The *Other* Ex-Ante Moral Hazard in Health*

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Abstract

It is well known that pooled insurance coverage can induce a form of ex-ante moral hazard: people make inefficiently low investments in self-protective activities. This paper identifies another ex-ante moral hazard that runs in the opposite direction: it causes people to choose inefficiently high levels of self-protection. This *other* ex-ante moral hazard arises through the impact that self-protective activities have on the reward for innovation. Lower levels of self-protection and the associated chronic conditions and behavioral patterns such as obesity, smoking, and malnutrition increase the incidence of many diseases for an individual. This increases the individual's consumption of treatments to those diseases, which increases the reward for innovation that an innovator receives. By the induced innovation hypothesis, which has broad empirical support, the increase in the reward for innovation in turn increases the rate of innovation, which benefits all consumers. As individuals do not take these positive externalities on the innovator and other consumers into account when deciding the level of self-protective activities, they each invest an inefficiently high level in self-protective activities. In the quantitative part of our analysis we show that for obesity the magnitude of this positive innovation externality roughly coincides with the magnitude of the negative Medicare-induced health insurance externality of obesity. The *other* ex-ante moral hazard that we identify can thus be as important as the ex-ante moral hazard that has been a central concept in health economics for decades. The quantitative finding also implies that the current Medicare-induced subsidy for obesity is approximately optimal. Thus the presence of this obesity subsidy is not a sufficient rationale for "soda taxes", "fat taxes" or other penalties on obesity.

Keywords: self-protection; prevention; moral hazard; innovation; induced innovation; reward for innovation; obesity; health insurance; Medicare.

JEL Classification Codes: I10, I18, D62, H23

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1 Introduction

Within economics, it is well-known that pooled insurance coverage can create a disincentive for the insured individual to invest in self-protective activities–a form of ex-ante moral hazard (Ehrlich and Becker, 1972). In health economics it is also well understood that insurance coverage can create also an ex-post moral hazard (Pauly, 1968; Manning et al., 1987). Both the ex-ante moral hazard and the ex-post moral hazard lead to a negative externality: the former causes people to invest insufficiently in self-protection, while the latter causes people to consume health care resources at an inefficiently high level.

In this paper, we identify a distinct second form of ex-ante moral hazard that runs in the opposite direction from the one examined by Ehrlich and Becker (1972). It causes people to devote an inefficiently high level of resources to self-protective activities.

This *other* ex-ante moral hazard arises through the impact that self-protection has on the reward for innovation. Lower levels of self-protective activities such as exercise and healthy diet and the associated chronic conditions and behavioral patterns such as obesity, smoking, and malnutrition increase the incidence of many diseases for an individual. This increases the individual's consumption of treatments to those diseases, which increases the reward for innovation that an innovator receives. By the induced innovation hypothesis, which has broad empirical support (see below), the increase in the reward for innovation in turn increases the rate of innovation of treatments to those diseases. Because consumers capture some of the surplus created by pharmaceutical and other medical innovation, this additional innovation benefits all people who are afflicted with any of those diseases.

A lower level of self-protection thus has two positive external impacts: it directly increases the reward for innovation which benefits the innovator, and it indirectly induces additional innovation which benefits other consumers. Because people do not account for these positive externalities when they decide their levels of self-protection, this mechanism–the *other* ex-ante moral hazard–causes people choose inefficiently high levels of self-protection.

We refer to the combined external effect from a lower level of self-protective activities through the increase in the reward for innovation and through induced innovation as the "innovation externality". As our formal analysis shows, the presence of a positive innovation externality does not rely on the assumption that there is an underinvestment in innovation from the perspective of total surplus, holding the level of self-protective activities constant.[1]

[1] The only case when there is no positive innovation externality is when there is a large enough overinvestment in innovation that the increase in the reward for innovation leads to a decrease in total surplus. Given the empirical evidence on private vs. social returns to R&D (see e.g. Jones and Williams, 1998, and Bloom et al., 2007) it seems very unlikely that this special case applies in practice.

Our analysis concerns the innovation of new goods for which the reward for innovation from each consumer is increasing in the consumer's consumption of the good. Accordingly, the innovator's marginal revenue from any consumer, including the consumer who is marginal in terms of the consumer's level of self-protective activities, is always above the marginal social cost. This gap between marginal revenue and social cost, together with the presence of self-protective activities that influence the intensity of demand, is the impetus for the existence of the *other* ex ante moral hazard and the associated optimal subsidy for lower levels of self-protective activities. This gap between marginal revenue and social cost is also the reason why our analysis differs from the famously erroneous analysis of pecuniary external economies and diseconomies of scale in the production of existing goods by Pigou (1912). Contrary to what Pigou asserted, taxes or subsidies for consumption are not warranted in the cases he examined because the producer's revenue from the marginal consumer is equal to the marginal social cost (see Young, 1913, and e.g. Liebowitz and Margolis, 1995). In contrast, for newly invented goods this marginal revenue and the marginal social cost are different.

The central role of the reward for innovation in our analysis is also a reason why we focus our analysis of the *other* ex-ante moral hazard on health. As is well known, the share of revenue that is reward for innovation (i.e. in excess of marginal costs) is much greater in the pharmaceutical industry than in most if not even all other industries. The potential of the innovation externality to drive a large wedge between the privately and socially optimal levels of self-protective activities is thus greater in health than in any other context.

The economic efficiency consequences of the ex-ante moral hazard examined by Ehrlich and Becker (1972) depend on what extent marginal health care costs are shared through insurance and on how elastic self-protective activities are with respect to the associated benefits. Similarly, the economic efficiency consequences of the ex-ante moral hazard that we identify depend on the size of the innovation externality and on how elastic self-protective activities are with respect to the associated benefits. Unfortunately, it is very hard to obtain reliable measures of how elastic self-protective activities are with respect to the associated benefits and, consequently, the evidence on this central concept in health economics is scant.

For this reason, in terms of quantitative analysis, we limit the scope of this paper to the measurement of the magnitude of the innovation externality and how large it is in comparison with the pooled health insurance externality. The comparison provides an assessment of the relative importance of the two forms of ex-ante moral hazard in health. Moreover, in most economic models of externalities–including the model that we present–the optimal policy depends only on the magnitude of the external effect and is independent of the relevant

behavioral elasticity. Quantifying the innovation externality thus goes a long way toward determining the optimal policy. Measurement of the extent to which marginal health care costs are shared and measurement of the size of the innovation externality are also sufficient to capture the distributional consequences of the two opposing externalities which are also of interest.

While the innovation externality and the associated *other* ex-ante moral hazard apply to health behavior in general, we present the analysis in the context of obesity, which is known to increase the prevalence of many diseases and the associated medical expenditures. This focus enables us to keep the analysis concrete and efficiently quantify the innovation externality of obesity to demonstrate that the *other* ex-ante moral hazard is also quantitatively important.

In the theoretical part of our analysis we present a model which allows us to characterize the magnitude of the innovation externality of obesity in terms of straightforward and empirically malleable economic concepts. The quantitative part of our analysis shows that the magnitude of the positive innovation externality of obesity roughly coincides with the negative Medicare-induced health insurance externality of obesity. From a theoretical perspective this finding implies that the *other* ex-ante moral hazard that we identify can be quantitatively as important as the ex-ante moral hazard examined by Ehrlich and Becker (1972) which has been a central concept in health economics for decades. From a policy perspective this finding implies that in the U.S. the current (Medicare-induced) subsidy for obesity is approximately optimal for people who are covered with private insurance before old-age, and thus the presence of this subsidy is not a sufficient rationale for imposing "fat taxes", "soda taxes" or other penalties on obesity.

The balance of this paper proceeds in the standard order–literature review, theory, data, quantitative application, and conclusion.

2 Related Literature

2.1 Obesity, Disease, and Health Expenditures

Americans are increasingly overweight or obese. The proportion of adults classified as obese increased from 12.0% in 1991 to 20.9% in 2001 (Mokdad et al., 1999, 2003; Wang and Beydoun, 2007).

Obesity is associated with an increased risk of a range of chronic conditions, including diabetes, hypertension, heart disease, and stroke (Kasper et al., 2004). In some cases, there are solid biochemical and physiological reasons to suppose that the association is causal,

such as in the case of diabetes. In other cases, the evidence is murkier. Here, we do not attempt to settle (nor are we capable of settling) the debate over which of these relationships are causal. Instead, our aim is to show that if the effect of obesity on disease prevalence is causal and obesity therefore has a negative Medicare-induced health insurance externality then obesity has also a positive innovation externality of roughly equal magnitude. Because either externality is present only for diseases for which the relationship is causal, the extent to which the relationships are causal is unlikely to significantly change the relative comparison of the two opposing externalities of obesity. For this reason we are comfortable with limiting the scope of our analysis to not include an analysis of to what extent the associations between obesity and disease prevalence represent causal effects.

Not surprisingly, also expected health care expenditures are higher for obese individuals than for normal weight individuals. A large number of studies document this fact. The vast majority of these studies use convenience samples consisting of individuals from a single employer or a single insurer (Elmer et al., 2004; Bertakis and Azari, 2005; Burton et al., 1998; Raebel et al., 2004). There are a few studies that use nationally representative data. Finkelstein et al. (2003) use data from the linked National Health Interview Survey (NHIS) and Medical Expenditure Panel Survey (MEPS) to estimate that annual medical expenditures are $732 higher for obese than normal weight individuals.[2] Sturm (2002), using data from the Health Care for Communities (HCC) survey, finds that obese individuals spend $395 per year more than non-obese individuals on medical care.

This is a large literature, which space constraints prevent us from surveying in more detail. The many studies that we do not discuss here vary considerably in generality but they all reach the same qualitative conclusion that obesity is associated with higher medical care costs.[3] None of this literature attempts to address whether the relationship between obesity and associated health care expenditures are causal. We do not attempt to settle this issue here and, for the same reasons outlined above on the link between obesity and disease

[2] On an aggregate level, approximately half of the estimated $78.5 billion in medical care spending in 1998 attributable to excess body weight was financed through private insurance (38%) and patient out-of-pocket payments (14%). Thorpe et al. (2004) use MEPS data to estimate how much of the $1,100 increase between 1987 and 2000 in per-capita medical expenditures is attributable to obesity. Using a regression model to calculate what per-capita medical expenditures would have been had 1987 obesity levels persisted to 2000, they conclude that about $300 of the $1,100 increase is due to the rise in obesity prevalence.

[3] Some of the studies we reviewed, but arbitrarily do not discuss here include Bungam et al. (2003), Musich et al. (2004), Quesenberry et al. (1998), Thompson et al. (2001) and Wang et al. (2003). There are also studies of obesity-related medical expenditure differences in an international setting. Both Sander and Bergemann (2003), in a German setting, and Katzmarzyk and Janssen (2004), in a Canadian setting, find higher medical expenditures for obese people. The analysis by Michaud et al. (2009) takes also into account the impact of obesity on longevity.

prevalence, we do not need to settle it.

