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Pain and Pleasure

Murat Aydede
Department of Philosophy
University of British Columbia

If you look at anybody's typical list of emotions, you won't see pains and pleasures among them. Indeed, even among the atypical emotions that people working on emotions regularly cite, pains and pleasures show up only rarely. And yet, very few people will fail to acknowledge the critical, or perhaps the essential, role pains and pleasures play in our emotional lives. This chapter will explain the sense in which pains and pleasures are elementary forms of emotions.

Let's first distinguish pain and pleasure *experiences*, properly so-called, from their *sources* — typically, the physical objects, events, activities, etc., that cause such experiences. Smelling a rose is a pleasure, getting pricked by a rose bush thorn a pain — it is said. But it is *primarily* the experiences generated by these events that are said to be pleasant or painful. These experiences are mental events or episodes caused by various physical stimuli. It is harmless, in fact sometimes quite appropriate, to extend the terms to refer to such stimuli in most ordinary contexts as causes of such experiences. But here we will focus on pains and pleasures as experiences. As experiences, they are presumed to be conscious mental episodes.[1]

Pain

In ordinary parlance, pain experiences are sometimes divided into what may intuitively be called 'sensory' and 'emotional' pains (for example, pains due to a paper cut or sprained ankle versus intense grief or frustration — this distinction is sometimes marked as 'physical' versus 'psychological' pain). Sensory pains are those experiences that involve the (normal or abnormal) activity of nociceptive mechanisms in the nervous system, whereas emotional "pains" don't involve such activity — although sensory and emotional pains seem to share the affective-motivational mechanisms (see below; Eisenberger 2012). The practice of calling negatively valenced intense non-sensory experiences 'pains' are generally avoided in pain science and clinical settings, and I will follow this practice

[1] Positive and negative affect as occurs in pain and pleasure experiences need not be conscious. For the purposes of this chapter, I'll have conscious experiences in mind in what follows except when it matters — see below, and Chapter ?? this volume.

and concentrate on sensory pains.[2] The International Association for the Study of Pain (IASP) defines 'pain' as follows:

> **IASP Definition of 'pain'**:
> Unpleasant sensory and emotional experience associated with potential or actual tissue damage, or described in terms of such damage. (IASP, 1979/2011)

Although there have been minor controversies about proper wording and the apparent appeal to *linguistic* description, this definition has been well received and widely accepted.[3] It captures all the major components of pains.

Sensory. Despite some controversy over the degree of its specificity (Perl 2011, Woolf & Ma 2007, Basbaum 2011), it has been generally accepted that there is a somewhat specialized nociceptive input system from the bodily periphery (nociceptors) to the central brain mechanisms that process somatosensory information about a spectrum of noxious stimuli (mechanical, chemical, thermal) that might cause actual or potential tissue damage. This system and the aspect of the pain experience it subserves is identified with the *sensory-discriminative* component of sensory pain which encodes various sensory qualities, intensity, bodily location and temporal characteristics of noxious stimuli. This system is, to some extent, comparable to other sensory input systems we have such as visual, auditory, gustatory, olfactory and various tactile sensory submodalities. The job of sensory systems is to transduce energy forms impinging on bodily surfaces (receptors) and thereby provide information about a range of physical parameters (stimuli) in the environment of the organism to the central brain mechanisms for further processing. We will use 'sensory' in this relatively technical sense, and 'sensation' to refer to the immediate experiential output of sensory systems registering the detection of relevant range of stimulus features proprietary to specific modalities.[4]

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[2] Examples of 'emotional pains' are typical negative emotions (when intense) such as grief, sadness, dread, anxiety, embarrassment, jealousy, shame, feelings of social exclusion, romantic loss, etc. So, obviously, emotional pains are paradigm cases of emotions. See Corns (2015) for a fairly persuasive argument that the so-called social pain (and the like) is not pain properly speaking — it's a "pain" only by courtesy of sharing the affective-motivational aspect of pain experiences and its underlying mechanisms (see also Eisenberger 2012) — see below.

[3] For a survey of these criticisms and defence of the IASP definition in its historical context, see Aydede (2017). Wright (2011) is also very useful.

[4] Sensory pains are sometimes divided into nociceptive, neuropathic, nociplastic pains — all can be chronic (see IASP 2011, Kosek et al 2016). Melzack and Wall's classic 1996 book, *The Challenge of Pain*, is still good as an entry to the overall organization of the sensory-discriminative aspect of pain.

