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A review of mathematical modeling of addiction regarding both (neuro-) psychological processes and the social contagion perspectives

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ABSTRACT

Addiction is a complex biopsychosocial phenomenon, impacted by biological predispositions, psychological processes, and the social environment. Using mathematical and computational models that allow for surrogative reasoning may be a promising avenue for gaining a deeper understanding of this complex behavior. This paper reviews and classifies a selection of formal models of addiction focusing on the intra- and inter-individual dynamics, i.e., (neuro) psychological models and social models. We find that these modeling approaches to addiction are too disjoint and argue that in order to unravel the complexities of biopsychosocial processes of addiction, models should integrate intra- and inter-individual factors.

1. Introduction

Addiction involves an interplay of many factors: biological in the form of genetic predispositions, psychological related to the processes of choice and craving, social by for example peer influence, and societal factors concerning the availability of substances (Heilig, Epstein, Nader, & Shaham, 2016; Reiter, Heinz, & Deserno, 2017; Heyman, 2009; Prom-Wormley, Ebejer, Dick, & Bowers, 2017). The interplay between these factors makes it intrinsically hard to create theories of addiction. One approach to handle this complexity and make progress on theory is formal, mathematical, modeling (Borsboom, van der Maas, Dalege, Kievit, & Haig, 2021). Formal modeling does not allow ambiguities and thus forces us to be very precise. Formal models can be extended and integrated into each other, allowing for accumulation of knowledge. Models allow us to engage in surrogative reasoning (Swoyer, 1991; Robinaugh, Haslbeck, Ryan, Fried, & Waldorp, 2020) as they are representations of the real system and by studying these representations we can infer and learn about the behavior of the real system.

This review aims to provide psychological and social scientists insight into the others' approaches, assumptions, and methods. Rather than being a systematic, exhaustive review, it is a discussion of a diverse

selection of interesting behavioral-focused models. In addition, it offers a way of classifying models from these domains. We start with formal psychological models of addiction. The second section focuses on models taking the role of the social environment into account. We conclude this review by discussing the findings and suggesting future model-development.

2. (Neuro-) psychological theories and mathematical implementations

Psychological theories and models of addiction can roughly be divided into two classes: theories representing the mechanisms that generate the desire to self-administer drugs, and theories of the decision-making and self-control mechanisms that ultimately result in whether or not the desired drug is administered. These conceptual theories are often synergistic with others and could be integrated using formal models, something recent research has called for Robinson and Berridge (1993) and Gueguen, Schweitzer, and Konova (2021).

Theories are explained briefly and a selection of formal models representing or using these theories are reviewed. For a more extensive explanation of a selection of theories, we refer to the review of Bickel

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et al. (2018). Tables 1 and 2 give an overview of all models discussed, including their scope both in terms of type of drug and stage of addiction described. They also include the type of validation performed and give an indication of the level of abstraction used. A high level of abstraction indicates that phenomena and patterns are described in an abstract, simple way. This makes it easier to learn about that representation but comes at the cost of some realism. Finally, the tables include which type of mathematical framework is used, giving an indication of the type of the main variables and methods used. While we discuss some models that focus on the psychophysiological effects of drug intake, we refer to the recent work by Mollick and Kober (2020) for a more in-depth review of models of this type.

2.1. Drug desire mechanisms

We can divide the first class of theories—mechanisms that generate the desire to self-administrate drugs—furthermore into two subclasses: a class of theories in which dopamine plays a vital role in the reward signal system, investigating the dynamics which result in the ‘high’ of drug intake, and a class of theories based on the assumption that the brain aims to keep homeostasis, counteracting the drug’s effects. We discuss these two subclasses below.

2.1.1. Dopamine function models

Behavior highly depends on the processes of identifying, judging, and pursuing goals. The idea that the dopaminergic system is a central part of these processes has been longstanding (McClure, Daw, & Read Montague, 2003; Keramati, Ahmed, & Gutkin, 2017). There are clear pharmacodynamic differences in the way different drugs increase

Table 1

Structure of models and their characteristics. *Drug* shows whether the model is applied to a specific drug, or if it describes a general phenomena in addiction. *Stages* shows which stage of the addiction process the models describe. Method explains the mathematical framework the models are based on, giving an indication of the type of variables used. The *Scale* is an arbitrary indication of the level of modeling. At 1: pharmacokinetic models, modeling real molecules in the brain, 2: pharmacokinetics with arbitrary units, 3: Cognitive, 4: Abstract Cognitive, where representation of variables are abstract, 5: Behavioral: focused not on correct inner workings but on resulting behavior. *Validation* methods are as follows: Qualitative (QL): validation matches behavior as qualitatively observed, QL-D: qualitative behavior matched with population level data. QN: quantitative data such as questionnaires, and ANML: animal behavior data.

