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Review

The uncertainty processing theory of motivation

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ABSTRACT

Most theories describe motivation using basic terminology (drive, 'wanting', goal, pleasure, etc.) that fails to inform well about the psychological mechanisms controlling its expression. This leads to a conception of motivation as a mere psychological state 'emerging' from neurophysiological substrates. However, the involvement of motivation in a large number of behavioural parameters (triggering, intensity, duration, and directedness) and cognitive abilities (learning, memory, decision, etc.) suggest that it should be viewed as an information processing system. The uncertainty processing theory (UPT) presented here suggests that motivation is the set of cognitive processes allowing organisms to extract information from the environment by reducing uncertainty about the occurrence of psychologically significant events. This processing of information is shown to naturally result in the highlighting of specific stimuli. The UPT attempts to solve three major problems: (i) how motivations can affect behaviour and cognition so widely, (ii) how motivational specificity for objects and events can result from nonspecific neuropharmacological causal factors (such as mesolimbic dopamine), and (iii) how motivational interactions can be conceived in psychological terms, irrespective of their biological correlates. The UPT is in keeping with the conceptual tradition of the incentive salience hypothesis while trying to overcome the shortcomings inherent to this view.

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1. Introduction

How is motivation induced in the brain? Several motivational concepts have emerged over the twentieth century, owing to the requirement for psychology and neuroscience to explain how the brain generates behaviour and modulates cognition. Two major categories of concepts can be roughly distinguished in the literature. Firstly, energy concepts consider that motivation is part of a homeostatic mechanism energising behaviour. They are at the origin of drive theories, which hold that motivation literally pushes organisms to regulate physiological imbalances and thereby

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satisfy their needs [106,112,113,130,139,239]. Secondly, incentive concepts suggest that motivation is the process that temporarily enhances sensitivity to specific stimuli and produces goal-directed behaviours. Incentive theories generally consider that motivation interacts with the mental representation of a goal object in order to induce expectancy [31,33,72,78,198,232–234]. Motivation therefore appears as quite a complex concept since it has implications for a wide range of phenomena, from mere behaviour to high-level cognition.

These theories capture essential properties of motivational phenomena while describing motivation as a psychological state: drive [113], pleasure [41], 'wanting' [198], emotion [39], etc. Without disputing this interpretation, it must be noted that a psychological state denotes a temporary disposition, which may only be present or absent at any given time in an animal; it is uninformative with respect to the psychological processes that are responsible for its occurrence. The 'wanting' brain system has received much attention in these last two decades (for reviews, see [26,68,197,207]). There is a body of evidence that 'wanting' is mainly mediated by mesolimbic dopamine in the nucleus accumbens, an input system of the striatum. But this does not explain how 'wanting' is psychologically produced. For instance, it is undeniable that the ability to 'want' a reward requires some anticipation of that stimulus, but the way in which anticipation contributes to 'wanting' remains unspecified. Overall, drive and incentive theories seem to describe functional states because they basically insist on the behavioural and perceptual effects of a motivational disposition; they do not attempt to identify the psychological processes that are likely to underpin motivation. As a result, it remains unclear why motivation is shown to extensively affect behaviour and cognition, how motivation can be directed towards specific objects, and how motivations can interact with each other.

The hypothesis, here, is that motivation is the brain's solution to the processing of uncertainty about psychologically significant (appetitive or aversive) stimuli—a view referred to as the uncertainty processing theory (UPT). It is now well accepted that the less uncertain an event is, the more information this event carries [88,220], increasing the opportunity to deal with it effectively. According to this hypothesis, motivation is an information processing (cognitive) system including anticipation and attention, and devoted to collecting information about particular events in order to optimise the reaching of goals. In this view, motivation is not merely assumed to support an animal's activity, just as seeking behaviour does not consist in a behavioural need: seeking is the uncertainty-reducing behavioural strategy controlled by motivation. The UPT insists on the psychological, rather than biological, significance of motivational goal objects. Indeed, animals can be easily conditioned to seek saccharin, even though this substance has no nutritional value [13], but the same could not, strictly speaking, be said of vitamins and trace elements, despite their biological relevance.

The UPT is the theoretical extension of a previously described model of motivational interactions [5–7], and it may be viewed as an improvement on the incentive salience hypothesis [28]. It is suggested that the cognitive and neuropharmacological substrates of motivation are those involved in uncertainty, anticipation, and attention. This view aims to develop an approach where the effects of motivation on behaviour and cognition can be credibly deduced. In addition, highlighting these processes is seen as necessary in order to understand how motivations acquire their object specificity, as well as how they interact together. Before developing the UPT, a critical description of drive and incentive theories is presented. This article mainly focuses on how motivation is induced in the brain rather than on how motivation is learned about a given stimulus.

2. Drive theories

In the first half of the twentieth century, there was a requirement to introduce a scientifically exploitable motivational concept in psychology and ethology, with a view to explaining change in reactivity when faced with constant stimulation. This gave rise to a set of theories in which motivation appeared as a drive providing the 'energy' required to trigger, maintain, and direct behaviour for an organism to achieve its need-related goals—e.g. eating, drinking, copulating, etc. [113,139,264]. Drive reduction was then assumed to follow need satisfaction, placing the organism in a state of rest before this drive or another energises behaviour again. More recently, different authors have restated such a general view [106,112,239]. Drive is assumed to be part of a homeostatic mechanism. In the body, homeostasis is everywhere, from genes to complex physiological regulations, and maintains equilibrium among different parameters (e.g. gene activation, blood pressure, hormonal rates, etc.) by inducing positive or negative feedback adjustments after any change in these parameters. Drive, then, is seen as a signal for behaviour to occur when the physiological state – e.g. blood glucose and cell water rates – of an individual no longer reaches a given set point. At first sight, the homeostatic drive theory seems to be appropriate for the explanation of hunger and thirst, and it was also generalised to other motivations, such as sex and aggression.

The use of the word 'drive' in the literature may slightly differ from author to author. For instance, Hull [113] used it to denote nonspecific energy while Lorenz [139] considered that there was such a thing as behaviour-specific drive energy. For Hull [113], the function of drive was to trigger behaviour, while others suggested that it was also able to maintain and direct behaviour [22,76,123,185,264]. Here is not the place to envisage these subtle differences among the interpretations. The concept of drive simply appeared to be straightforward and powerful – two important criteria in determining the relevance of a scientific theory – because it was in line with the available data. However, drive has been steadily revealed not to be as strong as was initially believed. The concept of drive has been successful for decades due to technological limitations that prevented researchers from collecting precise data about the neurobiological correlates of behaviour.

Conceptually, the so-called motivational energy of drive remained mysterious, but assuming its existence offered a convenient theoretical framework, from which a number of consequences sounded intuitively acceptable. Just as train engines used to require calorific energy from burning coal in order for their mechanics to run, it appeared reasonable to imagine that organisms needed some 'fuel' for behaviour to occur. However, energy concepts do not tell us why organisms behave as they do, because these concepts tautologically suggest that the expression of an activity involves the activation of its drive without allowing an independent measure of that drive. It looks like a vague analogy with energy concepts used in physics. Hinde [102] showed that motivational energy is not presented as physical energy but is nevertheless described in its terms: discharge, store, flow, sparking over, etc. Motivational energy, then, has appeared to be inadequate for physiological explanations, and this has probably contributed to the decline of the scientific claims of classic ethology [48]. While classic ethology was confined to the study of fixed-action patterns in response to specific stimuli, psychology contented itself with the study of the ability of conditioned cues to induce new responses in pigeons and rats. In both cases, energy concepts led scientists to limit their investigations within a stimulus–response (S–R) paradigm. These concepts cannot account for behavioural flexibility, that is, changes in strategy that circumstances may induce in the achievement of a goal. S–R learning tells us that a particular action is consolidated when it is repeatedly associated with a specific stimulus, but it neither

explains why this action is performed nor why animals sometimes decide to behave in a way that is not prescribed by the previously learned S–R associations [31]. When rats do not act within the constraints of experimental design, like that imposed by a Skinner box, they are indeed able to vary their responses to reach a goal according to circumstances (e.g. immediately running away, going round an obstacle, climbing up an object, digging through soil, etc.), even though none of these responses have previously been reinforced.

Although most theorists admitted that organisms had multiple drives [101,139,149,226], the concept of drive was considered as real progress compared with the traditional S–R approach to behaviour, in which a stimulus was supposed to activate a specific learned response. The drive concept seemed to avoid the number of S–R rocketing because each drive is a path that different stimuli and different responses have in common. However, I would put forward some doubts regarding this being a conceptually viable solution for the brain. On account of the large number of stimuli to consider, how can the brain quickly select an appropriate response for a given stimulus without reviewing its whole 'database' of S–R pairings? This difficulty is the motivational version of another one, called the frame problem [146,188], which has remained unsolved in artificial intelligence for forty years. Without going into detail, the frame problem is that of determining which representations are irrelevant in a situation, in order to avoid checking them when we acquire any new information. For instance, opening a window may allow insects to come inside or may produce a draught that causes sheets of paper to blow away. Such consequences are realistic and can be easily deduced. In contrast, opening the window will not change the size of the room, the number of chairs, the colour of the grass, and so on. Although we cannot think about all the consequences of any given action (e.g. it is very unlikely, though possible, that opening the window will kill you because of chemical pollution outside), it is obvious that we do not consider them when they are judged too improbable or simply absurd. Checking such hypotheses would lead to a combinatorial explosion of information processing that would paralyse any cognitive system, even facing such an insignificant event as opening a window. The same is true about drives, assumed to convey a large number of responses from sensory stimulation: as it is a very general process they are unable to select an appropriate form of output.

Empirically, data which argue against the homeostatic nature of drives have accumulated. Some suspicion has emerged with respect to aggression [9], which does not fit the definition of need, but this also seems to be confirmed about hunger and thirst. Firstly, it has been shown that eating behaviour can be elicited by the opportunity to consume food rather than by the exclusive occurrence of a need for food [244,255]. Similarly, rats that are allowed access to a saccharin solution drink more liquid than the quantity biologically required [13]. Secondly, when hungry and thirsty, the behaviours allowing animals to satisfy these needs always stop before the physiological deficiencies – i.e. low rates of blood glucose or of water cell – have disappeared [25]. Finally, the drive reduction hypothesis has been imperilled by the observations that intravenous feeding and the introduction of food or water directly into the stomach are ineffective at reducing appetite in animals and humans [157,164,236,257]. Another type of motivated behaviour sometimes presented in homeostatic terms is drug seeking [130,225]. According to this view, the presence of withdrawal symptoms induce negative feedback, informing the individual that seeking and taking drug is necessary to (try to) reach physiological equilibrium again. However, this does not explain why drug seeking and taking is more frequently reported to occur during euphoria, that is, when the effects of the drug are still there [199].

To date, no drive-dedicated neurons have been highlighted. There is even a body of evidence to argue against the existence of drive centres in the brain. For instance, electrical stimulation of the lateral hypothalamic region was shown to be related to motivation [173], but interestingly, the activation of one single site is able to activate different motivated behaviours depending on the individual's disposition, experience, and opportunities for acting [177,238,241]. Conversely, while some individuals exclusively show eating behaviour whatever the hypothalamic site involved, others exhibit drinking, sexual or aggressive action patterns according to the availability of stimuli and the individual receptiveness to these stimuli. For instance, a rat, for which lateral hypothalamic electrodes only elicited eating behaviour when food was present in its cage, began to drink after a few days when the food was replaced by a waterspout during electrical stimulation [238]. Drinking behaviour then persisted when the food was put back while the rat's hypothalamus was activated. On account of all these problems, drive theories are not a good option for achieving the purposes of this paper.

