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

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Abstract

We develop a theory of self-control based on the evidence reported in Hare, Camerer and Rangel (2009). One brain system computes *the goal value of consumption and another brain system can* modulate *this value by transmitting information regarding the high-order considerations (e.g., healthiness or long term payoffs). We determine the optimal modulation and consumption strategy of the individual as a function of the cost of information transmission. The model has several testable behavioral implications. First, choices can be affected by distractors or tasks orthogonal to the decision. Second, the likelihood of consuming a tempting good endogenously depends on environmental cues and, in particular, on how tempting the good is expected to be. And third, costly modulation can cause under-regulation (self-indulgence) but also over-regulation (self-restraint). The model sheds also light on documented behavioral anomalies in the context of eating disorders, present-biased preferences and habit formation.*

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

JEL Classification: D03, D87.

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1 Introduction

Consumption of goods with short term and long term consequences has been an important research topic in economics and psychology. 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 studied the neural correlates of temptation and short- vs. long-term choices. 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 more recent findings suggest 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.

The goal of this paper is to formalize the self-control idea described in this recent strand of neuroscience experiments and 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 ventromedial prefrontal cortex (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, its incorporation into value is facilitated by some regions of the dorsolateral prefrontal cortex (dlPFC). Self-control is defined as a 'value modulation', that is, a transmission of information about the high-order attribute to vmPFC facilitated by dlPFC. This information transmission is costly but allows a more informed decision. Using this constrained optimization model, we first determine the optimal modulation and choice strategy of the individual.[2] We

[1] These include, but are not limited to, hyperbolic discounting theories (Strotz (1956), 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.).

[2] In that respect, the formal model is close 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

then derive implications that depart from self-control theories of hyperbolic discounting, cue-triggered behavior or myopic vs. farsighted systems.

The full characterization of the optimal information transmission and consumption constitutes the main theorem of the paper (Theorem 1). Technically, we show that information about the high-order attribute is not transmitted (that is, value is not modulated) when the realization is in a certain compact set H. When there is no modulation, value is computed assuming the expectation of the high-order attribute within that set. The conditions for no modulation, that is, the size and location of H, depend on the cost of attention, the relative importance of the high-order attribute, and perhaps more importantly the environment, that is, the distribution from which that attribute is drawn.

This simple result has a number of new behavioral implications, which we discuss in section 3. First, the likelihood of value modulation decreases in the presence of distractors or other tasks orthogonal to the decision and increases with the help of reminders regarding health considerations or long term benefits (Corollary 1). This is in line with the recent experimental findings in neuroeconomics (Hare et al., 2011) that link self-control to health cues. Second, the environment affects the decision to modulate value signal and consume the good. For example, a highly negative health signal about a tempting good is more likely to be transmitted if the situation is such that the individual is not supposed to encounter such unhealthy goods than if he is expected to (Corollaries 2 and 3). This relates to 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: the environment determines the expectation of the high-order attribute, which in turn influences the decision to transmit information and consume. Third, the theory predicts that costly value modulation always results in excessive consumption of very unhealthy goods but also in insufficient consumption of mildly unhealthy ones (Corollary 4). While over-consumption is a standard result in models of self-control, under-consumption usually is not. It implies that over- and under-regulation are two sides of the same coin: the very same reasons that induce a self-indulgent behavior can also be responsible for a self-restraint one.

In section 4, we use the main theorem to study choice in several paradigms that are known to involve the same brain systems as in [HCR] and are likely to feature similar modulation principles. Our first example is the symmetric case of healthy aversive food. Next, we apply our theory to decisions with long term consequences. In line with Luo et al. (2009), we reinterpret the high-order consideration as the date where the health effect

(2017) or Caplin, Dean and Leahy (2018).

is suffered (instead of the level). It follows that an exponential discounting individual subject to costly value modulation behaves like a hyperbolic discounting individual with no cost of value modulation, at least when comparing goods with short-term and long-term negative consequences (Corollary 5). In other words, decision making in our model will be insensitive to future outcomes, a behavior that is often described as 'decreasing impatience' (Strotz (1956); Ainslie (1975); Prelec (1989)).

In section 5, we extend the model to study choice anomalies. We analyze the case of eating disorders and we endow the decision-maker with an incorrect perception of the distribution of health ratings. If an individual 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), his modulation decision will be compromised resulting in systematic under-consumption in the first case and systematic over-consumption in the second (Corollary 6). This contrasts with the case of correct perceptions in which over- and under-consumption occur for the same individual depending on the health realization. We also analyze modulation and consumption of addictive substances. We model habit formation in the standard Becker and Murphy (1988) tradition: 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 propensity 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.

It is important to note that the theory we propose is rooted in neuroscientific evidence and the model itself shares few features with traditional models of self control and related topics. 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 (Fudenberg and Levine (2006), Brocas and Carrillo (2008)). Instead, the individual is modeled as 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 (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 (Laibson (2001)). Instead, it

affects endogenously his decision. Overall, our model cannot be directly compared to the existing literature. A notable reason is that, with the exception of the few studies grounded on neuroscience evidence (Bernheim and Rangel (2004), Brocas and Carrillo (2008)), 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. Said differently, while we cannot generate predictions that do not emerge from the interplay between the brain systems that we model (e.g., the effects of self-confidence on willpower), we can still predict a large range of behavior (e.g., under and over consumption) because our model does not arbitrarily restrict attention to some.

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 direct implications of the theory: the effect of distractors and environmental cues on modulation and choice, and the possibility of under- and over-regulation. We then expand the analysis to other paradigms that involve the same brain regions and are likely to be governed by a similar mechanism. These include choices in the aversive food domain and inter-temporal choices (section 4). We also address behavioral anomalies related to self control, such as eating disorders, addiction and impulsive behavior (section 5). In section 6, we address final comments. Proofs of theorem and corollaries are relegated to the appendix.

2 The model

2.1 The basic decision problem

We propose a theoretical model of self-control based on the *multi-attribute* neurophysiological approach of [HCR]. In their experiment, each participant was first asked to rate snacks for taste and health separately, and then asked to choose between pairs of snacks.[3] They 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. Moreover, dlPFC was more active when the healthiest option was chosen. Last, vmPFC and dlPFC exhibited functional connectivity when the healthiest choice was made.

[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.

According to this experimental evidence, value signals are encoded by one system (some regions of vmPFC), and this value dictates choices. This system receives and encodes information regarding basic attributes (taste). A second system (some regions of dlPFC) can modulate vmPFC and affect the final choices by facilitating the incorporation of information regarding high-order attributes (healthiness). If self-control is not exercised (i.e., value is not modulated), information about the high-order attribute is not transmitted and choice is dictated exclusively by taste. If self-control is exercised (i.e., value is modulated), then information about healthiness is transmitted and both attributes are taken into consideration when making a choice. In the paper, we put special emphasis in tightening our theory as close as possible to this neurobiological evidence.

