Value computation and modulation: a neuroeconomic theory of self-control as constrained optimization *

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Abstract

We develop a theory based on the evidence reported in Hare, Camerer and Rangel (2009) to explain consumption of goods that feature a low-order attribute (e.g., taste) and a high-order attribute (e.g., health). One brain system with access to the low-order attribute computes *the goal value of consumption while another brain system can* modulate *this value, at a cost, by transmitting information regarding the high-order attribute. We determine the optimal modulation and consumption strategy as a function of the cost of information transmission and the environment. We show that in healthy environments, modulation is used to signal surprisingly unhealthy goods so as to trigger abstinence when consumption would ordinarily occur. Conversely, in unhealthy environments, modulation is used to signal surprisingly healthy choices so as to trigger consumption when abstinence would ordinarily occur. From an outside perspective, individuals may appear to under-regulate their choices (self-indulgence) but also to over-regulate them (self-restraint). Both modulation and decisions are affected by factors orthogonal to the decision problem. In particular, taxing executive functions results in less modulation and more inefficient behavior. Finally, the model can shed light on issues related to eating disorders, present-biased preferences, habit formation and compulsive behavior.*

Keywords: neuroeconomic theory, multiple brain systems, self-control, cue-triggered behavior, self-regulation.

JEL Classification: D03, D87.

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1 Introduction

Rational consumer behavior presupposes that individuals make choices consistent with an unambiguous underlying ranking of options (preferences). Many observations run counter to the predictions of rational choice. A prominent example is the consumption of goods with both short term and long term consequences that often features a tendency to overweight short term effects. Numerous inefficiencies in the context of economic choice are attributed to this tendency. In recent years, scholars have proposed formal models to better understand time-inconsistencies, self-control problems and other forms of preference reversals frequently observed in the data.[1]

In parallel, neuroscientists and neuroeconomists have extensively studied the neural correlates of economic choices and have identified the ventromedial prefrontal cortex (vmPFC) as a critical region in studies involving food items, trinkets and money (Clithero and Rangel (2014)). Some studies have extended paradigms to address behavioral anomalies under temporal delays. McClure et al. (2004, 2007) proposed a dual-valuation explanation roughly consistent with the quasi-hyperbolic model, where one system weights immediate rewards and another weights all rewards. Kable and Glimcher (2007) challenged that approach and, instead, argued in favor of a single-valuation explanation where one system responds to a combination of magnitude and delay of the reward. The subsequent research suggests that one system computes goal value and another system can modulate this value by exercising 'self-control' (Knoch and Fehr (2007), Hare et al. (2009), Luo et al. (2009), Figner et al. (2010), Hare et al. (2011)). In this case, exerting self-control typically means integrating high-order considerations –such as health or other long term consequences– in the decision.

Here, we formalize the evidence reported in this last strand of experiments and we discuss its behavioral implications. For this, we build a model of decision-making that incorporates the two distinct processes emphasized in this literature, *value computation* and *value modulation*. The model closely follows the experimental setup by Hare, Camerer and Rangel (2009) (hereafter [HCR]). According to their study, some regions of the vmPFC perform 'value computation', and the consumption decision depends on this value. Ideally, this value computation integrates all the attributes of the good. The key issue is that information about the high-order attribute is not encoded directly in this region. Instead,

[1] These include hyperbolic discounting (Prelec (1989), Laibson (1997), Carrillo and Mariotti (2000), etc.), decision theoretic models of temptation (Gul and Pesendorfer (2001), Dekel, Lipman and Rustichini (2009), etc.), cue-triggered theories of consumption (Laibson (2001), Bernheim and Rangel (2004)), and dual-self models of intertemporal choice (Thaler and Shefrin (1981), Fudenberg and Levine (2006), Brocas and Carrillo (2008), etc.).

its incorporation into value is facilitated by some regions of the dorsolateral prefrontal cortex (dlPFC). Information transmission to the vmPFC, or 'value modulation', is costly but allows a more informed decision.[2] It is worth noting that we do not necessarily associate the low-order attribute to a short term payoff and the high-order attribute to a long-term payoff. The brain is blind to these interpretations. Hence, the theory is applicable to other paradigms that feature conflicts between low- and high-order attributes, such as eating disorders and compulsive behavior. Using the constrained optimization model, we first determine the optimal modulation and choice strategy of the individual. We then derive implications.

The full characterization of the optimal mechanism that governs information transmission and consumption constitutes the main theorem of the paper (Theorem 1). We show that information about the high-order attribute is not transmitted (that is, value is not modulated) when its realization is in a certain compact set H. In the absence of modulation, value is computed under the correct inference that not transmitting information is optimal, that is, the health rating falls in H. Overall, the vmPFC computes the correct goal value based on both attributes when modulation occurs, while it assigns the health rating an average value over H when modulation does not occur. The conditions for no modulation depend on the cost of attention and the relative importance of the high-order attribute. Perhaps more importantly, it also depends on the environment, that is, the distribution from which that attribute is drawn. This information is critical for the inference process and in the calculation of the expected health rating when no modulation occurs. We interpret this as *contextual information*.

This result has a number of implications not covered by the experimental design in [HCR], since the environment and other elements of the model are taken as given in the study. These novel implications are, in principle, general properties that should govern any decision involving the interplay between the vmPFC and the dlPFC. We discuss them in sections 3 and 4.

The key driver of behavior and variations in behavior is the environment in which the decision takes place. Indeed, the latter affects both the decision to modulate the value signal and the decision to consume. Given the cost of modulation, it is optimal to modulate only when the true realization is "far away" from the average rating that will be assigned under no modulation (Corollary 1). Hence, when the environment features a low frequency of unhealthy items, the optimal mechanism dictates to modulate only bad ratings and to

[2] In that respect, the formal model bears some resemblance to the rational inattention theory, where a rational individual trades-off cost and expected value of information (see e.g. Sims (2003), Caplin and Dean (2015), Martin (2017), Caplin, Dean and Leahy (2018) and the discussion in section 2.5).

assign a good average rating whenever there is no modulation. Here, no modulation is often followed by consumption, and modulation is used to signal it is not safe to consume. This scenario refers to the standard self-control narrative. By contrast, when the environment features a high frequency of unhealthy items, the optimal mechanism dictates to modulate only good ratings and to assign a bad average rating whenever there is no modulation. Therefore, no modulation is often followed by no consumption, and modulation is used to signal it is safe to consume. This scenario is closer to a willpower scenario, where the individual sets his mind to not consume by default. Overall, the objective of modulation is simply to override a rule-based process that accounts for contextual information, and it is used to trigger a more informed response. These context effects logically imply that the very same good may be consumed in one environment but not in another (Corollary 2). We can see the parallel with the literature on cue triggered behavior, which argues that environmental cues affect choices either by changing preferences (Laibson, 2001) or producing errors (Bernheim and Rangel, 2004). A notable key difference is that, in our model, the effect of cues in modulation and consumption is endogenous and optimized: the environment determines the expectation of the high-order attribute, which in turn influences the decision to transmit information and consume.

Interestingly, the likelihood of value modulation decreases as cognitive costs increase or health concerns decrease (Corollary 3). This implies that behavior is heterogenous and individual decisions can be traced to indicators of cognitive control or health status. Behavior can also be manipulated, in particular by taxing or helping executive functions, or by reinforcing health cues. This is in line with the experimental findings in neuroeconomics that link self-control to health cues (Hare et al., 2011).

The basic theory can be extended to address behavioral observations in natural settings. Section 4 takes the perspective of a researcher who observes consumption decisions over time. To capture variability in choices (as is usual in the econometrics tradition), we assume that cognitive costs vary and we study the aggregate patterns of consumption of an individual. The model predicts that costly value modulation always results in excessive consumption of highly unhealthy goods but also in insufficient consumption of lowly unhealthy ones (Corollary 4). While over-consumption is a standard result in models of self-control, under-consumption usually is not. In our model, over- and under-regulation are two sides of the same coin. Whenever modulation does not occur, health realizations that lie below or above the average health rating are treated symmetrically, resulting in the two kinds of inefficiencies.

Our theory can account for other paradigms featuring choices over multi-attributes. In particular, we can reinterpret the low-order attribute as an immediate positive reward and

the high-order attribute as a delayed negative consequence. It follows that an exponentially discounting individual subject to costly value modulation behaves like a hyperbolically discounting individual with no cost of value modulation (Corollary 5). In other words, 'decreasing impatience' endogenously emerges in our setting, instead of being an exogenous modeling feature, as in the standard behavioral economics literature (Strotz (1956); Prelec (1989); Laibson (1997)). It also implies that such dynamic reversal of preferences does not occur all the time, only for items with low- and high-order attributes.

Guided by recent evidence in the the context of behavioral disorders, section 5 extends the model to study the consequences of physiological dysfunctions on choice. In the case of *eating disorders*, physiological observations are consistent with an incorrect representation of the distribution of health ratings. If an individual wrongly "believes" that all goods are unhealthy (as, for example, patients suffering from Anorexia Nervosa) or no good is unhealthy (as, for example, patients suffering from Bulimia Nervosa), the modulation decision is compromised resulting in systematic under-consumption in the first case and systematic over-consumption in the second (Corollary 6). This sharply contrasts with the case of correct perceptions in which over- and under-consumption occur for the same individual depending on the health realization. In the case of *addictive substances*, physiological evidence is consistent with modeling features of habit formation in the tradition of Becker and Murphy (1988): total and marginal utility of current consumption are decreasing and increasing in the level of past consumption, respectively. In this framework, we show that an addicted individual will be less likely to incur the cost of modulating the value signal due to his higher incentive to consume. This implies that ignorance of long term health considerations will be more prevalent for addictive substances, not because of self-delusion or other irrational motives, but as the result of an optimal trade-off between the costs and benefits of signal modulation (Corollary 7). We last show that similar principles can apply to better understand a large range of behavioral and mental disorders reported to involve dysfunctions of vmPFC and dlPFC. These include behavioral addictions, compulsive shopping and attention-deficit disorders.

The theory we propose is rooted in neuro-scientific evidence and the model itself shares few features with traditional models of self control and related topics in Economics. First of all, the individual is not modeled as one entity (Becker and Murphy (1988), Laibson (2001), Gul and Pesendorfer (2001)), a succession of time-inconsistent selves (Carrillo and Mariotti (2000), Benabou and Tirole (2004)), or a hierarchical organization with conflicting entities (Thaler and Shefirn (1981), Brocas and Carrillo (2008)). Instead, the individual is a collection of entities that do share the same objective but have different access to relevant information. Second, the individual is not assumed to act in different

modes (as in Bernheim and Rangel (2004)). Instead, the entities that participate in the decision look for an efficient solution at all times. Third, the environment does not affect directly the preferences of the individual (as in Laibson (2001)). Instead, it affects endogenously his decision. For these reasons, the causal mechanisms that lead to behavior in our framework are different from those emphasized in the previous literature, even when predictions are similar. With the exception of the few studies grounded in neuroscience evidence, the vast majority of models have been developed to fit an observed behavior via plausible assumptions rather than to describe a documented mechanism and determine which behavior follows. Economists have traditionally relied on behavioral observations because the causes of behavior were not directly observable. With easier access to evidence, it is now possible to put the theories to the test, not only in terms of their predictions but also in terms of their assumptions. To better illustrate the benefits of modeling physiology, we discuss the plausibility, similarities and differences of existing theories in terms of modeling assumptions in section 2.5 and in terms of predictions in section 4.4. These exercises allow us to appreciate the key methodological advantages of modeling physiology: the ability to offer a unified framework to account for a large array of behaviors traditionally captured through different theories and models (e.g., self-control, saliency, preference reversal, cue-driven behavior) as well as the possibility to unveil the endogenous relationships between them.

Last, even though the methodology is novel in economics, viewing behavior as the result of constrained decision-making implemented by several systems in the brain with different access to information is natural in cognitive neuroscience. While theoretical models in that line of study are computational, and therefore not based on optimization techniques, they recognize that choices result from a trade-off between choice accuracy (or efficiency) and costs (cognitive or metabolic). Critical to the relevance of these models is plausibility: the physiology must support the narrative. We discuss several conceptual frameworks outlined in cognitive sciences in section 2.5.

The paper is organized as follows. In section 2, we present the basic neurophysiological model of value modulation and value computation and we derive the main result. In section 3, we discuss the direct implications of the theory in terms of both the physiological response and the associated behavior. In particular, we study the effects of variations in the primitives of the model and we address alternative reinterpretations that encompass other decision-making paradigms over items with low- and high-order attributes. In section 4, we address behavior from the perspective of a researcher who observes consumption decisions in real life settings and tries to infer consumption patterns and their motivations. Section 5 investigates dysfunctions of the modulation model to address anomalies related to self

control, such as eating disorders, addiction and compulsive behavior. Concluding remarks are gathered in section 6. Proofs of theorem and corollaries are relegated to the appendix.

2 Model

2.1 The decision problem

We propose a theoretical model of self-control based on the *multi-attribute* neurophysiological evidence described in [HCR]. In the experiment, each participant was asked to rate snacks for taste and health separately before completing in the scanner a binary choice task involving these snacks.[3] Participants also indicated the strength of their decision on a scale, providing a measure of their goal values. Some participants chose the healthiest option often while others did not, and the difference in choices was reflected in activity patterns in dlPFC and vmPFC. More precisely, activity in vmPFC was correlated with participants' goal values regardless of their final choices and it reflected the health rating only for subjects who made the healthiest choices. The dlPFC was more active when the healthiest option was chosen. Last, vmPFC and dlPFC exhibited functional connectivity when the healthiest choice was made, indicating that both were part of the same network.[4]

According to this experimental evidence, value signals are encoded by one system (regions of vmPFC), and this value dictates choices. This system receives and encodes information regarding basic attributes (taste in this experiment). A second system (regions of dlPFC) can modulate vmPFC and affect the final choices by facilitating the incorporation of information regarding high-order attributes (healthiness in this experiment). If value is not modulated, information about the high-order attribute is not transmitted and choice is dictated exclusively by taste. If value is modulated, then information about healthiness is transmitted and both attributes are taken into consideration.

To capture this neurobiological evidence, we consider an individual choosing whether to consume ($z = 1$) or not consume ($z = 0$) a "tempting" good with a taste attribute $\theta \in [0, 1]$ and a health attribute $h \in [0, 1]$.[5] The utility of the individual is additively

[3] A reference item was selected for each subject on the basis of the ratings. This item was rated neutral in both dimensions. Each choice involved the reference item and one of the other rated snacks.

[4] This evidence has been corroborated in subsequent studies (Hare et al, 2011, Hutcherson et al, 2012, Chen et al., 2018).

[5] In real settings, choices are often continuous. We adopt a binary setting because our goal is to design a model that captures the existing experimental evidence and that can provide testable predictions for subsequent studies. The existence evidence relies on binary paradigms, so we do not have a clear basis for modeling continuous choice. In Appendix A we show that, provided optimization is carried out by vmPFC in the same fashion when more than two options are present, the main insights hold when we extend the model to continuous consumption choices.

separable and given by the following formulation:[6]

$$\begin{cases} \theta - \alpha h & \text{if} \quad z = 1 \\ 0 & \text{if} \quad z = 0 \end{cases} \tag{1}$$

By definition of being tempting, the good is pleasurable ($\theta \geq 0$) but unhealthy ($h \geq 0$), with $\alpha \in (0, 1)$ capturing the importance of healthiness relative to taste. A high α may reflect the health concerns of an individual who suffers from a diet-related medical condition, although it can also reflect an intrinsic preference for a healthy lifestyle.[7]

To model the context in which the decision takes place, we assume that the attributes θ and h are drawn from continuous and differentiable probability distribution functions $x(\theta)$ and $f_e(h)$, and we denote by $X(\theta)$ and $F_e(h)$ the cumulative distribution functions. We use subscript e to parametrize the distribution function of health, from now on referred to as the "environment". Differences in environment reflect differences in health contexts (e.g., a meal at a fast food vs. a meal at a vegan restaurant). Environments can be ordered using the familiar monotone likelihood ratio property (MLRP):

$$\frac{d}{dh}\left(\frac{f_e(h)}{f_{e'}(h)}\right) < 0 \quad \forall\, e' > e.$$

According to this formulation, items are drawn from a more unhealthy distribution (stochastically higher values of h) in environment e' compared to environment e.

2.2 Representation of information in the brain

Given an environment (a context) and an item (a stimulus), the brain *constructs* the value of the item and implements a decision. In our case, the construction of the value requires representing two attributes. Following [HCR], we posit that this operation is done vía an interplay between two systems. On the one hand, the dlPFC is responsible for encoding a signal regarding the high-order attribute, the healthiness h. It then decides whether to transmit that signal to the vmPFC or not. This transmission of information is called *value modulation*. On the other hand, the vmPFC is responsible for encoding a signal regarding the basic attribute, the taste θ. It also determines the goal value of the item based on all the information available: the taste θ, and the health h provided that the dlPFC transmits it.

[6] Setting the value of not consuming to 0 is a normalization. The analysis trivially extends to cases where an alternative known consumption is offered or, equivalently, where the 'virtuous' act of not consuming provides a fixed and known utility.

[7] Identical support for θ and h together with $\alpha \in (0, 1)$ ensure an interior optimal solution, where consumption is always optimal if $\theta = 1$ and never optimal if $\theta = 0$. More general formulations would exogenously introduce corner solutions of limited interest for our theory.

This aggregation of information is called *value computation*. Under modulation, the goal value incorporates both the health and taste attributes (appropriately weighted) whereas under no modulation it includes only the taste attribute. The consumption decision is made as a function of this goal value.[8] From now on, we will generically call $\mathcal{M}$ the system responsible for value modulation and $\mathcal{C}$ the system responsible for value computation, and defer to section 2.4 a more in-depth discussion of the brain regions involved.

The literature (including [HCR]) has evidenced that signal modulation is *not pervasive*, which suggests that it must be costly. It is generally understood that decision tasks are plagued with limitations due to the cognitive costs associated to information processing (McGuire and Botivnick (2010), Shenhav et al. (2017)). These costs arise from the limited budget of metabolic and attentional resources. Evidence of these costs are tracked to regions involved in cognitive control, such as the dlPFC (McGuire and Botivnick (2010), Shenhav et al., (2013)). More precisely, activity in these regions has been shown to correlate with measures of cognitive costs (reported experienced difficulty, manipulation of memory or attentional load, etc.). To capture this feature, we introduce a cost of modulation c internalized by system $\mathcal{M}$.

The evidence reported in [HCR] also suggests that signal modulation is *discriminative*, that is, it depends on the realization of the health parameter. This means that system $\mathcal{M}$ is "sophisticated enough" to anticipate how system $\mathcal{C}$ computes the goal value, and uses this knowledge to decide whether to send the information. The remaining (but crucial) issue is to determine the way in which system $\mathcal{C}$ interprets an absence of modulation. The evidence is mute on this point. We can entertain two possibilities. One of them is a mechanistic approach where system $\mathcal{C}$ incorrectly takes at face value that no information implies no health concerns (formally, it assumes $h = 0$). The other presupposes that system $\mathcal{C}$ uses information about the environment e and correctly 'infers' that no information transmission from system $\mathcal{M}$ still indicates some positive value of h.[9] The first option, quite reasonable as a first approximation, is in fact unsatisfactory for several reasons. From a theory standpoint, $h = 0$ is only one of many plausible "non-rational" ways to (not) incorporate information, and there is no ground for adopting this focal point rather than any other. More importantly, it will become clear from the discussion in section 3 that this option is also unsatisfactory from both physiological and empirical viewpoints. In the rest of the article, we will focus on the Bayesian model with correct expectations

[8] The set-up is analogous to a team problem, where two agents with a common objective have access to information regarding different features of a joint project. One agent is in charge of the final decision whereas the other must decide whether to transmit his information at a cost.