Unpleasant. Sensory pain experiences normally have also an *affective-motivational* component interfaced with their (nociception-based) sensory-discriminative component.[5] This component gives the normal sensory pain experiences their negative affective (or, hedonic) tone or character. It's in virtue of this component that pains are said to be unpleasant, hurtful, awful, agonizing, bad, or simply, 'painful'. The job of the affective-motivational component is to move the organism to deal with the unfolding (actual or potential) noxious events happening in or around the tissue that is being physically threatened and make the organism learn from its experience about how to behaviorally deal with such stimuli. The neurophysiological mechanisms subserving the affective component of sensory pains are complex and less well-understood relative to our knowledge of the nociceptive systems and are mostly comprised by partially overlapping but nevertheless distinct brain structures such as the midbrain and limbic system structures, basal ganglia, as well as parts of insular, prefrontal, and cingulate cortices. Most of these are phylogenetically older and thought to underlie emotions and motivation (behavior, motor output) as well as certain forms of learning. Thus, (normal)[6] sensory pains have always an affective-motivational component in addition to a sensory-discriminative component. The authors of the IASP definition have decided to use the word 'unpleasant' to denote this negative affective character that immediately modifies or attaches to the pain's sensory component and makes the pain experience directly relevant to motivation and moral considerations.

Emotional. It is an open question what the word 'emotional' contributes to the IASP definition beyond the word 'unpleasant' does. Indeed, there is a long explanatory *Note* added to the IASP definition that reads "[pain] is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience" (IASP, 1979/2011), which makes it sound like a pain experience is an emotional experience in virtue of being unpleasant and nothing else. This interpretation would not be incorrect but may be misleading. Arguably, there is a minimal sense in which any experience with an immediate negative or positive affect (hedonic valence) attached — maybe with some noticeable intensity — is an emotional experience, and

[5] See Melzack and Casey (1968) for the classic statement of the different components of pain. They included a *cognitive-behavioral* component in addition to sensory-discriminative and affective-motivational components. The prevalence and clinical importance of secondary affect (see below) may justify the addition of this component to the overall characterization of pain — see below for more discussion.

[6] This qualification is necessary in light of certain pain syndromes such pain asymbolia as well as pains experienced after certain brain lesions and surgical procedures such as prefrontal lobotomies and cingulatomies, where patients sincerely report pains but do not seem to find their pains bothersome, unpleasant, or bad. Some of these cases seem to involve sensory pains with nociceptive activity with diminished or completely absent affective-motivational component (Berthier et al 1988, Price 2000). Note that the IASP definition excludes pains that are not unpleasant.

thus an emotion. But most pains involve emotion in a more robust sense than this. Consider, for example, that you discover through a painful tactile encounter a developing lipoma under your arm. Your lymph cancer had been in remission for three years. The pain experience you felt upon pressing on the bump under your skin had an immediate negative affect — it hurt. But this experience was clearly much more 'painful' than the momentary physical hurt you felt: it also involved a sudden, deeper, sickening dread that overcame you for fear of the cancer coming back. Several negative emotions are caused here: fear, anxiety, panic, and more. The first brief hurt is not properly described as *suffering*. In the clinical literature, the former brief hurt or unpleasantness is sometimes called the '*primary* affect' (or, moment-to-moment affect) and the latter set of emotions '*secondary* affect' (Fields 1999, Price 2000). This distinction is most at home in cases of recurrent and persistent pains. Most often it is the secondary affect that grounds the suffering involved in chronic cases and is the major cause of decline in life-quality and well-being and generates most of the psychological, social and economic stigmas associated with chronic pain. In our example, the secondary affect is due to further processing of the information contained in the brief tactile/nociceptive encounter in light of your beliefs, expectations, values, desires, etc. The huge negative affective/emotional impact of this experience is due to what the sensory pain may *mean* to you given your concerns and doxastic background. Secondary affect involves heavier cognitive processing and is thus variable depending on various degrees of individual and contextual parameters. Thus, in light of most pains' having a secondary affect to varying degrees and complexities, pain may be said to be an emotional experience in a more robust sense, which is probably what the authors of the IASP definition had in mind.