2.2 Health Insurance, *Ex Ante* Moral Hazard, Induced Innovation

That obesity is associated with higher health care expenditures is only a necessary first step in establishing the traditional *ex-ante* welfare loss from obesity through health insurance. In the case of employer-provided health insurance, for instance, Bhattacharya and Bundorf (2005) show that differences in wages between obese and non-obese workers with employer-provided health insurance undo nominal risk pooling between the workers. Without pooling, there is no health insurance externality from obesity. This argument does not extend to public insurance, such as Medicare, where there is clearly pooling and the associated transfer from thinner to heavier individuals and no wage mechanism to undo it. Even in the case of public insurance, though, obese individuals are likely to pay higher out-of-pocket medical expenditures because of cost-sharing in insurance coverage. Being obese therefore imposes costs on the person holding the weight.

Bhattacharya and Sood (2007) show that, in pooled health insurance, if the elasticity of body weight with respect to the transfer from thinner to heavier individuals (induced by insurance) is zero, there is no welfare loss from the *ex-ante* externality. Unless the subsidy induced by insurance causes someone to become heavier, the insurance transaction is a costless transfer. With the exception of Rashad and Markowitz (2006), there has been little work attempting to measure the size of this key elasticity.

We are not aware of any work that has identified the *other* ex-ante moral hazard that we examine or has attempted to estimate the size of the associated innovation externality. The closest related study is Lakdawalla and Sood (2007) who examine the effect of extending drug insurance on welfare through induced innovation. In comparison, we focus on the ex-ante moral hazard effect of induced innovation.

The induced innovation hypothesis was first examined by Hicks (1932) and Schmookler (1966). Empirical investigations of this hypothesis in the pharmaceutical industry include Acemoglu and Linn (2004), Finkelstein (2004), Lichtenberg and Waldfogel (2003), and Yin (2008), which all find support for the hypothesis. Moreover, in Bhattacharya and Packalen (2008a), in which the main focus is on the determinants of the direction of academic medical research, we find evidence of obesity-induced pharmaceutical innovation: obesity-epidemic induced increases in the prevalence of diseases is associated with increases in the introduction of pharmaceutical drugs that treat those diseases. Support for the induced innovation hypothesis is not limited to health care, as Newell et al. (1999) and Popp (2002) find support

for the induced innovation hypothesis in the energy sector.[4]

3 Theory

In this section we first present the model and characterize the equilibrium. We then determine the optimal subsidy implied by the innovation externality from a lower level of self-protection. Optimal policy is solved in terms of both consumer and total surplus. In the former case the optimal subsidy reflects only the indirect induced innovation effect. In the latter case the optimal subsidy reflects also the direct impact on the reward for innovation. In Section 3.4 we discuss several aspects which are not included in the model but are taken into account in the quantitative application.

3.1 The Model

Agents in the model consist of an innovator and N consumers. Decisions are made in three stages. In stage 1 consumers simultaneously and non-cooperatively choose their level of prevention (self-protective activities). In stage 2 first the innovator chooses the level of its R&D investments which determines the probability μ that the innovator is successful in developing a new medical care technology. Subsequently in stage 2 the success of these R&D investments and the health status of each consumer is revealed. In stage 3 consumers choose the level of medical care.

3.1.1 Consumers

In stage 1 each consumer faces a trade-off between prevention and leisure, which we denote by S and L, respectively. The consumer resource constraint in stage 1 is

$$S + L = H, \tag{1}$$

where H is the resource endowment in stage 1. For expositional convenience we assume that there are only two levels of prevention, high and low, which we denote by S_{NORMAL}

[4] Our analysis is also related to the studies on preference externalities by Waldfogel (2003) and George and Waldfogel (2003), which build on the theoretical contributions by Hotelling (1929), Spence (1976a,b) and Dixit and Stiglitz (1977), and which these examine the impacts of (racial) population characteristics on product variety (in newspapers and radio programming) through a market size effect. In contrast, we examine the effects of population characteristics on welfare through the innovation externality. Furthermore, in our case the preference externality is determined by consumers' decisions rather than inherent characteristics (to extent that body weight is in fact a decision).

and S_{OBESE}, respectively, and that the opportunity cost of increasing the level of prevention from low to high is one unit of leisure, which is formally stated as[5]

$$S_{NORMAL} - S_{OBESE} = 1. \tag{2}$$

We denote the number of individuals who choose the low level of prevention by n_{OBESE}.

Choosing the lower level of prevention leads to chronic conditions and behavioral patterns such as obesity, smoking, or malnutrition, which increase the probability of illness. We assume that prevention only influences each consumer's own probability of illness. Consumers who choose the high level of prevention have the probability of illness π_{NORMAL}. Consumers who choose the low level of prevention have the elevated probability of illness $\pi_{OBESE} > \pi_{NORMAL}$. The average probability of illness in the population (disease prevalence) is

$$\pi_{AVERAGE} = \pi_{NORMAL} + \frac{n_{OBESE}}{N} \times (\pi_{OBESE} - \pi_{NORMAL}). \tag{3}$$

Consumers who choose the low level of prevention and thereby have the elevated probability of illness π_{OBESE} receive a subsidy t. We refer to the subsidy t as the "obesity subsidy" in part to emphasize the fact that subsidizing lower levels of prevention requires the presence of an observable proxy variable for the level of prevention. The subsidy t is financed through a lump-sum tax T on all consumers. The budget balancing condition is

$$n_{OBESE} \times t = N \times T. \tag{4}$$

The obesity subsidy t and the lump-sum tax T enter the stage 3 resource constraint.

Let W denote the resource endowment in stage 3. In stage 3 consumers face a trade-off between consumption of medical care and consumption of other goods, which we denote by M and C, respectively. For illl consumers who purchase medical care and choose the low level of prevention the resource constraint in stage 3 is

$$M + C = W - T + t. \tag{5}$$

[5]In the model we do not include the possibility of innovations that decrease the relative cost of prevention. While some such preventative innovation does occur in the form of lower-calorie foods, diets, nutritional supplements, exercise machines, and so forth, we are not aware of arguments that would place the qualitative importance of such preventative innovation anywhere near the importance of the type of disease-driven treatment innovation that we model. Dranove (1988) observes that for many forms of prevention innovation patents are unavailable and property rights are undefined, and conjectures that in the U.S. prevention innovation receives only a tiny percentage of medical R&D dollars. Accordingly, we conjecture that innovation externality from the consumption of such preventative innovations are likely small relative to the innovation externality from the consumption of the type of disease-driven treatment innovations that we model.

For ill consumers who purchase medical care and choose the high level of prevention the resource constraint in stage 3 is

$$M + C = W - T. \tag{6}$$

For healthy consumers the corresponding budget constraints are simply

$$C = W - T + t \tag{7}$$

and

$$C = W - T. \tag{8}$$

To simplify the analysis, we rely on assumptions which imply that all ill consumers purchase medical care. These assumptions are introduced next.

Consumer utility is influenced in part by the consumer's choices in stages 1 and 3 and health status in stage 3. The relative cost of prevention is captured by the parameter θ, which measures the marginal utility of leisure and is heterogenous across consumers. For each consumer the value of the parameter θ is drawn from the distribution represented by the cumulative distribution function $F(\theta)$ for which $F'(\theta) > 0$ for all $\theta \in [0, \bar{\theta})$, where $\bar{\theta} \in R^{+}$.

Denoting the utility loss from illness by D, the utility function is

$$U(L, C, D) = \theta L + C + D.$$

For healthy consumers $D = 0$ and the utility is

$$U(L, C, D) = \theta L + C. \tag{9}$$

When the innovator is unsuccessful, consumers can only purchase old medical care technology, which reduces the utility loss from illness to D_0. Hence, for ill consumers who purchase the old medical care technology the utility is

$$U(L, C, D) = \theta L + C - D_0. \tag{10}$$

When the innovator is successful, consumers can alternatively purchase the new medical care technology which further reduces the utility loss from illness to $D_1 < D_0$. Hence, for ill consumers who purchase the new medical care technology the utility is

$$U(L, C, D) = \theta L + C - D_1. \tag{11}$$

For expositional convenience we assume that the price of the old technology is zero, and that the innovator's production costs–unlike its R&D costs–are zero. Hence, subtracting the right-hand side of the expression (10) from the right-hand side of the expression (11) reveals that each ill consumer's willingness to pay for the new technology is $D_0 - D_1$, and this willingness to pay is also the ex-post surplus from innovation. We denote the innovator's share of this surplus by s. The price of the new medical care technology is thus

$$M = s\left(D_0 - D_1\right). \tag{12}$$

We assume that consumers are risk-neutral.[6] Combining expression (12), the definitions of the probability of innovation μ and the probability of illness π_{OBESE}, the stage 1 resource constraint (1), the stage 3 resource constraints (5) and (7) for consumers who choose the low level of prevention, and expressions (9), (10) and (11) for utility yields the expression

$$\theta\left(H - S_{OBESE}\right) + W - \pi_{OBESE}\left[D_0 - \mu\left(1 - s\right)\left(D_0 - D_1\right)\right] + t - T \tag{13}$$

for the expected utility of a consumer with the low level of prevention. Similarly, combining expression (12), the definitions of μ and π_{NORMAL}, the stage 1 resource constraint (1), the stage 3 resource constraints (6) and (8) for consumers who choose the high level of prevention, and expressions (9), (10) and (11) for utility yields the expression

$$\theta\left(H - S_{NORMAL}\right) + W - \pi_{NORMAL}\left[D_0 - \mu\left(1 - s\right)\left(D_0 - D_1\right)\right] - T \tag{14}$$

for the expected utility of a consumer with the high level of prevention.

In the above expressions (13) and (14) for expected utility the first term represents the utility from leisure, and the rest of each expression represents the impacts of consumption and illness on utility. The factor $D_0 - \mu\left(1 - s\right)\left(D_0 - D_1\right)$ in the third term in both expressions (13) and (14) measures the utility loss from an illness. We denote this cost of illness by

$$C_{ILLNESS} \equiv D_0 - \mu\left(1 - s\right)\left(D_0 - D_1\right). \tag{15}$$

[6]This increases the tractability of the analysis and the ease of exposition. Moreover, this assumption enables us to abstract from insurance and the associated potential for ex-post moral hazard. There are two reasons why we believe that abstracting from the ex-post moral hazard induced by obesity is innocuous here. First, the elasticity of demand for health care is larger (in absolute value) for those without chronic conditions (Manning et al., 1987; Bajari et al., 2006). Second, Lakdawalla and Sood (2006) show that when it comes to pharmaceutical expenditures–which we examine in our empirical application–there may not be any *ex-post* moral hazard at all as co-payments make out-of-pocket prices close to marginal cost.

