given time t in the future, the individual faces the cumulative probability $1 - F(t)$ of not yet suffering a heart attack, deriving the utility $U[c(t), h(t)]$ from consumption. However, with probability $F(t)$, he or she will already suffer a heart attack by this time. Given the probability $1-\gamma$ of surviving the event, the individual will then derive utility of $\bar{U}$ from adhering to a low-fat diet. In addition, with probability $\dot{F}(t)$, a heart attack will occur exactly at time t, causing a psychological shock of size K. Notice that problem (1)-(3) involves a state variable, $F(t)$, which is subject to a dynamic constraint (2), and a control variable, $c(t)$, which is subject to a static constraint (3). The control variable influences the objective function (1) both directly (through its own value) and indirectly through its impact on the evolution of the state variable.

Substituting (2)-(3) into (1) and applying Pontryagin Maximum Principle, the problem at hand involves the maximization of the Hamiltonian (omitting the time notation)

$$H: \ (1-F)[U(c,\ Y-c)\ -\lambda(c,\ S)K]\ +F(1-\gamma)\bar{U}\ +\ q(1-F)\lambda(c,\ S), \tag{4}$$

where q represents the 'shadow price' (i.e., the individual's marginal evaluation) of the cumulative probability of suffering an attack by time t. Maximization requires that the shadow price movement over time obeys the differential equation

$$\dot{q}\ =-H_F+\delta q\ =\ U(c,\ h)\ -\ (1-\gamma)\bar{U}\ -\lambda(c,\ S)K\ +\ [\delta\ +\lambda(c,\ S)]q, \tag{5}$$

and that optimal high-fat consumption, c^*, satisfies the first-order condition[9]

$$H_c\ =\ (1-F)[U_c(c,\ h)\ -\ U_h(c,\ h)\ -\ \lambda_c(c,\ S)(K-q)]\ =\ 0 \tag{6}$$

Condition (6) implies that excess fat consumption is independent of F, but dependent on its

[9]The second-order condition, $H_{cc} < 0$, will be satisfied by the concavity assumption on the utility function and the convexity assumption on the hazard rate function, given that the utility function is separable in both arguments (i.e., that $U_{ch} = 0$). Otherwise, the second-order condition should be *assumed* to hold.

shadow price, q. Hence, the variation over time in the latter explains the variation over time in the former. However, it can be shown that the necessary conditions (5)-(6) are actually satisfied with *constant* values of c^* and q.[10] Substituting thus $\dot{q}=0$ in (5) yields

$$q = -\frac{U(c, h) - (1-\gamma)\overline{U} - \lambda(c, S)K}{\delta + \lambda(c, S)}, \tag{7}$$

the negativity of which reflects the fact that the accumulation of F raises the probability of an undesirable occurrence, therefore reducing its optimal expected value.

Substituting now (7) into (6) and rearranging, we obtain

$$MB \equiv U_c(c^*, h^*) - U_h(c^*, h^*) =$$

$$\lambda_c(c^*, S)\left[K + \frac{U(c^*, h^*) - (1-\gamma)\overline{U} - \lambda(c^*, S)K}{\delta + \lambda(c^*, S)}\right] \equiv MC. \tag{8}$$

Equation (8) implies that high-fat consumption at each period of time preceding an attack should be determined such that the marginal benefit from deviating from the recommended diet (MB) equates its marginal cost (MC). The marginal benefit is reflected through the additional utility derived from consuming a unit of high-fat products rather than a unit of low-fat products. The marginal cost (MC) is reflected through the additional risk of suffering a heart attack emanating from consuming an additional unit of high-fat products. The increased risk involves not only the harm of suffering a psychological shock, but also the discounted value of the future utility loss due to having to adhere, if surviving, to a low fat diet, net of the expected psychological shock of an attack that might occur even if avoiding the additional high-fat consumption.

[10] This is essentially due to the assumption of an infinite time horizon and of the fact that q and c are independent of F (although c determines the accumulation rate of F). For a detailed proof of the constancy over time of the optimal shadow price and control variable in a problem as such, see Kamien and Schwartz (1991, pp. 223-225), who demonstrate this for a monopolist's problem of setting a price which, if unrestrictively high, might attract the entry of rivals.