There is another way to carve the distinction between these two sorts of affect. We may call the primary affect that directly attaches to pain sensations 'sensory affect' as this affect is an immediate sensory-quality-dependent modification of a sensation which has a more direct psychophysical connection to the sensory intensity of pain. We may call the secondary affect 'cognitive affect' as it seems to be a function of not sensory quality per se but of more abstractly characterized information extracted and further processed by cognitive and conative mechanisms downstream of sensation. An advantage of characterizing the distinction in these terms is that we can generalize it beyond pain. All sorts of sensory modalities produce experiences with primary affect (positive as well as negative), 'sensory affect' may thus be applied to sensation-dependent immediate hedonic valence (positive or negative) — e.g., the pleasant taste of chocolate, smell of rose, etc. Similarly, all sorts of experiences (sensory or otherwise) generate secondary affect due to what information they convey in light of what concerns and background knowledge the agents have — e.g., anxiety of driving on the fast lane, feeling the sudden strong gust while gybing, etc. 'Cognitive affect' may usefully cover

such episodes — whether negative or positive.[7] Indeed, most salient members of cognitive affect are standard emotions, such as grief, anger, fear, joy, etc. Note that cognitive affect is affect *due to* cognitive processing and is so designated because of its relative independence of modality specific sensory qualities, and *not* because it lacks feelings or phenomenology — indeed many experiences with cognitive affect may be among the phenomenologically most intense affective feelings there are.

Pleasure

Unlike in the case of pain, there is no accepted definition of pleasure. Indeed, as far as I know, there isn't even an attempt to offer a definition in the field. Part of the reason for this is the polysemy of the word 'pleasure' in ordinary language. We have already distinguished above between pleasure experiences and their sources or objects, and pointed out that it is the experience (as a mental event or episode) that we'll have in mind when discussing pleasure here. Beyond this, the distinction we introduced between primary and secondary affect should shed some light on a further crucial distinction among pleasures. We called the affective-motivational component of pain that immediately attaches to the pain sensation in an *intensity and quality dependent way*, 'primary affect,' and attempted to generalize the notion to *all* sensory experiences with positive or negative hedonic valence by calling it 'sensory affect.' We can now define sensory pleasure simply as a pleasant sensation:

Sensory pleasure = sensation + positive affect
(or, slightly more precisely: positively valenced modality dependent sensory experience)[8]

[7] Philosophers sometimes use the term 'propositional' (especially in designating propositional pleasures) for this sort of affect. See Feldman (1992) among others. The 'cognitive/conative' processes involved need not be very complicated or volitional — all that is required is that the information extracted or obtained is subjected to one's background knowledge and concerns (this could be fast and relatively automatic in many or most cases).

[8] Remember that we are using 'sensation' in a restricted sense referring only to the immediate output of sensory mechanisms. Kent Berridge has this sort of sensory pleasure in mind when he writes: "Sweetness tastes nice. The pleasantness of a sweet taste is a gloss on the mere sensation, added by our brains to the sensory quality of sweetness" (2004a: 243 — see also Frijda 2001). Berridge talks about the sensory quality of sweetness, presumably meaning the sensation of sweetness. The term 'sweetness' may denote whatever constellation of physical features of molecules that gives rise to the characteristic sensation upon tasting sweet substances. Or it may denote the particular sensory quality (qualia) of one's experience upon tasting such a substance. The former is an objective, physical feature of substances (stimuli). The latter is a subjective sensory quality — a phenomenological constituent of one's sensory experience. Psychophysics is precisely that discipline that investigates the systematic relationships between these two kinds of occurrences when generalized to all modalities.

Examples would include sensory experiences that we find pleasant solely due to how they feel in terms of sensory qualities and their intensities such as the various taste or smell experiences of tasty food items.[9] Note that sensory pleasures would be an instance of the more general schema for sensory affect:

Sensory affect = sensation + primary affect (positive or negative)

It turns out that pain is unique in having its own proprietary sensory specialization about noxious stimuli. Sensory pleasures, by contrast, are experiences that are the immediate result of sensory mechanisms involved in *all the other* sensory modalities — when the affective-motivational mechanisms tag them positively as pleasant. The proper class to oppose sensory pleasures is thus, not pain, but the class of *sensory displeasures* (for example, the sensory experiences upon smelling awful smells, tasting bitter substances, hearing sudden loud sounds, etc. — in brief, unpleasant sensations):

Sensory displeasure = sensation + primary negative affect
(or, slightly more precisely: negatively valenced modality dependent sensory experience)

Pain is thus a subspecies of sensory displeasures with its own proprietary (nociceptive) sensory (sub)modality. In this respect, sensory pains and pleasures are quite different: the latter don't have any *sensory* system or modality proprietary to itself.[10]

Finally, we have pleasures instantiating positive *cognitive* affect as described above. These would be *non-sensory* experiences with positive affect due to their amodal informational content. Suppose I am a strawberry farmer, eagerly hoping to find my strawberries ready to be picked relatively early in the spring. I am not hungry or thirsty. I am in fact tired of eating strawberries, don't like them very much anymore. I bite the strawberry. I am very pleased by how it tastes. But this is not because I find the taste intrinsically pleasant, rather it is because the taste indicates that my strawberries are ready to be picked and sold — I am *pleased that* I will have a good return on the market. Although the experience is pleasant, the affect isn't solely due to the intrinsic sensory

[9] In ordinary speech, sometimes the terms 'physical' or 'sensual pleasure' are also used to denote sensory pleasures as characterized here.

