

# ♪ If you want to be happy for the rest of your life —like what you want, and want what you will like

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**Abstract:** The subject is wants vs. likes (wanting/desiring vs. liking what you experience). Economic choice theory assumes you have a ranking of bundles based on preference (higher-ranked bundles are preferred in that if two bundles are feasible, you choose the higher-ranked of the two). The question is whether this ranking is based on betterment/well-being or based on wants/desires. Two neurological theories of choice are outlined, along with their supporting, and conflicting, neurological evidence: the *error-prediction hypothesis* and the *incentive-salience hypothesis*. Both assume, and the evidence supports, that your ranking is based on wants/desires, not, at the point of choice, on which feasible bundle will increase your WB the most. Both address the issue of whether you will only want what you will like (want the most that which will increase your emotional WB the most) and whether you want everything that you will like. With the error-prediction hypothesis, wants are predominately a function of expected emotional-WB. With incentive-salience, this link is easily broken if you are in an enhanced physiological state (hungry, tired, curious, aroused, etc.) and, before you choose, a cue (signal) for one of the alternatives appears, making that alternative more salient, so you choose it even though you would have liked more the experience of one of the other alternatives. The implications for modeling choices, the pursuit of happiness, and ethics are discussed.

**Keywords:** happiness, wants/desires, likes, the reward prediction-error hypothesis, the incentive-salience hypothesis, choice, the neoclassical choice-model, ranking of bundles, emotional well-being, the mesolimbic dopamine system, the ventral tegmental area, and the nucleus accumbens.

I assume well-being (WB) has two components: emotional WB and life-satisfaction WB, where happiness and pleasurable sensations are types of emotional WB, a definition of WB due to Kahneman and Deaton (2010). I concentrate on emotional WB not because it is more, or less, important for WB than life-satisfaction WB, but because more is known about the neurobiology of emotional WB. That said—however you specifically define WB, the issue here is whether you choose the feasible bundle that gives you the most WB, or whether you choose the feasible bundle that you most want/desire.<sup>1</sup>

Neoclassical choice theory assumes *that at every point in time, you have a unique ranking of all possible bundles. Call this Assumption A.* And, it further assumes your ranking is based on your preferences in that you *prefer* higher-ranked bundles to lower-ranked bundles, where *prefer* means if two bundles were available, you would choose the higher-ranked bundle. Call this **Assumption B**.

Consider two possible variants of Assumption B:

*Assumption B1: Experiencing a higher-ranked bundle is better for you (provides more WB), from your perspective, than experiencing a lower-ranked bundle, so you choose it.*

Or

*Assumption B2: You want/desire a higher-ranked bundle more than a lower-ranked bundle, so you choose it.*

And

*If B1 and B2 are both assumed the two rankings must be the same.*

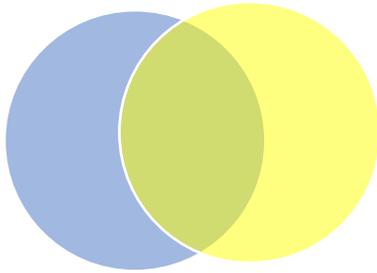
Assumption B1 assumes your ranking of bundles is based on *betterment* in that you are, from your perspective, better off (have greater WB) the higher the rank of the bundle you experience. That is, you will *like* more the experience of a bundle the higher its rank. In contrast, Assumption B2 assumes your ranking of bundles is based on *wants/desires* in that higher ranked bundles are desired more—they are more wanted.<sup>2</sup> The distinction between B1 and B2 is the distinction between getting what you want and experiencing what you will like. The blue area in Figure 1 represents bundles you want more than you will like, the yellow area bundles you like

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<sup>1</sup> Of course, choice might be based on neither of these two criteria, but that's a topic for a different paper.

<sup>2</sup> "Craving" is a synonym for wanting and desiring, a synonym with a negative edge, a synonym that Buddhists use to draw a critical distinction between wanting and liking (see, for example, [Stephen Batchelor](#) 2015). A Buddhist view is that getting what you want/crave does not increase WB.

more than you want. The overlap is bundles you will like to the same degree you want. The degree of overlap is an empirical question.



**Figure 1: Bundles wanted more than liked and liked more than wanted.**

Having two different rankings: one based on betterment and a different one based on wants violates Assumption A: you would not have a unique ranking.

For the purposes of this note, assume, as is standard in economics, that you will choose your highest-ranked feasible bundle. Accepting that here, without question, allows us to hone in on the distinction between wants and likes.

I investigate the neurological evidence, along with the two main neurological theories (*The reward prediction-error hypothesis* and the *incentive-saliency hypothesis*), on the extent to which you only want what you will like, and the extent to which you only end up liking what you first wanted. Whether these two assumptions hold is an empirical question.

But—many economists take as given, either B1, or B2, or both, or are vague. Which assumption is correct, or closest to correct, impacts on how to best model choices, impacts on ethics, and impacts on pursuing happiness (trying to increase your emotional WB)—the distinction between B1 and B2 is important for non-economists as well as economists—why we do what we do, and its implications are important. If, for example, B2 is correct and your B2 and B1 rankings do not line up, you are not necessarily maximizing your WB, even though you might think you are. And, a model that assumes B1 would explain your choices less well than a model based on B2. These issues are critical for those who think the moral objective is to increase WB (welfare economists and other welfare consequentialists). If your choices are driven by wants, and wants don't line up with likes, paternalistic intervention could increase your WB (make you happier).

While welfare economists and other welfare consequentialists assume the moral objective is to maximize overall WB (well-faring), some ethicists ([Peter Singer](#) being a well-known example) believe the moral objective is the fulfillment of wants and desires.<sup>3</sup> If the goal is happiness, the goal is liking what you experience, not want fulfillment. So, whether your ranking is based on wants, or likes, is important.