To formalize the theory, 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 $h \in [0, 1]$.[4] The utility of the individual is additively separable and given by the following simple formulation:[5]

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

By definition of being tempting, the good is pleasurable ($\theta \geq 0$) but unhealthy ($h \geq 0$). The parameter $\alpha \in (0, 1)$ captures the importance of healthiness relative to taste in the decision. For example, an individual in a diet or with a physical condition will exhibit a high α.[6]

We assume that 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", and we order the environments

[4] In real settings, individuals can often choose quantities. Our binary model cannot capture decisions in those settings. We adopt it because our goal is to design a model that captures the existing experimental evidence and that can provide testable predictions for new studies. We rely on evidence based on paradigms in which the decision-maker can either consume or not consume, 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 in a model with continuous consumption choices.

[5] 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.

[6] 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.

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' than in environment e.

2.2 Representation of information in the brain

We now model how the brain represents the information relevant to the decision. Following [HCR], we posit an interplay between two systems. The dlPFC encodes a signal regarding the high-order attribute, the healthiness h. It then decides whether to exert self-control, that is, to transmit that signal to the vmPFC or not. This transmission of information is called *value modulation*. The vmPFC encodes a signal regarding the basic attribute, the taste θ. It receives the information from dlPFC (or not) and determines the goal value of the good on the basis of the signals available. 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.[7] 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.

As has been evidenced in the literature (e.g., [HCR]), signal modulation is *not pervasive*, which suggests that it must be costly at least to a certain extent. Indeed, modulation requires energy and attention to be spent in the coding and decoding of information and this energy is not freely available. To capture this feature, we introduce a cost of modulation c internalized by system $\mathcal{M}$. Signal modulation is also *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. One possibility is a mechanistic approach where system $\mathcal{C}$ incorrectly takes at face value that no information implies no health concerns (formally, it

[7] The set-up is analogous to that of a novel team problem. The team is composed of two agents with a common objective, and each member is informed about one characteristic of a joint project. One agent may decide to send a costly message to the other to transmit his information while the other makes the final decision for the team based on the information available.

assumes $h = 0$). Alternatively, one may argue that system $\mathcal{C}$ realizes the importance of the environment e and correctly 'infers' that no information transmission from system $\mathcal{M}$ still indicates a certain level of the health parameter.[8] The first option, quite reasonable as a first approximation, is in fact unsatisfactory for theoretical, intuitive, neurophysiological and empirical 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. From an intuitive viewpoint, it seems that inferences about the high-order attribute under no modulation should be different for a savory, unhealthy good (e.g., potato chips) than for an unsavory, healthy good (e.g., cod liver oil). In other words, the general properties of the good– captured through e in our model– are always incorporated. From a neurophysiology standpoint and consistent with the previous argument, the evidence that will be reviewed in section 2.4 suggests that some information is updated and some inferences are made by system $\mathcal{C}$ even under no modulation. Last, from an empirical standpoint and as we will see in sections 3, 4 and 5, predictions of a theory based on correct expectations match empirical observations better than those of a theory based on an incorrect face value interpretation of no evidence. For the rest of the paper, we will focus on the bayesian model with correct expectations under no modulation. In the economics terminology, the environment e is "public information" whereas the realizations of health and taste, h and θ, are "private information" of $\mathcal{M}$ and $\mathcal{C}$ respectively.

It is important to emphasize that, contrary to the recent dual process literature (e.g., Fudenberg and Levine (2006), Brocas and Carrillo (2008) and Alonso et al. (2014)), in this model *there is no conflict between systems*. Both $\mathcal{M}$ and $\mathcal{C}$ care about representing the utility of the individual correctly. Some choices are suboptimal only because systems have different access to information and communication is costly. These features of the model are shared with the work by Cunningham (2013) on automatic vs. reflective judgment.[9]

Notice that our model displays a multi-attribute feature reminiscent of the theories by Köszegi and Szeidl (2013) and Bordalo et al. (2013). However, contrary to these models, we do not presuppose that the decision-maker has a biased view of some attributes or assigns specific weights to them. As we shall see, endogenous information processing will generate a tendency to overweight one attribute with respect to the other. This effect will

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

[9] The model is however substantially different from the evidence we want to capture. Indeed, it focuses on a paradigm involving two systems with access to different pieces of information. The first system makes a judgement observable by the second, creating an opportunity for the latter to make inferences and to aggregate information. A third system decides whether to pay a cost to delegate the decision to the more informed second system.

take place at equilibrium and to serve an efficiency requirement.

Finally, this one period setting with costly information transmission can be interpreted as a reduced form of a more general dynamic optimal information transmission model. The literature in computational neuroscience usually formalizes 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 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. 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)). This model 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 of the decision (Brocas (2012)). Making the bounds endogenous is useful because it allows to link behavior to experimental conditions. Our theory falls within the category of optimal information processing models. However, our goal is not just to show that some information is not incorporated when making a decision (a feature also present, by construction, in computational models). Our main objective is to predict under which conditions this occurs, and to discuss the behavioral and neurophysiological consequences. Given we are not interested in dynamic effects, we collapse the information transmission process in a one-shot model.

2.3 Modulation and choice

Under the "rational expectations" approach, system $\mathcal{C}$ computes goal value anticipating that no information transmission by system $\mathcal{M}$ has some informational content about the health realization. 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 are more subtle. 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 $f_e(h \mid h \in H_e)$ 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 in 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 between modulation and no modulation, it knows the health rating of the good but not its taste.[10] However, it anticipates correctly how system $\mathcal{C}$ will represent the value of consumption and recommend a decision. Therefore, the values from its viewpoint of modulating and not modulating the signal given a health rating h are:

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

respectively, where c (> 0) is the attentional effort or cost of signal modulation. The condition for the optimality of modulation is $V^+(h) > V^-(h)$, which can be rewritten as:

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

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

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

[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.

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

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

which facilitates significantly the analytical characterization of the optimal modulation strategy as function of c, α and e.

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 $E_e[h \,|\, h^o \leq h \leq h^o + 2\eta] = h^o + \eta$.

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

The first noticeable characteristic of the optimal modulation mechanism is that information about health is not transmitted when its realization is between a lower and an upper threshold. More importantly, these thresholds vary as a function of the environment. As graphically depicted in Figure 1, it is possible that good health ratings are not transmitted ($\underline{h}_{e_1} = 0$ and $\overline{h}_{e_1} < 1$, as in environment e_1), medium health ratings are not transmitted ($\underline{h}_{e_2} > 0$ and $\overline{h}_{e_2} < 1$, as in environment e_2), or bad health ratings are not transmitted ($\underline{h}_{e_3} > 0$ and $\overline{h}_{e_3} = 1$, as in environment e_3). The second feature is that optimal modulation 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.[11] The third general property is that, in the absence of bounds on health ratings, the set of realizations for which there is no modulation would be of constant

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

size. Formally, $\overline{h}_e - \underline{h}_e = 2\eta$ for all e although, as we just noticed, the placement would depend on the environment. This is depicted with the (equal sized) brackets in the three environments of Figure 1. In practice, modulation can be more or less widespread simply because of the limits on health realizations ($h \in [0,1]$). Overall, the extent of modulation is inversely related to η, the cost of self-control and the relative importance of taste.