[9] Needless to say, both approaches are *as if* abstractions of the decision processes involved. It is in no way implied that systems $\mathcal{M}$ and $\mathcal{C}$ literally perform such calculations.

under no modulation.

2.3 Modulation and choice

System $\mathcal{C}$ computes the goal value anticipating that no information transmission by system $\mathcal{M}$ still has some informational content. Formally, we use superscript "+" to denote modulation and "−" to denote no modulation. Under modulation, system $\mathcal{C}$ learns the health and taste realizations of the good and incorporates this information in the decision. The utility of consuming ($z = 1$) and not consuming ($z = 0$) are given by (1):

$$u_z^+(\theta, h) = \begin{cases} \theta - \alpha h & \text{if} \quad z = 1 \\ 0 & \text{if} \quad z = 0 \end{cases}$$

Under no modulation, inferences must be made. Denote by H_e the set of health realizations such that system $\mathcal{M}$ does not modulate the goal value, that is, does not transmit health information to system $\mathcal{C}$ under environment e (naturally, this set needs to be determined endogenously). Let $f_e(h \,|\, h \in H_e)$ be the revised probability distribution function when no information is transmitted: it is understood that $h \in H_e$ but the realization remains unknown. Also, let $E_e[h \,|\, h \in H_e]$ be the conditional expectation. Under no modulation, system $\mathcal{C}$ computes the goal value given the taste realization and the health inferred by the absence of information transmission. The expected utility of consuming ($z = 1$) and not consuming ($z = 0$) are now given by:

$$u_z^-(\theta) = \begin{cases} \theta - \alpha E_e[h \,|\, h \in H_e] & \text{if} \quad z = 1 \\ 0 & \text{if} \quad z = 0 \end{cases}$$

The optimal recommendation by system $\mathcal{C}$ under modulation (+) and no modulation (−) is based in each case on a comparison between the goal value of consuming and not consuming:

$$\begin{cases} z^+ = 1 \quad \text{iff} \quad u_1^+(\theta, h) > u_0^+ \quad \Leftrightarrow \quad \theta > \theta^+ \equiv \alpha h \\ z^- = 1 \quad \text{iff} \quad u_1^-(\theta) > u_0^- \quad \Leftrightarrow \quad \theta > \theta^- \equiv \alpha E_e[h \,|\, h \in H_e] \end{cases} \tag{2}$$

When system $\mathcal{M}$ decides whether to modulate or not, it knows the health rating of the good but not its taste.[10] However, it anticipates correctly how system $\mathcal{C}$ will represent

[10] Our model would also extend with minor modifications to the situation where the taste realization is public information, in which case $\mathcal{M}$ decides between modulation and no modulation under complete information of θ and h. Note that studies report evidence that vmPFC is involved in the computation of all types of values. Activity in dlPFC is reported in the presence of abstract high-order attributes (see e.g. Plassmann et at. 2007, Baumgartner et al. 2011, Hutcherson et al. 2012 and the meta analysis by Clithero and Rangel 2014), which does not preclude that dlPFC is only sensitive to those attributes.

the value of consumption and recommend a decision. Therefore, the values of modulating and not modulating the signal given a health rating h are respectively:

$$V^+(h) = \int_{\theta^+}^{1} (\theta - \alpha h) dX(\theta) - c \qquad \text{and} \qquad V^-(h) = \int_{\theta^-}^{1} (\theta - \alpha h) dX(\theta)$$

where c (> 0) is the cost of signal modulation. Finally, it is optimal to modulate if $V^+(h) > V^-(h)$, which can be rewritten as:

$$\Delta(h, E_e[h \,|\, h \in H_e]) \;\equiv\; \int_{\alpha E_e[h|h\in H_e]}^{\alpha h} (\alpha h - \theta) dX(\theta) \;>\; c \tag{3}$$

Fixing H_e, one can notice that the benefit of modulation, Δ, is convex in h and with a minimum at $E_e[h \,|\, h \in H_e]$:

$$\frac{\partial \Delta}{\partial h} = \alpha\Big(X(\alpha h) - X(\alpha E_e[h \,|\, h \in H_e])\Big) \gtreqless 0 \quad \text{iff} \quad h \gtreqless E_e[h \,|\, h \in H_e]$$

and

$$\frac{\partial^2 \Delta}{\partial h^2} = \alpha^2 \, x(\alpha h) \;>\; 0$$

Since the cost is constant, it means that the set of values such that system $\mathcal{M}$ chooses no modulation is necessarily compact.[11] Formally:

$$H_e = [\underline{h}_e, \overline{h}_e].$$

We further introduce the following parametric assumption.

Assumption 1 *$x(\theta)$ is uniform and $f_e(h)$ is strictly log-concave.*

There is of course a loss of generality in assuming a uniform distribution of taste. This, however, is not excessively problematic from an experimental viewpoint, since it is possible to pre-test items and select a set of goods that satisfies this property. Under Assumption 1, the modulation optimality condition (3) can be rewritten in a simpler form as:

$$\Big|h - E_e[h \,|\, h \in H_e]\Big| > \eta(c, \alpha) \;\equiv\; \frac{\sqrt{2c}}{\alpha} \tag{4}$$

which facilitates significantly the analytical characterization of the optimal modulation strategy as a function of the parameters of the model c, α and e. The main result of the article is the characterization of the optimal modulation mechanism.

[11] Although different tasks (e.g., mentally rotating an object vs. repeating a single digit number) may result in different cognitive costs, processing different values of attributes should not involve different costs. For this reason, we find the assumption of c fixed to be reasonable. From a theoretical perspective, it can be easily seen that our results would immediately extend to a cost function c increasing and concave in h.

Theorem 1 *For all* (c, α, e) *the no modulation set* $H_e = [\underline{h}_e, \overline{h}_e]$ *is unique and given by:*

$$\underline{h}_e = \max\{h^o, 0\} \quad \textit{and} \quad \overline{h}_e = \min\{h^o + 2\eta, 1\}$$

where h^o *is the solution of* $E_e[h \mid h^o \leq h \leq h^o + 2\eta] = h^o + \eta$.

If $E_e[h \mid h < 2\eta] < \eta$ *then* $\underline{h}_e = 0$ *and if* $E_e[h \mid h > 1 - 2\eta] > 1 - \eta$ *then* $\overline{h}_e = 1$.

The optimal modulation mechanism has three critical properties. First, information about health is not transmitted when its realization lies between a lower and an upper threshold. Importantly, these thresholds vary as a function of the environment, indicating that modulation is endogenous to the experimental conditions. Figure 1 illustrates the three possible distinct scenarii. In environment e_1, good health ratings are not transmitted ($\underline{h}_{e_1} = 0$ and $\overline{h}_{e_1} < 1$). In environment e_2, medium health ratings are not transmitted ($\underline{h}_{e_2} > 0$ and $\overline{h}_{e_2} < 1$). In environment e_3, bad health ratings are not transmitted ($\underline{h}_{e_3} > 0$ and $\overline{h}_{e_3} = 1$).

Second, the optimal modulation strategy is unique for any given c, α and e. This is a priori not obvious since it involves a fixed point argument: the benefit of modulation, Δ, depends on the set of health realizations for which there is no modulation, H_e, which itself depends on the benefit of modulation.[12]

Third, in the absence of bounds on health ratings, the set of realizations for which there is no modulation has a constant size (see Figure 1). Formally, $\overline{h}_e - \underline{h}_e = 2\eta$ for all e even though, as discussed above, the location of the interval in which no modulation occurs depends on the environment. In the presence of bounds on health realizations ($h \in [0, 1]$), modulation can be restricted. Overall, the extent of modulation is inversely related to η, which depends exclusively on the cost of self-control and the relative importance of health concerns. For future reference, it might be useful to remember that c and α (or jointly η) affect the size of the interval in which no modulation occurs while e affects the location of this interval on $[0, 1]$.

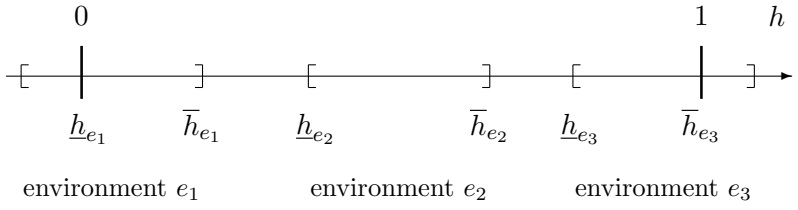

Figure 1. Different modulation regimes

[12] Unlike the first feature which is generally true, uniqueness requires Assumption 1, as reflected in (4).

At this stage, some important remarks are in order. First, based on the predictions of Theorem 1, it is possible to rule out alternative models. In particular, decisions would be context-independent at equilibrium under the (non-Bayesian) assumption that system $\mathcal{C}$ makes no inferences when the decision is not modulated. In that alternative specification, modulation would occur only when $h > \eta(c, \alpha)$. This would leave no role to the environment. It would also preclude modulation at the bottom as well as modulation at top and bottom (environments e_2 and e_3 in Figure 1). Alternatively, if we assumed that system $\mathcal{C}$ had direct access to h, or that there was no uncertainty about h, modulation would not occur. Thus, modulation and context effects are linked to both the existence of uncertainty and the ability to make inferences. In particular, the implications in section 3 would not hold under these alternative models.

Second, when decision-making only involves a low-order attribute, the decision is only reflected in $\mathcal{C}$ and the decision is always optimal. Cognitive costs and environment have no effect on the decision. This trivial case corresponds to standard economic decision-making represented by fully informed, unambiguous preferences over outcomes.

Last, we have intentionally abstained from referring to self-control. In the literature–including in [HCR]–modulation and the choice of the healthy alternative are interpreted as self-control. According to Theorem 1, modulation does not necessarily occur to signal bad health ratings and to trigger healthy choices. Not only modulation may signal good health ratings, but also healthy choices can occur in the absence of modulation. More generally, modulation and choices depend on the environment: the contextual representation (which affects the non-modulated goal value) may lead to healthy choices by itself. Overall, our setting encompasses but is broader than the standard self-control paradigm, as we will emphasize in sections 3 and 4.

2.4 The optimal mechanism as a dual process mechanism

The theorem predicts that both modulation and behavior depend on three critical factors: the environment, the cost of modulation and the concern for health. However, none of these factors have been modeled in [HCR]. Therefore, our theory offers not only the possibility to organize the existing evidence, but also to predict behavior in situations in which the experimenter can control and/or manipulate e, c and α (see section 3 for detailed implications for experiments). Key to these new tests is the ability to represent e and to implement modulation when appropriate given e. The objective of this section is to investigate the evidence that suggests such mechanisms are in place.

A simple reinterpretation of the optimal mechanism is as a dual-process: rule-based vs. cognitive. Consider a decision-maker who consumes in environment e with corresponding

known distribution $f_e(h)$. One process represents a rough outline of the high-order features of the environment in which consumption takes place. It is characterized by a default costless representation of the environment e that assesses the health context. Formally, it assigns to health the expected value $E_e[h \mid h \in H_e]$, and it triggers consumption only when the taste representation θ offsets that assigned health. In an unhealthy environment (e.g., a bar), the process represents a high $E_e[h \mid h \in H_e]$ and it tends to trigger abstinence (θ is likely to be below $\alpha E_e[h \mid h \in H_e]$). Overall, it takes the form of a *rule-based* process that recommends an action based only on the contextual information $E_e[h \mid h \in H_e]$). The other process represents with exactitude h, the high-order feature of the item to consume. It is characterized by a costly computation of h leading to an optimal choice, and can be viewed as a *cognitive* process. For a given modulation cost, the switch between the rule-based process and the cognitive process occurs when the rule is not appropriate and likely to generate a decision that departs substantially from the optimal one.

A natural question for the reader interested in the neurophysiology of the decision process is to determine the feasibility of this mechanism from a physiological viewpoint. [HCR] provides only evidence that $\mathcal{C}$ represents the goal value while $\mathcal{M}$ (sometimes) transmits higher order information. In particular, because the context is fixed, [HCR] does not provide evidence that vmPFC encodes contextual information. More generally, we would like to determine if there is additional evidence supporting the existence of (i) a rule-based process that encodes contextual information, (ii) a cognitive process that overrides the rule-based process to facilitate information transmission and (iii) system(s) that operate the switch between the two processes.

$\mathcal{C}$ and $\mathcal{M}$ are part of the cognitive process. Studies have shown that vmPFC, dlPFC and orbito-frontal cortex (OFC) are central actors of the goal value system (Hare et al. (2008), Plassmann et al. (2007)). Basic information about taste is computed in the mesolimbic regions involved in reward processing and sent to the vmPFC/OFC regions where the goal value is computed given the information received. As explained in section 2.2, dlPFC communicates costly information regarding the high-order health attribute to vmPFC over a range of values, while vmPFC encodes subjective taste and integrates the high-order attributes only when dlPFC sends that information (Hare et al. (2009)). Together, vmPFC and dlPFC ($\mathcal{C}$ and $\mathcal{M}$) are key regions of the cognitive process.

Contextual information is represented in $\mathcal{C}$. Experiments have shown that cues and frames affect the activity pattern in value-related regions of the OFC (Plassman et al. (2008), De Araujo et al. (2005)). In particular, Rudorf and Hare (2014) found that value reflected in vmPFC activity varies as a function of context. Also, the literature on habitual

control shows that the habit system (mostly the basal ganglia) assigns through repeated exposure values to actions performed in specific contexts, and is capable of selecting actions yielding the highest values (Niv and Montague (2008), Daw and O'Doherty (2013)). These results provide a framework to explain how associations between external factors (such as e) and actions are learned over time and how they are represented in each consumption episode. The evidence also indicates that a model that precludes inferences from the environment is not plausible. Therefore, we should expect activity in OFC/vmPFC regions to reflect $E_e[h \mid h \in H_e]$ and to change as the environment changes.

Behavior adjustments are implemented by $\mathcal{M}$. The medial frontal cortex (MFC) and the adjacent anterior cingulate cortex (ACC) have been shown to be involved in multiple situations involving executive control.[13] Converging evidence supports the view that the dorsal ACC and dlPFC achieve jointly cognitive control, with the dorsal ACC responsible for monitoring performance and dlPFC responsible for adjusting behavior (Botvinisk et al. (2001); Botvinik (2008); Kerns et al. (2004); Koechlin and Summerfield (2007); Koechlin et al. (1999, 2000), Shenav et al. (2017)). The decision to engage in cognitive control rather than rule-based control is driven by expectations about prospects and the eventual selection involves the dlPFC (Dixon and Christoff (2012), Bahlmann et al. (2012), Yee and Braver (2018)). This evidence is consistent with both the known anatomical and functional connections between the MFC/ACC and the PFC systems (Petrides (2005), Taren et al. (2011), Rushworth et al. (2011)).

Taken together, all this evidence suggests that a rule-based process is in place to represent contextual information and intrinsic motivation while a network of regions in ACC/MFC/PFC constructs and represents high-order information. Within those regions, the dlPFC implements the switch to the cognitive process to transmit the information when optimal. This mechanism is also consistent with our current understanding of dietary choices. These choices are regulated via either the habitual control system that uses contexts/actions associations built from past experience or the more flexible goal-directed control system capable of representing future consequences more accurately (Rangel (2013); Han et al. (2018)).

[13] These include, for example, motivation of behavior (Rushworth et al. (2007)), action monitoring to prevent undesirable actions (Bush et al. (2000), Paus (2001), Ridderinkhof et al. (2004)), behavioral change during option evaluation (Kolling et al. (2016), Shenav et al. (2016)), conflict resolution (Cole et al. (2014)), and representation of rule-context associations to implement executive control (Kouneiher et al. (2009)).

2.5 Similarities and differences with existing models

Before unpacking the physiological and behavioral implications of our model, we would like to take a step back and address the differences and similarities between our approach and other existing models in economics and cognitive neuroscience.

Two important notes are in order. First, the nature of modeling differs across fields. In economics, a model is a causal relationship between assumptions and consequences, and it is formalized as a constrained optimization. In most other fields, however, a model is a descriptive framework and it rarely uses an optimization strategy to link assumptions to consequences (although the description may still be consistent with an underlying optimization). Second, the approach to modeling is impacted by the available information. Natural science is usually interested in unveiling the mechanisms that underly observations, and researchers design experimental strategies to isolate and understand the "true" causal relationships. By contrast, the object of interests for economists has traditionally been behavior, thereby ignoring the causes for it. Instead, economists build models that are supposed to capture those causes, in a sort of untested black box. These distinctions are important because a neuroeconomic theory will lie at the intersection of these approaches: it cares about the true causal links (as in natural science fields) and it views them as an optimization that leads logically to behavior (as in economics).

Theories can be compared against each other and against the evidence in terms of their assumptions and their predictions. This section is concerned with the assumptions and the causal relationships that are explored. We relegate a discussion of the predictions to section 4.4.

2.5.1 Models in cognitive neuroscience

From the perspective of cognitive and computational neuroscience, people select a cognitive strategy that makes the best trade-off between costs and accuracy (Shenav et al., 2017). This calculation is formalized by the *value of computation* in the artificial intelligence literature and by the *expected value of control* in the cognitive control literature. The two approaches capture the decision problem as the difference between benefits and costs, and only differ in the interpretation of the costs: computational resources in the former (Lieder et al. 2014) and control costs in the latter (Bogacz et al. 2006; Shenhav et al. (2013)). This line of modeling is consistent with the physiological evidence reviewed above: behavior results from the choice between two processes (a rule-based process and a cognitive process) and the selection of the optimal trade-off between rewards and costs. Instead of assuming the existence of a trade-off, we model it from primitives and we link behavior to these primitives through optimization techniques.

Another approach sees behavior as the result of an information accumulation process: a decision is made when "enough" information supporting the decision is obtained. The drift-diffusion model (DDM) conceptualizes this idea by setting exogenous bounds towards which information is accumulated (Ratcliff and McKoon (2008)). Bounds capture the notion that information is not accumulated indefinitely due to costs in information processing. These costs can be interpreted as physiological constraints that prevent full accumulation, metabolic costs or cognitive control costs. The model has been applied successfully to fit behavior, including in value-based decisions (Krajbich and Rangel (2011)) and recent evidence suggests that it represents actual calculations performed by value regions (Domenech et al. (2017)). The DDM can be rationalized by standard models of optimal information accumulation in which bounds emerge endogenously as a function of the cost of accumulating information and the stakes (Brocas (2012)). Our model can be interpreted as a one-period, reduced-form optimal DDM in which starting with information about θ and the context, either accumulation occurs at cost c to learn h or no accumulation occurs.

