

# Why prevent when it does not pay? Prevention when health services are credence goods

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## Summary

This article identifies information asymmetries and the corresponding problem of overtreatment as a possible source of prevention and health disparities when patients differ with respect to their health risk. It analyzes preventive health behavior (primary prevention) and preventive health-care utilization (secondary prevention) in markets in which patients cannot determine whether they receive excessive secondary preventive treatment—that is, where health services are credence goods. The problem of overtreatment in such markets is considered as a possible pathway through which differences in health risk lead to disparities in primary and secondary prevention as well as the corresponding health outcomes. Patients with high health risks do not invest in primary prevention, because they anticipate to be provided with unnecessary secondary prevention. Patients with lower risks invest in primary but not in secondary prevention, resulting in health losses. Furthermore, when societal groups differ with respect to their exposure to overtreatment, and we consider socioeconomic status as a possible reason, we observe disparities in primary and secondary prevention as well as the resulting health outcomes, including the “social gradient.” Several implications for empirical research are discussed.

## KEYWORDS

credence goods, health disparities, health risk, prevention, socioeconomic status

## 1 | INTRODUCTION

Disparities in primary prevention behavior, the utilization of secondary preventive care, and the resulting health outcomes have been much reported and discussed (see, e.g., Marmot, 2015).<sup>1</sup> Although the reduction or elimination of such disparities both between and within countries has been a prime policy goal for years (CSDH, 2008), the disparities persist (Mackenbach, 2012). Many factors have been associated with these disparities; however, for the design of effective policies, the underlying causes and mechanisms linking these factors to disparities still need to be better understood (Adler & Rehkopf, 2008; Lleras-Muney, 2018). For instance, socioeconomic status (SES), although clearly associated with disparities in health and health behaviors (Cutler, Lleras-Muney, & Vogl, 2008; Marmot, 2015; Pampel, Krueger, &

<sup>1</sup>“Primary prevention consists of actions that reduce the occurrence or incidence of disease.[...] Secondary prevention consists of actions that reduce or eliminate the health consequences of a disease given its occurrence.” (Kenkel, 2000). Often, primary prevention comprises actions performed by patients, and secondary prevention comprises clinical services performed by physicians.

Denney, 2010), does not affect health directly. Given the vast amount of empirical research on disparities, theoretical frameworks that systematically analyze possible causal pathways from the identified factors to disparities appear to be lacking.

This article considers information asymmetries and the corresponding problem of overtreatment in health care as a possible source of disparities in prevention and health and as a possible pathway from low SES to these disparities.

The model presented in this article is motivated by the observation that health disparities involve both heritable and social factors. It considers patients to be heterogeneous with respect to their health risk and informed about this risk, possibly through genetic testing.<sup>2</sup> A motivation for this assumption is that genetic testing has greatly advanced in recent years and that “personalized medicine is bringing the genome into the clinic, and direct-to-consumer genetic testing is bringing it into the community” (Belsky, Moffitt, & Caspi, 2013).<sup>3</sup> Despite growing knowledge about the genetic components in health risks, they seem to be significantly under-researched in the social health sciences (Belsky & Israel, 2014).

We approach our analysis from the viewpoint of health care comprising credence goods. Because patients cannot assess whether they have received appropriate treatment even *ex post*, health care has credence qualities and is a “credence good” (Darby & Karni, 1973).<sup>4</sup> Credence qualities allow certain types of misbehavior by physicians to remain undetected. A common problem arising from this situation is *overtreatment* (sometimes referred to as overprovision), which occurs when physicians provide more treatment than necessary. Recently, field experiments have provided illustrative evidence for the existence of overtreatment in different health-care markets (Currie, Lin, & Zhang, 2011; Das, Holla, Mohpal, & Muralidharan, 2016; Gottschalk, Mimra, & Waibel, 2018).<sup>5</sup>

We consider the problem of overtreatment as a possible pathway through which differences in the interplay of heritable health risks and the different degrees to which societal groups are exposed to information asymmetries lead to disparities in prevention and health. After reviewing benchmark cases without asymmetric information (Section 3), we analyze the case of asymmetric information, when secondary prevention is a credence good (Section 4). We demonstrate how differences in health risks lead to the heterogeneous use of primary and secondary prevention and to disparities in health outcomes. Additionally, we compare two societal groups that differ with respect to their exposure to asymmetric information and the associated problem of overtreatment while considering SES to be a possible reason (Section 5). We selected SES as a factor for two reasons. First, SES is associated with information problems in the physician–patient relationship, which links it to the credence goods problem (Waitzkin, 1985; Willems, De Maesschalck, Deveugele, Derese, & De Maeseneer, 2005). Second, the link between low SES and bad health—often referred to as the *social gradient*—is a highly debated issue with significant relevance to policy (see, e.g. Marmot, 2015). Yet many questions remain open, both about the causal links between low SES and bad health and the heterogeneous effects of policy interventions across groups and settings (Adler & Rehkopf, 2008; Cutler et al., 2008; Lleras-Muney, 2018).

In our model, individuals in a population (“patients”) either require secondary preventive treatment or not but do not know whether they do or not. They differ with respect to the probability that they require this treatment (their “health risk”) and can engage in costly primary preventive behavior to reduce this risk (their ability to “prevent”). Regardless of their primary prevention decisions, patients can visit a physician for a checkup at which the physician can provide secondary preventive treatment (“treatment”). The physician treats all patients who require treatment. If a patient who requires treatment does not visit the physician for a checkup, the patient eventually suffers a severe health loss. In the credence goods market, physicians have an incentive to additionally provide treatment to patients who do not require it (“overtreatment”), because patients cannot verify whether treatment was necessary, even *ex post*. Many examples from real health markets relate to this setting, including dental, chronic, and mental diseases (see Section 2.2).

When there is no asymmetric information, or no credence goods problem and no overtreatment, all patients visit a physician for a checkup and receive treatment only when they need it. In this setting, patients above a certain health risk threshold find it worthwhile to prevent—in particular, high-risk patients prevent. We do not observe health disparities in this setting. However, in a setting with asymmetric information, where health care is a credence good and there is

<sup>2</sup>Thus, our article touches upon the evolving meta-field of *genoconomics* (Benjamin et al., 2012), which studies how genetic factors influence economic outcomes. See also Belsky and Israel (2014) for a more general view on the integration of genetic research into the social sciences. Although an interesting avenue for further research, patients’ responses to the availability of genetic risk data are not considered by us in this article. The issue of genomic medicine, referring to improvements in clinical treatment through the use of genetic information, is also not considered.

<sup>3</sup>The costs associated with DNA sequencing have decreased rapidly in recent years; see information provided by the National Human Genome Research Institute at <https://www.genome.gov/sequencingcostsdata/>.

<sup>4</sup>See Gottschalk (2018) for a recent discussion on the definition of credence goods.

<sup>5</sup>To illustrate the magnitude of the problem, in a recent experiment with 180 dentist visits, Gottschalk et al. (2018) found that the total cost of overtreatment recommendations exceeded the total cost of checkup visits. More generally, it has been estimated for the United States that up to 10% of health-care expenditures, a sum of more than \$200 billion per year, are due to overcharging and overtreatment (FBI, 2011).

overtreatment, matters are different (Section 4). Because patients anticipate being overtreated, visiting a physician for a checkup is worthwhile only for those with sufficiently high health risks. Patients who visit a physician do not prevent, however, as the anticipation of being unnecessarily treated undermines the benefits of prevention. In fact, patients whose health risks are sufficiently low do not visit a physician for a checkup and consequently risk suffering health losses, resulting in health disparities within the population depending on health risk. We further consider the role of SES such that the setting without overtreatment applies to high-SES patients and the setting with overtreatment to low-SES patients (Section 5).<sup>6</sup> A comparison reveals disparities with respect to prevention and health. Most importantly, patients with low SES suffer more severe health problems than high-SES patients, because they miss out on secondary prevention due to anticipated overtreatment.

The contribution of this article is to link overtreatment in health care—modeled as a consequence of the information asymmetries in credence goods markets—and heterogeneous (genetic) health risks with disparities in primary prevention, secondary preventive health care, and the resulting health outcomes. To the best of our knowledge, we are the first to conduct such an analysis. The model provides novel points of view that may help to better understand observed disparities and inform decision makers in public health. Furthermore, we hope that empirical researchers will be motivated to examine some of the model's empirical implications.