Model	Drug	Stages	Method	Scale	Val
Drug Desire Mechanisms	General, Nicotine, Cocaine, Opioids	Initial, Stable, Recovery		1–5	QL, QN, QL-D, ANML
Dopamine Function					
(Redish, 2004)	G	I	TDRL	2	QL
(Dezfouli et al., 2009)	C	I, S	TDRL	2	QL
(Redish et al., 2007)	G	I, R	TDRL	2	ANML
(Zhang et al., 2009)	G	I, S	TDRL	1	ANML
(Keramati et al., 2017)	C	I, S, R	HRL	1	ANML
Opponent Process					
(Ahmed & Koob, 2005)	C	I	Diff. Eq.	2	QL
(Amigó et al., 2008)	C	I	Diff. Eq.	3	QL
(Caselles et al., 2010)	C	I, S	Diff. Eq.	3	QL
(Bobashev et al., 2017)	N	I, S, R	Diff. Eq.	4	QL-D

Table 2

Continuation of 1, for decision-making models.

Decision Making Process						
Behavioral economics						
(Becker & Murphy, 1988)	G	I, S, R	Utility F	4	QL	
(Orphanides & Zervos, 1995)	G	I, S	Utility F	4	QL	
(O’Donoghue & Rabin, 1999)	G	I, S, R	Utility F	4	QL	
(Gruber & Koszegi, 2001)	N	I, R	Utility F	4	QL-D	
(Wang, 2007)	G	I, S, R	Utility F	4	QL	
(Field et al., 2019)	G	S, R	Diffusion	2	QL	
Dual Process						
(Bernheim & Rangel, 2004)	G	S	Utility F	4	QL	
(Siegelmann, 2011)	G	I, S, R	Functions	5	QL	
(Redish & Johnson, 2007)	O	I, S	TDRL Utility F	3	QL	
Strength Theory						
(Grasman et al., 2012)	N	I, S, R	Diff. Eq. Stoch.	3	QL-D	
(Grasman et al., 2016)	G	I, S, R	Diff. Eq. Stoch.	3	QL-D	
(Duncan et al., 2019)	G	S, R	Diff. Eq.	4	QL-D	
Multi-Level						
(Levy et al., 2013)	G	I, S, R	Diff. Eq.	3–5	QL-D	

dopamine levels, sometimes making these models not universal (Bad-iani, Belin, Epstein, Calu, & Shaham, 2011). For some addictive substances the centrality of the mesocorticolimbic dopamine system has even been questioned. Multiple different theories on the exact role of dopamine have been posited. The two main ideas are dopamine being a reward signal in *temporal difference reinforcement learning* (TDRL), and the theory of *incentive salience* or *incentive sensitization* (McClure et al., 2003). They share the result that increased dopamine activation increases the desire of choosing the action that leads to reward, and that addiction can be seen as the result of some dysfunction in the dopamine circuitry.

In TDRL approaches, an individual learns the value of different ‘states’: the outcomes of decisions depending on the previous state. These values are updated by prediction errors if outcomes occur that are different from the expected value. Redish (2004) models cocaine addiction by adding a noncompensable drug-induced dopamine surge to the TDRL model. Here, drugs produce a positive reward-error signal independent of the change in value function, making it impossible to learn a value function to mitigate this dopamine surge. The model then can describe how the pattern of initiation of self-administration can evolve within different availability constraints. However, in this model cocaine-induced dopaminergic responses do not accommodate at all and thus keep increasing infinitely, while biological compensation mechanisms likely limit the maximal effect of cocaine on the neural systems (Keramati et al., 2017; Redish, 2004). This issue is tackled by Dezfouli et al. (2009), who introduce a weighting factor on dopamine response as well as rewards, keeping the value of states finite. This way, the value of the drug choice is increased compared to other expected rewards, but the total remains finite. Relative expected rewards from the environment are then decreased, which can be seen as incentive salience of drug rewards in the incentive sensitization theory.

