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# Differing types of medical prevention appeal to different individuals

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# Differing types of medical prevention appeal to different individuals.\*

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#### Abstract

We analyse participation in medical prevention with an expected utility model that is sufficiently rich to capture diverging features of different prevention procedures. We distinguish primary and secondary prevention (with one or two rounds) for both fatal or non-fatal diseases. Moreover, we introduce a flexible relationship between the specific disease for which the prevention procedure is set up and the general background health of the individual. We show how these various possibilities change the comparative statics of the prevention decision and we test the differential predictions with data from SHARE (Survey of Health, Ageing and Retirement in Europe) about participation in mammography, dental caries screening and flu vaccination.

Keywords: screening, vaccination, expected utility, behavioral economics.

JEL Classification: D81, I12.

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## 1 Introduction

Medical prevention, i.e. vaccination and screening, has become increasingly important in the health care systems of advanced countries. Health practitioners are concerned about the relatively low participation rates, even for prevention measures that are generally considered to be cost-effective (such as flu vaccination for the elderly and breast cancer screening for women between 50 and 69 years old). A careful look at this participation pattern reveals huge interindividual and intercountry differences. Moreover, participation also varies widely between different procedures for the same individuals. Gaining a better understanding of the causes of these differences across individuals and types of prevention is definitely relevant from a policy point of view.

However, the importance of analysing medical prevention decisions goes beyond the policy aspect. The large degree of interindividual variation (and the claim by some that prevention decisions are suboptimal) also make it an interesting domain to test the theory of decision-making under uncertainty. Our main contribution to the literature is that we synthesize existing evidence on participation in medical prevention. Rather than focusing on one specific procedure, our aim is to build and test a model that is sufficiently rich so as to give some insights into the different results that are found with respect to different prevention procedures. We distinguish preventive care decisions along three dimensions. First, we compare in the same model both primary and secondary prevention (with one or two rounds).<sup>1</sup> The former refers to interventions that help avoid a given disease (e.g. vaccination), and the latter to measures that are aimed at detecting the presence of a disease in its early stages, so that early curative treatment becomes possible (e.g. cancer screening). Second, we distinguish between fatal (e.g. cancer) and non-fatal (e.g. dental caries) diseases. Third, we introduce into our model a flexible relationship between the specific disease for which the prevention procedure is set up and the general background health of the individual. In some cases, individuals may care more about the specific disease when their background health is worse (e.g. flu), in other cases they may care more when their background health is better (e.g. dental caries). We show how these various possibilities change the comparative statics of the prevention decision and test the differential predictions with data from SHARE (Survey of Health, Ageing and Retirement in Europe).

In this paper we stay in the tradition of the expected utility-approach to study individual preventive medical behaviour (see, among others, Dervaux and Eeckhoudt, 2004; Picone et al., 2004; Howard, 2005; Witt, 2008). The expected utility-model has recently come under sharp criticism. It is now widely accepted that it is unable to explain the real-world observations if one assumes a narrow specification

<sup>&</sup>lt;sup>1</sup>Note that we only focus on medical prevention procedures and do not consider lifestyle choices.

of utility (e.g. focusing only on health and income) and perfectly informed individuals. Taking a test imposes not only monetary (and time) costs, but also a psychological burden, which, according to the available surveys on motivations, may be crucial in explaining variations in preventive care participation (see, e.g., Whynes et al., 2007). Moreover, while the literature has shown that subjective probabilities influence individual decisions, it has also become clear that the subjective risk perceptions vary only very partially with objective risk factors (Carman and Kooreman, 2011). Therefore, the expected utility model only makes sense as an explanation of behaviour if all variables used in the model are individualspecific or an individual-specific interpretation of an objective parameter. This is acknowledged by most authors in the field, and we also adopt this interpretation. Of course, this means that our approach is necessarily incomplete. A complete model (even of the expected utility-kind) would require in addition an explanation of the subjective formation of beliefs and of the nature of the psychological costs.

Even more fundamentally, some recent papers in the behavioural economic literature have rejected the expected utility-model to build other realistic features into the analysis of screening and prevention decisions: hyperbolic discounting and myopia (Byrne and Thompson, 2001; Fang and Wang, 2010), lossaversion over changes in beliefs (Fels, 2011), biased perceptions of risks in a rank-dependent utility model (Etner and Jeleva, 2012) and anticipatory feelings (Oster et al., 2011). Some of these developments are very promising, but it would be overly ambitious to try to build a general model of different prevention decisions incorporating these mechanisms. For our comparative exercise, the expected utility model remains a convenient and flexible starting point. Moreover, it is still largely an open question as to how much the more sophisticated behavioural models add to the explanatory power of an (extended) expected-utility model, especially in cases of primary prevention and secondary prevention with screening as a necessary condition for treatment.<sup>2</sup> In any case, it remains useful to test how well the expected utility model can accommodate differences in the specific features of different medical prevention procedures.

In our empirical work we focus on three cases: breast cancer screening, dental caries screening and flu vaccination. We estimate probit models with the pooled data of the first two waves of SHARE. There have been previous empirical studies analysing the same prevention procedures with SHARE data (Maurer, 2009; Schmitz and Wübker, 2011 and Jusot et al., 2012 for influenza vaccination; Wübker, 2012a, 2012b and Jusot et al., 2012, for mammography; Listl, 2011 and Listl et al., 2012 for dental care). To the best of our knowledge, we present the first attempt to compare the results for the different procedures within a coherent theoretical approach, testing specific hypotheses about the differential comparative static effects. In accordance with the estimation strategies in Wübker (2012a) for breast cancer screening

 $<sup>^{2}</sup>$  The strongest arguments against using the expected utility model can be found in Oster et al. (2011). However, they analyse medical testing decisions for Huntington's disease – where at this moment no curative treatment is available.

and Listl et al. (2012) for dental care, we explain (part of) the intercountry differences through the introduction of institutional features that are specifically related to the prevention procedures analysed. These specific features can be related to the parameters from the theoretical model. This approach appears more promising than controlling for general characteristics of a country's health-care system (Jusot et al., 2012<sup>3</sup>). Regrettably, most studies do not attempt to incorporate institutional features (Listl, 2011, Maurer, 2009, Schmitz and Wübker, 2011 and Wübker, 2012b). Finally, some authors use information on the reported past behaviour in regard to (non-)participation in breast cancer screening (Wübker, 2012a, 2012b) and in preventive dental care (Listl et al., 2012) available in the third wave of SHARE to explain current behaviour. Since our theoretical model does not incorporate past behaviour, we have not used this information.

Section 2 describes our model with different types of disorders and characteristics of the process of medical prevention. Comparative static results for the prevention decision are derived in Section 3. Section 4 discusses the empirical testing of the hypotheses that are derived from the theoretical model. Section 5 concludes.

## 2 Type of disorder and characteristics of the process of medical prevention

We model prevention decisions related to a specific medical disorder. At the beginning of the period<sup>4</sup>, the individual believes that she will develop this disorder with probability p. The indicator representing its severity is denoted by m and takes one of four discrete values (0 < l < e < n), ranging from 0, i.e. deadly disorder, to n, when the individual does not suffer from the disorder. The values l and e indicate late and early stages of the disorder respectively. The stages are mutually exclusive. The costs of treatment for the individual (after accounting for potential government subsidies and health insurance coverage) are  $c_l$ and  $c_e$  respectively. If treated, the patient is cured of the illness, but relapse in the next period remains possible. Some diseases can be fatal when not treated, while others are not. The prevention behaviour of the individual determines whether the disease develops into early or late stage. Participation in a

 $<sup>^{3}</sup>$ None of the general characteristics used in this article turn out to have a significant effect for the explanation of flu vaccination and breast cancer screening.

<sup>&</sup>lt;sup>4</sup>In our model a 'period' is defined as the normal amount of time in which an individual has to choose whether or not to participate in prevention. For the flu, a period is a one-year interval, since an individual will have to decide to participate in prevention every year before the flu season starts. For breast cancer screening on the other hand, the normal screening interval is two years. Furthermore, we assume for simplicity and clarity that this amount of time corresponds to the period in which a disease can develop from a relatively harmless to a severe illness that requires curative care, or in case of a fatal disease, that might result in death. While this is true for many diseases such as e.g. the flu, this is not always the case. The assumption can however be relaxed and our model adapted so that the prevention period and the period of disease development do not necessarily coincide.

preventive program (either screening or vaccination) is taken to be a binary decision. We first describe in more detail how we model the type of disorder and then go into the characteristics of the process of medical prevention.

#### 2.1 Type of disorder<sup>5</sup>

During period t, the individual derives utility u(.) from income y and health, with  $u_1(y, h, m) > 0$  and  $u_{11}(y, h, m) \leq 0.^6$  To distinguish between different types of diseases, we introduce an index h representing the overall ("background") health of the individual, in addition to the indicator m representing the severity of the specific medical disorder. A better initial health status is translated as a higher index score h with  $u_2(y, h, m) > 0$  and  $u_{22}(y, h, m) \leq 0$ . In the occurrence of death, utility becomes zero, i.e.  $u(y, h, 0) = 0, \forall y, h$ .

Lifetime utility from period t onwards can be written as:

$$I(nf) \left[ u(y,h,m) + \beta (1 - p_{x,t+1}) V_{t+1} \right]$$
(1)

We introduce an indicator function I(nf) to indicate whether or not a disease is non-fatal. The indicator turns to zero if the individual dies from the specific medical disorder and equals 1 otherwise, while the individual's mortality risk from any other cause is given by  $p_{x,t+1}$ . Future utility  $V_{t+1}$  depends on the future streams of income and health, and is discounted with factor  $\beta$ . Of course, the future only matters if the individual survives into period t + 1.

Our two-dimensional representation of health allows us to distinguish between three types of specific disorders in terms of their interaction with the initial overall health status.

**Complements** Consider first the case of a rather minor medical problem, which does not affect the general health status of the individual: dental caries is an obvious example. In this case, it is natural to assume that "quality of the teeth" matters more for healthier individuals. This is represented in our model by

$$u_2(y, h, m_1) > u_2(y, h, m_2), \forall y, h \text{ if } m_1 > m_2$$
(2)

**Comorbidities** An alternative situation is the case of comorbidities, where the occurrence of the disease has a stronger effect on health if the background health status is worse. A good example is the

<sup>&</sup>lt;sup>5</sup> In this section, we drop the subscript t for notational convenience.

<sup>&</sup>lt;sup>6</sup>We define  $u_x(y, h, m)$  as the derivative of u(y, h, m) with respect to the  $x^{th}$  argument of u(.). Analogously,  $u_{xz}(y, h, m)$  is the cross derivative of u(y, h, m) with respect to the  $x^{th}$  and the  $z^{th}$  argument of u(.).

flu, since a healthy individual will suffer less from it than a sick individual, and runs a smaller risk of complications. If the utility loss due to the disorder is mitigated by a better initial health, this results in

$$u_2(y, h, m_1) < u_2(y, h, m_2), \forall y, h \text{ if } m_1 > m_2$$
(3)

**Independence** In principle it is also possible that the effect of the new disorder is largely independent of the initial overall health status, resulting in

$$u_2(y,h,m_1) = u_2(y,h,m_2), \forall y,h,m_1,m_2.$$
(4)

Perhaps an extreme diagnosis like that of a life-threatening cancer could be an example of independence, although in many cases comorbidities would be relevant with cancer also.

The classification of different diseases in one of the three categories is ultimately an empirical matter.

#### 2.2 Characteristics of the screening process

The default situation is one where the individual does not participate in preventive care. If the individual is hit by a disorder, she will find herself either in the late stage of the disease (m = l), which requires curative treatment (e.g. for the flu), or, in the case of a fatal disorder, she dies (e.g. for certain cancers). Her expected utility in the non-participation case can therefore be written as

$$EU^{non-participation} = (1-p) u^{HE} + p u^{S}, (5)$$

where the utilities in the healthy and sick states are given respectively by

$$u^{HE} = u(y,h,n) + \beta(1-p_{x,t+1})V_{t+1}$$
(6)

$$u^{S} = I(nf) \left[ u(y - c_{l}, h, l) + \beta (1 - p_{x,t+1}) V_{t+1} \right]$$
(7)

The individual has the opportunity to participate in primary or secondary preventive care. Primary prevention is aimed at avoiding or reducing the occurrence of a disease, e.g. through immunization. Secondary prevention aims to reduce the health consequences of a disease by early detection and treatment, e.g. cancer screening. First, we discuss the latter type and then show how the formal model can be reinterpreted to integrate the former.