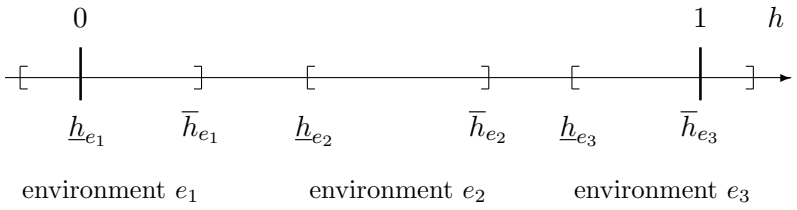

Figure 1. Different modulation regimes

Although these are interesting properties, the main value of our theory is that it provides a unified framework with new and testable implications for issues related to self-control problems. These implications are explored in sections 3, 4 and 5. But before, for the reader interested in the neurophysiology of the decision making process, we discuss the neural correlates of the modulation mechanism outlined in this section.

2.4 Neural correlates of value modulation and value computation

Consider a decision-maker who consumes in environment e with corresponding known distribution $f_e(h)$. Our result suggests that consumption is regulated via two different processes. One 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 health at $E_e[h \,|\, h \in H_e]$ and triggers consumption only when the taste representation θ offsets the health concern. In a bar, it captures an unhealthy environment ($E_e[h \,|\, h \in H_e]$ is high), and tends to trigger abstinence (θ is likely to be below $\alpha E_e[h \,|\, h \in H_e]$). It takes the form of a *rule-based* process that recommends an action based on contextual information only (here $E_e[h \,|\, h \in H_e]$). The other process represents the exact high-order characteristic of the item to consume h. It is characterized by a costly computation of h leading to an efficient 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. We are therefore looking for: (i) system(s) that implement the rule-based process, (ii) system(s) that implement the cognitive process,

and (iii) system(s) that operate the switch between the two.

Previous 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 –playing the roles of systems $\mathcal{C}$ and $\mathcal{M}$– are part of the cognitive process.

In parallel, several studies document both the ability to incorporate environmental features into value and to switch from rule-based effortless to cognitive effortful processes, the two other key elements to implement our mechanism. Recent 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)). Rudorf and Hare (2014) in particular found that value reflected in vmPFC activity varied 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 and actions are learned over time and how they are represented in each consumption episode. In particular, we should observe different activity patterns in OFC/vmPFC regions when subjects are asked to consume in different environments as a function of their prior experiences. Said differently, activity in those regions should reflect $E_e[h \mid h \in H_e]$ as e changes.

Other studies have identified key regions involved in rule setting and behavior adjustment. In particular, the medial frontal cortex (MFC) is involved in motivation of behavior (Rushworth et al. (2007)) and is known to participate in action monitoring to prevent undesirable actions (Bush et al. (2000), Paus (2001), Ridderinkhof et al. (2004)). Also, MFC has been shown to be involved in representing rule-context associations to implement executive control in lPFC (Kouneiher et al. (2009)). Converging evidence supports the view that MFC and dlPFC achieve jointly cognitive control, with MFC responsible for monitoring performance and dlPFC responsible for adjusting behavior.[12] This is consistent with

[12]Moreover, posterior lateral PFC regions have been found to select decisions as a function of contextual information while medial lateral PFC regions motivate decisions based on the temporal episode in which decision-making takes place (Koechlin et al. (2003)).

both the known anatomical and functional connections between the two systems (Petrides (2005), Taren et al. (2011)) and their documented joint contributions to cognitive control (Botvinisk et al. (2001); Botvinik (2008); Kerns et al. (2004); Koechlin and Summerfield (2007); Koechlin et al. (1999, 2000)).

Taken together, the evidence suggests that a rule-based process is in place to represent contextual information and intrinsic motivation. The process features PFC/MFC regions constructing and representing high-order information that is passed to vmPFC/OFC regions. Furthermore, systems involved in the rule-based process are functionally connected to those involved in the cognitive process used to adjust behavior according to task demands. More precisely, dlPFC selects which information is sent: a contextual (default) information independent of the current episode through the costless rule-based process, or a precise information about the food to be consumed through the costly cognitive process. This general description is in line with recent studies showing that the decision to engage in cognitive control rather than rule-based control is driven by expectations about prospects and the eventual selection involves dlPFC (Dixon and Christoff (2012), Bahlmann et al. (2012)). It is also consistent with our current understanding of how dietary 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)). Finally, it is in line with recent evidence showing that context is not only represented in vmPFC but also in the degree of connectivity between vmPFC and dlPFC (Rudorf and Hare (2014)). This evidence further suggests that dlPFC may be responsible for triggering the rule-based process necessary to compute value within the context, and for later operating the switch to the cognitive process when contextual information is insufficient.

Overall, our model predicts that, for a given environment e and a distribution $f_e(h)$, the rule-based process constructs the representation of the taste attribute θ and the contextual information $E_e[h \mid h \in H_e]$, anticipating that the exact h is not represented when the environment is e and the true health is $h \in H_e$. The final decision involves the comparison of these two quantities. We expect that the construction of $E_e[h \mid h \in H_e]$ involves PFC/MFC regions and is reflected in the activity of vmPFC/OFC regions. For instance, fixing the taste parameter, we expect the activity to be lower in an unhealthy environment than in a healthy one, following the construction of a worse health estimate (higher $E_e[h \mid h \in H_e]$). Our model also predicts that dlPFC will switch to a cognitive process when $h \notin H_e$ (an unexpected event occurs), as reflected by a higher activity in dlPFC and the general patterns observed in [HCR].

It is also worth noting that intuitive alternative models would yield radically different

predictions. In particular, if we assumed that system $\mathcal{C}$ did not make inferences from an absence of modulation, decisions would be context-independent at equilibrium. Modulation would occur when $h > \eta(c, \alpha)$ and would not occur otherwise, leaving no role to the effect of the environment. 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 the existence of uncertainty and the ability to make inferences.

3 Direct implications of the theory

3.1 Pointers and distractors

One key element of our theory is that modulation requires costly attentional effort. In the absence of a cost and 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 computing goal value. It is obviously difficult for an experimenter to estimate the cost of modulation. On the other hand, it is possible to study experimentally the effect of *variations* of this cost. The exercise can be performed across individuals. For example, a higher IQ or a greater capacity to focus attention should translate into a lower cost of modulation.[13] 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, asking the subject to multi-task, or emphasizing the health information of each product. The basic model has clear comparative statics predictions.

Corollary 1 - 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. Therefore, $d\eta/dc > 0$ means that modulation is more prevalent the lower the cost of exercising it. Similarly, $d\eta/d\alpha < 0$ means that modulation is less prevalent the lower the importance of healthiness compared to taste. These comparative statics are straightforward and not highly surprising. They are also consistent with existing neuro-experimental evidence. For instance, Hare et al. (2011) study the effect of health cues on the integration of health 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). Under that interpretation, their behavioral and physiological observations

[13] Recent evidence (Schmidt et al., 2018) also suggests that individual differences in the neuroanatomy of the dlPFC and the vmPFC predict differences in the ability to exercise self control.

are consistent with the predictions of Corollary 1. Indeed, the study reveals that with the help of health cues tasty but unhealthy snacks are avoided more often and dlPFC is more strongly activated.