Last, and more directly related to the case of self control, multi-attribute items have been modeled through a weight system (Hutcherson et al. (2015); Berkman et al. (2017)). The standard interpretation is that networks such as the lPFC influence those weights, hence shaping information accumulation and choice (Hare et al. (2011)). This captures the same effect as our one-period model. Because the health attribute is not always passed to system $\mathcal{C}$, the optimal mechanism operates as if the attributes have different weights depending on the circumstances. The role of system $\mathcal{M}$ is to set the conditions under which information regarding health is passed to system $\mathcal{C}$ given the cognitive costs. In our setting, these decisions are optimal.

2.5.2 Models in Behavioral Economics

Models in economics endow decision-makers with utilities that represent their preferences. In that tradition, individuals are one entity with set goals, and behavior is roughly classified in two categories: rational and anomalous. Self-control behavior has emerged as a systematic and significant departure, and the literature has focused on building models to rationalize it. While a few models have tried to reconcile it with the standard one entity approach by modifying axioms (e.g., Gul and Pesendorfer (2001)), the vast majority has proposed extended utility representations. Critically, most models have been built with no observation of the mechanisms that underly decision-making, and they have been retained for their ability to *fit* observed behavior.

The first and perhaps most common approach to self-control posits an inter-personal

conflict between selves, captured via hyperbolic discounting. Absent any commitment technology, the individual takes suboptimal actions, whether by succumbing to temptations when rewards are immediate and costs are delayed or by procrastinating when costs are immediate and rewards are delayed (O'Donoghue and Rabin (1999)). It has been shown that commitment strategies may alleviate these detrimental tendencies. For instance, and related to the current setting, imperfect information may be exploited. Indeed, by remaining strategically ignorant about payoffs (Carrillo and Mariotti (2000)) or by choosing whether to forget information (Benabou and Tirole (2004)), the individual can sometimes induce their future self to take superior actions. Such approaches are reminiscent of psychological effects such as optimism or self-confidence and psychological strategies such as willpower or self-reputation. However, the results are obtained under strict assumptions that are not necessarily true. In particular, hyperbolic discounting models cannot apply to settings in which the temporal separation between rewards and costs is not significant. They also feature an intrapersonal conflict while the current evidence ([HCR] and the literature reported here) supports the idea of cooperation under constraints. Last, the informational strategies are speculative. For example, imperfect memory (a necessary ingredient in Benabou and Tirole (2004)) seems orthogonal to the ability of individuals to exercise (or not) self-control. The evidence reported here suggests a much simpler and direct informational setup.

Another approach consists in modeling the individual as a dual-self, in which a long-term planner –who cares about the stream of payoffs– interacts with a short-term doer –who cares exclusively about the current payoffs (Thaler and Shefrin (1981), Brocas and Carrillo (2008)). This approach fits the evidence of brain modularity, whereby different systems (such as vmPFC and dlPFC) have different access to information and solve different subproblems. However, the hierarchical representation also assumes a conflict of objectives that may not necessarily reflect our current physiological evidence.

Some researchers have proposed to model a conflict between processes rather than a conflict between sub-entities (Benhabib and Bisin (2005), Bernheim and Rangel (2004)). This framework is reminiscent of the expected value control theory described earlier, and it features a process that leads to rational behavior and imposes cognitive control while another leads to suboptimal or dysfunctional outcomes. The framework is also reminiscent of our rule-based and cognitive processes. However, these models are normative: they do not model the informational constraints or the interplay between systems that rationalize the existence and function of the processes.

Economists have also thoroughly studied the problem of addiction (Becker and Murphy (1988), Laibson (2001), Bernheim and Rangel (2004)). Even though self-control and

addiction are linked, we view addiction as a dysfunction and we relegate its discussion to section 5. Notice, however, the parallel between the 'cues' in Laibson (2001) and the 'environment' in our paper. Our model rationalizes how contextual information enters the evaluation of options and shapes behavior, a feature that is taken as exogenous in Laibson (2001)'s model.

Self control and impulsive choice have also been approached through the lens of multi-attribute features and salience effects, reminiscent to those in our model (Köszegi and Szeidl (2013); Bordalo et al. (2013)). The studies presuppose that the decision-maker has a biased view of some attributes or assigns specific weights to them, which result in inefficiencies. As we shall see, endogenous information processing will generate a tendency to overweight one attribute with respect to the other. This effect will take place at equilibrium to serve an efficiency requirement.

Finally, our article is also related to the growing literature on rational inattention that started with Sims (2003). The basic premise of this elegant theory is that individuals optimize information acquisition by trading-off the expected value of information and its cost.[14] The literature has considered two main variants to study limited attention. Under the "consideration set" approach (see e.g., Caplin et al. (2011) and Manzini and Mariotti (2014)), individuals focus on a subset of the available options and ignore the rest. Recent laboratory experiments using a variety of non-choice data techniques suggest that this approach is consistent with the empirical evidence.[15] Unfortunately, there is still no consensus on how the consideration set is formed. Under the "imperfect perception" approach (see e.g., Krajbich et al. (2010) and Caplin and Martin (2015)), the decision maker forms statistical inferences and chooses the option that maximizes utility given an imperfect, constrained and noisy information accumulation process. As in the general rational inattention theory, information in our paper is costly and optimized. Our approach is closer to the imperfect perception strand, since we assume that the information flow is restricted and choices are optimized conditional on that information. A notable difference is that we assume a "game" between brain systems with different access to evidence but no conflict of interests. We then explicitly model the optimal information transmission given the cost-benefit tradeoff.

Overall, our model is firmly grounded on the neurophysiological evidence. It is therefore closer to the cognitive and computational neuroscience models and the process-based economic models than to the other behavioral economic theories. We also model features

[14] Notable applications include Woodford (2009) and Matejka and McKay (2015). We refer to Mackowiak et al. (2018) for a survey and Caplin et al. (2018) for a generalization of the Shannon cost function.

[15] The techniques include response times (Caplin and Martin (2016)), mousetracking (Brocas, et al. (2014), Brocas et al. (2018)) and eyetracking (Reutskaja et al. (2011)).

that are traditionally taken for granted, which allows us to unveil causal links more clearly. One key difference with most of the self-control literature is the *absence of a conflict of interests*. Both $\mathcal{M}$ and $\mathcal{C}$ care about representing the utility of the individual correctly. To our knowledge, this feature is shared only with the work by Cunningham (2013) on automatic vs. reflective judgment. In both models, the communication cost and restricted access to information results in behavior (choices in our case, judgements in Cunningham (2013)) that differs from what we would obtain if systems could pool information freely.[16]

3 Direct implications of the theory

In this section, we address the implications of the theory both in terms of what we should observe in experimental tests and in terms of the general properties of behavior.

Our theory can be tested directly by extending the food experimental paradigm of [HCR] to account for contexts. This can be achieved by incorporating several treatments, each representing an environment. For instance, a treatment may expose subjects primarily to healthy foods (environment e_1 in our theory) and another may expose them primarily to unhealthy foods (environment e_3 in our theory). The set of foods as well as their frequency of appearance would be disclosed ex-ante so that participants know their environment.[17] The experiment within each treatment would be the same as [HCR] and would permit the collection of both physiological and behavioral data. These implications are covered in sections 3.1 and 3.2. Our theory is also sensitive to the parameters α and c. The experimental paradigm in [HCR] could be extended to measure or manipulate these parameters, as addressed in section 3.3. Finally, the theory is relevant to predict physiological and behavioral patterns in other paradigms with low-order and high-order attributes for which decisions are known to be supported by similar value-based processes. These alternative applications are discussed in sections 3.4 and 3.5.

[16] In Cunningham (2013), the automatic system has access to low-order information and an extra piece of private information (implicit knowledge) while the reflective system has access to high-order information. The automatic system makes a judgement observable by the reflective system, creating an opportunity for the latter to make inferences and to aggregate information. Translated into our framework, the model would correspond to a case where $\mathcal{C}$ knows θ and an extra variable k, and signals this information to $\mathcal{M}$. System $\mathcal{M}$ knowns h and infers θ from the signal (and does not care about k). Cunningham (2013) is interested in judgements and biases defined as differences in expectations of each system compared to a third system that would aggregate all information. It does not formally model the decision process nor discusses which brain systems and physiological constraints this two-system approach is meant to represent.

[17] In such experiments, health and taste ratings are collected from participants in a pre-test stage. Participants are asked to rate many items and a personalized subset is retained. The procedure allows the experimenter to build distributions of values that are comparable across subjects.

3.1 Modulation regimes

Our model predicts that the cognitive process optimally overrides the rule-based process when contextual information departs significantly from the true information.

Corollary 1 - Modulation of the improbable. *Value modulation occurs when the realized health rating is unexpected: high if the expectation is low, low if the expectation is high, and high and low if the expectation is intermediate.*

If we fix c and α, the type of modulation depends on the distribution of the health rating. As the environment shifts towards stochastically more unhealthy goods (e increases), the optimal strategy of system $\mathcal{M}$ shifts from modulation only at the top, to never modulation or modulation at the top and bottom, and finally to modulation only at the bottom (e_1 to e_2 to e_3 in Figure 1). The intuition is as follows. By understanding when system $\mathcal{M}$ modulates the signal, system $\mathcal{C}$ makes inferences regarding the health rating. If $\mathcal{M}$ communicates only bad (good) health ratings, then $\mathcal{C}$ infers the rating is relatively good (bad) in the absence of modulation. Therefore, consumption is likely (not likely) to take place. Therefore, to prevent the individual from consuming highly unhealthy items, there are two possible strategies. The first consists in $\mathcal{M}$ communicating only bad ratings. In that case, the individual is likely to consume unless the rating is disclosed. The second strategy consists in $\mathcal{M}$ communicating only good ratings. Under that alternative, the individual is likely to not consume unless the rating is disclosed. When the item is drawn from an unhealthy distribution, the second strategy is optimal, as it requires to "pay" the modulation cost less often. When the item is drawn from a healthy distribution, the first strategy dominates for the same cost saving reasons. Overall, costly information transmission should be utilized when news is striking and unexpected. When news is expected, information is not transmitted, costs are spared and no-news is correctly interpreted as ordinary news.

The result is illustrated in Figure 2. The left graph corresponds to e_1, an environment where goods are typically healthy ($E_{e_1}[h \,|\, h < 2\eta] < \eta$) and the right graph to e_3, an environment where goods are typically unhealthy ($E_{e_3}[h \,|\, h > 1 - 2\eta] > 1 - \eta$). In the absence of a cognitive cost, optimal consumption occurs whenever $\theta > \alpha h$. In the presence of a positive c, consumption occurs when the taste parameter is above a cutoff, denoted $\bar{\theta}(h, c)$ (Figure 2, bold line). As described in equation (2), αh is the cutoff above which consumption occurs under modulation and $\alpha E_e[h \,|\, h \in H_e]$ is the cutoff above which consumption occurs under no modulation. In the healthy environment (e_1), modulation occurs when ratings are bad ($h \in [h^o + 2\eta, 1]$) and no modulation occurs when ratings are good ($h \in [0, h^o + 2\eta]$). By contrast, in the unhealthy environment (e_3), modulation

occurs when ratings are good ($h \in [0, h^o]$) and no modulation occurs when ratings are bad ($h \in [h^o, 1]$).[18]

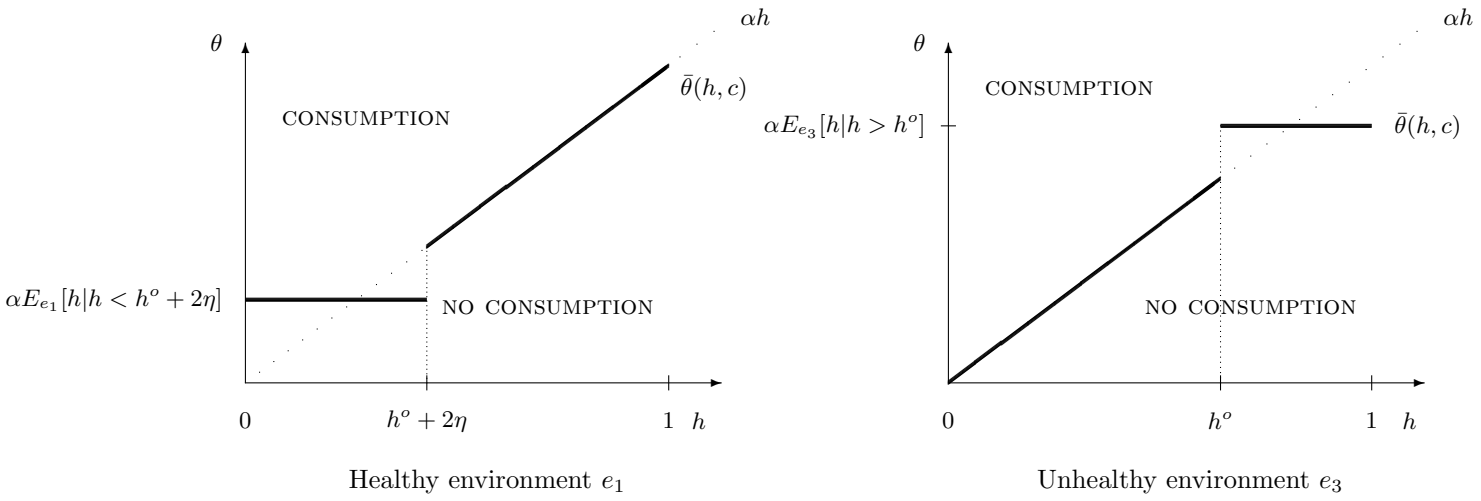

Figure 2. Consumption given taste and healthiness (bold line represents $\bar{\theta}(h, c)$)

As noted earlier, self-control, defined as the act of refraining from consuming an unhealthy tempting item, is only a special case of the modulation mechanism. It corresponds to environment e_1: in the absence of modulation, consumption of tasty foods often occurs, and modulation is used to prevent consumption of those that are truly unhealthy. By contrast, modulation in environment e_3 aims at inducing consumption of goods that are lowly unhealthy and which would otherwise not be consumed. In other words, optimal modulation is not a synonym of self-control. Its objective is to override contextual information when it is distant from the true information, so as to reverse rule-based behavior. When contextual information is likely to induce no consumption, behavior is reminiscent of willpower. As we will develop in the next section, an interesting implication is behavioral heterogeneity within an individual. A person will choose different modulation strategies depending on the environment, so that the same tempting good will be consumed in some situations but not in others.

3.2 Cue-dependent behavior

Our model predicts an endogenous relationship between environment and consumption, with choices depending on the context in which the decision is made.

[18] In the "intermediate" environment e_2 (not shown in Figure 2), health ratings are average (formally, $E_{e_2}[h \,|\, h < 2\eta] > \eta$ and $E_{e_2}[h \,|\, h > 1 - 2\eta] < 1 - \eta$) and modulation occurs when $h \in [0, h^o] \cup [h^o + 2\eta, 1]$.

Corollary 2 - Accounting for the environment. *The likelihood of consuming the tempting good depends not only on the health realization h but also on the environment e from which it is drawn.*

The environmental cue provides information about the distribution of the high-order attribute. If a decision-maker is offered a cocktail in a bar or a meal in a fast food parlor, chances are that the alcoholic and fat content of those goods are greater than if the items are presented by a health-conscious friend at a dinner party. The mere fact of knowing where the decision takes place endogenously affects behavior, simply because the environment affects the expectations about the health dimension. Intuitively, engaging cognitive resources to override these expectations is beneficial only when the true information is far from the expectation. Given different cues are associated with different expectations, the true information may be close enough in some cases and far in others. Therefore, the realized health rating of a good may be communicated to system $\mathcal{C}$ in one environment and not in another, leading to different consumption decisions. Experiments that provide cues to reassess the distribution of health attributes before making choices (Hare et al. (2011)) may trigger such mechanism.[19] Following the previous example, it implies that a cocktail at a bar may trigger a different response than *the same* drink at a friend's party.

The concept of environment in our proposed experimental setting is clear: it refers to the distribution of health ratings of the goods. In real-life settings, "meal at a fast food restaurant" or "meal at a friend's house" also provide clear contextual information. However, other settings may be less clear and the individual could, in principle, manipulate the perception of the context (e.g., represent this exact fast food restaurant vs. all fast food restaurants vs. all restaurants). In experiments where individuals are asked to attend to cues (focus on certain attributes, as in Hare et al. (2011)) or use general strategies (increase or decrease cravings, as in Hutcherson et al. (2012)), people may also differ in the way they represent contextual information.[20] This is important because the strategic representation of cues (which is not modeled here) may be part of a more general mechanism to reduce inefficient consumption.[21]

[19] In other words, cues may be understood as an implicit information reminder that the environment is likely to be unhealthy. Under that interpretation, dlPFC would potentially facilitate both the initial reassessment of the environment and the collection of health information after a choice is prompted.

[20] This is consistent with evidence in Maier and Hare (2019) that contrasted behavior and neural activity in a dietary food task and in an independent emotion regulation task. The evidence suggests that the processes utilized in both tasks to promote choices that conform with goals are related (tracked to medial and lateral PFC).

[21] Recent evidence suggests that combined volume of the dlPFC and vmPFC predicts dietary self-regulatory success in the presence of cues (Schimdt et al. (2018)).

The implications from sections 3.1 and 3.2 can be tested jointly. In a healthy environment featuring a high frequency of items with relatively good health ratings, dlPFC activation should be more prevalent when the most unhealthy items are presented and abstinence should follow whenever beneficial. This also should be accompanied by insufficient consumption of the lowly unhealthy items because good health ratings are not transmitted (and the decision-maker acts in expectation). By contrast, in an unhealthy environment featuring high frequencies of items with bad health ratings, dlPFC activation should be more prevalent when the least unhealthy items are presented, and consumption should follow whenever beneficial. This should be accompanied by over-consumption of the highly unhealthy items because, this time, bad health ratings are not transmitted.[22] A comparison of patterns of activation in the vmPFC in the different environments would also allow to test for the representation of contextual information $E_e[h \mid h \in H_e]$. Other things being equal, contrasts between treatments should reflect contextual information.

3.3 Pointers and distractors

This section studies changes in c and α in a fixed environment. Our theory predicts that both cognitive costs and health concerns are critical to the way information is processed to make a decision. In particular, modulation occurs only if it is costly to forego information. Given the congruence of objectives between $\mathcal{M}$ and $\mathcal{C}$, system $\mathcal{M}$ would always modulate the signal and system $\mathcal{C}$ would always incorporate that information in the goal value in the absence of a cost. Comparative statics have simple implications regarding behavior.

Corollary 3 - Manipulation of decisions. *Choices can be affected with the use of distractors and multi-tasking:* $\frac{d\eta}{dc} > 0$, $\frac{d\eta}{d\alpha} < 0$, $\frac{d^2\eta}{dc^2} < 0$, $\frac{d^2\eta}{d\alpha^2} > 0$ *and* $\frac{d^2\eta}{dc\,d\alpha} < 0$.