Redish, Jensen, Johnson, and Kurth-Nelson (2007) add a situation recognition process to the TDRL model. In TDRL, extinction is modeled by unlearning. This makes it impossible to quickly relearn associations,

and therefore normal TDRL is unable to model the rapid reacquisition of addictive behavior occurring during relapse (Bouton, Winterbauer, & Todd, 2012). The added recognition process categorizes observed cues into situations, making it possible to remember and recognize them and thus to model the rapid relearning of relapse accurately.

Zhang, Berridge, Tindell, Smith, and Aldridge (2009) model incentive salience based on an adjusted reinforcement model. They add a physiological factor, or 'gating parameter' κ , in the obtained reward of the value function. They discuss two ways in which this factor can be implemented: as a multiplication of the reward function or an addition to it using $\log\kappa$. This gating parameter depends on the physiological state and thus can represent either sensitization or satiation.

Keramati, Durand, Girardeau, Gutkin, and Ahmed (2017) continue on the model of Zhang et al. (2009) with the *homeostatically regulated reinforcement learning model*. Instead of a single internal variable κ , they propose that the reward is calculated depending on the internal state at the time of the action. Here the pathology is not in the rewards system but in the 'needs' machinery. They are able to show that seeking rewards is equivalent to the more fundamental objective that is physiological stability, and that a disruption can lead to over-consumption.

2.1.2. Opponent-process theory

The *opponent-process theory*, first described by Solomon and Corbit (1974), describes the dynamics of acquired motives, including addictive substance use (Koob and Le Moal (2008)). The theory is based on the assumption that the brain self-organizes to keep homeostasis: opposing hedonic processes and emotional arousal, regardless of whether they were generated by positive or negative stimuli. The opponent-process counteracts the effects of drug intake (different for each drug, and especially strong with opioids (Badiani et al., 2011)). It occurs on longer timescales and is therefore still active while the surge of the drug has subsided. The theory aims to explain phenomena such as falling into addiction, craving, building tolerance, and eventually overcoming addiction.

Ahmed and Koob (2005) use the opponent-process theory to integrate pharmacokinetic, pharmacodynamic, and motivational variables in a single model of cocaine intake. It uses a dual compartment system of nonlinear dynamics: one system to account for brain concentration and reward responsivity, and another threshold system which includes motivation to simulate intravenous self-administration. Using this they simulate accurately the allostatic effects on the reward function as well as sensitization.

Amigó, Caselles, and Micó (2008) connected this hedonic state with personality traits of introversion and extraversion. Certain personality types have a higher risk for certain substances (Conrod, Pihl, Stewart, & Dongier, 2000), and by using a nonlinear dynamics implementation of the opponent-process, they show how different personalities react differently to a single dose of cocaine. Caselles, Micó, and Amigó (2010) continue on this model by integrating the *unique personality trait theory* with the acute effect of drugs and addiction. Modeling periodical drug intake, they show that initially, a user is increasingly extroverted. However, over time habituation results in a decrease of activation level to below baseline levels, pushing the user to be more introverted.

Bobashev, Holloway, Solano, and Gutkin (2017) have created an abstract nonlinear dynamical model of smoking based on the opponent-process and the allostatic theory. They use a system of five differential equations, each dependent on the previous, with the first representing the hedonic result of drug input. Each 'process' has a delay over the one it is based on. They assign abstract theoretical meaning to these equations, going from short term hedonic drug effect to the opponent-process, habit, and eventually long term hedonic memory of the drug which has a timescale of years. Then using these processes, they define a threshold for self-administration actions and equations for levels of withdrawal and craving.

2.2. Decision-making theories

The previous section focused on the strength of desires and the mechanisms that make self-administration attractive. These theories do not yet incorporate cognitive aspects to addiction; individuals often know that the pleasure of taking drugs does not weigh up to the costs, yet they still choose to take them, making addictive behavior seem irrational and self-defeating (Stacy & Wiers, 2010; Bickel et al., 2018; Wiers, Le Pelley, Harbecke, & Herrmann-Pillath Routledge, 2020). In this section, different theories and formal models to describe the decision-making process resulting in this contradictory behavior are discussed. Broadly, these can be categorized into three classes: rational choice models based on behavioral economics, dual-process systems, and models that focus on self-control.