Marginal effects can also be tested in a controlled setting by suitably selecting and manipulating pointers and distractors.[14] According to Corollary 1, there are increasing returns in lowering the cost of modulation ($d^2\eta/dc^2 < 0$). Also, as individuals become more and more concerned with the health component of the good, value modulation increases but at a decreasing rate ($d^2\eta/dcd\alpha < 0$). Finally, subjects on a diet or with a delicate physical condition engage more in value modulation ($d\eta/d\alpha < 0$) but, again, at a decreasing rate ($d^2\eta/d\alpha^2 > 0$). More generally, the result shows that tasks which are orthogonal to the consumption decision (pointers, distractors, irrelevant tasks) can alter the benefit of modulating the goal value and, through this channel, affect the final choices.

3.2 Environmental cues

Traditional models of self-control 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 on the behavior of subjects is an exogenous feature of the model. Our model proposes an endogenous relationship between environment and consumption.

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.*

One way of thinking about environmental cues is in terms of the information they provide about the distribution of the high-order attribute from which the good is drawn. For example, if I am 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 I am offered them at a friend's dinner party. Under this interpretation, the environment in our model has an *endogenous* effect on the behavior of subjects. Indeed, for the optimal transmission of the high-order signal, not only the health realization (alcohol and fat content) but also

[14] Testing for marginal effects requires the use of cardinal measures. For instance, we can use the number of distractors to attend to as a proxy for c. We can also use indicators such as blood pressure and cholesterol levels as a proxy of how much weight is allocated to health attributes.

whether the subject typically faces healthy or unhealthy goods in this environment (bar vs. friend's house) matter. In our example, a cocktail at a bar may trigger a different response than *the same* drink at a friend's party. This results from the fact that the communication strategy of system $\mathcal{M}$ depends on its expectation regarding health, which in turn affects the interpretation by system $\mathcal{C}$ when no evidence is transmitted.[15] 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.[16] Interestingly, these decisions are also accompanied by different neural responses, which means that the theory has new testable implications at two levels, behavioral and neural. The effect of the environment in the choice of subjects has further implications.

Corollary 3 - 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.*

According to this result, not only the amount but even the type of modulation depends on the distribution of the health rating. Formally, 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 simple. 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 in the absence of modulation $\mathcal{C}$ assumes the rating is good (bad) and consumption is likely (not likely) to take place. Therefore, to prevent the individual from consuming highly unhealthy items, there are two possible strategies. $\mathcal{M}$ communicates only bad ratings, in which case the individual is likely to consume unless the rating is disclosed. Or $\mathcal{M}$ communicates only good ratings, in which case 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

[15] Notice the similarities between this result and the recent applications on "rational inattention" (see e.g. Caplin and Dean (2015), Martin (2017), Caplin, Dean and Leahy (2018)) where subjects decide whether to spend costly resources into incorporating signals as a function of the expected informational benefits.

[16] This mechanism may be at play in Hare et al. (2011). Health cues may be used to reassess the distribution of health attributes before any choice is requested. In other words, they 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. This interpretation differs from the cost reduction argument in section 3.1 but provides qualitatively similar results. Both interpretations also predict the same behavioral response and same neural correlates and are therefore consistent with the existing evidence.

the same cost saving reasons. Overall, since information transmission is costly it 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 ($E_{e_1}[h \mid h < 2\eta] < \eta$) and the right graph to e_3, an environment where goods are typically unhealthy ($E_{e_3}[h \mid h > 1 - 2\eta] > 1 - \eta$). With no modulation cost, optimal consumption occurs whenever $\theta > \alpha h$. With a positive modulation cost c, consumption occurs when taste is above a certain cutoff, denoted $\bar{\theta}(h, c)$ and represented in Figure 2 with a bold line. As described in equation (2), the taste cutoff above which there is consumption is αh under modulation and $\alpha E_e[h \mid h \in H_e]$ under no modulation. The key difference between environments is that in the healthy one (e_1), there is modulation for bad ratings ($h \in [h^o + 2\eta, 1]$) and no modulation for good ratings ($h \in [0, h^o + 2\eta]$). By contrast, in the unhealthy environment (e_3), there is modulation for good ratings ($h \in [0, h^o]$) and no modulation for bad ratings ($h \in [h^o, 1]$).[17]

In Corollary 1, we discussed a test of the effects of c and α on behavior. It is possible to induce variations on the environment to test this new aspect of the theory. For instance, one can start an experiment by presenting the goods from which choices are randomly drawn. The experimenter can manipulate the environment by choosing whether the majority of goods are healthy (e_1) or unhealthy (e_3). According to Corollaries 2 and 3, fixing the health of the current item, the amount and type of modulation– reflected both in the choices and the neural activity– should depend on the environment.

[17] There is also e_2, the environment (not shown in Figure 2) where goods have typically medium health ratings (formally, $E_{e_2}[h \mid h < 2\eta] > \eta$ and $E_{e_2}[h \mid h > 1 - 2\eta] < 1 - \eta$). In this environment, modulation occurs for $h \in [0, h^o] \cup [h^o + 2\eta, 1]$.

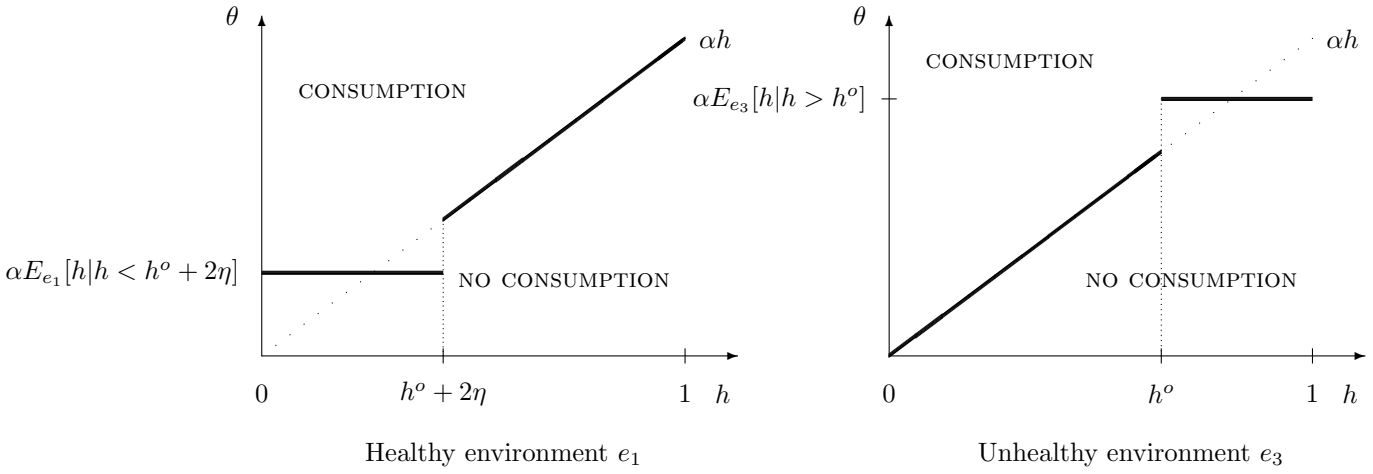

Figure 2. Consumption given taste and healthiness (bold line represents $\bar{\theta}(h, c)$)

In light of the literature previously reviewed (Kouneiher et al. (2009), Taren et al. (2011)), we conjecture that PFC/MFC regions make an initial assessment of the expected high-order information and gradually construct the representation of the contextual information based on the environment unless dlPFC (system $\mathcal{M}$) judges necessary to switch to the cognitive process. When the initial assessment suggests the health rating is likely to be bad, dlPFC triggers the cognitive process only if the actual draw goes against that prediction, and lets the rule-based process compute and send contextual information to vmPFC (system $\mathcal{C}$) otherwise.