Subtracting expression (14) from expression (13) and substituting $S_{NORMAL} - S_{OBESE} = 1$ shows that a consumer chooses the high level of prevention if and only if the condition

$$-\theta - t + (\pi_{OBESE} - \pi_{NORMAL}) \times C_{ILLNESS} \geq 0 \tag{16}$$

holds.[7] The first two terms in this expression (16) represent the cost of the high level of prevention in terms of effort and the loss of the obesity subsidy, respectively. The third term measures the benefit from the high level of prevention in the form of a lower probability of illness.

We denote the combined illness benefit and monetary penalty from the high level of prevention by

$$B_{NORMAL} \equiv (\pi_{OBESE} - \pi_{NORMAL}) \times C_{ILLNESS} - t. \tag{17}$$

Comparing this definition (17) of B_{NORMAL} with the condition (16) for the optimal prevention choice shows that individuals with $\theta \leq B_{NORMAL}$ choose the high level of prevention. The share of consumers $\frac{n_{OBESE}}{N}$ who choose the low level of prevention is therefore given by[8]

$$\frac{n_{OBESE}}{N} = 1 - F\left(B_{NORMAL}\right). \tag{19}$$

To measure the responsiveness of the share of consumers $\frac{n_{OBESE}}{N}$ who choose the low level of prevention to changes in the cost B_{NORMAL} of choosing the low level of prevention, we employ the concept the *cost-elasticity of obesity*, which is defined as

$$\varepsilon_{OBESE} \equiv -\frac{dn_{OBESE}}{dB_{NORMAL}} \frac{B_{NORMAL}}{n_{OBESE}}. \tag{20}$$

[7] Analogous to most analyses of externalities, we assume that the number of consumers N is large enough so that the impact that each consumer's own prevention decision has on its own expected utility through the impact on the probability of innovation μ can be ignored in deriving the condition (16) for the optimal prevention decision. The total induced innovation benefit of a single consumer's prevention decision, which is dispersed across all consumers, is still non-negligible provided that $s \in (0, 1)$.

[8] We assume that the number of consumers N is large enough so that the distribution function $F(\theta)$ is a good approximation of the distribution of the actual realizations of the parameter θ, which allows us to employ the distribution function $F(\theta)$ in the consumer optimum condition (19).

To avoid discussion of boundary equilibria in which either $\frac{n_{OBESE}}{N} = 0$ or $\frac{n_{OBESE}}{N} = 1$ without any obesity subsidy, we assume that the cumulative distribution $F(\theta)$ is such that when there is no subsidy, $t = 0$, the share of consumers who choose the low level of prevention is always positive but less than one regardless of the values s and μ. Formally, we assume that the conditions

$$1 - F\left[\left(\pi_{OBESE} - \pi_{NORMAL}\right) D_0\right] > 0 \qquad \text{and} \qquad 1 - F\left[\left(\pi_{OBESE} - \pi_{NORMAL}\right) D_1\right] < 1 \tag{18}$$

hold.

Combining the condition $\theta \leq B_{NORMAL}$ for the optimal prevention choice and expressions (13) and (14) for expected utility with low and high levels of prevention, respectively, as well as the budget balancing condition (4), yields the expression

$$A - N \times \int_0^{B_{NORMAL}} \theta F'(\theta)\, d\theta - N \times \pi_{AVERAGE} \times C_{ILLNESS} \tag{21}$$

for total expected consumer surplus, where $A \equiv N\left[(H - S_{OBESE}) \int_\theta \theta F'(\theta)\, d\theta + W\right]$ is a constant. The parameters t and s thus only influence consumer welfare through the utility loss from the high level of prevention and the utility loss from illness, which are reflected by the second and third terms in expression (21), respectively.

3.1.2 The Innovator

The innovator can increase its probability of success μ by increasing its R&D expenditures. One possible representation of the innovator's R&D cost function $C(\mu)$ is

$$C(\mu) = c_F + a\mu + \frac{b}{2}\mu^2, \tag{22}$$

where c_F, a, and b are parameters with $a > 0$ and $b > 0$. The assumption $a > 0$ implies that the marginal cost of increasing the probability of success is positive and captures the notion that increasing the probability of success requires additional resources. The assumption $b > 0$ implies that also the marginal cost of increasing the probability of success is increasing in the probability of success and captures the notion that it is increasingly more difficult to increase the probability of success due to the scarcity of fertile research ideas.

Given the expression (12) for the price of the new medical care technology and the expression (3) for the average probability of illness $\pi_{AVERAGE}$, the innovator's expected reward for success, which we denote by R, is

$$R = N \times \left[\pi_{NORMAL} + \frac{n_{OBESE}}{N} \times (\pi_{OBESE} - \pi_{NORMAL})\right] \times s \times (D_1 - D_0). \tag{23}$$

The innovator chooses the level of its R&D investment to maximize its expected profit

$$\Pi(\mu) = \mu R - C(\mu). \tag{24}$$

Given the cost function (22), the innovator's optimal probability of innovation is

$$\mu = \frac{a}{b} + \frac{1}{b}R, \tag{25}$$

provided that for the optimal μ given in this expression (25) the properties $\Pi(\mu) \geq \Pi(0)$ and $\mu \in [0,1]$ hold. This result (25) shows that for the quadratic cost function (22) the probability of innovation is increasing in the reward for success R. This result of course holds for cost functions more generally. Accordingly, below we rely on the more general reduced-form relationship

$$\mu = G(R), \tag{26}$$

where $G(R)$ is a differentiable function with $G'(R) > 0$, to capture the positive relationship between the probability of innovation μ and the reward for innovation R.[9]

A key determinant of the size of the induced innovation effect from self-protective activities is how responsive the rate of innovation is to changes in the reward for innovation. Accordingly, we frequently rely on the concept the *reward-elasticity of innovation*, which is defined as

$$\varepsilon_{\mu} \equiv \frac{d\mu}{dR}\frac{R}{\mu}. \tag{30}$$

The use of the reward-elasticity of innovation concept has two advantages. First, it is a more intuitive concept than a specific cost function. Second, the reward-elasticity of innovation is the parameter of interest in the empirical analyses of induced innovation.

[9] To avoid discussion of boundary equilibria in which the reward for innovation is either so small or so large that the probability of innovation is unresponsive to changes in the reward for innovation we assume that

$$G'(R) > 0 \text{ for all } R \geq 0 \qquad \text{and} \qquad G(\pi_{OBESE}(D_1 - D_0)) < 1, \tag{27}$$

where $\pi_{OBESE}(D_1 - D_0)$ is the highest possible value of the reward for innovation R when $t = 0$.

To avoid discussion of equilibria in which the probability of innovation is high enough without any reward for innovation so that the consumer surplus maximizing optimum is to set $s = 0$, we assume that

$$G(0) \in [0, \delta), \tag{28}$$

where δ is a small enough positive constant.

To avoid discussion of cases in which it is optimal from the total surplus perspective to set the obesity subsidy so high that all consumers choose the low level of prevention we assume that

$$\tilde{\mu} \times (1 + \varepsilon_{\tilde{\mu}}) \times \frac{D_0 - D_1}{D_0} < 1 \tag{29}$$

where $\tilde{\mu}$ denotes the probability of innovation when all consumers choose the low level of prevention, i.e. $\frac{n_{OBESE}}{N} = 1$, and $\varepsilon_{\tilde{\mu}}$ denotes the reward-elasticity of innovation when $\mu = \tilde{\mu}$. This condition (29) holds if this probability of innovation $\tilde{\mu}$ is sufficiently small, or if reward-elasticity of innovation $\varepsilon_{\tilde{\mu}}$ is sufficiently small, or if the innovation is incremental enough so that $\frac{D_0 - D_1}{D_0}$ is small enough.

3.2 Equilibrium

The endogenous variables are the probability of innovation μ and the share of consumers $\frac{n_{OBESE}}{N}$ with the low level of prevention. A Subgame-Perfect Nash Equilibrium satisfies the innovator optimum condition (26) and the consumer optimum condition (19), which can be rewritten as

$$\mu = G\left(N \times \left[\pi_{NORMAL} + \frac{n_{OBESE}}{N}\left(\pi_{OBESE} - \pi_{NORMAL}\right)\right] \times s \times \left(D_1 - D_0\right)\right) \tag{31}$$

and

$$\frac{n_{OBESE}}{N} = 1 - F\left[\left(\pi_{OBESE} - \pi_{NORMAL}\right) \times \left[D_0 - \mu\left(1 - s\right)\left(D_0 - D_1\right)\right] - t\right], \tag{32}$$

respectively.

Assumptions (18), (27), and (28) imply that when there is no obesity subsidy, $t = 0$, the consumer and innovator optimum conditions (31) and (32) intersect, and there thus exists an equilibrium that satisfies both conditions. Assumptions (18), (27), and (28) are also sufficient for there to exist an equilibrium that satisfies conditions (31) and (32) when the obesity subsidy t is set optimally because there is no gain from increasing this subsidy past the point at which all consumers choose the low level of prevention and the conditions (31) and (32) still hold.

A sufficient condition for the equilibrium to be unique and stable is that in any equilibrium the product of the slopes of the consumer and innovator optimum conditions (31) and (32) is less than one. In the appendix we show that this sufficient condition holds if

$$\varepsilon_\mu \times \varepsilon_{OBESE} \times \frac{\pi_{OBESE} - \pi_{NORMAL}}{\pi_{AVERAGE}} \times \frac{D_0 - D_1}{D_0} < 1 \tag{33}$$

holds in any equilibrium. This condition (33) holds if either of the two elasticities–the reward-elasticity of innovation ε_μ or the cost-elasticity of obesity ε_{OBESE}–is small enough, or if the impact of obesity on disease prevalence, $\frac{\pi_{OBESE} - \pi_{NORMAL}}{\pi_{AVERAGE}}$, is small enough, or if the innovation is incremental enough in the sense that $\frac{D_0 - D_1}{D_0}$ is small enough. In what follows we assume that condition (33) holds so that the equilibrium is unique, stable, and given by the equilibrium conditions (31) and (32).

3.3 Optimal Policy

We determine the optimal obesity subsidy t^* for the lower level of prevention both using the consumer surplus maximizing approach and the total surplus maximizing approach. In the

consumer surplus maximizing approach we assume that also the parameter s, which governs the division of ex-post surplus from innovation, is set to maximize consumer welfare. One important policy instrument that can be used to influence the parameter s in practice is patent duration. In the total surplus maximizing approach the lower bound for the optimal obesity subsidy is independent of the parameter s. In the total surplus maximizing approach we solve for the optimal obesity subsidy t^* for an arbitrary value of the parameter s.