[10] Notwithstanding the recent discovery of slow-conducting unmyelinated fibres "coding for pleasant touch" (Löken et al. 2009) — there are no fibres that "code" for mere pleasantness as an objective stimulus feature of the detected peripheral event. See Fulkerson (2016) for a critical discussion. The case of sexual orgasm as a pleasure is complicated — orgasms seem to have a sensory dependency that other sensory pleasures seem to lack. I don't think orgasms pose difficulties for the claim made in the main text, but I cannot pursue this matter here — see Georgiadis & Kringelbach (2012).

quality involved (this could even be unpleasant), but rather is a function of my background knowledge and preferences, previous experiences I've had, and the interpretation of current context (physical, social, economic), etc. In a certain sense, my pleasure has very little to do with this taste experience I've just had, it is the pleasure I derive from *learning that* my strawberries are ready for harvesting early and will bring me more profit than expected. We can call this sort of pleasure, non-sensory or cognitive pleasure. This is the positive affect that is brought about typically as a result of cognitive processing of information (including sensorially supplied information) in light of one's background beliefs, values, preferences, and can be phenomenologically quite real and intense and manifested in the form of an overwhelming feeling — but it can also be so fleeting that may not leave any phenomenological trace in your consciousness.[11]

The Common Element

So, what should we be talking about when we discuss pains and pleasures in the context of emotions? Both sensory and non-sensory (cognitive) pains and pleasures vary in a common bi-valent affective dimension that we may just simply call, *affect*. So, affect occurs as being *attached* to sensations and cognitions[12] — we may simply say that affect qualitatively *modifies* them. In fact, this seems to be the primary form that affect is manifested in.[13] Morten Kringelbach and Kent Berridge sum it up well when they write that the basic

> research [in affective neuroscience] has shown that pleasure is never merely a sensation nor a thought, but an additional hedonic gloss, which is the pleasure versus displeasure affect that is actively generated by the brain and attached to its sensory or cognitive object. This [positive or negative] hedonic gloss of an object is generated by the brain in dedicated networks of hedonic hotspots and coldspots. (2017: 198)

[11] For instance, I read in the campus newspaper that the administration has just voted to increase funding to the Library. I am pleased — happy to learn that. But I can't introspectively detect any feeling that I can identify with my feeling pleasure on this occasion.

[12] I am including perception in the cognition category as it requires some cognitive/conceptual uptake compared to sensation. Obviously, sensory versus cognitive affect is itself a continuum depending on how much cognitive uptake or information integration/extraction takes place. Also, there may be activity-based pleasures involving many sensations and cognitions but that cannot be reduced to them such as playing competent tennis, dancing, reading a well-crafted novel, taking a walk in the woods, etc. This is a topic for another occasion — see Frijda (2010).

[13] But "core affect" can also, it seems, occur on its own as in the case of generalized moods when we just simply feel joyful, or down, for no apparent reason.

We can summarize the general structure thus:

Sensation or cognition + affect (negative or positive)

It turns out that sensory pain is a little more specialized in that it has its own relatively specialized *sensory* (nociceptive) system detecting certain forms of physical disorders in or on the body.

Pain and pleasure understood as affectively modified experiences (in the broad sense involving sensations or cognitions or both) are *composite* states. We can study them as such or we may focus on the affective component in a more direct manner. In what follows, after briefly touching on pain, I'll take the second option and concentrate on affect, and get back to the larger question about emotion at the end.

History

Pain

When viewed this way, it is not at all surprising to see that there has been no unity in the history of the study of pain and pleasure. The history of pain research, for obvious reasons, has tended to mix up with medical research, and especially in the last 100 or so years, heavily blended with the research on the *sensory* nervous system, and this orientation is still dominant in the study of pain today, even though there is an increasing recognition of the importance of the affective-motivational aspect of pain and attempts to study it in relation to general negative (aversive) affect (Leknes & Tracey 2008).