2.2.1. Behavioral economics

Behavioral economic theory is based on the assumption that an individual acts rationally in its consumption of goods and services to maximize their utility: their satisfaction within their capabilities and resources. The value of a good depends on its benefit/cost ratio compared to all other possible actions. This concept has given economic researchers a generalizable modeling framework, which has been applied to many decision-making scenarios (Hommes, 2013). This behavioral economic framework was adjusted and applied to addiction by Becker and Murphy (1988) creating a new approach to studying addiction: the *theory of rational addiction* (RA) (Rogeberg, 2020; Ferguson, 2000).

In the RA model, individuals with nicotine addiction maximize value through balancing tolerance, withdrawal, and reinforcement: consumption of an addictive good gives a higher return on consumption in the future, while also decreasing baseline utility. This feedback can cause moderate use states to become unstable, and small deviations can lead to a large increase in consumption over time, or to sudden abstinence. The model can account for binges, responses on price change, anxiety and stress can precipitating addiction. However, in this model, only stopping 'cold turkey' can be used to end addiction.

Delay-discounting can be used to provide an explanation for impulsivity as well as loss of control as drug dependence increases (Bickel & Marsch, 2001). Later models added imperfections in the decision-making process, creating *imperfectly rational models* (Sloan & Wang, 2008). Examples are time-inconsistent preferences (O'Donoghue & Rabin, 1999; Gruber & Koszegi, 2001), wrongfully perceiving probabilities of results of their behaviors, or imperfect information about decisions (Wang, 2007). Other additions are models where, for example, youths have imperfect information about the propensities of becoming addicted, which results in experimentation escalating to addiction (Orphanides & Zervos, 1995).

Field et al. (2019) apply a different approach used in behavioral economics: *value-based decision making* (Rangel, Camerer, & Montague, 2008). In their rendition, they assume that an individual's speed and accuracy in the estimation of the perceived value are the results of an internal drift-diffusion process. In this process, internal evidence for each possible choice accumulates over time. Random noise is added representing uncertainty. When one of the possibilities has accumulated enough evidence to reach a threshold, it will be the chosen action. The rate of evidence accumulation is then different for substance-related choices for an addicted individual. However, self-control can counter the faster discounting and possibly change the outcome. Recovery can be accounted for by a change in parameters. As there is an interaction effect between multiple parameters, a simultaneous intervention on different vulnerabilities would yield the best results.

2.2.2. Dual-process system

The *theory of dual-processes*, or *dual-decision systems* (Bickel et al., 2007), hypothesizes that two different decision-making systems compete: the 'hot mode', the reward-driven, impulsive *subcortical*

system, pulling towards choosing short-term pleasure over long-term consequences, and the ‘cold mode’, the *prefrontal cortex executive control system*, capable of long-term planning, complex decision making, and behavioral inhibition (Metcalf & Mischel, 1999; Strack & Deutsch, 2004). These two systems are normally in balance, and a person is able to flexibly modulate behavior in accordance with many different situations. In certain cases the decision-making process can be ‘overruled’ by emotions, taking over from the normal objective value estimating process (Loewenstein, Weber, Hsee, & Welch, 2001). In the case of addiction, the impulsive system is hypothesized to dominate control (Bechara, 2005; Stacy & Wiers, 2010; Bickel et al., 2007). More recently the dual-process theory has received criticism on its explanatory strength and predictive power as a result of its binary nature (Keren & Schul, 2009), making the similar but more nuanced models of self-control, discussed in the section below, an interesting alternative.

A specific instance of a dual-process model is the model of Bernheim and Rangel (2004). It models environmental cues as stochastic processes that can cause either the hot or cold mode dominate control in the decision-makers’ brain. It then describes how the model accounts for distinctive features of addiction, such as unsuccessful attempts to quit, self-described mistakes, and the role of self-control. Furthermore, the authors give policy objectives based on their model.