The conclusion from both approaches is consistent with the conclusion that the optimal obesity subsidy t should be set no lower than the impact that choosing the lower level of prevention has on the reward for innovation. Formally, this derived lower bound for the optimal obesity subsidy t^* in the model is

$$t^* = \mu \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_1 - D_0)\,. \tag{34}$$

We base our quantitative analysis of the optimal obesity subsidy on this lower bound (34) for the optimal subsidy rather than on the exact total surplus maximizing obesity subsidy for three reasons. First, because the lower bound (34) for the optimal subsidy is also the consumer surplus maximizing obesity subsidy the quantitative results obtained using expression (34) are robust to the selection of the welfare criterion.

Second, from the perspective of total surplus the optimal obesity subsidy t^* is always at least (34) regardless of one's beliefs about how the parameter s is set. Thus, the analysis of the optimal obesity subsidy t^* is applicable even if one believes that the parameter s is not set to maximize total surplus due to, for example, political economy considerations.

Third, use of the exact expression for the total surplus maximizing obesity subsidy in the quantitative analysis would require calibrating values of the reward-elasticity of innovation ε_μ and the ratio of consumer and innovator surplus from innovation $\frac{1-s}{s}$. However, reliable and comprehensive estimates of these two parameters are not available. In terms of calibrating the parameter ε_μ it is important to keep in mind that empirical studies of induced innovation generally rely on a difference-in-difference methodology which yields estimates of the reward-elasticity of the composition of innovation rather than the parameter ε_μ which measures the reward-elasticity of the total extent of innovation. The point that the two reward-elasticities can be very different has been emphasized by Acemoglu and Linn (2004).

3.3.1 Consumer Surplus Maximizing Policy

Given expression (21) for consumer welfare, the consumer surplus maximizing approach solves

$$\max_{s,t} \left\{ -N \times \int_0^{B_{NORMAL}} \theta F'(\theta)\, d\theta - N \times \pi_{AVERAGE} \times C_{ILLNESS} \right\}. \tag{35}$$

The following result describes the optimum, which we denote by (s^*_{CS}, t^*_{CS}).

Proposition 1. *In the consumer surplus maximizing solution, neither consumers nor the innovator capture all of the ex-post surplus from innovation, and the optimal obesity subsidy is equal to the increase in the reward for innovation from the lower level of prevention. Formally,* $s^*_{CS} \in (0,1)$ *and* $t^*_{CS} = \mu \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_1 - D_0)$.

Proof. See the appendix.

To explain the intuition for the result on the optimal obesity subsidy t^*_{CS} we first need to discuss the optimum condition for s^*_{CS}.

Holding the probability of innovation constant, an increase in the innovator's share s of the ex-post surplus from innovation decreases expected consumer surplus.[10] An increase in the parameter s also influences consumer welfare through its impact on the probability of innovation. The parameter s influences the probability of innovation because it in part determines the reward for innovation.

Generally, an increase in the parameter s has both a direct and an indirect impact on the the reward for innovation. The direct impact is represented by the presence of the parameter s in expression (23) for the reward for innovation. The indirect impact arises through the impact that a change in the parameter s has on the number of individuals n_{OBESE} who choose the low level of prevention. This indirect impact is represented by the presence of the variable n_{OBESE} in expression (23) for the reward for innovation. However, because the cost of an illness is minimized at the consumer surplus maximizing optimum s^*_{CS}, and because the cost of an illness determines how many consumers choose the low level of prevention, at the optimum s^*_{CS} small changes in s have only a second-order impact on the number of consumers n_{OBESE} who choose the low level of prevention. The indirect effect of an increase in the parameter s on the reward for innovation can therefore be ignored when considering the optimum condition for s^*_{CS}.

[10] The property $s^*_{CS} > 0$, which means that consumers do not capture all of the ex-post surplus from innovation, follows directly from assumption (28) according to which the probability of innovation is small enough (or even zero) when the reward for innovation is zero. The property $s^*_{CS} < 1$, which means that consumers capture at least some of the ex-post surplus from innovation, follows from the fact that consumers benefit from innovation whenever $s \in (0,1)$ but do not benefit from innovation at all when $s = 1$.

Hence, at the consumer surplus maximizing optimum for the parameter s, a marginal increase Δs in the parameter s decreases consumer surplus by $\frac{\Delta s}{1-s}$ percent, holding the probability of innovation constant. The marginal increase Δs in the parameter s simultaneously increases the reward for innovation by $\frac{\Delta s}{s}$ percent, which increases the probability of innovation–and thereby also the benefit from innovation, holding the consumer's share $(1-s)$ of the ex-post surplus from innovation constant–by $\varepsilon_\mu \times \frac{\Delta s}{s}$ percent, where ε_μ is the reward-elasticity of innovation defined in expression (30). At the optimum s^*_{CS} these two impacts are equal,

$$\varepsilon_\mu \times \frac{1}{s^*_{CS}} = \frac{1}{1-s^*_{CS}}. \tag{36}$$

Intuitively, at the optimum s^*_{CS} any transfer from consumers to the innovator in the form of an increase in the reward for innovation must bring the consumers an equal benefit in the form of the benefit from additional innovation.

To derive the intuition for the optimal obesity subsidy t^*_{CS}, we now consider the external impact of a marginal increase in the number of consumers n_{OBESE} who choose the low level of prevention. When one consumer switches to the low level of prevention, the reward for innovation increases by

$$\frac{\pi_{OBESE} - \pi_{NORMAL}}{N \times \pi_{AVERAGE}} \tag{37}$$

percent. By the definition (30) of the reward-elasticity of innovation ε_μ this increase in the reward for innovation increases the extent of innovation by

$$\varepsilon_\mu \times \frac{\pi_{OBESE} - \pi_{NORMAL}}{N \times \pi_{AVERAGE}} \tag{38}$$

percent. As the expected total consumer surplus from innovation is

$$N \times \pi_{AVERAGE} \times \mu \times (1-s) \times (D_0 - D_1), \tag{39}$$

the impact of the increase in innovation on the expected consumer surplus is

$$\varepsilon_\mu \frac{\pi_{OBESE} - \pi_{NORMAL}}{N \times \pi_{AVERAGE}} \times N \times \pi_{AVERAGE} \times \mu \times (1-s) \times (D_0 - D_1), \tag{40}$$

which can be rewritten as

$$\varepsilon_\mu \times \frac{1-s}{s} \times \mu \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_0 - D_1). \tag{41}$$

Applying the property (36) of the optimal s^*_{CS} allows us to rewrite this expression (41) as

$$\mu \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_0 - D_1) . \tag{42}$$

This expression (42) for the external effect of a marginal increase in the number of individuals n_{OBESE} who choose the low level of prevention is the same as the expression for the optimal obesity subsidy t^*_{CS} in Proposition 1. As expected, because the parameter s was set optimally, an increase in the reward for innovation from the marginal consumer who switches to the low level of prevention brings an equal increase in total expected consumer surplus to all consumers in the form of the benefit from additional innovation.

3.3.2 Total Surplus Maximizing Policy

We first solve for the optimal obesity subsidy t for an arbitrary value of the parameter s, and then briefly discuss the optimal value of the parameter s. Given the expression (21) for consumer welfare and the expression (24) for the innovator's expected profit, the total surplus maximizing approach solves

$$\max_t \left\{ \mu R - C(\mu) - N \times \int_0^{B_{NORMAL}} \theta F'(\theta)\, d\theta - N \times \pi_{AVERAGE} \times C_{ILLNESS} \right\} . \tag{43}$$

The following result describes the optimum, which we denote by t^*_{TS}.

Proposition 2. *In the total-surplus maximizing solution, the optimal obesity subsidy is larger than the increase in the reward for innovation from the lower level of prevention if consumers and the innovator both capture a strictly positive share of the ex-post surplus from innovation, and the optimal obesity subsidy is equal to the increase in the reward for innovation from the lower level of prevention if the innovator captures the entire ex-post surplus from innovation. Formally,* $t^*_{TS} = \mu \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_1 - D_0) + \varepsilon_\mu \times \frac{1-s}{s} \times \mu \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_1 - D_0)$.

Proof. See the appendix.

The first term in the expression for the optimal obesity subsidy t^*_{TS} in Proposition 2 is the externality on the innovator. The size of this externality from a marginal increase in the number of consumers n_{OBESE} who choose the low level of prevention is equal to the associated increase in the reward for innovation from the consumer to the innovator. The second term in the expression for the optimal obesity subsidy t^*_{TS} in Proposition 2 is the

externality on other consumers. As expected, the expression for this part of the externality is the same as the expression (41) for the externality on other consumers in the consumer surplus maximizing case, which was derived before placing the restriction $s = s^*_{CS}$ on the parameter s.

Consider now the total surplus maximizing value s^*_{TS} of the parameter s. In our model $s^*_{TS} < 1$ so that in the optimum the innovator does not capture all of the ex-post surplus from innovation. This is in contrast with most models of innovation which typically do not consider the impact that the division of ex-post surplus from innovation has on prevention or other consumer investments that influence consumer demand. The intuition for the result $s^*_{TS} < 1$ is the following. When $s = 1$ consumers do not receive any surplus and thus a marginal decrease in parameter s does not decrease consumer surplus through its negative impact on the probability of innovation. Instead, at $s = 1$, a marginal decrease in the parameter s merely redistributes surplus from the innovator to consumers and, in addition, increases consumer surplus through the effect that the decrease in s has on the number of consumers n_{OBESE} who choose a low level of prevention. This implies that $s^*_{TS} < 1$.

It can also be shown that $s^*_{TS} > s^*_{CS}$, although we omit the formal proof here. The intuition for the result $s^*_{TS} > s^*_{CS}$ is the following. At $s = s^*_{CS}$, a small change in the parameter s has only a second-order impact on consumer surplus. Moreover, as was discussed above in Section 3.3.1, at $s = s^*_{CS}$ a small change in the parameter s has only a second-order impact on the number of consumers n_{OBESE} who choose the low level of prevention. Consequently, at $s = s^*_{CS}$ an increase always increases the reward for innovation. The innovator's surplus is obviously increasing in the reward for innovation and, therefore, at s^*_{CS} the total surplus is increasing in s. This is, intuitively, the reason for the result $s^*_{TS} > s^*_{CS}$.

The result $s^*_{TS} \in (s^*_{CS}, 1)$ and Proposition 2 together imply that when both policy parameters s and t are set to maximize total surplus, the optimal obesity subsidy t is strictly larger than the impact that a marginal increase in the number of individuals n_{OBESE} that choose the low level of prevention has on the reward for innovation.