Before turning to affect, it's important to mention a key turning point in the study of pain that revolutionized pain science that should be familiar to all. Ronald Melzack and Patrick Wall published two papers in the 1960's, one on the nature of cutaneous sensory mechanisms (1962), and one in 1965 proposing a partially speculative new theory of pain that came to be known as the Gate-Control Theory of pain. Later in 1968, Melzack along with Ken Casey published another paper on the "sensory, motivational, and central control determinants" on pain. The 1962 paper influentially argued against the specificity theory of nociceptive transmission, which was popular then. The Gate-Control Theory, despite lack of details, established the existence of gating mechanisms in the spinal cord, and heavy top-down effects from the brain on the spinal processing of nociceptive information. The 1968 paper proposed a model of pain with three different constitutive dimensions including, most famously, the affective-motivational dimension of pain. Although these works were the syntheses that depended on many pioneering works of others, these three papers, nevertheless, set the agenda for the subsequent pain research like no others in that era. During the following decades, pain science was never

the same: these works had a tremendous impact on both the quality and quantity of research on pain — their influence continues even today.[14]

Affect (positive or negative)

One could reasonably say that the history of scientific research on pleasure started in the second half of 19th Century with reward theories of learning in the context of adaptive behavior. In this regard, most theories generalized their principles to cover not only pleasure with reward (positive affect), but also displeasure/pain with punishment (negative affect). The focus was on the role pain and pleasure play in learning and motivation of behavior. Early theorists such as Alexander Bain (1865), Herbert Spencer (1870), James Baldwin (1894) formulated their views psychologically, without any hesitation of using mental terms like 'pain', 'pleasure', 'feelings of agreeableness' and so on. Indeed, Cason (1932) attributed to these thinkers what he called the "pleasure-pain theory of learning," according to which, "organisms select those modes of behavior which are accompanied or followed by pleasure and eliminate those that are accompanied or followed by pain" (440). This is a concise statement of how individual flexible behavior is acquired or learned, which assumes that pains and pleasures are somehow connected to motivation. Thorndike's well-known Law of Effect, in contrast, appears to be more *behavioral* rather than psychological:[15]

> Of several responses made to the same situation, those which are accompanied or closely followed by satisfaction to the animal will, other things being equal, be more firmly connected with the situation, so that, when [the situation] recurs, [the responses] will be more likely to recur; those which are accompanied or closely followed by discomfort to the animal will, other things being equal, have their connections with that situation weakened, so that, when it recurs, they will be less likely to occur. The greater the satisfaction or discomfort, the greater the strengthening or weakening of the bond. (Thorndike 1911: 244)

Behaviorists such as Clark Hull (1943, 1952) and B. F. Skinner (1938) in developing their principles of learning along similar lines tended to avoid the use of psychological words. Instead, they referred to objective stimuli by allegedly non-psychological words

[14] See Sufka & Price (2002) for a general evaluation of the Gate-Control Theory. Perl (2007) contains a very informative historical survey of pain research. Apkarian et al (2013) and Woo et al (2017) are good sources for an overall evaluation of most recent brain imaging studies.

[15] It depends on how one interprets the crucial terms such as 'satisfaction' and 'discomfort' in the quotation. In early formulations, Thorndike clearly had in mind psychological interpretation, but later on, in response to behaviorist criticisms, he dropped psychological terms in favor of merely behavioral repetition effects. See Berridge (2000).

like *'reinforcers'* or 'punishers' that function in establishing probabilities of certain types of behavior in response to stimulus conditions in the light of organism's conditioning history.

One of the major contributors to behaviorism's demise was its inability to explain satisfactorily how reinforcers reinforced, or how punishers punished, without introducing sufficiently rich set of internal motivating states. Hull thought that the reduction or increment in *drive* states produced by biological needs provides the mechanism for the changes in the rates of behavioral responses. Roughly, what is needed is a mechanism that will somehow track the effects of an animal's response to stimuli such that if they fulfill a certain criterion it will set in motion whatever further mechanism does the strengthening or weakening of this response type next time similar stimuli are encountered. What is needed, in other words, is a description of this criterion *independent* of the downstream strengthening or weakening effects on the response. The drive reduction theories such as Hull's take this criterion to be either the increase or reduction in biological needs or the reduction or increase in the drive states created by such needs. If among the consequences of the animal's behavior are the fulfillment of the animal's need or the reduction in the drive signal then the stimulus that prompted the behavioral response is a reward and therefore a reinforcer. If the response doesn't have need-fulfilling or drive-reducing consequences, then the stimulus isn't a reward and therefore shouldn't (positively) reinforce the response (it could even be a punisher). Drive reduction by satisfying biological needs is thus the proposed independent mechanism of how rewards reward (or, reinforcers reinforce) — and punishers punish in case there is increase in drive.