Redish and Johnson (2007) combine the dual-process system with temporal-difference reinforcement mechanisms and a situation-recognition component in an attempt to form a unified theory of decision-making processes. When the planning component reaches a goal with a high value, a strong desire, or craving is triggered. Their model also implements sensitization: in certain repeated situations it remembers that a high-value outcome can be reached. This triggers craving and the individual then limits the exploration of other possibilities, appearing as cognitive blinding, or obsession. The authors continue on this path by creating a theory for a unified framework for addiction, based on vulnerabilities in the decision process in the three systems (Redish, Jensen, & Johnson, 2008). While not a formal model, this theory could provide a good basis for future formal models of the decision-making process.

Siegelmann (2011) uses the dual-process system as a basis for a dynamical-systems model. It is composed of two main equations: one representing the internal processes such as craving, or strength of inhibition, and another one representing the actual decision-making process. Instead of a binary hot or cold mode, decisions are dependent on a scaling emotional-cognitive rationality factor as well as the physiological state. This factor is influenced by environmental drug cues and factors such as stress. It has a certain recovery rate as well, making it act similar to the self-control discussed in the following section.

2.2.3. Self-control and strength theory

In *strength theory*, self-control can be seen as an abstract finite resource, which costs effort to exert, which in terms of the dual-process view, limits the strength of the impulsive, short term decision-making system. When this resource runs out, an individual has impaired self-control, termed *ego-depleted* (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Baumeister, Vohs, & Tice, 2007). When ego-depleted, one is less capable of putting long term goals over short term desires. In the case of addiction, it means that they are less able to hold off their desire for self-administration. Many tasks cause ego-depletion: from regulating attention, making choices and thinking analytically, to overriding some desires in favor of others.

Grasman, Grasman, and van der Maas (2012, 2016) developed an abstract dynamic systems model using a two-state system of abstract notions of craving and self-control. Self-control can be decreased by the amount of craving or by giving into the craving, however, it is restored over time. They use a stochastic process to model external cues, which influence the amount of craving. These then determine the extent of addiction. Using empirically obtained data on alcohol and nicotine recovery rates to determine the parameters, the model is then able to

explain the bi-stability of the system, where an outside cue could drive the system state from non-addicted to addicted.

Duncan, Aubele-Futch, and McGrath (2019) use a mathematical approach to analyzing relapse-recovery cycles. They create a fast-slow dynamical system existing of two coupled differential equations: the ‘level of craving’ and the ‘mood’. Mood changes are a fast process while craving is slower, increasing over time of abstinence. This simple system has bifurcations, which represent falling into depression caused by abstinence, and relapse. Using the coupling of mood and craving this model is able to predict relapse frequency with psychologically relevant parameters, and it can be used to measure the relationships between treatment and relapse frequency.

2.2.4. Multi-scale models

Levy, Levy, Meyer, and Siegelmann (2013) combine multiple components of neuropsychology, cognition, and behavior in a single *knowledge repository* nonlinear dynamical model (Bobashev, Costenbader, & Gutkin, 2007). It is an extension of the rationality model of Siegelmann (2011) with added components such as stress, pain, and craving. Because the more than 70 parameters are separated into multiple components, each of which can be analyzed individually with empirical data, calibration and validation are claimed to still be attainable, although additional investigation is necessary. However, surrogate reasoning using such a complex model is very hard nonetheless. The preliminary main result of the model is that intervention efficacy is increased by combining pharmacological treatment along with counseling therapies.

3. Social models

Although the influence of social environments consists of complex processes and is hard to measure empirically (Birk et al., 2020), the social environment has a high impact on many stages of addiction (Heyman, 2009). Important factors for initial use include parental modeling of drug use and involvement with drug-using peers (Gorsuch & Butler, 1976). Peer pressure has influence on initiation as well as on drug abuse (Borsari & Carey, 2001; Larsen, Overbeek, Granic, & Engels, 2012). Social norms and stigmatization of illicit drugs and addiction can make it harder to seek or find help (Heyman, 2009). Also recovery is affected and social support is an essential part of the success of treatments (Meyers, Roozen, & Smith, 2011; Higgins, Heil, & Lussier, 2004). In addition, drug abuse impacts an individuals’ social network over time as well, which in turn has influence on the abusive behavior of the individual.