3.4 Estimation of the Optimal Obesity Subsidy

The expression (34) for the lower bound of the optimal obesity subsidy is equal to the impact of obesity on expected medical expenditures. The optimal obesity subsidy is thus

$$t^* = E\left(obese\right) - E\left(normal\right), \tag{44}$$

where $E\left(obese\right)$ and $E\left(normal\right)$ denote the medical expenditures for an obese person and for a normal weight person, respectively. This formulation (44) of the optimal subsidy is also advantageous because it allows obesity to impact both disease incidence and the intensity of medical expenditures for an illness.

3.4.1 Incorporating Marginal Costs

For expositional convenience in the formal model we have ignored marginal production and marketing costs as well as the fact that not all medical expenditures are spent on patent protected goods. A consideration of these aspects lowers the impact that a marginal increase in obesity has on the reward for innovation and the associated lower bound for the optimal obesity subsidy from expression (44) to

$$t^{*}=\left[E\left(obese\right)-E\left(normal\right)\right]\times R_{PATENT}\times\left(1-R_{MC}\right), \tag{45}$$

where R_{PATENT} is the share of medical care expenditures that are spent on patent protected (and previously patent protected brand-name) goods and R_{MC} is the share of medical care expenditures that covers marginal production and marketing costs.[11]

3.4.2 Age-Specific Medical Expenditures and Obesity Impacts

As is well known, health expenditures vary greatly by age. Moreover, as can be seen from our quantitative application below, also the impact of obesity on health care expenditures varies greatly by age. We thus calculate the optimal subsidy separately for each age group using the expression

$$t_{t}^{*}=\left[E_{t}\left(obese\right)-E_{t}\left(normal\right)\right]\times R_{PATENT}\times\left(1-R_{MC}\right), \tag{46}$$

where the subscript t denotes a specific age group. In Section 4 we use this expression (46), calibrated values of the parameters R_{PATENT} and R_{MC}, and estimates of the impact $E_{t}\left(obese\right)-E_{t}\left(normal\right)$ of obesity on pharmaceutical expenditures for each age group to

[11] Provided that both parameters s and t are set optimally (in terms of either consumer or total surplus), the result (34) for the lower bound of the optimal obesity subsidy does not change if one also takes into account the fact that while medical care innovation has world-wide benefits, the objective in U.S. policy is more likely set in terms of U.S. welfare as opposed to world-wide welfare (see the earlier version of this paper (Bhattacharya and Packalen, 2008b)). The parameter s is still set at the value for which a marginal increase in the reward for innovation from the relevant sub-population yields an equal increase in the benefit from innovation to this sub-population and, consequently, the optimal obesity subsidy t is still equal to (in the consumer surplus maximizing case) or greater than (in the total surplus maximizing case) to the impact that a marginal increase in obesity has on the reward for innovation.

obtain an estimate of the lower bound for the innovation externality of obesity from pharmaceutical innovation at different ages. Our empirical specification allows medical expenditures and the impact of obesity on medical expenditures to vary also across other characteristics such as race and gender (see Section 4).

3.4.3 Extent of Causal Impact on Expenditures, Individual-Specific Causality

Ideally, the optimal obesity would be calculated from the causal impact of obesity on medical expenditures. However, the question of to which extent the increase in medical expenditures that is attributed to obesity is causal is not important when the objective is to compare the relative sizes of the positive innovation externality of obesity and the negative health insurance externality of obesity: this relative comparison is unlikely to be significantly affected by to which extent the estimated increase in medical expenditures that is attributed to obesity is causal (see Section 2.1). Notice also that as long as there are some marginal individuals for whom the lack of preventative activities associated with obesity is a choice, expression (44) gives the optimal subsidy even if obesity is genetic for some people (and for whom an obesity subsidy is thus only a transfer).

3.4.4 Variation in Impact of Obesity across Diseases

In an earlier version of this paper (Bhattacharya and Packalen, 2008b) we also examined the impacts of variation in the effect that obesity has on disease incidence across diseases. This variation does not influence the total innovation externality of obesity and the associated optimal obesity subsidy but this variation does influence the size of the induced innovation externality of obesity on different sub-populations such as the normal weight and the obese. These externalities on different sub-populations depend on an unknown parameter, namely the ratio of the reward-elasticity of the composition of innovation and the reward-elasticity of the total extent of innovation. The results in Bhattacharya and Packalen (2008b) show that unless this ratio is very high, also the external effect of obesity is positivee both on the obese and on the normal weight.

4 Application: Innovation vs. Insurance Externalities

In this section we first calculate the innovation externality of obesity by age using the expression (46) for the lower bound of the optimal obesity subsidy. We only estimate the size of this externality from pharmaceutical innovation because of the relative difficulty of cali-

brating the parameters R_{PATENT} and R_{MC} for other forms of medical innovation. We then compare the cumulative innovation externality of obesity from pharmaceutical innovation with the cumulative Medicare-induced pooled health insurance externality of obesity from all health care expenditures.

4.1 Data

We use the Medical Expenditure Panel Survey (MEPS) data from years 2002-2005 to measure pharmaceutical expenditures and total health care expenditures by age and Body-Mass Index (BMI) group.[12] While MEPS data is available beginning from 1996, we only use the MEPS data from years 2002-2005 to eliminate concern over possible time effects in the pharmaceutical expenditures data. We use the following age groups: 0-18, 18-25, 25-30, 30-35, 35-40, 40-45, 45-50, 50-55, 55-60, 60-65, 65-70, 70-75, 75-80, 80+, and the following BMI groups: 18.5-25 (normal weight) and 30-50 (obese). For each age and body weight combination we allow expenditures to vary by sex and race (black/non-black). Accordingly, in calculating the innovation and insurance externalities of obesity we use the estimate of the average impact of obesity on expenditures within each age group.

4.2 Innovation Externality by Age

We first calibrate the parameters R_{PATENT} and R_{MC} for pharmaceutical innovation. Berndt (2001) reports that the share of off-patent generics is approximately 50% of dispensed drug units. Because brand-name drugs cost more than generics we calibrate the share of the marginal pharmaceutical revenue that goes to brand-name drugs at $R_{PATENT} = 0.80$. Reinhardt (2001) cites estimates for the pharmaceutical industry that place marketing and general administration costs at 35% of revenue and manufacturing costs at 27% of revenue, but notes that firms in the pharmaceutical industry often manufacture also other goods besides brand-name drugs. Estimating the share $1 - R_{MC}$ of the marginal revenue from brand-name drugs that is in excess of marginal costs is therefore difficult. We calibrate it at $1 - R_{MC} = 0.66$.

To calculate the external effect of obesity from pharmaceutical innovation using the expression (46) for the lower bound of the optimal obesity subsidy, we also estimate the impact $E_t(obese) - E_t(normal)$ of obesity on annual pharmaceutical expenditures by age group using the MEPS data. Of course, to quantify the two externalities more precisely

[12] BMI is the standard measure used to determine an appropriate weight in the medical literature. BMI is weight, measured in kilograms, divided by height, measured in meters, squared. Individuals with a BMI above 30 are considered obese and individuals with a BMI between 25 and 30 are considered overweight (National Institute on Health, 1998).

it would be necessary to sort out which of the associations between obesity and disease prevalence are causal. However, as we have argued above in Section 2.1, because either of the two opposing externalities of obesity (the innovation externality and the health insurance externality) is present only for diseases for which the relationship is causal, the extent to which the relationships are causal is unlikely to significantly change the relative comparison of the two opposing externalities of obesity, and for this reason we are comfortable with limiting the scope of our analysis to not include an analysis of to what extent the associations represent causal effects.

Our later objective is to compare the innovation externality of obesity with the Medicare-induced health insurance externality of obesity for individuals who are covered by private insurance before old-age. Accordingly, for ages 0-65 we estimate pharmaceutical expenditures using only data on individuals who are covered by private insurance, and for ages 65+ we estimate pharmaceutical expenditures using data on individuals who are covered by either public or private insurance.

The results are shown in Figure 1. The estimate of the innovation externality of obesity

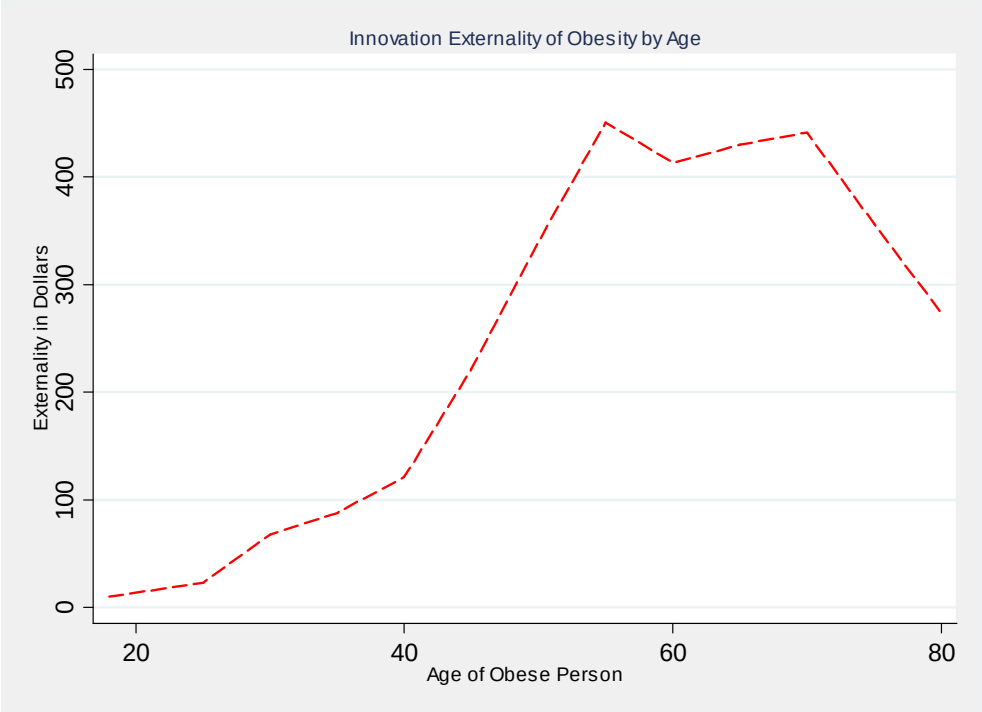

Figure 1: Innovation Externality of Obesity by Age.

increases sharply between age 40 and age 55. This is because the estimated impact of obesity on pharmaceutical expenditures increases sharply between age 40 and age 55, and

the innovation externality of obesity is calculated as a fixed percentage of the increase in a person's annual pharmaceutical expenditures that is attributed to obesity. The results also imply that the average and cumulative magnitudes of the innovation externality of obesity from pharmaceutical innovation over a lifetime are substantial.

4.3 Innovation vs. Insurance Externalities of Obesity

We now calculate the present value of the cumulative innovation externality of obesity from pharmaceutical innovation and compare it with the present value of the cumulative Medicare-induced health insurance externality of obesity from total health care expenditures.

