Just What Lies "Beyond the Pleasure Principle"?

Brian Johnson (Syracuse, NY)

Are there any developments in neuroscience since 1920 that might result in a revision of the concepts of the repetition compulsion and the death instinct? The observation Freud made that there is a force, "More primitive, more elementary, more instinctual than the pleasure principle which it overrides . . . independently of it and to some extent in disregard to it," is pursued by an examination of the neural pathways and signaling apparatus that underlie drive, pleasure, and cathexis. The conflict between drive and pleasure has been rediscovered and further explained biologically by Berridge and Robinson (2003). This conflict leads to an understanding both of the biological basis of the transference and of the neural underpinnings of why patients would seek an unhappy relationship with their psychoanalyst. In this neuropsychoanalytic theory paper it is suggested that happiness and emotional health are facilitated by the alignment of drive and pleasure, while neurosis is driven by urgently wanting relationships that cause pain and frustration based on a misalignment of two distinct neural systems. Within this framework, concepts of repetition compulsion and death instinct are adventitious. Understanding the neurobiology that underlies metapsychology can help us resolve disagreements and facilitate more accurate models of human functioning that guide our therapeutic interventions.

Keywords: cathexis; pleasure principle; transference; repetition compulsion; death instinct; SEEKING system

Neuroscience constrains theorizing. Our models must be possible within the limits of how brains work. Are there any developments in neuroscience that might cause us to modify metapsychology?

Our understanding of the neuroscience behind drive, pleasure, and cathexis is vastly improved. The relevance of the drive system to addiction has been a central focus of research since Panksepp first described the pathway for "SEEKING" in 1981. How is the SEEKING system of Panksepp correlated with the drive system of Freud? Shevrin (2003) discussed the congruence of Panksepp's SEEKING system and Freud's drive system. He said, in part,

> The classical view of motivation embodied in Freud's drive theory is supported independently by substantial neuroscience evidence. This independent evidence based on non-clinical methods demonstrates that two key presuppositions of clinical motivation theory, *motive pressure* and *functional equivalence*, have convergent validity. A clinical theory of motivation based on these assumptions acquires greater cogency. Based on this convergence, a theory of agency is presented as well as implications for our understanding of the primary process. In effect, I am proposing that motivation in all its forms, from drives to the so-called tamed motives, are the engines of agency, or better still, they *are* what we mean by agency. The word itself, *motive*, derives from the Latin *mover*e, to move or be in motion, as do most of our own words denoting activity. The first motives, whether conceived in developmental or evolutionary terms, were just that—motions, modes of action, simply moving about in the world. As active agents people and animals adaptively learn and prosper. But there is also a pathology of agency occurring when agency is hyperexcited and fixations in psychoanalytic terms, or sensitization of the NAS (nucleus accumbens shell) DA (dopamine) circuits in neuroscience terms, form and result in neurotic or psychotic primary process replacements of reality. Finally, the neuroscience evidence provides a neurophysiological and neuroanatomical grounding of drives.

Studying the neurobiology of addiction has allowed us to observe changes in the behavior of animals, including humans, whose aims have been altered from procurement of food, water, and sex (relationships) to the procurement of drugs. As described below, cathexis relies on drive combined with memory, and the

Brian Johnson: Department of Psychiatry, State University of New York Upstate Medical University, Syracuse, NY, U.S.A.
Correspondence to: Brian Johnson, Department of Psychiatry, State University of New York Upstate Medical University, 750 East Adams St, Syracuse, NY 13210, U.S.A (email: johnsonb@upstate.edu).

pleasure system depends on endogenous ligands of the opiate receptors released when certain survival behaviors are enacted. None of these systems is understood fully, but their neural organization is far better known than in Freud's time. I explain here the issue that Freud addressed in *Beyond the Pleasure Principle* in 1920. After elaborating some concepts relevant to the neural systems involved in drive, cathexis, and pleasure, I return to Freud's question of what lies beyond the pleasure principle and suggest that, with more information, we can solve his dilemma in a more elegant, more parsimonious, and even more Freudian way; more Freudian in that the solutions to what lies beyond the pleasure principle were already part of metapsychology by 1920, before Freud invented the concepts of repetition compulsion and death instinct.

Freud's dilemma in *Beyond the Pleasure Principle*, and ours

Why do we seek certain kinds of relationships? Every psychoanalyst has an answer that is based on the developmental history of his or her individual patients. As Freud described in *Beyond the Pleasure Principle*, the transference is a repetition of an earlier relationship that is remembered, not completely consciously, and is repeated. When the psychoanalyst listens carefully and without disrupting the patient's efforts, he or she begins to hear "the transference," the construction built on neutral stimuli emitted by the analyst of the type of person with whom the patient is seeking to engage. Psychoanalytic treatment resolves conflicts between the relationships a naïve person seeks out and the unfortunate complications of that person getting exactly what he or she unconsciously wants.

Freud assumed, as do many of us, that the goal of our drives was pleasure. However, his experience treating patients with psychoanalysis was that patients recreated dysphoric relationships with him. He began to call this the "compulsion to repeat":

> Only in rare instances can we observe the pure effects of a compulsion to repeat unsupported by other motives . . . the compulsion to repeat and instinctual satisfaction which is immediately pleasurable seem to converge into an intimate partnership. [1920g, p. 23]
>
> In the case of the person in analysis the compulsion to repeat the events of his childhood in the transference evidently disregards the pleasure principle in every way. [p. 36]

Notice the location of the word "and" above. It is sandwiched between the solution Freud was producing in this paper, "the repetition compulsion," and two entities that I describe later as unfortunately conflated, instinct and pleasure ("instinctual satisfaction which is immediately pleasurable"). Freud then described the force "beyond the pleasure principle" in a way that we will see rings even truer with more information from neuroscience.