Our model is connected to the broad literature on physician-induced demand (PID). The ability of physicians to influence demand due to informational advantages over patients has been recognized for decades (for a review, see; McGuire, 2000). Our credence goods model is based on the theoretical framework of Dulleck and Kerschbamer (2006). The importance of the credence goods setting for the PID hypothesis has been recognized by Calcott (1999) and De Jaegher and Jegers (2001), who show that the PID hypothesis is rooted in the cheap-talk literature from game theory and in the literature on credence goods. The issue of physician reimbursement in the context of credence goods has been analyzed by De Jaegher (2010) and Emons (2013). Our model also shares some aspects with that of Balsa and McGuire (2001), in which physicians treat members of different races differently because of statistical discrimination. Anticipating potentially biased treatment decisions, patients then adjust their visiting behavior accordingly. Little economic research has been conducted on the interaction of genetic health risks with primary and secondary prevention. Bardey and De Donder (2013) and Filipova-Neumann and Hoy (2014) have studied genetic health risks and primary prevention in the contexts of insurance. Wehby (2016) provides an overview of the few empirical studies that investigate the influence of genetics on primary prevention and health-care utilization.

## 2 | MODEL AND EXAMPLES

### 2.1 | Model

We consider a health-care expert market with physicians and risk-neutral, utility-maximizing patients (Dranove, 1988)<sup>7</sup>. Patients are either in a good or a bad health state. The bad health state can be interpreted as the early stage of a disease. Ex ante, patients differ with respect to the probability of being in the bad state. The probability is given by a patient's *type*  $h$ ,  $h \in [0, 1]$ . Correspondingly, the probability of being in the good state is  $(1 - h)$ . We assume a continuum of patients with mass 1, distributed on the interval  $[0, 1]$  according to the distribution function  $F(h)$  with density  $f(h) > 0$ . We suppose that this heterogeneity in risks reflects health differences that exist due to heritable (genetic) or other exogenous factors.<sup>8</sup> Therefore,  $F(h)$  can be interpreted as a genetic risk score (Belsky & Israel, 2014). We assume that patients know their respective health risks.

When health care is a credence good, there is asymmetric information, as patients cannot observe their health state but physicians can, through a costless diagnosis. In case a patient decides to visit a physician, the physician diagnoses the patient's state without diagnostic error. We abstract from costs associated with visiting and diagnosis. Physicians either carry out a preventive treatment at cost  $\kappa > 0$  or do not provide treatment. The price charged by physicians is denoted by  $t$ . We focus on the case  $t \in (\kappa, \alpha L + c)$ . The lower bound ensures that overtreatment is profitable; the upper bound guarantees positive demand in the credence goods market (see Appendix SA.2 for a detailed discussion of our assumptions).

<sup>6</sup>This analysis may generally be applied to factors other than SES that influence the extent to which patients are affected by overtreatment.

<sup>7</sup>We use the term *patient* to describe all individuals in a population, including those who do not visit a physician and who are often not included in data sets. This fact has to be considered when relating our theoretical predictions to empirical observations.

<sup>8</sup>For certain diseases, age may provide a proxy for such risks.

**TABLE 1** Patient utility depending on health state and treatment

		Not treated (not charged)	Treated (charged)
Patient state	Good	0 (appropriate)	$0 - t$ (overtreated)
	Bad	$-L$ (undertreated)	$0 - t$ (appropriate)

Each patient has the choice to engage in a costly prevention activity, in order to reduce the probability  $h$ . The prevention decision is modeled as a binary choice between prevention and no prevention. Prevention requires an effort with costs  $c > 0$ ; no prevention does not require any effort and is costless. With prevention, the patient reduces the ex ante probability of being in the bad health state by the factor  $(1 - \alpha)$  from  $h$  to  $\alpha h$ , where  $\alpha \in (0, 1)$ . With such modeling, the absolute reduction of the probability through prevention increases in the type  $h$  (for a discussion on the justification for this assumption, see ; Bardey & De Donder, 2013). A real-life analogy is that adopting solid dental hygiene practices with the aim of reducing caries has a greater impact (with respect to the absolute reduction in probability) on patients with high ex ante risk of caries than on those with low ex ante risk.

We assume that a patient in the bad state requires treatment to prevent a health loss  $L > 0$ , which occurs if the disease remains untreated. In dentistry, for example, this reflects the fact that when early oral diseases such as caries remain untreated, they lead to subsequent health problems that are painful and may require expensive treatment. The loss may more generally be interpreted as the cost of hospitalization, which could have been avoided by secondary prevention. A patient in the good state does not require treatment, corresponding to a dental patient without caries who does not need treatment. When patients receive the treatment they need, we say they have been treated appropriately. The appropriate treatment is to treat patients when they need treatment and to not treat them otherwise. Inappropriate treatment can occur in the form of overtreatment. When patients receive treatment, their utility (excluding treatment costs) is always zero, regardless of whether they needed the treatment or not. Overtreatment occurs when a patient in a good state receives treatment, is charged accordingly, and ends up with a utility of  $-t$ .

Table 1 illustrates the credence goods qualities of the medical service in our model. Ex post, after patients have been treated and charged, they cannot infer from their utility whether they were in a good or in a bad state. The utility is  $-t$  in both cases. The timing of the game is as follows:

1. Patients observe the price  $t$  and choose whether to invest in primary prevention or not;
2. Nature determines each patient's state (good or bad);
3. Patients decide whether to visit a physician for a checkup or not;
4. Physicians secretly learn the state of the visiting patients (diagnosis), provide treatment (secondary prevention) or not, and accordingly charge the price.

At the end of the last stage, utilities are realized. The solution concept for the multistage game is that of a subgame-perfect equilibrium. We introduce three tiebreaking rules to make exposition easier and ensure that we obtain unique equilibria in pure strategies. First, when patients are indifferent between visiting and not visiting a physician, they choose to visit. Second, when patients are indifferent between preventing and not preventing, they decide to prevent. Third, we assume that physicians choose the appropriate treatment in the case of indifference and that this is common knowledge. In addition, we do not permit patients to reject a treatment recommendation—that is, patients are committed to the physician's decision. This assumption is without loss of generality. The diagnosis is unobserved by the patients and therefore does not reveal any information to them. In equilibrium, there is no reason for a patient to visit a physician without the intention of being treated.

## 2.2 | Examples

The relevance of the presented setting can be illustrated through examples.<sup>9</sup> Generally, our model may apply to diseases of which the occurrence has a heritable component but can also be influenced by individual action such as healthy lifestyle

<sup>9</sup>We thank an anonymous referee and Albert Ma for their valuable input on this section.

choices (primary prevention) and for which possibilities of early clinical treatment exist (secondary prevention). Moreover, the following examples should not be viewed as perfect analogies to the presented model but as illustrations of the model's basic features.

Primary prevention in dental care consists mainly of toothbrushing and low sugar consumption. By engaging in these activities, patients can reduce their health risks with respect to the occurrence of common dental diseases such as caries. In addition, the occurrence of caries has a genetic component (Hassell & Harris, 1995). If untreated, this disease results in future health losses or expensive follow-up treatments. Caries can be detected in its early stages during a dental checkup visit. The dentist may diagnose a bad health state (caries in a tooth) and consequently treat the patient—for instance, by placing a filling (secondary prevention)—in order to prevent a tooth eventually needing extraction. Alternatively, the dentist may diagnose a good health state and find that no treatment is necessary. Overtreatment occurs when the dentist places a filling even in this case, despite diagnosing a good health state.

Further examples are seen in the occurrence of chronic diseases such as cardiovascular disease, diabetes, and colorectal cancer. Many of these diseases can primarily be prevented by physical activity, restrictive smoking, and low alcohol consumption.<sup>10</sup> An example for our model may be provided by type 2 diabetes.<sup>11</sup> The heterogeneity with respect to health risks relates to the heritable component of diabetes (Herder & Roden, 2011). Evidence from twin studies indicates that over 50% of observed variance in the occurrence of type 2 diabetes can be attributed to genetic factors (Wehby, Domingue, & Boardman, 2015). Patients may either have diabetes (bad state) or not (good state) without knowing and may consult a physician for a checkup. During the checkup, the physician tests for diabetes and either diagnoses a good state, in which no treatment is necessary, or a bad state. In the bad state, when type 2 diabetes is detected early, it can be treated with a combination of oral anti-diabetics and exercise therapy, resulting in the same health condition for the patient as that in the good state—a possibly simplifying assumption. The health loss  $L$  then reflects the bad health consequences that occur when a patient with diabetes does not receive treatment at all or receives it too late, such that treatment with insulin injections is warranted sooner than necessary, causing disutility. Overtreatment in this example constitutes the prescription of, for instance, oral anti-diabetics and exercise therapy, although the patient does not have diabetes (or is in the good state).