The present value of the cumulative innovation externality of obesity from the initial age t_0 to the terminal age T is

$$\sum_{t=t_0}^{T} \beta^{t-t_0} \times Innovation\_Externality_t, \tag{47}$$

where β is the discount factor and $Innovation\_Externality_t$ is the innovation externality of obesity at age t from pharmaceutical innovation which is given by the expression (46) for the lower bound of the optimal obesity subsidy. The present value of the cumulative Medicare-induced public health insurance externality of obesity from the initial age t_0 to the terminal age T is

$$\sum_{t=\min\{t_0,65\}}^{T} \beta^{t-t_0} \times m \times \left[T_t\left(obese\right) - T_t\left(normal\right)\right], \tag{48}$$

where m is the share of the marginal health care expenditures paid by Medicare, and $T_t\left(normal\right)$ and $T_t\left(obese\right)$ are the average annual health care expenditures at age t for the normal weight and for the obese, respectively, and are estimated from the MEPS data. We also calculate the present value of the cumulative Medicare-induced public health insurance externality of obesity from pharmaceutical expenditures alone.[13]

We calibrate the discount factor at $\beta = 0.97$ and set the initial age at $t_0 = 18$.[14] The share of health care expenditures covered by Medicare for people aged 65 and over is approximately

[13] The Medicare-induced insurance expenditure for pharmaceutical expenditures alone is calculated as $\sum_{t=\min\{t_0,65\}}^{T} \beta^{t-t_0} \times m \times \left[E_t\left(obese\right) - E_t\left(normal\right)\right]$, where $E_t\left(obese\right)$ and $E_t\left(normal\right)$ denote the annual pharmaceutical expenditures at age t for the obese and for the normal weight, respectively.

[14] We calibrate the initial age at 18 both because the impact of obesity on health expenditures is very small before age 18 and because we do not want to examine the additional implications of imposing taxes or subsidies on the behavior of minors.

50% in the MEPS data.[15] While this average rate may not coincide with the marginal rate, we assume that for people aged 65 and over medicare pays 50% of the increase in health care expenditures that is caused by obesity by setting $m = 0.5$. We calculate the two cumulative externalities as a function of the terminal age T.

The results are shown in Figure 2. For a person with terminal age 80, which roughly

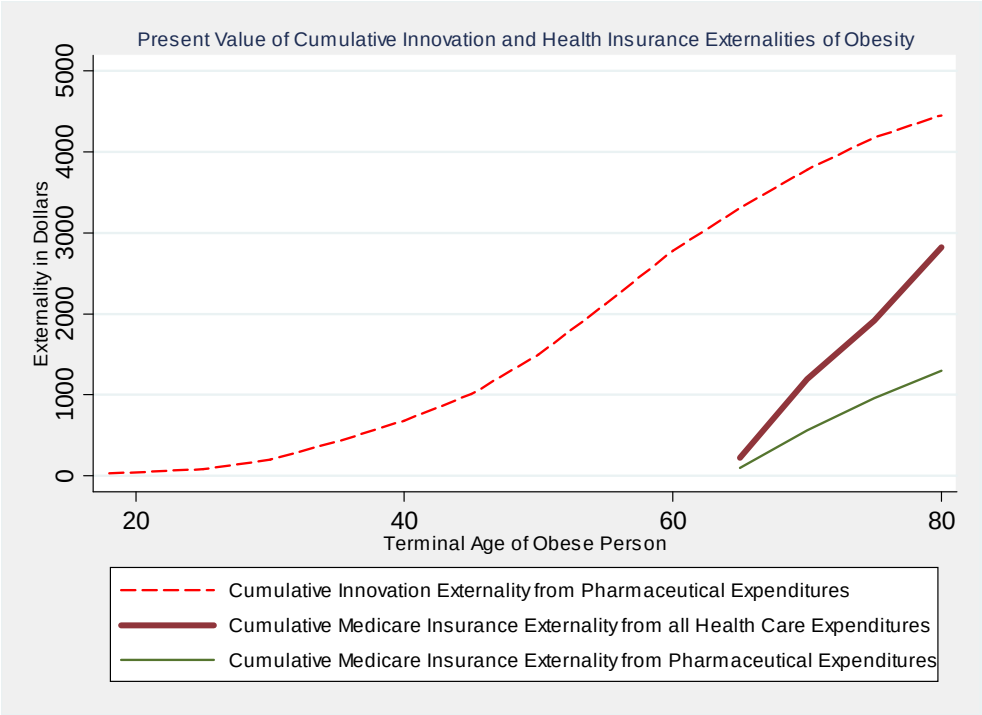

Figure 2: Present Value of Cumulative Innovation and Insurance Externalities of Obesity.

equals life expectancy, the present value of the (positive) cumulative innovation externality of obesity from pharmaceutical innovation is much larger than the present value of the (negative) Medicare-induced public health insurance externality from pharmaceutical expenditures and is similar in magnitude to the present value of the (negative) cumulative Medicare-induced public health insurance externality from all health care expenditures.[16]

This result is important for two reasons. First, it demonstrates that the *other* moral hazard in health that we identify can be quantitatively as important as the ex-ante moral

[15] Medicare is an old-age public insurance program and does not cover people aged 64 or younger. The proportion is presumably now higher than 50% since in 2006 Medicare started to cover pharmaceutical expenditures through its Part D program.

[16] To the extent that obesity reduces life expectancy (see e.g. Michaud et al., 2009), these reductions obviously have a larger impact on health expenditures at ages 65 years and older than at younger ages. Reductions in life expectancy that can be attributed to obesity therefore decrease the size of the Medicare-induced insurance externality of obesity more than the size of the innovation externality of obesity.

hazard examined by Ehrlich and Becker (1972) which has been a central concept in health economics for decades. Second, it implies that the magnitude of the Medicare-induced implicit pooled health insurance subsidy for obesity is roughly equal to the optimal subsidy for obesity that is implied by the innovation externality of obesity from pharmaceutical innovation. Accordingly, the presence of the Medicare-induced public health insurance externality of obesity is not a sufficient rationale for "soda taxes", "fat taxes" or other penalties aimed increasing the personal costs of obesity.

Of course, the exact value of the innovation externality of obesity is sensitive to the assumptions about the parameters. However, we believe that the conclusion that the magnitudes of the two opposing externalities of obesity are the same is robust. One reason why our estimate of the innovation externality of obesity likely underestimates the true magnitude of the externality is that in the above analysis we have ignored the innovation externality of obesity from all other medical expenditures than pharmaceutical expenditures which is also likely to be large. Another reason why our estimate of the innovation externality of obesity likely underestimates the true magnitude of the externality is that for reasons discussed in the beginning of Section 3.3 we have relied on the derived lower bound for the innovation externality rather than on the derived expression for the exact innovation externality.

5 Conclusion

In this paper we argue that an analysis of the ex-ante moral hazard should not stop at the disincentive effects of insurance on self-protective activities. To demonstrate that our argument is also quantitatively important we examine obesity as an example. A commonly held view is that since obesity is at least to some degree the result of an individual's decisions and an individual does not bear the full costs of obesity, public policies aimed at increasing the costs of obesity for an individual may be justified. Our analysis challenges this perspective on obesity.

Our theoretical argument is the following. Lower levels of prevention (self-protective activities) and the associated chronic conditions and behavioral patterns such as obesity, smoking, and malnutrition increase the incidence of many diseases. This increases the consumer's demand for treatments to those diseases, which increases an innovator's reward for innovation of treatments to those diseases. By the induced innovation hypothesis, which has broad empirical support, the increase in the reward for innovation increases the innovation of treatments to those diseases, which in turn benefits all consumers. Because individuals do not take these positive externalities on the innovator and other consumers into account

when deciding the level of preventative activities such as exercise and healthy diet, they invest too much in prevention. In other words, absent a policy intervention, individuals are too healthy.

Our quantitative application shows that the innovation externality of obesity from pharmaceutical expenditures alone roughly coincides with the Medicare-induced health insurance externality of obesity from total health care expenditures. This finding implies that the *other* ex-ante moral hazard that we identify can be quantitatively as important as the ex-ante moral hazard examined by Ehrlich and Becker (1972) which has been a central concept in health economics for decades. This quantitative result also implies that the Medicare-induced subsidy for obesity is roughly optimal and thus the presence of this subsidy is not an adequate justification for "soda taxes", "fat taxes" or other penalties on obesity.

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Appendix: Proofs

A.1 Proof of the Stability Condition (33)

The slopes of the innovator and consumer optimum conditions (31) and (32) are

$$\frac{d\mu}{d\frac{n_{OBESE}}{N}} = \frac{d\mu}{dR} \times N \times (\pi_{OBESE} - \pi_{NORMAL}) \times s \times (D_1 - D_0) \tag{49}$$

and

$$\frac{d\frac{n_{OBESE}}{N}}{d\mu} = F'(B_{NORMAL}) \times (\pi_{OBESE} - \pi_{NORMAL}) \times (1-s)(D_0 - D_1), \tag{50}$$

respectively. Applying the definition (30) of ε_μ and the expression (23) for R, expression (49) can be written as

$$\frac{d\mu}{d\frac{n_{OBESE}}{N}} = \varepsilon_\mu \times \mu \times \frac{\pi_{OBESE} - \pi_{NORMAL}}{\pi_{AVERAGE}}. \tag{51}$$

Applying the definition (20) of ε_{OBESE} and the expression (17) for B_{NORMAL}, expression (50) can be written as

$$\frac{d\frac{n_{OBESE}}{N}}{d\mu} = \varepsilon_{OBESE} \times \frac{n_{OBESE}}{N} \times \frac{(1-s)(D_0 - D_1)}{D_0 - \mu(1-s)(D_0 - D_1)}. \tag{52}$$

Substituting these expressions (51) and (52) into the stability condition $\frac{d\mu}{d\frac{n_{OBESE}}{N}} \times \frac{d\frac{n_{OBESE}}{N}}{d\mu} < 1$, the stability condition becomes

$$\varepsilon_\mu \times \mu \times \frac{\pi_{OBESE} - \pi_{NORMAL}}{\pi_{AVERAGE}} \times \varepsilon_{OBESE} \times \frac{n_{OBESE}}{N} \times \frac{(1-s)(D_0 - D_1)}{D_0 - \mu(1-s)(D_0 - D_1)} < 1. \tag{53}$$

Because $\mu \leq 1$ and $\frac{n_{OBESE}}{N} \leq 1$ by definition, and because

$$\frac{(1-s)(D_0 - D_1)}{D_0 - \mu(1-s)(D_0 - D_1)} \leq \frac{D_0 - D_1}{D_0} \tag{54}$$

holds for all s and μ, condition (33) is a sufficient condition for the stability condition (53) to hold.