#### 2.2.1 Secondary prevention

Secondary prevention allows early treatment of the disease (m = e) at a lower cost of treatment  $c_e < c_l$ . Let us take breast cancer screening as an example. In the typical case, mammograms are used in the first round. There are alternatives, such as self-control of the breasts or examination of the breasts by the general practitioner (GP). These different techniques entail different monetary, psychological (e.g. fear), physical (e.g. pain) and transaction costs (e.g. waiting and travel time). On the other hand, prevention can also give positive emotions such as reassurance. We indicate the intensity of the testing procedure by  $\alpha_1 > 0$ , the monetary cost by  $c_{\alpha 1}$  and the psychic cost by  $f(\alpha_1)$  (with  $\frac{\partial f(\alpha_1)}{\partial \alpha_1} > 0$ ). In many cases (including that of breast cancer), the first test round is not perfectly accurate and all individuals with a positive first diagnosis participate in a second test (e.g. breast tissue biopsy) with intensity  $\alpha_2$ , which is assumed to give perfect information. If the original diagnosis is confirmed, the individual is treated early at cost  $c_e$ . If the first test is negative, no subsequent test is taken. The additional monetary and psychic costs for the second round are given by  $c_{\alpha 2}$  and  $g(\alpha_1, \alpha_2)$  respectively with  $g_2(\alpha_1, \alpha_2) > 0$ . To cover the different cases we introduce an indicator function I(SR), taking the value 1 if the screening procedure involves a second round and zero otherwise. We thus write total monetary and psychic costs as  $c_{\alpha 1} + I(SR)c_{\alpha 2}$  and as  $f(\alpha_1) + I(SR)g(\alpha_1, \alpha_2)$  respectively.

In this setup, four states of the world are possible. Firstly, the disorder is correctly detected and treated early (true positive, TP). A second possibility implies that the disorder is falsely suggested in the first round while the individual does not have the disorder (false positive, FP); if no second round follows, the individual will be treated as if she actually suffers from the disorder. In a third state, the test rightly shows that the individual does not suffer from the disorder (true negative, TN). Lastly, the disorder can go undetected and evolves into the late or deadly stage (false negative, FN). The utility consequences of these different states are as follows:

$$u^{TP} = u(y - c_{\alpha 1} - c_e - I(SR)c_{\alpha 2}, h, e) - [f(\alpha_1) + I(SR)g(\alpha_1, \alpha_2)] + \beta(1 - p_{x,t+1})V_{t+1}$$
(8)

$$u^{FP} = u(y - c_{\alpha 1} - c_e - I(SR)(c_{\alpha 2} - c_e), h, n) - [f(\alpha_1) + I(SR)g(\alpha_1, \alpha_2)]$$

$$+\beta(1 - p_{x,t+1})V_{t+1} \tag{9}$$

$$u^{TN} = u(y - c_{\alpha 1}, h, n) - f(\alpha_1) + \beta(1 - p_{x,t+1})V_{t+1}$$
(10)

$$u^{FN} = I(nf) \times [u(y - c_{\alpha 1} - c_l, h, l) + \beta(1 - p_{x,t+1})V_{t+1}] - f(\alpha_1)$$
(11)

The probabilities of ending up in these different states depend on the effectiveness of the screening/prevention program. We model this effectiveness with the indicators that are used in the medical literature. Test sensitivity ( $se \in [0,1]$ ) is the probability that a test will be positive for an ill individual; test specificity ( $sp \in [0,1]$ ) is the probability that the test yields a negative result for an individual without the disorder. In terms of the numbers of true positive, false positive, true negative and false negative tests, these indicators can be expressed as:

$$se = \frac{N^{TP}}{N^{TP} + N^{FN}} \tag{12}$$

$$sp = \frac{N^{TN}}{N^{TN} + N^{FP}} \tag{13}$$

We can then write the probabilities to fall into a certain utility state in terms of p, sp and se:

$$p^{TP} = p \times se \tag{14}$$

$$p^{FP} = (1-p) \times (1-sp)$$
 (15)

$$p^{TN} = (1-p) \times sp \tag{16}$$

$$p^{FN} = p \times (1 - se) \tag{17}$$

From the point of view of the patient, test specificity sp is only relevant for those who are not hit by the disease, and its relative importance is given by the difference between  $u^{TN}$  and  $u^{FP}$ . On the other hand, test sensitivity se is important for those who are hit by the disease. In utility terms, the relevance of a larger value for se is expressed by the difference between  $u^{TP}$  and  $u^{FN}$ . If the first round gives perfect information we have  $N^{FP} = N^{FN} = 0$  and sp = se = 1. This makes the second round superfluous (I(SR) = 0).

In principle, one can expect that the medical prevention technology implies a positive relationship between *se* and *sp* on the one hand and test intensity  $(\alpha_1, \alpha_2)$  on the other. This relationship need not be monotonic, however. We will treat *se*, *sp*,  $\alpha_1$ , and  $\alpha_2$  as independent characteristics of the prevention process, but return to the possible relationships between them in the interpretation of our results.

#### 2.2.2 Primary prevention

Primary prevention (for example vaccination programs) reduces the probability of developing a disorder. We assume that a disease does not occur when vaccination is effective. Otherwise, the sick patient will be referred to late-stage treatment (or will die if the disease is fatal). Primary prevention involves only one round, i.e. I(SR) = 0. In this context, the notion of a false positive does not make sense; therefore  $N^{FP} = 0$  and sp = 1. There are three possible outcomes. The first is the situation where the individual would not have been hit by the disease, even if she were not vaccinated. This state can be called "true negative" as before, with utility  $u^{TN}$  as in eq. (10). It occurs with probability (1-p), as in eq. (16) with sp = 1. The second is the state where vaccination is not effective. This is analogous to the "false negative" for secondary prevention. It occurs with probability  $p^{FN}$  and the resulting utility is given by  $u^{FN}$  in eq. (11). When the disease would have occurred without vaccination, but the latter is effective, we are in a third state occurring with probability  $p \times se$ . This is analogous to the (effective) "true positive" state in the case of secondary prevention, but with a different utility outcome:

$$u^{EF} = u(y - c_{\alpha 1}, h, n) - f(\alpha_1) + \beta (1 - p_{x,t+1}) V_{t+1}$$
(18)

It is instructive to compare  $u^{EF}$  and  $u^{TP}$  in eq. (8) with I(SR) = 0. The "best" possible situation in the case of secondary prevention is that of early treatment, whereas the best possible situation with primary prevention is the avoidance of the disease. The latter therefore makes it possible to realise an additional utility gain. We can integrate primary and secondary prevention by rewriting  $u^{TP}$  from eq. (8) as follows

$$u^{TP} = u(y - c_{\alpha 1} - c_e - I(SR)c_{\alpha 2}, h, e) - [f(\alpha_1) + I(SR)g(\alpha_1, \alpha_2)] + \beta(1 - p_{x,t+1})V_{t+1} + I(prim) [u(y - c_{\alpha 1}, h, n) - u(y - c_{\alpha 1} - c_e - I(SR)c_{\alpha 2}, h, e)],$$
(19)

where the indicator function I(prim) = 1 in the case of primary prevention and zero otherwise. The last term between square brackets is the utility gain realized through primary prevention.

To improve the comparability of the states between primary and secondary prevention, we rewrite  $u^{FP}$ from eq. (9) in such way that it reduces to  $u^{TN}$  in case of primary prevention (I(SR) = 0, I(prim) = 1):

$$u^{FP} = u(y - c_{\alpha 1} - (1 - I(prim))(c_e + I(SR)(c_{\alpha 2} - c_e)), h, n) - [f(\alpha_1) + I(SR)g(\alpha_1, \alpha_2)] + \beta(1 - p_{x,t+1})V_{t+1}$$
(20)

#### 2.2.3 The full model

Bringing all the elements from this section together, we can formulate the expected utility in case of participation in a preventive care program:

$$EU^{participation} = p \times se \times u^{TP} + (1-p) \times (1-sp) \times u^{FP} + (1-p) \times sp \times u^{TN} + p \times (1-se) \times u^{FN}$$

The individual will participate in prevention if  $\Delta EU > 0$ , with

$$\Delta EU = EU^{participation} - EU^{non-participation}$$
  
=  $p \times se \times u^{TP} + (1-p) \times (1-sp) \times u^{FP} + (1-p) \times sp \times u^{TN}$   
 $+p \times (1-se) \times u^{FN} - p \times u^{S} - (1-p)u^{HE}.$  (21)

We have used three indicator functions to distinguish different possibilities: I(nf) = 1 for non-fatal diseases, I(SR) = 1 for a prevention procedure in two rounds and I(prim) = 1 in the case of primary prevention (which also involves I(SR) = 0 and sp = 1).

Before we analyse the prevention decision in detail, it is useful to consider the relative ranking of the different states. From eqs. (6)-(7), (9)-(11) and (19)-(20), it is immediately clear that  $u^{HE} > u^{TN} \ge u^{FP} \ge u^{TP}$  (where the equalities hold in case of primary prevention, i.e. if I(SR) = 0 and  $I(prim) = 1)^7$ , and that  $u^S > u^{FN}$ . It is sufficient to assume that  $u^{TP} > u^S$  to get a full ranking. For fatal diseases (with  $u^S = 0$ ), this assumption boils down to the innocuous premise that taking an effective preventive action to avoid death yields a positive utility outcome ( $u^{TP} > 0$ ). For non-fatal diseases, we derive from eqs. (19) and (7):

$$u^{TP} - u^{S} = \begin{cases} (1 - I(prim)) u(y - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h, e) \\ + I(prim) u(y - c_{\alpha 1}, h, n) - u(y - c_{l}, h, l) \\ - [f(\alpha_{1}) + I(SR)g(\alpha_{1}, \alpha_{2})], \end{cases}$$
(22)

A positive value implies that the utility gain due to early discovery and treatment instead of late treatment is larger than the psychological costs of the test. If this is not the case, non-participation will always be

<sup>&</sup>lt;sup>7</sup>Note that in the full model, all references to the false positive state disappear in the case of primary prevention (precisely because in the formal derivations  $u^{FP} = u^{TP} = u^{TN}$ ).

optimal. We can exclude this uninteresting case from our analysis and conclude that

$$u^{HE} > u^{TN} \geqslant u^{FP} \geqslant u^{TP} > u^S > u^{FN}.$$
(23)

The interpretation of the decision rule (21) becomes more convenient when we rewrite it as

$$\Delta EU = (1-p) \times \left[ u^{FP} - u^{HE} \right]$$
  
+  $p \times \left[ u^{FN} - u^{S} \right]$   
+  $(1-p) \times sp \times \left[ u^{TN} - u^{FP} \right]$   
+  $p \times se \times \left[ u^{TP} - u^{FN} \right].$  (24)

The first two terms in eq. (24) are obviously negative. They indicate the probability weighted utility loss due to a wrong screening diagnosis or an ineffective or unneccessary primary preventive action. The last two terms are positive and represent the utility gain from a correct diagnosis or successful preventive effort. Enhanced test specificity sp increases the probability of a correct diagnosis when one is not hit by the disease, and leads to a utility gain of  $(u^{TN} - u^{FP})$ . Increasing sensitivity se leads to more true positive results and less false negative results, and hence to a utility increase of  $(u^{TP} - u^{FN})$  when sick. This last term also includes the additional utility gain of primary prevention when I(prim) = 1.

## 3 Comparative statics of the prevention decision

Individuals that do not expect to die in the immediate future will be confronted for a given disease with multiple decision moments to participate in preventive care. The same decision problem will return in the next period and this process continues until the uncertain moment of death. This means that the individual decides whether or not to participate in prevention in the current period, taking into account future utility and future preventive effort. To model this full process, one would need a multi-period model. However, such a multi-period model is mathematically burdensome. We sketch its main features in Appendix 2, but here we focus on a simplified two period model that is sufficient to yield the main insights.

In this simplified model, we assume that the individual lives during two periods and dies at the end of the second period. In period 1, the individual decides whether or not to participate in the preventive program, while in period 2, the individual does not participate in prevention and simply gets utility from income and health. Of course, period 1 is the period of interest and in this period, we will analyse how differences or changes in individual characteristics and beliefs lead to adaptations in preventive behaviour.