A final example may be found in common psychological diseases such as stress, burnout, and depression and the corresponding mental health services. Primary prevention here consists of activities in individual stress prevention, such as reading books or accessing information provided by public institutions (Maslach, Schaufeli, & Leiter, 2001). Psychological diseases often follow sudden changes in life due to external factors, such as job loss. Sometimes, psychological treatment (secondary prevention) is warranted in order to prevent greater problems in the long run—for example, depression. With respect to the heterogeneity in the model, people face different genetic risks of being affected by a psychological disease. For instance, evidence from twin studies indicates that 30–40% of observed variance in major depression can be attributed to genetic factors (Wehby et al., 2015). A person affected by a sudden change in life may consult a psychologist to obtain a recommendation on whether psychological therapy is needed (bad state) or not (good state). A trained psychologist can diagnose the health state of a patient, whereas the patient has to rely on the information provided by the psychologist. In this example, overtreatment occurs when a patient who does not need psychological therapy receives therapy.

### 3 | BENCHMARKS

#### 3.1 | Benchmark I: No market for secondary preventive treatment ( $nM$ )

As a first benchmark, we assume that no secondary prevention treatment exists and denote this regime ( $nM$ ). The analysis reveals that prevention decisions involve a fundamental trade-off between benefits and costs. The availability of risk information alone does not mean that all individuals invest in primary prevention. Patients prevent if their type is above

<sup>10</sup>The effects of these prevention activities are less specific than the effects of toothbrushing in the case of dentistry, and our model assumes away possible externalities in this respect. Furthermore, basic health behaviors such as physical activity may sometimes be characterized as secondary rather than primary prevention, because they help to limit bad health consequences once a disease has already occurred.

<sup>11</sup>We thank an anonymous pharmacist for her input on this example.



a certain threshold, because only then the expected benefits of prevention outweigh its costs. Patients prevent if their expected utility with prevention,  $EU_p^{nM}$ , exceeds the expected utility without prevention,  $EU_{np}^{nM}$ —thus, if<sup>12</sup>

$$\begin{aligned}
 EU_p^{nM} &\geq EU_{np}^{nM} \\
 &\Leftrightarrow \\
 -\alpha hL - c &\geq -hL \\
 &\Leftrightarrow \\
 (1 - \alpha)hL &\geq c \\
 &\Leftrightarrow \\
 h &\geq \frac{c}{(1 - \alpha)L} := \hat{h}.
 \end{aligned} \tag{1}$$

$$h \geq \frac{c}{(1 - \alpha)L} := \hat{h}. \tag{2}$$

Inequality (1) contrasts the costs of prevention on the right-hand side with the benefits of prevention on the left-hand side. The right-hand side of (2) represents a patient of the marginal type who is indifferent between prevention and no prevention, denoted by the cutoff  $\hat{h}$ . The cutoff increases with the costs of prevention,  $c$ : The more expensive prevention is, the fewer patients decide to prevent. Conversely, the cutoff decreases with the efficiency of the prevention effort, measured by  $(1 - \alpha)$ : The more efficient prevention is, the larger are its benefits given its costs, and the more people decide to prevent. The cutoff also decreases with the costs  $L$ , as greater suffering from the untreated health problem implies larger benefits of prevention.

Because  $c > 0$ , there is always a positive share of patients who do not find prevention worthwhile. No patient in the population prevents when  $\hat{h} \geq 1 \Leftrightarrow \frac{c}{(1-\alpha)L} \geq 1$ . In this article, we focus on the more interesting case in which at least some patients prevent by assuming  $\frac{c}{(1-\alpha)L} < 1$  (*Assumption (A1)*; the assumptions made in this article are discussed in detail in Appendix SA.2). The benefits of prevention increase with type, whereas the costs remain equal for all patients. Hence, only patients with a sufficiently high type decide to prevent.

**Result 1.** *In the absence of a treatment technology, the population is divided into two groups. Types below  $\hat{h} := \frac{c}{(1-\alpha)L}$  do not prevent; types above  $\hat{h}$  prevent.*

We observe health differences in the population with respect to the occurrence of the loss  $L$  that increase with health risk.<sup>13</sup>

### 3.2 | Benchmark II: No asymmetric information ( $nA$ ), a case without overtreatment

Now, we consider a setting with secondary preventive treatment and without asymmetric information, denoted by  $nA$ . We assume that at the diagnosis stage, both the physician and the patients learn of the patient's state.

Hence, all patients decide to visit a physician for a checkup, but only patients in the bad state demand and receive treatment. This implies that there are no health disparities in this benchmark. Patients choose between the two strategies to visit and prevent,  $(v, p)$ , and to visit and not prevent,  $(v, np)$ . The expected utility of a patient with type  $h$  who does not prevent is given by

$$EU_{v,np}^{nA} = -ht. \tag{3}$$

The expected utility of a patient with type  $h$  who prevents is given by

$$EU_{v,p}^{nA} = -\alpha ht - c. \tag{4}$$

<sup>12</sup>We use the tiebreaking rule from Section 2.

<sup>13</sup>The literature distinguishes between health inequalities (or differences), which refer to “any measurable aspect of health that varies across individuals or according to socially relevant groupings,” and health disparities (or inequities), which are “systematic differences in health that could be avoided by reasonable means” (Arcaya, Arcaya, & Subramanian, 2015).

Patients benefit from prevention by saving on the treatment costs  $t$  with the probability  $(1 - \alpha)h$ , whereas the costs of prevention are  $c$ . A patient decides to prevent if

$$\begin{aligned}
 EU_{v,np}^{nA} &\leq EU_{v,p} \\
 &\Leftrightarrow \\
 -ht &\leq -\alpha ht - c \\
 &\Leftrightarrow \\
 h &\geq \frac{c}{(1-\alpha)t}.
 \end{aligned} \tag{5}$$

The marginal type is denoted by  $\tilde{h}$ :

$$\tilde{h} := \frac{c}{(1-\alpha)t}. \tag{6}$$

The population splits into two groups. All patients visit a physician for a checkup to get informed of their state, yet only high-risk types prevent, and low-risk types decide not to prevent. The mass of patients who demand treatment (bad-state patients) depends on the price, because the price influences prevention decisions. Patient behavior is illustrated in Figure 1. For types with  $h > \tilde{h}$ , the expected benefit of prevention,  $(1 - \alpha)L$ , is larger than the cost of prevention,  $c$ . For types with  $h < \tilde{h}$ , the opposite is true. In the case  $\tilde{h} \geq 1$ , which occurs if  $\frac{c}{(1-\alpha)t} \geq 1$ , no patient finds it worthwhile to prevent. The more interesting case is  $\tilde{h} < 1$ , which occurs when  $\frac{c}{(1-\alpha)t} < 1$ . In this case, a positive share of patients invests in primary prevention. In this article, we focus on this case by assuming  $\frac{c}{(1-\alpha)\kappa} < 1$  (Assumption (A2); see Appendix SA.2 for a discussion).

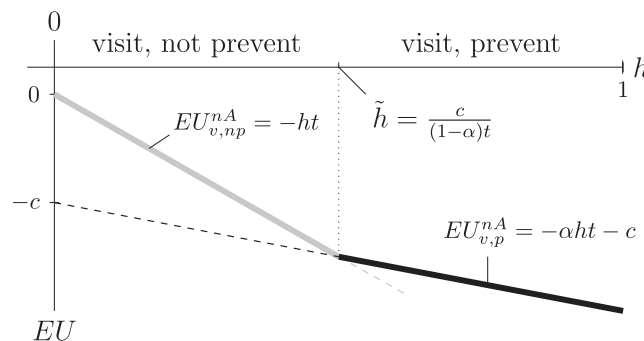
Social welfare is denoted by  $\omega_{nA}$  and given by

$$\omega_{nA} = \int_0^{\tilde{h}} f(h)(-ht)dh + \int_{\tilde{h}}^1 f(h)(-\alpha ht - c)dh + \pi_{nA}. \tag{7}$$

The first term in (7) represents the utility of patients who do not prevent, the second term represents the utility of patients who prevent, and the final term represents the profit of the physicians. The share of patients who prevent, denoted by  $P_{nA}$ , for  $t \in [\kappa, L]$ , is given by

$$P_{nA} = \int_{\tilde{h}}^1 f(h)dh = 1 - F(\tilde{h}). \tag{8}$$

The higher the treatment price, the more attractive is prevention, because a higher price yields higher expected savings through prevention. Therefore, the share of patients who prevent increases with price, as can be shown with  $\frac{\partial P_{nA}}{\partial t} = f(\cdot) \frac{c}{(1-\alpha)t^2} > 0$ . This substitution effect between prevention and treatment resembles the one described by Phelps (1978). As the price increases, types who change their prevention decision substitute treatment with prevention; in other words, they reduce the probability that they need (and receive) treatment by increasing their prevention effort.