A.1.2 Proof of Proposition 1

A.1.2.1 First-order condition for s

The first-order condition (FOC) for the optimal s in the optimization problem (35) is

$$-B_{NORMAL}F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{ds}-\frac{d\pi_{AVERAGE}}{ds}C_{ILLNESS}-\pi_{AVERAGE}\frac{dC_{ILLNESS}}{ds}=0 \tag{55}$$

Substituting the result

$$\frac{d\pi_{AVERAGE}}{ds}=\left(\pi_{OBESE}-\pi_{NORMAL}\right)\frac{d\frac{n_{OBESE}}{N}}{ds} \tag{56}$$

for $\frac{d\pi_{AVERAGE}}{ds}$ in the FOC (55) yields

$$-B_{NORMAL}F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{ds}-\left(\pi_{OBESE}-\pi_{NORMAL}\right)\frac{d\frac{n_{OBESE}}{N}}{ds}C_{ILLNESS}$$

$$-\pi_{AVERAGE}\frac{dC_{ILLNESS}}{ds}=0. \tag{57}$$

The consumer optimum condition (19) implies that

$$\frac{d\frac{n_{OBESE}}{N}}{ds}=-F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{ds}. \tag{58}$$

Substituting this result (58) for $\frac{d\frac{n_{OBESE}}{N}}{ds}$ in the FOC (57) yields

$$F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{ds}\left[-B_{NORMAL}+\left(\pi_{OBESE}-\pi_{NORMAL}\right)C_{ILLNESS}\right]$$

$$-\pi_{AVERAGE}\frac{dC_{ILLNESS}}{ds}=0. \tag{59}$$

Applying the definition (17) of B_{NORMAL} allows us to rewrite this FOC as

$$F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{ds}t-\pi_{AVERAGE}\frac{dC_{ILLNESS}}{ds}=0. \tag{60}$$

Substituting the result

$$\frac{dB_{NORMAL}}{ds}=\left(\pi_{OBESE}-\pi_{NORMAL}\right)\frac{dC_{ILLNESS}}{ds} \tag{61}$$

for $\frac{dB_{NORMAL}}{ds}$ allows us to rewrite the FOC (59) as

$$\left[F'\left(B_{NORMAL}\right)\left(\pi_{OBESE}-\pi_{NORMAL}\right)t-\pi_{AVERAGE}\right]\times\frac{dC_{ILLNESS}}{ds}=0. \tag{62}$$

Given the FOC (77) for the optimal t below, the factor in the brackets in the FOC (62) above for the optimal s is non-zero whenever the FOC (77) for the optimal t holds. Thus, the relevant FOC for the optimal s is

$$\frac{dC_{ILLNESS}}{ds}=0. \tag{63}$$

Applying the definition (15) of $C_{ILLNESS}$ allows us to rewrite this FOC as

$$-\frac{d\mu}{ds}\left(1-s\right)+\mu=0. \tag{64}$$

Given the definitions (15) and (17) for $C_{ILLNESS}$ and $B_{NORMAL,}$ respectively, the condition $\frac{dC_{ILLNESS}}{ds}=0$ implies that the property $\frac{dB_{NORMAL}}{ds}=0$ holds. Moreover, given the definition (17) of B_{NORMAL} and the expression (58) for $\frac{d\frac{n_{OBESE}}{N}}{ds}$, the property $\frac{dB_{NORMAL}}{ds}=0$ implies that the property $\frac{d\frac{n_{OBESE}}{N}}{ds}=0$ holds. Hence, the condition $\frac{dC_{ILLNESS}}{ds}=0$ implies that the property $\frac{d\frac{n_{OBESE}}{N}}{ds}=0$ holds. Thus, while in general the derivative $\frac{d\mu}{ds}$ is given by

$$\frac{d\mu}{ds}=\frac{d\mu}{dR}\left[\frac{\partial R}{\partial s}+\frac{\partial R}{\partial\frac{n_{OBESE}}{N}}\frac{d\frac{n_{OBESE}}{N}}{ds}\right], \tag{65}$$

at the optimum s_{CS}^{*}, for which the condition $\frac{dC_{ILLNESS}}{ds}=0$ and the property $\frac{d\frac{n_{OBESE}}{N}}{ds}=0$ hold, the derivative$\frac{d\mu}{ds}$ is given by

$$\frac{d\mu}{ds}=\frac{d\mu}{dR}\frac{\partial R}{\partial s} \tag{66}$$

holds. Substituting this result (66) for $\frac{d\mu}{ds}$ allows us to rewrite the FOC (64) as

$$-\frac{d\mu}{dR}\frac{\partial R}{\partial s}\left(1-s\right)+\mu=0. \tag{67}$$

Using the definition (23) of R yields

$$\frac{\partial R}{\partial s}=\frac{R}{s}. \tag{68}$$

Substituting this result (68) for $\frac{\partial R}{\partial s}$ allows us to rewrite the FOC (67) as

$$-\frac{d\mu}{dR}\frac{R}{s}\left(1-s\right)+\mu=0. \tag{69}$$

Applying the definition (30) of ε_μ allows us to rewrite this FOC as

$$\varepsilon_\mu \times \frac{1}{s} - \frac{1}{1-s} = 0. \tag{70}$$

A.1.2.2 First-order condition for t

The FOC for the optimal t in the optimization problem (35) is

$$-B_{NORMAL}F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{dt} - \frac{d\pi_{AVERAGE}}{dt}C_{ILLNESS} - \pi_{AVERAGE}\frac{dC_{ILLNESS}}{dt} = 0. \tag{71}$$

Substituting the result

$$\frac{d\pi_{AVERAGE}}{dt} = \frac{d\frac{n_{OBESE}}{N}}{dt}\left(\pi_{OBESE} - \pi_{NORMAL}\right)C_{ILLNESS} \tag{72}$$

for $\frac{d\pi_{AVERAGE}}{dt}$ allows us to rewrite the FOC (71) as

$$-B_{NORMAL}F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{dt} - \frac{d\frac{n_{OBESE}}{N}}{dt}\left(\pi_{OBESE} - \pi_{NORMAL}\right)C_{ILLNESS}$$

$$-\pi_{AVERAGE}\frac{dC_{ILLNESS}}{dt} = 0. \tag{73}$$

Substituting the result

$$\frac{d\frac{n_{OBESE}}{N}}{dt} = -F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{dt} \tag{74}$$

for $\frac{d\frac{n_{OBESE}}{N}}{dt}$ allows us to rewrite the FOC (73) as

$$F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{dt}\left[\left(\pi_{OBESE} - \pi_{NORMAL}\right)C_{ILLNESS} - B_{NORMAL}\right]$$

$$-\pi_{AVERAGE}\frac{dC_{ILLNESS}}{dt} = 0. \tag{75}$$

Applying the definition (17) of B_{NORMAL} allows us to rewrite this FOC as

$$F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{dt}t - \pi_{AVERAGE}\frac{dC_{ILLNESS}}{dt} = 0. \tag{76}$$

Applying the result $\frac{dB_{NORMAL}}{dt} = (\pi_{OBESE} - \pi_{NORMAL}) \frac{dC_{ILLNESS}}{dt} - 1$ allows us to rewrite the FOC (76) as

$$[F'(B_{NORMAL})(\pi_{OBESE} - \pi_{NORMAL})t - \pi_{AVERAGE}] \frac{dC_{ILLNESS}}{dt} - tF'(B_{NORMAL}) = 0. \tag{77}$$

This formulation of the FOC for the optimal t is used after expression (62) above to solve for the relevant FOC for the optimal s. Applying the result (74) again allows us to rewrite the earlier FOC (76) for the optimal t as

$$-\frac{d\frac{n_{OBESE}}{N}}{dt}t - \pi_{AVERAGE}\frac{dC_{ILLNESS}}{dt} = 0. \tag{78}$$

Substituting the result

$$\frac{dC_{ILLNESS}}{dt} = -\frac{d\mu}{dt}(1-s)(D_0 - D_1) \tag{79}$$

for $\frac{dC_{ILLNESS}}{dt}$ allows us to rewrite the FOC (78) as

$$-\frac{d\frac{n_{OBESE}}{N}}{dt}t - \pi_{AVERAGE}\left(-\frac{d\mu}{dt}(1-s)(D_0 - D_1)\right) = 0. \tag{80}$$

Substituting the expression $\frac{d\mu}{dt} = \frac{d\mu}{dR}\frac{dR}{dt}$ for $\frac{d\mu}{dt}$ and substituting the result

$$\frac{dR}{dt} = \frac{d\frac{n_{OBESE}}{N}}{dt}(\pi_{OBESE} - \pi_{NORMAL})s(D_0 - D_1) \tag{81}$$

for $\frac{dR}{dt}$ allows us to rewrite the FOC (80) as

$$\pi_{AVERAGE}\left(\frac{d\mu}{dR}(\pi_{OBESE} - \pi_{NORMAL})s(D_0 - D_1)\frac{d\frac{n_{OBESE}}{N}}{dt}(1-s)(D_0 - D_1)\right)$$

$$-\frac{d\frac{n_{OBESE}}{N}}{dt}t = 0. \tag{82}$$

Applying the definition (23) of R allows us to rewrite this FOC as

$$-\frac{d\frac{n_{OBESE}}{N}}{dt}t + \left(\frac{d\mu}{dR}R(\pi_{OBESE} - \pi_{NORMAL})\frac{d\frac{n_{OBESE}}{N}}{dt}(1-s)(D_0 - D_1)\right) = 0. \tag{83}$$

Applying the FOC (69) for the optimal s allows us to rewrite this FOC (83) for the optimal t as

$$\frac{d\frac{n_{OBESE}}{N}}{dt}\left[\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right)-t\right]=0. \tag{84}$$

This FOC for the optimal t yields the expression

$$t^*_{CS}=\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right) \tag{85}$$

for the consumer surplus maximizing obesity subsidy t^*_{CS}.

A.1.2.3 Second-Order Condition

We first show that $s^*_{CS}\in(0,1)$. Obviously, $s^*_{CS}<1$ as consumers do not receive any benefit from innovation if $s=1$. If $s=0$ then $t^*_{CS}=0$ as a positive t would not induce any innovation. Therefore, a sufficient condition for the result $s^*_{CS}>0$ is that the first derivative of the objective function in the relevant maximization problem (35) with respect to s is positive when $s=0$ and $t=0$. Substituting $t=0$ into the expression on the left-hand side of the FOC (62) for the optimal s, this condition can be written as

$$-\pi_{AVERAGE}\times\frac{dC_{ILLNESS}}{ds}>0 \tag{86}$$

when $s=0$. Applying the definition (15) of $C_{ILLNESS}$ allows us to rewrite this condition (86) as

$$-\pi_{AVERAGE}\left[-\frac{d\mu}{ds}\left(1-s\right)+\mu\right]\left(D_0-D_1\right)>0, \tag{87}$$

which holds at $s=0$ by assumption (28) according to which the probability of innovation μ is small enough (or zero) when the reward for innovation is zero, $R=0$, Hence, $s^*_{CS}\in(0,1)$, which in turn implies that the FOC for the optimal s yields the optimal s.

