MAIN ARTICLE

Grounding alcohol simulation models in empirical and theoretical alcohol research: a model for a Northern Plains population in the United States

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Abstract

The growing number of systems science simulation models for alcohol use (AU) are often disconnected from AU models within empirical and theoretical alcohol research. As AU prevention/intervention efforts are typically grounded in alcohol research, this disconnect may reduce policy testing results, impact, and implementation. We developed a simulation model guided by AU research (accounting for the multiple AU stages defined by AU behavior and risk for harm and diverse transitions between stages). Simulated projections were compared to historical data to evaluate model accuracy and potential policy leverage points for prevention and intervention at risky drinking (RD) and alcohol use disorder (AUD) stages. Results indicated prevention provided the greatest RD and AUD reduction; however, focusing exclusively on AUD prevention may not be effective for long-term change, given the continued increase in RD. This study makes a case for the strength and importance of aligning subject-based research with systems science simulation models.

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Additional Supporting Information may be found online in the supporting information tab for this article.

Introduction

System science is an increasingly popular approach to discern effective strategies for system-wide changes (Jalali *et al.*, 2021) that address public health concerns such as harms or consequences related to alcohol use (AU) (Mcgill *et al.*, 2021). Among these simulation modeling approaches, system dynamics (SD) has received great attention over the last two decades (Darabi and Hosseinichimeh, 2020). A key strength of SD is that it focuses on real-world stages of diseases or substance use transitions and dynamic transitions of

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individuals across those stages (Stringfellow *et al*., 2022). However, current AU-SD models often fall short of capturing the real-world stages and transitions that are frequent focuses of alcohol research and policy change.

Alcohol use system dynamics (AU-SD) models have a variety of applications, from examining biological and psychological processes (Clapp *et al*., 2018; Holder *et al*., 2005) to laws and policies (Matson *et al*., 2021). However, there is little focus on aligning AU-SD model structures with AU models in theoretical and empirical alcohol research (Chassin *et al*., 2013; Zucker, 2015). Alcohol researchers recognize the potential of systems models as springboards for effective actions towards change (Purshouse *et al*., 2018). SD models overcome the limitations of common methods used in alcohol research (frequentist statistics) to better evaluate a broader system of multiple policies for community-level change (Stockings *et al*., 2018). However, AU-SD models that are not grounded in alcohol research can have reduced application for policy testing. Prevention and intervention programs typically focus on specific stages of use, such as AU initiation, severe disordered drinking, or relapse (Hussong *et al*., 2018; Tanner-Smith and Lipsey, 2015). Aligning AU-SD models more closely with alcohol research allows for better use of the alcohol research as resource for model structure and parameterization and more rigorous model-based testing of such policies and programs.

The purpose of this article is to address gaps in these two bodies of literature by presenting an alcohol research-informed AU-SD model. We provide an overview of how AU is conceptualized within alcohol research and how this research can inform SD model structures and compare these conceptualizations to previous AU-SD models. We then present a quantitative research-based AU-SD model calibrated to historical data within a U.S. Northern Plains small metro area for parameterization. Finally, we demonstrate the potential of a research-informed AU-SD model by evaluating individual stages and transitions as specific leverage points for policy change, which correspond to common prevention and intervention efforts that target either risky AU or alcohol use disorder (AUD), as integrated within a broader preventative framework.

AU models within theoretical and empirical research and relevance to SD structure

Quantitative alcohol consumption, qualitative categories, and relevance for policy testing

AU is a quantitative construct defined by frequency (e.g. number of drinking days) and/or quantity (e.g. number of "standard drinks" (Kalinowski and Humphreys, 2016) per drinking episode/day). Researchers often conceptualize discrete qualitative categories (e.g. stages) of AU (Chassin *et al*., 2013;

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Epskamp *et al.*, 2022) based on quantity-frequency thresholds in relation to risk for harm and underlying pathology. AU stages include complete abstinence (never drinking), nonrisky drinking (NRD), risky drinking (RD), AUD, sobriety, remission, recovery, and relapse. Alcohol researchers typically consider lifetime abstinence (e.g. never drinkers) separate from abstention after drinking (Kerr *et al.*, 2017; Klatsky, 2008). NRD and RD are defined by specific consumption thresholds that correspond to statistically significant increases in risk for biopsychosocial harm (Dawson, 2011; Rehm *et al.*, 2021). Standard RD thresholds in the United States can be quantity-only (binge or heavy episodic drinking; (Livingston, 2013)) or quantity-frequency composites (heavy drinking (Dawson *et al.*, 2012)).

AUD is a unique stage defined by past-year experiences of at least two of 11 symptoms (e.g. increased tolerance, engaging in hazardous behavior when drinking, inability or refusal to cut down on drinking despite negative consequences (Hasin *et al.*, 2013)) indicative of an underlying pathology that drives heavy AU and increases risk of AU consequences. Most "RD-ers" do not experience AUD; however, there is a strong association between RD and AUD for those predisposed to AUD (Boness *et al.*, 2021; Palmer *et al.*, 2019). Thus, AUD is an important stage to include in models that focus on AU-related harms (e.g. alcohol-involved externalizing behavior (Elam *et al.*, 2022)) and intervention, especially given the relatively low levels of formal AUD treatment engagement and high variety in "natural" AUD recovery and remission (Kelly *et al.*, 2017; Tucker *et al.*, 2020b). Recovery has recently been redefined from a sobriety-only (shift to alcohol abstention) construct to a more inclusive construct that includes nonabstinence (Hagman *et al.*, 2022). This redefinition is based upon research distinguishing between AUD recovery (broad alcohol-related health improvement without AUD symptoms) and remission (limited alcohol-related health improvement with reduced AUD problems or symptoms), both of which can include nonabstinence (Tucker *et al.*, 2020a; Witkiewitz and Tucker, 2020).

Many AU-reduction programs target levels of risk and stage progression, such as preventing early AU initiation, reducing risk, onset and recurrence of RD or AUD, and subsequent harm (O'Connor *et al.*, 2018). This corresponds with general prevention frameworks of disease (Nelson *et al.*, 2022). However, cultural and societal encouragement of "acceptable" or "responsible" AU adds unique factors to prevent or reduce "dangerous" or "unacceptable" AU, changing the meaning of "prevention" (e.g. preventing any use vs. harmful use). Longitudinal AU trajectories are facilitated by multiple etiologies and contexts that can facilitate the initiation and progression of hazardous AU; program efficacy requires a fit between the severity of risk or behavior and etiological contexts of risk (Hussong *et al.*, 2018; Witkiewitz *et al.*, 2019). For example, screening, brief intervention, and referral for treatment approaches are effective RD intervention strategy to prevent the initial onset of AUD in high-risk individuals, but they do not sufficiently address

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the needs for AUD intervention (Knox *et al.*, 2019). AU reduction requires a multitiered approach targeting individuals' needs within each stage for full community-level change.