Finally, there is an interesting connection between these results and a strand of the literature that links self-control to intertemporal choices. As pointed out by Ainslie and Monterosso (2003) and formalized by Bodner and Prelec (2003), Benabou and Tirole (2004) and Mijovic-Prelec and Prelec (2010), succumbing to temptation today may signal the individual his likelihood of succumbing in the future. If each decision has a small cost but the accumulated stream is large, then by bundling behavior (that is, mentally comparing never succumbing to always succumbing) the individual can achieve self-control. Here we propose a different mechanism that links *indirectly* current choices to future opportunities even when decisions are not inter-temporally connected: the set of upcoming options, defined as the future likelihood of encountering high vs. low unhealthy goods, determines the distribution from which goods are drawn, which itself affects the current decision to modulate value signals and consume.

3.3 Imperfect self-regulation

Our next goal is to understand consumption choices better. We take the perspective of an experimenter who observes decisions over repeated trials and would like to retrieve the consumption patterns of the subject. Recall that $\bar{\theta}(h,c)$ represents the taste cutoff above which the individual consumes (Figure 2). The fact that $\bar{\theta}(h,c)$ is a step function is an artifact of our dichotomous model with binary consumption and binary choice of information transmission. To make the model more realistic, we now assume that the cost of modulation varies across trials, and this may not be directly observed by the experimenter. For example, some days it requires less attention to modulate the signal than others simply because it is easier to focus or there are fewer distractors present. Technically, a variable cost has the property of smoothing out $\bar{\theta}(h,c)$.[18]

To capture variability, we assume that c is stochastic, so that $\eta \equiv \sqrt{2c}/\alpha$ is also stochastic. 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.[19] The realization of c is known to both systems before the modulation and consumption decisions so that, in each trial, the optimal strategy presented in Theorem 1 applies. From the perspective of the experimenter who does not observe c, the relevant variable is the *average* taste cutoff above which the individual consumes. This is computed by integrating over all possible values of c. Denote $\bar{\theta}(h)$ the expected taste cutoff above which the individual consumes (i.e., the analogue of $\bar{\theta}(h,c)$). We obtain the following result.

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 $\bar{\theta}(h) < \alpha h$ for all $h > h_2$.*[20]

An individual with no cost of modulation consumes the good optimally, that is, when $\theta > \alpha h$. With a positive and stochastic cost of modulation, the individual exhibits on

[18] The function would be also smoothed out if we included stochastic changes in preferences, variable consumption quantities z or variable information transmission technologies (e.g., signals that are more or less precise). The results in this section would be similar under these alternative formulations.

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

[20] Under some conditions, $h_1 = h_2$ so that the cutoff separating under-consumption from over-consumption is unique. The conditions include, among others, $\eta \sim U[1/2,1]$ or $\eta \sim U[0,1]$ and $f_e(h)$ either monotonic or symmetric around its inflection point (see appendix C for details).

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, system $\mathcal{C}$ 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 which results in a strictly increasing expected taste cutoff function $\bar{\theta}(h)$. Following our previous example, 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 when $\eta \sim U[0,1]$ and $f_e'(h) \leq 0$ (in which case, $h_1 = h_2 = \hat{h}$, $d^2\bar{\theta}/dh^2 > 0$ and $\bar{\theta}(h) \gtrless \alpha h$ for all $h \lesseqgtr \hat{h}$).

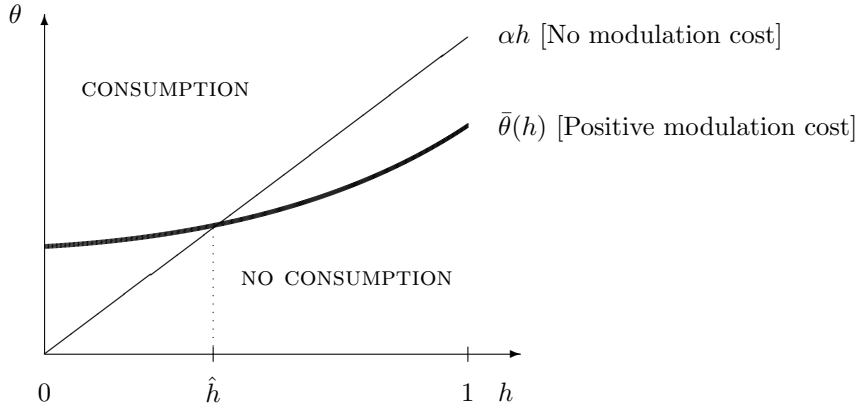

Figure 3. Expected consumption as a function of the health realization

Traditional models of imperfect self-regulation emphasize the natural 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 that the very same mechanism that induces self-indulgence can also result in self-restraint behavior.[21] Notice also that $d\bar{\theta}/dh < \alpha$ is *not* generally true, that is,

[21]The result has some resemblance to Benabou and Tirole (2004)'s model of compulsive behavior. The mechanisms, however, are very different. In their setting, self-control behavior depends on how likely the individual anticipates he will be subject to an inter-temporal conflict, which depends on what he

actual consumption in our model is not always less sensitive to health than optimal. In particular, whenever $h_1 \neq h_2$, consumption will necessarily be over-reactive to health in some interval. We therefore get the paradoxical result that costly attention can generate excessive sensitivity to the unattended variable.[22]

Remember that overconsumption arises when food items are tasty but very unhealthy because system $\mathcal{C}$'s assessment of the expected rating is better than the true rating. During this consumption event, the subject faces a tempting alternative and ends up succumbing to it. This case corresponds to the failure of willpower. Recent studies have shown that exertion of willpower is associated with the activation of dlPFC (Figner et al. (2010); Hare et al. (2009); Crockett et al. (2013)). In our case, dlPFC is not sending information to the goal value system when the rule-based process operates, which is in line with this evidence. Under-consumption arises when food items are healthy but not very tasty. In that case, $\mathcal{C}$'s assessment of the expected rating is not as good as the true rating, and the subject fails to consume an item that is good for his health. Consistently with the case of willpower failure, we conjecture that dlPFC is not activated when this occurs.

3.4 Testing the theory

The model outlined above closely follows the [HCR] experimental paradigm, and we can extend it in order to test the implications of the theory. A key element of our model is the predicted comparative statics across environments. Practically, we can construct two treatments to capture different environments. Formally, treatment T_1 exposes subjects primarily to healthy foods (environment e_1 in our theory) and treatment T_3 exposes subjects primarily to unhealthy foods (environment e_3 in our theory).[23] In order to make participants aware of their environment, we can ex-ante communicate them the set of goods and their frequency of appearance.[24] We can then conduct in each treatment a similar analysis as [HCR], by repeatedly offering choices drawn form the corresponding distribution.