The expected utility in period 2 is unaffected by individual behaviour and is characterized as follows:

$$V_2 = (1 - p_2) \times u(y_2, h_2, n) + I(nf) \times p_2 \times u(y_2 - c_l, h_2, l)$$
(25)

Let us now implement the decision rule (21) for the first period. This gives:

$$\Delta EU_{1} = p_{1} \times se \times \left\{ \begin{array}{ll} u(y_{1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h_{1}, e) - [f(\alpha_{1}) + I(SR)g(\alpha_{1}, \alpha_{2})] + \beta(1 - p_{x,2})V_{2} \\ + I(prim) [u(y_{1} - c_{\alpha 1}, h_{1}, n) - u(y_{1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h_{1}, e)] \end{array} \right\} \\ + (1 - p_{1}) \times (1 - sp) \times \left\{ \begin{array}{l} u(y_{1} - c_{\alpha 1} - (1 - I(prim)) (c_{e} + I(SR) (c_{\alpha 2} - c_{e})), h_{1}, n) \\ - [f(\alpha_{1}) + I(SR)g(\alpha_{1}, \alpha_{2})] + \beta(1 - p_{x,2})V_{2} \end{array} \right\} \\ + (1 - p_{1}) \times sp \times \{u(y_{1} - c_{\alpha 1}, h_{1}, n) - f(\alpha_{1}) + \beta(1 - p_{x,2})V_{2}\} \\ + p_{1} \times (1 - se) \times \{I(nf) [u(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l) + \beta(1 - p_{x,2})V_{2}] - f(\alpha_{1})\} \\ - (1 - p_{1}) \times [u(y_{1}, h_{1}, n) + \beta(1 - p_{x,2})V_{2}] \\ - p_{1} \times I(nf) \times [u(y_{1} - c_{l}, h_{1}, l) + \beta(1 - p_{x,2})V_{2}] \end{array} \right]$$

$$(26)$$

Remark that the expected utility for period 2 appears in the decision rule for period 1.

We will now derive the comparative statics for the utility difference  $\Delta EU_1$ . We first analyse the effects of the intertemporal structure of the problem, then consider the characteristics of the testing procedure and of the disease, and finally look at the personal characteristics income and health. To keep track of the reasoning, it may be convenient to look at Table 3, summarizing the results for the three cases that will be analysed in our empirical application: breast cancer screening (secondary prevention, fatal disease, comorbidities), dental caries screening (secondary prevention, non-fatal disease, complementarities) and flu vaccination (primary prevention, non-fatal disease, comorbidities).

The future It follows from (26) that

$$\frac{\partial \Delta E U_1}{\partial \beta} = p_1 \times se \times (1 - I(nf))(1 - p_{x,2})V_2 \ge 0$$
(27)

$$\frac{\partial \Delta E U_1}{\partial p_{x,2}} = -p_1 \times se \times \beta (1 - I(nf)) V_2 \leqslant 0$$
(28)

$$\frac{\partial \Delta E U_1}{\partial V_2} = p_1 \times se \times \beta (1 - I(nf))(1 - p_{x,2}) \ge 0$$
(29)

The future will only influence the prevention decision in the case of a fatal disease, i.e. if I(nf) = 0. Indeed, with a non-fatal disease, all the relevant consequences occur in the first period and every health state has the same prospects with respect to the future. For fatal diseases, prevention provides an opportunity to avoid death through early treatment and thus increases the probability to benefit from future utility. Participation in prevention rises as the present value of the utility gain related to prevention, increases. This happens when the level of future utility  $V_2$  or the weight  $\beta$  given to the future increase or the probability of dying from other causes  $p_{x,2}$  decreases.<sup>8</sup>

Characteristics of the testing procedure Starting from eq. (24), we derive

$$\frac{\partial \Delta E U_1}{\partial se} = p_1(u^{TP} - u^{FN}) > 0 \tag{30}$$

$$\frac{\partial \Delta E U_1}{\partial s p} = (1 - p_1)(u^{TN} - u^{FP}) \ge 0, \tag{31}$$

where the conclusions about the signs follow from eq. (23), and the equality in eq. (31) holds in the case of primary prevention. An improvement of preventive care characteristics, without additional monetary or psychological costs, always makes prevention more attractive.

It is instructive to look at the case of a perfect screening test with se = sp = 1. Opting to take this test results in

$$\Delta EU_1 = (1 - p_1)(u^{TN} - u^{HE}) + p_1(u^{TP} - u^S).$$

Even a perfect test will only be taken if  $(u^{TP} - u^S)$  is sufficiently large compared  $(u^{TN} - u^{HE})$ , i.e. to the monetary and psychological cost  $u(y_1 - c_{a1}, h_1, n) - f(\alpha_1) - u(y_1, h_1, n)$ . The relative importance of the latter term decreases with  $p_1$ .

The comparative static results are easy for the "cost" parameters  $\alpha_1, \alpha_2, c_{\alpha 1}$  and  $c_{\alpha 2}$ . We indeed have  $\frac{\partial u^{xx}}{\partial z} \leq 0$ , for  $z = (\alpha_1, \alpha_2, c_{\alpha 1}, c_{\alpha 2})$  and for xx = (TP, FP, TN, FN), with obvious equality for  $\alpha_2$  and

 $<sup>^{8}</sup>$  An important assumption that drives these results is that the frequency of prevention and the period of disease development coincide. If this is not the case, and e.g. prevention is recommended to be taken yearly while the disorder needs more than a year to develop from harmless to the late stage of the disorder, the prevention decision is taken in period 1 and potential curative treatment occurs in period 2. The consequence of this discrepancy is that the future will also matter for a non-fatal disease, and the marginal effects go in the same direction as described for a fatal disease.

 $c_{\alpha 2}$  if there is no second round. We therefore conclude that

$$\frac{\partial \Delta E U_1}{\partial \alpha_1} < 0 \tag{32}$$

$$\frac{\partial \Delta E U_1}{\partial c_{\sigma 1}} < 0 \tag{33}$$

$$\frac{\partial \Delta E U_1}{\partial \alpha_2} \leqslant 0 \tag{34}$$

$$\frac{\partial \alpha_2}{\partial EU_1} \leqslant 0, \tag{35}$$

where the equalities hold if I(SR) = 0. As could be expected, increased costs make preventive effort less attractive.

If an increase in  $\alpha_1(\alpha_2)$  leads to an increase in  $c_{\alpha 1}(c_{\alpha 2})$  the negative effects are reinforced. If, on the other hand, a policy change increases  $\alpha_1$  and/or  $\alpha_2$  and, at the same time, se and/or sp, positive and negative effects should be weighed against each other.

**Characteristics of the disease** The first relevant distinction is the one between fatal and non-fatal diseases. Starting from eq. (26), we see

$$\begin{aligned} \Delta EU_1 \mid_{fatal} -\Delta EU_1 \mid_{non-fatal} &= p_1 \times [u(y_1 - c_l, h_1, l) - u(y_1 - c_{\alpha 1} - c_l, h_1, l)] \\ &+ p_1 \times se \times [u(y_1 - c_{\alpha 1} - c_l, h_1, l) + \beta(1 - p_{x,2})V_2)] > 0. \end{aligned}$$

Not surprisingly, *ceteris paribus*, the expected utility of prevention is larger in the case of a fatal than a non-fatal disease.

The effect of a change in  $p_1$  is less straightforward. Taking the derivative of eq. (21), we get

$$\frac{\partial \Delta E U_1}{\partial p_1} = \left[ u^{HE} - (1 - sp)u^{FP} - sp \times u^{TN} \right] \\ + \left[ se \times u^{TP} + (1 - se)u^{FN} - u^S \right],$$
(36)

which has an obvious interpretation. The relative ranking of utility states in eq. (23) shows clearly that if the individual is healthy (states  $u^{HE}$ ,  $u^{TN}$ ,  $u^{FP}$ ), participation in prevention leads to additional costs and a utility loss, while if she is ill (states  $u^S$ ,  $u^{TP}$ ,  $u^{FN}$ ), it depends on the underlying parameters, such as the costs and the efficiency of the preventive procedures, whether prevention leads to a gain or a loss. As  $p_1$  increases there is a shift away from the utility loss when healthy, towards the utility gain or loss when sick. The former leads to a positive effect on participation in prevention, captured by the first term in eq. (36), while the latter may result in a positive or a negative effect on preventive behaviour, captured by the second term in eq. (36). The positive effect will dominate, i.e.  $\frac{\partial \Delta EU_1}{\partial p_1} > 0$ , for a fatal disease and for preventive procedures with a high sensitivity se and/or low screening costs  $c_{\alpha 1}$ . This can be formally shown by rewriting eq. (23) as follows:

$$\frac{\partial \Delta EU_1}{\partial p_1} = se(u^{TP} - u^{FN}) + u^{HE} - f(\alpha_1) - [(1 - sp) \times u^{FP} + sp \times u^{TN}] + I(nf) (u(y_1 - c_{\alpha 1} - c_l, h_1, l) - u(y_1 - c_l, h_1, l)).$$
(37)

The first and the second term in eq. (37) are positive.<sup>9</sup> This positive effect increases as *se* improves or *sp* decreases. For non-fatal diseases, we have to take into account the last term in eq. (37), which is negative. This negative term is, *ceteris paribus*, more important for low-income individuals or for individuals who are more risk averse with respect to income. However, in general it will be small, since preventive care is often subsidized or (partially) reimbursed, so that  $c_{\alpha 1}$  is small.

We can also draw conclusions for the effect of  $p_2$  on the probability of taking a preventive test in period 1. As noted before, it will only have an impact for fatal diseases. In that case, we get from eqs. (25) and (29) that

$$\frac{\partial \Delta E U_1}{\partial p_2} = -p_1 \times se \times \beta (1 - p_{x,2}) u(y_2, h_2, n) < 0$$

The intuition is obvious. Future utility  $V_2$  unambiguously decreases as  $p_2$  increases, since the individual is less likely to be healthy and more likely to be dead. As a result  $\Delta EU_1$  decreases and prevention becomes less interesting. This is in accordance with the conclusions from eq. (29)<sup>10</sup>

A last characteristic of the disease is the treatment cost, represented in the model by  $c_e$  and  $c_l$ . Starting from eq. (26), we get

<sup>&</sup>lt;sup>9</sup>Remark that  $u^{HE} - f(\alpha_1) - (1 - sp) \times u^{FP} - sp \times u^{TN} \ge 0 \Leftrightarrow u(y,h,n) \ge (1 - sp) \times [u(y - c_{\alpha 1} - (1 - I(prim))(c_e + I(SR)(c_{\alpha 2} - c_e)),h,n) - I(SR)g(\alpha_1,\alpha_2)] + sp \times [u(y - c_{\alpha 1},h,n)].$ 

Given that the individual can choose to participate in prevention in period 2 as well, she can counter partly the utility loss due to an increased risk of illness.

$$\begin{array}{lcl} \displaystyle \frac{\partial \Delta EU_1}{\partial c_e} &=& -p_1 \times se \times \left(1 - I(prim)\right) \times u_1(y_1 - c_{\alpha 1} - c_e - I(SR)c_{\alpha 2}, h_1, e) \\ && -(1 - p_1) \times (1 - sp) \times (1 - I(prim)) \times (1 - I(SR)) \\ && \times u_1(y_1 - c_{\alpha 1} - (1 - I(prim)) \left(c_e + I(SR) \left(c_{\alpha 2} - c_e\right)\right), h_1, n) \\ &\leqslant& 0 \\ \\ \displaystyle \frac{\partial \Delta EU_1}{\partial c_l} &=& I(nf) \times p_1 \times \left(u_1(y_1 - c_l, h_1, l) - (1 - se)u_1(y_1 - c_{\alpha 1} - c_l, h_1, l)\right) \end{array}$$

An increase in the cost of early treatment leads to a reduction in  $\Delta EU_1$  and, consequently, lowers the incentives for preventive action. The partial effect equals zero in the case of primary prevention, since early treatment does not exist in this setting. Higher curative (late stage) treatment costs have no effect for fatal diseases, since no cure is available. For non-fatal diseases the effect is ambiguous, since the costs can occur both in case of participation (state  $u^{FN}$ ) as in case of non-participation (state  $u^S$ ). However, if *se* is high enough and/or  $c_{\alpha 1}$  low, more expensive curative treatment increases the incentives for preventive effort. That was only to be expected. Prevention is the only possibility to avoid the larger cost, but this cost avoidance can only work if prevention is reasonably effective (*se* high enough) and screening costs are limited.

**Income and health** In general, the comparative statics effects of income and health depend on the cross-effect between both variables in the utility function. The empirical evidence on the sign of this cross-effect is mixed – and therefore most of the theoretical predictions remain ambiguous. To simplify, we impose in this section (as in the largest part of the theoretical literature) separability between the utility from income and from health, i.e. u(y, h, m) = v(y) + w(h, m). The results for the general model are given in Appendix 1. Using the separability assumption, we derive for actual income:

$$\frac{\partial \Delta E U_{1}}{\partial y_{1}} = (1 - p_{1}) \times (1 - sp) \times v_{1}(y_{1} - c_{\alpha 1} - (1 - I(prim))(c_{e} + I(SR)(c_{\alpha 2} - c_{e}))) \\
+ (1 - p_{1}) \times sp \times v_{1}(y_{1} - c_{\alpha 1}) \\
- (1 - p_{1}) \times v_{1}(y_{1}) \\
+ p_{1} \times I(nf) \times (v_{1}(y_{1} - c_{\alpha 1} - c_{l}) - v_{1}(y_{1} - c_{l})) \\
+ p_{1} \times se \times (1 - I(prim)) \times v_{1}(y_{1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}) \\
+ p_{1} \times se \times I(prim) \times v_{1}(y_{1} - c_{\alpha 1} - c_{l}) \\
- p_{1} \times se \times I(nf) \times v_{1}(y_{1} - c_{\alpha 1} - c_{l})$$
(38)

Given that  $v_1(.) > 0$  and  $v_{11}(.) \leq 0$ , the sum of the first three terms in this expression is positive. The next three terms are also positive. The last term is zero for fatal diseases. Therefore for fatal diseases the overall income effect is always positive. If the disease is non-fatal, this last term is negative and the sign of the overall income effect will depend on the relative size of the different monetary costs and benefits.