Empirical and theoretical alcohol research as a source to inform model structure and parameterization

SD modelers should consider the multiple stages and patterns of transitions between stages for developing structures. Research on AU initiation, persistence, and desistance (e.g. transitions in and out of stages) can be a rich resource. AU stages are usually positioned within a developmental model structure that starts from initiation, first sip/full standard drink (Jackson *et al.*, 2021), increasing use frequency/quantity, AUD (Deutsch *et al.*, 2017), and ending with recovery and/or relapse (Seeley *et al.*, 2019). However, movement across this continuum is not always a linear pattern of transitions between "nearest neighbor" stages. For example, a proportion of individuals report initiating AU through binge drinking (e.g. skipping NRD and initiating RD (Deutsch *et al.*, 2017; Sartor *et al.*, 2016)). People at higher AU stages (RD, AUD) demonstrate a variety of remission, recovery, and relapse patterns. Both treatment and nontreatment AUD populations demonstrate long-term patterns of transitions in and out of heavy drinking, abstinence, and AUD categories (Fan *et al.*, 2019; Maisto *et al.*, 2020; Tucker *et al.*, 2020a). Similar work demonstrates a variety of pathways for general RD populations (Koenig *et al.*, 2020; Lee *et al.*, 2018), often related to "maturing out" (Lee and Sher, 2018) of RD.

Few datasets provide the information needed to parameterize the full continuum of AU patterns within people and across time, requiring integration between data and literature. Survival/hazard models often provide percentages of individuals transitioning to stages and average time to transition (Koenig *et al.*, 2020; Seeley *et al.*, 2019). Longitudinal latent analyses (identifying "unmeasured" qualitative patterns or categories, e.g. mixture models, latent transition models) of AU patterns often report sample distribution of latent classes (Lee *et al.*, 2018; Maisto *et al.*, 2021). Parameters from such studies can be used to calculate rates (e.g. z percent of people transitioning between stages/classes X and Y during a certain duration) that can inform model flows.

SD modelers must consider a few caveats when using alcohol research data to parameterize AD-SD for models. Alcohol research is often conducted in high-risk samples to account for nonnormal distribution of RD and AUD. Similarly, most research on transitions is based on adolescent/young adulthood populations (Koenig *et al.*, 2020; Sartor *et al.*, 2016), given the normative onset of AU and occurrence of RD and AUD are most common during this age range (Jackson *et al.*, 2021; Lee *et al.*, 2018). Using statistics from studies with high-risk or clinical samples may require modification if

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applied to broader-population SD models (e.g. yearly transitions to AUD from RD may be lower when considering communities "at large" compared to a high-risk subpopulation). Modelers must also consider strategies for aggregating subgroups. For example, a single flow representing the transition from RD to AUD will include both initial AUD onset and relapses. A single "nondrinker" stock will aggregate lifetime abstainers, future drinkers who have not yet initiated, nondisordered infrequent drinkers who may be temporarily sober, and former drinkers in recovery from AUD or RD. In turn, higher aggregation of stocks will change the potential meaning of flows between stocks.

Representations of alcohol use within system dynamics literature

Table 1 displays examples of how AU is represented in prior qualitative and quantitative SD models of AU or specific AU-related behaviors (driving under the influence of alcohol), using a recent literature review (Mcgill *et al.*, 2021) and our own literature search (Google Scholar and EBSCO Host for "system dynamics" and "alcohol use" articles). AU is represented in a variety of ways, most commonly as a single variable (Matson *et al.*, 2021; Moxnes and Jensen, 2009). Quantitative models were more likely to have multiple AU stages (Apostolopoulos *et al.*, 2018; Mckelvie *et al.*, 2011). Most studies did not include operational definitions for variables, making it difficult to distinguish between stages (Apostolopoulos *et al.*, 2018; Scribner *et al.*, 2009). Of the two studies that included operational definitions, only one study (Mckelvie *et al.*, 2011) reported using alcohol research guidelines for these definitions (Office for National Statistics, 2012). All studies that included nondrinkers used a single stock (e.g. "abstainers": Hosseinichimeh *et al.*, 2022), which aggregated qualitatively unique subgroups (lifetime abstainers, people in recovery from heavy drinking or AUD).

Detail on data/information used for model structure rationale varied from thorough discussions of prior literature (Clapp *et al.*, 2018; Mubayi *et al.*, 2010) to minimal references (Tawileh *et al.*, 2008). Studies that utilized participatory model building often discussed professional and personal expertise; however, few studies mentioned participants' specific academic, professional, or personal expertise/experience with AU (Belue *et al.*, 2012; Deutsch *et al.*, 2021; Matson *et al.*, 2021). Studies that used alcohol research for structure rationale focused on AU influences (e.g. peer influences, alcohol marketing) or contexts and consequences (e.g. underage AU, outlet zoning, or intoxicated driving). Few studies referenced theoretical or empirical models of alcohol use patterns or trajectories. Almost all multiple-stage models used a linear structure that included only transitions between "nearest neighbor" stocks (e.g. lighter to heavier use (Apostolopoulos *et al.*, 2018)), with only one study providing multiple pathways through bidirectional flows (Mckelvie *et al.*, 2011).

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Table 1. Alcohol use system dynamics (AU-SD) models in prior literature

Study	Model type	Project focus	AU parameters	Operational definitions	Associations between AU parameters	Model structure sources/AU specificity and simulation data
Belue *et al*. (2012)	Causal Loop Diagram (CLD)	Underage AU	Underage drinking among high school children	NS	NA	Structure sources: Group model building sessions (Participant expertise: public health/program practitioners, specific experience in alcohol programming)
Deutsch *et al*. (2021)	CLD	Alcohol misuse	Alcohol Misuse	"A pattern of drinking that impacts one's life negatively, potentially harming health and relationships; an individual that continues to drink even after experiencing consequences, needing alcohol to cope with life or deal with daily functions, or sacrificing other aspects of life (activities, relationships) to drink," e.g. AUD symptoms	NA	Structure sources: Group modelbuilding sessions (Participant expertise: personal experience with AU and alcohol misuse)
Holder *et al*. (2005)	CLD	AU treatment	Drinking	NS	NA	Structure sources: Prior theoretical models on AU and drug behavior and prevention
Ip *et al*. (2012)	CLD	Prevention of hazardous AU	High-risk drinking Alcohol consumption	NS	NA	Structure sources: Prior literature on college drinking and prevention.
Matson *et al*. (2021)	CLD	Alcohol outlet zoning policy on community well-being	Alcohol intoxication	NS	NA	Structure sources: Group model-building sessions (Participant expertise: academic [AU expertise NS], professional [lawyers focusing on liquor issues] business owners, nonprofit org staff)
Suriyawongpaisal *et al*. (2021)	CLD	AU and AU-related harm (violence, driving while intoxicated)	Alcohol use Driving while intoxicated Alcohol-related violence	NS	AU → Driving while intoxicated AU → Alcohol-related violence	Structure sources: AU social/ community influences Theory of Change paradigm In-depth interviews (Participant expertise: policy champions and organizers with an alcohol prevention campaign).