3. Incentive theories

Theories that present motivation as a parameter responsible for change in an animal's receptiveness to specific environmental stimuli rather than as fuel for behaviour are traditionally referred to as incentive theories. These theories rely on a hypothesis by Tolman [233,234] that animals may form a connection – in Tolman's terminology, acquire a cathexis – between a motivational state (e.g. hunger or sex) and an appropriate goal (food or a partner) during consummatory behaviour, in order that they can subsequently maintain their interest in that goal. Bolles [33] thereby suggested that motivation is not caused by any drive, but takes its origin in the learned expectation of hedonic rewards: a conditioned stimulus (CS) repeatedly paired with an unconditioned stimulus (US) allows an animal to use the former to predict the latter. There has indeed been strong evidence that the ability of the CS to provide information about the US is more important than temporal pairing between a CS and a US for Pavlovian conditioning to occur ([126,193,242], see also [89]). However, assimilating anticipation to motivation remained unsatisfactory because, after all, we seem to be able to predict the arrival of motivating and non-motivating stimuli with the same ease.

A few years later, Bindra [31] decided to return to a more physiological interpretation of motivation while avoiding the shortcomings of drive theories. Bindra [31] thought that animals acquire sensory representations of the environment from the perceptual correlations which they detect among different objects and events. Owing to their regulatory effect on these sensory representations, motivational states were assumed to stimulate perception in order to indicate to the animal which behaviour to adopt in any given situation. This process was able to account for behavioural flexibility, unlike the S–R associations postulated by energy concepts. According to Bindra [31], electrical stimulation of the lateral hypothalamus may elicit various motivated behaviours because that stimulus does not inform the animal about the motor pattern to produce. This motor pattern (pressing a lever, crossing a labyrinth, etc.) depends on circumstances, that is, on what has been learned and what is perceived and considered of interest by the animal at the time. Animal behaviour is opportunistic. A wild animal indeed has more chance of survival if it decides to eat and drink when resources are available while being not very hungry and thirsty, because it is uncertain whether such opportunities will occur soon again. In the same vein, it has also been shown that a reward can partially replace another when the latter is absent. For example, cocaine self-administration in rats is significantly reduced when they are offered to run in a wheel, even though there is no time pressure for either task [58]. Removing the wheel leads the rats to self-administer cocaine again at the same rate as before, a sign that

these animals did not develop any motivation for cocaine during the wheel-running activity.

Although it may be recognised that 'motivational states influence the production of directed responses, not by a direct influence on motor outflow, but by influencing perceptual processes' [31], this view leads an important problem: motivational specificity is not accounted for. Indeed, if the neurobiological control of motivation is equivalent for different classes of rewards, it becomes difficult to determine how particular stimuli can interact with particular physiological states to appropriately guide behaviour [165]. Not only should the sight of food induce hunger in a thirsty animal, but also this animal would be unable to decide between food and water when confronted with the presence of both. Of course, it can be argued that a number of empirical results indicate that physiological cues may acquire control over behaviour, suggesting an influence of physiology on the specification of motivational objects [40,44,57,63,232]. For instance, Davidson [62] exposed rats to shock when they were deprived of food for 6 h and to no shock when they were deprived of food for 23 h, in order that these animals would learn to associate the arrival of shock with their state of deprivation. All rats were then deprived of food for 22 h and subjected to either condensed milk intubation or sham intubation one hour before testing. It was observed that the 6-h deprived rats froze more after condensed milk than sham treatment, whereas the reverse was obtained with the 23-h deprived rats. These results suggest that rats have learned to predict shock delivery depending on their state of deprivation. Davidson [63] therefore argues that physiological cues act as 'occasion setters' whose effect is to modulate the association strength between a CS and a US (see also [107]). But such a model is more inclined to explain vigour rather than directedness of behaviour, since it suggests that there is no a priori connection between occasion setters and specific unconditioned stimuli; this model is therefore subject to the same limitations as that of Bindra [165].

Incentive theorists have a propensity to be interested in the neurobiological determinants of motivation, and the characteristics of these determinants whose motivations are indistinguishable one from another lead to the interpretation of the very nature of motivation in terms of a 'common currency' [31,41,63]. Although it cannot be denied that motivations have to depend on a common 'language' to interact with each other and explain behavioural transitions (e.g. [5,191]), it seems quite obvious that this common currency is insufficient to capture the whole of the concept of motivation. Perhaps one way to reconcile the neurobiological (nonspecific) data with the behavioural (specific) data is to provide a better characterisation of motivation in terms of psychological processes. It is important to circumscribe the psychological processes that constrain an individual's receptiveness to specific stimuli depending on its physiological (e.g. deprivation) state, as well as on learning and context. Two modern incentive concepts have proved to be of special interest: incentive salience, which is known to affect Pavlovian processes [28], and instrumental incentive learning [72]. Both views provide a complementary account of how motivation is neuropharmacologically and cognitively organised in relation to behaviour.

3.1. Pavlovian incentive learning

In nature, animals devote a lot of time and effort to searching for different sources of reward—in particular, food, water, sexual partners, and social interactions. In laboratory, they can also be trained to develop an interest in less conventional sources of reward, such as drugs of abuse—e.g. cocaine, amphetamine, morphine, heroin, and alcohol. How do these stimuli become rewarding? Following the work by Berridge et al. [28,29], it is currently accepted that a reward is not a unitary process, so that answering this question requires some precision. Robinson and Berridge [198] distinguish the 'wanting' process, that would make animals more receptive to certain stimuli, from the 'liking' process, which would designate pleasure felt during stimulus consumption. 'Wanting' seems to mainly involve dopamine systems of the mesocorticolimbic brain region, that is, the ventral tegmental area's projections to the ventral striatum (nucleus accumbens) and to the prefrontal cortex. Dopamine would transform an animal's mental representations of specific stimuli into reward [26], a process called incentive salience. The 'wanting' process can be abolished using different techniques: pharmacological blockade of dopamine receptors [28], neurochemical destruction of dopamine projections [29,237], surgical lesions of the brain regions involved [28], electrical brain stimulation [27], and the study of mutant mice in which the gene enabling the synthesis of dopamine is absent [43]. Behaviour which characterises 'wanting' reduction or suppression is general apathy and difficulty (not due to motor deficiency) to initiate action for reward seeking. In contrast, 'liking' is concerned with GABA systems in the brainstem and opiate systems in the nucleus accumbens shell [28]. The same techniques can be used here, but the 'liking' process is behaviourally measured by reward-induced orofacial hedonic reactions during oral consumption [179,181,260]. The expressive patterns are hypothesised to be reliable indicators of the hedonic valence, from disgust to craving, attributed to a stimulus because there are correlations when it comes to exhibiting pleasure and displeasure among mammals. A number of empirical results show that 'wanting' and 'liking' can be double dissociated. For instance, destroying the mesolimbic dopamine projections using the neurotoxin 6-OHDA leads rats to develop profound aphagia, although they continue to like food, and even learn hedonic associations between food and other stimuli [29,237]. Conversely, electrical stimulation of the lateral hypothalamus keeps reward-seeking behaviours intact, but these animals exhibit negative hedonic facial reactions when they consume a reward [27].

Such results are clearly in favour of the involvement of dopamine in motivational processes. However, it is important to note that this neurotransmitter does not allow animals to learn the incentive value of a reward which has never been previously experienced. Dopamine modulates the previously acquired incentive value about a reward depending on different parameters, such as the individual's physiological state—e.g. food becomes more and more attractive as an animal grows hungrier and hungrier (see [165]). The 'wanting' process controls instrumental performance for a given US, and transfers the incentive value of this US to CSs that have come to reliably predict its occurrence. On account of the requirement of 'wanting' in Pavlovian learning, it is unsurprising that dopamine plays a role in anticipation. For instance, dopamine release is more intense before meal delivery than during feeding [99,145]. In conditioned animals, voltammetry studies show an increase in dopamine levels of the ventral tegmental area before presentation of sucrose [131], cocaine [94], and heroin [128] in comparison with the time these substances are consumed. In humans, PET studies indicate that the striatal release of dopamine is correlated with the anticipation of a video game [129] or amphetamine-induced drug wanting [65,135,174]. Substantial dopamine release is observed in inexperienced monkeys while food is in contact with their mouth, but dopamine ceases to be released as a response to food and becomes strongly related to a conditioned stimulus repeatedly associated with that food [8,137,213,217]. Conditioned stimuli support anticipation because they provide information about reward delivery; they can therefore be used as reliable predictors by animals [88].

A few years ago, Salamone and Correa [207] suggested that the 'wanting' process should be divided into two components, activational and directional. This view relies on the observation that the use of low doses of dopaminergic antagonists, or the neurotoxine

6-OHDA, is sufficient to suppress lever pressing, while food and water consumption remains unaffected [12,59,115,136,206,224]. Low doses of antagonists would impair the activational ('working to obtain') component, rather than the directional ('appetite to consume') component, of reward-related behaviour. For instance, Salamone et al. [208] used a T-maze choice task, in which one arm obstructed by a 0.44-m barrier contained four food pellets, while the other arm contained only two freely available food pellets. When control rats were tested in such conditions, they were observed to climb the barrier on most trials in order to obtain the high-density food. However, when rats received low doses of the D2 receptor antagonist haloperidol (0.1 mg/kg), they chose to climb the barrier less often and preferred to consume the food in the low-density arm. The authors concluded that these rats maintain their appetite to consume food but are much less disposed to work to obtain it. Without denying the possible effects of dopamine depletion on motor systems, it is difficult, however, to determine whether the activational/directional distinction is necessary to account for the data available. In the experiment reported above, how can we distinguish between an impairment of the 'wanting' to work for food and a reduction in the 'wanting' to approach food? If the 'wanting' to approach food is reduced, as Berridge et al. would suggest, it is logical that rats do not work for food—they are not sufficiently hungry to deploy the effort required to climb the barrier. In addition, the expression 'appetite to consume', assumed to characterise behaviour directedness, is confusing with regard to the traditional wanting/liking distinction. Whereas appetite explicitly refers to 'wanting', consumption is related to 'liking' and should not be considered as part of the 'wanting' process.

'Wanting' is presented as a general, dopamine-dependent process that increases an individual's receptiveness to stimuli whose incentive value has already been learned. Insofar as organisms learn about a lot of reward-based events during their lifetime, it is questionable whether 'wanting' can account for motivational specificity. The examples mentioned above indicate that dopamine has similar psychological effects irrespective of the type of reward involved: the 'reward circuit' in the brain is functionally undifferentiated. It has indeed been shown that approximately 75% of dopamine neurons are synchronically activated when animals experience physical contact with a hidden food or liquid during an exploratory phase [200] and the figure is up to 55–70% for conditioned visual and auditory stimuli [213]. Many dopamine neurons in the ventral tegmental area and substantia nigra (especially pars compacta) exhibit similar activations and depressions in situations to which the dopamine neurons of other regions do not respond at all [213]. Of course, dopamine nonspecificity caused by a high synchronicity among dopaminergic neurons should not lead us to think that all these neurons respond in a similar way to identical sources of stimulation. In the striatum, for example, several regions have been identified as being responsible for different categories of behaviour—in particular, the nucleus accumbens (involved in seeking behaviour), posterior dorsomedial striatum (involved in goal-directed behaviour), and dorsolateral striatum (involved in habit formation) (for reviews, see [80,247,262]). However, it is hard to believe that the dopamine-based 'wanting' process would be sufficient to encompass the concept of motivation as a whole. The problem has even become more complicated, as we know that dopamine release is also observed in animals confronted with aversive events, such as a multitude of stressful contexts [95,124,147,154,189]. Finally, it must be mentioned that dopamine release is facilitated by salient, novel stimuli, irrespective of their motivational value [108]. For instance, single-unit data showed midbrain dopamine release when subjects exhibited target-directed saccades in response to the new opening of the compartment door of an item of behavioural apparatus prior to appetitive conditioning [137]. In the light of these results, dopamine appears to be an insufficient pharmacological correlate of 'wanting' and motivation in general.