Recall from Theorem 1 that value modulation is inversely related to η. This means that modulation is more prevalent the lower the cost of exercising it ($d\eta/dc > 0$) and that there are increasing returns in lowering this cost ($d^2\eta/dc^2 < 0$). Also, as individuals become more and more concerned with health, value modulation increases but at a decreasing rate ($d^2\eta/dcd\alpha < 0$). Finally, subjects who are more concerned about the health effects of their diets engage more in value modulation ($d\eta/d\alpha < 0$) but, again, at a decreasing rate ($d^2\eta/d\alpha^2 > 0$). Overall, manipulations of c and α should affect the involvement of $\mathcal{M}$, hence the patterns of activation in dlPFC and vmPFC, as well as the decisions.

This is consistent with existing neuro-experimental evidence. In the context of dietary choices, Hare et al. (2011) study the effect of health cues on the integration of health

[22] Over- and under-consumption could be pinned down by comparing actual decisions with health and taste ratings collected in the pre-test stage.

attributes into decisions. Health cues make it less effortful for the individual to integrate health components (diminish c) and they also make the benefit of healthiness more salient (increase α). The study reveals that tasty but unhealthy snacks are avoided more often and dlPFC is more strongly activated in the presence of health cues. These behavioral and physiological observations are consistent with the predictions of Corollary 3. As noted earlier, these cues may also affect ex-ante context representations. The design in that study does not allow us to tease the two mechanisms apart. Nevertheless, the activation of dlPFC provides strong support for the attentional cost effect.

A more precise test of our theory requires considering each parameter in isolation. Even though it is obviously difficult to estimate the cognitive cost of modulation, it is possible to study the effect of variations of this cost. For example, a higher IQ and a greater capacity to focus attention should translate into a lower cost of modulation.[23] This suggests that cognitive measures such as IQ or working memory tests may be used as a proxy of c for each participant. Such a design would allow us to test the prediction across individuals. Perhaps more easily testable, comparisons can also be performed within individuals and across trials. In particular, the experimenter can manipulate the cost of attention by using distractors (e.g., asking participants to multi-task) or emphasizing the health characteristics of the products.[24] Last, it is also possible to study the effect of health concerns by collecting self-reported measures of the subject's quality of life as well as objective measures of the subject's health condition (e.g., blood pressure, cholesterol levels, BMI) to estimate α.[25]

3.4 Healthy aversive goods

The fundamentals of our theory relies on the interplay between goal value computation and representation of high-order attributes. The mechanism should extend to other decision featuring a low-order and a high-order attribute, such as goods that are aversive but provide health benefits to the decision-maker. Noting that vmPFC is known to encode the value of items with aversive goal value (Plassmann et al. (2010)), we hypothesize that adding a health component to the decision-problem (e.g., a medicine or a healthy food with poor taste) should trigger a similar trade-off as in section 2.3.

The analogue of the utility in (1) is

[23] This may be related to the individual differences in the neuroanatomy of the dlPFC and the vmPFC that predicts differences in the ability to exercise self control (Schmidt et al., 2018).

[24] Note that testing for marginal effects requires cardinal measures. The number of distractors to attend to may be a proxy of c.

[25] The implementation of such study should account for changes in biological and mood indicators during the day. In particular, the information should be collected always at a fixed time.

$$\begin{cases} -\theta + \alpha h & \text{if} \quad z = 1 \\ 0 & \text{if} \quad z = 0 \end{cases}$$

An immediate extension of Theorem 1 predicts three regimes and the solution exhibits the same qualitative properties as the ones discussed in sections 3.1, 3.2 and 3.3. When the health benefits are expected to be small, modulation occurs when the realized health benefits are large to promote consumption of the aversive good. When the health benefits are expected to be large, modulation occurs when the realized benefits turn out to be small to prevent the decision-maker from consuming aversive items inefficiently. Last, when the health benefits are expected to be moderate, modulation occurs optimally for low or high health realizations. Both physiological and behavioral predictions can be tested in the same way as previously discussed in the context of tasty unhealthy foods.

3.5 Temporal delays

High-order attributes generally represent information that is complex to assess. One example is temporal separation until the realization of the distant component of the good. Note that many classical theories around self-control in economics presuppose a time element (Carrillo and Mariotti (2000), Benabou and Tirole (2004)). Although this is not the interpretation in [HCR]'s multi-attribute model of self-control, it also corresponds to the more traditional neuroscience approach to dynamic choices and self-control (see e.g. Luo et al. (2009)).

From the physiological viewpoint, neuroimaging studies suggest that similar regions and processes as those detailed in the previous sections are involved in time discounting paradigms. In particular, ventral striatum, mPFC and OFC, together labeled as limbic reward areas, are activated in response to immediate rewards while regions in the lPFC are involved in inter-temporal trade-offs (McClure et al. (2004, 2007); Hare et al. (2014); Zha et al. (2019)). In apparent contradiction, Kable and Glimcher (2007) show that those limbic reward areas are also encoding delayed values. However, these findings can be reconciled. Indeed, they are both consistent with the hypothesis that system $\mathcal{C}$ (here, the limbic reward areas) encodes information about the immediate taste attribute but is also capable of representing general properties of the high-order attribute, the delayed health effects. In parallel, system $\mathcal{M}$ (here, the lPFC) has access to the high-order information and supervises decisions. We conjecture that a cognitive process involving these two regions is in place, with $\mathcal{M}$ sending precise information to $\mathcal{C}$ about delayed health ratings when the expected delays computed vía a rule-based process are likely to result in significant inefficiencies.

Our basic model can therefore be straightforwardly extended to encompass the time delay domain. Formally, suppose that consumption at date 0 has a fixed and known negative health effect equal to one which occurs at an unknown date t ($\in \{1, ..., \tau\}$) with τ finite but possibly large. Signal modulation by system $\mathcal{M}$ consists in the costly transmission of t, the delay between the time where the pleasurable taste is enjoyed and the time where the negative health effect is suffered. Under the standard assumption of exponential discounting of payoffs, the analogue of the utility described in (1) is now:

$$\begin{cases} \theta - \delta^t & \text{if} \quad z = 1 \\ 0 & \text{if} \quad z = 0 \end{cases} \tag{5}$$

where the low-order and high-order attributes are now taste and delay, θ and t, instead of taste and healthiness. Following the same methodology as in section 2.3, it is straightforward to show that modulation occurs when the realization of t is in a certain set T. At equilibrium, modulation dominates no modulation when:

$$\left| \delta^t - E[\delta^t \mid t \in T] \right| > \eta(c) \equiv \sqrt{2c} \tag{6}$$

The equilibrium modulation interval has similar properties to those in section 2.3, as evidenced by (4) and (6). Here again, the environment affects the decisions, and the same problem may result in different choices depending on the context. This is because information transmission occurs only when the realization of t is improbable. When the harm is likely to be suffered in a distant date, system $\mathcal{M}$ signals only near dates to avoid consuming goods with pernicious consequences in the short term. Naturally, the same logic also applies to other decisions in which pleasant consequences are immediate while unpleasant ones are delayed to an uncertain date, such as consumption and savings decisions for which present consumption is likely paired with future indebtedness. It also applies to paradigms involving a negative low-order immediate return and a positive high-order future benefit enjoyed at an uncertain date. These include aversive goods that grant future health benefits, or immediate efforts associated with future rewards.

Studies have shown that the mechanisms underlying value computation are largely independent of domains. Regions including vmPFC respond to changes in the magnitude of rewards in the goods, food or money domains (Bartra et al., 2013). They also represent costs and efforts (Lin et al, 2012; Aridan et al., 2019) as well as social rewards (Haller and Schwabe, 2014). Besides, the presence of more abstract or complex elements involve cognitive processes hosted by the PFC, often including the dlPFC (Hare et al. (2009); Hackel et al. (2020)). Therefore, the predictions of our theory could, in principle, also be tested in a large variety of decision-making problems involving multi-attribute goods.

4 Consumer behavior

In this section, we discuss how the properties of the trial-by-trial choices emphasized in section 3 map into general patterns of consumption that can be observed in empirical studies. We take the perspective of a researcher who infers the consumption tendencies from the decisions of an individual observed at different dates.

4.1 Preliminaries

We use our physiological model to generate day-to-day choices but we concentrate on what the researcher can observe. Choices at different dates are likely to be subject to different cognitive costs. For example, some days it requires less attention to modulate the signal simply because it is easier to focus or because fewer distractors are present. However, the researcher does not observe these differences. To capture variability, we assume that c is stochastic, so that $\eta \equiv \sqrt{2c}/\alpha$ is also stochastic.[26] Denote by $G(\eta)$ its cumulative distribution function. Assume $G(\eta)$ is strictly increasing and with a sufficiently wide support ($[0, \eta^*]$, with $\eta^* \geq 1$), to ensure that both modulation and no-modulation occur in equilibrium with positive probability.[27] The realization of c is known to both systems before the modulation and consumption decisions so that, in each consumption event, the optimal strategy presented in Theorem 1 applies. For simplicity, we take the environment as given and we report results that operate under all possible environments.

4.2 Imperfect self-regulation

We first look at this problem in the context of dietary choices similar to [HCR]. From the perspective of the researcher who does not observe c, the relevant variable is the *expected* taste cutoff above which the individual consumes given a health realization. We can compute this function, denoted $\bar{\theta}(h)$, by integrating $\bar{\theta}(h, c)$ from Figure 2 over all possible values of c. The following result describes the consumption patterns.

Corollary 4 - Self-indulgent and self-restraint behavior. *On average, the individual under-consumes low tempting goods and over-consumes high-tempting goods. Formally, there exist h_1 and h_2 with $0 < h_1 \leq h_2 < 1$ such that $\bar{\theta}(h) > \alpha h$ for all $h < h_1$ and*

[26] In spirit, this exercise is the same as specifying error terms in an empirical analysis to capture errors in choices or perceptions.

[27] Recall that, by definition, modulation is always optimal when $\eta = 0$ and by Theorem 1 modulation is never optimal when $\eta \geq 1$. Notice that all that matters for our theory is the stochasticity of η. Therefore, identical results are obtained when c is fixed and α varies.

$\bar{\theta}(h) < \alpha h$ *for all* $h > h_2$.[28]

In the absence of a cost of modulation, the individual consumes the good optimally, that is, when $\theta > \alpha h$. With a positive and stochastic cost of modulation, the individual exhibits on average under-consumption of low-tempting goods ($\bar{\theta}(h) > \alpha h$ for sufficiently small h) and over-consumption of high-tempting goods ($\bar{\theta}(h) < \alpha h$ for sufficiently high h).

The reason is simple and holds for any distribution $G(\cdot)$. When $\mathcal{M}$ modulates the goal value, the individual consumes optimally. When $\mathcal{M}$ does not modulate the goal value, $\mathcal{C}$ infers that health is in a certain set, and assumes the expectation within that set. Such expectation is necessarily above the true realization when h is sufficiently low and below the true realization when h is sufficiently high. Also, other things being equal, modulation is less frequent the higher the cost, so that the expected taste cutoff function $\bar{\theta}(h)$ is strictly increasing in h. Corollary 4 implies that a subject will succumb more often than optimal to beverages with high alcoholic content but that same individual will consume less often than optimal beverages with low alcoholic content. Figure 3 illustrates the result.[29]

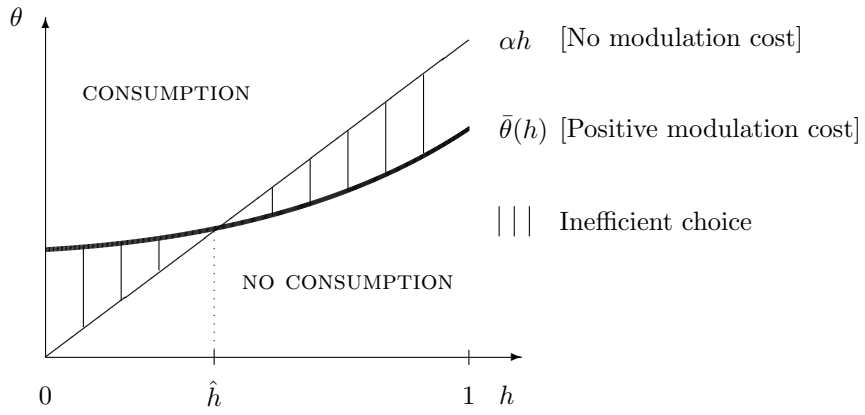

Figure 3. Expected consumption as a function of the health realization

Models of imperfect self-regulation emphasize the idea that subjects with self-control problems have a tendency to succumb into temptations and over-consume. Our theory has a novel implication. It suggests that the cost of modulating the goal value will result in either over-consumption or under-consumption, depending on the taste and health realizations. In other words, these opposite deviations are in fact two sides of the same coin, so

[28] Under some sufficient conditions, $h_1 = h_2$. This is the case, for instance, if $\eta \sim U[0,1]$ and $f_e(h)$ is either monotonic or symmetric around its inflection point (see appendix C for details).

[29] It assumes $\eta \sim U[0,1]$ and $f_e'(h) \leq 0$, so that $h_1 = h_2 = \hat{h}$, $d^2\bar{\theta}/dh^2 > 0$ and $\bar{\theta}(h) \gtrless \alpha h$ for all $h \lessgtr \hat{h}$.

that the very same mechanism that induces self-indulgence can also result in self-restraint behavior. Notice also that $d\bar{\theta}/dh < \alpha$ is *not* generally true, that is, actual consumption in our model is not always less sensitive to health than optimal (in particular, whenever $h_1 \neq h_2$, consumption will always be over-reactive to health in some interval). We therefore get the paradoxical result that costly attention can generate *excessive sensitivity* to the unattended attribute.[30] As a technical remark, notice that $\bar{\theta}(h, c)$ in Figure 2 is a step function, which is an artifact of our dichotomous model with binary consumption and binary choice of information transmission. Introducing a variable cost smoothes out that function. The results in this section would be similar if we modeled instead stochastic changes in preferences α, variable consumption quantities z, or variable information transmission technologies (e.g., signals with different precisions).

Remember that overconsumption arises when food items are tasty but very unhealthy. This case corresponds in our model to the failure of willpower: the dlPFC does not send information to the goal value system, and the subject ends up succumbing to the tempting alternative. This is in line with recent studies which show that exertion of willpower is associated with the activation of dlPFC (Figner et al. (2010); Hare et al. (2009); Crockett et al. (2013); He et al. (2019)).

4.3 Dynamic inconsistencies

A similar analysis can be made in the context of temporal delays, as described in section 3.5. We can determine the expected taste cutoff above which the individual consumes as a function of the delay between the pleasurable consumption and its health consequences, both with zero and positive cost of modulation. We obtain the following prediction.

Corollary 5 - Modulation of near and distant events. *Costly signal modulation generates over-consumption of goods with short term negative effects and under-consumption of goods with long term negative effects.*

From (5), it is immediate that an individual with no cost of modulation learns the realization of t and consumes whenever $\theta > \delta^t$. By construction, this corresponds to the traditional exponential discounting model. Denote by $\bar{\theta}(t)$ the analogue of $\bar{\theta}(h)$ when the high-order attribute is the date at which the health component is suffered. Using a straightforward change in variable, we can graphically depict in Figure 4 the analogue of Figure 3 in the (θ, t) space.

[30] A similar result is found in Chetty et al. (2007, section 7) in a different context: rational inattention to sale taxes due to cognitive costs can generate under- or over-sensitivity to taxes.

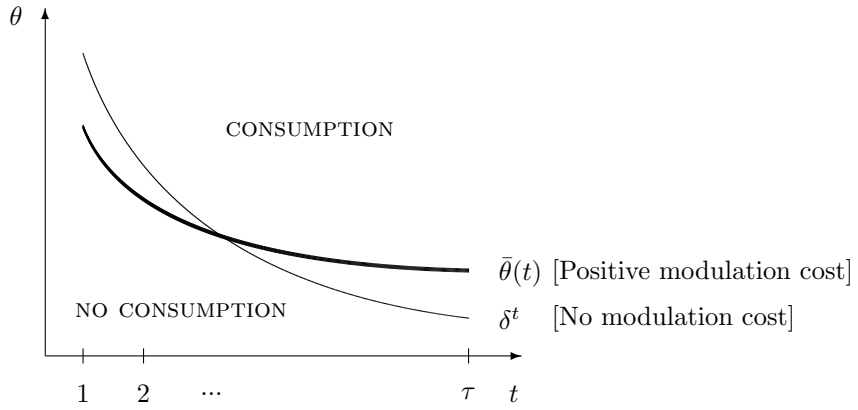

Figure 4. Expected consumption as a function of the *delay* realization

Relative to the case where modulation is costless, an individual will, on average, exhibit increased consumption when the negative health effect is at dates close to 1 and decreased consumption when the negative health effect is at dates close to τ. This means that the implicit discount function exhibits decreasing impatience, the most basic property of hyperbolic discounting. Indeed, compared to the exponential case, consumption is more frequent when the externality is in the immediate future and less frequent when it is in the distant one. This behavioral anomaly has long been noted in psychology and economics (Ainslie (1975); Prelec (1989); Laibson (1997)) and endogenously emerges in our model when the cost of modulation is positive.[31]

Naturally, the same logic also applies to decisions with a negative low-order immediate return and a positive high-order future benefit enjoyed at an uncertain date. Relative to the standard exponential discounting case, costly signal modulation generates under-consumption of aversive goods with short-term positive effects and over-consumption of aversive goods with long-term positive effects. In the case of efforts to complete tasks with delayed benefits, procrastination occurs for rewards in the near future. This prediction is reminiscent of the hyperbolic discounting literature (O'Donoghue and Rabin (1999)), except that we do not impose exogenous properties on the discounting function.

[31] Notice that, in our model, decreasing impatience always holds when comparing extreme dates (t close to 1 vs. t close to τ). Depending on $G(\eta)$ and $F_e(h)$, it may or may not apply to intermediate time intervals.

4.4 Similarities and differences with existing predictions

Our findings exhibit similarities and differences with the behavioral predictions in the literature. Here, we compare two that are particularly relevant.

Self-control

Classical theories based on hyperbolic discounting (e.g., Carrillo and Mariotti (2000)) predict that strategic manipulation of information can be used as a commitment device to achieve self-control in the context of temporal delays between benefits and costs. Still a few differences should be noted. First, because our theory is based on choices between alternatives that feature low-order and high-order attributes, it provides a unified framework to study decision problems that include but extend beyond the short-term vs. long term trade-off. In other words, temporal delays are not a necessary ingredient in our framework. Second, since future consequences are not systematically underweighted, there is not an a priori maladaptive consumption pattern that needs to be corrected. Third, our model places self-control within a broad class of regulatory strategies (top-down regulation). Therefore, we can also encompass situations in which (i) self-control behavior is the rule-based decision instead of the cognitive overrule and (ii) top-down regulation is used to trigger healthy consumption rather than prevent unhealthy consumption.

Our result on self-indulgent and self-restraint behavior also bears some resemblance to Bénabou and Tirole (2004)'s model of compulsive behavior. However, not just the assumptions (as discussed in section 2.5), but also the mechanisms are very different. In their setting, self-control behavior depends on how likely the individual anticipates that he will be subject to an inter-temporal conflict, which depends on what he will remember at the time of decision. Rules are the result of these beliefs and are used to prevent the individual from falling prey of his time inconsistency. In our model, the individual is not misguided by inconsistencies or bounded memory. Instead, under- and over-consumption result exclusively from an imperfectly informed valuation process.