> More primitive, more elementary, more instinctual than the pleasure principle which it overrides. [p. 23]
>
> . . . not, indeed, in opposition to the pleasure principle, but, independently of it and to some extent in disregard of it. [p. 35]

Freud thought that he had no explanation to provide from his existing metapsychology (we will see that this was not right) and therefore entered the realm of speculation:

> It seems, then, that an instinct is an urge inherent in organic life to restore an earlier state of things. [p. 36]
>
> . . . the aim of all life is death. [p. 38]

If we might then agree that there is most certainly a force beyond the pleasure principle, and that it is clearly present in the miserable relationships that patients in psychoanalysis set up with us as a routine matter, what other explanation might we conceive of? Does there now exist enough neuroscience knowledge to explain the material, neurobiological basis for the development of an unpleasurable transference?

The drive system as observed in addictive illness

The Freudian metapsychological concept of drive might best be instantiated in neuroscience as activity in the ventral tegmental dopaminergic SEEKING system (Panksepp, 1998, p. 168). "The SEEKING disposition, independent of world events, would also have its own hedonic properties, not the 'pleasure of satisfaction', but 'enthusiastic positive excitement', 'interest', 'desire', and 'euphoria'" (Alcaro, Huber, & Panksepp, 2007, p. 294). This system, which arises in the midbrain, synapses in the nucleus accumbens, and then stimulates centers in the frontal cortex (Figure 1), gives energy to animals in their pursuit of incentives such as food, water, and sex (Robinson & Berridge, 1993). This system helps us in identifying resources in our environment and moving toward them effectively.

Freud suggested that the energy of the neuronal system exists in a "facilitated" or "cathected" state related to the memory of experiences (Freud, 1950 [1895], pp. 300–301). He believed that the brain runs on "particu-

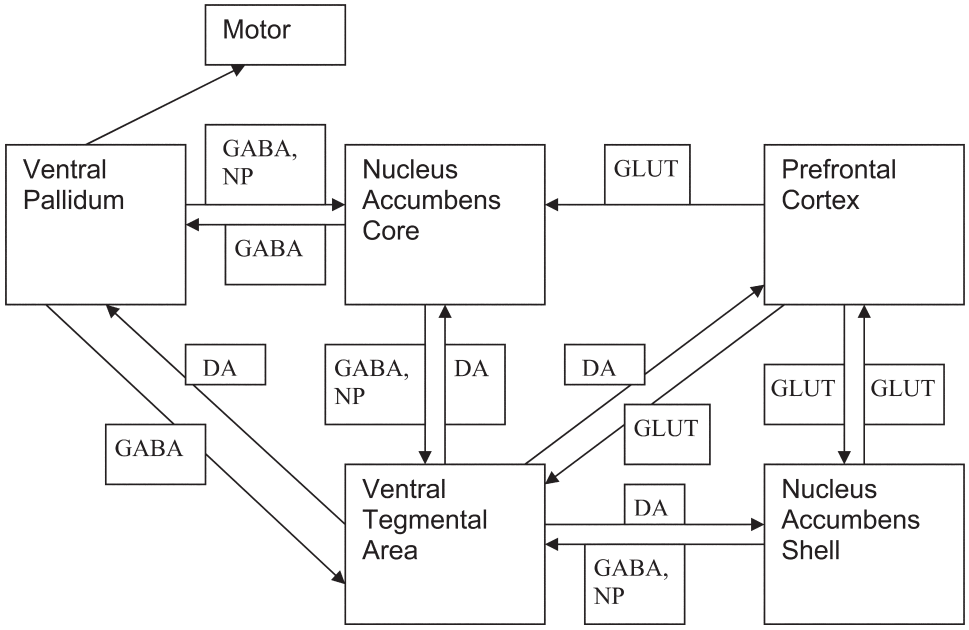

Figure 1. Addiction allows us to look at the drive system without people being involved. DA = dopamine, GABA = gamma-aminobutyric acid, NP = neuropeptide, GLUT = glutamate. (After Kalivas & Volkow, 2005.)

lar chemical substances" that affect "the amounts of energy and their distribution in the mental apparatus" (Freud, 1940a [1938], p. 182). This concept might well be instantiated in modern neuroscience by the concept of long-term potentiation (LTP) of neurons (Kandel, 2006). AMPA (alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid) receptors are converted to more easily triggered, higher-energy phosphorylated NMDA (N-methyl-D-aspartic acid) receptors by repeated glutamatergic stimulation. In the high-energy phosphorylated state, these receptors are far more easily triggered by glutamate molecules, opening ion channels that provoke neuronal depolarization.

How does LTP apply to drive in a human situation? If you are walking down the road without having eaten, VTA dopamine will be stimulating prefrontal areas to be on the lookout for sources of food, even if you are not consciously aware of being hungry. If the smell of pizza wafts through the air, long-term potentiation of neural systems will connect your memories of loving pizza with other memories of obtaining pizza. Glutamatergic neurons relay this information back to the VTA (Figure 1), which excites more specific SEEKING—not for any source of food, but for pizza. Frontal eye fields that are linked with memories of storefront shops help you scan the environment for the source of the smell, in case you want to stop in for a slice (sensorimotor connection: Kandel, 2006; frontal eye fields: Barbas, Ghashghaei, Rempel-Clower, & Xiao, 2002). The connections of the drive system, the sensory memories, and your visual ability to discern sources of pizza in your environment have been facilitated or cathected by the long-term transformation of glutamate receptors into a phosphorylated state that results in intense firing of all involved neurons when the smell of pizza has been transmitted to your thalamus. Once you have feasted on pizza, you will never forget the smell (Freeman, 1983; Staubli, Izrael, & Xu, 1996). When you are hungry, you will involuntarily seek out the source of that smell.