3.2. Instrumental incentive learning

According to the S–R theory, with which the drive concept is in keeping, instrumental learning stems from a mere association (temporal pairing) between a stimulus and a response. An animal is not assumed to establish a causal relationship between them or to pursue any goal when it has learned to press a lever for reward obtaining. Although it is a well-documented fact that the acquisition of S–R habits results from extensive training [80,262], other forms of instrumental learning have been highlighted [17]. One of them, instrumental incentive learning, denotes the process allowing a value to be assigned to a goal—a view that brings a new light on Tolman's cathexis theory and the study of goal-directed behaviour in general. The instrumental incentive learning theory predicts that an animal will choose a reward (e.g. food) in accordance with its motivational state (hunger) provided that the animal has had the opportunity to experience this reward in the appropriate motivational state beforehand. Such a prediction can be tested using the so-called irrelevant incentive effect [14,71,259]. In one experiment, Dickinson and Dawson [70] trained hungry rats to perform two different actions to get a relevant reward (some food) and an irrelevant one (a 20% sucrose solution). After learning, the motivational state of rats was shifted from hunger to thirst and their performance was assessed in extinction under thirst. It appeared that the incentive value assigned to both rewards under hunger – that is, a high value for food and a lower one for the sucrose solution – was curiously maintained under thirst; rats did not seem to learn that the sucrose solution could quench their thirst when thirsty if they were exposed to this solution when hungry. If thirsty rats were then allowed access to a sucrose solution before training, an increase in their performance was observed when tested in extinction while thirsty. This indicates that preexposure to the sucrose solution was necessary for rats to learn its relevance in relation to thirst. Similarly, devaluation of food with lithium chloride immediately after a training session will only reduce the subsequent instrumental performance of rats for that food provided that these animals are re-exposed to the devalued food prior to the extinction test [15].

It is important to note that the motivational strength of rats that are consuming a food reward for the first time determines their subsequent response rate to lever press in an extinction test [14]. Preexposure to food in food-deprived rats leads to a high response rate, whereas preexposure in non-deprived rats significantly lowers the response rate. Interestingly, the acquired instrumental performance then becomes insensitive to motivational shifts. The instrumental performance of rats whose deprivation state was switched from a high to low level remains similar to that of rats maintained at a high-deprivation state. Such insensitivity of rats to motivational shifts persists unless they have the opportunity to learn about the changed value of the reward in the new deprivation state. As Balleine [14] put it: 'in the absence of incentive learning, an animal trained while hungry and thus presumably inclined to assign a high incentive value to the reinforcer does not immediately learn that this value is conditional on its high-deprivation state and continues to respond in a manner appropriate to this value when shifted to a low-deprivation state'. This fact – that the instrumental performance cannot be directly related to motivational strength – contradicts a main prediction of energy models, that the greater the drive, the more intense is the behavioural response. In contrast, animals have to experience a reward in different motivational states in order to learn to alter their performance.

Despite substantial evidence for the existence of an instrumental incentive learning process across a variety of motivational systems [14,70,72,138], it remains unclear how rewards exert their

influence on appetitive behaviour. One plausible hypothesis is that the insular cortex, which contains gustatory representations [37,203], is involved in the incentive memory for food rewards. The value assigned to a food indeed seems to depend on its taste because lesions in the insular cortex suppress instrumental incentive learning [18]. However, it must be noted that the insular cortex is not involved in assigning a new incentive value to a food reward, but it does control the ability to store information about the changed value of this food reward. Rats initially trained to consume a new item of food under conditions of hunger fail to appropriately reduce their response to that food when stated after being subjected to lesions in the insular cortex [17].

Contrary to incentive salience ('wanting'), the instrumental incentive learning process is insensitive to dopamine antagonists, such as pimozide and α-flupenthixol [74]. It may result from hedonic reactions ('liking'), which are also dopamine-independent. This would explain why establishing physical contact with a reward is necessary in order to learn its incentive value. In all likelihood, a vitamin-deficient rat has no innate knowledge of the food containing the missing vitamin, and can only learn this information provided that the rat has the opportunity to ingest the required food [204]. It is, of course, questionable whether hedonic reactions provide information about pleasure, or consist of a mere sensory reaction to food. Nader et al. [165] suggest that hedonic reactions result from sensory discrimination because, if they were really related to pleasure, they should be expected to affect behaviour. These authors consider that motivation ('wanting') is not double dissociated with pleasure ('liking') but with sensory discrimination. For instance, rats that have to decide whether to turn left or right in a T-maze for different concentrations of sucrose and water maintain their discriminative abilities at the same accuracy level as saline-injected rats after receiving a high dose of pimozide when the cues were water and a 0.0024% sucrose solution. Similarly, when rats with large lesions of the nucleus accumbens have to respond to visual cues indicating the size of an expected reward, their reaction times and response rates depend on the reward magnitude, similar to control subjects [16,38]. Admittedly, the results of these experiments show that abolishing motivation – using a pharmacological or surgical method – does not affect the sensory discrimination ability of animals. However, this does not call into question the hypothesis that hedonic systems play a role in the discrimination task. If hedonic reactions are a reliable indicator of pleasure and allow a value to be assigned to a reward, then the brain substrates responsible for pleasure and those controlling the acquisition of a new incentive value for a given reward should tally.

4. Summary and perspectives

Theories of motivation exist to explain how organisms can behave, while taking into account multiple influences from their physiological state, knowledge about the environment, and the opportunities they have to act. In a sense, motivation is the 'conductor' of behavioural (and cognitive) flexibility, which depends on the individual's ability to process and articulate information from different sources. Any theory of motivation is therefore expected to explain how this can happen. However, it is hard to imagine that current theories of motivation are capable of providing such a unified view.

Three questions seem to be of special interest when it comes to the analogy of motivation as a 'conductor' of behavioural/cognitive flexibility. Firstly, how does motivational specificity emerge from nonspecific neurobiological causal factors? Motivational theories have in common that they provide no convincing framework allowing us to interpret motivational specificity. The link between physiological and neuropharmacological parameters and the organism's goals are unsatisfactory because the psychological concepts used to characterise motivation are nonspecific in themselves. To say that motivation is drive [113] or 'wanting' [28] or pleasure [41] is hardly more informative than to say that memory is informational retention. There is no doubt that motivation is somehow related to these different notions, but it is important to wonder about the concept's capacity to distinguish one motivated behaviour from another. Secondly, how are motivational interactions carried out? The nonspecificity of motivational concepts has a negative impact on the ability to explain motivational interactions; it is hard to understand how one motivation can become solicited while another is inhibited at the same time. Presupposing the existence of distinct, interdependent motivational systems [101,149] is not an acceptable solution because this evades the problem of neurobiological nonspecificity highlighted above. Thirdly, how can motivation affect behaviour and cognition in a broad sense? Motivation is assumed to trigger/modulate behaviour and increases receptiveness to certain stimuli. However, it is also involved in the directedness of behaviour, and influences many cognitive abilities, such as learning, memory, and decision. Any theory of motivation is expected to account for that. In the next section, I will set out the uncertainty processing theory (UPT) of motivation, a general framework that attempts to provide an original answer to these three questions. The UPT derives from a model that was previously developed to explain some particularities of motivational interactions in the fields of ethology [5,6] and psychopharmacology [7].

5. Motivation from an information processing perspective

On the one hand, most theories take it for granted that motivation is an elementary process arising before any cognitive processing of information. On the other hand, it is assumed that motivation actively controls goal-directed behaviours and many cognitive abilities. In my view, these two assertions contradict each other. Elementary processes are limited in scope; they are not expected to distinguish between a wide range of abilities—e.g. spinal reflexes may be necessary to escape danger, but they are not responsible for the way in which danger is dealt with thereafter, or for anything else. Thus, motivation can be reasonably hypothesised to be more complex than usually believed.

The UPT suggests that motivation is the brain's solution to the problem of environmental uncertainty relative to psychologically significant events, whether appetitive or aversive. These events correspond to those whose reinforcing value (positive or negative) has been previously fixed by past experience. One motivational function would be to reduce uncertainty about them through the recruitment of anticipatory and attentional resources, as well as the expression of seeking behaviour (Fig. 1). Seeking behaviour, whether characterised by approach ('pleasure' seeking) or avoidance ('relief' seeking), is indeed required to collect information for goal achievement and therefore contributes to reducing uncertainty. As an information processing system, motivation would then allow organisms to acquire more thorough knowledge about the conditions of the next occurrence for psychologically significant events. This would result in the organism's ability to highlight such events as goal objects. The neuropharmacological substrates of uncertainty, anticipation, and attention are now described, in order to show how their interactions may result in a unified motivational process.

Before discussing the neuropharmacological substrates of uncertainty, anticipation, and attention, which are at the heart of motivational phenomena according to the UPT, it is important to note that these substrates are not assumed to reflect any direct causal relationship with the functioning of the mind: the way the brain works does not informs us in a straightforward way as to

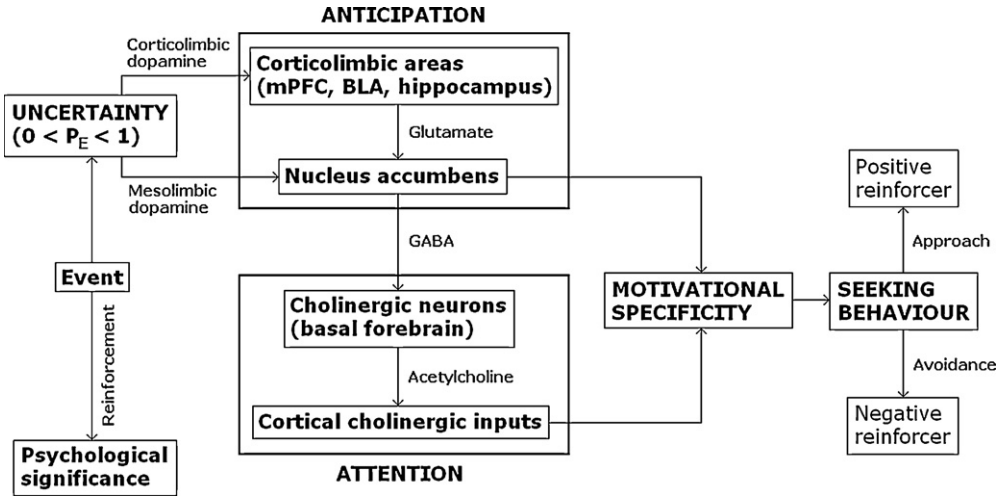

Fig. 1. The uncertainty processing theory of motivation. According to the UPT, motivation consists of an information processing system involving different psychological processes working together. Its expression results from uncertainty about an event ($0 < P_E < 1$) where the past experience of the organism revealed its psychologically significant character (appetitive or aversive). The induced motivation is said to highlight this event as a goal object by reducing uncertainty about the conditions of its next occurrence. Dopamine's role is hypothesised to be crucial for this effect to occur: dopamine is believed to code the event's uncertainty and facilitate availability of anticipatory and attentional resources, in order to allow the organism to acquire knowledge about the world through information-seeking behaviour. Wanting would be the final common path of this information processing. Details relative to the neuropharmacological substrates of these processes are provided in the text (see Sections 5 and 7). *Abbreviations*: P_E (probability of a psychologically significant event), mPFC (medial prefrontal cortex), BLA (basolateral amygdala).

how the mind works. However, the brain mechanisms have well-demonstrated psychological effects (desires, pleasures, aversions, etc.), and it is often difficult to explain why organisms behave as they do without referring to both aspects. The neural correlate of a psychological/behavioural process is a brain component that is accompanied by this kind of specific process on a regular basis. For instance, when it is said that cortical cholinergic neurons are involved in attentional processes [212], this does not mean that these neurons are only recruited for attention or that attention relies only on their activation. This means that cortical cholinergic neurons and attention tend to go hand in hand—because pharmacologically or surgically altering their functioning has some predictable effects on attentional abilities [100,235]. The UPT is in keeping with the view that the mind does not causally reflect brain functioning because the unity of motivational experience is 'dissected' into several psychological components, which emerge from interconnections among different neural circuits.