A first order Taylor expansion around  $y_1$  allows us to formulate approximate conditions for  $\frac{\partial \Delta E U_1}{\partial y_1}$  to be positive. The results are summarized in Table 1.<sup>11</sup> For non-fatal diseases, income will have a positive effect if the (private) monetary costs for participation in prevention (costs for screening, vaccination and unneccessary early treatment) outweigh the savings in terms of curative treatment costs. If monetary costs are larger than monetary benefits, this will have a negative effect on the incentives for prevention, and, with a concave utility function, the negative impact will be more pronounced for poorer persons. This explains the positive income effect on the expected utility gain from prevention. If costs are less than the benefits, an analogous reasoning yields a negative income effect. The conditions in Table 1 are easily interpreted. In most realistic cases of secondary prevention we may expect a positive income effect. If, for example, curative treatment and early treatment are equally well covered by health insurance, any monetary cost for prevention, as minor as it might be, leads to a positive income effect. In the case of primary prevention, the conditions for a positive income effect are stricter.

We can also draw conclusions about the effect of  $y_2$  on the expected utility gain of taking a preventive test in period 1. It will only have an impact for fatal diseases:

$$\frac{\partial \Delta EU_1}{\partial y_2} = p_1 \times se \times \beta (1 - p_{x,2})(1 - p_2)u_1(y_2, h_2, n) > 0$$

<sup>&</sup>lt;sup>11</sup>The details of the calculations are given in Appendix 3.

Disease type	I(nf) = 0, I(prim) = 0/1, $I(SR) = 0/1$	I(nf) = 1, I(prim) = 1,  I(SR) = 0	I(nf) = 1, I(prim) = 0, I(SR) = 1	I(nf) = 1, I(prim) = 0, I(SR) = 0
Taylor condition $y_1$	always positive effect	$c_{\alpha 1} \ge p_1 \times se \times c_l$	$\begin{array}{c} c_{\alpha 1} + c_{\alpha 2} \times (p_1 \times se + \\ (1 - p_1)(1 - sp)) \ge p_1 \times \\ se \times (c_l - c_e) \end{array}$	$c_{\alpha 1} + c_e \times (1 - p_1)(1 - sp) \ge p_1 \times se \times (c_l - c_e)$

Table 1: Taylor conditions for positive income effect of  $y_1$  on participation in period 1

By analogy with eq. (38), it is clear that  $\frac{\partial \Delta EU_1}{\partial y_2} \ge 0$  for a fatal disease, which allows us to conclude that the income effect of  $y_2$  has a positive effect on participation. The obvious intuition is that an income rise increases future utility and makes actual preventive effort more beneficial.

Keeping the assumption of additive separability of the utility function, the comparative static expressions for initial health  $h_1$  are given by:

$$\frac{\partial \Delta EU_1}{\partial h_1} = p_1 \times se \times (1 - I(prim)) \times w_1(h, e) + p_1 \times se \times I(prim) \times w_1(h, n) - p_1 \times se \times I(nf) \times w_1(h, l).$$
(39)

The sign of this expression depends heavily on the type of illness and prevention, as well as on the interaction between h and m as laid out in section 2. An overview of the different possibilities is given in Table 2. Note that these results offer an alternative explanation for the puzzle of Wu (2003), who found a positive effect of health for participation in breast cancer screening and a negative effect for flu vaccination. Wu pointed at psychological factors such as fear and anxiety, varying discount rates or GP advice according to health status to explain this discrepancy. Our model provides an easy explanation within the context of a standard expected utility model, based on the type of prevention and the disease's characteristics.<sup>12</sup> Comorbidities decrease the incentives for healthier individuals to participate in prevention, since they can recover more easily. Complementarities have the opposite effects on preventive behaviour. This mechanism breaks down if the disease has a fatal outcome.

<sup>&</sup>lt;sup>12</sup>Similar arguments are given by Mullahy (1999) and Maurer (2009).

Table 2: Overview of the expected effect of health on preventive action according to disease and prevention type

Disease type	I(nf) = 0, I(prim) = 0/1,	I(nf) = 1, I(prim) = 0/1,
Interaction health	I(SR) = 0/1	I(SR) = 0/1
Complements	positive	positive
Comorbidities	positive	negative
Independence	positive	no effect

The effect of future health on participation in prevention is similar to the effect of future income. A better general health status in the future makes it worthwhile to pursue prevention in the current period. This has however only an impact for fatal diseases.

$$\frac{\partial \Delta E U_1}{\partial h_2} = p_1 \times se \times \beta (1 - p_{x,2})(1 - p_2) u_2(y_2, h_2, n) > 0$$
(40)

## 4 Empirical analysis

For our empirical illustration, we analyse three types of disorders and their corresponding preventive care programs: breast cancer, dental caries and flu. In the next subsection we briefly describe these three disorders and we summarize the corresponding behavioural hypotheses. We then present the available data used in the empirical analysis. Finally, we present the results.

#### 4.1 Setup of the empirical exercise

#### 4.1.1 Three procedures

Breast cancer: I(nf) = 0, I(prim) = 0, I(SR) = 1, comorbidities or independence in health. Breast cancer is the most common cancer among European women. It accounts for almost one in three new cancer cases and one in six cancer deaths. One in nine women develops breast cancer at some point in her life, and this fraction has increased over the years. Although primary prevention is not yet an option, it is possible to detect breast cancer and the chances of survival increase the earlier the cancer is treated. For this reason, many countries have set up a preventive screening program. The most common prevention program consists of two screening rounds. Given the nature of breast cancer, we assume that late treatment of cancer results in death during the period.

**Dental caries:** I(nf) = 1, I(prim) = 0, I(SR) = 1, complements in health. The second application of our general model is preventive dental care. In comparison with cancer screening, dental

program is a less common and a less well-known secondary preventive care program. The set up of the program is as follows. An (asymptomatic) individual visits the dentist preventively (without feeling pain or having dental-related problems). The dentist screens for dental caries and dental plaque. If the dentist observes irregularities (first round), an X-ray picture (second round) will be taken. The second round either confirms (true positive) or disproves the first observation (false positive). The difference to breast cancer screening is that the individual will not die from a dental disorder. In the case of no prevention or a false negative result, there will be curative treatment of the advanced dental problem.

Flu: I(nf) = 1, I(prim) = 1, I(SR) = 0, comorbidities in health. Our final application refers to flu vaccination. This is one of the best-known and most studied examples of primary preventive care. Flu is a common infectious disease that causes general discomfort for most and death for some. In line with the public opinion, we consider flu to be a non-fatal disease. Since the disease is infectious, immunization brings about positive externalities. Most developed countries provide subsidized vaccination programs for certain vulnerable groups within the population, such as chronically ill individuals or the elderly. In addition to government programs, a number of companies also provide vaccination programs.

#### 4.1.2 Hypotheses and empirical specification

Participation in prevention is a discrete decision. In our theoretical model we assumed that individual *i* participates if  $\Delta EU_1^i > 0$ , with  $\Delta EU_1^i$  given in eq. (26). Adding a stochastic component  $\varepsilon_i$  capturing idio-syncratic factors, missing variables and measurement errors, we can write the probability of participation as

$$P(i \ participates) = P(\Delta EU_1^i + \varepsilon_i > 0) = P(\Delta EU_1^i > -\varepsilon_i).$$

If we assume the random term to be normally distributed, this results in a standard probit model. The comparative static hypotheses about  $\Delta EU_1$ , as derived in the previous section, can then be rephrased directly as hypotheses on the probability of participation.<sup>13</sup>

Table 3 gives an overview of these hypotheses. We distinguish between hypotheses that will be tested directly with the available data, hypotheses that will be "tested" indirectly through (sometimes remote) proxies and hypotheses that cannot be tested.

 $<sup>^{13}</sup>$ Belkar et al. (2006) show that neglecting to distinguish between "aware" and "unaware" individuals may lead to a selection effect. However, they also show that the problem is not very serious if "censoring is modest and positive dependence between awareness and choice is substantial" (p. 44). This is likely to be the case with our data.

Effect on participation		Disorder					
in prevention (period 1)	Breast cancer	Dental caries	Flu				
Hypotheses (tested directly)							
Increase $h_1$	positive	positive	negative				
Increase $y_1$	positive	ambiguous (likely positive)	> ambiguous (likely positive)				
Increase $p_{x,2}$	negative	no effect	no effect				
Hypotheses (tested indirectly)							
Increase $\beta$	positive	no effect	no effect				
Increase $\alpha_1$	negative	negative	negative				
Increase $c_{\alpha 1}$	negative	negative	negative				
Increase $p_1$	positive	ambiguous (likely positive)	ambiguous (likely positive)				
Increase $V_2$	positive	no effect	no effect				
Increase $p_2$	negative	no effect	no effect				
Increase $y_2$	positive	no effect	no effect				
Hypotheses (not tested)							
Increase se	positive	positive	positive				
Increase sp	positive	positive	-				
Increase $\alpha_2$	negative	negative	-				
Increase $c_{\alpha 2}$	negative	negative	-				
Increase $c_l$	no effect	ambiguous (likely positive)	ambiguous (likely positive)				
Increase $c_e$	negative	negative	-				

Table 3: Overview of the theoretical hypotheses

#### 4.2 Data

Our individual microdata are taken from SHARE. We combine them with information about the specific features of the prevention programs in different countries, which has been collected from macrosources. Table 4 gives an overview of the relevant data and shows how they are related to the variables in our theoretical model.

Individual data Our individual data come from the first (2004-2005) and second (2006-2007) wave of the Survey on Health, Ageing and Retirement in Europe (SHARE). SHARE is a micro-data set, targeted at individuals aged 50 years and over. It covers more than 30,000 non-institutionalized individuals from 14 European countries and Israel. A household is selected in a random procedure, but with the specific requirement that at least one individual is aged 50 years or over. SHARE provides comparable and detailed individual and household information. A full description can be found in Börsch-Supan et al. (2005).

The dependent variables are binary variables equal to one if the individual has had a specific type of prevention in the last (two) year(s). The type of procedures include mammograms for women, preventive dental care<sup>14</sup> and flu shots. Reported participation rates are 54%, 41% and 33% respectively. Despite using two rounds of SHARE, no panel structure can be easily implemented. Data on participation in breast

 $<sup>^{14}</sup>$  We set preventive dental care equal to one if individuals reported visting a dentist in the last twelve months for preventive use or prevention and treatment combined. The value is set to zero if the individual has not seen a dentist or has seen them only for treatment. Our empirical results are not very different when using an alternative specification with a value equal to one if the dentist is contacted for prevention use only and zero otherwise.

#### Table 4: Overview of the data

		Disorder						
Data	Breast cancer	Dental caries	Flu					
$h_1$		– Subjective health status						
	– Objective	e health variables: ADL, BMI,	Specific diseases					
$y_1$	– Equ	ivalent household income, broa	adly defined					
$\beta$		– Expressed hope for the fut	ture					
$p_{x,2}$	- M	ortality risk over the next 1,2,	5,10 years					
$\alpha_1$	– Probability of receiving		– Belonging to country risk					
	an invitation letter		group for flu					
	– Population based pro-							
	gram completed							
$c_{\alpha 1}$			– Free or subsidized vacci-					
			nation					
$p_1, p_2$		- Age						
	– Past cancer diagnosis	- Dentures	- Belonging to country risk					
			group for flu					
	– Age and country specific	– Trouble biting						
	breast cancer incidence and							
	mortality rates							
$V_2$		– Age						
$y_2$	- Equ	ivalent household income, Edu	ication, Age					
Other	Education, Partner, House o	wner, Nationality, Gender, Sm	oker, Wave and Country dummies					

cancer screening<sup>15</sup> and flu vaccination were collected through a self-administered drop-off questionnaire.<sup>16</sup> No respondent received the drop-off questionnaire in both waves, therefore we are limited to a pooled cross-sectional analysis for breast cancer screening and flu vaccination. In the case of dental prevention, an important number of individuals answered the question on participation in preventive dental care in both waves. We account for this by pooling all observations but correcting for the correlation between the answers of the individuals that have two entries.