Looking ahead, the neurological pathways for wanting and the neurological pathways for liking, while related, are different, and a considerable amount of evidence indicates that wants and likes often do not line up. There is agreement (in neuroscience) that choice is driven, at the decision point, by wanting, not liking. The debate in the neurology-of-choice literature is whether wants are predominately determined by how much you expect you would like each of the alternatives, or whether your link between wants and expected likes is sometimes (or often) broken, broken when cues are encountered when you are in an enhanced physiological state (tired, hungry, aroused, curious, angry, etc.) A cue is a potentially-influential observation that occurs right before you make a choice. Many cues are consciously observed (you hear the phone ring), but some cues you only record subliminally. For Pavlov's hungry dogs, cues included bells that rang before the food arrived. For a thirsty you, a cue could be you seeing someone enjoying a Pepsi rather than a Coke. Given you are thirsty, and you see, at the next booth, someone enjoying a Pepsi, you want the Pepsi, and order it even though you would have liked the Coke more. You don't even have to be aware that you saw that person drinking that Pepsi.

## Choosing/seeking, pleasure, and the mesolimbic dopamine system (the MDS)<sup>4</sup>

Economists use the word “choose”, neuroscientists often use the word “seek”. While we often like what we sought/chose, and seek/choose what we will like, you can choose what you will not like, and like what you did not pursue. Choosing/seeking is behavior; liking, by itself, is

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<sup>3</sup> For Singer, the objective is to fulfill an individual's *interests*, and this is achieved by giving people what they prefer, even if it involves less pleasure and more pain, or even less emotional WB. Singer defines preferences as “wants, needs, and desires” (Singer 2011) Singer does not advocate for the maximization of aggregate WB, so while he is consequentialist, he is a *preference-fulfillment consequentialist* (a *preference utilitarian*), not a WB utilitarian nor a welfare consequentialist. Singer is the most famous and infamous living utilitarian.

1. <sup>4</sup> For a short introduction to the brain see “Perception Lecture note: the brain ([David Heeger](#) 2014). The notes include great pictures and diagrams—a nice introduction to the brain.

not a behavior; it's a feeling. (When I throw the ball, Giacomo (my dog) seeks it (chooses to pursue it); at academic cocktail parties, many of us seek the wine rather the soda—what we seek is a choice.) The path you choose is the result of seeking, **not** necessarily liking.

Meso is Greek for middle, the brain's approximate center. Limbic refers to the part of the brain that includes the under chamber (the hypothalamus), the seahorse (the hippocampus), and the almond (amygdala).

You have clumps of neuron cell bodies in the VTA (ventral tegmental area) of your brain (Fig. 1). ["Ventral tegmental", from Greek, means "belly-covering"; it's the area of the brain that sits on the floor of the midbrain, covering its belly.] These neurons have long axons that project into your prefrontal cortex and nucleus accumbens. Since they all emit dopamine, the clumps and their axons are the *mesolimbic dopamine system*, the MDS.

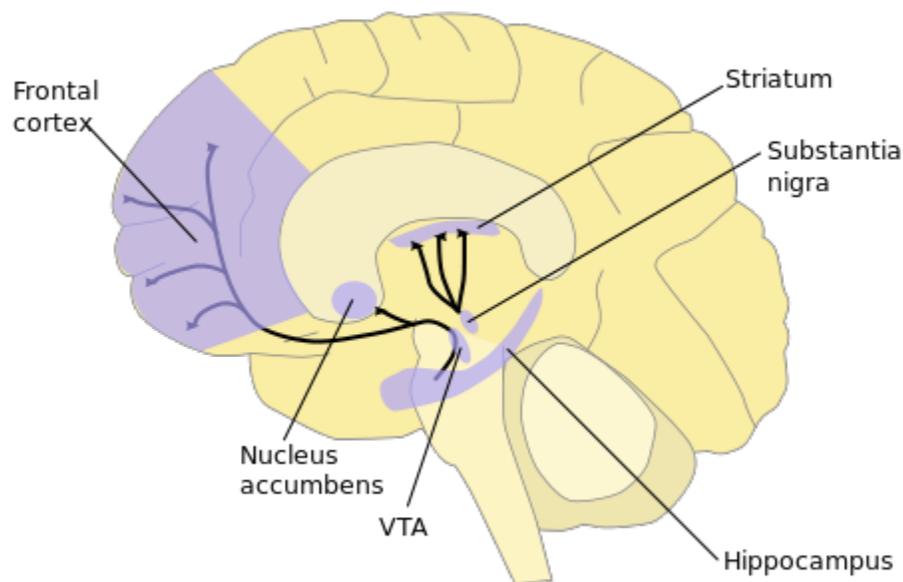


Figure 2: The mesolimbic dopamine system (Kringelbach and Berridge 2012)

When these neurons fire, their axons<sup>5</sup> (the black line starting in the VTA that branches out (in purple) when it enters the frontal cortex) release dopamine in the frontal cortex. If a rat pushes a

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<sup>5</sup> Every neuron (neuron) has a transmission tower called an *axon*, and one or more antennas (receiving towers), called *dendrites*. If enough neurotransmitters land on a neuron's dendrites, the neuron fires (an electrical impulse travels down the neuron to its transmission tower (the axon)), and the end of the axon (the axon terminal) shoots out neurotransmitters that attach to the dendrites on nearby neurons.

reward-less bar, but the researcher electrically stimulates the rat's mesolimbic dopamine axons using electrodes implanted in those axons, dopamine will be released in the frontal cortex, causing the rat to quickly learn to keep pushing the bar, sometimes repeating it until it falls over in exhaustion—the rat will continue to push the bar even if it causes a painful electrical shock. However, if the rat is given a drug that blocks the effect of the researcher-released dopamine,<sup>6</sup> the rat never learns to push the bar. If the bar press is paired with a reward, and the rat has been given a dopamine blocker, the rat never learns to push the bar, even though it likes the reward.