Drive reduction theories have proven to be empirically false: there have been many demonstrations that merely satisfying the biological nutrient needs, thus presumably reducing the drive, by for example intravenous or intragastric feeding, don't stop motivated eating or drinking behavior (Miller & Kessen 1952, Nicolaidis & Rowland 1976, Myers et al 1998, among others). The electrical brain stimulation experiments conducted by James Olds and Peter Milner and many others in the 50's and 60's (Olds & Milner 1954, Olds 1977) also decisively showed that animals can be motivated to act and taught to act in ways which have nothing to do with natural drive reduction by need satisfaction. Drive reduction is neither necessary nor sufficient for reinforcement.

The failure of drive theories encouraged the development of *incentive* theories of motivation and learning (e.g., Pfaffman 1960, Young 1966, Bolles 1972, Bindra 1978, Dickinson 1985, Dickinson & Balleine 1994, Toates 1994, Panksepp 1998, Berridge & Robinson 1998, Berridge 2004b). The basic tenet of these theories is that organisms respond to stimuli not because they are reinforced to do so according to a strict behaviorist reading of the Law of Effect, but because their responses are pieces of

behavior emitted due to the incentive values of the environmental cues (conditioned stimuli — CS) as well as the expected hedonic values of rewards they predict (unconditioned stimuli — UC), which usually involves experiential encounter with the reward during consummatory activity. It is possible to interpret these theories as a way of returning to the pleasure-pain theory of learning. Incentive theories interpret rewarding and punishing qualities of stimuli psychologically as their capacity to impact agents hedonically or affectively. As we move around and behave in the world, we acquire expectancies about what stimuli and what behavioral responses will result in experiences with hedonic/affective value (with positive or negative valence). Experienced hedonic value is what seems to determine the incentive value for those objects, events, conditions in the world that, when interacted with in the right way, tend to bring about the affectively (hedonically) valued experiences. Learning, then, consists in learning when to attribute incentive value to what worldly conditions.

Historically, this function of affect has taken to imply motivation. Learned hedonic expectancies have been thought to necessarily bring along with them motivation. Indeed, the idea that hedonic value (positive or negative affect) is inherently motivating seems quite intuitive: it sounds like a truism when it is said that all organisms capable of experiencing affect tend to *pursue* experiences with positive affect and *shun* those with negative affect. It was this "truism" that led to the view that dopamine is the neurotransmitter of pleasure. Olds and Milner's 1954 brain stimulation experiments on rats showed that rats can be made to eagerly and repeatedly press a lever upon delivery of tiny electrical currents in certain areas in their hypothalamus and septum at the expense of food, water, sex, and other naturally rewarding stimuli. Robert Heath (1963) and others later ran similar experiments on humans who reported various desires and urges and tended to similarly self-stimulate continuously. Olds (1956) described the brain sites as "pleasure centers" and the term quickly got stuck with the popular imagination for a while. Roy Wise (1980) along with others identified many stimulation sites as dopaminergic and commented: "I wonder if OIds' notion [pleasure center], though admittedly simplistic, does not merit a closer look since it now seems clear that blockade of the dopamine synapse can uniquely block the rewarding impact as objectively defined… and since direct activation of this synapse is rewarding in its own right" (1980: 94). Pretty much all the *actual evidence* strongly pointed to *motivation* ('wanting', pursuing, acting) and the dopamine's role in it, and yet the brain centers and the role of dopamine got labeled as *hedonic* ('liking', pleasure) mainly because of the natural dual assumptions that pleasure inherently motivates and that strong motivation is a direct measure of hedonic impact.

Current Evidence (about positive affect)

Recent research in affective neuroscience seems to show that these assumptions may not be correct if interpreted as expressing more than mere causal connections. Since the early 1990's, there has been growing evidence that the pleasure/liking circuitry (for merely hedonic phenomena) in the brain is connected to but distinct from the circuitry subserving wanting/desiring (motivation). There is now a growing consensus that the mesocorticolimbic dopaminergic system, which was once thought to directly underlie pleasure, is not directly responsible for 'liking' whatever the primary sensations the animal may be undergoing.[16] (This system projects from a region in the midbrain to the nucleus accumbens, NA, and some neighboring areas.) Rather, the circuitry that seems essential for liking consists of groups of "hotspots" that are functionally connected to each other and are to be found in the rostrodorsal quadrant of the medial shell of NAc and the posterior part of ventral pallidum (VP) as well as in the parabrachial nucleus (Pecina et al 2006; Smith & Berridge 2007). The NA hotspots receive major input from the infralimbic cortex, which itself interacts heavily with orbitofrontal, prelimbic and anterior cingulate cortices. NA has strong projections to VP (as well as parts of amygdala). VP hotspots project to the paraventricular nucleus of the thalamus that in turn have connections to the infralimbic cortex and other frontal areas, thus closing the loop. This circuitry interacts with the dopamine systems but also uses different neuromodulators such as enkephalin and anandamide, as well as glutamate and GABA (intersystems) (Castro & Berridge 2014a,b). There are also cold spots in NA and VP. The terms 'hot spot' and 'cold spot' were used by Pecina and Berridge (2005) to denote tiny anatomical regions where microinjections of opioids significantly increase or decrease hedonic reactions. It is thought that these functionally connected hubs of hotspots act in unison to stamp a sensation as pleasant in that if any hub in the circuitry defects or issues a veto, the sensation that is being processed is denied the 'pleasant' stamp or no increase in hedonic impact occurs (Berridge & Kringelbach 2013).