5.1. Uncertainty

Most changes in the environment are due to variables about which organisms are ignorant, so that a large number of events appear uncertain to them. This means that uncertainty is not an attribute of the events themselves; it is the psychological state of an organism confronted with events whose unfolding, consequences or time of next occurrence is difficult to assess. This also means that the distribution of environmental resources may vary independently of an organism's knowledge about the world at any given time, and therefore that uncertainty may imperil survival. On account of that, it is reasonable to think that uncertain events with some psychological significance are motivating by their very nature. It is even argued that some degree of uncertainty is required for motivation to occur (see Sections 5.2 and 6). Of course, organisms are motivated to approach the things they like (e.g. food, partner, drug, etc.) and to avoid the things they do not like (e.g. predator, noxious odour, electric shock, etc.). But it seems that the uncertainty concerning the next occurrence of these things is necessary to induce motivation about them. For instance, a paramecium, which constantly ingests organic particles homogeneously distributed in stagnant sources of water, has probably no need to be motivated for food. Similarly, a rat or dog subjected to unavoidable electric shock stops try to flee [219]. In contrast, gambling is a very motivating activity in some people because of the uncertainty associated with money gain [89].

Although uncertainty seems to have motivational properties, the fact that it has potentially harmful consequences for survival also suggests that the basic role of motivation is to allow organisms to reduce uncertainty about the liked and disliked events. In classical conditioning, the repeated association between a CS and a US lead the motivated animal to learn to use the former stimulus as a reliable predictor of the latter [88,194,214]. For a prey, predicting the strategies of its predators reduces the risk of being attacked and killed [92,141,187]. This section will set out the neuropharmacological effects of uncertainty in the brain, and attempts to show why motivation is affected by this process. The next two sections explain how anticipation and attention are required for motivation, and strongly contribute to reducing uncertainty about the world.

How does the brain process uncertainty? There is evidence that uncertainty induces phasic dopamine responses in the prefrontal cortex and the ventral striatum from the ventral tegmental area. For instance, Fiorillo et al. [81] have shown that slow, sustained activation of dopamine neurons of ventral midbrain areas developed with increasing reward uncertainty, while such activation did not occur when reward had low uncertainty (Fig. 2). As previously discussed, these brain regions are known to be strongly involved in motivation. Amazingly, however, current theories of dopamine's role make no direct reference to uncertainty, although the uncertainty-related effect of dopamine appears obvious in most of them. Deciding between these theories is often a difficult task; all are confirmed by some facts and contradicted by others [7,26,69,116]. Given that their exhaustive description cannot be reported here, only a few facts showing their probable incompleteness will be mentioned.

Firstly, the hedonic theory [252] suggests that dopamine is the pharmacological cause of pleasure and is therefore released during reward consumption. Although dopamine release is sometimes observed at that moment [249], it occurs with much higher intensity before the arrival of an expected reward [97,145,215,254], and the pharmacological blockade of dopamine receptors in the brain fails to reduce reward consumption [115], as well as the associated orofacial taste reactivity patterns [24]. Nowadays, the hedonic theory is no longer believed to account for dopamine's role. Secondly, the error prediction theory asserts that dopamine release codes

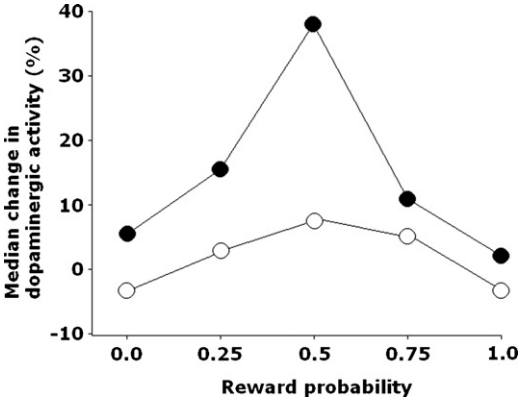

Fig. 2. Effects of reward probability on dopaminergic activity in the midbrain region in monkeys. The sustained activation of dopamine neurons reaches its maximum value at $P = 0.5$, when uncertainty is at its maximum level. It is less pronounced at $P = 0.25$ and 0.75, and totally absent at $P = 0.0$ and 1.0, when uncertainty of reward delivery is nil. The black and white circles represent data collected using two different monkeys. Modified after Fiorillo et al. [81].

the unexpected reward delivery [214]. Not only are dopamine discharges recorded after the appearance of an unexpected reward, or that of a CS cueing reward, but also depression in dopamine rates is observed when reward is undelivered at the time predicted by the CS [183,214]. The theory therefore suggests that anticipation is responsible for dopamine release. However, phasic dopamine responses are so fast – i.e. 50–100 ms – that it is unlikely for an organism to generate any prior cognitive processing of information [192]. Thus, dopamine release must necessarily precede anticipation. Thirdly, the 'wanting' theory states that dopamine increases receptiveness to particular stimuli by converting the mental representation of a 'cold' stimulus into a reward [28]. Despite the amount of evidence for this view, it fails to convincingly explain why the ability to determine the incentive value of reward remains intact after surgical lesions of dopamine receptors in the ventral striatum [16,38] and why dopamine depletion increases the number of lever presses of rats in a progressive-ratio schedule [36]. Fourthly, the reinforcement theory predicts that dopamine enables animals to make an effort to work for reward [207]. The theory is supported by many findings, but this seems to be essentially due to the difficulty in establishing operational tests of the reinforcement process [26,69,116]. The word 'reward' is indeed commonly used 'to denote the undifferentiated effects of reinforcement and motivational arousal' [253]. For this reason, it appears to be as scientifically unusable as the concept of arousal can be [79,210]. Finally, according to the delay theory, dopamine indicates that reward does not immediately follow the CS [47]. For example, lesions of the nucleus accumbens core induce impulsive responding for reward, that is, a preference for small, immediate rewards over larger, delayed rewards [47]. However, the presence of a delay between a CS and the US may be insufficient for dopamine release to occur as such. A conditioned light stops causing a phasic dopamine response after animals have learned the delay between its occurrence and that of the reward [216].

In summary, three out of the five theories described above see dopamine's role in relation to reward expectation—i.e. error prediction, 'wanting', and delay theories. Although none of them capture how dopamine and expectation interact together, they make room for the hypothesis that dopamine is specifically concerned with uncertainty: an expected stimulus is a stimulus that is not there but is assumed to occur at a given time. Such a stimulus is therefore uncertain to an extent that depends on the individual's past experience with it. I hypothesise that when an animal has been exposed to a limited number of CS–US learning trials, the CS is known to be followed by a phasic dopamine response because reward-obtaining behaviour is only contingent on this stimulus—the animal cannot be sure that the US will be delivered. However, after extensive training, the phasic dopamine response ceases for the CS in a fixed temporal schedule because the US is represented as almost certain in this case. In line with this interpretation is the observation that extensive training does not suppress dopamine release when the interval of time between the CS and the US is randomly variable [216]. In other words, the basic function of dopamine would be to code uncertainty—i.e. to represent uncertainty for the brain. As organisms are ignorant about many things in the environment (e.g. Where to find food? When to find a partner? How to avoid a predator? How to interpret a new event? etc.), it is hardly surprising that dopamine release is produced for a wide range of phenomena, irrespective of their motivational value [108]. The present view is therefore more in line with the available data than current theories of dopamine's role, which suggest that dopamine somehow consists of a reward signal. The claim that dopamine codes reward is restrictive and leads to the odd conclusion that this neurotransmitter is an unreliable signal [192].

Only ignorance-based uncertainty has been considered. However, different types of uncertainty can be distinguished, and the influence of other neurotransmitters should be taken into account. Expected uncertainty arising from the unreliability of predictive cues in a task is known to be correlated with acetylcholine release [50,186,256]. This differs from mere ignorance because uncertainty is here due to uncontrollable parameters in the task, which can therefore never be optimally learned. For example, when a cue predicts the location of a target with a given probability, acetylcholine release is higher on incorrectly-cued trials than on correctly-cued trials. In contrast, unexpected uncertainty arises when significant changes occur in the environment and violate the individual's expectations. It is correlated with norepinephrine release and can be highlighted in an attentional shifting task [32]. In the linear maze navigation, in which visual cues unexpectedly indicate the route that rats must take to proceed within a maze, the norepinephrine agonist idazoxan increases the detection of cue-shifts and the learning of new cues [67]. High levels of acetylcholine and norepinephrine are therefore hypothesised to suppress the top-down, expectation-driven information relative to bottom-up, sensory-induced cues [20,196,210], whereas depletion in these neurotransmitters seems to suppress experience-dependent neural plasticity [19,21].

Such subtle differences in the conception of uncertainty are blurred, but would deserve to be considered in appropriate models [266]. These differences are not taken into account in the present article. Although expected uncertainty in probabilistic tasks and unexpected uncertainty may be somehow involved in motivational processes, they imply that organisms are already anticipating the actions leading to a goal when uncertainty arises, and that uncertainty suppresses anticipation. But the next section will rather show how uncertainty may also prepare anticipation, contributing indirectly to goal achievement. Ignorance-based uncertainty is hypothesised to be the releasing factor of motivation in changing environments, where the location of reinforcers (positive and negative) is often not known in advance, whereas expectation-based uncertainties impede activity and should thereby reduce motivation in a motivated organism.

5.2. Anticipation

The ability to anticipate objects and events in a changing, uncertain environment is necessary in order to stay alive. This means that the knowledge an animal has about the environment can alter its performance. For instance, Sibly and McFarland [222] studied time sharing between feeding and drinking in Barbary

doves (*Streptopelia risoria*) and concluded that alternation of the two behaviours was determined by the intensity of the product Deficit × Availability of food and water. Lester [134] noted, however, that this model only describes how alternation depends on immediate physiological and environmental factors; it does not explain what happens when useful information about the availability of resources is lacking. Lester [134] showed, in fact, that 'doves learned to expect rewards at their initial levels of availability and that their response when availability changed was affected by this prior learning'. How does anticipation affect motivation? And how is anticipation related to uncertainty? The studies of goal-directed behaviour offer an interesting approach to these two problems.

Packard [175], and Packard and McGaugh [176], showed that after receiving moderate training (32 trials) to turn right to obtain some food in a cross maze surrounded by environmental cues, rats adapt their behaviour by deciding to turn left when they are placed in the opposite arm of the maze (place strategy). This trajectory change indicates that rats take context into account to reach food: their behaviour is goal-directed. However, after receiving extensive training (56 trials), rats seem to be unable to exploit these orienting cues and continue to turn right in the maze when the start signal arises (response strategy): their behaviour is no longer goal-directed but has become a stimulus-driven habit. There is evidence that habits are not goal-directed behaviours. While devaluing the taste of food with quinine suppresses lever press in rats that have received a limited number of training trials, the procedure has no effect in overtrained rats [3,17,52]. The sensitivity of performance to manipulation of outcome value after moderate training is a sign that behaviour is controlled by goal anticipation. In the same vein, degrading the action-outcome contingency by making food freely available to rats, results in a suppression of lever presses in moderately trained animals, but not in extensively trained ones [17,53,96]. Once again, goal anticipation affects behaviour when animals receive only a few learning trials. These observations indicate that habits consist of the abolishing of goal-directed (flexible) actions in favour of stimuli-driven (inflexible) actions. Habits seem to be formed when reinforcer delivery reaches (quasi) certainty due to the ceasing of the uncertainty-induced anticipatory process. For instance, fixed interval reinforcement schedules, where a response is rewarded after a given time interval, tend to produce habits, that is, a low change in response rate when reward rate is altered [73]. In contrast, habits do not develop, even after extensive training, in a procedure of concurrent reinforcements using fixed intervals for both rewards [53]. Persistence of goal-directed actions here is likely due to the need for animals to allocate anticipatory and attentional resources to both tasks; developing a routine in one task would necessarily abolish the animal's ability to carry out the other. These studies show us that the behaviours concerned with anticipation and uncertainty are precisely those that we commonly recognise as being motivated.