In a recent study published in the journal *Nature* (Pessiglione et al. 2006), human subjects had to quickly and repeatedly choose between two symbols on a computer screen. After each choice, the individual was shown whether they had won £1. The probability of winning was always higher for one of the symbols (e.g. one was 60% and the other 40%), so the object of the experiment was to see how quickly the subject figured this out. Subjects who had taken a dopamine-enhancing drug were the quickest to learn, subjects on a dopamine inhibitor were the slowest, and those on a placebo were in between.

We, and rats, are motivated to seek alternatives that cause the dopamine to synapse with receptive neurons in our frontal cortex. This is good from an evolutionary perspective if the actions that produce a dopamine rush are actions that increase reproduction. The pursuit of sex releases dopamine. Cocaine, nicotine, and methamphetamines are fast ways to cause the dopamine to flow, causing many of us to pursue these drugs—including lab rats.<sup>7</sup> But snorting coke does not typically confer an evolutionary advantage. [Keith Richards, an affluent father of many, being an exception to this rule.] Cravings for sugar can be as strong as the cravings for cocaine, and seeking sweet and high-calorie foods used to convey an evolutionary advantage. [Note that foods with high concentrations of sugar easily ferment into alcohol.]

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<sup>6</sup> A drug that blocks the receptors on neuron's dendrites from accepting (bonding with) the released neurotransmitter.

<sup>7</sup> Here, "flow" is the firing rate of dopamine neurons (a typical way to measure neural activity). Dopamine's effectiveness is enhanced by how long it remains in the synapses (before reuptake) and by the receptiveness of the dendrites on the recipient neurons.

*How much dopamine is released when you get the reward? How much when an expected reward does not occur? How much when you observe a cue that makes a reward more, or less, likely.*

Initial evidence came from an experiment with monkeys in the 90's (Schultz, Dayan, and Montague 1997, Schultz 1998): if when a light starts flashing the monkey pushes the left bar he gets some juice. While learning the task, the dopamine only flows when he gets the juice (when the reward arrives). However, after he has learned to associate the light (a cue) with the juice, the dopamine flows not when he gets the juice but when the light flashes—when the monkey knows he will get the juice if he pushes the bar. fMRI (*functional magnetic resonance imaging*) indicates it's the same for us: after learning, dopamine isn't released by the reward itself (biting into and tasting a great burger) but prior to you know it will happen, when you realize a desire/want will be fulfilled, not when it's fulfilled.

If, after the monkey learns to associate the light with the juice, the expected juice does not appear when he pushes the bar, there is a burst of dopamine when the light flashes but then the dopamine level drops below baseline right after the juice should have arrived—reducing his motivation to push the bar again. Alternatively, if more than the expected amount of juice is delivered, its arrival causes a second burst of dopamine—increasing his motivation to push the bar again. Dopamine release motivates behavior: one interpretation is if the reward is better than expected, more dopamine flows, if the reward is less than expected, less flows.

Summarizing to here: when the cue for a positive outcome occurs, there is a burst of dopamine and after, when the outcome occurs, there is either a positive burst, a negative burst, or no burst. And, one theory assumes (the error-prediction hypothesis) that the magnitude and direction of the burst depends on whether the outcome was better than expected, worse than expected, or as expected.

One additional wrinkle is important. Imagine an experiment where there is more than one cue (e.g., five different symbols), and each is associated with a different probability of the monkey getting the juice (e.g. 0%, 25%, 50%, 75%, and 100%). When cued, the amount of dopamine that flows depends on which cue is presented: the higher the probability associated with the cue, the more dopamine, indicating, maybe, that the dopamine that flows at cue time signals how much information is in the cue. See Figure 3 from this experiment by Fiorillo,

Tobler, and Schultz (2003) where, for example, the second graph shows the dopamine spike when the cue appears for a 25% chance of getting the juice, and the dopamine spike when the juice then appears. The five figures are for a single neuron in Monkey “A”.

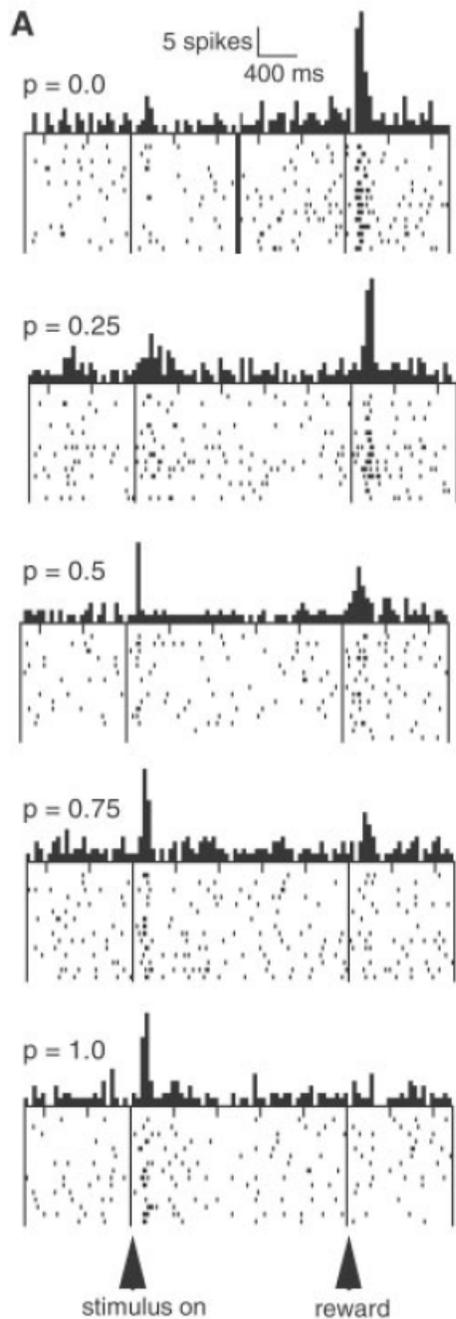


Figure 3 (Fiorillo, Tobler, and Schultz 2003)

When the monkey sees the cue that signals the reward will arrive with certainty, there is a big spike when that cue appears, but no spike when the certain reward arrives (the 4<sup>th</sup> graph).