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 dynamic inconsistencies or bounded memory. Instead, under- and over-consumption result exclusively from an imperfectly informed valuation process.

[22] 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.

[23] Naturally, we could also add an intermediate treatment T_2 which exposes subjects primarily to foods with average health ratings (environment e_2).

[24] Health and taste ratings can be collected from individuals in a pre-test stage to personalize the set of goods for each subject. We can have each good in the set presented with equal or different probabilities, and this can be communicated beforehand.

The predictions of Theorem 1 and Corollary 3 can be tested *both at the behavioral and neural level*. In the healthy environment T_1, we should observe similar levels of consumption for goods with medium to very healthy rating. We should also observe comparatively higher activity in dlPFC for unhealthy goods. Conversely, in the unhealthy environment T_3, we should observe similar levels of consumption for goods with medium to very unhealthy rating and higher activity in dlPFC for healthy goods.

Corollary 1 can be tested by adding a cognitive task to be performed while making food choices, as a way to manipulate attention (cost c). The experimenter can also focus the attention of the subjects on the health component of their choices in order to manipulate the importance of the health attribute (weight α). It is also possible to simultaneously combine the manipulation of attention (c and α) and environment (e). Finally but importantly, the predictions of Corollary 2 can be tested by comparing across treatments the likelihood that subjects consume the same item (or two different items with the same health rating) as well as the difference in dlPFC activation.

4 Modulation in other domains

Our theory is based on the taste vs. health self-control paradigm, for which there is evidence both the behavioral and physiological levels. In this section, we extend it to other related paradigms, and discuss how that the principles described in Theorem 1 apply to new situations. We will however restrict attention to cases in which the roles of vmPFC and dlPFC have been established, and to issues that fall directly within the scope of the model.

4.1 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 low-order and a high-order attributes, 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 (Plassman et al. (2010)), we hypothesize that adding a health component (e.g., a medicine or a certain healthy foods) to the decision-problem should trigger a similar trade-off as in section 2.2.

We can model that situation by setting the utility of consumption to $-\theta + \alpha h$ under modulation and $-\theta + \alpha E[h \,|\, h \in H_e]$ under no modulation. An immediate extension of Theorem 1 predicts three regimes. When health benefits are expected to be low, modulation occurs when health benefits are large to promote consumption of the aversive good.

When health benefits are expected to be high, modulation occurs when benefits turn out to be small to prevent the decision-maker from consuming aversive items inefficiently. Last, when health benefits are expected to be moderate, modulation occurs optimally for low or high health realizations. These predictions can be tested in the same way as it was discussed in section 3.4.

4.2 Dynamic inconsistencies

The difficulty for individuals to incorporate high-order motivations may come simply from the problem of correctly evaluating the temporal distance until the realization of the distant component of the good. Although this is not the interpretation in [HCR]'s multi-attribute model of self-control, it corresponds to the more traditional neuroscience approaches to dynamic choices and self-control (see e.g. Luo et al. (2009)). Our basic model can be straightforwardly extended to encompass this alternative interpretation. Indeed, 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 effects are suffered. Under the standard assumption of exponential discounting of payoffs, the analogue of the utility described in (1) is:

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

where the basic and high-order components of the model are now taste and delay, θ and t, rather than 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 equations (4) and (6). In particular, there is information transmission 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, resulting in no modulation and possibly inefficient consumption decisions when the health realization occurs in the distant future. When the harm is likely to be suffered in a near date, $\mathcal{M}$ signals only distant dates and inefficiencies occur when the health realizations occurs in the near future.

As in section 3.3, we assume that c is stochastic. 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 conclusion.

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 5 the analogue of Figure 3 in the (θ, t) space.

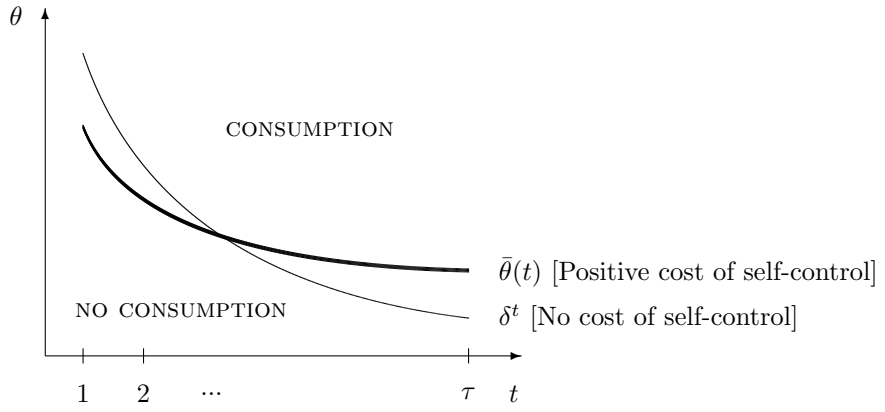

Figure 5. Expected consumption as a function of the *delay* realization

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 when the cost of modulation is positive exhibits the most basic property of hyperbolic discounting: consumption is more frequent than in the exponential case 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)). However, notice that pure hyperbolic discounting requires decreasing impatience at all dates, that is, a marginal rate of substitution that falls monotonically between *all* consecutive periods. Following the argument in section 3.3, the property always holds in our model for extreme dates (t close

to 1 vs. t close to τ) but not necessarily for the others. Therefore, for some intermediate time intervals, discounting may exhibit the opposite increasing impatience. It would be interesting to test experimentally this prediction.

Even though the specific paradigm outlined in this section has not been studied in the literature, recent neuroimaging studies suggest that similar regions and processes as those detailed in the previous sections are involved. In particular, time discounting studies show that 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); McClure et al. (2007)). In apparent contradiction, according to Kable and Glimcher (2007) those limbic reward areas are also encoding delayed values. 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, that is, 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 via a rule-based process are likely to result in significant inefficiencies.

Naturally, the logic of this section also applies to decisions with a negative "low-order" immediate return and a positive "high-order" future benefit enjoyed at an uncertain date. In that 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.

5 Behavioral disorders

Our study looks at "rational self-control," in the sense that a lack of self-control results from a rational trade-off between costs and benefits. If vmPFC and dlPFC play their intended role, self-control is exercised only under certain circumstances. In some cases, however, these regions exhibit dysfunctions. Patterns of activation are different from what we would expect and behavior becomes anomalous. In this section, we address such dysfunctions by altering the basic model. We derive new behavioral predictions resulting from changes in modulation properties.