The most sophisticated understanding of the drive system has been produced by researchers trying to understand why individuals with addiction pursue the drugs that are destroying them. Drugs of addiction provoke an artificial drive state, often referred to as "craving," that motivates them to seek the drugs that impinge on the drive system. However, this drive system does not exist to help animals or humans procure addictive drugs that will destroy them. The drive system exists to help animals pursue basic needs, but it is also connected to the pursuit of social relationships (Depue & Morrone-Strupinsky, 2005; Insel, 2003; Kalivas & Volkow, 2005; Panksepp, 1981, 1998). We will make a detour through drug addiction research to understand

how chemicals can sensitize the drive system, causing an unreasonable urge to find and use substances. We then return to the pursuit of relationships.

None of the following explanations involve the process of initial exposure to drugs. This has been discussed elsewhere, and some of the forces perpetuating addiction are more complex than the brain pathways discussed below (Johnson, 1993, 1999, 2003). However once introduced into the body, addictive chemicals upregulate the dopamine pathway originating in the ventral tegmentum (Robinson & Berridge, 1993). The increase in energy in this system produces learning as information, expressed as dopamine barrages from the ventral tegmentum to the nucleus accumbens shell (NAS) that cause long-term potentiation of accumbens neurons (Kalivas & Volkow, 2005). A key gene regulator is delta fosB (Zacharion et. al., 2006), because it is not transiently expressed but appears to be a permanently upregulated feature of this process. Delta fosB modulates the synthesis of AMPA glutamate receptor subunits and cell-signaling enzymes (Kalivas & Volkow, 2005; Nestler, 2005). By recurrent stimulation of AMPA receptors, they are turned into phosphorylated and much more easily triggered NMDA receptors. This change represents information in the system. In turn, information is passed upward to the prefrontal areas, causing them to scan the environment for the now-desired substance and to evaluate the desirability of various cues in the environment that may result in the ingestion of the drug. Other rewards become relatively less interesting. Finally, downgoing glutamatergic fibers from the prefrontal and insular areas innervate the nucleus accumbens core, where they motivate behaviors intended to procure the desired substance. Glutamatergic fibers from these areas also innervate the ventral tegmental area (VTA) so that "exciting" environmental events charge the drive system with energy to pursue the now-desired chemical.

This process does not happen all at once. The first event is the increase in neurotransmission from the VTA to the NAS (Nestler, 2005) (Figure 2). One addicted person after another will tell the story that they had no idea that they were addicted at first. The increased attraction to the addictive drug is initially unconscious. The VTA/accumbens directs motor areas to begin SEEKING. Learning involves glutamatergic and dopaminergic inputs from higher centers to the accumbens core, leading to increased dendritic spine density (Robinson & Kolb, 1997). The final state of addiction involves the prefrontal cortex seeking cues that the organism has learned to predict the availability of the drug and commanding the accumbens core and subsequent motor areas to search for the drug (Kalivas & Volkow, 2005; Panksepp, Nocjar, Burgdorf, Panksepp, & Huber, 2004). It is only when the entire behavioral ensemble is present that persons become

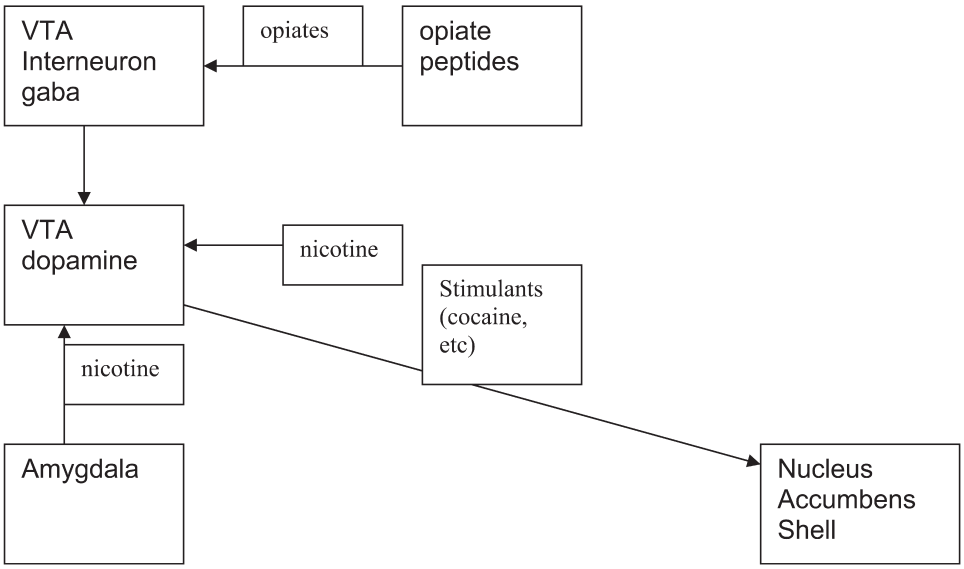

Figure 2. How do drugs that are so destructive become urgently wanted? Nicotine stimulates the ventral tegmental area (VTA) dopaminergic signal to the nucleus accumbens shell (NAS); stimulants (cocaine, methamphetamine) block the dopamine reuptake transporter protein, increasing the dopaminergic signal to the NAS; opiates inhibit the gamma-aminobutyric acid (GABA) tonic inhibition of VTA interneurons against the VTA, increasing dopaminergic signal to the NAS. (After Nestler, 2005.)

fully conscious of their addicted state. By then it is too late to reverse the neurobiological process; one can only contend with it—use drugs or not, but the urge will always be present (Robinson & Berridge, 2000). By the point of end-stage addiction, it may be that the most important focus of action is not the cortex but, rather, the accumbens core (Everitt & Robbins, 2005). The point about the accumbens core is that this is a subcortical center that organizes behavior. In order for behavior to be conscious, it must involve cortical areas (Barbas et al., 2002; Freud, 1920g). Hence, addicted persons can be goal-directed and yet unconscious regarding the reason for their behaviors.