**FIGURE 1** Patient utility depending on health risk and patients' strategy choices without overtreatment, in a market without asymmetric information ( $nA$ )

The share of patients who are treated—those who are diagnosed with a bad state—is denoted by  $B_{nA}$  and is given by

$$B_{nA} = \int_0^{\tilde{h}} hf(h)dh + \alpha \int_{\tilde{h}}^1 hf(h)dh. \quad (9)$$

At prices between  $\kappa$  and  $L$ , the share of treated patients is composed of the mass of patients who do not prevent (types below the cutoff  $\tilde{h}$ ) and the mass of patients who prevent (types above the cutoff). The profit of the physicians is given by multiplying physician demand and the profit per treatment,  $t - \kappa$ :

$$\pi_{nA} = (t - \kappa)B_{nA}. \quad (10)$$

**Result 2.** Consider the market without asymmetric information with a price  $t \in (\kappa, L]$ . In equilibrium, all patients visit a physician for a checkup and receive secondary preventive treatment when a bad state is diagnosed. The population is divided into two groups. Types below  $\tilde{h} = \frac{c}{(1-\alpha)t}$  prevent; types above  $\tilde{h}$  do not prevent. The share of patients who prevent is given by  $1 - F\left(\frac{c}{(1-\alpha)t}\right)$ .

### 3.2.1 | Efficiency and disparities with respect to primary prevention

The primary prevention decisions in our model are efficient only when the price  $t$  equals the marginal costs  $\kappa$ . Intuitively, at the efficient prevention level, the expected costs of the marginal patient not preventing,  $\kappa\tilde{h}$ , equal the expected costs of the marginal patient preventing,  $-(\alpha\kappa\tilde{h} + c)$ . We thus obtain

$$\begin{aligned} -\kappa\tilde{h} &= -(\alpha\kappa\tilde{h} + c) \\ &\iff \\ \tilde{h} &= \frac{c}{(1-\alpha)\kappa} = \tilde{h}(t = \kappa). \end{aligned} \quad (11)$$

With prices above marginal costs, we observe excessive prevention by the types between the cutoffs  $\tilde{h}(t)$  and  $\tilde{h}(\kappa)$ . From a societal point of view, patients should prevent only if the benefits of prevention, the expected reduction in treatment costs  $(1 - \alpha)h\kappa$ , outweigh the costs of prevention  $c$ —that is, if  $(1 - \alpha)h\kappa > c$ . We may call these inefficient prevention decisions “disparities,” because they could be avoided through marginal cost pricing.

## 4 | THE CREDENCE GOODS MARKET: THE CASE WITH OVERTREATMENT

In many real-world situations in health care, diagnostic results are the privileged knowledge of the physician. Such asymmetric information occurs, for instance, when a physician is better trained than the patient with respect to finding and interpreting symptoms or with respect to evaluating results displayed by medical equipment.<sup>14</sup> In such cases, the treatment recommendation of a physician is a credence good, and patients cannot detect overtreatment. An example is a dental filling (Gottschalk et al., 2018): even after treatment, patients usually cannot evaluate whether they needed a filling (bad state) or not (good state). In our model, physicians use this informational advantage to overtreat visiting patients who are in the good state. When patients get overtreated, they are not able to infer their state from the realized utility even after they have visited a physician (see Table 1). In line with other credence-goods health-care models (e.g., De Jaegher & Jegers, 2001), we assume that treatment is verifiable, because patients are usually able to verify the treatment they receive and the billing for the treatment.<sup>15</sup>

<sup>14</sup>It can also occur due to procedural practices as illustrated by an example from dentistry based on the personal experience of the author: In some cases, after taking an X-ray of the patient, the dentist leaves the patient in order to analyze the X-ray in private in another room and returns with a diagnosis.

<sup>15</sup>Dulleck and Kerschbamer (2006) have emphasized the importance of the two institutions of verifiability and liability in credence goods markets. Liability describes institutional features that hold a physician liable for not appropriately treating a patient in need. Liability prohibits undertreatment; however, overtreatment is still possible. In health-care markets, liability may or may not be a given, depending on how easy it is to prove misconduct before a court. In our model, as long as verifiability holds, it is irrelevant whether liability holds or not, because undertreatment is not an issue in our setting.



**TABLE 2** Physician payoffs and behavior

	Setting	No asymmetric information	Credence goods market
Patient state	Good	0	$t - \kappa$
		Appropriate treatment	Overtreatment
	Bad	$t - \kappa$	$t - \kappa$
		Appropriate treatment	Appropriate treatment

Note. An overtreatment inducing price,  $t > \kappa$ , is assumed.

**TABLE 3** Patient payoffs

	Setting	No asymmetric information	Credence goods market
Patient state	Good	0	$-t$
		Appropriate	Overtreatment
	Bad	$-t$	$-t$
		Appropriate treatment	Appropriate treatment

Note. An overtreatment inducing price,  $t > \kappa$ , is assumed.

Tables 2 and 3 summarize the payoffs in the different information settings for a physician and patients, respectively.

Patients decide whether to prevent or not and whether to visit a physician for a checkup or not. They have four possible strategies: Not visiting and not preventing ( $nv, np$ ), not visiting and preventing ( $nv, p$ ), visiting but not preventing ( $v, np$ ), and visiting and preventing ( $v, p$ ). Patients always choose ( $v, np$ ) over ( $v, p$ ), because they obtain the same payoff  $-t$  given they visit a physician, regardless of their state.<sup>16</sup> Prevention is of no benefit in this case, but costs occur, as  $c > 0$ . Thus, ( $v, p$ ) will not be considered in the following. A patient of type  $h$  obtains the following respective expected utility from the three remaining strategies.

$$EU_{nv,np}^{CG} = -hL \quad EU_{nv,p}^{CG} = -\alpha Lh - c \quad EU_{v,np}^{CG} = -t. \quad (12)$$

#### 4.1 | Patient behavior with overtreatment

The patient population in the credence goods market splits into three groups, divided by two cutoffs. Patients in the group with the lowest risks decide neither to visit nor to prevent—strategy ( $nv, np$ ). Patients with the highest risks visit but do not prevent—strategy ( $v, np$ ). The remaining patients do not visit but prevent—strategy ( $nv, p$ ). Figure 2 illustrates this result.

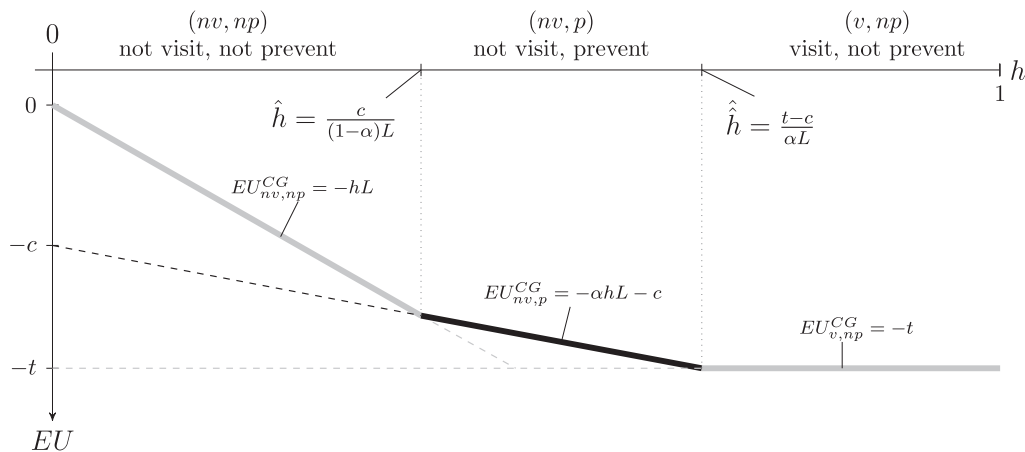
The cutoff  $\hat{h}$  divides the patient groups who choose the strategies ( $nv, np$ ) and ( $nv, p$ ), respectively; the cutoff  $\hat{\hat{h}}$  divides the patient groups who choose ( $nv, p$ ) and ( $v, np$ ), respectively. The cutoff between the two strategies without visiting ( $nv, np$ ) and ( $nv, p$ ) is the same as in the first benchmark, because it involves the same trade-off:  $\hat{h} = \frac{c}{(1-\alpha)L}$ . The cutoff between the two strategies ( $nv, p$ ) and ( $v, np$ ), derived in Appendix SA.1, is given by

$$\hat{\hat{h}} = \frac{t - c}{\alpha L}. \quad (13)$$

The cutoff decreases, which implies that some patients substitute primary with secondary prevention, when the costs of primary prevention,  $c$ , increase. The cutoff increases with the price for treatment,  $t$ , because a larger price makes prevention relatively more attractive. The more efficient prevention is, or the lower the  $\alpha$ , the more beneficial prevention becomes as an alternative to visiting a physician, and the cutoff hence increases.