To show that the FOC for the optimal subsidy t yields the optimal subsidy t we show that when the FOC for the optimal t holds, we prove two results. First we show that the second derivative of the objective function in the relevant maximization problem (35) with respect to the subsidy t is negative. We then show that when the subsidy t is given by the expression (34) for the subsidy t_{CS}, the share of individuals who choose the low level of prevention is less than one, $\frac{n_{OBESE}}{N}<1$. Together these two results imply that the FOC for the optimal t yields the optimal t.

Given the expression for the first derivative of the objective function with respect to the subsidy t on the left-hand side of the FOC (84) for the optimal subsidy t, the second

derivative of the objective function is negative when the FOC holds if the condition

$$\frac{d^2 \frac{n_{OBESE}}{N}}{dt^2}[0] + \frac{d\frac{n_{OBESE}}{N}}{dt}\left[\frac{d\mu}{dt}(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1) - 1\right] < 0 \tag{88}$$

holds when the FOC holds. Because $\frac{d\frac{n_{OBESE}}{N}}{dt} > 0$ this condition (88) can be rewritten as

$$\frac{d\mu}{dt}(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1) < 1, \tag{89}$$

which can be rewritten as

$$\frac{d\mu}{dR}\frac{dR}{d\frac{n_{OBESE}}{N}}\frac{d\frac{n_{OBESE}}{N}}{dt}(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1) < 1. \tag{90}$$

Substituting the result

$$\frac{dR}{d\frac{n_{OBESE}}{N}} = (\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1) \tag{91}$$

for $\frac{dR}{d\frac{n_{OBESE}}{N}}$ allows us to rewrite condition (90) as

$$\frac{d\mu}{dR}(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1)\frac{d\frac{n_{OBESE}}{N}}{dt}(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1) < 1. \tag{92}$$

Applying the definitions (30) and (20) for ε_μ and ε_{OBESE}, respectively, allows us to rewrite this condition as

$$\varepsilon_\mu \mu \frac{(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1)}{R}\varepsilon_{OBESE}\frac{n_{OBESE}}{N}\frac{(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1)}{B_{NORMAL}} < 1. \tag{93}$$

Substituting the definitions (23) and (17) for R and B_{NORMAL}, respectively, allows us to rewrite this condition as

$$\varepsilon_\mu \mu \frac{\pi_{OBESE} - \pi_{NORMAL}}{\pi_{AVERAGE}}\varepsilon_{OBESE}\frac{\frac{n_{OBESE}}{N}(\pi_{OBESE} - \pi_{NORMAL})\, s\,(D_0 - D_1)}{(\pi_{OBESE} - \pi_{NORMAL}) \times [D_0 - \mu\,(1 - s)\,(D_0 - D_1)] - t} < 1 \tag{94}$$

Substituting the expression (34) for the optimal obesity subsidy t^*_{CS} for the obesity subsidy t allows us to rewrite the condition (94) as

$$\varepsilon_\mu \mu \frac{(\pi_{OBESE} - \pi_{NORMAL})}{\pi_{AVERAGE}}\varepsilon_{OBESE}\frac{n_{OBESE}}{N}\frac{(D_0 - D_1)\, s}{D_0 - \mu\,(D_0 - D_1)} < 1. \tag{95}$$

Because $\mu \leq 1$ and $\frac{n_{OBESE}}{N} \leq 1$ by definition, and because the property (54) holds for all s and μ, a sufficient condition for this condition (95) to hold is that the condition

$$\varepsilon_{\mu} \times \varepsilon_{OBESE} \times \frac{\pi_{OBESE} - \pi_{NORMAL}}{\pi_{AVERAGE}} \times \frac{D_0 - D_1}{D_0} < 1 \tag{96}$$

holds. This condition (96) is the same as the stability condition (33), and hence holds by assumption.

We now show that when the subsidy t is given by the expression (34) for the subsidy t_{CS}, the share of individuals who choose the low level of prevention is less than one, $\frac{n_{OBESE}}{N} < 1$. Substituting the expression (34) for the subsidy t^*_{CS} for t in the expression (32) for the share of individuals $\frac{n_{OBESE}}{N}$ who choose the low level of prevention yields

$$\begin{aligned}
\frac{n_{OBESE}}{N} &= 1 - F[(\pi_{OBESE} - \pi_{NORMAL}) \times [D_0 - \mu\,(1-s)\,(D_0 - D_1)] && (97)\\
&\quad -\mu \times s \times (\pi_{OBESE} - \pi_{NORMAL})\,(D_0 - D_1)] \\
&= 1 - F\left[(\pi_{OBESE} - \pi_{NORMAL}) \times [D_0 - \mu\,(D_0 - D_1)]\right] && (98)\\
&< 1 - F\left[(\pi_{OBESE} - \pi_{NORMAL}) \times [D_0 - 1\,(D_0 - D_1)]\right] && (99)\\
&= 1 - F\left[(\pi_{OBESE} - \pi_{NORMAL}) \times D_1\right] && (100)\\
&< 1, && (101)
\end{aligned}$$

where the first equality follows from the second part of assumption (27) and the last inequality follows from the second part of assumption (18).

A.1.3 Proof of Proposition 2

The first-order condition to the maximization problem (43) is

$$\begin{aligned}
&\frac{d\left[\mu R - C\left(\mu\right)\right]}{d\mu}\frac{d\mu}{dt} + \mu\frac{dR}{dt} \\
&-N \times B_{NORMAL} F'\left(B_{NORMAL}\right)\frac{dB_{NORMAL}}{dt} \\
&-N\frac{d\pi_{AVERAGE}}{dt}C_{ILLNESS} - N\pi_{AVERAGE}\frac{dC_{ILLNESS}}{dt} = 0. && (102)
\end{aligned}$$

Because the probability of innovation μ is chosen optimally by the innovator, the property $\frac{d[\mu R - C(\mu)]}{d\mu} = 0$ holds, which implies that the first term on the first line of the FOC (102) is zero. Aside from the presence of the factor N, the combined second and third line of the

FOC (102) is the same as the FOC (71) for the subsidy t that maximizes consumer surplus. Following the same steps as in solving the FOC for the consumer surplus maximizing subsidy t, the combined second and third line of the FOC (102) for the total surplus maximizing subsidy t can be written the same as the FOC (83) for the consumer surplus maximizing subsidy t. Applying these results, and also the result (81) for $\frac{dR}{dt}$, enables us to rewrite the FOC (102) as

$$\mu\left[N\frac{d\frac{n_{OBESE}}{N}}{dt}\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right)\right]$$

$$-N\frac{d\frac{n_{OBESE}}{N}}{dt}t+N\left(\frac{d\mu}{dR}R\left(\pi_{OBESE}-\pi_{NORMAL}\right)\frac{d\frac{n_{OBESE}}{N}}{dt}\left(1-s\right)\left(D_0-D_1\right)\right)=0. \quad (103)$$

Applying the definition (30) of ε_μ enables us to rewrite this FOC as

$$-\frac{d\frac{n_{OBESE}}{N}}{dt}N[t-\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right)$$

$$-\varepsilon_\mu\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)\left(1-s\right)\left(D_0-D_1\right)]=0. \quad (104)$$

This FOC for the optimal subsidy t implies that the optimal subsidy t is

$$t=\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right)+\varepsilon_\mu\frac{1-s}{s}\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right). \quad (105)$$

We next show that the first derivative of the objective function in (43) is positive when $t=0$, so that $t^*_{TS}>0$. Using the FOC (104), the first derivative of the objective function in the maximization problem (43) is

$$\frac{d\frac{n_{OBESE}}{N}}{dt}N[\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right)$$

$$+\varepsilon_\mu\frac{1-s}{s}\mu\left(\pi_{OBESE}-\pi_{NORMAL}\right)s\left(D_0-D_1\right)] \quad (106)$$

when $t=0$. The factor $\frac{dn_{OBESE}}{dt}$ is positive because both the direct effect (through t) and the indirect effect (through μ) of an increase in t on B_{NORMAL} are negative and thus the effect of an increase in t on $\frac{n_{OBESE}}{N}$ is positive. Moreover, all factors inside the brackets in expression (106) are positive. Hence, expression (106) is positive, which implies that $t^*_{TS}>0$.

We now show that when the optimal subsidy is so high that all consumers choose the low level of prevention, $\frac{n_{OBESE}}{N}=1$, the first derivative of the objective function in (43) is positive. Given the assumption $F'\left(\theta\right)>0$ for all $\theta\in[0,\bar{\theta})$ and the expression $\frac{n_{OBESE}}{N}=1-F\left(B_{NORMAL}\right)$ for the number of consumers who choose the low level of prevention, a

necessary and sufficient condition for all consumers to choose the low level of prevention is that $B_{NORMAL} = 0$. Applying the definition (17) of B_{NORMAL}, the condition $B_{NORMAL} = 0$ holds if and only if $t = \tilde{t}$, where

$$\tilde{t} = (\pi_{OBESE} - \pi_{NORMAL}) [D_0 - \mu (1 - s) (D_0 - D_1)]. \tag{107}$$

Substituting this expression (107) for t in the expression (104) for the first derivative of the objective function in (43) yields

$$-\frac{d\frac{n_{OBESE}}{N}}{dt} (\pi_{OBESE} - \pi_{NORMAL}) N[D_0 - \mu (D_0 - D_1) - \varepsilon_\mu \mu (1 - s) (D_0 - D_1)], \tag{108}$$

which can be rewritten as

$$-\frac{d\frac{n_{OBESE}}{N}}{dt} (\pi_{OBESE} - \pi_{NORMAL}) D_0 N[1 - \mu(1 + \varepsilon_\mu (1 - s))\frac{D_0 - D_1}{D_0}]. \tag{109}$$

In this expression (109) the factor $\frac{d\frac{n_{OBESE}}{N}}{dt} (\pi_{OBESE} - \pi_{NORMAL}) D_0 N$ is obviously positive and the expression in brackets is positive by assumption (29). Hence, the expression (109) is negative, which implies that the optimal subsidy t^*_{TS} is smaller than the subsidy $\tilde{t}$ given by expression (107) which induces all consumers to choose the low level of prevention.

This result $t^*_{TS} < \tilde{t}$ and the previous result $t^*_{TS} > 0$ together imply that the optimal obesity subsidy t^*_{TS} is given by the solution to the FOC for the optimal obesity subsidy.