Building on the description above, and a number of trenchant experiments Robinson and Berridge (1993; Berridge & Robinson, 1998, 2003) now have a model of the drive for addictive drugs that parses the motivational system into three parts: wanting, hedonic valence; and learning. Wanting is not a conscious process, and they give examples such as that addicted individuals will work for low doses of stimulants or morphine even though the doses are so low that they produce no subjective effects and no autonomic responses. Berridge and Robinson (2003) cite an experiment where thirsty people exposed to subliminal views of a happy face will describe no change in subjective feeling or mood yet consume more fruit drink moments later. The faces affect wanting in a way that is not conscious.

There is much evidence (Berridge & Robinson, 2003; Panksepp, 1998) that the basis of "liking" is the endorphin/opioid system. In a classic experiment to demonstrate the nonmotivated hedonic system, Berridge and Robinson (1998) cut the ventral tegmental dopaminergic SEEKING pathway in rats and showed evidence of smiling facial responses to sucrose drinks, despite the fact that the rats had no interest in eating. With their drive system cut, the rats still showed evidence of pleasure. We learn to look for things we enjoy, and that learning involves the orbitofrontal, insular, cingulate gyrus, hippocampal, and amygdalar regions (Volkow, Fowler, & Wang, 2004), but the key connection is with the nucleus accumbens core using mainly AMPA/kainate, NMDA, and dopamine receptors (Hernandez, Andrzejewski, Sadeghian, Panksepp, & Kelly, 2005).

Berridge and Robinson (2003) then ask the question: "People often have an explicit cognitive expectation that they will want the things they like. Is this true?" They suggest that this is *not* the case; rather, the wanting system evolved first to guide creatures without consciousness toward stimuli that suggested the availability of food or sex. They suggest that the hedonic system evolved independently so that complex animals could weigh the availability of multiple rewards in the environment, and make decisions regarding the desirability of each, and then initiate the motor sequence required to maximize benefit.

Cocaine induces an artificial drive by blocking the dopamine reuptake transporter protein, resulting in much-increased stimulation of nucleus accumbens neurons. The entire brain is then reorganized. If you smell pizza when you are hungry, and notice that your cocaine dealer is standing next to the pizza parlor, the drive for cocaine will be more powerful than the drive for food (Volkow, Fowler, Wang & Goldstein, 2002). Many addicted patients make the comment, "I don't know why I do cocaine, I don't like how it makes me feel." There is less survival value in using cocaine than in eating pizza. The pursuit of cocaine is not for pleasure, it is a drive. After repeated exposure, the urge to buy and use cocaine is stronger than the urge to buy and eat pizza. This involves a combination of drive and memory.

A neurobiological explanation, then, of why addicted individuals would pursue drugs when the results of using them is so catastrophic is that an imbalance is created between the inhibitory frontal cortical systems and the glutamatergic prefrontal areas that stimulate a demand to the nucleus accumbens core to snap into action to pursue the drug (Bechara, 2005**;** Kalivas, Volkow, & Seamans, 2005). This glutamatergic frontal input also increases the dopaminergic tone at the VTA, producing an excitement in the person to pursue the drug. This ensemble then gives the pressure to act a compulsive quality that is difficult to resist. Multiple authors have noted the similarity of pathways in addiction and compulsion (e.g., Everitt & Robbins, 2005; Kalivas & Volkow, 2005; Panksepp et al., 2004). In summary, our understanding of drug addiction is that the drive system can be more powerful than anything else; pleasure can be disregarded if the drive to use a drug is powerful enough.

The neurobiology of pleasure

As noted above, the subjective sense of pleasure is organized around the endorphin/opiate receptor (OR) system (Panksepp, 1998; Robinson & Berridge, 1993). Endorphins cause increased interpersonal warmth, well-being, and peaceful calmness. Human females administered naltrexone, which blocks ORs, spend increased time alone and report decreased pleasure in social interactions (Depue & Morrone-Strupinski, 2005). Pally (2000) suggested that endorphins "addict" us to human relationships, as demonstrated by the "reunion

response" in primates. Mother/baby pairs hug each other at a measurable rate. Administration of naltrexone increases the rate of reunion, while morphine decreases the rate, as if the monkey pairs are attempting to maintain stimulation of their ORs in a steady state (Pally, 2000). Panksepp (1998) suggested that the pain of mourning involves loss of OR stimulation previously maintained by the relationship. He also suggests that the calming pleasure of touch is produced by stimulation of endorphins (Panksepp & Moskal, 2005).

The calming effect of endorphins apparently has to do with ORs present in the parasympathetic areas, the right ventral orbitofrontal areas with vagal output, and receptors in the periaqueductal grey, rostral ventrolateral medulla, parabrachial nucleus, and locus coeruleus; all of these turn down sympathetic/adrenergic signals (Depue & Morrone-Strupinski, 2005). Hence, the endorphin system is responsible for the pleasure of consummation: a drink when we are thirsty, good food when hungry, sex when we are full of desire—all accompanied at their ending with a parasympathetic glow of relaxation.

But the pleasure system is tied to the drive system. Many of the ORs of the brain are located in the VTA and NAS, where they potentiate glutamate and dopamine processes. Mu OR activity at the VTA during anticipation of reward increased dopamine release to the NAS. Dopamine Type 1 (D1) receptors are necessary for place conditioning to opiates: rats whose D1 receptors are blocked do not learn to go to the side of the T where opiate is administered (all studies here cited by Depue & Morrone-Strupinski, 2005). With this mention of learning, we must consider the next item: learning to be attached to persons—cathexis.

The neurobiology of cathexis

In order to develop an attachment to a person that is remembered and sought—a cathexis—one must remember the interactions that one has had with that person. The above discussion of subcortical areas involved in drive and pleasure—VTA, NAS, accumbens core, periaqueductal grey, etc.—suggests that attachment would not necessarily have anything to do with cortically mediated consciousness. In fact, the way in which attachment is made has to do with the drive system and the pleasure system as they interact. Rats prefer partners that they have mated with before. OR antagonists block the development of this partner preference. In other words, rats need to remember that mating was a pleasant, not just a driven, experience. An odor paired with morphine administration in 4-day-old rats causes conditioned preference, and this effect is blocked by naltrexone, which blocks their ORs (Depue & Morrone-Strupinski, 2005)—the same idea, the experience of the odor has to be paired with the experience of pleasure. This pairing of a sensory experience with another sensory experience increases synaptic connectivity, thereby altering the firing patterns of neurons. This is the molecular basis of learning (Kandel, 2006).