A sufficient condition for nonadherence is that at the level of the recommended diet, $\bar{c}$, the marginal benefit from nonadherence exceeds the marginal cost. This will be the case if

$$U_c(\bar{c}, \bar{h}) - U_h(\bar{c}, \bar{h}) > \lambda_c(\bar{c}, S)[K + \frac{\gamma \bar{U}}{\delta}] . \qquad (9)$$

Because $\lambda(\bar{c}, S) = 0$, the marginal cost of nonadherence at $\bar{c}$ reflects, in addition to the psychological shock, just the utility loss of future consumption if not surviving an attack, discounted by the individual's time preference rate, δ. Given that condition (9) holds, the individual will opt to consume high-fat products in excess of the prescribed diet, raising the hazard rate to an above zero level. As nonadherence increases, the hazard rate will follow suit, shortening the expected time until a forthcoming attack. Consequently, the future must be discounted at a higher rate than the regular time preference factor, which increases with the level of nonadherence. As is evident from equation (8), this acts to moderate the marginal cost of nonadherence, stimulating a greater consumption of high-fat products.[11] In sum, the hazard rate is not just a deterrent to nonadherence. It also attributes a lower value to future loss the greater the deviation from the recommended diet. This drives the individual to behave less respectfully towards his or her future, offering an economic explanation for what seems to be an example of sheer irrational behavior.

As intuitively expected, equation (8) implies that an increase in the psychological shock, K, or in the marginal hazard rate, $\lambda_c(c, S)$, increases the marginal cost of nonadherence and thus acts to discourage excess fat consumption. However, an increase in the hazard rate itself (holding its marginal rate constant) *reduces* the future loss component of the marginal cost, acting to stimulate excess fat consumption. Consequently, a increase in any of the external risk factors, S, which (assuming $\lambda_S > 0$ and $\lambda_{cS} \geq 0$) may affect both the hazard rate and its marginal rate, will have an ambiguous effect on the extent of deviation from the recommended diet. Interestingly, if $\lambda_{cS} = 0$ or is relatively small, an increase in an external

[11]Notice that MC may be rewritten as $\lambda_c\{[U(c^*, h^*) - (1-\gamma)\bar{U} + \delta K]/(\delta + \lambda)\}$, which eliminates λ from the numerator of the future loss, thus highlighting its role as a discount factor of future utilities.

risk factor may actually *increase* high-fat consumption, rationalizing the psychological notion that if a person knows that the end is near, he or she will seek to increase the quality of the time still left, adhering to the prophet's complaint ("eat, drink, and be merry, for tomorrow we die", *Isaiah*, 22:13) rather than to a restricting diet.

Notice finally that the greater is $\bar{c}$, the lower will be the future loss from an attack, since the less restrictive will be the post-attack diet. Hence, the greater is $\bar{c}$, the lower will be the marginal cost of nonadherence, and the greater the pre-attack consumption of high-fat products. This suggests that a skillful physician may do better by prescribing a *stricter* than necessary diet, so as to induce a lower level of high-fat consumption. Such a strategy requires, however, that the physician has reasons to believe that the patient is likely to deviate from the prescribed diet. Otherwise, if condition (9) does not hold, a stricter than necessary prescription might have the opposite effect, driving the patient to nonadherence.

III. Self-protection

A major reason for the high mortality rates following heart attacks is the delay occurring in diagnosing an attack and obtaining emergency treatment.[12] Much of the blame falls on patients themselves, who often delay several hours or even days before seeking treatment. Some patients are simply unable to face the fact that they have had a heart attack, and others tend to interpret the symptoms as mild disorders.[13] While it may be rational for individuals to delay diagnosis of a given symptom if the marginal damage from doing so is relatively low, it can be shown that such behavior is irrational when suspecting a heart attack.[14] Adhering to the rationality premise, we thus assume that in case of suspecting a

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[12] Many of the heart attack deaths are due to ventricular fibrillation (chaotic electrical disturbance) of the heart that occurs before the victim can reach any medical assistance or an emergency room. When paramedics arrive, medication and/or electrical shock to the heart can be promptly administered to convert ventricular fibrillation to a normal heart rhythm. Once in an emergency room, electrical disturbances are treated through administration of medications to dissolve blood clots and open blood vessels, and/or balloon angioplasty to open an obstructed artery. Therefore, 90 to 95 percent of heart attack victims who make it to the hospital survive (American Heart Association, 1993).