More generally, and as emphasized earlier, one strength of the temporal version of our theory is its ability to endogenously derive –rather than assume– a behavior consistent with decreasing impatience. Relatedly, it has been reported that discounting is domain-dependent (Frederick et al. (2002)), which remains a puzzle in the behavioral economics literature. An obvious but unsatisfactory way to capture this observation is to assume a domain-specific discount function. Our theory shows that the characteristics of the decision problem (whether the options have low- or high-order attributes) has a direct impact on the subject's regulation strategy and therefore on his evaluation of inter-temporal trade-offs. This mechanism naturally results in domain-dependencies.

Context effects

Traditional models of self-control have ignored the fact that the environment can affect the choices of individuals, an issue long recognized in the psychopharmacological literature, notably for addictive substances (Zinberg (1984); Falk and Feingold (1987); Caprioli et al. (2007)). Realizing this shortcoming, the economics literature has proposed more comprehensive models where the environment provides *cues* that either change preferences (Laibson, 2001) or trigger mistakes (Bernheim and Rangel, 2004). In both cases, the effect of cues is an exogenous feature of the model. Our study demonstrates that, by modeling the physiological mechanism, it is possible to provide a plausible explanation of why context endogenously affects consumption decisions. As we have shown, they are logical consequences of an efficient information processing mechanism. This feature is reminiscent of recent applications of rational inattention theory, as reviewed earlier, where individuals decide whether to spend costly resources into incorporating information as a function of its expected benefits. The advantage of our model is to provide a brain-based microfoundation that rationalizes these mechanisms.

We have also noted that the context can be an explicit cue (an objective distribution as in our theory) or a more informal one. Informal cues are left to the interpretation of individuals. This has been first addressed in the self-control literature with time delays by Ainslie and Monterosso (2003) and formalized by Bodner and Prelec (2003) and Mijovic-Prelec and Prelec (2010). The research assumes that the individual achieves self-control by 'bundling decisions' and mentally comparing never succumbing to always succumbing instead of comparing succumbing today to not succumbing today.

Overall, our theory encompasses many classic behavioral predictions derived previously. Because our model is based on physiological evidence, it has the merit to rationalize certain assumptions made in earlier studies (e.g., the effect of cues) and to revisit speculative modeling features (e.g., the role of information). It also provides a new framework to think about self-control in particular (e.g., constrained physiology rather than decreasing impatience) and decision-making in general (e.g., cooperative top-down regulation rather than multiple selves). It places also the earlier predictions in a broader framework.

5 Behavioral disorders

So far we have described the physiological mechanism that underly "normal" behavior. This mechanism features a specific interplay between systems $\mathcal{C}$ and $\mathcal{M}$. In some cases, however, these systems exhibit dysfunctions. When this happens, patterns of activation are different from what we would expect and behavior becomes anomalous. In this section,

we study dysfunctions by modifying the basic model. We derive new behavioral predictions resulting from changes in modulation properties.

5.1 Eating disorders

Neuroimaging studies (Brooks et al., 2012; Lowe et al, 2019) show that eating disorders are tied to failures of self-regulation and to an imbalance between mesolimbic regions (which include the vmPFC) and prefrontal regions involved in cognitive evaluations (which include the dlPFC). Patients with Anorexia Nervosa (AN) show low stimulation in mesolimbic regions and high stimulation in prefrontal regions and their behavior is associated with a strict refusal to eat. By contrast, patients with Bulimia Nervosa (BN) show the reverse pattern and they exhibit binge eating (van Kuyck et al. (2009), Kaye et al. (2009), Kaye et al. (2011), Brooks et al. (2011), Foerde et al. (2015)). Consistent results are obtained for the case of obesity: mesolimbic regions are found to be hypersensitive in response to food stimuli while regions of the prefrontal cortex are found to have activation deficits. Interestingly, dlPFC shows reduced activation during self-control as a function of body mass index (Han et al, 2018). Current stimulation techniques have also shown a causal effect of PFC on obesity (Hall et al., 2017; Lowe et al, 2017; Mostafavi, 2020).[32]

In the context of our model, this literature suggests that the true values of taste and health are distorted and the brain makes decisions under an *incorrect perception* of attributes. In this section, we offer a model of suboptimal self-regulation that captures this feature. Formally, h is drawn from environment e with distribution $f_e(h)$ in $[0,1]$ but it is perceived to be drawn from environment p with distribution $f_p(h)$ in $[0,1]$. for simplicity, we consider two polar cases for this (incorrect) perceived environment: extremely healthy $p = \underline{p}$ such that $E_{\underline{p}}[h] \to 0$ and extremely unhealthy $p = \overline{p}$ such that $E_{\overline{p}}[h] \to 1$. Importantly, the true realization of h is correctly assessed by system $\mathcal{M}$ and, if transmitted, also by system $\mathcal{C}$.[33] As in section 4.2, we assume that attention costs vary across trials so that $\eta \sim G(\eta)$. We then study how the choice of modulation and the expected consumption given the true realization of health (i.e., the cutoff $\bar{\theta}(h)$) depend on the perceived environment p. We have the following result.

Corollary 6 - Dysfunctions. *When $p = \underline{p}$, there is over-consumption for all $h \in (0,1]$. When $p = \overline{p}$, there is under-consumption for all $h \in [0,1)$.*

[32] Studies using transcranial magnetic stimulation also showed that symptoms were reduced in AN patients (feeling fat or anxious) and BN patients (suffering binge eating disorders) in conjunction with stimulation of dlPFC (Van den Eynde et al. (2010); Van den Eynde et al. (2013); Dalton et al. (2020)).

[33] One natural question is whether and, if so, how fast individuals realize that their perceived distribution about health is incorrect. We do not address this issue here.

According to this result, an individual with unrealistically positive prior beliefs about the health rating will over-consume, even when the health realization is not as good as expected. By contrast, an individual with unrealistically negative prior beliefs will under-consume even when the health realization is not as bad as expected. Formally and as graphically depicted in Figure 5, when $p = \underline{p}$ then $\bar{\theta}(h; \underline{p}) < \alpha h$ for all $h \in (0, 1]$ and when $p = \overline{p}$ then $\bar{\theta}(h; \overline{p}) > \alpha h$ for all $h \in [0, 1)$.

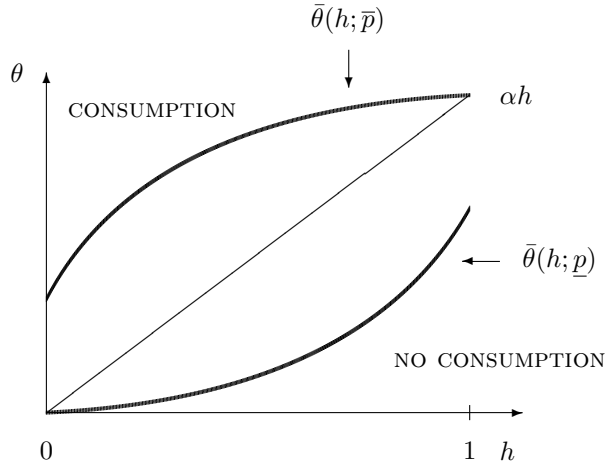

Figure 5. Expected consumption with incorrect perceived environments $\overline{p}$ and $\underline{p}$

It is important to realize that over- and under-consumption are not trivially driven by the fact that goods are always incorrectly seen as fully healthy and fully unhealthy, respectively. As we can see from Figure 5, consumption is decreasing in h in both cases ($\bar{\theta}$ increasing in h). An interior realization of h is an extremely low perceived probability event under either $\underline{p}$ or $\overline{p}$ but it occurs generically given the true environment e. When it happens, the individual with perception $\underline{p}$ consumes more often than optimal–though not always–whereas the individual with perception $\overline{p}$ consumes less often than optimal–though still sometimes. Indeed, depending on the attention cost c, system $\mathcal{M}$ will sometimes modulate the signal and sometimes not. Modulation triggers optimal consumption by system $\mathcal{C}$. No modulation triggers highly optimistic beliefs under $\underline{p}$, inexorably followed by consumption, and highly pessimistic beliefs under $\overline{p}$, inexorably followed by abstinence. The final expected consumption is an appropriately weighted combination of the choices made under modulation (optimal behavior) and no modulation (extreme behavior). Overall, the result shows that prior beliefs are key in shaping modulation and consumption. Incorrect perceptions can have an impact on choice not because realizations are incorrectly interpreted, but because information is not transmitted when it should be.

The findings are consistent with the existing evidence on AN and BN. These two conditions are characterized by extreme patterns of behavior in which subjects systematically under- and over-consume. In our model, these behaviors emerge when goods are believed to be extremely unhealthy ($\overline{p}$) and unusually healthy ($\underline{p}$), respectively. More specifically, inefficiencies result from a dysfunction of the rule-based mechanism because contextual information is represented through a biased assessment of the environment. AN patients act according to a rule-based mechanism that represents an excessively unhealthy environment, thereby triggering under-consumption. Conversely, BN patients act according to a rule-based mechanism that represents an unusually healthy environment, thereby triggering over-consumption. Our model also predicts that the behavior of AN and BN patients would not change if information about the true health consequences of a good is provided. Indeed, this information will not be processed as long as decisions are based on incorrect assumptions about the environment. Experimentally, we should observe no reduction in over- or under-consumption following the disclosure of health information.[34]

The theory could be tested by replicating [HCR] across populations with different eating disorders. We could measure differences across distributions of health ratings to elicit biased perceptions. Trivially, we would expect dramatic differences in behavior between AN and BN patients. More interestingly, absence of dlPFC activity (no modulation) should be associated with consumption in BN patients and with abstinence in AN patients.

5.2 Substance addiction

Addiction to substances has long been investigated in relation to self-control problems.[35] From a physiological viewpoint, studies of consumption patterns in addicts point to an interplay between vmPFC and dlPFC. They show that addiction disrupts functions in both areas that are fundamental for regulation and decision-making (Koob and Volkow (2010); Goldstein and Volkow (2011)).

To capture this feature, we propose the simplest possible extension of our basic model. The individual chooses consumption during two periods, 1 and 2. In period 1, the utility is given by (1). In period 2, the utility depends on both present and past consumption decisions. More precisely, denote by $u_{zz'}$ the utility in period 2 of consumption choice z' ($\in \{0,1\}$) given a consumption choice z ($\in \{0,1\}$) in period 1. To reflect the patterns

[34] Evidence also suggests that dysfunctions may be exacerbated through repeated consumption of unhealthy foods (Lowe et al., 2019). These dynamic effects would be interesting to model.

[35] Behavioral addictions, such as gambling and internet addictions, share many similarities. We will discuss them in section 5.3.

of activity in vmPFC, we assume that:

$$\begin{array}{ll} u_{01} = \theta - \alpha h & \\ u_{00} = 0 & \end{array} \quad \text{and} \quad \begin{array}{ll} u_{11} = p\,\theta - \alpha h & \\ u_{10} = -q\,\theta & \end{array} \tag{7}$$

where $p < 1$ and $p + q > 1$. In words, if the individual abstains in period 1 ($z = 0$), his utility in period 2 remains unchanged. If the individual consumes in period 1 ($z = 1$), then both the utility of consumption and the utility of abstinence are reduced ($u_{11} < u_{01}$ and $u_{10} < u_{00}$). This accounts for the physiological evidence on "habituation" and "craving", respectively. We further assume that the marginal utility of consumption at date 2 is higher after consumption in period 1 than after abstinence ($u_{11} - u_{10} > u_{01} - u_{00}$). While we do not have direct evidence of this assumption, we conjecture that it is represented in the brain since it is necessary to derive an addictive behavior. As can be seen immediately, the model exhibits the traditional basic features of addiction and habit formation (Becker and Murphy (1988)).[36]

We focus on the decision in period 2. If the subject abstains in period 1, the choice in period 2 is the same as in Theorem 1. By construction, if information about health is transmitted from system $\mathcal{M}$ to system $\mathcal{C}$, consumption in period 2 is more likely to occur after consumption in period 1 than after abstinence, as in traditional models of habit formation (formally, $\theta > \alpha h/(p+q)$ vs. $\theta > \alpha h$). A more interesting question is to determine whether the decision to modulate the goal value is affected by past consumption. To answer that question, we determine the value of modulation and no modulation after period 1 consumption:

$$V_1^+(h) = \int_0^{\frac{\alpha h}{p+q}} -q\,\theta d\theta + \int_{\frac{\alpha h}{p+q}}^1 (p\,\theta - \alpha h) d\theta - c$$

and

$$V_1^-(h) = \int_0^{\frac{\alpha E_e[h|h\in H_e]}{p+q}} -q\,\theta d\theta + \int_{\frac{\alpha E_e[h|h\in H_e]}{p+q}}^1 (p\,\theta - \alpha h) d\theta$$

Again, modulation dominates no modulation when $V_1^+(h) > V_1^-(h)$, which translates into:

$$\begin{aligned} \Delta_1(h, E_e[h|h \in H_e]) &\equiv \int_{\frac{\alpha E_e[h|h\in H_e]}{p+q}}^{\frac{\alpha h}{p+q}} \Big(\alpha h - (p+q)\theta\Big) d\theta > c \\ &\Leftrightarrow \Big|h - E_e[h|h \in H_e]\Big| > \eta_1(c, \alpha, p, q) \equiv \frac{\sqrt{2c(p+q)}}{\alpha} \end{aligned} \tag{8}$$

[36] A natural extension would be to build a dynamic version, where future decisions play the role of high-order information represented at a cost in dlPFC. It is unclear however whether a distinction exists between the representation of a future benefit (the direct utility from consuming an addictive good in the future) and a future cost (its health consequence), that is, whether those may be independently modulated.

which naturally leads to the following result.

Corollary 7 - Under-modulation of addictive goods. *For addictive goods, signal modulation is less prevalent given past consumption than given past abstinence.*

The result is obtained by direct inspection of equations (4) and (8). Because the marginal effect of taste on current consumption increases with past consumption ($p + q > 1$), the incentives to modulate the signal decrease with past consumption. Indeed, increasing the marginal value of taste makes consumption more desirable which, in turn, makes it less valuable to spend the cost of transmitting the exact health realization (since it has a lower impact on decision). Overall, the set of health realizations for which there is no modulation expands. Naturally, the effect of over-consumption in period 2 is more severe the stronger the addictive properties of the good.

Note that as the individual is exposed to an addictive good, his decision-making may switch from a regime in which there is modulation in some interval to a regime in which modulation is never optimal. Hence, ignorance of health consequences is *endogenously more frequent* for addicted than for non-addicted individuals. The result is not based on an irrational self-delusion motive but, instead, on the optimal decision to avoid a costly signal transmission that is unlikely to affect consumption.

Studies have reported hypo-activity of the dlPFC in drug-addicts, and sometimes interpreted it as an impairment due to drug exposure (Goldstein and Volkov, 2011). Our model suggests that hypo-activity may be simply the natural consequence of the interplay between $\mathcal{M}$ and $\mathcal{C}$. It also indicates that helping the subjects' attention (i.e., decreasing c) should increase communication between systems $\mathcal{M}$ and $\mathcal{C}$. In that respect, recent evidence has shown that a weak direct current applied non-invasively over the dlPFC produces both behavioral and brain functional changes in vmPFC in drug addicts (Nakamura-Palacios et al. (2016)). Such clinical intervention may constitute an efficient way of combatting addiction.

5.3 Impulsive compulsive spectrum disorders

Dysfunctions of the reward system and cognitive processes supported by the dlPFC have been observed in a variety of behavioral disorders. These dysfunctions are often grouped under the umbrella of 'Impulsive Compulsive Spectrum Disorders' and they are characterized by maladaptive impulses and/or an impairment of executive control. Behavior usually features decisions for which only some attributes are taken into consideration, even though other aspects should also be incorporated.

5.3.1 Neuropsychiatric conditions

Patients with Attention Deficit and Hyperactivity Disorder (ADHD) tend to not represent the consequences of their decisions and act impulsively, responding almost exclusively to the features of imminent rewards or costs. This behavior has been found to be associated with a dysfunction of the dlPFC (Petrovic (2016)) and there is evidence that its stimulation improves behavior (Siniatchkin (2017)). Such disorder may be simply due to a disproportionate cost of modulation that reduces the range of decisions regulated by dlPFC. Said differently, behavior may be modeled as in the main analysis of section 2.2, except that it relies on a very high cost.

In the case of Borderline Personality Disorder (BPD), impulsivity affects the behavior of patients, this time due to an imperfect representation of self-image and emotions. BPD has been associated with different patterns of activity in the vmPFC and dlPFC along with other structures such as the amygdala (Berdahl (2010)). Similar phenomena are observed in obsessive-compulsive disorder (OCD) and schizophrenia (Cavallaro et al. (2003)).

There is converging evidence that disorders featuring a dysfunction of emotional regulation share common neural correlates (Sebastian et al. (2014)), which overlap with the systems recruited to evaluate the attributes of a decision. There is also a strong correlation between these dysfunctions: ADHD patients are more likely to succumb to a form of addiction or to become bulimic compared to control populations (Urcelay and Dalley (2011), Ziobrowski et al. (2018)). In the same vein, a large fraction of the population with schizophrenia abuse drugs and/or alcohol (Batel (2000)). These correlations suggest that behavioral anomalies in ADHD, BPD, OCD and schizophrenia may stem from a single (dysfunctional) mechanism.

In light of our theory, we could design appropriate experimental paradigms in which disorders naturally manifest (e.g., the inability to complete tasks in the case of ADHD) and study whether, compared with control participants, the behavior of patients (i) is compatible with biased motives (as in the case of eating disorders) or (ii) can be captured by a difference in attentional costs (as predicted by Corollary 3).[37]

5.3.2 Behavioral addictions

Growing evidence suggests that behavioral addictions resemble substance addictions in terms of both the characteristics of behavior (craving, tolerance) and their neurobiological underpinnings. For instance, gambling addiction features disruptions of vmPFC and

[37] For an interesting theoretical model of task-switching and attention switching in ADHD patients, see Landry (2021).

dlPFC (Reuter et al. (2005); Moccia et al., 2017) and can be helped by transcranial modulation of the dlPFC (Soyota et al., 2019). Similar findings have been made for internet addiction (Brand et al., 2014), video game addiction (Palaus et al., 2017) and internet gaming disorders (He et al., 2020). Compulsive shopping has also been shown to share similarities with substance use disorders (Probst and van Eimeren, 2013; Lejoyeux and Weinstein, 2010; Raab, 2011).

It is important to note that the disorders collected under the umbrella "Impulsive Compulsive Spectrum Disorders" feature fundamentally different dysfunctions. Neuropsychiatric conditions are not characterized by craving, tolerance and more generally the building up of a habit. They are better captured by a distorted perception, as in the variant described in section 5.1. By contrast, behavioral addictions are closer to substance addiction, as in the variant described in section 5.2.