As described by Depue and Morrone-Strupinski (2005), inputs from many higher centers impinge on the NAS; as many as 30,000 cortical and limbic neurons synapse on a single dendrite. The NAS integrates these inputs and sends information on with further compression until it reaches the dorsomedial thalamus, from which motor actions eventuate (Figure 3). Behaviors that have cortical inputs are organized by subcortical structures.

Memory in the amygdala depends on classical stimulus reinforcement: cues acquire positive and negative status. Animals associate contextual features with reinforcement: light conditions, physical features, spatial relations. Memory inputs from the hippocampus are more time-related, such as associations between spatial and contextual interrelations of environmental stimulus and reinforcement. The medial orbitofrontal cortex (MOFC) evaluates the survival value and the desirability of events. The MOFC has inputs from the fusiform gyrus that relate to the structure of faces, as well as inputs from the superior temporal gyrus and sulcus on facial expression and body language (all above from Depue & Morrone-Strupinski, 2005). By combining memories of different modalities of relational interactions inputs from the higher centers, the drive system becomes tuned to a certain kind of person—a look, smells, movements, sensory and behavioral impingements of all types. These memories generate the type of person one finds appealing—just whom one might be looking for when that stranger walks into the room. A key item in these memories has to do with pleasure generated by opiate receptors and with long-term potentiation of AMPA receptors turned into NMDA receptors. This construction of drive, pleasure, and memory is shown in Figure 4.

The transition from more fundamental systems to pursue food, water, and sex, and the pursuit of relationships, is modulated by hormones (Depue & Morrone-Strupinsky, 2005; Insel, 2003; Panksepp, 1998). Hormones having to do with sex also have receptors in the drive system (VTA, NAS). Two hormones that have been studied are oxytocin (OT) and argenine vasopressin (AVP), which are released during mating (OT but not AVP in females). OT triples the baseline

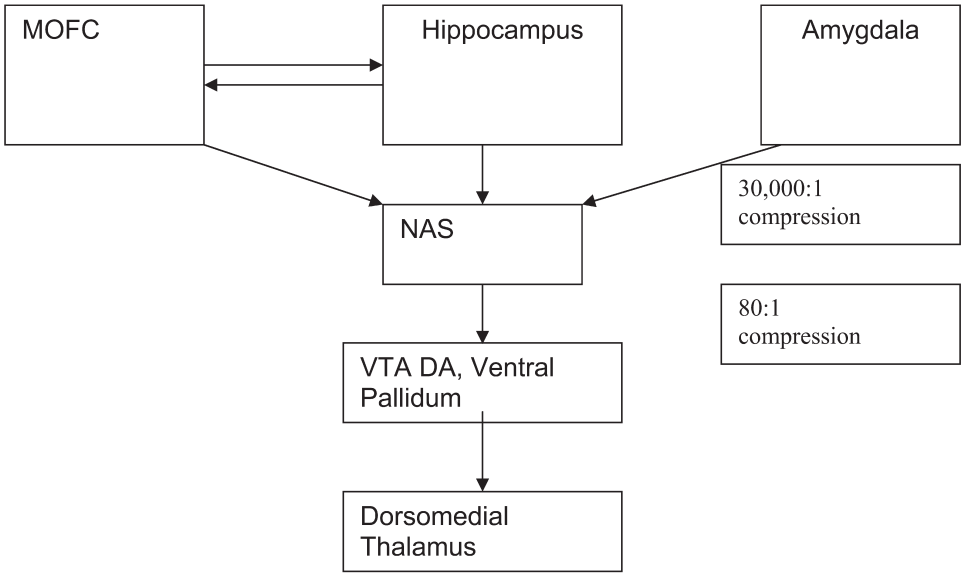

Figure 3. The cathexis system. This figure shows how nonconscious inputs of various types arising from cortical and limbic centers are summated in subcortical centers that drive nonconscious behaviors. MOFC = medial orbitofrontal cortex, NAS = nucleus accumbens shell, VTA = ventral tegmental area of the midbrain, DA = dopamine; "compression" indicates that many cortical and limbic neurons impinge on lower (nonconscious) neural centers to produce behaviors. (After Depue & Morrone-Strupinski, 2005.)