[13] See Taylor (1995) for a discussion of delay behavior in MI episodes.

[14] See Yaniv (1997b).

heart attack, the individual will be seeking *prompt* diagnosis and treatment. It thus follows that the individual may also consider the possibility of self-protection against involuntary delay through measures that may help expedite diagnosis and treatment.[15] These include subscribing to an emergency call-in center which may provide round-the-clock cardiac diagnosis by phone (thus giving timely warning of the onset of an attack) and/or to a modern intensive care ambulance service (which adjusts its vehicle fleet and paramedics to the number of subscribers, enabling immediate response), insuring with a private medical center which guarantees surgery with no waiting list, or even relocating closer to a major medical center.

Suppose then that delay in diagnosis an attack and obtaining treatment, D, determines the probability of dying in case of an attack, $\gamma(D)$. More specifically, suppose that the probability of dying from an attack is an increasing, convex function of diagnosis and treatment delay, thus $\gamma'(D) > 0$ and $\gamma''(D) > 0$. Suppose further that the individual assesses the public system delay to be $\overline{D}$, but that he or she may act to reduce delay through subscribing to private emergency services and/or expending on other types of delay-reducing measures. Total expenditure, $\varphi(D)$, is assumed to be a decreasing, convex function of delay, thus $\varphi'(D) < 0$ and $\varphi''(D) > 0$, where $\varphi(\overline{D}) = 0$. That is, the shorter the desired delay, the larger the expenditure (which increases at increasing marginal rates). Allowing the individual to determine both the desired levels of delay, D^*, and high-fat consumption, c^*, his or her problem becomes:

$$Max \quad \int_0^\infty e^{-\delta t}\{[1-F(t)]U[c(t), h(t)] + F(t)\Omega(t)\overline{U} - \dot{F}(t)K\}dt \tag{10}$$

$$subject\ to: \ \dot{F}(t) = [1-F(t)]\lambda[c(t), S], \tag{11}$$

$$and: \quad \Omega(t) \equiv \int_0^t \{1-\gamma[D(k)]\}\dot{F}(k)dk \tag{12}$$

$$h(t) = Y - c(t) - \varphi[D(t)], \ c \geq \overline{c}, \ D \leq \overline{D}. \tag{13}$$

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[15] Self-protection, defined as measures taken to reduce the *probability* of a loss, was first analyzed in the literature by Ehrlich and Becker (1972), as an alternative to market insurance and to self-insurance which reduce the *size* of a loss.

Notice that if an attack has already occurred by time t, the individual's probability of surviving at that time depends on the level of delay existing at the particular time in the past when the attack occurred. Hence, expected utility at time t if already suffering an attack by that time is $\Omega(t)\overline{U}$. Notice also that if an attack has already occurred by time t, the individual will not expend on delay-reducing measures at that time, since adhering to the prescribed diet ensures, by assumption, that a second attack will not occur in the future.

Substituting (11)-(13) into (10), the Hamiltonian to be maximized is

$$H: (1-F)\{U[c, Y-c-\varphi(D)]-\lambda(c, S)K\} + F\,\Omega\overline{U} + q(1-F)\lambda(c, S), \qquad (14)$$

where, substituting (11) into (12), $\Omega \equiv \int_0^t \{1-\gamma[D(k)]\}[1-F(k)]\lambda\,[c(k), S]dk$.