6 Concluding remarks

We have built a novel theory of value-based decision-making where the options are characterized by a conflict between their low- and high-order attributes. A typical example is the decision to consume a tempting unhealthy good, where the taste is a low-order easy to represent characteristic and the health consequence is a high-order difficult to represent characteristic. Following the behavioral and neurophysiological evidence of dietary choices reported in [HCR], we have modeled the interplay between two critical systems. The goal value system has direct access to information about the low attribute and is in charge of integrating information about both attributes. The cognitive system collects information about the high-order attribute and facilitates its representation, through costly modulation, in the goal value system. Because modulation is costly, it does not always occur.

We have derived the properties of this interplay, their neural correlates and the resulting constrained optimal behavior. We have highlighted the importance of the environment in triggering value modulation and therefore endogenously affecting consumption decisions. In some environments, the mechanism involves modulation of unhealthy attributes to prevent their consumption, and it takes the form of self-control. In others, unhealthy food avoidance is implemented via context representations. It does not require modulation and is reminiscent of willpower. More generally, we have emphasized that self-indulgence and self-restraint are two sides of the same coin: they are both the result of the same costly value modulation mechanism. Our theory extends to other paradigms that feature the same key conflict between attributes. In the context of current (low order) rewards

and delayed (high order) unpleasant consequences, the optimal modulation mechanism is also affected by context effects and we observe inefficiencies consistent with decreasing impatience.

Because our model is not designed to fit behavior and rather describes its causes (modularity of brain systems with specific functions and constraints), it is capable of providing a unified account of observed choices in the context of self-control, time discounting, and impulsivity. Given the convergent neurophysiological evidence that behavioral anomalies are dysfunctions of the mechanism we describe, we have expanded the model to capture these dysfunctions and predict behavior in the context of substance addictions, eating disorders, behavioral addictions and, more generally, compulsive disorders. We have shown that, although behavior in those paradigms may seem impulsive, out-of-control or over-focused on some elements of the decision, it is the result of an internally consistent information processing. Only, the information technology is anomalous, distorted or excessively costly.

We have also drawn a parallel with the modeling strategies and the predictions of classical models in economics. Existing theories of decision-making are insightful but incomplete. Behavioral economics uses introspection, casual observation and empirical observation of choices to infer causes of behavior. This strategy has led to a multiplicity of competing stories. By contrast, our model is disciplined by the neurobiological evidence, which provides direct access to the causes.[38] This approach has two critical methodological advantages. First, modeling correctly the interplay of systems that contribute to a decision allows us to unveil the true mapping between causes of behavior and behavior itself, and to understand how this mapping responds to exogenous variations. Taking neurophysiological evidence into consideration shows that a single unified framework can account for behaviors usually generated under different incompatible axioms or different irreconcilable models. These behaviors are all consequences of the same mechanism applied to different environments. Second, understanding causal features is critical to make both out-of-sample predictions of behavior and policy recommendations. We have shown that behavior results from internally consistent mechanisms that are responsive to contextual information and cues. This suggests that corrective measures should focus on these tools. These advantages provide a clear argument regarding the value of neuroscience for economics (Gul and Pesendorfer (2008); Camerer (2008)).

Our approach is also complementary (not substitute) to computational neuroscience. Indeed, one could build a mechanical model where system $\mathcal{M}$ automatically sends health

[38]Some contributions using this neuroeconomic theory approach include Bernheim and Rangel (2004), Brocas and Carrillo (2008), Caplin and Dean (2008), Alonso et al. (2014) and, more recently, Landry (2021), Landry and Webb (2021) and Webb (2019).

"warnings" to system $\mathcal{C}$ for certain realizations of h. This seems a more plausible implementation of what is actually going on in the brain than the 'as if' optimization mechanism developed in the paper. The trouble with such approach is that it provides little guidance as to when the health information should be transmitted. As a first approximation, one could argue that warnings should be sent for health realizations above a certain threshold. The added value of our optimization approach is to show that optimal information transmission generically requires both a threshold and a cap, and that these values depend on the cost of modulation, the relative importance of health, and the distribution from which the health parameter is drawn. Therefore, a computational model should incorporate those features, even if it does so in a mechanical model.

There are also numerous alleys for future research both to better understand the physiological underpinning of behavior and to build more accurate models and inform interventions.

First, the theory outlined here is closely related to the evidence in [HCR], and focuses on the modulatory mechanisms involved in a simple paradigm. We have intentionally left aside many important issues to highlight that mechanism, most notably dynamic considerations in the case of addiction or eating disorders. Recent literature suggests that repeated exposure to unhealthy food affects dysfunctions of prefrontal regions, indicating that these dysfunctions are reinforced through time (Lowe et al., 2019). Also, decision-makers may gradually update information about the environment. Such learning can interact with the ability to evaluate costs and benefits of consumption and to exercise modulation. For example, individuals exposed to an unhealthy environment at a young age may develop different habits when older.

Second, it would be also interesting to investigate the paradigm in the less well-documented case of children and adolescents. Studies have shown that dlPFC is one of the last region to mature, causing a gradual development of attention throughout childhood and adolescence (Gathercole et al. (2004), Gogtay et al. (2004)). It is unclear how self-control can occur before dlPFC is mature.[39] More generally, there is converging evidence that child and teen behavioral disorders (such as ADHD and proclivity to drug addiction) are linked to age-related deficits in regulatory mechanisms in the brain (Sonuga-Barke et al., 2012; Bava and Tapert, 2010). There is also evidence that self-control problems together with dysfunctions of the dlPFC are associated with delinquency (Meldrum et al.,

[39]Brain plasticity is still imperfectly understood, and only a few studies have been conducted with children using food choice paradigms. Van Meer et al. (2017) have reported different dlPFC signals in children compared to adults while Lim et al. (2016) have shown that dlPFC encodes the projected maternal food choice. Bruce et al. (2016) have reported that health ratings did not predict the choices of children who were asked to watch food commercials beforehand, while vmPFC was significantly more activated.

2018). It is plausible that disruptions of the mechanism outlined in the paper, such as an over-reaction of system $\mathcal{C}$ (Nock et al. (2017)), are causing anomalous behavior during adolescence that perdure throughout life.

Third, neuroscience studies have shown that cues have an effect on self-control (Hare et al. (2011), Hutcherson et al. (2012)). Our theory offers an opportunity to organize our understanding of the mechanisms at play. Cue manipulations consist in asking subjects to alter their thinking process regarding their decisions (focus on one attribute, vary craving). In principle, these manipulations may affect three components of our model: (i) the attention cost c (it is less costly to evaluate the health component when directed to focus on it), (ii) the health concern α (the preference for a healthy lifestyle may be reinforced when asked to reduce craving) and (iii) the environment e (the perception of the context may be affected by the request to focus on one's own emotions). Variations in these three components should be reflected in changes in behavior. They should also be reflected in changes in physiological responses. Indeed, (i) and (ii) should affect the range of modulation, resulting in more dlPFC activity while (iii) should affect the level of activation in vmPFC in the absence of modulation. Testing these specific contrasts in existing studies should help identify which mechanism is triggered by each manipulation. It may also be useful to design a cue manipulation that targets one specific mechanism explicitly. A concern with existing studies is that subjects do not receive guidance regarding how to achieve their goals. The specific mechanisms employed are unclear and may vary across subjects. As discussed earlier, attentional costs may be manipulated with distractors, the effect of health concerns may be tested via a between subject design that focuses on differences in lifestyle or health status, and variations in the distribution of health ratings can be used to manipulate the environment. Given the relevance of cues for self-control behavior, it would be useful to learn their modus operandi.

Last, heterogeneity in behavior is another important aspect to investigate further. Studies have shown that differences in brain anatomy are responsible for some aspects of cognitive functions and decision-making (Cachia et al. 2018; Pagliaccio 2020), including in issues related to eating disorders (Schmidt et al., 2018). Our model relies on several parameters that reflect potential heterogeneity. It would be interesting to determine the relationship between these parameters and the differences in brain volume. For instance, anatomical differences may be associated with differences in cognitive control abilities resulting in differences in attentional costs. Given the existing evidence that eating disorders is associated with cognitive flexibility (Perpina et al., 2017), understanding heterogeneity may help us link an even broader set of observations to the same underlying mechanisms.

Appendix A

Consider a continuous quantity model where the subject's utility of consuming q units is:

$$u_q = \theta v(q) - \alpha h q$$

where the benefit of consumption is increasing and concave in quantity ($v' > 0$ and $v'' < 0$).

Denote by $q^+(\theta, h)$ and $q^-(\theta)$ the optimal consumption recommendation by system $\mathcal{C}$ under modulation $(+)$ and no modulation $(-)$. Following the same steps as in the binary case, these consumption levels are given by:

$$q^+(\theta, h) = \arg\max_q \ \theta v(q) - \alpha h q \quad \text{and} \quad q^-(\theta) = \arg\max_q \ \theta v(q) - \alpha \, E_e[h \,|\, h \in H_e] \, q \quad (9)$$

Therefore, for system $\mathcal{M}$ the value of modulating and not modulating the signal given a health rating h are:

$$V_q^+(h) = \int_\theta \Big(\theta \, v(q^+(\theta,h)) - \alpha h q^+(\theta,h)\Big) dX(\theta) - c \quad \text{and} \quad V_q^-(h) = \int_\theta \Big(\theta \, v(q^-(\theta)) - \alpha h q^-(\theta)\Big) dX(\theta)$$

Optimal modulation, $V^+(h) > V^-(h)$, can be rewritten as:

$$\Delta_q(h, E_e[h \,|\, h \in H_e]) \equiv \int_\theta \Big[\Big(\theta \, v(q^+(\theta,h)) - \alpha h q^+(\theta,h)\Big) - \Big(\theta \, v(q^-(\theta)) - \alpha h q^-(\theta)\Big)\Big] dX(\theta) \ > \ c$$

Using the envelope theorem, we get:

$$\frac{\partial \Delta_q}{\partial h} = \alpha \int_\theta \Big(q^-(\theta) - q^+(\theta,h)\Big) dX(\theta) \quad \text{and} \quad \frac{\partial^2 \Delta_q}{\partial h^2} = -\alpha \int_\theta \frac{dq^+(\theta,h)}{dh} \, dX(\theta) \qquad (10)$$

Differentiating the first-order condition in (9) we get:

$$\theta \, v''(q^+) \frac{dq^+}{dh} = \alpha \ \Leftrightarrow \ \frac{dq^+}{dh} < 0 \ \text{ and } \ q^+(\theta, E_e[h \,|\, h \in H_e]) = q^-(\theta) \qquad (11)$$

Combining (10) and (11), it is immediate to conclude that:

$$\frac{\partial \Delta_q}{\partial h} \gtreqless 0 \ \text{ iff } \ h \gtreqless E_e[h \,|\, h \in H_e] \ \text{ and } \ \frac{\partial^2 \Delta_q}{\partial h^2} > 0$$

implying, once again, that the set of values such that system $\mathcal{M}$ chooses no modulation is necessarily compact ($H_e = [\underline{h}_e, \overline{h}_e]$).

Appendix B

Proof of Theorem 1

Step 1. Δ is convex with a minimum at $E_e[h \mid h \in H_e]$. This implies that H_e is necessarily a compact set. As a result, there are only four possible types of equilibria depending on whether no modulation is exerted when h is at the bottom (called **B** and formalized as $H_e = [0, h^{**}]$), at the middle (**M**, with $H_e = [h^o, h^{oo}]$), at the top (**T**, with $H_e = [h^*, 1]$) or always (**A**, with $H_e = [0, 1]$).[40] Naturally $h^*, h^{**}, h^o, h^{oo} \in (0, 1)$[4]. We now characterize those sets and provide conditions for existence.

Bottom (**B**): $H_e = [0, h^{**}]$ where h^{**} is the value such that $\Delta(h^{**}, E_e[h \mid h \leq h^{**}]) = c$. Using (4), we get:

$$h^{**} - E_e[h \mid h \leq h^{**}] = \eta \tag{12}$$

where $\eta(c, \alpha) = \sqrt{2c}/\alpha$. It is well-known that if $f_e(h)$ is log-concave then $x - E_e[h \mid h \leq x]$ is increasing in x (see e.g. Bagnoli and Bergstrom (2005)), so h^{**} is unique and $dh^{**}/d\eta > 0$. The conditions for this equilibrium to exist are $\Delta(0, E_e[h \mid h \leq h^{**}]) < c$ and $\Delta(1, E_e[h \mid h \leq h^{**}]) > c$, which can be rewritten as:

$$E_e[h \mid h \leq h^{**}] < \eta \tag{B1}$$

and

$$E_e[h \mid h \leq h^{**}] < 1 - \eta \tag{B2}$$

Middle (**M**): $H_e = [h^o, h^{oo}]$ where h^o and h^{oo} are the values such that $\Delta(h^o, E_e[h \mid h^o \leq h \leq h^{oo}]) = c$ and $\Delta(h^{oo}, E_e[h \mid h^o \leq h \leq h^{oo}]) = c$ respectively. Using (4), we get:

$$E_e[h \mid h^o \leq h \leq h^{oo}] - h^o = \eta \quad \text{and} \quad h^{oo} - E_e[h \mid h^o \leq h \leq h^{oo}] = \eta$$

Combining them, we get $h^{oo} = h^o + 2\eta$ and therefore:

$$E_e[h \mid h^o \leq h \leq h^o + 2\eta] = h^o + \eta \tag{13}$$

We will prove later that, in this equilibrium, h^o must be unique. The conditions for this type of equilibrium to exist are $\Delta(0, E_e[h \mid h^o \leq h \leq h^o + 2\eta]) > c$ and $\Delta(1, E_e[h \mid h^o \leq h \leq h^o + 2\eta]) > c$, which can be rewritten as:

$$E_e[h \mid h^o \leq h \leq h^o + 2\eta] > \eta \tag{M1}$$

[40] Never no modulation (that is, always modulation or $H_e = \emptyset$) cannot be an equilibrium if $c > 0$. Indeed, for any belief distribution that follows a deviation to no modulation, there exists a value h characterized by the expectation of such belief such that $\Delta = 0$ ($< c$), making the deviation to no modulation profitable.

and

$$E_e[h \mid h^o \leq h \leq h^o + 2\eta] < 1 - \eta \tag{M2}$$

Top (**T**): $H_e = [h^*, 1]$ where h^* is the value such that $\Delta(h^*, E_e[h \mid h \geq h^*]) = c$. Once again using (4), we have:

$$E_e[h \mid h \geq h^*] - h^* = \eta \tag{14}$$

If $f_e(h)$ is log-concave then $E_e[h \mid h \geq x] - x$ is decreasing in x (Bagnoli and Bergstrom, 2005), so h^* is unique and $dh^*/d\eta < 0$. The conditions for this type of equilibrium to exist are $\Delta(0, E_e[h \mid h \geq h^*]) > c$ and $\Delta(1, E_e[h \mid h \geq h^*]) < c$, which can be rewritten as:

$$E_e[h \mid h \geq h^*] > \eta \tag{T1}$$

and

$$E_e[h \mid h \geq h^*] > 1 - \eta \tag{T2}$$

Always (**A**): $H_e = [0, 1]$. The conditions for this equilibrium to exist are $\Delta(0, E_e[h]) < c$ and $\Delta(1, E_e[h]) < c$, which can be rewritten as:

$$E_e[h] < \eta \tag{A1}$$

and

$$E_e[h] > 1 - \eta \tag{A2}$$

Step 2. We now prove that an equilibrium exists and is unique for all parameter values of η and for all strictly log concave distributions $F_e(\cdot)$. From (h^{**}, h^o, h^*) as defined in (12)-(13)-(14) and the equilibrium conditions $(B1)$-$(B2)$-$(M1)$-$(M2)$-$(T1)$-$(T2)$-$(A1)$-$(A2)$, we have three cases.

Case 1: $\eta \geq 1$. Trivially, the unique equilibrium is **A** for all $F_e(h)$.

Case 2: $\eta \in [1/2, 1)$. In this case, $1 - \eta \leq \eta$ so **M** can never be an equilibrium.

• If $E_e[h] < 1 - \eta$ (that is, $1 - E_e[h] > \eta$), then a **B**-equilibrium exists and is unique. Indeed, $x - E_e[h \mid h \leq x]$ increasing in x implies there exists a unique $h^{**} < 1$ such that $h^{**} - E_e[h \mid h \leq h^{**}] = \eta$. Furthermore, $E_e[h] < 1 - \eta$ implies that a **A**-equilibrium cannot exist. Finally, $E_e[h] < 1 - \eta$ implies $E_e[h] < \eta$. $E_e[h \mid h \geq x] - x$ decreasing in x implies $E_e[h \mid h \geq x] - x < \eta$ for all x so a **T**-equilibrium does not exist either.

• If $E_e[h] > \eta$, then a **T**-equilibrium exists and is unique. Indeed, $E_e[h \mid h \geq x] - x$ decreasing in x implies there exists a unique $h^* > 0$ such that $E_e[h \mid h \geq h^*] - h^* = \eta$. Furthermore, $E_e[h] > \eta$ implies that a **A**-equilibrium cannot exist. Finally, $E_e[h] > \eta$ implies $1 - E_e[h] < \eta$. $x - E_e[h \mid h \leq x]$ increasing in x implies $x - E_e[h \mid h \leq x] < \eta$ for all x so a **B**-equilibrium does not exist either.

• If $E_e[h] \in [1-\eta, \eta]$, then a **A**-equilibrium exists. $E_e[h \,|\, h \geq x] - x$ decreasing in x implies $E_e[h \,|\, h \geq x] - x < \eta$ for all x so a **T**-equilibrium does not exist. $x - E_e[h \,|\, h \leq x]$ increasing in x implies $x - E_e[h \,|\, h \leq x] < \eta$ for all x so a **B**-equilibrium does not exist either.

To summarize, when $\eta \in [1/2, 1)$ the equilibrium is unique given $F_e(\cdot)$ and η:

$$\begin{array}{ll} \mathbf{B} & \text{if} \quad E_e[h] < 1-\eta \\ \mathbf{A} & \text{if} \quad E_e[h] \in [1-\eta, \eta] \\ \mathbf{T} & \text{if} \quad E_e[h] > \eta \end{array}$$

Case 3: $\eta \in (0, 1/2)$. In this case, $\eta < 1-\eta$ so **A** can never be an equilibrium.

In a **B**-equilibrium, $h^{**} - E_e[h \,|\, h < h^{**}] = \eta$ and $E_e[h \,|\, h < h^{**}] < \eta$. Therefore, $h^{**} < 2\eta$. Since $x - E_e[h \,|\, h \leq x]$ is increasing in x, then $2\eta - E_e[h \,|\, h < 2\eta] > \eta$ or:

$$E_e[h \,|\, h < 2\eta] < \eta \tag{1'}$$

In a **M**-equilibrium, $E_e[h \,|\, h^o \leq h \leq h^{oo}] - h^o = \eta$ and $h^{oo} - E_e[h \,|\, h^o \leq h \leq h^{oo}] = \eta$. Therefore, $h^{oo} = h^o + 2\eta$, and therefore:

$$E_e[h \,|\, h^o \leq h \leq h^o + 2\eta] = h^o + \eta \tag{2'}$$

In a **T**-equilibrium, $E_e[h \,|\, h > h^*] - h^* = \eta$ and $E_e[h \,|\, h > h^*] > 1-\eta$. Therefore, $h^* > 1 - 2\eta$. Since $E_e[h \,|\, h < x] - x$ is decreasing in x, $E_e[h \,|\, h > 1-2\eta] - (1-2\eta) > \eta$ or:

$$E_e[h \,|\, h > 1-2\eta] > 1-\eta \tag{3'}$$

Note that $f_e(h)$ strictly log-concave and differentiable implies single-peakedness of $f_e(h)$ resulting in three possible cases:[41] (i) $f_e'(h) < 0$ for all h (i.e. $F_e''(h) < 0$), (ii) $f_e'(h) > 0$ for all h (i.e. $F_e''(h) > 0$), or (iii) $f_e'(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ (i.e. $F_e''(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$).