Alternatively, the lower the probability that the cue means a reward, the smaller the spike for that cue, but the larger the spike when the reward appears.

In a 2008 economics paper, [Andrew Caplin](#) and [Mark Dean](#) suggest dopamine “update[s] the ‘value’ humans and animals attach to different actions and stimuli, causing a change in the probability of choosing each action.” That is, the sequence of dopamine release associated with an outcome is a measure of the value of the outcome relative to its expected value. A caution: here don’t interpret “value” to necessarily mean providing pleasure; a better word would be “reinforcing”, as in choices that result in the release of dopamine are likely to be repeated; the choice might, or might not, produce pleasure. We now turn to that issue.

### Wanting versus liking:

It has been known for over sixty years that releasing dopamine causes wanting/seeking, and this led to the reasonable conjecture that dopamine release causes both wanting and pleasure (liking). If the conjecture is correct, it would mean. neurons are both wanting and liking neurons. This conjecture was accepted as true. If true, Assumptions B1 and 2 line up.

However, findings over the last few decades by the neuroscientist [Kent Berridge](#), and others, contradict the conjecture. Based on their findings, the neural pathways for liking are separate from the neural pathways for wanting, and choice is driven by wanting rather than liking. That is, choice is based on Assumption B2 (based in desire/wanting, not necessarily betterment (B1))

The Berridge lab separated liking a food from wanting a food by measuring liking (or disliking) in terms of facial and mouth expressions (lip smacking vs. yuck face). [The idea of using expressions to measure emotions goes back to Darwin’s 1873 book, [\*The Expression of the Emotions in Man and Animals\*](#).] If you block or destroy the relevant dopamine axons, a rat will not seek food: it will starve rather than walk across the cage for food. But, the rat will eat with relish a sweet food placed on its tongue, expressing all the gestures a hungry carnivore exhibits when enjoying a cheeseburger—the food is liked but was not wanted. In rats whose dopamine axons have not been severed, stimulating these axons causes more dopamine to be released, which causes the craving for food to increase, but not the liking (no additional the lip smacking).

*Research on humans and animals suggests that liking and wanting [seeking] are mediated by separate circuits in the brain. Berridge and his colleagues have, for example, shown that how much you like a sweet is independent of how much dopamine is flowing. Drugs like antipsychotics that inhibit dopamine activity reduce people's desire for pleasure, but don't make that pleasure less intense (Bear et al. 2007)*

Consider the distinction between the sex drive and the pleasure of sex. As people age, their sex drive (their seeking) diminishes, but when sex occurs it's enjoyed, maybe more than when they were eighteen. Alternatively, consider being an aroused adolescent desperately wanting sex who is taking an antidepressant that blocks the ability to orgasm—an awful, un-liked state.

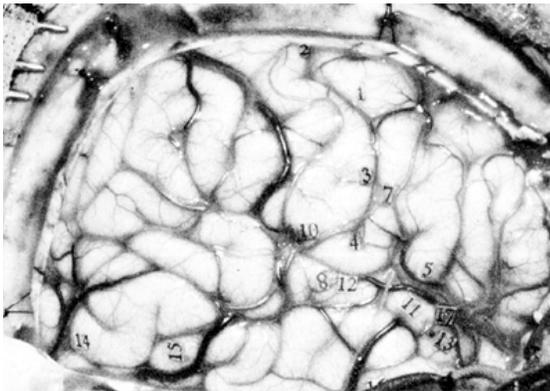


Figure 4: The exposed brain of a conscious subject—Wilder Penfield <http://www.cns.nyu.edu/~david/courses/perception/lecturenotes/brain/brain-slides/penfield-brain.gif>

Researchers occasionally get to cut or stimulate neural pathways in humans, typically to reduce extreme seizures. The surgeons temporarily implant electrodes in the exposed brain to help locate the origin of the seizures—the patient is necessarily awake but feels no pain (the brain has no pain receptors). Different parts of the brain are electrically stimulated and the patient is asked what they feel. If allowed to choose where and how often to self-stimulate, the patients typically stimulate sites that produce an experience they describe as a potential for pleasure that never comes. Sites that produce a pleasurable sensation are stimulated less often.

My friend Bob recently paid a thousand dollars for a power meter for his bicycle (don't even ask). He had to have it but, as he would readily admit, he knew that having a power meter was not going to increase his WB. There is a famous, and made-up, philosophical example of the extent to which desiring and liking can differ. Imagine a highly-addicting drug that when taken causes neither pain nor pleasure, but, once you are addicted, every morning you wake up desiring it, a feeling which is neither pleasurable nor painful. Once addicted, if you don't take the drug within an hour of waking, you experience pain. So, if you take the drug every morning the

addiction and the drug cause neither pleasure nor pain (your emotional WB is unaffected). If the drug were free and if your ranking was determined by wanting/desiring rather than liking, you would choose to become addicted: why? —because you would fulfill a lot of desires for free. The example is from the philosopher [Derek Parfit](#) (1984). You want your seeking and liking systems in sync. You don't want to be like [Marcel Proust's](#) famous fictional self, Charles Swann, in [Swann's Way](#) (1913), who spent his life desiring a woman he knew he would not like.

Neuroscientists agree that wanting is driven by the mesolimbic dopamine system but disagree, somewhat, on how dopamine affects wanting. And, the disagreement is critical to choice theory, ethics, and the pursuit of happiness.

How much you want alternative A versus B could depend on your recollection of how each previously affected your emotional WB, could depend on how much you expect each would affect your emotional WB, or could depend on want/desires that you are experiencing that are unattached to the rewards you expect each alternative would produce.<sup>8</sup> Given that the neuroscientists say choice is based on wants, advocates of choice theories that rank paths based on betterment (experienced WB) should hope that your wants/desires are in sync with your WB expectations, and should hope you experience the WB you expected to experience.