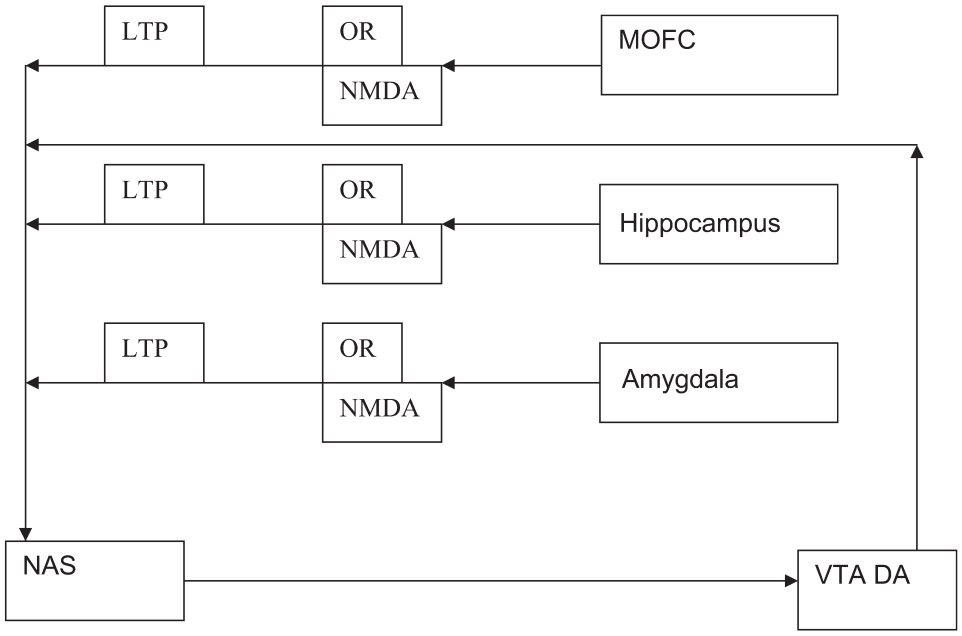

Figure 4. Integration of drive, pleasure, and memory in cathexis. This figure is a simplification meant to illustrate how the way in which drive and memory is instantiated within the brain, and influenced by experience (NMDA potentiation) and pleasure (opiate receptors) to produce cathexis. Note the reverberating interactions of subcortical drive centers (VTA, NAS), limbic memory structures (amygdala, hippocampus) and the frontocortical MOFC. LTP = long-term potentiation, OR = opiate receptor, NMDA = N-methyl-D-aspartic acid (the high-energy phosphorylated and easily triggered long-term potentiated receptor that had formerly been an AMPA receptor), MOFC = medial orbital frontal cortex, NAS = nucleus accumbens shell, VTA = ventral tegmental area of the midbrain, DA = dopamine. (After Depue & Morrone-Strupinski.)

release of endorphin during mating, making bonding more pleasurable. Self-administration of heroin is potentiated by OT. OT antagonists disrupt initiation of maternal behavior, but not its continuation. Similarly OT levels are increased in sexually naïve rats exposed to potential partners, but not sexually experienced rats. OT potentiates acquisition of hippocampal-dependent spatial learning and memory, and the mechanism of OT-related memory stimulation is thought to involve receptors on cholinergic neurons in the basal forebrain that innervate amygdala, hippocampus, and neocortex. (All studies in this paragraph cited by Depue & Morrone-Strupinski, 2005.)

Prairie and pine voles form partner preferences and pair bonds after mating, whereas montane and meadow voles do not. Dense OT receptors are found on the NAS of the prairie vole, but not on the NAS of the montane vole. OT given centrally to female prairie voles facilitates the development of a partner preference in the absence of mating. A selective OT inhibitor before mating blocks formation of the partner preference without inhibiting mating; prairie voles given an OT inhibitor resemble montane voles—they mate normally but show no lasting interest in their mate (Insel, 2003). OT is the hormone that causes humans to feel bonding following sexual intercourse (Panksepp, 1998). The equivalent hormone for prairie vole males is AVP; inhibiting it before mating blocks affiliation, and administering it centrally in the absence of mating causes males to form partner preference. Both dopamine and a hormone are necessary for partner-preference formation (Insel, 2003).

Another hormonal system involved in affiliation is the prolactin system. Prolactin release is essential for the formation of maternal bonding with infants postpartum (Panksepp, 1998).

Again, the drive system is required for all these behaviors, while the emotional quality of the experience and the degree of expression of these social behaviors depends on the concomitant engagement of hormones in the drive system (Panksepp & Moskal, 2005). While hormones have a facilitating effect on drive expression, it is the drive system itself that is crucial for remembering and desiring. Female prairie voles showed a 50% increase in NAS dopamine within 15 minutes of mating, and the dopamine levels remained high for 3 hours. Dopamine antagonists do not block mating, but infusion of D2 antagonist eticlopride into the nucleus accumbens blocked partner preference in the presence of mating. (Both studies cited in Depue & Morrone-Strupinski, 2005.) Dopamine is released in the nucleus accumbens of mother rats following pup exposure. VTA or nucleus accumbens lesions disrupt maternal behavior (Insel, 2003). Day 8 postpartum females prefer pups to cocaine, whereas, at Day 16, cocaine appears more attractive than pups; the change is induced by hormonal fluctuations (Insel, 2003). The VTA and the NAS interact using glutamate and dopamine to form incentive-encoded contextual memory ensembles predictive of affiliative reward (Depue & Morrone-Strupinsky, 2005).

Without dopamine input, one might see the partner one has had sex with and ask, "What's your name?" So, cathexis has to do with an ensemble of drive, pleasure, memory, and hormones—but there is no cathexis without drive.

What about the psychoanalytic notion of a "cathexis" as a libidinal investment? McIntosh (1993) traced the evolution of Freud's concept of cathexis:

> in mental functions something is to be distinguished—a quota of affect or sum of excitation—which possess all the characteristics of a quantity (though we have no means of measuring it), which is capable of increase, diminution, displacement and discharge, and which is spread over the memory-traces of ideas somewhat as an electric charge is spread over the surface of a body. [Freud, 1894a, p. 60]

I believe the neurobiological discussion above is true to this fundamental concept; the energy of cathexis comes from the drive system. The memory comes from diverse inputs—including hippocampal, amygdalar, frontal—that are combined in a compressed way into subcortical centers that produce behaviors related to both the hedonic value and the drive intensity.

McIntosh (1993) said:

> The ideal of a cathexis as something both mental and physical is the foundation for an overall theory, which takes the form of what is nowadays called a "mind–brain identity theory." Two completely worked-out theories of psychic activity, one psychological and the other neurological, are presented and equated point by point. In these terms, a cathexis is both a motivational impetus which increases in intensity, is directed toward the idea of an object, and is satisfied via one kind or another of gratifying activity—and at the same time a charge of electrochemical energy which is built, infuses portions of the brain, and is discharged via a set of neurological processes. . . . In 1898 he wrote to his friend Fliess that he had given up any attempt to establish a physiological basis for his theories, and was resolved hence forth to conduct his inquiries solely on the psychological level, and he stuck to that resolution for the rest of his life. [pp. 682–683]

My assertion is not that there must be "two completely worked-out theories of psychic activity, one psychological and the other neurological . . . presented and

equated point by point," but, rather, that metapsychology must be constrained by neurobiology: a theory has to have a possible basis in the brain. I am asserting that it is possible to return to Freud's question of how psychology and physiology are related and to generate a correlation between the two levels of thinking.