The occurrences of waves of drug epidemics such as the prescription opioid crisis spreading over a population are a clear example of the magnitude of the effects a social environment can have (Heyman, 2009), and confirm that in order to accurately represent addictive behavior in a formal model, the impact of social environments should be taken into account. Therefore, this section focuses on inter-individual models, divided into *epidemiological* models and *agent-based* models. Epidemiological models treat the spread of addictive behavior via social interactions as similar to the spread of infectious diseases. Agent-based models consider each individual, or agent, to make its own decisions depending on individual variations in psychology and local environment.

3.1. Epidemiological models

The spread of infectious diseases and *social contagious issues* such as addiction have many similar characteristics; the use of the word *epidemic* when talking about the rapid spread of drug-use is indicative. Mathematical epidemiology is a field of research that is well-developed, and using the experience in this field offers obvious opportunities when modeling addiction and other social contagion issues (Pastor-Satorras, Castellano, Van Mieghem, & Vespignani, 2015). Epidemiology aims to

understand the prevalence and distribution of a disease on a population-level. It distinguishes individuals only by placing them into different compartments of physiological state, with certain rules on how transitions between compartments occurs. We discuss two subclasses of epidemiological models: non-structured models that ignore the exact social structure of individuals in a population, and structured models that incorporate spatial or social structures in a population.

3.1.1. Non-structured epidemiological models of addiction

White and Comiskey (2007) created a model using three states: 'susceptible'¹, 'using while not in treatment', and 'using while in treatment'. They demonstrated that the spread was most dependent on the rate at which the susceptible population transfers to the addicted one. Rather than focusing on recovery, reducing this initial descent into addiction is highly important, and they conclude that prevention should be the highest priority on policies. Battista (2015) adds a recreational-use population to this model. Battista, Pearcy, and Strickland (2019) also adjusts the model of White and Comiskey (2007) by adding a prescribed but not addicted population, modeling the opioid epidemic.

A series of models on alcohol abuse started with the work of Sánchez, Wang, Castillo-Chávez, Gorman, and Gruenewald (2007). They created a model consisting of 'susceptible individuals', 'problem drinkers', and 'temporarily recovered individuals'. Buonomo, Giacobbe, and Mulone (2019) extend this by splitting problem drinkers into people that admit they have a problem and those who do not, resulting in a stable binge-drinking endemic state. Similarly, Manthey, Aidoo, and Ward (2008) apply this to model the spread of problematic drinking behavior in college. Walters, Straughan, and Kendal (2013) add complete recovery and look into what policymakers should focus on to reduce the total population of problem drinkers. Their results are similar to the ones of White and Comiskey (2007) and Manthey et al. (2008) in that prevention is better than cure. It is important to note however, that while the models discussed above can be good representations, they are all purely theoretical and have not been validated.

3.1.2. Structured epidemiological models on addiction

Spatial compartment models in epidemiology separate the population into different states, but also in spatial compartments. While in these compartments homogeneous mixing still occurs, there is added complexity in the mixing and spread to and from spatial compartments. A simple implementation of this is the model of Mubayi, Greenwood, Castillo-Chávez, Gruenewald, and Gorman (2010), that continues the work of Manthey et al. (2008) by adding a low and high-risk drinking environment. Moderate drinkers can move in between them, making it possible to better study the effects of increased recruitment to high-risk environments. They conclude that policies reducing transitions to initial use and heavy use should be prioritized.

Social network structures representing the specific social connections of individuals are even more accurate descriptions of connectivity. Recently, these social networks can be obtained via empirical data, such as the Framingham Heart Study (Mahmood, Levy, Vasan, & Wang, 2014; Christakis & Fowler, 2013) or AddHealth Study (Jeon & Goodson, 2015; Harris & Udry, 2018). Therefore *network epidemics* can be used to improve models on spreading dynamics (Pastor-Satorras et al., 2015). The work done using these data confirms that the spread of social contagious issues can be modeled accurately with epidemiological methods.

Bahr, Browning, Wyatt, and Hill (2009) model social contagion of obesity, which arguably has similar neurobiological properties as addiction (Volkow, Wang, Tomasi, & Baler, 2013). They implement multiple structures, such as a compartmental square lattice, and hypothetical social networks with similar characteristics as real-world social

networks such as small-world and random networks (Newman, 2010). They find that obesity can quickly spread in the whole population on all structures, but that pinning the BMI of some healthy individuals can stabilize clusters.