5.1 Modulatory dysfunctions and eating disorders

Neuroimaging studies (Brooks et al., 2012) show that eating disorders are tied to failures of self-regulation and to an imbalance between mesolimbic regions (ventral striatum, which has an activity correlated with vmPFC) and prefrontal regions involved in cognitive evaluations (dlPFC, MPFC, OFC and ACC).[25] Patients with Anorexia Nervosa (AN) show low stimulation in mesolimbic regions and high stimulation in prefrontal regions associated with a strict refusal to eat whereas patients with Bulimia Nervosa (BN) exhibit the reverse pattern predicting 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.[26]

In this section, we offer a model of suboptimal self-regulation by assuming that individuals have *incorrect perceptions* of the environment. Formally, we assume just as before that h is drawn from environment e with a distribution $f_e(h)$ in $[0,1]$. However, the individual perceives that it is drawn from environment p with a distribution $f_p(h)$ in $[0,1]$. 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 $\mathcal{C}$.[27] As in section 3.3, 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 $h \in [0,1)$.*

According to this result, an individual with unrealistically positive prior beliefs about the health rating of goods will over-consume them, even when the health realization is not as good as expected whereas an individual with unrealistically negative prior beliefs will under-consume them even when the health realization is not as bad as expected. Formally and as graphically depicted in Figure 4, 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 $h \in [0,1)$.

[25] As will be discussed later, similar structures are implicated in the case of drug and behavioral addictions (Reuter et al. (2005)).

[26] 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)).

[27] 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.

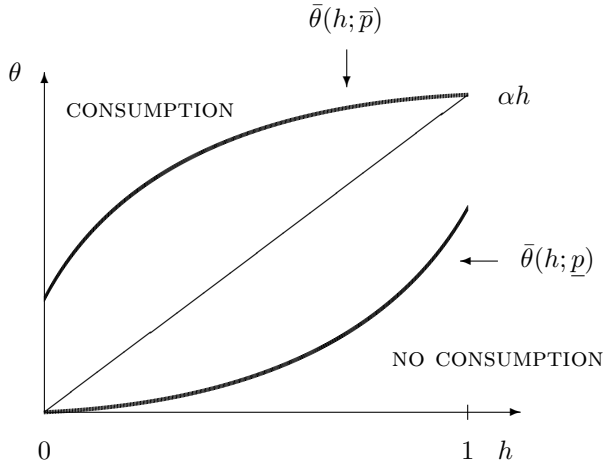

Figure 4. Expected consumption with incorrect perceived environments $\overline{p}$ and $\underline{p}$

It is crucial 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 form Figure 4, consumption is decreasing in h in both cases. An interior realization of h is an extremely low perceived probability event under either $\underline{p}$ or $\overline{p}$ but 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–. The reason is simple. Consider $h \in (0,1)$. As a function of the attention cost c, system $\mathcal{M}$ will sometimes modulate the signal and sometimes not. Modulation triggers optimal consumption by system $\mathcal{C}$, that is, whenever $\theta > \alpha h$. No modulation triggers highly optimistic beliefs and always consumption under $\underline{p}$ or highly pessimistic beliefs and never consumption under $\overline{p}$. The final expected consumption is an appropriately weighted combination of the choices under modulation and no modulation. Overall, the result shows that prior beliefs are key in shaping modulation and consumption. Incorrect perceptions can have an impact in choice not because realizations are incorrectly interpreted, but because information is not transmitted when it should be. This also means that making individuals aware of h, the health effects of the good they are considering, is not enough to prevent suboptimal choices: as long as perceived environments are incorrect, information transmission (and therefore decisions) will be distorted.

The findings are consistent with the existing evidence on AN and BN. These two conditions are characterized by extreme behavior patterns in which subjects systematically under- and over-consume, respectively. In our model, these behaviors emerge for subjects

whose prior belief is that all goods are 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. As developed above, this prediction is consistent with recent findings that suggest that eating disorders emerge from a dysfunction of the processes in charge of representing the attributes and the contextual information. AN patients compute low taste attributes and act according to a rule-based mechanism that represents an excessively unhealthy environment, thereby triggering under-consumption. This is reflected by a low activity in the mesolimbic regions (low θ) and a high activity in regions involved in rule-based processing (high $E[h]$). BN patients compute high taste attributes and act according to a rule-based mechanism that represents an unusually healthy environment, thereby triggering over-consumption. This is reflected by a high activity in the mesolimbic regions (high θ) and a low activity in regions involved in rule-based processing (low $E[h]$). By focusing on the effect of health expectations on choices, our model captures the dysfunction of the rule-based process.[28] Our model also predicts that the behavior of AN and BN patients should not be affected by providing information about the true health consequences of a good. According to our model, 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.

Testing for these effects would involve replicating the experiment detailed in section 3.4 across populations with different eating disorders. The presence of a biased perception could be detected by providing all participants the same objective details about the health attributes of options before asking them to rank items for healthiness. We could then compare the distribution of health ratings across populations. More importantly, we could correlate neural activity and behavior. According to our theory, the absence of dlPFC activity in AN patients should be associated with no consumption whereas in the case of BN patients no modulation should always result in consumption.

5.2 Addiction

We now extend the basic model to account for the possibility of habit formation or inter-period consumption externalities. The model presented here is intentionally simple to be able to study the direct implications of the self-control mechanism discussed in this

[28] A richer model of expectations regarding the taste parameter would be necessary to also capture the dysfunction occurring in the mesolimbic regions.

article.[29] Suppose that the individual chooses consumption during two periods, 1 and 2. In period 1, the individual's utility is given by (1) just as in the basic model. 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. In keeping with the most traditional definition of addiction and habit formation (Becker and Murphy (1988)), we assume that:

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

where $p < 1$ and $p + q > 1$. In words, if the individual abstains in period 1 ($z = 0$), his period 2 utility remains unchanged. If the individual consumes in period 1 ($z = 1$), then the total utility of consumption in period 2 is reduced ($u_{11} < u_{01}$) but the marginal utility is increased ($u_{11} - u_{10} > u_{01} - u_{00}$). Notice that the individual is subject to "craving", that is, a disutility of abstaining in period 2 after having consumed in period 1 ($u_{10} < u_{00}$). Also, craving is increasing in the realization of taste θ.

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. Also 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 (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, self-control dominates no self-control when $V_1^+(h) > V_1^-(h)$, which translates into:

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

[29] 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 future benefits (the direct utility from consuming an addictive good in the future) and future costs (its health consequences), that is, whether those may be independently modulated.

and we obtain 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). The fact that the marginal effect of taste in current consumption increases with past consumption ($p+q > 1$), also implies that the incentives to modulate the signal decrease with past consumption. Indeed, increasing the marginal value of taste makes consumption more desirable, which implies that spending the cost of knowing the exact health realization is less valuable (since it has a lower impact on decision). Overall, the set of health realizations for which there is no modulation expands. Moreover, as the individual is exposed to an addictive good, his decision-making may switch from a regime in which modulation is optimal at the top or the bottom, to a regime in which modulation is never optimal. The direct consequence of both effects is that blissful ignorance of health consequences of consumption 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 costly signal modulation.

Interestingly, the current evidence on the neurobiology of addiction points to the importance of both vmPFC and dlPFC, our main candidates for systems $\mathcal{C}$ and $\mathcal{M}$, respectively. The mechanisms underlying addiction are intricate. However, recent studies show that addiction disrupts PFC functions that are fundamental for regulation and decision-making (Goldstein and Volkow (2011)). In particular, activity in vmPFC decreases with the severity of addiction. This pattern of activity is consistent with our modeling of addiction: a given item becomes less desirable when it has been consumed often in the past. Hypoactivity of dlPFC is also noted and interpreted as a deficiency of inhibitory control in drug-addicted individuals. Our model suggests that hypoactivity may well be the natural consequence of the interplay between the two systems, rather than the result of an impairment due to drug exposure.