Why? Habits and goal-directed behaviours are controlled by different brain regions. The dorsolateral striatum in rats (or putamen in humans) is crucial for the learning of S–R associations, and renders animals insensitive to outcome devaluation and action–outcome contingency degradation, leading to the development of habits [66,109,247,263]. In contrast, the dorsomedial striatum (or caudate in humans), especially its posterior region, is necessary for the learning of action–outcome associations, which are sensitive to outcome value and its expectancy, and are therefore goal-directed [109,132,247,263]. Inactivation of the dorsolateral striatum leads the behaviour of rats in the cross maze to remain flexible after extensive training. On the contrary, lesions of the posterior dorsomedial striatum result in behavioural rigidity compared with controls, even early in training [261]. Similar results are obtained after inactivation of the hippocampus [176], medial prefrontal and orbitofrontal cortex [55,251], dorsal anterior cingular cortex [10], and mediodorsal nucleus of the thalamus [56], suggesting privileged connections of these brain regions with the dorsomedial striatum [262]. It is important to note that both habits and goal-directed behaviours depend on nigrostriatal dopamine, whose projections ascent from the substantia nigra to the dorsal striatum. The role of nigrostriatal dopamine therefore needs to be nuanced. However, it is now well demonstrated that nigrostriatal dopamine is only moderately involved in motivational phenomena [209,119]. While habits are clearly non-motivated behaviours, goal-directed behaviours are only induced in individuals who are already motivated. In other words, motivation supports goal directedness through anticipation, but it must have been induced prior to the expression of any goal-related behaviour.

As discussed, motivation seems to be correlated with the release of mesolimbic dopamine in the nucleus accumbens [28,69]. The production of mesolimbic dopamine in situations requiring anticipation of rewards and aversive events is well known and has already been set out, so will not be considered again here [26,47,95,189,190,240,265]. We also mentioned the short latency of dopamine responses, which suggests that these responses precede the expression of anticipatory processes [192]. Before considering how dopamine can affect anticipation, it is important to briefly describe the neuropharmacological substrates of anticipation.

How is anticipation implemented in the brain? Several corticolimbic areas, especially the prefrontal cortex, basolateral amygdala, and hippocampus, are known to be involved in anticipatory processes [46,80]. For instance, the delay following a CS in animals trained to work for reward induces expectancy that is processed by prefrontal neurons [49,205]. Watanabe [243] showed that a majority of reward-dependent delay neurons were more active in monkeys which had been preexposed to a preferred as opposed to a non-preferred food reward (banana vs. lettuce), and suggested that the difference in motivational value may explain a discrepancy in retention, retrieval, and anticipation. Lesions of the basolateral amygdala increase impulsivity, given that animals choose small, immediate rewards over larger, delayed rewards [250], and impair cocaine and heroin seeking under a second-order schedule [114,246]. These consequences are also observed following lesions of the nucleus accumbens core, which is massively connected to the basolateral amygdala [45,80]. Although lesions in this brain region do not prevent animals from acquiring conditioned responses [98,180], these responses lack flexibility and are insensitive to subsequent changes – devaluation and revaluation – in reward value [98]. This indicates that the basolateral amygdala is necessary for a CS to retrieve the value of the associated US [46]. The hippocampal formation, projecting mainly to the nucleus accumbens shell, seems to underlie conditioning to contextual cues [218], and may therefore alter their motivational impact on reward seeking. Inactivation of the dorsal hippocampus prevents the restoration of response to cocaine contextual stimuli after extinction [87].

All these corticolimbic areas not only interact using glutamate, but also send glutamatergic projections to the nucleus accumbens [80,116,177]. As the nucleus accumbens, the prefrontal cortex – in particular its medial region – receives dopaminergic inputs from the ventral tegmental area. This is the reason why dopamine should be important for anticipation to occur, although it is in no way a foregone conclusion that dopamine is the neurotransmitter of anticipation. Dopamine would be necessary to facilitate anticipation in different corticolimbic areas, such as the prefrontal cortex and related forebrain regions, owing to ascending dopamine projections from the ventral tegmental area. This would increase the availability of cognitive resources for organisms to deal with these events due to the activation of appropriate mental representations, and would therefore reduce uncertainty about the

achievement of goals. In addition, this theory holds that the nucleus accumbens is the convergence zone for anticipatory signals because it receives inputs from the areas where anticipation is processed, and has indirect connections with motor systems. There is evidence for an excitatory role for glutamatergic NMDA receptors in the nucleus accumbens on dopamine efflux [82,111]. The basolateral amygdala not only sends a dense projection that synapses in close apposition to accumbal dopamine varicosities [122], but also indirectly regulates accumbal dopamine efflux in altering dopamine neuronal firing in the ventral tegmental area via glutamatergic afferents to the medial prefrontal cortex, which in turn projects to this area [121,229]. According to Ikemoto and Panksepp [116], the role of the nucleus accumbens is therefore 'to facilitate flexible approach responses by modulating incentive motivation processes'. This interpretation is compatible with that defended here since the nucleus accumbens receives signalling from brain regions responsible for flexible cognitive processes.

In summary, mesocorticolimbic dopamine is hypothesised to code uncertainty and to facilitate anticipatory processes in several corticolimbic areas. Uncertain events do indeed solicit both dopamine and anticipation, whereas events whose occurrence is (almost) certain stop producing dopamine and anticipation. This suggests that uncertainty facilitates anticipation, and that these two processes are necessary in order for motivation to occur. The nucleus accumbens is the convergence zone for anticipatory signals, and is therefore involved in the expression of flexible behavioural responses, such as seeking. This explains why the nucleus accumbens is essential to motivation, and also why motivation is necessary to collect information about the world.

5.3. Attention

Assuming that motivation requires uncertainty and anticipation, these two processes remain insufficient because they do not account for motivational specificity. While uncertainty may be related to any event, anticipation is based on the individual's background knowledge at any given time. There is no requirement that this background knowledge be appropriate to encompass the conditions under which the motivating event occurred. It is argued that receptiveness to particular stimuli also depends on the ability to recognise opportunities to act by focussing one's attention on these stimuli in the environment. In other words, attention would be necessary for an animal to become receptive to specific stimuli, whereas attentional focus is traditionally presented as a mere consequence of motivation (e.g. 'this dog is looking for food because it is hungry'). The neuropharmacological substrate of attention and its connection with anticipation will be discussed here.

A substantial body of evidence demonstrates the involvement of cortical cholinergic inputs in the discrimination and processing of sensory information, which are defining features of attentional performance. Acetylcholine, the product of cholinergic neurons, has been shown to control selective, sustained, and divided attention, and essentially originates in the basal forebrain [210,235]. Infusion of the cholinotoxin 192 IgG-saporin in the basal forebrain impairs detection of visual stimuli [148] and impedes the allocation of cognitive resources in double task performance [235].

Two types of cholinergic receptors, muscarinic and nicotinic, can be distinguished, both pharmacologically and psychologically. Mirza and Stolerman [159] trained rats in a five-choice serial reaction time task, in which animals had to push a tray flap to collect a food pellet after a visual stimulus was presented randomly in one of five locations. Muscarinic receptors, essentially found in the forebrain, and nicotinic receptors, massively located in the brainstem, appeared to control different behavioural parameters. Subcutaneous injection of mecamylamine, a nicotinic receptor antagonist, increased correct response latency at the highest dose (5 mg/kg). This result indicates that mecamylamine impairs reaction time, making the early stage of stimulus evaluation more difficult. In contrast, injection of scopolamine, a muscarinic receptor antagonist, induced severe deficiency in the expression of correct responses at the highest dose (0.1 mg/kg). Scopolamine impairs accuracy, suggesting a possible involvement of muscarinic receptors in discrimination and motivation, that is, in a later-stage process required for response selection. Both antagonists also had effects in common, such as an increase in omission errors, and decreases in the number of completed trials and of anticipatory responses. Although both types of cholinergic receptors can be found in different brain locations, such as the ventral tegmental area [170,221], only the status of cholinergic projections from the basal forebrain to the cortex is here considered to be due to their undeniable role in attention.

Cortical cholinergic inputs do not reflect a strong topographic organisation, because all cortical areas and layers receive cholinergic fibres in variable proportions [210,212]. They receive afferent projections from various brain regions, in particular amygdala, hippocampus, nucleus accumbens, substantia nigra, locus ceruleus, raphe nucleus, and the pedunculopontine nucleus [210,267]. Cortical cholinergic inputs have been shown to enhance neuronal responses to stimuli in visual, auditory, somatosensory, and prefrontal cortices [4,120,156,163]. In a sense, the role of acetylcholine in attention does not seem to be more specific than that of dopamine in reward. For instance, tonic acetylcholine release appears to be correlated with wakefulness and arousal [125,144], which clearly consist of object-undirected psychological states. However, acetylcholine is also known to enhance the activation of cortical cells that react to the preferred features of a stimulus while inhibiting less prominent responses to other features [210], allowing attentional resources to be used in an effective way. Detection, identification, and the processing of sensory stimuli result from phasic acetylcholine release [155,161,195]. Deficits observed in the ability of individuals to filter irrelevant stimuli 'are attributed to pathological increases in the activity of cortical cholinergic inputs' [211]. Thus, acetylcholine may be viewed as a sort of pharmacological resource that organisms can devote to such-and-such a stimulus depending on the opportunities which they have to act. This property is adaptive since it permits organisms to decide which stimuli are or are not relevant in a situation.

How focussed attention can emerge from such a nonspecific pharmacological factor is assumed to depend on what organisms are able to anticipate in an uncertain environment. The reader will recall that there is evidence that environmental uncertainty induces dopamine release in the nucleus accumbens and that dopamine production can be enhanced by anticipation from the prefrontal cortex, basolateral amygdala, and hippocampus. Sarter and Bruno [210,211] report convincing data showing that dopamine indirectly brings about the greater excitability of cortical cholinergic inputs arising from the basal forebrain when accumulating in the nucleus accumbens. I called the processing of this information cascade the 'dopaminergic–cholinergic pathway' [7] and attempted to highlight its crucial role in motivation. How does this pathway operate? The nucleus accumbens has an inhibitory GABAergic projection to the cholinergic neurons of the basal forebrain. This GABAergic projection is more strongly connected to basal forebrain cholinergic neurons [160] than, for instance, that originating in the amygdala [178]. Perhaps because of the presence, in the nucleus accumbens, of D2 receptors whose action is inhibitory [93], mesolimbic dopamine tends to inhibit the action of GABA in the basal forebrain. On account of the dopaminergic inhibition of the GABA projection, mesolimbic dopamine indirectly increases the excitability of cholinergic neurons of the basal forebrain, causing disinhibition of cortical acetylcholine efflux.

This view is supported by many results. For instance, the systemic administration of amphetamine and apomorphine, two drugs known to enhance dopamine production, reduces GABA release in the basal forebrain [35]. In the nucleus accumbens, haloperidol and sulpuride, two D2 receptor antagonists, have been shown to reduce the FG 7142-induced acetylcholine efflux, but no effect is observed to occur after the injection of the D1 receptors antagonist SCH 23390 [162]. Similarly, the administration of haloperidol blocks the acetylcholine efflux in the frontoparietal cortex, a region receiving ascending projections from the nucleus accumbens, whereas this effect is less marked with SCH 23390. It can therefore be hypothesised that only D2, but not D1, receptors in the nucleus accumbens are involved in the regulation of cortical acetylcholine [162]. Such a result is consistent with the observation that D2 receptors, mainly localised in the striatum [217], exert an inhibitory control over the GABA neurons projecting to cortex [212]. Interestingly, none of these dopaminergic antagonists affect the basic level of acetylcholine efflux in either the nucleus accumbens or the frontal cortex [162], suggesting the existence of a minimal dopamine-independent acetylcholine release.