Hill, Rand, Nowak, and Christakis (2010b) apply an infectious disease model to the social network of the Framingham Heart Study data. They use obesity as a case study, but mention that their model applies to smoking cessation and alcohol consumption and abstinence as well. They add a possibility for spontaneous non-social infection to the SIR model, creating the SISa model. Using regression of the data they obtain values for the parameters in the SISa model and show that the obesity epidemic may be driven by increasing rates of becoming obese both via spontaneous as well as by social transmission. Hill et al. (2010b) subsequently use the fitted SISa model to make predictions and analyze the course of the spread. While in other statistical-physics-based models obesity quickly reaches 100% (Bahr et al., 2009), the network-based SISa predicts an endemic state of 42%, or between 25% and 54% with 95% confidence (Hill et al., 2010b). Using sensitivity analysis, they show that the spontaneous infection and recovery rates have much larger effects than the social infections for the obesity prevalence, but the interpersonal transmission rates are increasing and do contribute significantly to the overall prevalence. While substantial, these rates are not high enough for clustering to occur. Hill, Rand, Nowak, and Christakis (2010a) apply the SISa model also on emotional state and perform similar analyses. They find that the emotional states are also social-contagious; content and discontent can spread between socially connected individuals.

3.2. Agent-based models

Agent-based models (ABM) are a class of complex system models existing of interacting autonomous individuals, or *agents*. Instead of looking at a system as a whole, as in SIR models, ABMs define rules and interactions for each agent. These agents then interact with each other and with the environment of their simulated, simplified world. These rules and interactions can range in complexity and can be extended to include stochasticity, learning, and many other mechanisms. Agent-based models are most useful if the agents' interactions impact the emergent behavior of the system, when spatial dynamics are applicable, time-symmetry is not satisfied, and when the agents can adapt to interventions and changes. All of these are very relevant to the social spreading of addiction, which makes it a promising field (Castellani, Barbrook-Johnson, & Schimpf, 2019). Behaviors that are well-described using ABMs in addiction are mostly the adaptations agents make to changes in the physical environment. Examples are how agents adapt to changes in availability, or how different housing and drug-related locations impacts heavy use.

Below we review ABMs on substance use and abuse. They can be divided into two subclasses: epidemic ABMs that extend on the epidemiological modeling discussed above, and stylized ABMs implementing a simulated environment with complex interacting agents, attempting to re-create and predict behavior of addiction.

3.2.1. Epidemic agent-based models

Gorman, Mezic, Mezic, and Gruenewald (2006) have created a model that is a mix between an ABM and a SIR model on a one-dimensional lattice. Each spatial compartment can be seen as a certain house, room, or pub. Next to the SIR states, some agents are set to always be abstaining. The agents can stochastically move about the lattice, with the probability of starting to drink depending on the relative amount of drinkers at their site. Similarly, stopping depends on the number of people already abstaining.

Ip et al. (2005) apply a similar idea to a college population, simulating the drinking culture at a college. The multiple compartments are the different starting years, with every year there an influx of freshmen and outflux of the senior population. Interactions are different

¹ Note that the epidemiological term 'susceptible' describes healthy individuals that can become addicted, and does not indicate individual differences in terms of susceptibility to addiction.

depending on each compartment, and parameters and initial conditions are set to match qualitative survey data. Different scenarios were tested to see the effects of higher conductivity or relapse numbers.

3.2.2. Stylized agent-based models

Stylized ABMs have fully individual agents with complex, heuristic decision making, and a complex environment to interact with Epstein (2012). These models range widely in their complexity and can reach an ontology of over 50 variables (Lamy, Perez, Ritter, & Livingston, 2005). Many different subjects have been researched using these ABMs. Methods vary significantly, and psychological foundation on the agents' rules are often lacking, as the field is not yet mature. The subjects that have been researched using stylized agent-based models are: drug epidemics (Agar & Wilson, 2002), effects of the workings of a local heroin market (Hoffer, Bobashev, & Morris, 2009; Hoffer, Bobashev, & Morris, 2012), alcohol-related problems (Lamy et al., 2005), college students' personality and party drinking behavior (Garrison & Babcock, 2009), weekend drug-related harms (Moore et al., 2009; Scott et al., 2016), and binge drinking (Giabbanelli & Crutzen, 2013). Their main characteristics are summarized in Table 3.