As for the explanatory variables, we are particularly interested in variables that allow us to distinguish between the different models: health state, discount factor, mortality risk and income. We supplement this with various control variables.

SHARE contains subjective and objective health information. We created a dummy variable "sick" for individuals who report to have a fair to poor health (as opposed to good, very good or excellent health). The objective health information comes from an index of limitations to six activities of daily living<sup>17</sup>, the BMI score and the presence of specific diseases. A higher score corresponds to more limitations.

 $<sup>^{15}</sup>$  For breast cancer screening, we restrict our sample to women without a history of breast cancer.

 $<sup>^{16}</sup>$  All other SHARE data discussed below were collected using a computer assisted personal interviewing (CAPI) program. A self-administered drop-off questionnaire can be biased, since lower socio-economic groups tend to be underrepresented. Therefore, the answers to the drop-off questionnaire might not be representative of the population. However, Jusot et al. (2012) point out that prevalence rates obtained in the drop-off questionnaire correspond to available published OECD population data for most countries.

 $<sup>^{17}</sup>$  The activities that are used are: dressing, walking across a room, bathing or showering, eating, getting in and out of bed and using the toilet.

There is no good information about the subjective discount factor, but SHARE contains a dummy variable equal to one if the individual expresses hope or a desire for the future. We (tentatively) interpret a positive value as indicating a more future-oriented attitude, i.e. a larger  $\beta$ .

The mortality risk  $(p_{x,2})$  is captured by the question: "What is the chance that you will live to be age X or more?". We adjust 'certain' answers of 0 and 100 percent survival to slightly uncertain answers of 0.01 and 99.99 percent. The age of the individual, the probability and age of survival allow us to estimate an individually comparable probability of dying in the next ten years using a Weibull specification. In order to compute the entire Weibull distribution, one would need additional information, such as the answer to the same question for another age of survival. This information is lacking. However, the lack of information can be overcome by an assumption on the death rate. We assume that older people are more likely to die, and different values are tried out to approximate the shape of this relation.<sup>18</sup> There exists doubt as to whether or not the answers to survival questions have predictive value for real longevity. Moreover, sceptics point at a heaping of responses at focal-point values of 0, 50 or 100 percent, which hints at biased responses.<sup>19</sup> For our purpose, however, it is not crucial whether or not individual beliefs are an accurate reproduction of reality, since the prevention decisions of individuals will be influenced by their subjective beliefs including biases.

Income is broadly interpreted as equivalent household income (using the square root equivalence scale), comprised of labour and retirement income as well as income from wealth (dividends, rental income etc.). We use reported (not imputed) income data and filter out households with zero or extremely high reported income. All amounts are expressed in euros using the exchange rates provided by SHARE, and subdivided into deciles across the different European countries.

Other individual variables used in the empirical model are age (in classes of 5 years), education (ISCED-97 scale, with levels 5 and 6 merged), gender, partner, house owner, nationality, (past) smoker. These control variables capture elements of awareness, prevalence, need, subjective beliefs and risk aversion. Moreover, age is also an indirect measure of future utility  $V_2$  and education can be interpreted as an indicator of future income  $y_2$ .

Finally, there are a limited number of variables specific to the type of prevention. In the empirical

<sup>&</sup>lt;sup>18</sup> The CDF of the Weibull distribution has the following form:  $1 - e^{-\left(\frac{x}{\lambda}\right)^k}$  with k the shape parameter or death rate, x the time to death, and  $\lambda$  the scale parameter. With the survival probability and two age points, i.e. current age and age of survival, we can compute x and either  $\lambda$  or k, but we need an assumption on the other parameter. The death rate is more suitable for assumptions than the scale parameter. k = 1 implies a constant death rate at all ages, while k > 1 corresponds to an increase of the death rate with age. In our empirical analysis we perform a sensitivity analysis for  $k \in [1, 4]$ . We use k = 2 as standard value in the empirical results.

<sup>&</sup>lt;sup>19</sup>On the other hand, an individual has access to superior information about herself than is incorporated in a life table. For a discussion, see e.g. Peracchi & Perotti (2011) or Wübker (2012b). Peracchi & Perotti (2011) using SHARE data and Smith et al. (2001) using HRS data find evidence that subjective beliefs about longevity relate to observed survival patterns.

analysis of breast cancer prevention, we take up an indicator for whether or not the individual has had a positive cancer diagnosis (except breast cancer) in the past. We believe that the experience of another cancer will increase the subjective belief (and/or objective risk) of developing breast cancer. The model for dental prevention is enlarged with a variable indicating whether or not the individual experiences biting problems and has dentures.

**Macro data** Previous work with SHARE (see, e.g., Schmitz and Wübker, 2011) has introduced a wave dummy and country dummies to estimate the effect of intercountry variation that is not captured by the included variables. Yet these all-embracing country dummies do not allow one to distinguish between cultural, policy related or other behavioural differences across countries. While they are necessary (and are also present in our model), we enrich the SHARE data with information about health policies and health indicators from other sources. These can be seen as rough and indirect measures of  $\alpha_1$  and  $c_{\alpha_1}$ . These additional data are not individual specific but group or region specific. Due to missing data or lack of comparable information on health policies, Israel and Switzerland are left out of the analysis and only data from the 13 remaining countries are used (Austria, Belgium, Czech Republic, Denmark, France, Germany, Greece, Ireland, Italy, the Netherlands, Poland, Spain, Sweden).

For breast cancer, the WHO GLOBOCAN project provides age and country specific information on incidence and mortality rates for 2008. These rates are expressed as cases per 100,000 individuals, but we rescale them to cases per 1,000 individuals. The report on cancer screening in the European Union gives information on the type of screening program (population-based or opportunistic<sup>20</sup>) and the implementation status<sup>21</sup> (von Karsa et al., 2008). In Germany, Denmark and Italy, population based programs are administered at a regional level, with varying progress in program implementation. We include the region-specific information on the implementation status of the breast cancer screening program in our dataset. Moreover, von Karsa et al. provide details on the country target group for screening and on the chances of receiving an invitation letter per country. All of this information was matched with the characteristics of the individuals in our sample. For Spain and Sweden, regional differences in target group definitions were taken into account.

 $<sup>^{20}</sup>$ By population-based screening, we refer to an organized screening program (with a specified target group, a specific screening test, intervals, quality assurance, monitoring and other procedures) managed by an organization at a national or regional level. In addition to the high degree of organization, every eligible individual served by the screening program is individually identified and personally invited to attend screening. Opportunistic screening on the other hand refers to screening outside an organised program and without personal invitation. The initiative to perform a screening examination is taken either by the individual or the health-care provider. Opportunistic screening may or may not be performed according to the public screening policy (if one exists), e.g. it may be applied to individuals outside the targeted population or according to a different screening technique.

 $<sup>^{21}</sup>$ It takes time to set up a population-based program. By implementation status, we refer to the progress made in this process. The starting point is a planning phase, followed by a pilot project, a rollout over the entire region/country and finally a completed population-based screening program.

Comparable information for dental prevention is limited or inaccurate. Therefore, we do not add additional macro information to the available micro data.

Information on influenza vaccination policies and country differences can be found on the website and in the publications from the VENICE project<sup>22</sup>. We use information on two types of country specific definitions of target groups, eligible for vaccination: a first based on age (e.g. individuals aged 65 and over), a second based on existing medical conditions (e.g. individuals with chronic lung diseases)<sup>23</sup>. In addition, we can distinguish three reimbursement schemes: free vaccination, partially-subsidized vaccination, or no subsidies.

#### 4.3 Results

Table 5 presents the averaged individual marginal effects of the participation determinants for the different types of preventive care. As mentioned already, the regression results are controlled for country and wave specific unmeasured factors. We prefer the fixed effect specification to a random effects specification, since the latter assumes independence between the policy covariates and the country effects. In the case of dental prevention, we correct for the correlation between the responses of individuals that answered in both waves. The analysis is performed on a large number of individuals: 11,547 individuals for breast cancer screening, 34.620 individuals for dental prevention of whom 14,609 have entries in both wave 1 and 2, and 21,750 individuals for flu vaccination. The analysis confirms results previously obtained with SHARE by Schmitz and Wübker (2011), Jusot et al. (2012) and Wübker (2012a, 2012b), but some explanatory variables differ to match better our theoretical model.

**Direct testing of hypotheses** We hypothesized that background health would have a positive effect on screening for breast cancer and for dental caries and a negative effect for flu vaccination. These effects are indeed found in Table 5. Those reporting less than good health or having difficulties in activities of daily living, show a lower probability of participation in dental prevention and in breast cancer screening. The marginal effect of self-assessed health is not significant in the latter case. The effect of health on the participation in flu vaccination has the opposite sign. It is highly significant for self-reported health and significant at the 10% level for the ADL index.<sup>24</sup>

Controlling for education levels, we find overall a positive effect of income on participation in the

<sup>&</sup>lt;sup>22</sup>VENICE is an acronym for Vaccine European New Integrated Collaboration Effort.

<sup>&</sup>lt;sup>23</sup>In many countries a third group based on 'at-risk-jobs' exists.

 $<sup>^{24}</sup>$  SHARE also contains information on bmi and the presence of some specific diseases (e.g. diabetes, chronic lung disease, cardiac disease, asthma). Introducing this information does not add much to the explanatory power of our model. The effects of BMI or the specific diseases are in line with the effects of ADL and subjective health. Moreover the pattern of significant effects for the other variables changes only to a limited degree.

	breast cancer screening	dental prevention	flu vaccination
Variables	Marginal effects	Marginal effects	Marginal effects
Self-assessed health (Ref. $=$ good, v	very good or excellent)		
less than good	-0.003	$-0.024^{***}$	0.028***
Other health and behavioural indica	tors		
ADL index	$-0.081^{**}$	$-0.072^{***}$	$0.047^{*}$
smoked in the past	0.037***	0.006	0.025***
currently smokes	$-0.062^{***}$	$-0.067^{***}$	$-0.035^{***}$
Importance of the future			
prob. death in 10 years	$-0.064^{***}$	-0.013	0.016
expresses hope for future	0.029**	$0.038^{***}$	0.004
Education (Ref. = no degree)			
primary	0.029	0.029**	-0.014
lower secondary	$0.065^{***}$	0.075***	-0.018
upper secondary	0.102***	$0.127^{***}$	-0.005
post secondary, non tertiary	0.101***	0.131***	0.006
tertiary	0.102***	0.172***	0.008
Income (Ref. = decile 1)			
decile 2	-0.020	-0.006	-0.028*
decile 3	-0.024	-0.019*	-0.002
decile 4	-0.003	-0.003	0.004
decile 5	-0.004	-0.000	-0.002
decile 6	0.035*	0.004	0.001
decile 7	0.026	0.039***	0.024
decile 8	0.036*	0.040***	0.024
decile 9	0.030	0.053***	0.030**
decile 10	0.046**	0.053***	0.026*
$age (Ref. = 50 \ to \ 54)$	0.040	0.000	0.020
under $40$	$-0.223^{***}$	0.023	-0.038
40 to 44	-0.086**	$-0.048^{**}$	-0.037
45 to 49	0.006	-0.048 -0.030**	-0.037 $-0.039^*$
55 to 59	0.018	-0.030 -0.005	-0.039 $0.026^{**}$
60 to 64	0.005	-0.003 -0.004	0.020
65 to 69	-0.068**	-0.004 $-0.013^*$	0.106***
70 to 74	$-0.127^{***}$	-0.013 -0.044***	0.100 $0.165^{***}$
75 to 79	-0.127 -0.284***	-0.044 $-0.075^{***}$	0.206***
80 to 84	-0.284 · · · · · · · · · · · · · · · · · · ·	-0.075*** -0.114***	0.233***
	-0.379 $-0.396^{***}$	$-0.134^{***}$	0.233 $0.234^{***}$
85 or over Other indicators	-0.390	-0.134***	0.234
Other indicators	0.051***	0.001***	0.096***
partner	0.051***	$0.021^{***}$ $0.027^{***}$	0.026***
house owner	-0.003		-0.008
foreigner, EU nationality	-0.062	-0.022	-0.015
foreigner, non-EU nationality	$-0.170^{***}$	$-0.096^{***}$	$-0.069^{*}$
female	-	0.048***	0.021***