Conversely, there is evidence that dopamine release may occur in the nucleus accumbens without influencing cortical acetylcholine efflux. Neigh et al. [166] showed that rats perfused with an artificial cerebrospinal fluid and transferred into a new environment exhibit more dopamine efflux in the nucleus accumbens shell than rats maintained in a familiar environment. In addition, they observed a potentiation of dopamine release in rats that received an intra-accumbens injection of amphetamine, irrespective of the environment (new or familiar) to which these rats were exposed. However, despite a transitory effect of novelty on acetylcholine efflux in the medial prefrontal cortex in rats that received the fluid, as well as those that received amphetamine, there was no potentiation of this effect in rats perfused with amphetamine. This unexpected result that dopamine efflux fails to induce a production of cortical acetylcholine (see also [100]), seems to be incompatible with the observations that systemic amphetamine- and novelty-induced cortical acetylcholine efflux can be blocked using dopamine antagonists [1,2,64]. Neigh et al. [166] suggest that the transfer of rats into a new environment was probably insufficient to stimulate the glutamatergic afferents to the nucleus accumbens, a pharmacological influence known to modify dopamine release. Indeed, it has been independently shown that novelty was unable to increase glutamatergic efflux in the dorsal striatum [104]. In these conditions, mesolimbic dopamine is insufficient to inhibit GABAergic projections ascending to the acetylcholine neurons of the basal forebrain.

Although this latter experiment provides no information about the motivation of rats to carry out a task, the UPT interprets the situation above as an example of uncertainty without anticipation. A new environment is uncertain, and thereby produces dopamine efflux, irrespective of amphetamine injections. Once released, dopamine is assumed to facilitate anticipation. But here there is nothing to anticipate, since animals are placed in an unknown environment. The absence of anticipation therefore prevents glutamate release from the prefrontal cortex, basolateral amygdala, and hippocampus to the nucleus accumbens. In accordance with Neigh et al.'s [166] interpretation, dopamine levels then remain insufficient to prevent the inhibitory effect of GABAergic projections to the cholinergic neurons of the basal forebrain. Several results do indeed indicate an effect of anticipation on cortical acetylcholine release. The anticipation of an appetising meal releases acetylcholine in the hippocampus and cortex of rats [117]. After being trained for five weeks to obtain food during a fixed 2-h period, the extracellular concentrations of acetylcholine in rats show an increase of 49% in the prefrontal cortex and 55% in the hippocampus during the 40 min preceding food presentation [90]. In this experiment, acetylcholine was also recorded in these two brain regions during food consumption, but the cholinergic antagonist abecarnil (0.1 mg/kg, i.p.) only prevented the acetylcholine efflux when rats were anticipating food delivery. An assessment of the functional state of central $GABA_A$ receptors in the prefrontal cortex, hippocampus and septum using the binding [^{35}S]TPBS indicated that the acetylcholine efflux during the anticipatory phase, but not consumption, was sensitive to the activation of $GABA_A$ receptors [90]. Increased efflux of mesolimbic dopamine (50–75%) and cortical acetylcholine (150–200%) are observed to occur when rats perform a fixed number of licks to a citric acid solution in order to gain access to a cheese-flavoured food [167]. In accordance with the hypothesis of a dopaminergic–cholinergic pathway, the intra-accumbens perfusion of tetrodotoxin (TTX), a specific blocker of voltage-gated sodium channels, prevented these two effects, but administration of TTX in the dorsal striatum did not block performance-associated cortical acetylcholine efflux. Unexpectedly, the intra-accumbens perfusion of the D2 receptors antagonist sulpiride was ineffective in blocking the cortical acetylcholine efflux, suggesting that activation of the D2 receptors is not necessary for acetylcholine efflux to occur. This seems to contradict the prediction that these receptors are essential to activate the dopaminergic–cholinergic pathway. But it is important to note that Neigh et al. [167] perfused sulpiride in the shell region of the nucleus accumbens. Despite evidence that projections to the basal forebrain massively stem from the shell rather than the core region [210], their influence on the release of cortical acetylcholine appears to be limited. For instance, Himmelheber et al. [100] showed that amphetamine-induced dopamine affected the performance of rats in a sustained attentional task by stimulating cortical cholinergic neurons when the perfusion of amphetamine was carried out in the nucleus accumbens core, but had no effect when the perfusion took place in the nucleus accumbens shell.

5.4. Motivational specificity and seeking behaviour

There are many occasions for an organism (animal or human) to exhibit motivated behaviours, each motivation being directed towards a specific object—food, partner, drug, money, etc. In fact, motivation is even more specific than that: omnivorous animals can be motivated for carbohydrates rather than for lipids and proteins at any given time, one sexual partner is not necessarily equivalent to another, etc. What is the origin of motivational specificity? This question would have seemed superfluous to a number of pioneer researchers, who were convinced that motivational specificity was hardwired in the brain. However, this question now stands out in the light of modern behavioural and psychopharmacological investigations, which indicate that motivational objects are not fixed in advance [14,26]. In addition, we showed in a previous section that electrical brain stimulation was unable to highlight motivational centres [177,238]. Indeed, motivation is presumably controlled by mesolimbic dopamine in the nucleus accumbens and related areas, irrespective of its nature and valence. A possible way of thinking about motivational specificity is that offered by the UPT, since this theory attempts to describe the psychological processes involved in the expression of motivation.

According to the UPT, motivational specificity results from the allocation of anticipatory and attentional resources to a psychologically significant object whose degree of uncertainty is higher than zero. This assertion requires a few comments. When an animal has the opportunity to establish physical contact with such an object in the appropriate physiological (e.g. deprivation) state, it learns the object's incentive value, as well as its sensory features. This allows the object in question to acquire some psychological significance (or hedonic value), which can be mentally represented. Many theorists would certainly consider that the conditions for motivated behaviour to occur are therefore present. Indeed, the

learned 'association' between the animal's physiological state and the object's incentive value seems to be sufficient to trigger seeking behaviour—i.e. approach or avoidance depending on the degree of pleasure felt during physical contact. This is not the view taken here. The fact, for an animal, to have learned what it likes and dislikes in the environment is not enough to organise the animal's seeking behaviour. After all, if motivation is the process controlling goal-directed actions, it cannot be content with initiating this type of action. If this were the case, it would be impossible to operationally differentiate between motivated and non-motivated behaviours, such as habits and even instincts (i.e. inherited action patterns). Not only does motivation initiate particular courses of action, but it also regulates their intensity, duration, and orientation. For this reason, both goal-directed and seeking behaviours are motivated. In nature, seeking is often required in order to learn how to reach a particular goal, whether it is a stimulus to approach (i.e. the animal seeks pleasure) or a stimulus to avoid (i.e. the animal seeks relief). Seeking behaviour is indeed related to exploration, and there is evidence that exploration does not consist of a mere behavioural need, but rather of a strategy for obtaining information about the world. Forkman [83] showed that when Mongolian gerbils (*Meriones unguiculatus*) are offered access to 35 seeds on a black surface or on a similar surface where the seeds are dispersed in sawdust and among barks, they seem to prefer – i.e. they spend more time – in the latter as opposed to the former environment. Gerbils, then, have a preference for an uncertain environment, in which they are offered the opportunity to find the location of seeds. Such a preference is beneficial for survival in nature and suggests that seeking behaviour is the way for motivation of any kind to be expressed. Seeking is the behavioural strategy resulting from the activation of motivation as an information processing system, and allowing organisms to collect information about the world in order to facilitate goal reaching.

In summary, anticipation and attention contribute to reducing the uncertainty of goal achievement through seeking behaviour. The expression of anticipatory processes is not only required to interpret any object representation as a goal, but it is also necessary to form 'hypotheses' about the behaviour to adopt in any given circumstance. In addition, attentional processes are essential to extract relevant data relative to goal achievement, and therefore to 'test' behavioural hypotheses. According to this view, it is unsurprising that the introduction of food or water directly into the stomach does not suppress appetite in animals and humans, as there is no involvement of anticipation and attention in this process (see Section 2). Similarly, it can be explained that unexpected events, such as noxious tail pinch, may fail to elicit a detectable increase in striatal dopamine concentration [248]. According to the UPT, an unexpected event is an event whose probability of occurring was nil (or at least, close to zero), that is, not uncertain at all. As a result, no dopamine signal is produced. The capacity of anticipation and attention to provide information about the world has the logical consequence of highlighting specific objects whose incentive value has already been learned; they allow receptiveness to these objects to be increased through mental rehearsal. Thinking repeatedly about an appetising food or a sexually attractive mate may dispose an individual about these stimuli. To consider anticipation and attention as parts of motivational processes explains why these processes are not homeostatic, that is, why organisms can become motivated by particular stimuli in the absence of the appropriate physiological state (see Section 2).

6. Motivation requires uncertainty

The suggestion that motivation requires some degree of uncertainty in order to occur may sound excessive. However, assuming that uncertainty about an event is due to ignorance of what comes next, goal attractiveness can be shown to result from uncertainty-related changes that take place in external and/or internal variables. It has been suggested that a rat overtrained to learn to press a lever or run a cross maze for food reinforcement develops S–R habit, which is non-motivated behaviour because the overtrained rat's uncertainty of food delivery becomes nil. This does not mean that the rat is no longer able to exhibit food seeking (i.e. motivated behaviour) when the environmental conditions are altered—e.g. the required number of lever presses is increased or food is unexpectedly placed in the opposite arm of the maze. In this case, the animal perseveres in responding as previously – habits are 'residuals' on goal-directed behaviours – but may use new strategies when it realises that food does not follow: the lever is pressed a larger number of times, the rat explores the surroundings, etc. For the rat, the fact that habitual responding no longer results in food means that its delivery is not as certain as initially learned, leading to reinstatement of food-seeking behaviour. Similarly in humans, gambling is motivating because despite there being a lot of losers, some people earn much money, making the outcome uncertain. If there were only losers, gambling would be non-attractive because the probability to win would be zero. If there were a guarantee to earn much money every time, gambling would end up losing its attractiveness because access to financial resources that is too easy dampens the interest in money.

Of course, it can be argued that the need for money, as well as the need for food, is a much more convincing source of motivation than uncertainty. Accordingly, food seeking occurs in food-deprived individuals, and gambling exists because people consider that they do not have enough money. At first sight, deprivation (or the need for something) is unrelated to the level of uncertainty which an individual may encounter. However, this viewpoint can be reversed if we adopt an evolutionary perspective—in which notions such as deprivation and deficit do not really make sense. For instance, to say that hunger and thirst are physiological deficits hardly explains how they can solicit motivated behaviours to eat and drink. It is more advantageous to suggest that hunger and thirst are bodily indicators that the environment is poor in food and water; these indicators would provide the organism with an estimate of the degree of certainty with which appropriate resources are expected to be found in this environment. As a result of deprivation strength, individuals therefore come to exhibit information-seeking behaviour – which may consist of moving to another place – more or less vigourously. Supporting the hypothesis that deprivation is an indicator of uncertainty is the observation that there is a correlation between food and water deprivations and dopamine release in the midbrain region [165]. Since in this case there is no particular reward to point to, the UPT's proposal that dopamine codes uncertainty is reinforced. In addition, the stimulating effects of drugs that induce dopamine release may be better characterised as enhancement of exploration rather than general activity, because the actions that result depend on environmental conditions [116].

In summary, the assumption that motivation requires some degree of uncertainty has theoretical justification, even though empirical investigations are necessary to determine the exact conditions of its application.