4. Discussion

In this paper, we reviewed psychological and social approaches to modeling addiction. We found that psychological models, summarized in Table 1 and 2, often focus on drug desire mechanisms and decision-making processes. The impact of the social environment was often ignored or implemented as a one-directional covariate. Validation too proves to be challenging. Most models either depend solely on (ever-changing) theoretical consensus, or are based on animal data. Psychological models often describe specific processes, and while they can be accurate descriptions of some aspects of addiction, they are often too specific. Improvements on integrating these existing psychological models could already provide an interesting way to create more intricate and more general models.

Social models on the spread of addiction often apply epidemiological modeling. This field has seen great progress in the past decades, but severe limitations remain. As the exact structure of social networks is unclear, many successful models in epidemiology completely forgo this structure. In addition, the exact process of contagion of addiction is not well understood, especially compared to the tractable spread of virus particles in traditional epidemiology. These limitations result in restrictive assumptions and thus abstract implementations. While this works well when examining populations as a whole, it may be less insightful when more detailed processes such as intra-individual dynamics are included. Agent-based models are promising as they are capable of implementing both intra- and inter-individual dynamics. However, this field is not yet mature regarding addiction; models implementing a social environment are scarce and the psychological foundation of the agents' decision-making dynamics is very limited.

In conclusion, while it is clear that psychological processes as well as social interactions play a pivotal role in all stages of addiction, our review suggests that the two main modeling approaches are currently disjoint; we found no published computational model that incorporated both psychological and social dynamics. To improve our understanding of the biopsychosocial issue of addiction, it is critical to understand the feedback loops that social interactions have on the inner workings of an individual, as well as the influence the individual has on his peers (Heilig et al., 2016; Reiter et al., 2017).

Future work should focus on implementing progress made in both fields. Integrating certain models from both disciplines described in this review could already provide more intricate models. For example, an agent-based model of interacting agents in a social network (Gueguen et al., 2021) where the agents are equipped with sound decision-making behavior based on psychological theory, could lead to advancements in both fields. Alternatively, recent research has suggested that the

Table 3

Summary of Stylized Agent-Based Models. *Complexity*: an arbitrary measure for how complex the model is, going from 1–3. *Focus*: the focus of their results: 'Policy' scenarios and recommendations or finding 'Patterns' that can be explained by the ABM. Abbreviations: 'Psy-Stim' are Psycho-Stimulants, 'Pop' stands for Population-level data and 'Ethno' stands for Ethnographic data

Model	Drug	Complexity	Focus	Validation
Epidemic ABM				
(Ip et al., 2005)	Alcohol	1	Patterns	Pop
(Gorman et al., 2006)	Alcohol	1	Patterns	–
Stylized ABM				
(Agar & Wilson, 2002)	Heroin	1	Patterns	Ethno
(Lamy et al., 2005)	Alcohol	3	Policy	–
(Hoffer et al., 2009)	Heroin	2	Policy	Ethno
(Garrison & Babcock, 2009)	Alcohol	2	–	–
(Moore et al., 2009)	Psy-Stim	2	Policy	Ethno
(Hoffer et al., 2012)	Heroin	3	Patterns	Ethno
(Giabbanelli & Crutzen, 2013)	Alcohol	1	Patterns	Survey
(Scott et al., 2016)	Alcohol	3	Patterns	Survey

dynamics of social decision-making are of similar cognitive nature to some of the internal models described in this review (Rilling & Sanfey, 2011; Sanfey, 2007; Cacioppo et al., 2007; Smith & DeCoster, 2000), making modeling social decision-making as well as drug-use behavior within an integrated model an interesting way forward as well.

By combining the modeling work of psychological and social scientists in this review, we aim to present both sides a view into the others' approaches, assumptions, and methods. We believe that the complexity of addiction can only be unraveled when both these approaches are taken into account, and encourage the simultaneous modeling of intra- and interpersonal dynamics to further the understanding of addictive behavior.

CRedit authorship contribution statement

Maarten W.J. van den Ende: Conceptualization, Writing – original draft, Writing – review & editing. **Sacha Epskamp**: Conceptualization, Writing – review & editing. **Michael H. Lees**: Conceptualization, Writing – review & editing. **Han L.J. van der Maas**: Conceptualization, Writing – review & editing. **Reinout W. Wiers**: Writing – review & editing. **Peter M.A. Sloot**: Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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