Consider intervals $[a, b] \subset [0, 1]$ and $h \in [a, b]$, we have the following relationship:[42]

$$F_e''(h) \lessgtr 0 \quad \Rightarrow \quad (b-a)\frac{F_e(b) + F_e(a)}{2} \lessgtr \int_a^b F_e(h)dh \quad \Leftrightarrow \quad E_e[h \,|\, a < h < b] \lessgtr \frac{a+b}{2} \tag{4'}$$

Combining (1')-(2')-(3') with (4'), there are three subcases:

[41] Given $f_e(h)$ is positive, strict log concavity of $f_e(h)$ is equivalent to strict concavity of $log(f_e(h))$, i.e. $f_e''(h)f_e(h) < (f_e'(h))^2$. Suppose there exists x such that $f_e'(x) = 0$, then it has to be the case that $f_e''(x) < 0$. At most one value satisfies this property.

[42] The first implication relies on obvious properties of increasing and concave as well as increasing and convex functions. The second equivalence is obtain by integrating $\int_a^b F_e(h)dh$ by parts.

(3i) $F_e''(h) < 0$ for all h. Only $(1')$ can be satisfied so only **B** can be an equilibrium. Furthermore, $h^{**} < 1$ if and only if $1 - E_e[h] > \eta$ which is always true since $F_e''(h) < 0$ implies $E_e[h] < 1/2$ $(< 1 - \eta)$.

(3ii) $F_e''(h) > 0$ for all h. Only $(3')$ can be satisfied so only **T** can be an equilibrium. Furthermore, $h^* > 0$ if and only if $E_e[h] > \eta$ which is always true since $F_e''(h) > 0$ implies $E_e[h] > 1/2$ $(> \eta)$.

(3iii) $F_e''(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$. Fix η and $F_e(\cdot)$ and suppose that $\eta\, F_e(2\eta) < \int_0^{2\eta} F_e(h)dh$ (condition $(1')$ holds). This means there is $h^{**} < 1$ such that a **B**-equilibrium exists. Furthermore, it is by construction such that $F_e''(2\eta) < 0$ (or $2\eta > \tilde{h}$). This equilibrium is unique if $(2')$ and $(3')$ never hold, that is, if:

$$\eta[F_e(x + 2\eta) + F_e(x)] < \int_x^{x+2\eta} F_e(h)dh \qquad \forall x \in (0, 1 - 2\eta] \tag{5'}$$

Fix x and notice that $F_e''(x + 2\eta) < 0$ which implies $F_e'(x + 2\eta) < \frac{F_e(x+2\eta)-F_e(x)}{2\eta}$ for all x. If $F_e''(x) < 0$, then $F_e''(h) < 0$ for all $h \in [x, x + 2\eta]$ and therefore $(5')$ automatically holds as in case (3i). If $F_e''(x) > 0$, then $F_e'(x) < \frac{F_e(x+2\eta)-F_e(x)}{2\eta}$. This means that $\eta[F_e'(x) + F_e'(x + 2\eta)] < F_e(x + 2\eta) - F_e(x)$. So, as x increases the left hand side of $(5')$ increases at a lower rate than the right hand side of $(5')$. Hence, if the inequality holds at $x = 0$ it also holds at all $x > 0$ and no **M** or **T** equilibrium exists.

Suppose now that $\eta[1+F_e(1-2\eta)] > \int_{1-2\eta}^1 F_e(h)dh$ (condition $(3')$ holds). This means there is $h^* > 0$ such that a **T**-equilibrium exists. Furthermore, it is by construction such that $F_e''(1-2\eta) > 0$ (or $1-2\eta < \tilde{h}$). This equilibrium is unique if $(1')$ and $(2')$ never hold, that is, if:

$$\eta[F_e(1 - x) + F_e(1 - 2\eta - x)] > \int_{1-2\eta-x}^{1-x} F_e(h)dh \qquad \forall x \in [0, 1 - 2\eta] \tag{6'}$$

Fix x and notice that $F_e''(1-2\eta-x) > 0$ which implies $F_e'(1-2\eta-x) < \frac{F_e(1-x)-F_e(1-2\eta-x)}{2\eta}$ for all x. If $F_e''(1-x) > 0$, then $F_e''(h) > 0$ for all $h \in [1-2\eta-x, 1-x]$ and therefore $(6')$ automatically holds as in case (3ii). If $F_e''(1-x) < 0$, then $F_e'(1-x) < \frac{F_e(1-x)-F_e(1-2\eta-x)}{2\eta}$. This means that $\eta[F_e'(1 - x) + F_e'(1 - 2\eta - x)] < F_e(1 - x) - F_e(1 - 2\eta - x)$. So, as x increases the left hand side of $(6')$ decreases at a lower rate than the right hand side of $(6')$. Hence if the inequality holds at $x = 0$ it also holds at all $x > 0$ and no **M** or **B** equilibrium exists.

Suppose last that $\eta[F_e(h^o + 2\eta) + F_e(h^o)] = \int_{h^o}^{h^o+2\eta} F_e(h)dh$ (condition $(2')$ holds). Consider the function $M(x) = \int_x^{x+2\eta} F_e(h)dh - \eta[F_e(x + 2\eta) + F_e(x)]$. It represents the area between the curve $F_e(x)$ between x and $x + 2\eta$ and the line joining points $(x, F_e(x))$

to point $(x+2\eta, F_e(x+2\eta))$. By construction, the line is above the curve whenever $x < \tilde{h} - 2\eta$ and therefore we have $M(x) < 0$. By contrast, the line is below the curve when $x > \tilde{h}$ and therefore, we have $M(x) > 0$. For all $x \in [\tilde{h} - 2\eta, \tilde{h}]$, there exists a unique point $k(x)$ where the line and the curve cross. The line is above the curve on $[x, k(x)]$ and it is above the curve on $[k(x), x + 2\eta]$. Let $\underline{M}(x)$ be the area between the curve and the line on $[x, k(x)]$ and $\overline{M}(x)$ the area between the curve and the line on $[k(x), x+2\eta]$, we have $M(x) = \underline{M}(x) + \overline{M}(x)$, $\underline{M}(x) < 0$ and $\overline{M}(x) > 0$. By construction, as x increases, $k(x)$ decreases and therefore both $\underline{M}(x)$ and $\overline{M}(x)$ increase. Assume there exists a point x^* such that $M(x^*) = 0$. This point must lie in $[\tilde{h} - 2\eta, \tilde{h}]$ and must be such that $\underline{M}(x^*) = -\overline{M}(x^*)$. Given the previous points, $M(x) < 0$ for all $x < x^*$, and $M(x) > 0$ and for all $x > x^*$.This proves that h^o is unique when it exists. It also proves that $M(0) < 0$ (condition (1') does not hold) and $M(1-2\eta) > 0$ (condition (3') does not hold).Therefore, if a **M** equilibrium exists, no **B** or **T** equilibrium can exist. Last, note that by construction the two cutoffs h^o and $h^{oo} = h^o + 2\eta$ need to move away from each other when η increases. Therefore, we necessarily have $h^{o'}(\eta) < 0$ and $h^{oo'}(\eta) > 0$.

To summarize, when $\eta \in (0, 1/2)$ the equilibrium is unique given $F_e(\cdot)$ and η:

$$\begin{array}{ll} \mathbf{B} & \text{if} \quad E_e[h \,|\, h < 2\eta] < \eta \\ \mathbf{M} & \text{if} \quad E_e[h \,|\, h < 2\eta] > \eta \quad \text{and} \quad E_e[h \,|\, h > 1-2\eta] < 1-\eta \\ \mathbf{T} & \text{if} \quad E_e[h \,|\, h > 1-2\eta] > 1-\eta \end{array}$$

Proof of Corollary 1: immediate and therefore omitted.

Proof of Corollary 2

We now identify the properties of the equilibrium for a given η as a function of the distribution. It is well-known that MLRP, that is, $\left(\frac{f_e(h)}{f_{e'}(h)}\right)' < 0 \quad \forall e' > e$, implies $E_{e'}[h \,|\, a < z < b] > E_e[h \,|\, a < z < b]$. Suppose now that we are on a **B**-equilibrium. Denote h_e^{**} the no-self control cutoff given environment e. We have: $h_e^{**} - E_e[h \,|\, h \le h_e^{**}] = \eta$ and $h_{e'}^{**} - E_{e'}[h \,|\, h \le h_{e'}^{**}] = \eta$. Suppose $h_{e'}^{**} = h_e^{**} - \delta$ with $\delta > 0$. Then:

$$h_e^{**} - E_e[h \,|\, h \le h_e^{**}] = h_e^{**} - \delta - E_{e'}[h \,|\, h \le h_e^{**} - \delta]$$

Log-concavity of $f_e(h)$ implies that $E_{e'}[h \,|\, h \le h_e^{**} - \delta] > E_{e'}[h \,|\, h \le h_e^{**}] - \delta$. Combining this inequality with the previous equation, we get:

$$E_{e'}[h \,|\, h \le h_e^{**}] < E_e[h \,|\, h \le h_e^{**}]$$

which is a contradiction. Therefore, $h_{e'}^{**} > h_e^{**}$. The same argument demonstrates that in a **M**-equilibrium $h_{e'}^{o} > h_e^{o}$ and $h_{e'}^{oo} > h_e^{**}$ and in a **T**-equilibrium $h_{e'}^{*} > h_e^{*}$. Thus, as the

distribution shifts towards higher values of h in a MLRP sense, we move from **B** to **A** to **T**-equilibrium if $\eta \in [1/2, 1)$ and from **B** to **M** to **T**-equilibrium if $\eta \in [0, 1/2)$.

Proof of Corollary 3

We analyze the quantity of modulation as a function of η.

Step 1. We first study the sequence of equilibria for a given distribution $F_e(\cdot)$ as a function of η.

Case 1: $\eta > 1$. The equilibrium is always **A**. There is no modulation.

Case 2: $\eta \in (1/2, 1)$. This is immediate from Step 2 - case 2 in the proof of the main theorem. If the distribution is such that $E_e[h] < 1/2$, the equilibrium is **B** when $\eta \in (1/2, 1 - E_e[h])$ and **A** when $\eta \in (1 - E_e[h], 1)$. If the distribution is such that $E_e[h] \geqslant 1/2$, the equilibrium is **T** when $\eta \in (1/2, E_e[h])$ and **A** when $\eta \in (E_e[h], 1)$.

Case 3: $\eta \in (0, 1/2)$. We have 3 subcases

(2i) $F_e''(h) < 0$ for all h. The only equilibrium for all $\eta \in (0, 1/2)$ is **B**. This is immediate from the previous proof.

(2ii) $F_e''(h) > 0$ for all h. The only equilibrium for all $\eta \in (0, 1/2)$ is **T**. This is also immediate.

(2iii) $F_e''(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$. Let

$$A(\eta) = \eta F_e(2\eta) - \int_0^{2\eta} F_e(h)dh$$

$$B(\eta) = \eta[1 + F_e(1 - 2\eta)] - \int_{1-2\eta}^{1} F_e(h)dh$$

We remind the reader that $A(\eta) < 0$ in a **B**-equilibrium and $B(\eta) > 0$ in a **T**-equilibrium. We have $A(1/2) = B(1/2) = E_e[h] - 1/2$ and $A(0) = B(0) = 0$. Note that $A''(\eta) = 4\eta f_e'(2\eta)$, hence $A(\eta)$ is convex for all $\eta \leqslant \tilde{h}/2$ and concave for all $\eta > \tilde{h}/2$. We also have $A'(\eta) = 2\eta f_e(2\eta) - F_e(2\eta)$ and given by construction $hf_e(h) > F_e(h)$ for all $h < h$, we deduce that $lim_{\eta \to 0} A'(\eta) > 0$. Combining these findings, when $E_e[h] > 1/2$, $A(\eta) > 0$ for all $\eta \in (0, 1/2)$. When $E_e[h] < 1/2$, there exists a unique $\underline{\eta}$ such that $A(\eta) > 0$ for all $\eta < \underline{\eta}$ and $A(\eta) < 0$ for all $\eta \in (\underline{\eta}, 1/2)$.

Note also that $B''(\eta) = 4\eta f_e'(1 - 2\eta)$, hence $B(\eta)$ is concave for all $\eta \leqslant (1 - \tilde{h})/2$ and convex for all $\eta > (1 - \tilde{h})/2$. We also have $B'(\eta) = -2\eta f_e(1-2\eta) + 1 - F_e(1-2\eta)$ and given by construction $(1 - h)f_e(h) > 1 - F_e(h)$ for all $h > \tilde{h}$, we deduce that $lim_{\eta \to 0} B'(\eta) < 0$. Combining these findings, when $E_e[h] < 1/2$, $B(\eta) < 0$ for all $\eta \in (0, 1/2)$. When $E_e[h] > 1/2$, there exists a unique $\overline{\eta}$ such that $B(\eta) < 0$ for all $\eta < \overline{\eta}$ and $A(\eta) > 0$ for all $\eta \in (\overline{\eta}, 1/2)$.

Overall, when $E_e[h] > 1/2$, the equilibrium is **M** when $\eta < \overline{\eta}$ and **T** when $\eta \in (\overline{\eta}, 1/2)$. When $E_e[h] < 1/2$, the equilibrium is **M** when $\eta < \underline{\eta}$ and **B** when $\eta \in (\underline{\eta}, 1/2)$.

To summarize, as η increases, the sequence of equilibria given $F_e(\cdot)$ is:

$$\begin{array}{ll} \textbf{(M) B A} & \text{if} \quad E_e[h] < 1/2 \\ \textbf{(M) T A} & \text{if} \quad E_e[h] > 1/2 \end{array}$$

where parentheses indicate that the equilibrium may not occur for some distributions. Recall that **(M)** is a possible equilibrium only when there exists $\tilde{h} \in (0,1)$ such that $F_e''(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$.

Note that $A(\underline{\eta}) = 0$ implies that $h^o(\underline{\eta}) = 0$ and $h^{oo}(\eta^*) = 2\underline{\eta}$. Combining (1') and $A(\underline{\eta}) = 0$ implies $h^{**}(\underline{\eta}) = 2\underline{\eta}$ as well. Therefore, as η increases, the transition from **M** to **B** is smooth. Similarly, $B(\overline{\eta}) = 0$ implies that $h^o(\overline{\eta}) = 1 - 2\overline{\eta}$ and $h^{oo}(\overline{\eta}) = 1$. Combining (3') and $B(\overline{\eta}) = 0$ implies $h^*(\overline{\eta}) = 1 - 2\overline{\eta}$. Therefore, as η increases, the transition from **M** to **T** is also smooth.

Step 2. Note that h^{**} increases in η and therefore, there is less modulation in **B** equilibria as η increases. Similarly, h^* decreases in η and therefore, there is less modulation in **T** equilibria as η increases. When **(M)** exists, no modulation occurs only between h^o and $h^{oo} = h^o + 2\eta$. As η increases, the interval in which no modulation occurs increases. Given the transition from equilibria **(M)** and **B** or **(M)** and **T** are smooth, the quantity of modulation decreases continuously as η increases.

Proof of Corollary 4

Assume $E_e[h] < 1/2$. There are 2 possible cases.

Case 1. $f_e'(h) < 0$ for all h or $f_e'(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium does not exist. From (12), for all $\eta < 1 - E_e[h]$, the equilibrium is **B** and there exists $h^{**}(\eta)$ such that $h^{**}(\eta) - E_e[h \,|\, h < h^{**}(\eta)] = \eta$. For all $\eta \geqslant 1 - E_e[h]$, the equilibrium is **A** and $H_e = [0,1]$. For all h, there exists $i(h)$ such that $h^{**}(i(h)) = h$ and such that the equilibrium is **B** and modulation occurs at h when $\eta < i(h)$, the equilibrium is **B** but no modulation occurs at h when $\eta \in (i(h), 1 - E_e[h])$ and the equilibrium is **A** and no modulation occurs at h when $\eta > 1 - E_e[h]$. Given $h^{**}(\eta)$ is increasing, $i(h')$ is increasing. For each h', there are three consumption scenarii: (i) when $\eta < i(h')$, consumption takes place if $\theta > \alpha h'$; (ii) when $\eta \in (i(h'), 1 - E_e[h])$, consumption takes place when $\theta > \alpha E_e[h \,|\, h' < h^{**}(\eta)]$; (iii) when $\eta > 1 - E_e[h]$, consumption takes place when $\theta > \alpha E_e[h]$. Overall, for all h', consumption takes place when $\theta > \bar{\theta}(h)$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{i(h')} \alpha h' \, dG(\eta) + \int_{i(h')}^{1-E_e[h]} \alpha E_e[h \,|\, h < h^{**}(\eta)] \, dG(\eta) + \int_{1-E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

Note that $\bar{\theta}(0) > 0$ and $\bar{\theta}(1) < \alpha$. We also have

$$\frac{d\bar{\theta}}{dh'} = \alpha\, G(i(h'))\left[1 + \frac{g(i(h'))}{G(i(h'))} i(h') i'(h')\right] > 0 \tag{15}$$

Case 2. $f'_e(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium exists. For all $\eta < \underline{\eta}$, the equilibrium is **M** and there exists $h^o(\eta)$ such that $E_e[h | h^o(\eta) \leqslant h \leqslant h^o(\eta) + 2\eta] = h^o + \eta$. For all $\eta \in [\underline{\eta}, 1 - E_e[h]]$, the equilibrium is **B** and there exists $h^{**}(\eta)$ such that $h^{**}(\eta) - E_e[h | h < h^{**}(\eta)] = \eta$. For all $\eta > 1 - E_e[h]$, then $h^{**} = 1$. Note that the unique health rating that solves $E_e[h | h^o(\eta) \leqslant h \leqslant h^o(\eta) + 2\eta] = h^o + \eta$ when $\eta \to 0$ is $\tilde{h}$. There are three cases: (i) $h' < \tilde{h}$ and modulation occurs only for low values of η in the **M** equilibrium; (ii) $h' \in [\tilde{h}, 2\underline{\eta}]$ no modulation occurs when η is high enough in **M**, and modulation never occurs in the **B** equilibrium. (iii) $h' > 2\underline{\eta}$ and modulation occurs always in the **M** and only if η is not too high in the **B** equilibrium.