The necessary condition for the evolution of the shadow price over time now becomes

$$\dot{q} = U(c, h) - \Omega\,U(\overline{c}, \overline{h}) - \lambda(c, S)\{K - F[1-\gamma(D)]\,\overline{U}\} + [\delta + \lambda(c, S)]q, \qquad (15)$$

whereas the necessary first-order condition determining c^* and D^* are given by[16]

$$H_c = (1-F)[U_c(c, h) - U_h(c, h)] - (1-F)\lambda_c(c, S)\{K - F[1-\gamma(D)]\overline{U} - q\} = 0 \qquad (16)$$

$$H_D = (1-F)[-\varphi'(D)U_h(c, h) - F\lambda(c, S)\gamma'(D)\overline{U}] = 0, \qquad (17)$$

A sufficient condition for self-protection is that at the level of the present delay, $\overline{D}$, the marginal cost of self-protection falls short of the marginal benefit. This will be the case if

$$-\varphi'(\overline{D})U_h(c, h) < F\lambda(c, S)\gamma'(\overline{D})\overline{U}, \qquad (18)$$

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[16] The second-order conditions, $H_{cc} < 0$, $H_{DD} < 0$, and $H_{cc}H_{DD} - H_{cD}^2 > 0$ are assumed to hold.

where $h = Y - c$ [as $\varphi(\overline{D}) = 0$]. Obviously, an incentive for self-protection will exist if, at $\overline{D}$, the marginal expenditure on reducing delay, $\varphi'(\overline{D})$, is sufficiently small or if the marginal reduction in the probability of dying from an attack, $\gamma'(\overline{D})$, is sufficiently large.

Unlike the previous problem, where q and c^* were independent of F and could be shown to be constant over time, equations (15)-(17) reveal that the shadow price as well as optimal delay and high-fat consumption are now dependent on F, which increases over time. Consequently, all critical variables will now vary over time. In particular, totally differentiating (16) and (17) with respect to time, t, we obtain

$$\frac{dD^*}{dt} = \frac{H_{cc}\lambda(c, S)\gamma'(D)\overline{U}\dot{F} + H_{Dc}\lambda_c(c, S)\{\dot{q} + [1-\gamma(D)]\overline{U}\dot{F}\}}{H_{cc}H_{DD} - H_{cD}^2} \tag{19}$$

$$\frac{dc^*}{dt} = \frac{-H_{DD}\lambda_c(c, S)\{\dot{q} + [1-\gamma(D)]\overline{U}\dot{F}\} - H_{cD}\lambda(c, S)\gamma'(D)\overline{U}\dot{F}}{H_{cc}H_{DD} - H_{cD}^2}, \tag{20}$$

where $H_{cD} = H_{Dc} = -\varphi'(D)(U_{ch} - U_{hh}) - F\lambda_c\gamma'(D)\overline{U}$, which is ambiguous, given that high-fat consumption is a normal good [i.e., $U_{ch} - U_{hh} > 0$]. Moreover, the sign of $\dot{q}$ is indeterminate from (15) alone.[17] However, since (2') implies that $\dot{F}$ is always positive, both (19)-(20) will be negative if $H_{cD} = H_{Dc} > 0$ and $\dot{q} + [1-\gamma(D)]\overline{U}\dot{F} < 0$, whereas (19) will be negative and (20) will be positive if the converse holds; that is, if $H_{cD} = H_{Dc} < 0$ and $\dot{q} + [1-\gamma(D)]\overline{U}\dot{F} > 0$. Hence, while one cannot rule out the perverse possibility that both optimal delay and optimal high-fat consumption increase over time or that the former increases while the latter decreases, it is possible to identify conditions ensuring that both

[17] Still, it is unlikely for $\dot{q}$ to be positive, because this would imply that the optimal marginal value of the cumulative probability of suffering a heart attack *increases* with time (i.e., that the accumulated risk becomes more desirable as time progresses).

optimal delay and optimal high-fat consumption decrease over time or that optimal delay decreases while optimal high-fat consumption increases. The former possibility is of particular interest as it implies that dietary adherence and self-protection can actually be *complements*: increased protection can be accompanied with increased adherence.