Table 5: Determinants in the take-up of prevention

\* p<0.10, \*\* p<0.05, \*\*\* p<0.01 Database: SHARE, wave 1 and wave 2

	breast cancer screening	dental prevention	flu vaccination
Variables	Marginal effects	Marginal effects	Marginal effects
breast cancer specific			
diagnosed cancer (except breasts)	0.084***	_	-
in country target group	0.063**	_	—
prob. receiving invitation letter	0.170***	_	—
pop. based program complete	0.188***	_	—
age and country specific incidence	-0.003	_	—
age and country specific mortality	$0.107^{*}$	_	—
dental specific			
dentures	_	$-0.122^{***}$	-
trouble biting	_	$-0.049^{***}$	-
flu specific			
risk group based on age	_	-	0.080***
risk group based on illness	_	_	$0.065^{***}$
free vaccination	_	_	$0.180^{***}$
subsidized vaccination	_	_	$0.089^{***}$
wave and country dummies (Ref. =	- Greece)		
Austria	0.173***	0.102***	$0.119^{***}$
Germany	-0.039	$0.295^{***}$	0.101***
Sweden	-0.042	0.310***	$0.149^{***}$
The Netherlands	-0.054	$0.267^{***}$	0.243***
Spain	-0.060	$-0.056^{***}$	$0.222^{***}$
Italy	-0.010	$-0.048^{***}$	0.208***
France	0.007	$-0.033^{***}$	$0.186^{***}$
Denmark	$-0.289^{***}$	$0.372^{***}$	$0.105^{***}$
Belgium	$-0.103^{**}$	$0.105^{***}$	$0.306^{***}$
Czech Republik	0.092***	$0.156^{***}$	$0.046^{**}$
Poland	0.001	$-0.118^{***}$	$-0.118^{***}$
Ireland	-0.076*	$0.048^{***}$	$0.252^{***}$
wave 1	-0.008	$-0.012^{***}$	0.010
Pseudo $R^2$	0.216	0.206	0.179
No. of observations	11,547	49,229	21,750

 $\frac{1}{p < 0.10, ** p < 0.05, *** p < 0.01}{Database: SHARE, wave 1 and wave 2}$ 

three cases. The effects are least pronounced for flu vaccination.<sup>25</sup> This is also in line with the theoretical predictions.

Also as expected, mortality risk over 10 years is an important predictor in the model of breast cancer screening, but has no significant effect in dental prevention or flu vaccination. Sensitivity analysis confirms these results. The signs and significance do not alter if we change the survival time from 10 years to 5, 2 or 1 year(s) nor if we change the death rate gradually from k = 1 to k = 4. Only in the model of flu vaccination, an increase in mortality risk over 10 years with  $1 \le k < 1.3$ , slightly increases the probability of vaccination at a significance level of 10%. However, since SHARE is oriented towards individuals aged 50 or over, higher values for k, i.e. an increase in the probability of dying as one ages, are more probable.

Indirect confirmation of hypotheses As described before, the proxy used to represent the discount factor is much less precise. It confirms the hypotheses for breast cancer screening and flu vaccination but, contrary to the hypothesis, expressing hope for the future has a positive effect on dental prevention. This is perhaps not surprising. As discussed in footnotes (4) and (8), we assume that the screening interval and the period of disease development coincide. For preventive dental care, this is not necessarily the case. Preventive check-ups are recommended on a yearly basis, whereas the development of dental carries to a severe problem can take up more than a year. Somebody that decides to participate in prevention now, has to realize she can avoid more important curative costs in a future period. This discrepancy between screening and disease development periods may explain why prospects for the future have a positive effect on prevention of dental caries.<sup>26</sup>

Age is an important predictor of participation in prevention. The age pattern in our data is depicted in Figure 1. Flu vaccination clearly increases with age while the age-profile for breast cancer screening shows an inverse U-shape with highest participation probabilities between the ages of 45 to 65. We also find an inverse, but less pronounced, U-shape for dental caries.

These patterns reflect a mixture of various effects. First, age is related to the probability of disease  $p_1$ . This has led in many countries to the targeting of prevention towards particular age groups, so that information and financial incentives interact with need. In our analysis, we controlled for age targeting in breast cancer screening and flu vaccination (see below). The strongly declining participation in breast cancer screening at higher ages cannot be explained by the change in  $p_1$ . The most obvious explanation

 $<sup>^{25}</sup>$  If we subdivide income into deciles by country instead of for all countries together, similar results apply.

 $<sup>^{26}</sup>$  A different explanation for a positive effect for expressing hope for the future on dental prevention is that while dental caries is (of course) a non-fatal disease, preventing it has still an "investment"-effect, which is absent from flu vaccination (that has to be repeated regularly). This aspect is missing in our model and is consistent with a positive effect on prevention of dental carries.

is the decline of future utility  $V_2$  as people grow older. This is a second important effect.<sup>27</sup> It does not play in the case of flu vaccination and it perhaps plays somewhat in the case of dental caries.

Education is a significant determinant of preventive care utilization. Participation in breast cancer screening is lowest for individuals with no degree or primary education, it increases significantly until the upper secondary level and then remains at the same (highest) level. Participation in dental prevention is even more strongly related to schooling, and increases with each additional step in the schooling system. The probability of vaccination is less influenced by education, although a significant difference exists between primary and lower secondary on the one hand, and tertiary education on the other. These results also reflect a mixture of different effects, such as awareness, understanding and risk assessment. Education may also capture "permanent income", and hence the effect of  $y_2$ .<sup>28</sup> We can test the relative importance of cognitive abilities, since SHARE provides information on recall, verbal fluency and mathematical reasoning.<sup>29</sup> As can be seen in Table 9 in Appendix 4, increased cognitive ability along all three dimensions has a positive effect on participation in dental prevention, while only verbal fluency has a significant positive effect on flu vaccination. Breast cancer screening is unrelated to the used indicators. In line with the results of Cutler and Lleras-Muney (2010), the education effect decreases when measures of cognitive ability are added to the model. Education effects drop by around 10% for breast cancer screening and 25% for dental prevention. The marginal effects of other variables are affected in magnitude as well, however without changing direction or significance levels.

For each type of prevention, we included specific variables. As shown in Table 5, most of these variables have significant effects. The same is true for the specific health policy variables, that can be linked to  $\alpha_1$  and  $c_{\alpha_1}$ .

An earlier diagnosis of non-breast cancer increases the probability of participation in breast cancer screening on average by 8 percentage points. This can be explained by higher subjective beliefs of developing breast cancer or by an increased attention on the part of the health care providers. Furthermore, we observe large positive effects on participation if the individual has a higher probability of receiving an invitation letter and if the country or region has fully enacted a population based program (vs. an ongoing enrollment or a non-population based program). Finally, we observe that the participation deci-

<sup>&</sup>lt;sup>27</sup>In a similar model, Howard (2005) derives the conclusion that there is a "maximum" age, above which screening is no 

on education.

 $<sup>^{29}</sup>$ Recall is measured by the number of words an individual can recall from a list of 10 words that has been shown some minutes before. Verbal fluency is defined as the number of different animals an individual can enumerate within one minute (with a maximum of 100). The mathematical index is calculated by means of four mathematics questions with varying difficulty. An individual who was able to correctly answer the most difficult question obtained a score of 1, an individual who was unable to answer the most difficult question, but could correctly answer the second most difficult question got a score of 0.75 and so on. An individual who could not answer any question correctly got a score of 0.

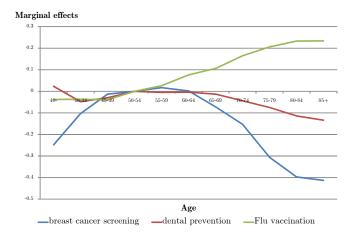


Figure 1: The marginal effect of age on participation in prevention.

sion is positively affected by age and country-specific mortality rates, while incidence rates do not have a significant effect.

Preventive dental care is negatively related to having dentures and having trouble biting. This is not surprising, since the former probably reduces the need for regular preventive care, while the latter requires curative rather than preventive care.

Many countries define different risk groups for flu vaccination based on age, on existing illnesses and on professions that have interactions with vulnerable groups. The exact definitions differ from country to country. We replicated the target groups based on age and existing illnesses for each country. The probability of taking up a vaccine increases if an individual belongs to a target group. Finally, we observe that monetary stimuli, such as free vaccination or subsidzed vaccination, clearly increase the probability of receiving flu shots and this on average by 18 and 9 percentage points respectively.

Lifestyle may be correlated with risk taking behaviour or with the assessment of probabilities. In all three models, the marginal effects for past smokers show an increased tendency towards participation (though non-significant for dental prevention), while current smokers have a lower participation probability. This is consistent with our model if past smokers acknowledge that their past behaviour gives them an increased health risk and they update their subjective beliefs correspondingly, while current smokers *ceteris paribus* apply lower subjective probabilities of health risk than their non-smoking counterparts.

Preventive behaviour is positively related to having a partner and negatively related to being of a non-EU nationality. The latter is not surprising, since transaction costs might be higher for foreigners: they profit less from information campaigns and are less familiar with local procedures and habits. A similar effect is observed for foreigners originating from other EU countries, but it is less pronounced and not significant. Women are *ceteris paribus* more likely to participate in flu vaccination and dental prevention.

We added country dummies to control for missing policy variables and cultural and behavioural differences. Denmark and Ireland lag behind the other countries in breast cancer screening participation because they combine a negative country effect with the lack of a completed population-based program. The negative country effects for Belgium, Sweden and the Netherlands are mitigated by a fully-implemented population-based screening program. Austrian women are *ceteris paribus* more likely to participate in breast cancer screening than their European peers. We observe large intercountry differences for dental prevention. The Northern European countries are characterized by relatively higher rates of dental prevention. Poland performs worst. Finally, we observe that Belgium, Ireland and the Netherlands *ceteris paribus* report the highest flu vaccination rates, whereas Poland and Greece lag behind.

Finally, SHARE also makes it possible to calculate an index of the quality of the doctor that is seen by the individual respondent. It has been shown by Maurer (2009), Schmitz and Wübker (2011) and Wübker (2012b) that the quality of the GP has a positive effect on participation in prevention.<sup>30</sup> This is easily understood, as good doctors will increase the awareness of the patients, or, when needed, will themselves take the initiative to suggest prevention. We also experimented with this variable, and it has a significant positive effect on participation for mammography and flu vaccination, while being insignificant for caries prevention. These results stand to reason. Introducing the variable does not alter considerably the overall pattern for the other variables (the most pronounced change is a decrease of the income effect for dental prevention). Since the new variable is not available for all individuals in all countries, using it reduces the number of observations by more than 20% for breast cancer screening and influenza vaccination and by more than 60% for dental prevention. We therefore decided not to include it in our reported results, but interested readers can find the results in Table 8 in Appendix 4.

## 5 Conclusion

We analysed participation in medical prevention with an expected utility model. Rather than focusing on one specific intervention, we aimed to explain the differences between different procedures within one coherent model. This model is sufficiently flexible to distinguish primary and secondary prevention (with one or two rounds) for either fatal or non-fatal diseases. Moreover, we integrated the idea of different disease types characterized by a different interaction with background health. The model yields different

 $<sup>^{30}\,\</sup>mathrm{We}$  adopt the physician quality index as proposed by Wübker (2012b).

predictions in the different cases. We tested these predictions with individual data from SHARE and the model performed reasonably well.

We believe that it is important to construct a flexible theoretical model. It allows the bringing together of various insights from the literature, and the validation of hypotheses for different cases, which is an essential test for the usefulness of a theoretical approach.<sup>31</sup> In this respect, the expected utility model (broadly interpreted) seems to be an interesting starting point for further developments. These developments should go in two directions.

First, on the theoretical side, there are by now sufficient indications in the literature that the expected utility model cannot explain all of the empirical regularities, not even when it is interpreted – as in our model – in a purely subjective way, taking due account of biases in the perception of costs and probabilities. It is definitely necessary to integrate the main insights from the behavioral literature. However, we do believe that also in this approach the focus should not be on specific papers describing specific *ad hoc* mechanisms to explain certain facts about one specific procedure, but on the construction of a more general model. The ultimate goal of theory is to bring some coherency into the mass of disparate obsevations.

Second, on the empirical side, we often had to rely on ill-defined proxies. Crucial parameters such as the subjective rate of time preference or the subjective perception of probabilities were not available in our data. Future work should try to collect direct measures of these parameters.<sup>32</sup> Using such well-designed measures would allow a more convincing testing of the hypotheses.

<sup>&</sup>lt;sup>31</sup>A similar position is taken by Howard (2005, p. 893).

 $<sup>^{32}</sup>$ Examples in the literature are Bradford et al. (2010) for time preferences and Carman and Kooreman (2011) for subjective probabilities.