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Table 1. Continued

Study	Model type	Project focus	AU parameters	Operational definitions	Associations between AU parameters	Model structure sources/AU specificity and simulation data
Apostolopoulos *et al.* (2018)	Stock and Flow (SF)	Risky AU on college campuses	Abstainers	NS	Abstainers ↔ Occasional Drinkers	Structure sources: NS
			Occasional drinkers		Occasional Drinkers ↔ Regular Drinkers	Simulation data: NS, datasets examples are mentioned
			Regular drinkers		Regular Drinkers ↔ High-Risk Drinkers	
			High-risk drinkers			
Mckelvie *et al.* (2011)	SF	Reducing alcohol-related hospital admissions	Abstinent	Definitions taken from General Lifestyle Survey Guidelines	All parameters have bidirectional associations with each other	Structure sources: NS
			Lower Risk	Lower risk: ≤21/≤14 units per week for men/women		
			Increasing Risk	Increasing risk: 22–50/14–35 units per week for men/women		Simulation data: Nationally representative datasets (General Lifestyle Survey)
			Higher Risk	Higher risk: >50/>35 units per week for men/women		
Moxnes and Jensen (2009)	SF	Adolescent event-level drinking	Alcohol intake/ bottles of alcohol consumed	Alcohol intake/bottles: # of bottles of beer consumed	NA	Structure sources: Prior AU/binge drinking literature Simulation data: Experimental data
Mubayi *et al.* (2011)	SF	AU behavior in college	Light drinkers	Light drinker frequency/quantity ≤3 drinks per session, ≤1 session per month	Light drinkers → moderate drinkers	Structure sources: Authors' prior models
			Moderate drinkers	Moderate drinker frequency/quantity 3–5 drinks per session, ≥1 session per month OR 1 session per week and ≤3 less per session	Moderate drinkers (low-risk environment) ↔ moderate drinkers (high-risk environment) Moderate drinkers → heavy drinkers	Binge drinking and social influence AU research Simulation data: National college sample datasets
			Heavy drinkers	Heavy drinker frequency/quantity 4–5 drinks per session, ≥1 day per week OR >5 drinks ≥1 day per month		

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Table 1. Continued

Study	Model type	Project focus	AU parameters	Operational definitions	Associations between AU parameters	Model structure sources/AU specificity and simulation data
Scribner *et al.* (2009)	SF	Risky AU on college campuses	Abstainers	Abstainers: nondrinkers	Abstainers ↔ Light drinkers	Structure sources: Prior literature on AU in college contexts
			Light drinkers			
			Moderate drinkers	Light drinkers: NS	Light drinkers ↔ Moderate drinkers	
			Problem drinkers			Simulation data: Social norms marketing research project data
			Binge drinkers	Moderate drinkers: NS	Light drinkers ↔ Binge drinkers	
				Heavy episodic drinkers: 4/5 drinks per session at least 1× in past 2 weeks	Light drinkers → Problem drinkers	
				Problem drinkers: those endorsing at least 2 of the 4 problem-drinking indicators from the CAGE questionnaire (common clinical alcohol problem screening tool).	Moderate Drinkers ↔ Binge drinkers	
					Moderate Drinkers → Problem drinkers	
					Problem Drinkers → Abstainers	
Clapp *et al.* (2018)*	CLD and SF	Event-level drinking	CLD: Drinking	GAC: Alcohol residing in gastrointestinal tract	NA	Structure sources: Authors' alcohol research
			SF: Alcohol ingestion			Prior AU literature on event-level biopsychosocial models
			GAC			Simulation data: Authors' alcohol research
Hosseinichimeh *et al.* (2022)	CLD and SF	Drinking While Intoxicated (DWI)/Peer influence on binge drinking	Abstainers	NS	Abstainers ↔ Drinkers who do not drive after drinking	Structure sources: Group model building (participant expertise: academic experts, AU expertise NS)
			Drinkers who do not drive after drinking		Drinkers who do not drive after drinking ↔ Drinkers who drive after drinking	
			Drinkers who drive after drinking		Drinkers who drive after drinking → Never DWI again	AU/intoxicated driving, and peer influence literature
			Former drunk		Drinkers who binge but do not drive	

(Continues)

Table 1. Continued

Study	Model type	Project focus	AU parameters	Operational definitions	Associations between AU parameters	Model structure sources/AU specificity and simulation data
			drivers with DWI charge (never DWI again)		after drinking →Total drinkers who binge	
			Drinkers who binge and drive after drinking		Drinkers who binge and drive after drinking → Total drinkers who binge	Simulation data: AU/intoxicated driving literature
			Drinkers who binge but do not drive after drinking		Never DWI again → Total drinkers	
			Total drinkers who binge		Drinkers who drive after drinking → Total drinkers Drinkers who do not drive after drinking → Total drinkers	National datasets
			Total drinkers			
Salmon *et al.* (2020)	CLD and SF	Preventing driving while intoxicated (DUI)	CLD: Incidental use	NS	CLD: Incidental use → alcohol addiction Incidental use → drivers under influence of alcohol	Structure sources: Group model building, (Participant expertise: policy, academic, transportation experts, AU expertise NS)
			Alcohol addiction		Alcohol addiction → drivers under influence of alcohol	AU/intoxicated driving literature
			Drivers under influence of alcohol SF: # of people habitually misusing alcohol Habitual DUIers		SF: # of people misusing alcohol → habitual DUIers	Simulation data: Not specified
			CLD		NA	Structure sources: NS

(Continues)

Table 1. Continued

Study	Model type	Project focus	AU parameters	Operational definitions	Associations between AU parameters	Model structure sources/AU specificity and simulation data
Tawileh *et al.* (2008)	CLD and SF	AU/misuse prevention	Total alcohol consumption ↓ legal age drinker consumption ↑ legal age drinker consumption SF Total Alcohol consumption ↓ legal age drinker consumption ↑ legal age drinker consumption	Consumption based on unit measure of drinking (8 g of alcohol)		Simulation data: Nationally representative datasets

*NS, Not Specified; NA, Not available – e.g. no associations between AU parameters.

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Current study

Although alcohol research can serve as a useful resource for developing AU-SD models, especially for policy testing of strategies grounded in alcohol research, there are clear gaps in how AU is represented in both bodies of literature. The purpose of this article is to address these research gaps by presenting an AU-SD model that aligns with current alcohol use research. We demonstrate a model that represents the continuum of AU stages and flexibly allows for diverse transitions between stages. We further demonstrate how such models can be parameterized using an integrated dataset that draws from both datasets and alcohol use research, calibrating our model to historical data representing a specific community within the Northern Plains United States as part of a larger community-based system dynamics project. Finally, we provide a demonstration of how this model can be utilized in testing and analysis by examining transitions between stages as specific leverage points. Our policy tests correspond to common strategies to reduce AU associated with higher rates of harm, examining both an overarching preventative (primary, secondary, tertiary) framework (Nelson *et al*., 2022) and individual focus on RD and AUD stages. We simulate our model to examine primary prevention (prevention of risk for onset of disease, e.g. prevention of RD), secondary prevention (reducing risk progression, preventing risk progression to disease onset, or preventing risk recurrence, e.g. promoting transitions out of RD, preventing transitions back to RD, and preventing transition to AUD), and tertiary prevention (reducing disease progression and preventing recurrence, e.g. promoting transitions out of AUD and preventing transitions back to AUD).