There are thousands of contributions to the debate about the repetition compulsion and the death instinct (Hoffman, 2004). By describing the brain mechanisms that underlie the psychoanalytic concepts of drive, pleasure, and cathexis, and citing the Berridge and Robinson (2003) concept of separate neural systems for wanting and liking, I am setting on a material base the metapsychological discussion that comes next.

Just what lies beyond the pleasure principle?

How might these facts inform our understanding of neurosis? It gives us an idea of the machinery underlying drive, pleasure, and cathexis in a way that informs our activity with patients. Current arguments among psychoanalysts have no means of resolution if the only basis of adjudication is "clinical experience" (Johnson, 2006). If we can use the neuroscience outlined above, we have a material basis for our understanding of patients. In particular, we note that Freud made exactly this step in moving from neuroscience researcher to psychoanalyst: he took his understanding of the brain from his late-nineteenth-century laboratory and applied it to the patients he was seeing as a clinician. This approach has been termed "dual-aspect monism" by Solms and Turnbull (2002). The concept is not to reduce psychology to neuroscience, but to correlate empathic psychoanalytic observations with nomothetic biological studies. Both are accorded validity at their own levels of observation and theory-building, but there must be a congruence where there are overlapping observations. For example, Shevrin (1997) discussed the conflict between the degradation of the drive concept within psychoanalytic theory at the same time that neuroscience was making discoveries that supported its validity. His point was that if we are finding neuroscience evidence that Freud was right about drives, we would not want to simultaneously be making psychoanalytic observations and metapsychological theories that delete drive theory from psychoanalysis.

Our twenty-first-century neuroscience gives evidence that there is a more primitive drive system that is connected by learning to early human relationships. Our goals in finding persons to be close to were shaped by experiences with caregivers/parents/mother. There is nothing clear or conscious about why these relationships were sought: the basis of the learning could have to do with odors, shapes, static or active facial attributes—we have to be mindful that there are things that we could never guess. In fact, one experiment demonstrated that women preferred the odor of men with different immune systems, as shown by having a different human leukocyte antigen (HLA) type, thought to confer superior immune systems to her offspring. In addition, changing the internal hormonal milieu by being on birth control hormones changed the preference to the odor of a man with similar HLA type (Wedekind, Seebeck, Betters, & Paeke, 1995).

There is no standard way to treat a patient with psychoanalysis, because everyone's goal in forming relationships is so particular. The psychoanalyst must remain relatively unresponsive and wait for the patient to show, by making much of nonspecific stimuli in the interaction, just what kind of relationship he or she is seeking—what kind of cathexis has been built from early memories that will be triggered by the presence of the psychoanalyst. To mention a few patients at random, one woman sought a relationship with me where she was sure to be attacked and betrayed, as she was with her mother. One male patient sought to form a relationship with me where he was treated like a sick little girl, a stance that he fantasized as a child to prevent attacks from much older brothers and a father, all of whom were brutal and had alcoholism. One patient sought to form a relationship with me where he appeared submissive but was actually dominating me by canceling hours and was having relationships with all the women he wanted despite his marriage. In these treatment relationships, I tried not to distort the relationship they were seeking with me by explaining that I am a nice person; rather, I used interpretations to show how the unconscious cathexis was in conflict with the more conscious hedonic system, in the context that their problem was that living while unconsciously seeking these kinds of relationships had caused symptoms and made them unhappy.

Anyone who has gone on a diet, only to find him/herself eating a chocolate bar, is aware that conscious intention is in conflict with other forces. The kind of material base described here for psychoanalysis makes using our technique to treat addiction a natural approach (Dodes, 2003; Mann, 2002). But in addition, the neuroscience relationship of drive and relatedness helps to resolve some of the arguments regarding whether relationships are the basis of drives, or whether drives are the basis of relationships (Shevrin, 1997). Freud was right: drives come first. As we follow the development of the brain, we see that the VTA/nucleus accumbens system is active at birth, while frontal inhi-

bition takes 25 years of development before it is fully functional (Schore, 2003). One aspect of amelioration of neurotic conditions has to do with placing drives and affiliational urges dominated by neural structures in subcortical areas under the control of cortical inhibition through the technique of interpretation of the unconscious.

Berridge and Robinson (2003), discussing the distinction between "wanting" and "liking," rediscovered Freud's observation that the differentiation of drive and hedonic brain systems gives us a material basis for intrapsychic conflict. Freud had a complex understanding of this conflict as the basis for defense and for the dynamic unconscious. Conflict is generated by the drive system, shaped in goal by memories of early relationships, being opposed by a more complex and conscious (but certainly not fully conscious) hedonic system. The patient comes to psychoanalytic treatment saying, "My choices are making me sick and unhappy." Defenses are easy to observe using this dichotomy: one watches one's patients' associations as they seek things that they unconsciously know will come into conflict with health and happiness. The drive system is, as Freud said, "More primitive, more elementary, more instinctual than the pleasure principle which it overrides . . . not, indeed, in opposition to the pleasure principle, but, independently of it and to some extent in disregard of it." We all want what we want, urgently, and we must have it that minute—even if we have to pay for it later. This is the nature of the drive system. The extreme manifestation of this system is seen in persons who are desperately unhappy about what cocaine has been doing to their lives, yet who desperately want cocaine.

One way to explain the difference between emotional health and neurosis is that happiness is a byproduct of functioning well; the drive system and the pleasure system line up seamlessly and, as Freud remarked, we never even notice that there are two systems operating because they are perfectly coupled: we want what we like. One way to explain neurosis is that the drive system is seeking relationships based on old models of pleasure and affiliation—relationships that became cathected because of a thousand minor events or childhood fantasies that caused long-term potentiation of neurons in the cathexis system. These connections now trigger the seeking of relationships that are dysfunctional under current living conditions.