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## Appendix 1: Alternative relation income and health

Empirically, there is no consensus upon the sign of the interaction between health and income in the utility function. We therefore assumed separability in our base model. In this appendix we explore alternative assumptions. A positive relationship implies  $u_{12}(y, h, m) > 0$ , i.e. an individual enjoys an additional unit of income more when she is in a better general health state, or vice versa, when the individual earns a higher income, she values health more. A negative relationship is defined as  $u_{12}(y, h, m) < 0$ , which means that an individual enjoys an additional unit of income more when she is in a worse general health state, or vice versa, when the individual has a lower income, she values health more.

The general comparative static results are (to be compared with eqs. (38) and (39)):

$$\frac{\partial \Delta EU_{1}}{\partial y_{1}} = p_{1} \times se \times \begin{cases}
(1 - I(prim)) \times u_{1}(y_{1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h_{1}, e) \\
+ I(prim) \times u_{1}(y_{1} - c_{\alpha 1}, h_{1}, n) \\
- I(nf) \times u_{1}(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l)
\end{cases} + (1 - p_{1}) \times \begin{cases}
(1 - sp) \times u_{1}(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l) \\
+ sp \times u_{1}(y_{1} - c_{\alpha 1}, h_{1}, n) \\
- u_{1}(y_{1}, h_{1}, n)
\end{cases} + p_{1} \times I(nf) \times \begin{cases}
u_{1}(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l) \\
- u_{1}(y_{1} - c_{l}, h_{1}, l)
\end{cases}$$
(41)

$$\frac{\partial \Delta EU_{1}}{\partial h_{1}} = p_{1} \times se \times \begin{cases}
(1 - I(prim)) \times u_{2}(y_{1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h_{1}, e) \\
+ I(prim) \times u_{2}(y_{1} - c_{\alpha 1}, h_{1}, n) \\
- I(nf) \times u_{2}(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l)
\end{cases} + (1 - p_{1}) \times \begin{cases}
(1 - sp) \times u_{2}(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l) \\
+ sp \times u_{2}(y_{1} - c_{\alpha 1}, h_{1}, n) \\
- u_{2}(y_{1}, h_{1}, n)
\end{cases} + p_{1} \times I(nf) \times \begin{cases}
u_{2}(y_{1} - c_{\alpha 1} - c_{l}, h_{1}, l) \\
- u_{2}(y_{1} - c_{l}, h_{1}, l)
\end{cases} \end{cases}$$
(42)

As can be seen in eqs. (41) and (42), there are three terms to be considered, the sign of which depends on the relation between y and h on the one hand, and between h and m on the other. An overview for

<b>Relation</b> $y$ and $h$	relation $h$ and $m$	Term 1	Term 2	Term 3	Overall effect
Positive	Complements	positive	negative	no effect	ambiguous
	Comorbidities	positive	negative	no effect	ambiguous
	Independence	positive	negative	no effect	ambiguous
Independence	Complements	positive	no effect	no effect	positive
	Comorbidities	positive	no effect	no effect	positive
	Independence	positive	no effect	no effect	positive
Negative	Complements	positive	positive	no effect	positive
	Comorbidities	positive	positive	no effect	positive
	Independence	positive	positive	no effect	positive

Table 6: Overview of the partial effects of health in case of a fatal disease

Table 7: Overview of the partial effects of health in case of a non fatal disease

<b>Relation</b> $y$ and $h$	<b>Relation</b> $h$ and $m$	Term 1	Term 2	Term 3	Overall effect
Positive	Complements	positive	negative	negative	ambiguous
	Comorbidities	ambiguous	negative	negative	ambiguous
	Independence	positive	negative	negative	ambiguous
Independence	Complements	positive	no effect	no effect	positive
	Comorbidities	negative	no effect	no effect	negative
	Independence	no effect	no effect	no effect	no effect
Negative	Complements	ambiguous	positive	positive	ambiguous
	Comorbidities	negative	positive	positive	ambiguous
	Independence	negative	positive	positive	ambiguous

health is given in Table 6 for a fatal disease and in Table 7 for a non-fatal disease.

The partial effects for income are less complicated. For a fatal disease, the effect of income is positive, no matter how the relationship between income and health is specified. For a non-fatal disease, the first term is ambiguous, while the second and third terms are positive. The overall effect is unknown. However, the first term can be ranked according to the relationship between y and m. The value of the first term and therefore the overall partial effect will be *ceteris paribus* higher the more positive the relationship between y and m.

## Appendix 2: The T period model

Our simplified two period model can be generalized to a multi-period model. In our approach, decisions in the different time periods are independent of past decisions. See, e.g. de la Mata (2011) and Etner and Jeleva (2012) for a richer dynamic specification. Take the number of periods to be T; T can be individually specific. We assume that in period T the individual dies, so that  $V_T = 0$ . We solve the problem backwards. We can define  $\Delta E U_{T-1}$  similarly to the expression in eq. (26):

$$\Delta EU_{T-1} = p_{T-1} \times se \times \left\{ \begin{array}{l} [u(y_{T-1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h_{T-1}, e) - f(\alpha_{1}) - I(SR)g(\alpha_{1}, \alpha_{2})] \\ + I(prim) [u(y_{T-1} - c_{\alpha 1}, h_{T-1}, n) - u(y_{T-1} - c_{\alpha 1} - c_{e} - I(SR)c_{\alpha 2}, h_{T-1}, e)] \end{array} \right\} \\ + (1 - p_{T-1}) \times (1 - sp) \times \left\{ \begin{array}{l} u(y_{T-1} - c_{\alpha 1} - (1 - I(prim)) (ce + I(SR) (c_{\alpha 2} - c_{e})), h_{T-1}, n) \\ - f(\alpha_{1}) - I(SR)g(\alpha_{1}, \alpha_{2}) \end{array} \right\} \\ + (1 - p_{T-1}) \times sp \times [u(y_{T-1} - c_{\alpha 1}, h_{T-1}, n) - f(\alpha_{1})] \\ + p_{T-1}(1 - se) (I(nf) u(y_{T-1} - c_{\alpha 1} - c_{l}, h_{T-1}, l) - f(\alpha_{1})) \\ - (1 - p_{T-1}) \times u(y_{T-1}, h_{T-1}, n) - p_{T-1}I(nf) u(y_{T-1} - c_{l}, h_{T-1}, l) \\ + p_{T-1} \times se \times (1 - I(nf)) \beta(1 - p_{x,T})V_{T} \end{array} \right.$$
(43)  
$$= \Delta CPEU_{T-1} \\ + p_{T-1} \times se \times (1 - I(nf)) \beta(1 - p_{x,T})V_{T}$$

We can subdivide  $\Delta EU_{T-1}$  into two terms: a first term captures the current period difference in expected utility ( $\Delta CPEU$ ) and a second term represents future utility. We can also define  $V_{T-1}$ , which captures expected utility from period T-1 onwards. Since expected utility depends on preventive behaviour, we introduce an indicator function  $I_{T-1}(part)$  that equals 1 if the individual participates in prevention in period T-1 (i.e.  $\Delta EU_{T-1} > 0$ ) and zero if the individual does not take part in prevention (i.e.  $\Delta EU_{T-1} < 0$ ).

$$V_{T-1} = (1 - p_{T-1}) \times u(y_{T-1}, h_{T-1}, n) + p_{T-1}I(nf) u(y_{T-1} - c_l, h_{T-1}, l) + I_{T-1}(part)\Delta EU_{T-1} = (1 - p_{T-1}) \times u(y_{T-1}, h_{T-1}, n) + p_{T-1}I(nf) u(y_{T-1} - c_l, h_{T-1}, l) + I_{T-1}(part)\Delta CPEU_{T-1} + I_{T-1}(part) \times p_{T-1} \times se \times (1 - I(nf)) \beta(1 - p_{x,T})V_T$$
(45)

It is clear from eqs. (44) and (45), that all references to future utility disappear for a non-fatal disease (I(nf) = 1). Therefore, in that case the prevention decision depends only on current period variables, and the comparative static results are the same as in the two period-model.

The analysis for fatal diseases is more challenging, since for them future utility will not disappear from the model if the individual participates in prevention. For any period t, we can characterize the decision process and the payoff as follows:

$$\begin{split} \Delta EU_t &= \Delta CPEU_t + p_t \times se \times \beta \times (1 - p_{x,t+1}) \times \\ &\sum_{i=t+1}^{T-1} \left[ I_i \left( part \right) \Delta CPEU_i + (1 - p_i) \, u_i^{HE} \right] \times \prod_{j=t+1}^{i-1} \left[ p_j \times se \times \beta \left( 1 - p_{x,j+1} \right) I_j \left( part \right) \right] \, (46) \end{split}$$
with
$$\begin{aligned} & (47) \\ \Delta CPEU_t &= p_t \times se \times \left\{ \begin{array}{c} \left[ u(y_t - c_{\alpha 1} - c_e - I(SR)c_{\alpha 2}, h_t, e) - f(\alpha_1) - I(SR)g(\alpha_1, \alpha_2) \right] \\ + I(prim) \left[ u(y_t - c_{\alpha 1}, h_t, n) - u(y_t - c_{\alpha 1} - c_e - I(SR)c_{\alpha 2}, h_t, e) \right] \end{array} \right\} \\ &+ (1 - p_t) \times (1 - sp) \times \left\{ \begin{array}{c} u(y_t - c_{\alpha 1} - (1 - I (prim)) \left( ce + I(SR) \left( c_{\alpha 2} - c_e \right) \right), h_t, n \right) \\ - f(\alpha_1) - I(SR)g(\alpha_1, \alpha_2) \end{array} \right\} \\ &+ (1 - p_t) \times sp \times \left[ u(y_t - c_{\alpha 1}, h_t, n) - f(\alpha_1) \right] - p_t(1 - se)f(\alpha_1) \end{aligned} \right\} \\ &+ (1 - p_t) \times u(y_t, h_t, n) \end{aligned}$$

$$V_t = \sum_{i=t}^{T-1} \left[ I_i \left( part \right) \Delta CPEU_i + (1 - p_i) \, u_i^{HE} \right] \times \prod_{j=t}^{i-1} \left[ p_j \times se \times \beta \left( 1 - p_{x,j+1} \right) I_j \left( part \right) \right] \end{aligned}$$

In the case of a fatal disease, expected utility consists of current period expected utility and future utility. Future utility becomes more important when the individual expects to live longer (T - t larger), when the individual is more future-oriented ( $\beta$  larger), when the mortality risk for other diseases is lower  $(p_{x,j+1} \text{ smaller})$  and when the benefit from prevention is more important  $(p_j \times se \text{ larger})$ . In the expression for future utility, we take into account the individual's future prevention decisions. If prevention has a positive payoff in the future, this payoff will be taken into account for current decisions as well.

**Comparative statics** The comparative statics are similar to those in the two period model. Note, however, that age (or, more accurately the individual's time horizon T - t), becomes relevant in the generalized model. In what follows, we look at the comparative statics of fatal diseases.

With respect to the **future**, we can conclude that the partial effects have the same sign, but since  $V_t$  gets smaller as an individual ages, the effect of  $\beta$  and  $p_{x,t+1}$  decreases over time.

For the **disease characteristics** and **treatment costs**  $z \in \{se, sp, \alpha_1, \alpha_2, c_{\alpha 1}, c_{\alpha 2}, c_e\}$ , we see that  $sgn\left(\frac{\partial \Delta CPEU_t}{\partial z}\right) = sgn\left(\frac{\partial \Delta CPEU_j}{\partial z}\right)$  with  $t \leq j < T$ , so that  $sgn\left(\frac{\partial \Delta EU_t}{\partial z}\right) = sgn\left(\frac{\partial \Delta CPEU_t}{\partial z}\right)$  and the partial effects have the same sign as in the two period model, but the time horizon and the future prevention decision will influence the magnitude of the effect. We assume for simplicity that the test characteristics and the costs of treatment are the same in each period. However, this assumption can be relaxed.

A higher subjective probability of having the disorder in period t still leads to an increase in

participation in the same period. However, the effect of  $p_{t+1}$  on the probability of participation in period t is not the necessarily the same as in the two periods model:

$$\frac{\partial \Delta EU_t}{\partial p_{t+1}} = p_t \times se \times \beta \times (1 - p_{x,t+1}) \times \left[ I_{t+1} \left( part \right) \frac{\partial \Delta CPEU_{t+1}}{\partial p_{t+1}} - u_{t+1}^{HE} \right] \\ + p_t \times se^2 \times \beta^2 \times (1 - p_{x,t+1}) \times (1 - p_{x,t+2}) \times I_{t+1} (part) \times \\ \sum_{i=t+2}^{T-1} \left[ I_i \left( part \right) \Delta CPEU_i + (1 - p_i) u_i^{HE} \right] \times \prod_{j=t+2}^{i-1} \left[ p_j \times se \times \beta \left( 1 - p_{x,j+1} \right) I_j \left( part \right) \right]$$

We can distinguish between two terms. On the one hand there is a direct effect in period t + 1, which is negative. If  $p_{t+1}$  increases, the individual is more likely to die, and utility decreases. This decrease cannot be countered by the direct positive effect of prevention in period t + 1. On the other hand, there might be an indirect effect of prevention in the subsequent periods. If the individual participates in prevention in period t + 1 ( $I_{t+1}(part) = 1$ ), she reduces the risk from dying and gains utility in periods t+2, t+3, ..., T-1. The total utility gained depends, amongst other factors, on the participation decision in the subsequent periods and the time horizon. The second term can have a positive indirect effect on the participation decision in period t. The overall effect is ambiguous.