## The wanting/liking disagreement, put simply: the incentive-salience hypothesis vs. the reward prediction- error hypothesis

One hypothesis (the *incentive-salience hypothesis*) is dopamine influences choice by **directly** influencing **wanting**. The other hypothesis (the *reward prediction-error hypothesis*) is dopamine **indirectly** influences choice by influencing **expected emotional-WB**, and wanting is determined by expected emotional-WB. There is data both in support and in conflict with each hypothesis. Much of the data is consistent with both hypotheses. A critical distinction is whether dopamine directly drives learning: the reward-prediction hypothesis says yes, incentive-salience

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<sup>8</sup> Neuroeconomists, and interestingly non-economic neuroscientists, use the word *utility*: separating *decision utility* (the measure (of value) you use to decide which alternative to choose) from *experienced utility* which is how emotional WB is affected by the alternative chosen. Their experienced utility is what I have defined as emotional WB. So, in terms of terminology, they separate what you will like from what you choose. They then separate decision utility into its possible components: *remembered utility* (how it felt last time it happened), *predicted/expected utility*, and *wanting (incentive salience)*.

says no. “Chapter 18: From Experienced Utility to Decision Utility” in the textbook *Neuroeconomics* critiques, compares, and debates the two hypotheses. Interestingly, the chapter is jointly written by Kent Berridge and [John O’Doherty](#). O’Doherty, the director of the Caltech Brain Imaging Center, advocates for the reward prediction-error hypothesis and Berridge for the incentive-salience hypothesis, which he first proposed.

### The incentive-salience hypothesis

This hypothesis is most associated with Kent Berridge and his lab. *Incentive salience* is a fancy term for serious wanting (a strong urge—conscious, or not) as compared to run-of-the-mill wanting. The hypothesis is that your *incentive* is to choose that which is most *salient* (striking, attention grabbing). An initial motivator for the hypothesis was the Berridge finding that rats could be manipulated, by removing or blocking dopamine, to not want what they would like, and manipulated, with dopamine, to want what they will not like, implying that wants are not always based on expected emotional WB. After they discovered that wanting is separable from liking, and wanting is driven by dopamine, the lab turned towards identifying what part of the brain produces liking? Two emotional/pleasure hotspots have been identified.

### The sensation of pleasure

So, if dopamine isn’t the pleasure neurotransmitter, and if the mesolimbic dopamine system, MDS, isn’t the pleasure pathway, what causes the sensation of pleasure? Recent research by Berridge and others has identified two *hedonic hotspots* (the red spots in Fig. 4): “One of these lies in a subregion of the nucleus accumbens called the *medial shell*”, which is a small part of the MDS (see Fig. 11). “A second is found within the *ventral pallidum*, the VP, a deep-seated structure near the base of the forebrain that receives most of its signals from the nucleus accumbens” (Kringelbach and Berridge 2012). The ventral pallidum is between the nucleus accumbens and the amygdala. Axons connect the ventral pallidum and the nucleus accumbens to each other, and both have axons projecting to the orbitofrontal cortex.

## Paths to Pleasure

Pleasure is a complex experience that encompasses everything from anticipation and desire to sensation and satisfaction. So it's no surprise that several brain regions work together to generate this warm glow of good feeling.

### Wanting and Liking

A neural circuit (blue) that begins near the brain stem and reaches out to the forebrain was once thought to be the sole mediator of pleasure. But it is actually more focused on desire. In addition to this pathway, several so-called hedonic hotspots, including two shown here (red), interact to create a sense of liking. A quilt of cortical regions (pink) then translates information received from the "wanting" and "liking" circuits into conscious pleasure and adjusts this feeling based on inputs from other brain regions.