7. Uncertainty: an interface between wanting and reinforcement?

To suggest that mesolimbic dopamine is an uncertainty-induced pharmacological parameter facilitating anticipation is in accordance with the presumed role of dopamine in 'wanting' [28]. The 'wanting' process 'anticipates reward and motivates instrumental behaviors' [168]. This theory does not indicate how dopamine and

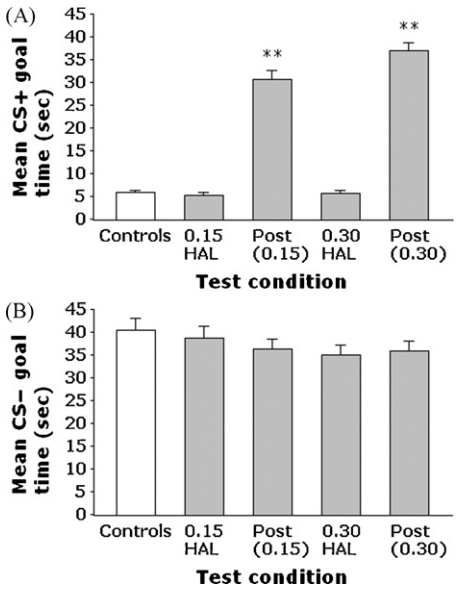

Fig. 3. (A) The performance of haloperidol-injected rats (0.15 and 0.30 HAL) is similar to that of control animals when a CS predicts the presence of a reward (intravenous heroin) in a goal box. But the performance of the rats is decreased the next day (0.15 and 0.30 POST), whereas animals are tested in the absence of haloperidol. (B) When the CS predicts the absence of reward in the goal box, no alteration of performance is observed in any condition. Modified after McFarland and Ettenberg [151].

anticipation interact together, but the view presented in this article may bring an answer to this problem. However, a number of researchers consider that dopamine is, in fact, more inclined to affect reinforcement than motivation [36,38,207]. To my knowledge, McFarland and Ettenberg [151] obtained one of the most convincing results in favour of a role for dopamine in reinforcement. These authors trained rats to run an alley to reach a goal box in which they received intravenous heroin. In one group, a conditioned olfactory stimulus (CS+) predicted the availability of heroin in the goal box, whereas in another group a second olfactory stimulus (CS−) predicted its absence. After training, the effect of haloperidol (0.15 and 0.30 mg/kg, i.p.) was tested on running performance the first day. The performance of the rats was also recorded the next day in a haloperidol-free condition. The results for the CS+ trials (Fig. 3A) indicated that haloperidol had no effect on the behavioural performance of the rats compared to control animals on the first day, suggesting that dopamine does not affect the motivation to reach the goal box. In contrast, rats ran more slowly than controls on the next day, when no haloperidol was administered. Haloperidol therefore seems to have decreased the reinforcing (pleasant) effect of heroin, leading to a reduced motivation to reach the box. No difference in performance was observed between test animals and control animals for the CS− trials (Fig. 3B).

At first sight, these results appear incompatible with the 'wanting' hypothesis, as haloperidol did not impair the running performance of rats on the first day; these animals 'want' heroin as much as the control animals do. Of course, haloperidol only prevents the activation of D2 receptors; it does not act on D1 receptors, which are also present in abundance in the nucleus accumbens [214,247] and have been shown to play a role in motivated behaviour [34,77,169]. D1 receptors might therefore compensate the inactivation of D2 receptors, due to interactions existing between them [127], explaining why motivation has remained unaffected. However, another interpretation – which reconciles both the reinforcement and 'wanting' hypotheses with respect to dopamine's role – is favoured. Before considering this interpretation, an important comment must be made. McFarland and Ettenberg's [151] data can be interpreted in terms of incentive learning (see [17]), a view not considered by these authors. They rightly argue that 'it is only after subjects experience the reinforcer under dopamine antagonist challenge that one typically sees a weakening in their behavioural output—an interpretation suggestive of a motivational change that is only secondary to a deficit in reinforcement function'. As previously discussed, preexposure to a reward in a given motivational state does indeed determine the behavioural vigour of an animal in obtaining that reward thereafter.

What follows is a unified interpretation. Assuming that mesolimbic dopamine is the way for the brain to represent uncertainty about an event, the haloperidol-induced decrease in dopamine sensitivity should have led rats to underestimate uncertainty about obtaining heroin in the goal box. The reduction in heroin uncertainty on the first day, therefore, would have suppressed the reinforcing property of this reward the next day. How? On the first day, rats received a haloperidol injection (which inhibited D2 receptors) after being subjected to a moderate number of training trials. This moderate number of trials was probably insufficient to cancel out the rats' motivation – i.e. to induce habit formation – for the running task. But it is reasonable to think that this motivation was reduced following the haloperidol injection. In human addicts, for instance, the use of haloperidol results in decreased craving [23,84]. Nevertheless, the performance of rats remained similar to that of the control animals because they were not given the opportunity to experience heroin reward in this new motivational state. It is only during physical contact that the rats updated – i.e. reduced – the reinforcing value of heroin. The next day, when the rats' motivation was adjusted to the reinforcing value of heroin acquired the day before, these animals therefore ran more slowly. Uncertainty would then play a role in reinforcement [81], though indirectly. This viewpoint is reasonable because reward uncertainty is reinforcing in many situations, from the mere preference for variable over fixed schedules in rats to gambling in humans [89].

It is possible to reconcile the motivational and reinforcement processes with respect to dopamine's role when dopamine release in the mesocorticolimbic region is assumed to result from uncertainty. Both processes require uncertainty and dopamine, but not in a similar way. While dopamine is assumed to code uncertainty in the brain and is directly involved in motivation, this neurotransmitter does not constitute the substrate of reinforcement, at least in the mesocorticolimbic region. Underestimation (or overestimation) of uncertainty about a stimulus would have the indirect effect of decreasing (or increasing) its reinforcing value. The next section examines the consequence of the UPT as pertains to the interpretation of motivational interactions.

8. Motivational interactions

Carrying out two activities simultaneously is often a difficult task. At first sight, the reasons for such a difficulty are to be found in physical or motor incompatibility—e.g. feeding and drinking at the same time is impossible, and fighting and fleeing involve two antagonistic behavioural patterns. However, a reduction in performance is also observed when both activities are carried out using two compatible sensorimotor pathways. For instance, in humans, when two types of reactions (motor vs. verbal) have to be produced in response to particular stimuli (auditory vs. visual), a decrease in reaction times is observed compared with control subjects carrying out both tasks separately ([171], see also [11,103,228]). In animals, the performance of rats in an operant task leads them to underestimate temporal information ([133], see also [75,142,268]). Interference is due to the fact that motivations are in competition with each other to express themselves. Even in the case of

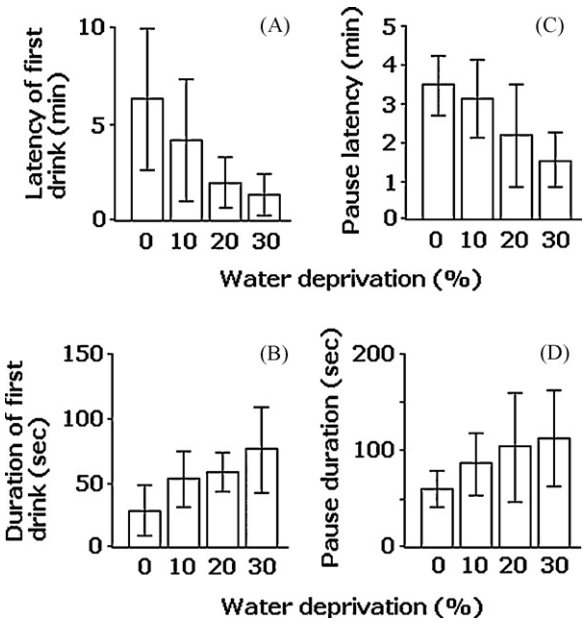

Fig. 4. The interaction between feeding and drinking is gradual (nuanced) rather than abrupt (sudden) in rats. The latency and duration of both behaviours depend on the strength of the motivations involved. Modified after Roper and Crossland [202].

physical/motor incompatibilities, if one motivation could not really overtake the other, organisms would probably try to feed and drink or fight and flee at the same time, leading to pathological patterns of behaviour. I argue for the view that the process of motivational inhibition is related to anticipation and attention whatever the situation, and that its consequences on behaviour are easy to derive from the UPT. Before developing this view, different conceptions of motivational inhibition will be set out.

8.1. Conceptions of motivational inhibition

The processes of motivational inhibition were extensively studied a few decades ago [51,60,86,91,105,110,134,140,150,172]. One issue that has needed to be resolved was to determine whether motivational inhibition is gradual (nuanced) or abrupt (sudden). Gradual inhibition means that different motivations may be simultaneously present in an individual, whereas abrupt inhibition means that the expression of one motivation suppresses that of any other. In the former case, the time of activity transition depends on the intensity of conflicting motivations, whereas it is independent of that intensity in the latter case. Despite much controversy on this topic, most studies seemed to indicate that activity transitions are gradual rather than abrupt—and that those which show the opposite were methodologically ambiguous [105]. Roper and Crossland [202] demonstrated this fact through the study of how hunger interacts with thirst in rats. If the transition between feeding and drinking is gradual, it should occur at a time in accordance with a particular strength of thirst. In contrast, this transition should be independent of thirst if it is abrupt. Their results indicated that, depending on the degree of water deprivation of rats; latency before drinking decreased; the duration of the first drink increased; latency before the first stop in feeding decreased; and the duration of the first stop increased (Fig. 4A–D). As activity transition is contingent on the strength of thirst for each parameter, the gradualist model is preferable. In any case, it is unlikely that activity transitions are generally abrupt because then each new activity would appear sudden and frenetic, and this is not borne out by most observational data [201].

A more recent, neurobiological approach to the problem of motivational inhibition stems from the work by Redgrave et al. [153,191]. They suggest that the input nuclei of basal ganglia (e.g. nucleus accumbens) select the most salient inputs from different corticolimbic areas, while the output nuclei (e.g. substantia nigra pars reticulata) select the appropriate motor channels. These selection mechanisms require the existence of a 'common input currency' so that all input signals can be compared with each other. They would implement the principle of 'winner-takes-all'. The inputs can be induced not only by a target stimulus in the environment (e.g. some food or a predator), but also by contextual stimuli whose influence is able to boost the effect of the target stimulus. The theory is that selection is caused by inhibitory signals from the striatum [158,223], preventing concurrent signals from affecting the final output. When a decision has been made, a mechanism is assumed to quickly clean switching in order to enable basal ganglia to be sensitive to a newflow of information. For that to happen, excitatory inputs to the subthalamic nuclei are thought to stimulate the basal ganglia's output nuclei ascending to the brainstem and thalamic areas before the next occurrence of the striatal inhibitory signals. These excitatory inputs are thought to reset the system, making a new selection process possible. Owing to its effect on the responsiveness of striatal neurons, the theory holds that dopamine controls the timing of behavioural selections and would induce readiness of corticostriatal synapses to change these selections. This view is an interesting solution to the action selection problem. However, it contents itself with offering a neurophysiological interpretation of motivational interactions—just as older studies offered a simple behavioural approach to that process. If motivations are psychological phenomena, a psychological theory of their interactions also needs to be formulated.

To my knowledge, only one psychological theory of motivational interactions has been put forward: Cabanac [41] holds that pleasure is the common currency guaranteeing the trade-off necessary for achieving the ranking of concurrent motivations. In other words, in his view, organisms decide to commit to the activity that maximises pleasure. For instance, under some conditions, hens favour gaining access to gain access to a nest-site over gaining access to food [54], but they prefer feeding to dust-bathing or foraging [184]; pigs prefer feeding to thermoregulating [118], while steers prefer thermoregulating to feeding [143]; sheep prefer drinking to feeding [227], addicted rats take ethanol to the detriment of feeding and of social hierarchy [258], etc. While classical ethologists, such as Tinbergen [231], postulated that the satisfaction of the most urgent need takes priority over any other, they provided no information about the ranking process of these needs. The pleasure maximisation principle appears to be the solution to this problem, as pleasure is assumed to be a sort of psychological indicator of the need's strength. Cabanac [41] points out that '[p]leasure serves [...] to provide the motivation for eliciting behaviour that optimises physiological processes'.