The partial effect of current **income** does not change. The effect of a future marginal change in income  $y_j$  with t < j < T still has a positive effect on participation in the current period. Finally, the partial effect of both current and future **health** in case of a fatal disease is always positive, as long as health between the periods is independent or positively correlated.

## Appendix 3: First order Taylor expansion

We start from eq. (38) and perform a Taylor expansion around  $y_1$ , yielding:

$$\begin{aligned} \frac{\partial \Delta EU_1}{\partial y_1} &= v_1(y_1) \times p_1 \times se \times (1 - I(nf)) \\ &- v_{11}(y_1) \begin{bmatrix} c_{\alpha 1} \times (1 - p_1 \times (1 - se) \times (1 - I(nf))) \\ + c_{\alpha 2} \times (1 - I(prim)) \times (p_1 \times se \times I(SR) + (1 - p_1) \times (1 - sp) \times I(SR)) \\ + c_e \times (1 - I(prim)) \times (p_1 \times se + (1 - p_1) \times (1 - sp) \times (1 - I(SR))) \\ &- c_l \times p_1 \times se \times I(nf) \end{bmatrix} \end{aligned}$$

Since  $v_1(y_1) > 0$  and  $v_{11}(y_1) \leq 0$ , we can easily derive the conditions for  $\frac{\partial \Delta E U_1}{\partial y_1} \geq 0$  for all disease and prevention types.

For I(nf) = 0:

$$\begin{aligned} \frac{\partial \Delta EU_1}{\partial y_1} &= v_1(y_1) \times p_1 \times se \\ &-v_{11}(y_1) \begin{bmatrix} c_{\alpha 1} \times (1-p_1 \times (1-se)) \\ +c_{\alpha 2} \times (1-I(prim)) \times (p_1 \times se \times I(SR) + (1-p_1) \times (1-sp) \times I(SR)) \\ +c_e \times (1-I(prim)) \times (p_1 \times se + (1-p_1) \times (1-sp) \times (1-I(SR))) \\ &> 0 \end{aligned}$$

For I(nf) = 1, I(prim) = 1, I(SR) = 0:

$$\frac{\partial \Delta EU_1}{\partial y_1} = -v_{11}(y_1) \left[ c_{\alpha 1} - c_l \times p_1 \times se \right]$$
$$\frac{\partial \Delta EU_1}{\partial y_1} \geqslant 0 \Leftrightarrow c_{\alpha 1} - c_l \times p_1 \times se \geqslant 0$$
$$\Leftrightarrow c_{\alpha 1} \geqslant p_1 \times se \times c_l$$

For I(nf) = 1, I(prim) = 0, I(SR) = 1:

$$\begin{aligned} \frac{\partial \Delta EU_1}{\partial y_1} &= -v_{11}(y_1) \begin{bmatrix} c_{\alpha 1} + c_{\alpha 2} \times (p_1 \times se + (1 - p_1) \times (1 - sp)) \\ + c_e \times (p_1 \times se) - c_l \times p_1 \times se \end{bmatrix} \\ \frac{\partial \Delta EU_1}{\partial y_1} &\geqslant 0 \Leftrightarrow \frac{c_{\alpha 1} + c_{\alpha 2} \times (p_1 \times se + (1 - p_1) \times (1 - sp))}{+ c_e \times (p_1 \times se) - c_l \times p_1 \times se \geqslant 0} \\ \Leftrightarrow \frac{c_{\alpha 1} + c_{\alpha 2} \times (p_1 \times se + (1 - p_1) \times (1 - sp))}{\geqslant p_1 \times se \times (c_l - c_e)} \end{aligned}$$

For I(nf) = 1, I(prim) = 0, I(SR) = 0:

$$\begin{aligned} \frac{\partial \Delta EU_1}{\partial y_1} &= -v_{11}(y_1) \times [c_{\alpha 1} + c_e \times (p_1 \times se + (1 - p_1) \times (1 - sp)) - c_l \times p_1 \times se] \\ \frac{\partial \Delta EU_1}{\partial y_1} &\geqslant 0 \Leftrightarrow c_{\alpha 1} + c_e \times (p_1 \times se + (1 - p_1) \times (1 - sp)) - c_l \times p_1 \times se \geqslant 0 \\ \Leftrightarrow c_{\alpha 1} + c_e \times (1 - p_1) \times (1 - sp) \geqslant p_1 \times se \times (c_l - c_e) \end{aligned}$$

Appendix 4: Extra empirical results

Variables	<b>breast cancer screening</b> Marginal effects	dental prevention Marginal effects	flu vaccination Marginal effects	
GP quality index	0.060***	0.019	0.134***	
	0.000	0.019	0.154	
Self-assessed health (Ref. = $good$ , ver		0.099***	0.010**	
less than good	-0.008	$-0.033^{***}$	0.019**	
Other health and behavioural indicato		0.000**	0.040	
ADL index	-0.086*	$-0.082^{**}$	0.042	
smoked in the past	0.032**	0.005	0.028***	
currently smokes	$-0.056^{***}$	$-0.061^{***}$	$-0.031^{***}$	
Importance of the future	a a mediate			
prob. death in 10 years	-0.077***	-0.015	0.005	
expresses hope for future	$0.037^{***}$	$0.041^{***}$	0.009	
$Education (Ref. = no \ degree)$				
primary	-0.000	0.022	-0.016	
lower secondary	0.043	$0.070^{***}$	-0.023	
upper secondary	0.082***	$0.129^{***}$	-0.009	
post secondary, non tertiary	$0.106^{***}$	$0.132^{***}$	-0.015	
tertiary	$0.077^{***}$	0.186***	0.007	
$Income \ (Ref. = decile \ 1)$				
decile 2	-0.024	0.003	-0.031*	
decile 3	-0.023	-0.031*	-0.002	
decile 4	-0.006	0.003	0.014	
decile 5	-0.008	-0.007	0.003	
decile 6	0.034	-0.004	0.006	
decile 7	0.011	0.011	0.026	
decile 8	0.045**	0.029*	0.034**	
decile 9	0.029	0.016	0.035**	
decile 10	0.052**	0.021	0.035	
$age \ (Ref. = 50 \ to \ 54)$	0.052	0.021	0.020	
under 40 $(Ref. = 50, 10, 54)$	$-0.224^{***}$	$0.114^{*}$	0.067	
	-0.224 · · · · · · · · · · · · · · · · · · ·	$-0.078^{**}$	$-0.067 \\ -0.058$	
40 to 44 45 to 49	0.008	-0.018	-0.038 -0.032	
55 to 59	0.032*	-0.003	0.021*	
60 to 64	0.022	-0.015	$0.074^{***}$	
65 to 69	$-0.056^{*}$	$-0.029^{**}$	0.103***	
70 to 74	$-0.132^{***}$	$-0.055^{***}$	0.159***	
75 to 79	$-0.242^{***}$	$-0.091^{***}$	0.191***	
80 to 84	$-0.326^{***}$	$-0.156^{***}$	$0.203^{***}$	
Other indicators				
partner	$0.052^{***}$	$0.017^{**}$	$0.024^{***}$	
house owner	-0.007	$0.031^{***}$	-0.006	
foreigner, EU nationality	-0.067	-0.035	-0.015	
foreigner, non-EU nationality	$-0.208^{***}$	-0.076*	$-0.093^{**}$	
female	_	$0.056^{***}$	$0.017^{**}$	
breast cancer specific				
diagnosed cancer (except breasts)	0.085***	_	_	
in country target group	0.062**	_	_	
prob. receiving invitation letter	0.130***	_	_	
pop. based program complete	0.189***	_	_	
age and country specific incidence	0.109	_	_	
age and country specific mortality	0.040	_	_	
· · ·	0.040	_	_	
dental specific		$-0.116^{***}$		
dentures	—		_	
trouble biting	-	$-0.026^{***}$	_	
flu specific			0 00044-4-	
risk group based on age	—	-	0.083***	
risk group based on illness	—	_	0.059***	
free vaccination	—	—	0.161***	
subsidized vaccination	-	-	0.089***	
wave and country dummies added				
Pseudo $R^2$	0.190	0.205	0.168	
No. of observations	9,008	17,086	16,986	

Table 8:	Determinants	(extended	with C	GΡ	quality inc	.dex) ii	n the	take-up	of prevention

Variables	<b>breast cancer screening</b> Marginal effects	dental prevention Marginal effects	flu vaccination Marginal effects
Measures of cognitive ability		<u> </u>	
recall	-0.001	0.006***	-0.001
verbal fluency	0.001	0.001***	0.002***
mathematics index	0.025	$0.054^{***}$	0.020
Self-assessed health (Ref. = $good$ , very		0.001	01020
less than good	-0.002	$-0.019^{***}$	0.030***
Other health and behavioural indicator		0.015	0.000
ADL index	-0.081**	$-0.054^{**}$	$0.050^{*}$
	0.037***	-0.034 0.004	0.024***
smoked in the past	$-0.061^{***}$		
currently smokes	-0.061	$-0.066^{***}$	$-0.035^{***}$
Importance of the future	0.001.444	0.010	0.01=
prob. death in 10 years	$-0.061^{***}$	-0.010	0.017
expresses hope for future	0.028**	$0.031^{***}$	0.002
$Education (Ref. = no \ degree)$			
primary	0.024	0.018	-0.019
lower secondary	$0.058^{**}$	$0.055^{***}$	-0.025
upper secondary	0.092***	$0.101^{***}$	-0.016
post secondary, non tertiary	0.092***	$0.102^{***}$	-0.009
tertiary	0.090***	$0.137^{***}$	-0.005
$Income \ (Ref. = decile \ 1)$			
decile 2	-0.021	-0.005	$-0.029^{**}$
decile 3	-0.024	-0.017	-0.002
decile 4	-0.004	-0.003	0.002
decile 5	-0.005	-0.001	-0.003
decile 6	0.035*	0.003	-0.002
decile 7	0.026	0.035***	0.021
decile 8	0.036*	0.035	0.021
decile 9	0.029	0.048***	0.027*
decile 10	$0.044^{**}$	$0.048^{***}$	0.021
$age (Ref. = 50 \ to \ 54)$	0.00.1***	0.010	0.010
under 40	$-0.224^{***}$	0.018	-0.040
40 to 44	$-0.086^{**}$	$-0.048^{**}$	-0.036
45 to 49	0.006	$-0.031^{**}$	-0.044 **
55 to 59	0.019	-0.003	$0.028^{***}$
50 to 64	0.005	-0.002	$0.078^{***}$
65 to 69	$-0.068^{**}$	-0.007	0.110***
70 to 74	$-0.126^{***}$	$-0.036^{***}$	$0.169^{***}$
75 to 79	$-0.279^{***}$	$-0.062^{***}$	0.211***
80 to 84	$-0.375^{***}$	$-0.100^{***}$	0.241***
85 or over	$-0.395^{***}$	$-0.113^{***}$	0.244***
Other indicators	0.000	0.110	0.244
	0.051***	0.019***	0.026***
partner	-0.004	0.025***	-0.009
house owner			
foreigner, EU nationality	-0.058	-0.017	-0.012
foreigner, non-EU nationality	$-0.167^{***}$	$-0.085^{***}$	$-0.063^{*}$
female	—	$0.049^{***}$	$0.023^{***}$
breast cancer specific			
diagnosed cancer (except breasts)	0.083***	-	_
in country target group	0.063**	_	-
prob. receiving invitation letter	0.172***	—	—
pop. based program complete	0.191***	_	_
age and country specific incidence	-0.003	_	_
age and country specific mortality	0.105*	_	_
dental specific			
lentures	_	$-0.119^{***}$	_
rouble biting		$-0.046^{***}$	_
0	—	-0.040	—
flu specific			0.000444
risk group based on age	—	—	0.080***
risk group based on illness	—	_	0.065***
free vaccination	—	_	0.181***
subsidized vaccination	—	-	$0.089^{***}$
wave and country dummies added			
Pseudo $R^2$	0.215	0.207	0.179
No. of observations	11,506	48,912	$21,\!661$

Table 9: Determinants	(extended	with	$\operatorname{cognitive}$	abilities)	) in	the take-up	of prevention

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