Methods

Model development

Model structure

Figure 1 displays an overview of the AU model structure, highlighting the unique contributions of the current model based on prior AU-SD models as discussed above (excluding some population parameters, e.g. death rates and net migration). Contributions include both novel stocks or flows that, to our knowledge, have not been included in prior AU models or stocks and flows that have been expanded upon to align with theoretical and empirical alcohol research. Model development was guided by (1) empirical and theoretical AU models as discussed in the introduction and expanded upon in Table S1 in the online supporting information, (2) empirical AU studies on transitions between stages to inform inflows and outflows (see Table S2), and (3) matching operational definitions of stage/stocks with the historical

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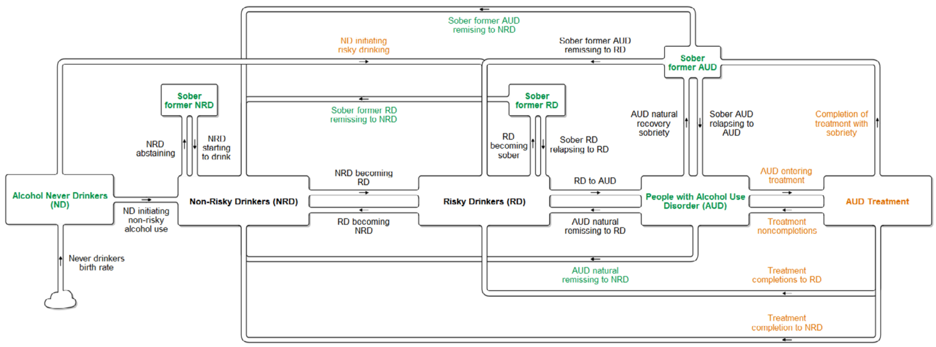

Fig. 1. Alcohol–specific structure of the model, representing stages (stocks) and transitions (flows). Some population-level parameters (e.g. death rates, net migration) are excluded for ease of viewing. Full model with all variables available in the online supporting information. Orange variables represent novel contributions to existing SD models (e.g. absent variables in other models, such as treatment or flows from AUD to lower-drinking stocks). Green variables represent extensions of existing SD models (e.g. variables that have existed in prior AU-SD work in some form but have been changed to align with the alcohol use literature, such as sober and abstaining stocks or alcohol use disorder) [Color figure can be viewed at wileyonlinelibrary.com]

data we were using for parameterization (see below). Our model included a single NRD and RD stage, given the larger amount of research that discusses broad "risk" and "nonrisk" AU (Epskamp *et al.*, 2022) with less information on transitions between multiple RD and NRD categories (e.g. different categories of NRD and RD frequency such as "regular" drinking or frequent heavy episodic drinking). Table S2 displays the operational definitions for all stocks. To address aggregation within stocks, and as common in alcohol research, we separated lifetime abstainers and nonabstainers (those who have never had a single standard drink), as well as sobriety transitions from NRD, RD, and AUD. Multiple sobriety stages allowed us to represent the unique transitions between any AU and sobriety. Flows allowed individuals to move from most sobriety stages to either NRD or RD (e.g. sober former RD to NRD), providing less aggregation of individuals with distinct pathways within stages and transitions.

As many studies examine transitions of specific populations, such as AUD relapse (Maisto *et al.*, 2020), calculating accurate outflows from a single "sober" stage to AUD involved considering relapse and remission rates alongside the proportion of people transitioning to sobriety from AUD. Although we were required to make such considerations for some flows (e.g. the inflow of AUD from RD must consider both relapse and initial onset), our goal was to reduce aggregation when possible. We included two potential pathways for recovery/remission: either "naturally" (moving to sober AUD or either drinking stage) or transitioning from untreated to "in treatment" (and then to either sober AUD or drinking stages), given the high rate of people who recover without treatment and the wide variety of recovery pathways that include gradual reductions in drinking (Tucker *et al.*, 2020a).

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We made two adjustments to the model during development. An initial model that included separate inpatient and outpatient treatment modalities (given a difference in time spent in treatment) forced trade-offs in fit between the consumption and treatment components, requiring consolidation of the treatment stock. Treatment flows were calculated by averaging inpatient and outpatient flows, accounting for the proportion of people in inpatient versus outpatient treatment. Second, we calculated age-weighted inflows and outflows for AUD and RD stages. Studies we used for parameters used adolescent and young adult samples (Koenig *et al.*, 2020; Seeley *et al.*, 2019). However, this ignores other people within RD and AUD stages, including older RD-ers who have never and will never experience AUD, those in remission from AUD, or those with persistent AUD (Fan *et al.*, 2019). Using the National Survey on Drug Use and Health (NSDUH) online data analysis tool to obtain age ranges for stages, we adjusted our calculated rates for RD and AUD inflows and outflows by considering the proportion of 18–25-year-olds, who make up the majority of AUD cases as indicated by NSDUH data (Substance Abuse and Mental Health Services Administration, 2020), in comparison to those younger than 18 or older than 25.

Data and model development and quantification

Our model represented the total population of a Northern Plains United States target community. Most parameters were informed by regional data from nationally representative datasets and prior research. In the absence of such evidence, we estimated parameters through model calibration, replicating historical data of model stages. Table S2 in the online supporting information displays our data and sources for model parameterization and operational definitions. Historical data was compiled by multiplying the viable population of our target community spanning from 2016 to 2019 from census and vital statistics data (Centers for Disease Control and Prevention, 2019; Survey, 2019) to percentages of individuals in specific categories and transitions to categories (when possible) as derived from the restricted NSDUH dataset (Substance Abuse and Mental Health Services Administration, 2021b) or the Treatment Episode Dataset-Discharges (TEDS-D (Substance Abuse and Mental Health Services Administration, 2021a)). Table S3 presents our historical data and in-depth detail regarding parameter calculations. When we had less confidence in the collected estimates from prior research, we considered ±10% uncertainty around the reported values and estimated these uncertainty coefficients as part of model calibration.

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Model analysis

We simulated changes in RD and AUD from 2019 to 2024, given historical trends from 2016 to 2019. We tested multiple approaches to reduce population (community)-level RD and AUD, as aligned with broad disease prevention/health promotion frameworks for substance use (Nelson *et al.*, 2022). Specifically, we examined primary prevention (preventing initial risk for disorder), secondary prevention (mitigating existing risk or initial symptoms, e.g. prevention intervention and preventing recurrence of risk), and tertiary prevention (managing progression of disease through treatment and reducing recurrence, e.g. intervention, treatment, and relapse prevention). Model tests included considering both natural recovery and formal treatment pathways for AUD and considering strategies emphasizing only sobriety versus sobriety and low-risk drinking (Tucker and Witkiewitz, 2021). Table 2 provides further detail on individual strategies.