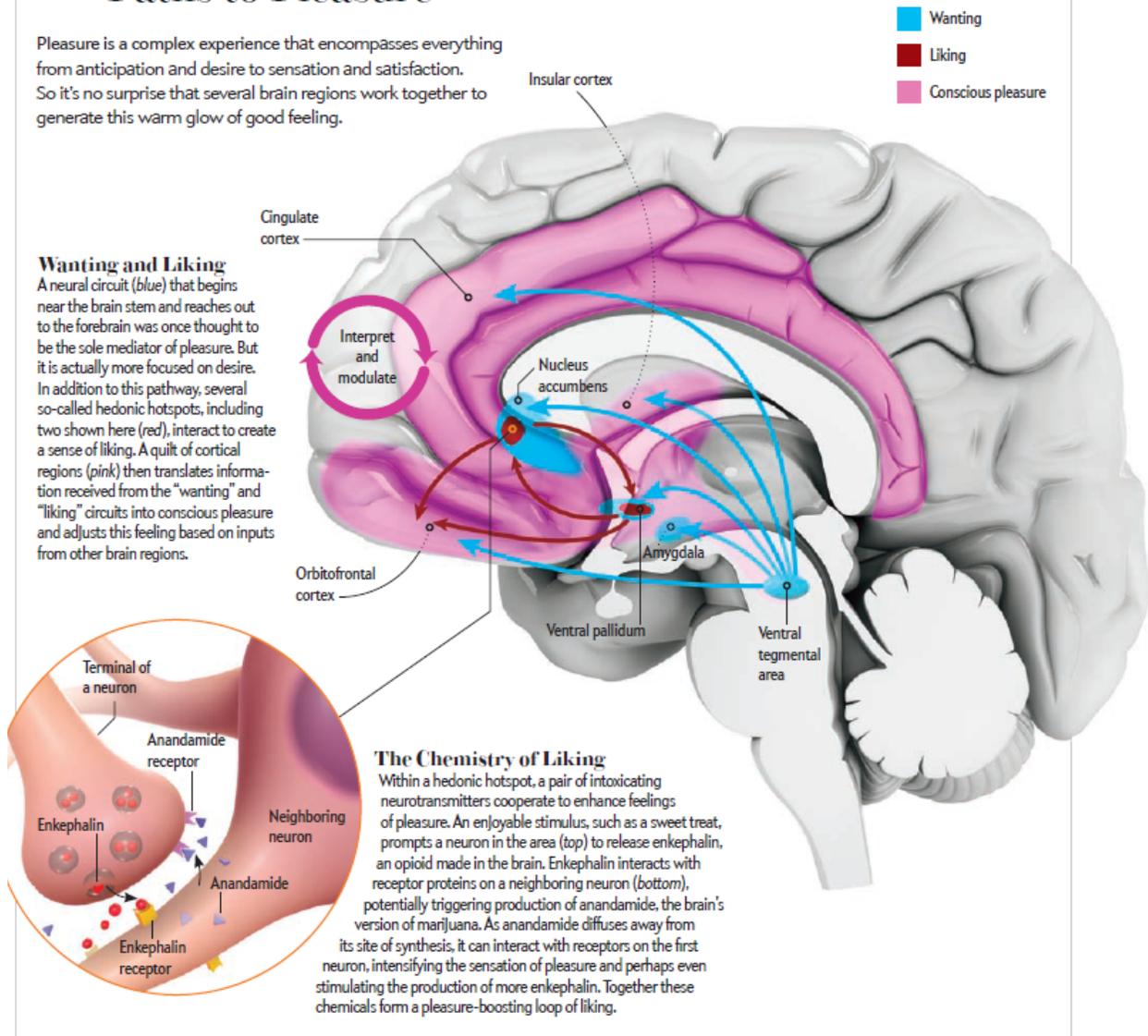


Figure 5: Kringelbach and Berridge (2012)

So, why are these two regions identified as hedonic hotspots? In brains, at least in rodent brains, bathing the neurons in these areas with the neurotransmitter enkephalin makes a sweet taste sweeter, so does bathing them in the neurotransmitter *anandamide*, “the brain’s version of the active ingredient in marijuana. [“Ananda” is Sanskrit for bliss.] The release of enkephalin causes anandamides to flow, which can feedback on the axon terminal causing more enkephalin to flow (a positive feedback loop). Destroying a rat’s ventral pallidum makes a sweet taste

yucky. [Many rats get parts of their brains destroyed in the interests of neuroscience.<sup>9</sup>] Stimulating the ventral pallidum can make something yucky produce pleasure, and the VP and medial shell light up when people experience pleasure. Enkephalin and anandamide are opiates: pleasure is all about bathing the right neurons in the right drugs.

The axons projecting from the VP and the medial shell terminate in the *orbitofrontal cortex* where the experience of conscious pleasure is produced (the *pleasure gloss*). The orbitofrontal cortex “adjusts this feeling based on inputs from other brain regions” (Kringelbach and Berridge 2012). Neural activity in the orbitofrontal cortex (observed by neuroimaging), is highly correlated with the sensation of pleasure, and there is some evidence that stimulating this region can cause you to feel deep pleasure—where do I sign up?

Supporting these findings, some people experience more pleasure than others because they consume more stuff that makes the enkephalin and anandamide flow. Some because of genetics and prior experiences: they have more and better enkephalin/anandamide synapses. A lucky 20% of Americans have a FAAH gene mutation that increases the flow of anandamide.<sup>10</sup> The gene encodes for the production of an enzyme called *fatty acid amide hydrolase*, FAAH, (Duke University 2012, Gunduz-Cinar et al. 2013, Friedman 2015, Dincheva et al. 2015). This enzyme breaks down anandamide. The mutation causes less FAAH to be produced, so less anandamide is broken down, so more of it flows. People and mice with this mutation tend to be less anxious, and recover from bad experiences more quickly.<sup>11</sup> So to experience pleasure, you want parents who were good at experiencing pleasure, and you want experiences that strengthen the pleasure synapses you were born with.

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<sup>9</sup> The pleasure parts of your brain and the pleasure of a rat’s brain are quite similar. Of course, unlike a rat, you have a large cortex, giving you the ability to ruminate about what you feel. But a rat doesn’t need these higher brain regions to experience pleasure, and neither do you. In the 1940’s and 50’s thousands of people got frontal lobotomies (a procedure that intentionally destroys cortex) and while many of them then suffered from bad decision making, they didn’t experience a decrease in their ability to experience pleasures—they still liked food and sex.

<sup>10</sup> The probability of inheriting this gene mutation varies by ethnic group; while it’s 21% for Americans of European descent it’s 14% for Han Chinese living in China and 45% for Yoruban Nigerians (Friedman, referring to data from the [HapMaP](#) project).

<sup>11</sup> Researchers (Dincheva et al. 2015) recently inserted the mutated gene into mice to see how their behavior would be affected. Compared to normal mice they were more adventurous, and had better connections between their amygdala (a fear-processing center) and their prefrontal cortex. Then mice were conditioned to be afraid of a neutral tone achieved by pairing it with an electric shock, they were later all repeatedly exposed to the same tone but without the shock. The mutated mice more quickly learned to not be afraid of the tone. The researchers got the same results when they compared humans with and without the gene mutation.

## The incentive-salience hypothesis assumes dopamine directly affects wanting, not learning

Most neuroscientists, including the incentive-salience advocates, agree that wanting is typically determined by remembered WB and expected WB. Quoting from the text

### *Neuroeconomics*

*That is, we ordinarily desire an outcome to exactly the same degree that we predict the outcome will be liked, and most predictions about future experienced utility [WB] are based on how liked the outcome was in the past.* (Glimcher and Fehr 2012)

[There is a 2012 economic study that supports, to a degree, this conclusion (Benjamin et al. 2012). The study asks hypothetical choice questions; for example, “Would you choose a job that pays \$80K/yr. and you only have time to sleep 7.5 hrs./night, or a job that pays \$140K with only 6 hours of sleep?” After you answer, the researcher asks which scenario would make you happier. The study estimates that in 83% of the respondents chose the alternative that they expected would generate the most happiness. If we make the leap and assume the subjects always chose their most-wanted/desired alternative, the study implies that 83% of the time the alternative the subject wants the most was the alternative they expected to like to most.]