The result also suggests that helping the subjects' attention (i.e., decreasing c) should enhance the incentives to restore 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. Also, an increase in p and q results in less modulation in period 2, that is, over-consumption in period 2 is more severe if the good consumed in period 1 has stronger addictive properties.

5.3 Impulsive behavior

Dysfunctions of the reward system and cognitive processes supported by the dlPFC have been observed in a variety of behavioral disorders featuring abnormal decisions other than consumption decisions. Dysfunctional behavior in those cases tend to feature decisions for which only some attributes are taken into consideration, while other aspects should also be incorporated. Attention and hyperactivity disorders belong to that category: patients 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 dysfunction of the dlPFC (Petrovic (2016)) and there is evidence that stimulation of the dlPFC improves behavior (Siniatchkin (2017)). Such disorder may be simply due to an excessive (outlier) 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.

Patients suffering from borderline personality disorder (BPD) display similar impulsive behavior that affect their relationships, 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), Paret et al. (2016)). Similar phenomena are observed in obsessive-compulsive disorder (OCD) and schizophrenia (Cavallaro et al. (2003)). Overall, 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 are not simply the result of high modulation costs, or at least not in the case of all patients.

In light of our theory, we could design appropriate experimental paradigms in which disorders naturally manifest (e.g. task completion 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 1).

6 Conclusion

We have built a novel theory of self-control based on the neurophysiological experiment of [HCR]. Our model provides new testable implications regarding self-control. We highlight the importance of the environment in triggering value modulation and therefore endogenously affecting consumption decisions. We also argue 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. Finally, our theory sheds light on some related phenomena. For instance, we propose that eating disorders are the result of an incorrect perception of the general health properties of goods. Moreover, we show that decreasing impatience can be rationalized by a positive cost of modulation, and ignorance of health considerations naturally occurs more frequently for individuals suffering from addiction.

Even though the direct conclusions of our theory pertain to the case of self-control (section 3), the broader implications discussed in light of existing behavioral and physiological evidence (sections 4 and 5) suggest that self-control is an illustration of a general mechanism. This mechanism may apply to other choices requiring the aggregation of several attributes known more or less immediately to the goal value system. Note in particular that the vmPFC has been constantly reported to be at the core of the value system while the dlPFC is recruited in complex decisions featuring higher-order concerns. Hence, we conjecture that the present theory could be applied to study other decision-making paradigms for which simple (low-order) and complex (high-order) attributes are relevant. Overall, a non-negligible share of goal value driven behavior of the population may be explained by a common information mechanism.

In our multi-attribute context, an efficient information processing strategy is to allocate resources to better know those attributes only if the expected benefit of such operation exceeds its cost. The mechanism produces choices that, given the true attributes, sometimes reflect only a subset of them. In the context of the paradigms we have focused on, this is interpreted as a self-control problem, a tendency to procrastinate or an impulsive tendency. When this information mechanism is subject to distortions or further constraints, as in some behavioral and mental disorders, the tendency to not reflect all attributes of a decision is enhanced resulting in anomalous behavior from the perspective of an outside observer. Note however that, although behavior may seem impulsive, out-of-control or over-focused on some elements of the decision, it is the result of a constrained optimal and internally consistent information processing. Only, the information technology is anomalous, distortive or excessively costly.

The theory outlined here is closely related to the evidence in [HCR] and focuses on

the modulatory mechanisms involved in self control. We have intentionally left many issues aside to highlight that mechanism. Other considerations may be important in some applications. Notably, these include dynamic concerns in the case of addiction. Also, decision-makers may learn gradually the characteristics of their environment. Such learning can interact with the ability to evaluate costs and benefits of consumption and to exercise self-control. These issues constitute interesting alleys for future neuroscientific and theoretical research.

An interesting path would also be to investigate the paradigm in the less well documented case of children and adolescents. Studies have shown that the 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 take place before dlPFC is mature. Brain plasticity is still not very well understood and only a few studies in children provide findings in food choice paradigms. For instance, van Meer et al. (2017) have reported different dlPFC signals in children compared to adults. Lim et al. (2016) have shown that dlPFC encodes the projected maternal food choice. Moreover, 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 in those subjects.

More generally, the article illustrates the value of a recent methodological approach that combines the constrained optimization tools of microeconomic theory with the insights obtained from neuroscience experiments. Indeed, existing theories of decision-making (temptation, self-control, etc.) are insightful but incomplete. Behavioral economics uses introspection, casual observation and empirical observation of choices to refine those models. However, different behavioral theories can sometimes be constructed to explain the same departure. In our methodology, the discipline of the model comes from the neuroscience experimental evidence. The model has a rich set of testable predictions, both behavioral and neurophysiological, which can form the basis of new experiments.[30] In that respect, this line of investigation provides an argument regarding the value of neuroscience for economics that has not been emphasized much in the recent methodological debates (Gul and Pesendorfer (2008); Camerer (2008)).

Finally, we view this approach as complementary (not substitute) to computational neuroscience. Indeed, one could build a mechanical model where system $\mathcal{M}$ automatically sends health "warnings" to system $\mathcal{C}$ for certain realizations of h. This seems a more plau-

[30]Some contributions using this neuroeconomic theory approach include Brocas and Carrillo (2008), Caplin and Dean (2008), Alonso et al. (2014) and, more recently, Landry (2017), Landry and Webb (2018) and Webb (2018).

sible 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 is supposed to 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 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. Once this is understood, one can think of a mechanical implementation along the computational neuroscience lines described above.

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 $H_e = [0, 1]$).[31] 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:

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

[31] 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:[32] (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:[33]

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

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

[32] 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.

[33] 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

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 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: $h_e^{**} - E_e[h \,|\, h \leq h_e^{**}] = \eta$ and $h_{e'}^{**} - E_{e'}[h \,|\, h \leq h_{e'}^{**}] = \eta$. Suppose $h_{e'}^{**} = h_e^{**} - \delta$ with $\delta > 0$. Then:

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

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

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

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: immediate and therefore omitted.

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

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

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 $\bar{\theta}(1) < \alpha$.

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

Case 1. When $\eta^* = 1$ and $G(\eta) = \eta$, we get:

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

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.

Case 2i When $\eta^* = 1$ and $G(\eta) = \eta$, 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 $\eta^* = 1$ and $G(\eta) = \eta$, 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. This case is similar to case 1. We have $\frac{d^2\bar{\theta}}{dh'^2} > 0$.

Case 3. Assuming $\eta^* = 1$ and $G(\eta) = \eta$, we get:

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

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

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

Case 4.

Case 4i: When $\eta^* = 1$ and $G(\eta) = \eta$, 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 $\eta^* = 1$ and $G(\eta) = \eta$, 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. This case 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 satisfying cases 1 and 3. When the extra property P^* holds, $\underline{h} = \overline{h}$ also for distributions satisfying cases 2 and 4.

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