We first compared a baseline model (in which we made no changes) to 11 individual strategies and compared final rates at the simulation end in 2024. All model testing comparisons (modification of flows) were set at 25%. As we did not have a specific type of program or policy (e.g. a peer support program for recovery maintenance) that we were examining, we selected 25% change as it is equivalent to a significant moderate effect for AU primary, secondary, and tertiary prevention programs (e.g. within a random experimental design, behavior change for the experiment group were 25% more likely to become sober compared to the control group) (O'connor *et al.*, 2018; Tanner-Smith and Lipsey, 2015). Therefore, our question for this set of model tests was: "when considering prevention programs that have demonstrated similar efficacy, what leverage points corresponding to stage-specific types of program will provide the most benefit *to the community at large*?"

We then evaluated 12 combinations of preventing onset, managing symptoms, and preventing recurrence of RD (e.g. primary and secondary prevention) or AUD (secondary and tertiary prevention) by the simulation end in 2024. Prevention is best provided by a combination of preventing onset, reducing progression, and preventing recurrence, as addressing only those who are currently experiencing or who are at risk of experiencing negative health issues is insufficient for full community reduction of such issues. We wanted to consider the most powerful combinations of these strategies and further understand combined strategies that focus on individual stages of RD and AUD. We did not combine AUD and RD-specific strategies, given distinctions between disordered and nondisordered drinking, in both health practice (e.g. access to formal treatment, screening tools (O'connor *et al.*, 2018, Venegas *et al.*, 2021)) and conceptual/empirical research (e.g. differences in severity of risk progression to AUD), and how this corresponds to specific types of programs focused on addressing specific stages.

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Table 2. Model tests for leverage points, and corresponding prevention approach strategy and example individual-level programs

	Strategy #	Policy test	Specific strategy	Individual-level programs examples	Prevention approach
RD	S1	Decreasing RD inflow from NRD	Preventing risk for onset of disease by preventing NRDers to transition to RD	Life skills training in school and university settings, social norm interventions, educational programs, universal policies (Cho and Cho, 2021; Lammers *et al.*, 2011; Martineau *et al.*, 2013; Werch *et al.*, 2000)	Primary (prevent risk)
	S2	Decreasing RD inflow from never-drinkers	Preventing risk for onset of disease by preventing NRDers to transition to RD and never-drinkers to initiate drinking through RD		
	S3	Increasing outflow of RD to Sober RD	Reducing risk for onset of disease through promoting transition out of RD to sobriety	Screening, Brief Intervention (Knox *et al.*, 2019; Tansil *et al.*, 2016)	Secondary (reduce risk progression /prevention intervention)
	S4	Decreasing Sober RD outflow to RD	Preventing recurrence of risk for onset of disease by preventing former RDers who transitioned to sobriety from returning to RD	Peer support, social support groups (harm-reduction and sobriety focused) (Marlatt *et al.*, 2011; Tracy and Wallace, 2016)	Secondary (prevent risk recurrence)
	S5	Decreasing Sober RD outflow to RD + NRD	Preventing recurrence of risk for onset of disease by preventing former RDers who transitioned to sobriety to return to any drinking	Peer support, social support groups (sobriety focused) (Kelly *et al.*, 2020)	
AUD	S6	Decreasing outflow of RD to AUD	Reducing progression of risk to onset of disease by preventing RDers to experience more harm and problematic AU	Screening, Brief Intervention, Referral to Treatment (Knox *et al.*, 2019; O'connor *et al.*, 2018)	Secondary (prevent risk progression to disease onset)
	S7	Increasing outflow of treatment to Sober AUD	Reducing continuation of disease after onset by improving the efficacy of formal AUD treatment to promote people with AUD who are in formal treatment to complete treatment as sober individuals.	Evidence-based patient-appropriate formal inpatient/ outpatient treatment (comprehensive, culturally tailored), pharmacological treatment (MacKillop et al., 2022; Ray *et al.*, 2019; Witkiewitz *et al.*, 2019)	Tertiary (reduce disease progression)
	S8	Increasing Outflow of AUD to Sober AUD	Reducing continuation of disease after onset by promoting natural recovery of AUD without formal treatment methods to sobriety	Sobriety-focused informal treatment, peer support, promoting recovery capital (Bassuk *et al.*, 2016; Kelly *et al.*, 2020)	
	S9	Increasing outflow of AUD to Sober AUD + NRD	Reducing continuation of disease after onset by promoting natural recovery of AUD without formal treatment methods to sobriety or to reduce alcohol consumption to nonrisky drinking levels	Sobriety and harm-reduction focused informal treatment or peer support and recovery capital promotion (Tucker and Witkiewitz, 2021)	

(Continues)

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Table 2. Continued

Strategy #	Policy test	Specific strategy	Individual-level programs examples	Prevention approach
S10	Decreasing outflow of Sober AUD to AUD	Preventing recurrence of disease by preventing AUD relapse for individuals who have become sober after AUD	Pharmacological treatment, peer support, recovery support groups (sobriety and harm reduction focused), recovery housing or communities, and continuing treatment (Bassuk *et al*., 2016; Kaplan *et al*., 2010; Spanagel and Vengeliene, 2012)	Tertiary (reduce disease recurrence)
S11	Decreasing outflow of Sober AUD to AUD + RD	Preventing recurrence of disease and risk for disease recurrency by preventing AUD and RD relapse for individuals who have become sober after AUD	Pharmacological treatment, peer support, recovery support groups (sobriety focused), recovery housing or communities, and continuing treatment (Polcin *et al*., 2010; Rinck *et al*., 2018)	

For example, promoting transitions out of RD to sobriety and reducing transitions from RD to AUD are both secondary AUD prevention strategies with a goal of reducing risk progression. However, programs will differ based upon target populations' predisposition for AUD (e.g. addressing a higher number of needs, contexts, and risk factors for RD-ers with higher predisposition to progress to AUD compared to RD-ers with lower predispositions but similar patterns of harmful drinking). Our question for this set of model tests was "when considering efforts to address a full spectrum of preventing, managing, and reducing the recurrence of drinking that is associated with harm and hazard, what are the multiple strategies that will provide the most impact for the overarching body of risky drinkers in the community?"

As our final step, we evaluated strategies within the broad framework of AUD prevention (e.g. primary, secondary, and tertiary prevention of disease), by examining the synergy of the best RD-reduction and AUD-reduction combination strategies (e.g. the best RD-focused combination strategy and the best AUD-focused combination strategy). We probed strategies and combinations through two-way sensitivity analysis to investigate the sensitivity of model outcomes to changes in intervention parameters, which helped explore potential synergies and nonlinearity that may not have been observed in our analysis of intervention. We used Latin Grid method and changed the parameters from zero to 100%, with an increment of 10%, in the desired direction. Here, we can consider the percentage of flows in terms of both efficacy of program and resources required for program efficacy (for implementation, fidelity, and sustainability). Thus, the question we focus on for this test is: "what is the optimal efficacy we need for either RD or AUD programs to jointly maximize community benefit in reducing overall harmful

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Table 3. Model analyses for testing RD and AUD strategies, focusing on primary prevention, intervention, and secondary prevention strategies, and the combinations of these strategies