**But sometimes**, according to the incentive-salience hypothesis, your level of wanting/desiring for one of the alternatives is hyped because a cue for that alternative occurs when you are in a physiological state (hungry, aroused, tired, etc.) that increases your receptiveness to the cue. For example, you are thirsty and see someone enjoying a Pepsi, rather than a Coke. When a cue occurs and you are receptive, the dopamine spike is compounded causing you to want the alternative associated with that cue above and beyond what would be warranted by its expected effect on your emotional WB. [Remember that dopamine is released when a cue for the outcome occurs, which is before the outcome is experienced.] The clock striking six is a cue for Giacomo that dinner will soon arrive and a cue for me that wine will soon arrive. A second cue for Giacomo is the smell of his burger cooking. These cues trigger the release of dopamine, directly and excessively increasing our desire for the alternative associated with those cues, making me overly inclined to drink, and Giacomo overly inclined to stand near his bowl.

Experiments in the Berridge lab (Wyvell and Berridge 2000) have measured the joint effect of a cue and physiological receptiveness. “Rats were first trained to press one of two levers to obtain sucrose pellets. They were separately conditioned to associate a Pavlovian cue (30 sec. light) with free sucrose pellets.” A few days later, the rats had different amounts of

amphetamines injected into their nucleus accumbens, “putting the rat’s brain in an elevated state of dopamine activation.” The rats were then set free to press, or not press, one or both of the levers. No sucrose pellets were rewarded or given, but the cue was presented at intervals. All the rats pressed both levers, but pressed more the lever previously associated with the sucrose pellets. When the light went on (the cue), they all pressed the sucrose lever even more (but not the other lever more), and the number of additional sucrose-lever presses increased with the amount of amphetamines the rat had received. Between cues, the amphetamines had no effect on the number of presses, so the amphetamines only influenced cue-specific behavior. Based on a separate test, the amphetamines did not increase liking, so lever pushing was not increased by liking, only by wanting. Summarizing, the cue increased sucrose-lever pushing more, the more the rat’s physiological state was amped up—the cue became more salient the more the rat was amped up.

You might imagine that incentive salience is expected emotional-WB multiplied by some constant  $k$ , so when  $k$  equals 1 you only want what you expect to like, but if  $k$  is more, or less than 1, wanting will deviate from liking. And, cues can affect  $k$  depending on your physiological state. The implication for choice theory is that what you choose will not always make you better off, and this is more likely to occur when you experience cues and are in a receptive physiological state. You often make bad choices when you are tired, aroused, intoxicated, etc., and a cue for the bad alternative appears.

Consider again the 83% estimate noted above, keeping in mind that in that study the choices were hypothetical, there was no cueing (no pictures of comfortable beds were flashed on the screen), and there is a presumption that the subjects were not off their physiological baseline (e.g. tired), making one imagine that in the real world (where people are often tired (or stressed or excited, or curious) and cues are numerous) wanting and expected liking line up significantly less than 83% of the time. Whether getting it wrong, at least 17% of time is a big deal or not is a matter of interpretation. Some choices (like Coke or Pepsi) probably don’t have much of an effect on long-term WB, but some choices (e.g. having a child) do.

In a recent paper, Franz and Christian (2016) proposed a choice model where choice is based in the salience of each alternative (more salient alternatives are ranked higher), so it is a model consistent with the incentive-salience hypothesis. They formulated the model as a way to explain why you have the ranking you have, and, maybe more importantly, why your ranking

changes over time (the salience of an alternative can vary over time even as its effect on your WB remains constant). There is not much literature in economics on the creation of your ranking.

#### The reward prediction-error hypothesis:

This hypothesis, proposed by [Wolfram Schultz](#) at Cambridge, is that dopamine flows when your expectation of future rewards needs correcting. That, is dopamine flows whenever new information arrives that indicates your current expectations are wrong. New information comes in two forms: the magnitude of your last reward, and sometimes with a cue before the next reward is expected to appear. A temporary increase in firing rate (a positive spike) indicates that what you expect in terms of future rewards has been in error and your expectation can be improved by expecting more; a negative spike indicates that what you expect in terms of future rewards was in error and can be improved by expecting less, and no spike indicates no prediction error. The dopamine spike that occurs when a cue appears sends the message that you should expect more, and the magnitude of that spike is larger the more the cue is associated with the reward (see Figure 3). In the absence of the cue, there is no pre-reward spike because there is no basis for correcting your expectations. A positive dopamine spike when the reward arrives signals that the reward was better than expected so you should expect even more going forward. A negative dopamine spike signals the opposite. The hypothesis is that the deviation in the dopamine flow causes recipient neurons in your frontal cortex, and other part of your brain, to start the process of bringing expected WB more in line with the WB you will experience. [Recollect that the axons of these dopamine neurons project widely.] Discussions of this hypothesis typically use the word *learning*: you learn, through reinforcement, better what to expect. When the prediction error is large, there is more to learn.

The primary evidence for the hypothesis that dopamine flow is a gauge of prediction error are the observations, discussed above, that the dopamine spike when a cue is presented is larger the more the cue predicts the reward (there is new information that the reward is more likely). And, the observation that the dopamine spike when the reward occurs (up, down, or not at all) depends on how much the reward deviates from the expected reward. These two observations are evidence for the hypothesis in that they are what the hypothesis predicts. They are not proof because they are consistent with other hypotheses as well, including, arguably, the incentive-salience hypothesis.