A number of studies, attempting to modify diet behavior of large populations, have concluded that while cholesterol levels were significantly reduced immediately after the intervention, a substantial number of participants reverted at later stages to their former behavior, even with intensive individualized instruction. That is, adherence to dietary prescriptions in the general population tends to fall off over time.[18] The possibility that self-protection and dietary adherence are complements suggests that it could be possible for public health policy to enhance adherence to low-fat diets through providing incentives for subscribing to private emergency services. To formally examine this suggestion, suppose, for simplicity, that $\varphi(D) = \pi(\overline{D} - D)$, where π denotes the price of reducing delay by an additional unit of time. Totally differentiating (16) and (17) with respect to π, and evaluating the results at $D = \overline{D}$ [assuming that the entry condition (18) holds as an equality], we obtain

$$\frac{dD^*}{d\pi}|_{D=\bar{D}} = \frac{-H_{cc}U_h(c, h)}{H_{cc}H_{DD} - H_{cD}^2} \tag{21}$$

$$\frac{dc^*}{d\pi}|_{D=\bar{D}} = \frac{H_{cD}U_h(c, h)}{H_{cc}H_{DD} - H_{cD}^2}. \tag{22}$$

While the sign of (21) is unambiguously positive, implying that a fall in π at $\overline{D}$ will induce self-protection ($D^* < \overline{D}$), the sign of (22) is ambiguous, depending on the sign of H_{cD}: if H_{cD} is negative, self-protection will be accompanied with increased nonadherence, whereas if H_{cD} is positive, self-protection will be accompanied with increased adherence. Quite

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[18] See Reeves et al. (1983) and Southard et al. (1992).

interestingly, and contrary to prior intuition, subscribing to a self-protection scheme must not necessarily give rise to a "moral hazard" effect in the form of stimulating health negligence behavior. That is, not only can dietary adherence and self-protection be complements in the sense that they both increase over time, but also in the sense that a fall in price which induces the latter enhances the former.[19] It thus follows that public health policy, informed of private preferences, might be able to reduce both the risk of a heart attack and the risk of dying from an attack through subsidizing the price of private emergency services.

IV. Concluding Remarks

The present paper has applied a rational expected-utility-maximization model to inquire into the individual's decision to deviate from a prescribed low fat diet, despite bearing an increased risk of suffering a heart attack in the future. The analysis has given rise to a number of interesting conclusions regarding dietary nonadherence and public health policy. First, the risk of undergoing a heart attack assumes a double-agent role in the rational decision model: while acting to deter high-fat consumption, it also enhances nonadherence *through discounting future costs at a rate that is positively dependent on the extent of* deviation from the recommended diet. In its latter role as a discount factor which increases with the level of nonadherence, the risk of an attack drives the individual to behave less respectfully towards the future. Second, the greater the level of external risk factors, such as smoking, diabetes, obesity, or genetic predisposition, the greater may be excess fat consumption, as the shorter the expected time left for the individual to enjoy life. Third, a skillful physician may induce lower consumption of high-fat products through prescribing a stricter than necessary diet. Fourth, contrary to intuition, dietary adherence and self-protection to reduce delay in diagnosis and treatment of an attack can be complements, in the sense that they both increase over time and that engaging in the latter enhances the

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[19] Notice, however, that once the individual engages in self-protection, further reductions in π must not necessarily increase adherence, since it will also give rise to a positive income effect on high-fat consumption, given that the latter is a normal good.

former. Public health policy may thus help reduce both the risk of an attack and the risk of dying from an attack through providing incentives for high-risk individuals to subscribe to delay-reducing emergency services.

The model developed in this paper may be extended in several ways, although at the cost of substantial mathematical complexity. First, rather than *assuming* that the individual learns his lesson after the first attack, adhering thereafter to the recommended diet, the possibility of post-attack nonadherence, at the risk of suffering a second attack, may be allowed for. It would be interesting to see whether optimal post-attack adherence is indeed greater than its pre-attack counterpart, or whether it may be rational for the individual to exhibit even a greater disrespect towards his or her future. Secondly, the possibility of permanent physical and emotional impairments following an attack may be incorporated into the model, rather than restricting its consequences to the extreme cases of death and complete recovery. This could make the model relevant to the case of brain stroke as well, the risk factors for which overlap heavily with those for a heart attack, but which is more likely to result in significant neurological and cognitive impairments. Finally, self-protection measures which reduce not just the risk of dying from an attack but also the risk of an attack itself, such as aerobic exercising, may be incorporated into the model, contributing to a more comprehensive understanding of the nonadherence phenomenon.

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