Case 2i: $h' < \tilde{h}$, there exists $i^o(h') < \underline{\eta}$ such that $h^o(i^o(h')) = h'$ and modulation occurs only when $\eta < i^o(h')$. Consumption takes place on average when $\theta > \bar{\theta}(h')$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{i^o(h')} \alpha h'\, dG(\eta) + \int_{i^o(h')}^{\underline{\eta}} \alpha E_e[h \,|\, h^o(\eta) < h < h^{oo}(\eta)]\, dG(\eta)$$

$$+ \int_{\underline{\eta}}^{1-E_e[h]} \alpha E_e[h \,|\, h < h^{**}(\eta)]\, dG(\eta) + \int_{1-E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

We have

$$\frac{d\bar{\theta}}{dh'} = \alpha\, G(i^o(h'))\left[1 - \frac{g(i^o(h'))}{G(i^o(h'))} i^o(h') i^{o'}(h')\right] \tag{16}$$

Note that $h^o(\eta)$ decreases in η and therefore $i^o(h')$ decreases which proves that $\frac{d\bar{\theta}}{dh'} > 0$. Note also that $\bar{\theta}(0) > 0$.

Case 2ii: $h' \in [\tilde{h}, 2\underline{\eta}]$, there exists $i^{oo}(h) < \underline{\eta}$ such that $h^{oo}(i^{oo}(h)) = h$. Consumption takes place on average when $\theta > \bar{\theta}(h)$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{i^{oo}(h')} \alpha h'\, dG(\eta) + \int_{i^{oo}(h')}^{\underline{\eta}} \alpha E_e[h \,|\, h^o(\eta) < h < h^{oo}(\eta)]\, dG(\eta)$$

$$+ \int_{\underline{\eta}}^{1-E_e[h]} \alpha E_e[h \,|\, h < h^{**}(\eta)]\, dG(\eta) + \int_{1-E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

We have

$$\frac{d\bar{\theta}}{dh'} = \alpha\, G(i^{oo})(h')\left[1 + \frac{g(i^{oo}(h'))}{G(i^{oo}(h'))} i^{oo}(h') i^{oo'}(h')\right] \tag{17}$$

Note that $h^{oo}(\eta)$ increases in η and therefore $i^{oo}(h')$ increases which proves that $\frac{d\bar{\theta}}{dh'} > 0$.

Case 2iii: $h' > 2\underline{\eta}$, there exists $i(h')$ such that $h^{**}(i(h')) = h'$. Consumption takes place on average when $\theta > \bar{\theta}(h)$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{i(h')} \alpha h' \, dG(\eta) + \int_{i(h')}^{1-E_e[h]} \alpha E_e[h \,|\, h < h^{**}(\eta)] \, dG(\eta) + \int_{1-E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

which we know to be increasing in h'. Note also that $\bar{\theta}(1) < \alpha$.

To sum up, when $E_e[h] < 1/2$, the function $\bar{\theta}(h')$ is increasing in h' and such that $\bar{\theta}(0) > 0$ and $\bar{\theta}(1) < \alpha$. Therefore, there exist $\underline{h} \in (0,1)$ and $\bar{h} \in (0,1)$ such that $\bar{\theta}(h') > \alpha h$ for all $h' < \underline{h}$ and $\bar{\theta}(h') < \alpha h'$ for all $h' > \bar{h}$.

Assume now that $E_e[h] > 1/2$. There are 2 possible cases.

Case 3. $f_e'(h) > 0$ for all h or $f_e'(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium does not exist. From (14), for all $\eta < E_e[h]$, the equilibrium is **T** and there exists $h^*(\eta)$ such that $E_e[h \,|\, h > h^*(\eta)] - h^*(\eta) = \eta$. For all $\eta \geqslant E_e[h]$, the equilibrium is **A** and $H_e = [0,1]$. For all h, there exists $j(h)$ such that $h^*(j(h)) = h$ and such that the equilibrium is **T** and modulation occurs at h when $\eta < j(h)$, the equilibrium is **T** but no modulation occurs at h when $\eta \in (j(h), E_e[h])$ and the equilibrium is **A** and no modulation occurs at h when $\eta > E_e[h]$. Given $h^*(\eta)$ is decreasing, $j(h')$ is decreasing. For each h', there are three consumption scenarii: (i) when $\eta < j(h')$, consumption takes place if $\theta > \alpha h'$; (ii) when $\eta \in (j(h), E_e[h])$, consumption takes place when $\theta > \alpha E_e[h \,|\, h > h^*(\eta)]$; (iii) when $\eta > E_e[h]$, consumption takes place when $\theta > \alpha E_e[h]$. Overall, for all h', consumption takes place when $\theta > \bar{\theta}(h)$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{j(h')} \alpha h' \, dG(\eta) + \int_{j(h')}^{E_e[h]} \alpha E_e[h \,|\, h > h^*(\eta)] \, dG(\eta) + \int_{E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

Note that $\bar{\theta}(0) > 0$ and $\bar{\theta}(1) < \alpha$. We have

$$\frac{d\bar{\theta}}{dh'} = \alpha \, G(j(h')) \left[1 - \frac{g(j(h'))}{G(j(h'))} j(h') j'(h') \right] > 0 \tag{18}$$

Case 4. $f_e'(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium exists. For all $\eta < \overline{\eta}$, the equilibrium is **M** and there exists $h^o(\eta)$ such that $E_e[h | h^o(\eta) \leqslant h \leqslant h^o(\eta) + 2\eta] = h^o + \eta$. For all $\eta \in [\overline{\eta}, E_e[h]]$, the equilibrium is **T** and there exists $h^*(\eta)$ such that $E_e[h | h < h^*(\eta)] - h^*(\eta) = \eta$. For all $\eta > E_e[h]$, then $h^* = 0$. Again, the unique health rating that solves $E_e[h | h^o(\eta) \leqslant h \leqslant h^o(\eta) + 2\eta] = h^o + \eta$ when $\eta \to 0$ is $\tilde{h}$.

There are three cases: (i) $h' > \tilde{h}$ and modulation occurs only for low values of η in the **M** equilibrium; (ii) $h' \in [1-2\overline{\eta}, \tilde{h}]$ no modulation occurs when η is high enough in **M**, and modulation never occurs in the **T** equilibrium. (iii) $h' < 1-2\overline{\eta}$ and modulation occurs always in the **M** and only if η is not too high in the **T** equilibrium.

Case 4i: $h' > \tilde{h}$, there exists $j^{oo}(h') < \overline{\eta}$ such that $h^{oo}(j^{oo}(h')) = h'$ and modulation occurs only when $\eta < j^{oo}(h')$. Consumption takes place on average when $\theta > \bar{\theta}(h')$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{j^{oo'}(h')} \alpha h' \, dG(\eta) + \int_{j^{oo}(h')}^{\overline{\eta}} \alpha E_e[h \,|\, h^o(\eta) < h < h^{oo}(\eta)] \, dG(\eta)$$

$$+ \int_{\overline{\eta}}^{E_e[h]} \alpha E_e[h \,|\, h > h^*(\eta)] \, dG(\eta) + \int_{E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

We have

$$\frac{d\bar{\theta}}{dh'} = \alpha \, G(j^o(h')) \left[1 + \frac{g(j^{oo}(h'))}{G(j^{oo}(h'))} j^{oo}(h') j^{oo'}(h') \right] \tag{19}$$

Note that $h^{oo}(\eta)$ increases in η and therefore $j^{oo}(h')$ increases which proves that $\frac{d\bar{\theta}}{dh'} > 0$. Note also that $\bar{\theta}(1) < \alpha$.

Case 4ii: $h' \in [1-2\overline{\eta}, \tilde{h}]$, there exists $j^{oo}(h) < \overline{\eta}$ such that $h^o(j^o(h)) = h$. Consumption takes place on average when $\theta > \bar{\theta}(h)$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{j^{o}(h')} \alpha h' \, dG(\eta) + \int_{j^{o}(h')}^{\overline{\eta}} \alpha E_e[h \,|\, h^o(\eta) < h < h^{oo}(\eta)] \, dG(\eta)$$

$$+ \int_{\overline{\eta}}^{E_e[h]} \alpha E_e[h \,|\, h < h^{**}(\eta)] \, dG(\eta) + \int_{E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

We have

$$\frac{d\bar{\theta}}{dh'} = \alpha \, G(j^o)(h') \left[1 - \frac{g(j^o(h'))}{G(j^o(h'))} j^o(h') j^{o'}(h' \right] \tag{20}$$

Note that $h^o(\eta)$ decreases in η and therefore $j^o(h')$ decreases which proves that $\frac{d\bar{\theta}}{dh'} > 0$.

Case 4iii: $h' < 1-2\underline{\eta}$, there exists $j(h')$ such that $h^*(j(h')) = h'$. Consumption takes place on average when $\theta > \bar{\theta}(h)$ where

$$\bar{\theta}(h') = \int_{\eta=0}^{j(h')} \alpha h' \, dG(\eta) + \int_{j(h')}^{1-E_e[h]} \alpha E_e[h \,|\, h > h^*(\eta)] \, dG(\eta) + \int_{E_e[h]}^{\eta^*} \alpha E_e[h] dG(\eta)$$

which we know to be increasing in h'. Note also that $\bar{\theta}(0) > 0$.

When $E_e[h] > 1/2$, the function $\bar{\theta}(h')$ is increasing in h' and such that $\bar{\theta}(0) > 0$ and $\bar{\theta}(1) < \alpha$. Therefore, there exist $\underline{h} \in (0,1)$ and $\bar{h} \in (0,1)$ such that $\bar{\theta}(h') > \alpha h$ for all $h' < \underline{h}$ and $\bar{\theta}(h') < \alpha h'$ for all $h' > \bar{h}$.

Proof of Corollary 5: This corresponds to a straightforward change of variable. Note that δ^t is monotonic (decreasing) in t. Let $m = \delta^t$ and replace h by m in the previous proofs. More precisely, there is no modulation when $m \in M$ where

$$\Delta(m, E_e[m \,|\, m \in M]) = \int_{E_e[m \,|\, m \in M]}^{m} (m - \theta) d\theta > c$$

which is convex in m. Therefore M is compact, and given monotonicity of δ^t, T is also compact. The rest of the proofs are straightforward adaptations of the previous ones and therefore omitted.

Proof of Corollary 6

Under beliefs $\underline{p}$, we have $E_{\underline{p}}[h] \to 0$. For each η, modulation occurs at the top and $h^{**}(\eta) \to \eta$. Overall, consumption takes place on average when $\theta > \bar{\theta}(h'; \underline{p})$ where

$$\bar{\theta}(h'; \underline{p}) = \int_0^{h'} \alpha h' \, dG(\eta) \leqslant \alpha h'$$

which is increasing in h' and such that $\bar{\theta}(0) = 0$ and $\bar{\theta}(1) < \alpha$.

Under beliefs $\overline{p}$, we have $E_{\overline{p}}[h] \to 1$. For each η, modulation occurs at the bottom and $h^*(\eta) \to 1 - \eta$. Overall, consumption takes place on average when $\theta > \bar{\theta}(h', \overline{p})$ where

$$\bar{\theta}(h', \overline{p}) = \int_0^{1-h'} \alpha h' \, dG(\eta) + \int_{1-h'}^{\eta^*} \alpha \, dG(\eta) \geqslant \alpha h'$$

which is increasing in h' and such that $\bar{\theta}(0) > 0$ and $\bar{\theta}(1) = \alpha$.

Proof of Corollary 7: immediate and therefore omitted.

Appendix C

Sufficient conditions in Corollary 4

The proof consists in studying the variations of $\bar{\theta}(h)$ when $\eta^* = 1$ and $G(\eta) = \eta$. We go through the same cases as in the proof of Corollary 4.

Case 1. Distributions satisfying $E_e[h] < 1/2$ and $f_e'(h) < 0$ for all h or $f_e'(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium does not exist.

$$\frac{d\bar{\theta}}{dh'} = \alpha\, i(h')\left[1 + i'(h')\right] \quad \text{and} \quad \frac{d^2\bar{\theta}}{dh'^2} = \alpha\, i'(h')\left[1 + i'(h')\right] + \alpha\, i(h')i''(h') \tag{21}$$

From the definition of $i(h')$, we have $h' - E_e[h|h < h'] = i(h')$, which implies:

$$i'(h') = 1 - \frac{\partial}{\partial h'} E_e[h|h < h'] \quad \text{and} \quad i''(h') = -\frac{\partial^2}{\partial h'^2} E_e[h|h < h']$$

Also:

$$E_e[h|h < h'] = h' - \frac{\int_0^{h'} F_e(h)dh}{F_e(h')} \quad \Rightarrow \quad \frac{\partial}{\partial h'} E_e[h|h < h'] = \frac{\int_0^{h'} F_e(h)dh}{F_e(h')} \frac{f_e(h')}{F_e(h')} = i(h')\mathcal{J}(h')$$

where $\mathcal{J}(h') = f_e(h')/F_e(h')$ which is decreasing in h'. Therefore

$$i''(h') = -\Big(1 - i(h')\mathcal{J}(h')\Big)\mathcal{J}(h') - i(h')\mathcal{J}'(h')$$

Using these equalities we obtain

$$\frac{d^2\bar{\theta}}{dh'^2} = 2\alpha\,[i'(h')]^2 - \alpha\,[i(h')]^2\mathcal{J}'(h') > 0.$$

Combining $\bar{\theta}(0) > 0$, $\bar{\theta}(1) < \alpha$, $\frac{d\bar{\theta}}{dh'} > 0$ and $\frac{d^2\bar{\theta}}{dh^2} > 0$, we get that there exists a unique value $\bar{h} \in (0,1)$ such that $\bar{\theta}(h') > \alpha h$ for all $h' < \bar{h}$ and $\bar{\theta}(h') < \alpha h'$ for all $h' > \bar{h}$.

Case 2. Distributions satisfying $E_e[h] < 1/2$ and $f_e'(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium exists

Case 2i : when $h' < \tilde{h}$, we have

$$\frac{d^2\bar{\theta}}{dh'^2} = \alpha\, i^{o'}(h')\left[1 - i^{o'}(h')\right] - \alpha\, i^o(h')i^{o''}(h') \tag{22}$$

Consider functions that satisfy the following condition P*: $f_e(x) = f_e(1-x)$ for $x < \tilde{h}$ when $\tilde{h} < 1/2$ and $f_e(x) = f_e(1-x)$ for $x > \tilde{h}$ when $\tilde{h} > 1/2$.

When P* is satisfied, we also have $h^o(\eta) = \tilde{h} - \eta$ and therefore $i^o(h') = \tilde{h} - h'$ and therefore $\frac{d^2\bar{\theta}}{dh'^2} < 0$.

Case 2ii: when $h' \in [\tilde{h}, 2\underline{\eta}]$, we have

$$\frac{d\bar{\theta}}{dh'} = \alpha\, i^{oo}(h')\left[1 + i^{oo'}(h')\right] \quad \text{and} \quad \frac{d^2\bar{\theta}}{dh'^2} = \alpha\, i^{oo'}(h')\left[1 + i^{oo'}(h')\right] + \alpha\, i(h')i^{oo''}(h') \tag{23}$$

When P^* is satisfied, we also have $h^{oo}(\eta) = \tilde{h} + \eta$ and therefore $i^{oo}(h') = h' - \tilde{h}$ and therefore $\frac{d^2\bar{\theta}}{dh'^2} > 0$.

Case 2iii. when $h' > 2\underline{\eta}$, this is similar to case 1. We have $\frac{d^2\bar{\theta}}{dh'^2} > 0$.

Case 3. Distributions satisfying $E_e[h] > 1/2$ and $f'_e(h) > 0$ for all h or $f'_e(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium does not exist.

$$\frac{d\bar{\theta}}{dh'} = \alpha\, j(h')\left[1 - j'(h')\right] \quad \text{and} \quad \frac{d^2\bar{\theta}}{dh'^2} = \alpha\, j'(h')\left[1 - j'(h')\right] - \alpha\, j(h')j''(h') \tag{24}$$

From the definition of $j(h')$, we have $E_e[h|h > h'] - h' = j(h')$, which implies:

$$j'(h') = \frac{\partial}{\partial h'}E_e[h|h > h'] - 1 \quad \text{and} \quad j''(h') = \frac{\partial^2}{\partial h'^2}E_e[h|h > h']$$

Also:

$$E_e[h|h > h'] = \frac{1 - h'F_e(h') - \int_{h'}^1 F_e(h)dh}{1 - F_e(h')} \;\Rightarrow\; \frac{\partial}{\partial h'}E_e[h|h > h'] = \frac{1 - h' - \int_{h'}^1 F_e(h)dh}{1 - F_e(h')}\frac{f_e(h')}{1 - F_e(h')}$$

that is

$$\frac{\partial}{\partial h'}E_e[h|h > h'] = j(h')\mathcal{K}(h')$$

where $\mathcal{K}(h') = f_e(h')/(1 - F_e(h'))$ which is increasing in h'. Therefore

$$j''(h') = \Big(j(h')\mathcal{K}(h') - 1\Big)\mathcal{K}(h') + j(h')\mathcal{K}'(h')$$

Using these equalities we obtain

$$\frac{d^2\bar{\theta}}{dh'^2} = -2\alpha\left[j'(h')\right]^2 - \alpha\left[j(h')\right]^2\mathcal{K}'(h') < 0.$$

As in case 1, there exists a unique value $\bar{h} \in (0, 1)$ such that $\bar{\theta}(h') > \alpha h$ for all $h' < \bar{h}$ and $\bar{\theta}(h') < \alpha h'$ for all $h' > \bar{h}$.

Case 4. Distributions satisfying $E_e[h] > 1/2$ and $f'_e(h) \gtrless 0$ for all $h \lessgtr \tilde{h}$ such that a **M** equilibrium exists

Case 4i: When $h' > \tilde{h}$, we have

$$\frac{d^2\bar{\theta}}{dh'^2} = \alpha\, j^{oo'}(h')\left[1 + j^{oo'}(h')\right] + \alpha\, j(h')j^{oo''}(h') \tag{25}$$

When P^* is satisfied, we also have $h^{oo}(\eta) = \tilde{h} + \eta$ and therefore $j^{oo}(h') = h' - \tilde{h}$ and therefore $\frac{d^2\bar{\theta}}{dh'^2} > 0$.

Case 4ii When $h' \in [1-2\bar{\eta}, \tilde{h}]$, we have

$$\frac{d^2\bar{\theta}}{dh'^2} = \alpha\, j^{o'}(h') \left[1 - j^{o'}(h')\right] - \alpha\, j^{o}(h') j^{o''}(h') \tag{26}$$

When P* is satisfied, we also have $h^o(\eta) = \tilde{h} - \eta$ and therefore $j^o(h') = \tilde{h} - h'$ and therefore $\frac{d^2\bar{\theta}}{dh'^2} < 0$.

Case 4iii. When $h' < 1 - 2\bar{\eta}$, this is similar to case 3. We have $\frac{d^2\bar{\theta}}{dh'^2} < 0$.

Overall, when η is drawn from a uniform distribution on $[0,1]$, $\underline{h} = \overline{h}$ for distributions of the health parameter that satisfy cases 1 and 3. When the extra property P^* holds, we also have $\underline{h} = \overline{h}$ for distributions satisfying cases 2 and 4.

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