If we now turn to the "repetition compulsion" and the "death instinct," we can see that these terms are adventitious. As Freud so clearly explained, a compulsion is a motor act initiated to undo an unconscious hostile impulse. There is nothing "compulsive" about the repetition compulsion. What Freud had been observing in the transference was the eternal conflict between pleasure and the drive to recreate remembered relationships or previously pleasurable fantasies. As he stated, there is no better way to observe this conflict than directly in the room as the patient associates about the psychoanalyst. Our advantage in being able to explain this now has to do with the advances in neuroscience that allow a rough idea of the brain systems that underlie drive, pleasure, and cathexis. Occam's razor dispenses with the death instinct—no need to create an explanation that is already elegantly and parsimoniously explained by metapsychological concepts of drive, pleasure, and cathexis that are well supported by neuroscience research. The concepts of "repetition compulsion" and "death instinct" were created to fix a problem that, in retrospect, never existed.

Cathexis is the result of drive combined with learning and hormones. Rat mothers cannot care for their pups without dopamine release in the VTA/nucleus accumbens, and mother–baby snuggling has something to do with prolactin and oxytocin in the mother and endorphins in both mother and infant (Panksepp, 1998). Cathexis probably has something to do with upregulation of delta fosB resulting in long-term potentiation of the same systems that invoke attachment to drugs—except the target is a specific kind of person.

In *Beyond the Pleasure Principle* (1920g), Freud noted that the hedonic system allows for much of the human behavior that we observe. He described the "repetition compulsion" as the urge to repeat rather than remember and described how this repetition compulsion was present in his transference relationships with his patients. He described the transference as the engine of change in psychoanalysis.

With the above neuroscience explanation, we can understand that the repetition compulsion is the result of drive, learning, and hormones interacting to form "desired" types of persons. In life we are all prone to unconsciously seek these persons, but in psychoanalysis, if the analyst is not too active, the patient begins to describe the exact experiences he or she seeks with this analyst. There is no need to go any further; the repetition compulsion is not really a "compulsion," it is simply the result of early shaping of objects by drive and memory that will subsequently be sought. By the time Freud began to invoke the "death instinct" to explain why people behave self-destructively beyond the pursuit of pleasure, he had moved beyond his clinical evidence, as shown by the odd arguments he began to make about the wishes for death of unicellular organisms. Seeking cocaine is not a manifestation of the death instinct; it is a manifestation of an illness in

the ventral tegmental dopaminergic SEEKING system. Wanting a mother/analyst who will abuse and betray one, a man wanting to appear as his analyst's sick little girl, or wanting to have an analyst whom you can "beat" by not showing up, are all manifestations of pursuing goals that were laid down a long time ago and need to come up for conscious review and conscious decision-making. It is drive and cathexis—the Panksepp SEEKING system, the Berridge and Robinson "wanting" system—that lies beyond the pleasure principle.

The time required for "working through" becomes the time it takes to give up on goals that are urgently desired (think drug addiction) and yet are dysfunctional. The chocolate bar turns on craving via the drive system. It is not eaten because one "has to" lose weight, but only when one has unambivalently decided that one "wants" to lose weight. The drive system is often more powerful than the hedonic system, and one has to be fully conscious and completely committed to the goal of being aggressively athletic rather than eating chocolate bars. If one can see one's urges to have a dysfunctional relationship with one's analyst, it improves one's ability to make good relationships outside of therapy (Hoglend et al., 2008).

Psychoanalysts have held on to their concepts of drive, unconscious, transference, conflict, defense, etc. despite a century of intellectual storms of protest against these entities. The neuroscience to back up these concepts has arrived. Twenty-first-century neuropsychoanalysis is a "psychology for neurologists." This approach has some valuable implications for psychoanalytic theory.

We can see that there has been a dispute in psychoanalysis about whether to correlate metapsychology with neurobiology, as Freud did, or base it on concepts that are solely developed from experiences with practice. By relying on the later, without the requirement of outside validation by other methods (Villa, Shevrin, Snodgrass, Bazan, & Brakel, 2006), we travel from materialism to idealism. A materialist approach invites constant interchanges with members of other disciplines and gives legitimacy to psychoanalysis within the culture because it is allied with science. The idealist approach—developing concepts from treatment without any way of confirming them or discarding them—leads to scientific and cultural isolation; psychoanalysis begins to have a religious quality where differences of opinion can only be settled by argument and persuasion. The present paper shows that this conflict within psychoanalysis began with Freud. His concepts of "repetition compulsion" and "death instinct" were developed with a combination of observation during practice and speculation, unrelated to his understanding of neuroscience.

This approach also bears on the discussions in psychoanalysis about "one-person" versus "two-person" psychology. The neurobiological understanding of brain systems involved in drive, pleasure, and cathexis leads to the conclusion that drive is unrelated: it is a one-person event. Pleasure and cathexis are interpersonally generated; a simple caress by another can cause a surge of endorphins that is calming and reassuring. I am sure the voice of the psychoanalyst can cause an endorphin surge. The psychoanalyst tries to provide a relationship that is attuned, quiet, steady, responsive—to form a therapeutic alliance. Neurotic patients are seeking a relationship that is guaranteed to create a feeling of frustration because their drive system is cathected to old objects. Hence, a patient will create a similar transference neurosis with different psychoanalysts, but the response of the psychoanalyst is unique in that his or her technical skill in propitiating a therapeutic alliance will allow or not allow that patient to do the work of observing the transference problems that he or she is experiencing. If the therapeutic alliance can hold the frustration of the transference, the psychoanalysis will go on until drive and pleasure are sufficiently coordinated to allow the patient to leave the treatment functioning well.

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