	RD strategies					AUD strategies					
	Primary (prevent risk)		Secondary (reduce risk progression)	Secondary (reduce risk recurrence)		Secondary (prevent risk progression to disease)	Tertiary (reduce disease progression)			Tertiary (reduce disease recurrence)	
By stage	Strategy (S)1	S2	S3	S4	S5	S6	S7	S8	S9	S10	S11
By prevention framework / Combined strategies	↓ NRD to RD	↓ (Never drinker + NRD) to RD	↑ RD to Sober RD	↓ Sober RD to RD	↓ Sober RD to (RD + NRD)	↓ RD to AUD	↑ Treatment to Sober AUD	↑ AUD to Sober AUD	↑ AUD to (Sober AUD + NRD)	↓ Sober AUD to AUD	↓ Sober AUD to (AUD + RD)
C1	25%										
C2		25%									
C3		25%	25%								
C4		25%	25%	25%							
C5		25%	25%		25%						
C6						25%					
C7						25%	25%				
C8						25%	25%			25%	
C9						25%	25%				25%
C10						25%		25%			
C11						25%			25%		
C12						25%			25%		25%

Note: ↓, decrease; ↑, increase; e.g. Strategy 1 presents a 25% reduction in the flow from NRD to RD.

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drinking?" Table 3 presents the specific policy tests for all individual strategies (S1-11) and strategy combinations (C1-12).

All model analyses were conducted in Vensim DSS software (version 8.2.1). All Vensim files are available in the online supporting information. We designed an online, interactive model interface to run the model without any software requirements—available at https://mj-lab.mgh.harvard.edu/alcohol-misuse-model/

Results

Model analysis

The model replicated historical data (Figure S1), with the mean absolute percentage error (MAPE) and coefficient of determinant (R^2), averaged across all stages, of 8% and 96.8%, respectively. Estimated model parameters are presented in Table S4 in the online supporting information.

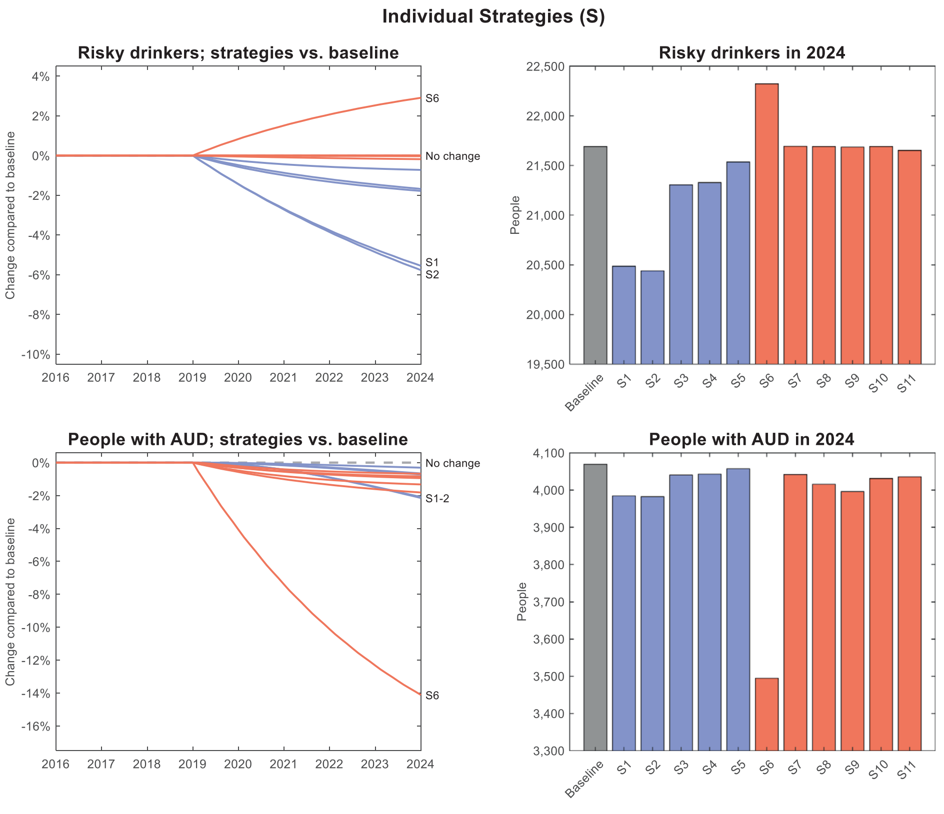

Fig. 2. Analysis of individual strategy (S) for people with RD (above) and AUD (below), including 2016–24 trends compared to baseline (left) and 2024 outcomes for all scenarios (right). See Table 2 for each strategy change [Color figure can be viewed at wileyonlinelibrary.com]

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Figure 2 displays 2016–24 trends and final 2024 outcomes for RD and AUD stages, comparing individual strategies. Primary prevention had the strongest impact on RD. Reducing the number of NRD-ers who transitioned to RD (S1) resulted in reducing the RD stock by almost 6% by 2024; there was little change when adding the strategy to reduce never-drinkers' initiation to RD (S2). RD-focused secondary prevention strategies, including prevention intervention (e.g. intervening on risk by promoting sobriety prior to AUD onset, S3) and reducing risk recurrence (reducing the return to RD or any drinking after sobriety, S4-5) provided much weaker changes (~2% reduction). Primary prevention had a small effect on reducing AUD (~2%), with RD-specific secondary prevention strategies providing minimal impact. AUD-focused secondary prevention (e.g. preventing AUD onset by reducing RD flow to AUD, S6) provided the strongest reductions to AUD (~12%), but also resulted in an approximate 2% increase in RD. Tertiary prevention strategies, both promoting formal or natural recovery (S7–9) or preventing relapse (S10–11), provided much less change for AUD and RD.