Most of the evidence for the separability of these two systems comes from various experiments that show that the liking system can be modulated (enhanced or inhibited) independently of the wanting system, and vice versa (Berridge 1999; Berridge

[16] Kent Berridge and his colleagues have been most vocal about this evidence: see Berridge 1996, 1999, 2006; Berridge & Robinson 1998, 2003; Pecina et al. 2006; Berridge & Kringelbach 2011, 2015. The collection edited by Berridge and Kringelbach (2010) contains very useful chapters reflecting the state of the art. See also Salamone et al. (1997, 2002), Ikemoto & Panksepp (1996, 1999), among others. Berridge and his colleagues distinguish liking/wanting, which they take to be consciously experienced, from core 'liking'/'wanting' (with quotes) that are not necessarily conscious. I'll ignore this for convenience and use the words without quotations and leave open their status as conscious or not. Needless to say, conscious liking/disliking will be underlain by additional neural structures and mechanisms, especially the orbitofrontal and insular cortices.

& Kringelbach 2011). For instance, you see motorically capable dopamine-depleted rats starving themselves to death in the presence of readily available food — they are not motivated to eat in the sense that they seem to lack a desire for food. But when these rats are force-fed, they exhibit orofacial movements clearly indicative of them liking the taste (Berridge & Robinson 1998). There are also experiments where you can increase or decrease liking without a corresponding change in wanting, or you can make rats work hard to get and consume food or sucrose solution without them liking their taste (Pecina et al. 2006). Similar disassociations seem to exist in humans — indeed addiction is taken to be the overreaction of an overly sensitized 'wanting' (dopamine) system to contextual cues surrounding drug use without corresponding proportional hedonic impact (Robinson & Berridge 1993; Robinson & Berridge 2008).

If affect (hedonic valence) and motivation are only causally connected and can be dissociated, then the question arises as to what the function of sensory affect is — what is the functional signature of hedonic valence within the mental economy of the liking/wanting/acting agent? The function of conative states like wanting or desiring (motivation) is relatively straightforward: move the agent in a way that will tend to bring about the worldly content of these states (when combined with other information about the perceptual environment and past experience, etc.). For instance, if I now want to have a beer to quench my thirst, *ceteris paribus*, I will behave, shortly after, in a way that will bring about *what* I want (the propositional content — that I drink beer). When decoupled from motivation, the function of liking (affect in general) becomes somewhat less obvious.

Function

One natural proposal is that hedonic valence is a "teaching signal" of sorts:[17] it tells the agent to 'want,' or form a 'desire' to bring about, what is thus valenced — this involves, and for most animals, *exhausts* learning when and how to perform those sequences of actions similar to those that have actually lead to the obtaining of the valenced experience. Liking helps attribute *incentive salience* to environmental stimuli and sustain it (Berridge 1996, Dickinson & Balleine 2010). The sustaining bit is important. A learning-capable agent that acts out of an existing want or desire (learned, acquired, or otherwise) needs to somehow track the consequences of its behavior, that is, whether its actions

[17] I am using this expression *not* to refer to the phasic dopamine signals that are hypothesized to code prediction errors in expected reward. The role of phasic dopamine signals in midbrain structures (VTA, SN) has been controversial but they look a lot like the teaching/learning signals postulated by many reinforcement learning algorithms (see, e.g., Schultz 1997, 2016 for a defense; see Berridge 2012, and Berridge & O'Doherty 2014 for general critical assessment).

result (or have resulted) in the satisfaction or frustration of its 'desires' — generating more 'likes' or 'dislikes.' Plausibly, this is the other side of the same coin — of learning what desires to form on the basis of experienced valence. So, experienced valence is also a signal for desire satisfaction or frustration (cf. Schroeder 2004). Thus, although the mechanisms for affect and motivation are separate, they causally interact. We quite generally want what we like, and, more often than not, we like what we want.