Intuitively, patients with high health risks—“high types”—are more likely to visit a physician in order to avoid the likely loss  $L$ . Because of the prospect of being overtreated, there is no incentive for high types to prevent, although they would prevent in the absence of overtreatment. Types below  $\hat{\hat{h}}$  choose not to visit a physician at all, as visiting is too expensive for them, given that visiting helps them to avoid the loss  $L$  with a comparatively low probability only. Types between

<sup>16</sup>This is due to the assumption that all physicians always overtreat if it is profitable. If we assumed that only some physicians overtreated, then this may not be the case anymore—see Appendix SA.2.1 for a discussion.



**FIGURE 2** Patient utility depending on health risk and patients' strategy choices with overtreatment, in the credence goods market (CG). We assume  $t \in (\kappa, \alpha L + c)$

$\hat{h}$  and  $\hat{\hat{h}}$  decide to prevent, as for them, the benefits of prevention—the saved loss  $L$  with additional probability  $(1 - \alpha)$ —are larger than its costs. Patients with low health risks—types below  $\hat{h}$ —neither visit nor prevent, because they experience the loss  $L$  with only a small probability, such that preventing is more expensive than beneficial.

Social welfare in the credence goods market, denoted by  $\omega_{CG}$ , is given by

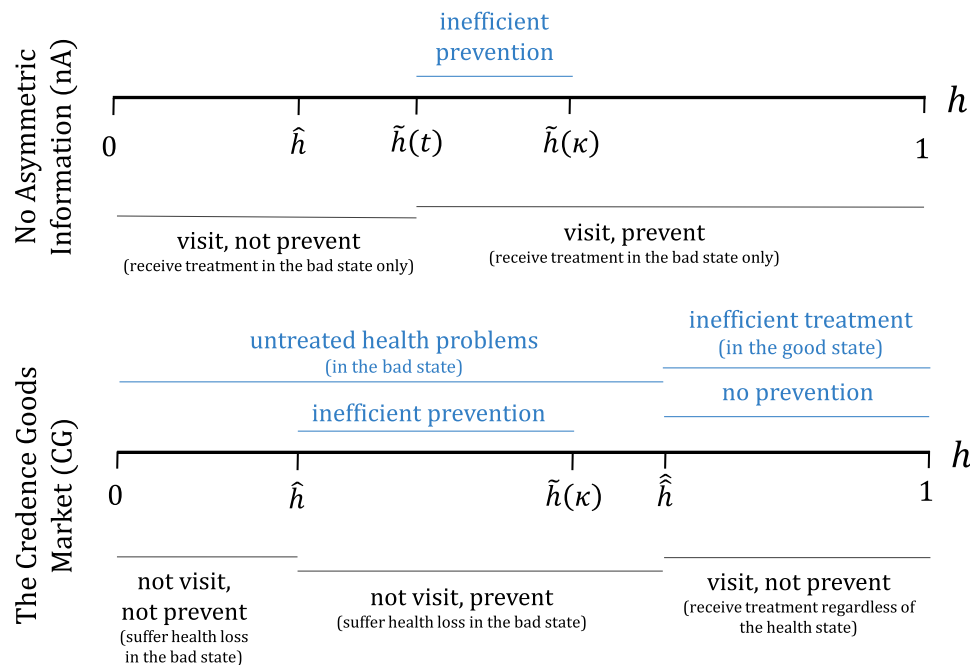
$$\omega_{CG} = \int_0^{\hat{h}} f(h)(-hL)dh + \int_{\hat{h}}^{\hat{\hat{h}}} f(h)(-\alpha hL - c)dh + \int_{\hat{\hat{h}}}^1 f(h)(-t)dh + \pi_{CG}, \quad (14)$$

where  $\hat{h} = \frac{c}{(1-\alpha)L}$  and  $\hat{\hat{h}} = \frac{t-c}{\alpha L}$ . The first term in (14) represents the utility of patients who choose strategy  $(nv, np)$ . The second term represents the utility of patients who choose strategy  $(nv, p)$ . The third term indicates the utility of patients who choose strategy  $(v, np)$ . Finally,  $\pi_{CG}$  indicates the profit of the physicians. Comparing (14) and (7) reveals that at a given price  $t$ , welfare is unambiguously lower in the credence goods market than in the market without asymmetric information.

An important difference compared with the case without asymmetric information lies in the willingness to pay for treatment. Without asymmetric information, all patients diagnosed with a bad state are willing to pay  $L$  for treatment, whereas all patients in the good state have no positive willingness to pay. In the credence goods market, counterintuitively, the willingness to pay of patients with the highest type,  $h = 1$ , who require treatment in any case, is  $\alpha L + c$  and thus below  $L$ <sup>17</sup>. The reason is that in the credence goods market, the alternative strategy to visiting and not preventing,  $(v, np)$ , is not visiting and preventing,  $(nv, p)$ . At the price  $\alpha L + c$ , the highest type with  $h = 1$  is indifferent between the two strategies. This is in contrast to the case without asymmetric information, where the prevention decision is independent of the visiting decision.

Evidence from several health settings suggests that patient populations indeed split into groups with different strategies according to our model. With respect to dental care, a study from Switzerland (FSO, 2017) reports that, within a calendar year, only around one third of the population both visited a practice for preventive dental hygiene (primary prevention) and went to a dentist for a recommended checkup (where secondary prevention may be provided). About 28% of the population neither invested in primary prevention nor had a dental checkup. Furthermore, around 10% of the population invested only in dental hygiene, and 30% had only a dental checkup. Within the framework of our model, the first group is not included, due to our assumption that all physicians always overtreat if it is profitable; see Appendix SA.2.1 for a discussion on our results when this assumption is relaxed. Another example is the case of preventive colorectal cancer screenings, where only a minority of the population decides to undergo the testing procedure at all (Hamman & Kapinos, 2016). However, to the best of our knowledge, no study yet has related these strategies to exogenous health risks, which would be an interesting test of the implications of our model.

<sup>17</sup>The fact that  $\alpha L + c < L$  follows from Assumption (A1).



**FIGURE 3** Patient strategies (gray) and inefficiencies (blue) in *nA* and *CG*. Legend:  $\hat{h} = \frac{c}{(1-\alpha)L}$ ,  $\tilde{h} = \frac{c}{(1-\alpha)t}$ ,  $\tilde{h}(\kappa) = \frac{c}{(1-\alpha)\kappa}$ , and  $\hat{\hat{h}} = \frac{t-c}{\alpha L}$ . Illustration for  $t \in (\kappa, L]$  in *nA* and  $t \in \left(\frac{\alpha L c}{(1-\alpha)\kappa} + c, \alpha L + c\right)$  in *CG* [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

## 4.2 | The inefficiencies of the credence goods market

Figure 3 illustrates patient behavior and the resulting inefficiencies for both the setting without asymmetric information (*nA*) and the credence goods market (*CG*). The only inefficiency in *nA* is excessive prevention by types between  $\tilde{h}$  and  $\tilde{h}(\kappa)$  due to prices above marginal costs (see Section 3). This is illustrated in the upper part of Figure 3. In *CG*, three additional inefficiencies occur as illustrated in the lower part of Figure 3.<sup>18</sup> Importantly, patients who do not visit a physician due to the high costs of related overtreatment (types below  $\hat{\hat{h}}$ ) suffer from untreated health problems in case they realize a bad state. Additionally, there is both inefficiently much prevention (types between  $\hat{h}$  and  $\tilde{h}(\kappa)$ ) and insufficiently little prevention (types between  $\hat{\hat{h}}$  and 1). The reason for the latter is that overtreatment destroys prevention incentives for patients who decide to visit a physician. Finally, we observe excessive medical treatment in *CG*, because of overtreatment.

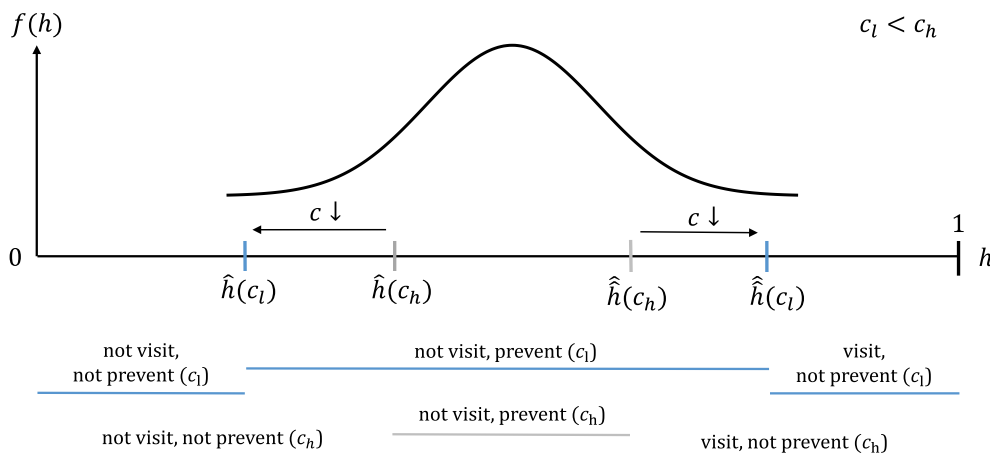
The results from this section are summarized in Proposition 1, where we denote  $\frac{\alpha L c}{(1-\alpha)\kappa} + c$  by  $t_x$ .