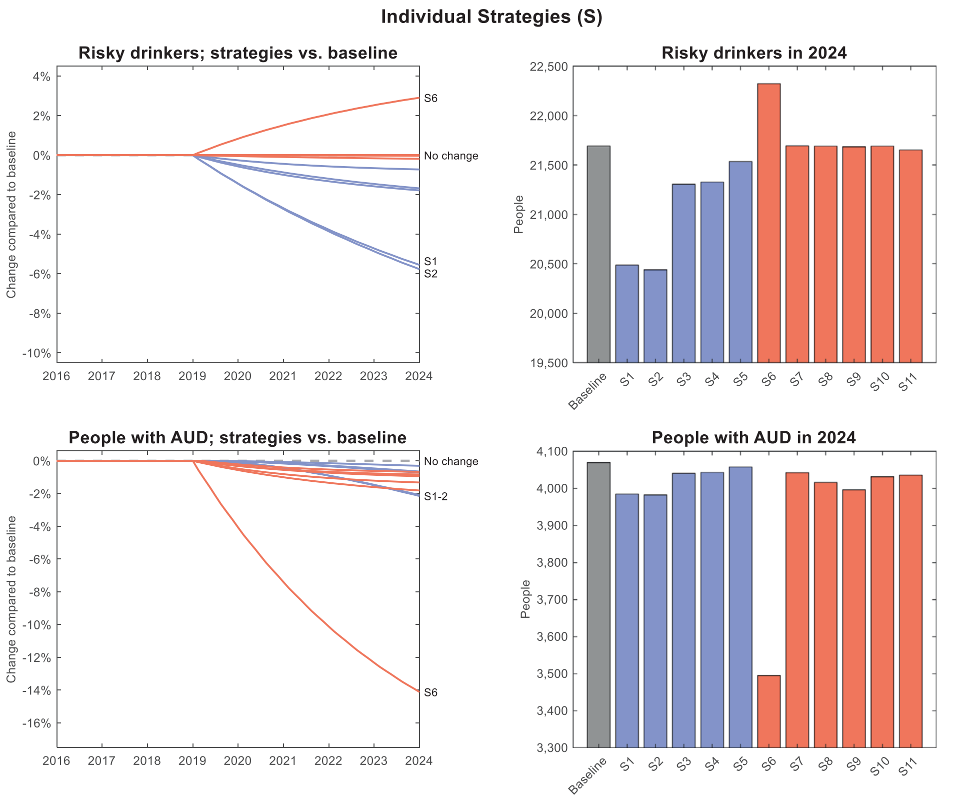

Fig. 3. Analysis of combined strategies (C) for people with RD (above) and AUD (below), including 2016–14 trends compared to baseline (left) and 2024 outcomes for all scenarios (right). See Table 2 for the combination of strategies [Color figure can be viewed at wileyonlinelibrary.com]

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Figure 3 presents tests for combinations of RD-focused or AUD-focused strategies on RD and AUD. The most effective strategy combination for RD (C4) included reducing the flow of people transitioning to RD from NRD or never drinking (S2), supporting transitions out of RD to sobriety (S3), and preventing transitions from sobriety back to RD, but not necessarily enforcing complete sobriety (S4). This combination provided an approximately 9% reduction in RD in 2024, or about a 3% greater change than focusing only on preventing RD onset (S1–2). There were also minor reductions in AUD when focusing exclusively on combinations of RD strategies; C4 was the best-performing RD combination strategy for reducing AUD, resulting in an ~3% reduction by 2024.

The most effective combined strategies to reduce AUD (C12) involved reducing the flow of people transitioning from RD to AUD (S6), encouraging natural recovery for individuals with AUD through both sobriety and remission to NRD (S9), and preventing relapse of AUD by preventing individuals who have become sober after AUD to either transition back to AUD or start RD again (S11). Although the secondary prevention strategy of preventing AUD onset appeared to drive most of the change, changing the flows involved in combination C12 by 25% resulted in an ~17% decrease in AUD in 2024 compared to baseline, or about 3% larger change compared to preventing onset alone (S6). Implementing only AUD strategies resulted in an ~3% increase for RD in 2024.

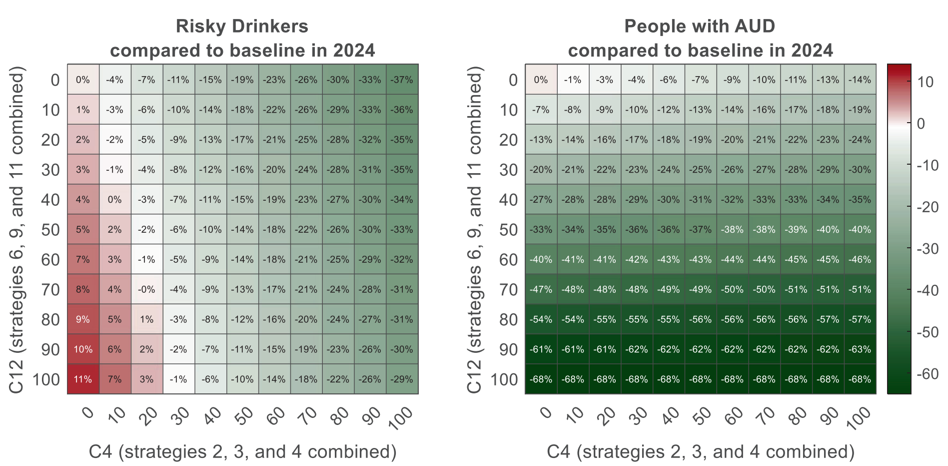

Fig. 4. Comparing the outcomes of C4 and C12 combination of strategies. See Table 2 for definitions of C4 and C12. Components of C4 and C12 are changed between 0 and 100%. Color code represents percent change of the outcome compared to the projected baseline in 2024, with positive outcomes (reduction) in green and negative outcomes (increase) in red [Color figure can be viewed at wileyonlinelibrary.com]

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Sensitivity analysis

Figure 4 presents the comparison of best-combined strategies to reduce RD (C4) and AUD (C12). Results show that when examined alone, C4 provides reductions in both RD (up to a 37% reduction) and AUD (up to a 14% reduction). However, the best AUD strategy has a clear "dampening" effect on C4 effects for both RD and AUD in multiple ways. First, results show that while C12 reduced the number of people with AUD, it increased RD if C4 is not implemented. Implementing both C12 and C4 together, where C4 consists of at least 30%–40% change, results in decreasing RD. However, implementing C12 strategies appears to reduce the effect of C4 strategies on RD, such that as C12 increases, C4 results in smaller reductions in RD. For example, when C4 flows are changed by 30%, RD reductions are highest when C12 flows are unchanged (11% RD reduction) and lowest when C12 flows are changed by 100% (1% RD reduction). Second, for every additional 10% change in C12 strategy flows, the impact of C4 on providing additional AUD reductions becomes weaker. For example, when C12 flows are changed by 20%, changing C4 flows can reduce AUD up to an additional 11% (from −13% to −24%). However, when C12 flows are changed by 80%, changing C4 flows will only provide up to a 3% additional reduction (from −54% to −57%).

Discussion

AU is a substantial global burden of disease and death (Peacock *et al.*, 2018); however, efforts to reduce AU and related harms have varying success (Knox *et al.*, 2019; Tanner-Smith and Lipsey, 2015). Effective strategies require considering the unique contexts of individual stages and the system of underlying mechanisms and external contexts facilitating long-term behavioral stability or change (Hussong *et al.*, 2018; Witkiewitz *et al.*, 2019). As AU-SD models are often used to identify and evaluate high-efficacy strategies (Matson *et al.*, 2021; Tawileh *et al.*, 2008), findings can be further enhanced by accounting for "sensitive periods" within the AU continuum (e.g. leverage points for an optimal change). For example, preventing the onset of AUD compared to RD will require different approaches, such as identifying RD-ers at high risk for AUD (Knox *et al.*, 2019) versus reducing social environments and norms that promote transitions from NRD to RD (Hussong *et al.*, 2018). Finally, using a stage-like structure can provide additional understanding of the multiple ways in which specific programs may or may not have effects. For example, although a program that promotes sobriety among RD-ers simultaneously reduces AUD onset, programs focusing on preventing AUD onset do not inherently promote sobriety for RD-ers (e.g. reduction in alcohol use problems/AUD risk but not complete transition to sobriety or transitioning out of RD (Tanner-Smith and Lipsey, 2015)).