Despite the existence of much data supporting this view (see [42]), I would question whether motivation is a simple function of pleasure—the pleasure maximisation principle is too straightforward. Firstly, as already shown, the motivational strength developed concerning a stimulus seems to depend on the degree of pleasure experienced during its consumption [74,151,152], but there is evidence that motivation and pleasure are controlled by different neurotransmitters in the brain [28]. Secondly, the most motivating stimuli can only produce the maximally pleasant activities when there is no cost to the decision. The pleasure of carrying out an activity may differ from that of obtaining reward, and this may therefore affect performance. For instance, studies on concurrent reinforcements implicitly suggest that increasing the price of a reward (i.e. the number of lever-press responses required to obtain it) decreases motivation for that reward, because a decrease

in consumption arises proportionately to demand [30]. The pleasure maximisation principle stipulates that an increasing demand becomes more and more unpleasant, and therefore undermines the animal's motivation to obtain the reward. But is this what really happens? When a cock accepts pressing a lever once to see a congener in an adjacent cage, but does not accept a higher number of lever presses to obtain the same reward [230], that is, after increasing the price to be paid, can we say that the higher demand reduces the cock's motivation to see a congener? Or does this merely mean that the cock's motivation is not sufficiently strong at the beginning to allow the animal to respond to a higher demand? In this latter case, the observed reduction of consummatory acts is not due to a decrease in the animal's motivation, but to a rise in its reactivity threshold; the critical motivational value required for the lever-press behaviour to occur has been enhanced. There is evidence for this interpretation. For instance, Pelchat et al. [182] showed that electric shocks coupled with the access to a sucrose solution led rats to avoid consuming this solution. However, they also showed that rats remain attracted by the sucrose solution when they could access it without receiving shocks. Such a result clearly indicates that the reduced reactivity of rats is due to the knowledge-associated negative consequence of sucrose-directed behaviour rather than to a shift of motivation from a high to low level. The effect of electric shocks on drinking seems to be similar to that induced by the increase in the number of lever presses required to obtain a reward: both events raise the price to pay to an intolerable level, so that animals prefer to give up the activity – which has become unpleasant – despite their motivation remaining intact.

8.2. Attentional control of motivational interactions

According to the UPT, the ability of one motivation to inhibit another is unrelated to pleasure, but is due to the limitation of attention in organisms anticipating two different events within the same timeframe. This interpretation has been made into a model [5–7] and will be only briefly discussed here. Neuropharmacologically, mesolimbic dopamine is considered to be the main causal factor of motivation, and motivation is assumed to correspond to the activation of the dopaminergic–cholinergic pathway. It is indeed important to remember that mesolimbic dopamine, which is presumably necessary to facilitate anticipation, is known to excite cholinergic neurons of the basal forebrain, which project to the cortex in order to control attention [212]. The more a stimulus activates the cholinergic–dopaminergic pathway, the higher the motivation for that stimulus then becomes. As the UPT includes attention in motivation, it is therefore unsurprising that the limitation of attentional resources – i.e. the fact that it is hard for an organism to pay attention to more than one event at a time – has an impact on motivational interactions: the more a motivation is activated, the more the activation of another, concurrent motivation is difficult. This means that the activation of the dopaminergic–cholinergic pathway by an event makes this pathway less available for another event, pre-inhibiting its potential motivation. The UPT conceives mesocorticolimbic dopamine and acetylcholine as the pharmacological common currencies required for motivations to be compared with each other [24,210].

The process described above is represented by means of an attentional threshold whose intensity value for a motivation depends on the number of motivations solicited in an individual at any given time. Fig. 5A shows that the intensity of the attentional threshold is close to zero for one motivation when this motivation is the only one to be activated at the time in the organism (e.g. the search for food is the single thing of interest at the time), so that the attentional threshold of this motivation's causal factors can normally be reached with low mesolimbic dopamine levels. However,

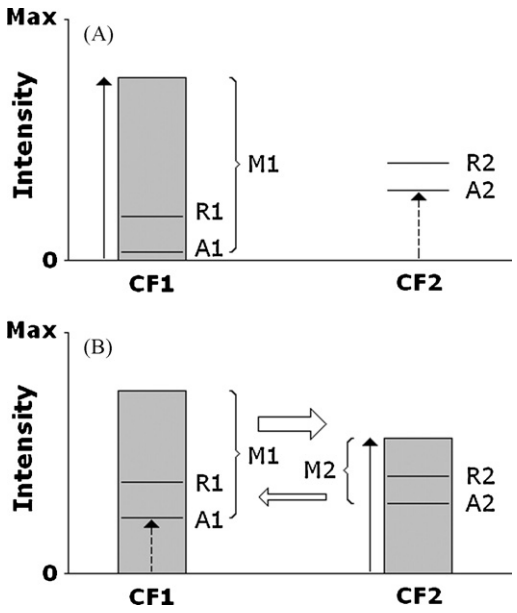

Fig. 5. (A) The increase in causal factors (CF1) of a motivation beyond its attentional threshold (A1) induces motivation M1. This raises the attentional threshold (A2) of another potential motivation, even in the absence of causal factors (CF2). This is a case of pre-inhibition. (B) If CF2 overcomes the A2, a new motivation M2 is induced. In turn, M2 inhibits M1 by raising the A1. It may be observed that both reactivity thresholds (R1 and R2) are raised by the attentional thresholds, as the model postulates that the former are necessarily of higher intensity than the latter. Simultaneous solicitation of M1 and M2 generates attentional interference (white arrows), which here is strongest from M1 to M2.

this motivation makes the appearance of concurrent motivations in raising the attentional threshold of concurrent causal factors more difficult. This attentional threshold cannot really be defined operationally in an experiment, but it can be indirectly assessed through the individual's behaviour. Indeed, the theory suggests the existence of a reactivity threshold, whose intensity value for a motivation indicates the degree of inhibition of a behavioural response. The higher the reactivity threshold's intensity value, the more a behavioural response is then inhibited. The reactivity threshold's intensity value is assumed to be always higher than that of the attentional threshold—allowing organisms to pay attention to the object of their action before carrying it out. When two motivations compete to express themselves in behaviour, they inhibit each other by raising their attentional threshold, and this has the consequence of raising their reactivity threshold as well. The latter is indeed 'pushed' by the attentional threshold in each motivation (Fig. 5B). This process may explain why two tasks carried out simultaneously exhibit higher reaction times than when they are performed separately. In addition, attentional interference resulting from two conflicting motivations may explain the appearance of errors and inaccuracies in performance. Interestingly, the model captures these two effects of the limitation of attention on behaviour – i.e. increased reaction times and the presence of errors/inaccuracies – through two distinct mechanisms. As shown, there is evidence that both effects are pharmacologically controlled by two distinct cholinergic receptors in the brain. While the blockade of nicotinic receptors impairs reaction times, that of muscarinic receptors affect accuracy of responding in rats [159].

In summary, motivational interactions and their behavioural effects are believed to depend on how an organism's limited attentional resources are shared among different tasks, a process controlled by the dynamical property of attentional thresholds. When a motivation increases, because the anticipatory attention process boosts it, this causes the inhibition of the organism's other motivations, owing to a rise in their attentional threshold. Con-

versely, when a motivation decreases because its object becomes of weaker interest, there is a disinhibition of the organism's other motivations due to a lowering of their attentional threshold.

A number of results can be found in the animal and human literature to support this interpretation of motivational interactions [58,85,245], and I would like to illustrate it through a study by Culshaw and Broom [61]. These authors assessed the responsiveness of 6-day-old chicks to sudden illumination of a 3-W torch-bulb during feeding and preening. This stimulus brings about a startle response that tends to interrupt (inhibit) the ongoing activity for a time. In one group, bulb illumination happened two seconds after the chicks had started feeding or preening, and in the other group, it occurred at the end of the feeding or preening bout. The results indicated that the chicks disturbed at the end of the feeding bout remained motionless for longer, fixating the illuminated bulb, than the chicks disturbed at the beginning. According to the UPT, the easier inhibition of feeding at the end of the bout is due to the decreased intensity of this behaviour's motivation, causing a decrease in the attentional threshold of any other concurrent motivation's causal factors. Disinhibition of the motivation directed towards bulb illumination (mild fear?) therefore leads the chicks to pay more attention to the bulb. During preening, the observations were similar and remained compatible with the UPT's predictions: the chicks disturbed at the end of the bout ceased their activity almost immediately while those disturbed at the beginning continued to preen for some time, though more 'frantically', after bulb illumination. The UPT can also explain this higher frequency preening at the beginning of the bout. Such an abnormal pattern would be due to the inability of the preening motivation to totally inhibit the chicks' motivation directed towards the bulb, bringing about an attentional focus of chicks on its illumination. Preening is therefore performed with obvious signs of nervousness. Why are these signs of nervousness observed with preening, but not with feeding? The model predicts that the increased rate of attentional thresholds depends on the priority of conflicting motivations. This increased rate is assumed to be of stronger intensity when it originates from a high-priority motivation and moves towards a low-priority motivation [5]. It is undeniable here that feeding is of a higher priority than preening in relation to the bulb-directed motivation. As a result, the motivation to feed raises the attentional threshold of the bulb-directed motivation more than the motivation to preen, leading to an absence of attentional interference on the feeding activity at the beginning of the bout. In contrast, such attentional interference does occur in the case of preening.

9. Conclusion

According to the UPT, motivation is an information processing system – which includes anticipation and attention – whose role is to reduce uncertainty about psychologically significant events through information-seeking behaviour. This view is in keeping with incentive theories of motivation while attempting to go beyond their interpretation of motivation in terms of an elementary disposition which arouses behaviour and cognition prior to any cognitive processing of information. Psychologically, the UPT relies on the assumption that anticipation, attention and some degree of uncertainty are necessary for any motivation to occur—rather than seeing them only as consequences of motivated behaviour. Neuropharmacologically, it presupposes that mesocorticolimbic dopamine is the way for the brain to code uncertainty—rather than a reward signal. The UPT aims to solve three conceptual problems which cause trouble for traditional theories. Firstly, if we understand motivation to be the increased availability of cognitive resources in circumstances of uncertainty, this explains, by definition, why cognition and behaviour are affected in a broad sense. In contrast, if motivation was seen as an elementary disposition, the processes responsible for cognitive and behavioural arousal would still be inaccessible. Secondly, the theory is advanced that motivational specificity originates in the ability to anticipate and pay attention to particular stimuli because these two processes highlight specific objects; traditional theories provide a nonspecific picture of motivation that cannot explain goal-directed behaviour without assuming the existence of hardwired drives. Thirdly, a psychopharmacological mechanism of motivational interactions, compatible with the way in which we see motivational specificity, is provided. To my knowledge, no other theory has attempted to offer such a unified framework.

The UPT is not just conceptual; the theory is also of interest because it makes original predictions. Assuming that dopamine codes uncertainty and facilitates anticipation, an experimental set-up (allowing the effects of uncertainty and anticipation to be double dissociated or added up) is therefore expected to affect performance in different ways. For instance, the behaviour of haloperidol-injected rats which must escape an aversive stimulus should be the same as that of control rats, because escape requires neither uncertainty nor anticipation. In contrast, rats which must avoid the aversive stimulus following a conditioned signal should exhibit altered behavioural patterns due to the involvement of anticipation and to low uncertainty in active avoidance. Finally, the maximal alteration of behavioural patterns should appear in those rats which must avoid an aversive stimulus that does not systematically follow the conditional signal, that is, where uncertainty is much higher. Several methodologies attempting to dissociate the effects of uncertainty and anticipation can be imagined. On a different note, cocaine doses received using a random interval schedule – hence, uncertain – are expected to reduce food consumption in rats, as compared with cocaine-administered animals on a yoked schedule. The uncertainty of cocaine delivery should indeed lead rats to allocate fewer anticipatory and attentional resources to food in the random schedule as opposed to the yoked schedule. This prediction is opposed to that of theories assuming that dopamine codes unexpected reward [214] or consummatory pleasure [252]. It is unclear whether the 'wanting' hypothesis [28] is in accordance with the UPT's prediction, because 'wanting' refers to an unconscious preparatory motivational signal. Here, the rats explicitly want cocaine—at least, those working in the random interval schedule. The UPT is a theory of wanting (without quotation marks), which is assumed to consist of the conscious extension of 'wanting'. Other predictions, as well as reinterpretations of findings already described in the literature, can be found elsewhere [6,7].

Overall, the UPT constitutes a coherent articulation of different phenomena which play a central role in behaviour: anticipation, attention, reactivity, and dopamine. All these phenomena are known but some of them often remain undifferentiated in those theories which do not really explain how motivated behaviour can emerge from their interconnections. Appropriate experiments are necessary to determine whether the UPT is a true alternative to traditional incentive theories of motivation. Two such experiments are currently taking place.

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