**Proposition 1.** *Consider the credence goods market (CG) and assume that prices are fixed at a level at which incentives for overtreatment prevail and at which demand is positive—that is,  $t \in (\kappa, \alpha L + c)$ . Several inefficiencies occur: Patient types above  $\hat{\hat{h}}$  (if  $t > t_x$ ) or  $\tilde{h}(\kappa)$  (if  $t < t_x$ ) do not prevent; types between  $\hat{h}$  and  $\tilde{h}(\kappa)$  (if  $t > t_x$ ) or  $\hat{h}$  (if  $t < t_x$ ) prevent; types below  $\hat{\hat{h}}$  remain untreated in the bad state; and types above  $\hat{\hat{h}}$  receive treatment in the good state.*

We consider it an interesting avenue for further research to compare societal groups that differ with respect to the parameters of the model within the setting *CG*. For instance, risk distributions and prevention benefits with regard to many diseases differ between genders (Baggio, Corsini, Floreani, Giannini, & Zagonel, 2013). Moreover, socioeconomic characteristics such as income may influence the costs of prevention,  $c$ , or the size of the health loss,  $L$ , in comparisons between or within societies. We refer to this discussion only briefly with the following example.

A reduction in the costs of prevention,  $c$ , has similar effects as an increase in the benefits of prevention, which corresponds to a reduction of  $\alpha$ , and vice versa, illustrated in Figure 4. As  $c$  (or  $\alpha$ ) decreases, the cutoff  $\hat{h} = \frac{c}{(1-\alpha)L}$  decreases, whereas the cutoff  $\hat{\hat{h}} = \frac{t-c}{\alpha L}$  increases; in other words, more patients among those who do not visit choose to prevent—that is, choose  $(nv, p)$  over  $(nv, np)$ , and fewer patients opt for checkup visits instead of prevention—that is, choose  $(nv, p)$  over

<sup>18</sup>For low prices,  $t \in (\kappa, \frac{\alpha L c}{(1-\alpha)\kappa} + c)$ , we have  $\hat{\hat{h}} < \tilde{h}(\kappa)$ , and the figure has to be adjusted accordingly.



**FIGURE 4** A decrease in prevention costs for a hypothetical risk distribution; analogue for a decrease in  $\alpha$ , which corresponds to an increase in prevention benefits [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]

( $v, np$ ). Suppose, merely for illustration, that women face lower costs or higher benefits of prevention than men.<sup>19</sup> This would imply a higher share of women who prevent and do not visit, compared with men. Correspondingly, we would observe a lower share of women making up the two groups below  $\hat{h}$  and above  $\hat{h}$ , respectively, implying not only higher rates of primary prevention but also higher rates of the loss  $L$ . This provides a possible solution to the empirical puzzle from dentistry that women prevent more yet more often suffer severe health problems (Berteau, Staehelin, Dratva, & Stutz, 2007).

## 5 | WHEN OVERTREATMENT AFFECTS ONLY SOME PATIENTS: THE CASE OF SOCIOECONOMIC STATUS (SES) (NA VS. CG)

What are the implications of our model when we compare the two worlds with and without asymmetric information? An interesting discussion may be initiated by phrasing this question in terms of a comparison of markets in which the extent of asymmetric information between physicians and patients is small for some and large for other patients. In other words, the upper part of Figure 3 applies to some patients, whereas the lower part of the figure applies to others. In what follows, we propose SES to be a possible channel from the setting without asymmetric information ( $nA$ ) to the credence goods setting ( $CG$ ); however, readers should keep in mind that the analysis more generally applies to situations in which some patients are more exposed to overtreatment than other patients, for whatever reason.<sup>20</sup>

We propose that  $CG$  applies to patients with low SES, whereas  $nA$  applies to patients with high SES.<sup>21</sup> We postulate that a large information asymmetry is associated with low SES for several reasons. Generally, Waitzkin (1985) finds that the patient's SES is one determinant of information transmission in the physician–patient relationship. More precisely, Willems et al. (2005) demonstrate that high-SES patients are able to elicit more information from their physician. Moreover, high-SES patients may be better able to communicate symptoms to physicians (Brekke, Holmås, Monstad, & Straume, 2018) and are potentially better able to detect overtreatment recommendations through indirect cognitive skills (Cutler & Lleras-Muney, 2010). Also, high SES may lead to higher presumed patient information on the physician side (Van Ryn & Burke, 2000) and to longer consultations (Brekke et al., 2018). By suggesting that health disparities related to SES are rooted in the credence goods problem and associated overtreatment, our analysis provides a new point of view to the discussion on the social gradient—referring to the finding that lower SES is associated with worse health outcomes (Cutler et al., 2008; Marmot, 2015).<sup>22</sup>

<sup>19</sup>For instance, a recent study by Althoff et al. (2017) suggests that with an increase in physical activity, the occurrence of obesity decreases more rapidly for women than for men.

<sup>20</sup>There is, for instance, a large literature researching the disparities in treatment decisions among racial and ethnic groups; see Balsa and McGuire (2001) and Van Ryn and Fu (2003) for a review.

<sup>21</sup>We use the term SES broadly, although we acknowledge that empirical research requires more specific definitions of the term (Braveman et al., 2005).

<sup>22</sup>See Lleras-Muney (2018) for a recent review and discussion of Marmot's main arguments.

Assume that, all else equal<sup>23</sup>, there are high- and low-SES patients and that high-SES patients are exposed to the setting  $nA$ , whereas low-SES patients are exposed to the setting  $CG$ . In other words, the upper part of Figure 3 applies to high-SES patients, whereas the lower part of the figure applies to low-SES patients. We can derive several predictions from this comparison, which are presented in the following section (a summary table is also provided in Appendix SA.3, Table S4). We relate these predictions to some empirical findings that serve as illustrations. We do not claim to have reviewed the enormous empirical literature on the topic.

Our basic prediction with respect to supply-side behavior is that low-SES patients are overtreated more often than high-SES patients, as there is no overtreatment in  $nA$  (high SES), in contrast to  $CG$  (low SES).<sup>24</sup> Hence, health-care costs related to secondary prevention are on average higher for low-SES patients, conditional on checkup visits, which is in line with findings from England that health-care expenditures are concentrated on low-SES people (Cookson, Propper, Asaria, & Raine, 2016). Our model also predicts that all patients who visit and are in need of treatment receive treatment; thus, the health gradient is visible only when the whole population is considered, including those who visit for checkups and those who do not. This may prove to be useful information for empirical work, which often uses data provided by health-care providers. For instance, Brekke et al. (2018) found no health gradient among patients who consulted general practitioners.

With respect to disparities in health outcomes, our model suggests—corresponding to the social gradient—that low-SES patients more often suffer from the loss  $L$  due to missed secondary prevention. In  $CG$  (low SES), patient types below the cutoff  $\hat{h}$ , in anticipation of being overtreated, do not make checkup visits. In  $nA$  (high SES), however, all patients visit a physician for a checkup, and hence, all patients who need treatment receive treatment. If the health loss,  $L$ , refers to hospital treatment, this finding is potentially connected to the evidence that low-SES individuals are several times more likely than high-SES patients to require preventable hospital visits (Kangovi et al., 2013). Another link to this prediction is the finding from England that people of higher SES “tend to present to health-care providers at an earlier stage of illness” (Cookson et al., 2016).

In relation to these points, our analysis suggests that low-SES patients on average make fewer checkup visits, because in  $CG$  (low SES), only patient types above the cutoff  $\hat{h}$  visit, whereas in  $nA$  (high SES), all patients visit. This aggregate prediction seems to be consistent with the finding that in most Organisation for Economic Co-operation and Development countries, patients with higher SES, as measured by income, are more likely to visit general practitioners and dentists and have cancer checkups (Doorslaer, Koolman, & Jones, 2004; Devaux, 2015). Regarding econometric studies, our framework proposes an interaction between health risks and patient information (with respect to being able to evaluate the treatment recommendation). For low risks, we expect that lower information asymmetries (high SES) lead to more checkup visits; however, for high risks, we expect that lower information asymmetries do not affect the visiting decision. Possible interactions between health risk and information are typically not considered in empirical studies that test the effect of patient information on physician visits. This may be one reason why, for instance, Kenkel (1990) finds that information is associated with a higher likelihood of visits, whereas Schmid (2015) finds no such effect. Our framework, moreover, distinguishes between checkup visits with secondary preventive treatments and other visits with non-preventive treatments that are a consequence of the health loss  $L$ , whereas this distinction is not made in many empirical studies.