A problem with testing the reward prediction-error hypothesis is that while neuron firing rates can be directly observed (what Fiorillo did), prediction errors cannot, so are estimated based on assumptions, assumptions that not everyone accepts. Many studies show that firing rates are correlated with estimates of prediction errors, but no current studies prove that firing rates are correlated with actual prediction errors. Berridge, for example, points out that if the incentive-salience hypothesis is correct, and if lab subjects' psychological states are all the same and constant through all cue/reward trials (e.g., all the rats are equally thirsty, or not), the data produced will be consistent with the reward prediction-error hypothesis and the researcher will mislabel the level of wanting for the level of the prediction error. In most learning experiments, the physiological state is the same across subjects. So, according to Berridge, a distinction between level of wanting and level of prediction error will not appear. A distinction could only appear in an experiment if there was variation in physiological states across subjects (e.g. different degrees of thirst or arousal), and most studies have not included such variation.

A finding in conflict with the reward prediction-error hypothesis is that you can learn in the absence of dopamine (e.g. rats are "able to learn a new dislike for a sweet taste that was originally liked" (Berridge and O'Doherty 2013) even after most of their dopamine neurons are destroyed).<sup>12</sup>

All said, many neuroscientists support the reward prediction-error hypothesis.

An implication of the reward prediction-error hypothesis is that dopamine does not directly drive wanting, but indirectly influences wanting by updating expected emotional WB. With respect to Assumption B1 (bundles are ranked in terms of betterment) and Assumption B2 (bundles are ranked in terms of how much they are wanted/desired/), the reward prediction-error hypothesis, and its supporting data, is consistent with paths being ranked on the basis of wanting/desiring/ (B2), and consistent with desiring based on expected WB. In addition, the hypothesis assumes a neurological mechanism that is continuously using new information to make your expected WB more reflective of what you will actually experience. If the reward

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<sup>12</sup> But it seems that some types of learning do require dopamine (Berridge and O'Doherty 2013).

error-prediction hypothesis is correct, your ranking of paths is based, mostly, on expected betterment.

## Summarizing:

Both hypotheses assume that choice is driven by wanting/desiring, and that wanting is often driven by the level of expected WB associated with each alternative. The link between wanting and expected WB is weakest if the incentive-saliency hypothesis is correct, particularly when cues precede the choice and the chooser is in a physiological state that make her hyper-receptive to the cues. In such cases, the incentive-saliency hypothesis predicts you will often choose something other than what you would have liked the most. In the reward-prediction hypothesis, dopamine drives learning (changing expected values when new information arrives). In incentive-saliency, dopamine drives wanting, not learning.

The neurological data supports the conjecture that choices are based, at the decision point, on wants rather than likes (Assumption B2, not B1). I am no expert in judging between the two hypotheses, but find the incentive-saliency hypothesis appealing and intriguing, more appealing to me, because it is more in line with models and data from psychology that indicate we do often act against our own WB, particularly when we are in an elevated physiological state (for a review, see Morey (2017): Chapters 6-8). It is also in line with literature on addiction that suggests how much you want something does not necessarily reflect how much you will like something. For example, in one interesting study (Leyton et al., 2005),

*cocaine users were given a drug that lowered their dopamine levels. In the lowered dopamine state, cues indicating the availability of the drug were rated as less desirable. When given the drug, however, the users' feelings of euphoria and the rate of self-administration were unaffected. That is, with reduced dopamine, study participants still liked the drug in the same way (reinforcement was unchanged), even though they didn't particularly want it. (Gazzaniga, Ivry, and Mangun 2014).*

Or the reverse, the recovering addict encounters a positive cue while in an elevated physiological state, which makes them temporally want the drug more than they will like the drug. So, they take the drug even though they know that their past use was a mistake, and know that taking it now is a mistake (Bernheim and Rangel 2004)

Also recollect the Buddhist distinction between craving/wanting and WB.

As a choice modeler, and as a welfare economist, I find the incentive-saliency hypothesis both more intriguing and more troublesome because, if it's correct, you and I often do not

maximize our WB. (The reward prediction-error hypothesis is more in line with us each maximizing our WB, so I guess that I should hope the incentive-salience hypothesis, but not the reward prediction-error hypothesis, is eventually rejected. That said, seeing apple carts upset spikes my emotional WB when I do not have too many apples in the game.)

What about economists and ethicists who do not care about WB. If you are an economist who models choices, and if you only want to predict behavior, not caring how an individual's choices affect them, assume B2, it is consistent with both hypotheses, and the data.

If you are an ethicist (economic or otherwise) for which want/desire fulfillment is the ethical objective (rather than maximizing WB) go with B2: it is consistent with both hypotheses (both assume B2), and it implies that individuals pursue your ethical objective (want fulfillment), at least from their own perspective.

Of course, findings in neuroscience are coming fast and furious, and new research could negate or confirm the findings reported above. One possibility is that maybe dopamine plays two roles: in some parts of your brain it might be a gauge of prediction error and in other parts a gauge of salience. Gazzaniga, Ivry, and Mangun (2014) describing a study by Matsumoto and Hikosaka (2009) that recorded firing rates for dopamine neurons in the brain stem:

*One subset of dopamine neurons responded in terms of valence. These cells increase their firing rate to stimuli that are predictive of reward and decrease their firing rate to aversive stimuli (Figure 12.19a). A greater number of dopamine neurons, however, were excited by the increased likelihood of any reinforcement, independent of whether it was a reward or a punishment, and especially when it was unpredictable (Figure 12.19b). The first response class is similar to what would be expected of neurons coding prediction errors, the second to what would be expected of neurons coding salience or signaling things that require attention.*

In closing, I leave it to the reader to decide when, and if, it would be better for economists to be explicit, or not, about what they assume your ranking of bundles is based on. My preference would be for specifying whether preference is based on wants or likes.

If this were a self-help paper,

And your goal is to increase your WB, be aware that your choices are based on your wants and desires, and the alternative you want won't necessarily increase your WB as much as some other alternative, and might even decrease it. So, ask yourself why you want to dump your spouse/job/car for a different model, and question whether the replacement will substantially, and permanently (or at least for a long time) increase your WB. And, don't commit to an alternative when you are in a hyped-up physiological state—you often are—and look for cues

that might affect what you desire. That smile at the bar, when you are intoxicated and aroused, might lead to a WB-reducing choice.

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