Further research on the function of affect is likely to reveal in the future that there is a deeper unitary role affect plays in learning, motivation, and subjective wellbeing.

Impact

The discovery of dissociable underlying mechanisms has obvious important implications for a better understanding of affective disorders such as depression, clinical anxiety, and bipolar disorders as well as addiction and obsessive-compulsive behavior. For example, the well-received *incentive sensitization theory* of addiction (Robinson & Berridge 1993, 2008) directly came out of hypotheses about the separability of affect from motivation — one way to characterize addiction is as a big increase in motivation to seek and consume substances that is vastly disproportionate to the increasingly diminishing affective payoff. The advances in basic affective neuroscience are poised to deliver surprising results about the causes of various emotional and affective disorders, which promises not only to greatly facilitate proper, faster, and more detailed diagnosis but also to offer huge potential for developing treatment options. There is increasing research on the extensive mechanisms shared by the brain's default-mode network and the affective circuitry, both of which are connected, unsurprisingly, to pervasive affective disorders such as depression and anhedonia in general. There is evidence that optimal metastability in the brain's large-scale dynamical oscillation plays a role in subjective affective wellbeing. (Kringelbach & Berridge 2017).[18]

[18] There are historical precedents to this sort of approach to affective wellbeing. Sigmund Freud himself noted that his own 'Pleasure Principle' is substantially the same as Gustav Fechner's 'Principle of Constancy' (1983): "In so far as conscious impulses always have some relation to pleasure or unpleasure, pleasure and unpleasure too can be regarded as having a psycho-physical relation to conditions of stability and instability. . . According to this hypothesis, every psycho-physical motion rising above the threshold of consciousness is attended by pleasure in proportion as, beyond a certain limit, it approximates to complete stability, and as attended by unpleasure in proportion as, beyond a certain limit, it deviates from complete stability; while between the two limits, which may be described as qualitative thresholds of pleasure and unpleasure, there is a certain margin of … indifference" (quoted in Freud 1920/1961: 2). It would be interesting to sort out the similarities and differences between this tradition and the emotion psychologists (e.g., Russell 2003, 2009) who promote the notion of a "core affect" as the foundation out of which emotions are constructed.

Conclusion

Above, we had a schema about the general structure of pains and pleasures:

[sensation or cognition or both] + affect[19]

The question to ask is:

(Q) Does every instance of this schema yield an emotion?

In the absence of clear and uncontroversial criteria about what qualifies as an emotion, it is hard to answer this question. If I rely on my pre-theoretical intuitions, I am inclined to answer it in the negative. But we all know that when it comes to emotions, people's intuitions (pre-theoretical or otherwise) are all over the place. Perhaps we can simply stipulate that any sensory affect is an *elementary* emotion. This would be fine but it shifts the main research question in emotion theory: what, then, makes a mental episode into a *non*-elementary emotion? If we can set the sensory case aside in this way by designating them as elementary, perhaps we can identify all cognitive affect with emotions? This suggestion is probably better — as all emotions are known to involve some cognitive uptake about what is going on in the environment of their emoters, which is usually not a modality specific affair and involves cognitive appraisals. But what about very simple forms of cognitive affect? For example, I've just learned there is less car theft in my neighbourhood this year than last year — I am certainly *pleased that* this is so. Have I undergone an emotion? Saying yes would seem to stretch the meaning of 'emotion'. The best we can justifiably say, it seems to me, is that some pains and pleasures are emotions, some not — and leave it at that for present purposes.

The key point not to lose sight of is that the core brain mechanisms that generate negative or positive affect as attached to mental processes and the mechanisms connecting affect to learning, decision-making, motivation, and action, are the core building blocks of all emotions. We need to investigate what further elaboration of the basic affective processes is needed to explain the full range of emotions.[20]

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[19] Positive or negative affect. It is plausible that affect is a dimension of all mental events even when they seem affectively neutral. One way of putting the point is to say that there are no affectless mental events but there are affectively neutral ones. It makes sense to posit affect in this sense as always present, and "commenting" or glossing on all mental episodes with intentional content that contribute to learning, decision making and action. It should be obvious why this is particularly crucial in *sensing* or *perceiving* our environment and acting in it quickly and efficiently.

[20] This seems to be the general direction that Kringelbach and Phillips (2014) are recommending for the future of emotion research. Panksepp's work has also been generally supportive of this sort of direction based more on the biological and evolutionary considerations — see Panksepp & Watt (2011) for a review,

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