In our model, low-risk patients with low SES do not visit a physician for a checkup. Consequently, visiting low-SES patients on average have larger health risks than visiting high-SES patients, which may be interpreted as a possible source of unobserved heterogeneity in studies that rely on data collected at health-care providers and thus exclude individuals who decide not to visit at all.

With respect to primary and secondary prevention patterns, our analysis suggests that the share of high-SES patients who both prevent and visit a physician for a checkup, strategy  $(v, p)$ , is larger than the corresponding share of low-SES patients (which is zero). Furthermore, the share of low-SES patients who neither prevent nor visit a physician for a checkup, strategy  $(nv, np)$ , is larger than the corresponding share of high-SES patients (which is zero). Last, the respective shares of low-SES patients who either only prevent or only visit a physician, strategies  $(nv, p)$  and  $(v, np)$ , are larger than the corresponding shares of high-SES patients.

<sup>23</sup>In particular, we assume that risk distributions for low-SES and high-SES patients are equal, although the research in this area, which is sparse, suggests possible correlations between SES and health risk (Belsky et al., 2013; Wehby, 2016).

<sup>24</sup>Recent evidence for this is provided by Brekke et al. (2018) and Gottschalk et al. (2018) for the specific settings of diabetes and dental treatments, respectively.

The total share of individuals who invest in primary prevention may be larger, equal, or lower in either group, depending on the risk distribution function  $F(\cdot)$ . Hence, our model does not necessarily reflect the evidence of a social gradient with respect to primary prevention behavior (Pampel et al., 2010).<sup>25</sup> The share of patients for whom prevention is inefficient is larger among low-SES patients ( $CG$ ) than high-SES patients ( $nA$ ). The reason is that patient types between  $\hat{h}$  and  $\tilde{h}$ , for whom prevention is inefficient, prevent in  $CG$  (low SES) but not in  $nA$  (high SES); furthermore, patient types above  $\hat{h}$ , for whom prevention would be efficient, prevent in  $nA$  (high SES) but not in  $CG$  (low SES). Hence, our model also predicts a social gradient with respect to efficient prevention decisions.

One referee pointed to a notable yet subtle prediction regarding the patient types between  $\hat{h}$  and  $\tilde{h}(t)$ . When these types are of high SES, they do not invest in primary prevention, whereas those of low SES do. This leads to the prediction that patient types between  $\hat{h}$  and  $\tilde{h}(t)$ —and these are comparatively low health risks—prevent less when they have a high SES compared with a low SES. Empirical support for this prediction seems to be scarce. However, there seems to be no contrary evidence either. One reason may be that this prediction is subtle. For an empirical test, a researcher would have to order health risks gradually to determine the two cutoffs, but the use of exact risk information, such as genetic risk scores, has so far been limited in the social health sciences (Belsky & Israel, 2014). Additionally, the binary modeling of prevention leads to a potential overstatement of the prevention differences between the two groups.<sup>26</sup>

## 6 | CONCLUSION

This article identified information asymmetries and the corresponding problem of overtreatment as a possible source of prevention and health disparities when patients differ with respect to their exogenous (genetic) health risks. It presented a theoretical model in which health services are modeled as credence goods—that is, physicians have an informational advantage over patients with respect to whether secondary preventive treatment is necessary or not and in which the problem of overtreatment persists. The model shows that primary prevention behavior and secondary preventive health-care consumption patterns in the presence of overtreatment differ from those in markets without asymmetric information, in which overtreatment is not a problem. The credence goods market incorporates several inefficiencies that do not occur in markets without asymmetric information. We used the model to discuss disparities in primary and secondary prevention as well as the related health outcomes when societal groups differ with respect to their exposure to overtreatment and considered SES as a possible reason.

The model adds novel points of view to discussions on prevention and health disparities, which are highly relevant politically. It provides several testable implications with respect to the role of exogenous (genetic) risk in health behavior and the interaction of health risks, information problems, and overtreatment as a source of health disparities. Naturally, the conclusions provided by our abstract analysis are valid only within our theoretical framework. We nevertheless hope that our analysis lets researchers and public health professionals appreciate that health disparities being caused by patient behavior may be better understood when the provision of health care is taken into account.

We hope that our analysis motivates empirical work. Regarding the analysis of individual health behavior and its interaction with health risk, health risk data is not used much yet in the social health sciences including health economics. More could be done with existing data sets, and further advances in the availability of genetic risk data, such as genetic risk scores, are promising (Belsky et al., 2013; Belsky & Israel, 2014). As regards the analysis of the extent to which different groups, such as groups of different SESs, are affected by overtreatment, the major challenge is that overtreatment is hard to measure in the first place, especially with aggregated data. Aggregated data do not usually reveal whether a single treatment was necessary or not. In this respect, field studies (Currie et al., 2011; Das et al., 2016; Gottschalk et al., 2018) and empirical work using patient-level microdata in specific settings (Brekke et al., 2018) are promising. Although such studies are limited to specific settings, they seem to be well-suited to enhance our understanding of the extent and causes of overtreatment. Besides empirical work, laboratory experiments could be used to test some of the implications of our model.

<sup>25</sup>If we relax our assumption of equal risk distributions, our model is more likely to predict a prevention gradient if low SES is associated with higher health risks.

<sup>26</sup>If we modeled prevention continuously—for instance, with marginally decreasing returns and linear costs of prevention—patient types below the respective cutoffs would invest in prevention, and the investment would gradually increase with the type; we thank an anonymous referee for suggesting this discussion.



Regarding policy implications, the problem of overtreatment lies at the core of the problems identified in our conceptual framework. Therefore, we advocate that the reduction or elimination of overtreatment be a policy target. Within our framework, this would have to be achieved by reducing or eliminating information asymmetries with respect to the knowledge about whether treatment is necessary or not. More than proposing specific policies, however, our analysis calls for caution with respect to policies that do not address this specific information asymmetry or the problem of overtreatment. One problem is that policies may just redistribute overtreatment between societal groups without reducing its total level. As an illustration, a much debated issue is the expansion of health insurance coverage (see Kreider, Manski, Moeller, & Pepper, 2015, for dental care). The recommendation to expand insurance coverage often rests upon the presumption that it can help to reduce inequalities in health-care utilization (Devaux, 2015). However, insurance has been shown to potentially increase the problem of overtreatment when health services are credence goods (Huck, Lünser, Spitzer, & Tyran, 2016). In our framework, insurance would therefore potentially imply that both low-SES and high-SES patients face the problem of overtreatment. This would eliminate utilization inequalities and health disparities between the two groups but at the cost of high-SES patients being worse off than before.

A policy often proposed is to better inform certain population groups about the benefits of primary or secondary prevention. In our model, this would have no effect, because patients are already informed. In the same spirit, we need to reject the idea that the availability of personalized health-risk information, such as that obtained through genetic testing, will unambiguously lead to better health behaviors (see also Evans, Meslin, Marteau, & Caulfield, 2011), at least once better health literacy among patients is achieved (McBride, Koehly, Sanderson, & Kaphingst, 2010). In our model, insufficient prevention by high-risk patients and health disparities due to missed secondary prevention are the result of a reaction to anticipated overtreatment, even though patients perfectly understand their health risk.

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## REFERENCES

- Adler, N. E., & Rehkopf, D. H. (2008). U.S. disparities in health: Descriptions, causes, and mechanisms. *Annual Review of Public Health*, 29, 235–252.
- Althoff, T., Hicks, J. L., King, A. C., Delp, S. L., Leskovec, J., & Sosič, R. (2017). Large-scale physical activity data reveal worldwide activity inequality. *Nature*, 547(7663), 336–339.
- Arcaya, M. C., Arcaya, A. L., & Subramanian, S. (2015). Inequalities in health: Definitions, concepts, and theories. *Global Health Action*, 8(1), 27106.
- Baggio, G., Corsini, A., Floreani, A., Giannini, S., & Zagonel, V. (2013). Gender medicine: A task for the third millennium. *Clinical chemistry and Laboratory Medicine*, 51(4), 713–727.
- Balsa, A. I., & McGuire, T. G. (2001). Statistical discrimination in health care. *Journal of Health Economics*, 20(6), 881–907.
- Bardey, D., & De Donder, P. (2013). Genetic testing with primary prevention and moral hazard. *Journal of Health Economics*, 32(5), 768–779.
- Belsky, D. W., & Israel, S. (2014). Integrating genetics and social science: Genetic risk scores. *Biodemography and Social Biology*, 60(2), 137–155.
- Belsky, D. W., Moffitt, T. E., & Caspi, A. (2013). Genetics in population health science: Strategies and opportunities. *American Journal of Public Health*, 103(S1), S73–S83.
- Benjamin, D. J., Cesarini, D., Chabris, C. F., Glaeser, E. L., Laibson, D. I., Gudnason, V., ..., & Lichtenstein, P. (2012). The promises and pitfalls of genoeconomics. *Annual Review of Economics*, 4, 627–62.

- Bertea, P. C., Staehelin, K., Dratva, J., & Stutz, E. Z. (2007). Female gender is associated with dental care and dental hygiene, but not with complete dentition in the swiss adult population. *Journal of Public Health*, 15(5), 361–367.
- Braveman, P. A., Cubbin, C., Egerter, S., Chideya, S., Marchi, K. S., Metzler, M., & Posner, S. (2005). Socioeconomic status in health research: One size does not fit all. *Jama*, 294(22), 2879–2888.
- Brekke, K. R., Holmås, T. H., Monstad, K., & Straume, O. R. (2018). Socio-economic status and physicians' treatment decisions. *Health Economics*, 27(3), e77–e89.
- Marmot, M., Friel, S., Bell, R., Houweling, T. A. J., Taylor, S., & Commission on Social Determinants of Health and others (2008). Closing the gap in a generation: health equity through action on the social determinants of health. *The Lancet*, 372(9650), 1661–1669.
- Calcott, P. (1999). Demand inducement as cheap talk. *Health Economics*, 8(8), 721–733.
- Cookson, R., Propper, C., Asaria, M., & Raine, R. (2016). Socio-economic inequalities in health care in England. *Fiscal Studies*, 37(3–4), 371–403.
- Currie, J., Lin, W., & Zhang, W. (2011). Patient knowledge and antibiotic abuse: Evidence from an audit study in China. *Journal of Health Economics*, 30(5), 933–949.
- Cutler, D. M., & Lleras-Muney, A. (2010). Understanding differences in health behaviors by education. *Journal of Health Economics*, 29(1), 1–28.
- Cutler, D. M., Lleras-Muney, A., & Vogl, T. (2008). Socioeconomic status and health: Dimensions and mechanisms. (NBER Working Paper No. 14333. Tech. Rep.): National Bureau of Economic Research, Cambridge, MA.
- Darby, M. R., & Karni, E. (1973). Free competition and the optimal amount of fraud. *Journal of Law and Economics*, 16(1), 67–88.
- Das, J., Holla, A., Mohpal, A., & Muralidharan, K. (2016). Quality and accountability in health care delivery: Audit-study evidence from primary care in India. *American Economic Review*, 106(12), 3765–99.
- De Jaegher, K. (2010). Physician incentives: Cure versus prevention. *Journal of Health Economics*, 29(1), 124–136.
- De Jaegher, K., & Jegers, M. (2001). The physician–patient relationship as a game of strategic information transmission. *Health Economics*, 10(7), 651–668.
- Devaux, M. (2015). Income-related inequalities and inequities in health care services utilisation in 18 selected OECD countries. *The European Journal of Health Economics*, 16(1), 21–33.
- Doorslaer, E. v., Koolman, X., & Jones, A. M. (2004). Explaining income-related inequalities in doctor utilisation in Europe. *Health Economics*, 13(7), 629–647.
- Dranove, D. (1988). Demand inducement and the physician–patient relationship. *Economic Inquiry*, 26(2), 281–298.
- Dulleck, U., & Kerschbamer, R. (2006). On doctors, mechanics, and computer specialists: The economics of credence goods. *Journal of Economic Literature*, 44(1), 5–42.
- Emons, W. (2013). Incentive compatible reimbursement schemes for physicians. *Journal of Institutional and Theoretical Economics JITE*, 169(4), 605–620.
- Evans, J. P., Meslin, E. M., Marteau, T. M., & Caulfield, T. (2011). Deflating the genomic bubble. *Science*, 331(6019), 861–862.
- FBI (2011). Financial crimes report to the public: Fiscal years 2010–2011. (Tech. rep.): Federal Bureau of Investigation, Washington, DC.
- FSO (2017). *Schweizerische Gesundheitsbefragung*. Switzerland: Federal Statistical Office.
- Filipova-Neumann, L., & Hoy, M. (2014). Managing genetic tests, surveillance, and preventive medicine under a public health insurance system. *Journal of Health Economics*, 34, 31–41.
- Gottschalk, F. (2018). What characterizes credence goods? A critical look at the literature. ETH Zurich, Center of Economic Research (CER-ETH). (SSRN Working Paper). <https://dx.doi.org/10.2139/ssrn.3114257>
- Gottschalk, F., Mimra, W., & Waibel, C. (2018). Health services as credence goods. A field experiment. ETH Zurich, Center of Economic Research (CER-ETH). (SSRN Working Paper). <https://dx.doi.org/10.2139/ssrn.3036573>
- Hamman, M. K., & Kapinos, K. A. (2016). Colorectal cancer screening and state health insurance mandates. *Health Economics*, 25(2), 178–191.
- Hassell, T. M., & Harris, E. L. (1995). Genetic influences in caries and periodontal diseases. *Critical Reviews in Oral Biology & Medicine*, 6(4), 319–342.
- Herder, C., & Roden, M. (2011). Genetics of type 2 diabetes: Pathophysiologic and clinical relevance. *European Journal of Clinical Investigation*, 41(6), 679–692.
- Huck, S., Lünser, G., Spitzer, F., & Tyran, J.-R. (2016). Medical insurance and free choice of physician shape patient overtreatment: A laboratory experiment. *Journal of Economic Behavior & Organization*, 131, 78–105.
- Kangovi, S., Barg, F. K., Carter, T., Long, J. A., Shannon, R., & Grande, D. (2013). Understanding why patients of low socioeconomic status prefer hospitals over ambulatory care. *Health Affairs*, 32(7), 1196–1203.
- Kenkel, D. (1990). Consumer health information and the demand for medical care. *The Review of Economics and Statistics*, 74(4), 587–595.
- Kenkel, D. S. (2000). Prevention, (1st ed.). In Culyer, A. J., & Newhouse, J. P. (Eds.), *Handbook of health economics* (chapter 31, Vol. 1, pp. 1675–1720). Elsevier, Washington, DC
- Kreider, B., Manski, R. J., Moeller, J., & Pepper, J. (2015). The effect of dental insurance on the use of dental care for older adults: A partial identification analysis. *Health Economics*, 24(7), 840–858.
- Lleras-Muney, A. (2018). Mind the gap: A review of the health gap: The challenge of an unequal world by Sir Michael Marmot. *Journal of Economic Literature*, 56(3), 1080–1101.
- Mackenbach, J. P. (2012). The persistence of health inequalities in modern welfare states: The explanation of a paradox. *Social Science & Medicine*, 75(4), 761–769.

- Marmot, M. (2015). *The Health Gap: The Challenge Of An Unequal World*. London: Bloomsbury Publishing, London.
- Maslach, C., Schaufeli, W. B., & Leiter, M. P. (2001). Job burnout. *Annual Review of Psychology*, 52(1), 397–422.
- McBride, C. M., Koehly, L. M., Sanderson, S. C., & Kaphingst, K. A. (2010). The behavioral response to personalized genetic information: Will genetic risk profiles motivate individuals and families to choose more healthful behaviors? *Annual Review of Public Health*, 31, 89–103.
- McGuire, T. G. (2000). Physician agency. *Handbook of Health Economics*, 1, 461–536.
- Pampel, F. C., Krueger, P. M., & Denney, J. T. (2010). Socioeconomic disparities in health behaviors. *Annual Review of Sociology*, 36, 349–370.
- Phelps, C. E. (1978). Illness prevention and medical insurance. *Journal of Human Resources*, 13, 183–207.
- Schmid, C. (2015). Consumer health information and the demand for physician visits. *Health Economics*, 24(12), 1619–1631.
- Van Ryn, M., & Burke, J. (2000). The effect of patient race and socio-economic status on physicians' perceptions of patients. *Social Science & Medicine*, 50(6), 813–828.
- Van Ryn, M., & Fu, S. S. (2003). Paved with good intentions: Do public health and human service providers contribute to racial/ethnic disparities in health? *American Journal of Public Health*, 93(2), 248–255.
- Waitzkin, H. (1985). Information giving in medical care. *Journal of Health and Social Behavior*, 26(2), 81–101.
- Wehby, G. (2016). How genetics can inform health economics. In (Ed.), *The Oxford handbook of economics and human biology* (pp. 366–384). Oxford University Press, New York, NY.
- Wehby, G. L., Domingue, B. W., & Boardman, J. D. (2015). Prevention, use of health services, and genes: Implications of genetics for policy formation. *Journal of Policy Analysis and Management*, 34(3), 519–536.
- Willems, S., De Maesschalck, S., Deveugele, M., Derese, A., & De Maeseneer, J. (2005). Socio-economic status of the patient and doctor–patient communication: Does it make a difference? *Patient Education and Counseling*, 56(2), 139–146.

## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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