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Our goal was to present an AU model that more closely aligns with empirical and theoretical alcohol research, using established operational definitions and representing the distinct patterns of AU marked by transitions between stages. Our model replicated historical trends while incorporating the broad stages and transitions between stages that have been considered part of the holistic continuum of AU across the lifespan. We accounted for multiple transition patterns evidenced within empirical literature by adding complimentary sobriety stocks for each drinking stage. These stages can provide valuable insight into the efficacy of programs that promote recovery through diverse pathways (Tucker and Simpson, 2011; Tucker *et al.*, 2020a). Adding more pathways between stages also allowed for representing a wider variety of known AU patterns, such as "skipping" experimental stages of AU by initiating AU onset through RD (Deutsch *et al.*, 2017), maturing out of RD (Lee and Sher, 2018), and multiple AUD recovery pathways (Witkiewitz and Tucker, 2020). Using operational definitions and stages, as described in the relevant alcohol research literature, allowed us to better utilize publicly available data and alcohol research for parameterization. Finally, by using the common definitions and constructs for model development and parameterization within alcohol research, our model can be easily used by alcohol researchers and SD modelers alike.

Probing prevention and intervention strategies at different stages

Given the lack of feedback loops, AU influence auxiliary variables, and specific programs to test, we discuss our results in terms of broad insight and application for future research, rather than definitive findings on effective areas for policy change. Our results indicated that focusing on preventing onset (primary prevention, secondary prevention) leads to the greatest reductions for RD and AUD compared to strategies for intervening within stages or preventing recurrence of stage transitions. This is not surprising given the fact that onset-prevention flows were the largest intervenable flows in the model, but this result does align with research demonstrating the benefit of earlier prevention as one of the strongest strategies for reducing overall disease (Arango *et al.*, 2018). However, given the costs and burdens associated with hazardous or problematic levels of AU (Rehm *et al.*, 2021), prevention strategies across the continuum cannot be ignored. Currently, there is little research on cumulative impact of individual stage-based strategies on a community-wide level (Stockings *et al.*, 2018). SD models can provide important information on the most effective synergies between programs and potential consequences of not considering them. For example, "over-investing" resources in effective early prevention programs may provide weaker benefits for heavy drinking communities with social environments that serve as incentives to transition to and remain in stages of hazardous drinking (Karriker-Jaffe *et al.*, 2020; Sudhinaraset *et al.*, 2016). However,

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overinvestment in programs that address current heavy drinkers will also reduce benefits by the larger number of individuals transitioning into these stages.

We explored differences between the multiple recovery or remission pathways. For AUD, promoting natural recovery that also allowed for low-risk drinking, in combination with preventing individuals who transitioned to sobriety from AUD to either relapse to AUD or transition to RD, provided the greatest reduction in AUD. This approach aligns with more recent discussions about the multiple pathways to AUD recovery and recognition of the large proportion of individuals who recover from AUD without treatment (Tucker *et al.*, 2020a; Witkiewitz and Tucker, 2020). Sobriety may not be necessary for all individuals, especially those with lower vulnerability for chronic or severe AUD (Witkiewitz *et al.*, 2019). Our best-combined strategy to reduce RD included primary prevention, promoting transitions to sobriety, and preventing individuals who transitioned from RD to sobriety from transitioning back to either RD or NRD. Coupling AUD prevention with RD intervention to sobriety may be an effective and integrative approach for individuals at high risk for AUD. This approach aligns with a contemporary discussion about prevention across the continuum of risk, especially within primary-care settings (Carvalho *et al.*, 2019; Knox *et al.*, 2019).

Finally, we tested a multistage approach and evaluated effective strategies across the continuum of prevention. Focusing on AUD exclusively resulted in increased RD, and although RD strategies provided minor reductions in AUD, RD strategies required a much higher rate of efficacy (e.g. 40% change in RD flows) compared to AUD strategies (e.g. 10% change in AUD flows). This effect can be, at least in part, explained by the fact that reducing any outflow to a stage will result in accumulation in that stage; reducing the outflow from RD to AUD will undoubtedly create an increase in RD that will require proportionally higher rates of change from other inflows and outflows to neutralize this increase. However, the dampening effect of AUD strategies on the effect of RD strategies for reducing both RD and AUD (at least for short-term projections) indicates that a careful balance must be considered to maximize reductions in both stages while accounting for finite resources for strategy implementation and a wide variety of options for and efficacy of AU-reduction programs and policies (O'connor *et al.*, 2018; Tanner-Smith and Lipsey, 2015). Focusing exclusively on clinical thresholds of hazardous AU for prevention/intervention efforts may insufficiently address the cumulative burden attributed to overall hazardous drinking.

Limitations

This research is subject to several limitations. First, our intent was to develop and evaluate a model structure for AU that addresses gaps in the current literature and be broadly used for SD projects that evaluate AU and

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AU consequences, rather than testing a complete SD model that would include feedback loops. Adding feedback loops may have influenced some of the policy tests, e.g. the bidirectional association between the population of hazardous drinkers and hazardous-drinking social influences on promoting transitions in or out of hazardous-drinking stages. Our model also did not account for potential gender, age, or race differences in AU patterns between and within stages (Banks and Zapolski, 2018; Lee *et al*., 2018; Wilsnack *et al*., 2018), as our focus here was to test a general model structure, based on both literature and data, using the entirety of a specific population. Further testing, including probing for differences between groups, is beyond the scope of the current article. However, our current model is well suited to account for AU pattern differences between groups in multiple ways (e.g. subscripts, separate models) to further probe leverage point equity as well as efficacy (Deutsch *et al*., 2022). Although TEDS data allowed us to refine our analyses down to site core-based statistical area, we were constrained to analyzing the restricted NSDUH data at the state level, which lead to lower precision for some parameters. As nationally representative datasets differ in reported trends and percentages of drinking behaviors such as binge drinking (Grucza *et al*., 2018), using a different dataset may have provided us with different results that could have impacted both model fit and findings.

We were also required to make some assumptions with our model. We constrained all individuals younger than 12 to the never-drinker stage. Longitudinal drinking patterns are stable for under-12 nonabstainers compared to adolescents/adults (Donovan and Molina, 2013), and NSDUH parameters are not generalizable to this age group. We excluded some flows that are seldom represented in data or literature to minimize the number of parameters and parameters requiring optimization, including transitions from sober NRD to RD or AUD and incomplete treatment transitions to stages other than AUD. However, our modeling strategy required trade-offs between which transitions were the most common and most important to explicitly model, and those that could be "aggregated" into other pathways.

Conclusions

Although generalized solutions exist, AU-reduction efforts are often most successful when tailored for different AU stages (Hussong *et al*., 2018; O'connor *et al*., 2018). Currently, most alcohol research utilizes frequentist statistical approaches that provide fine-grained understanding of individual constructs but have limited ability to capture complex systems. Systems science is a complementary tool to discern highly effective strategies in ways that overcome such limitations. However, this requires stronger alignment with the alcohol research used to develop stage-specific programs and policies. Despite limitations, our results indicate that this can be achieved within

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quantitative SD simulation models. As systems science becomes more visible within public health and behavioral science disciplines, grounding model constructs and systems in theoretical and empirical research will be critical in bridging the gap between visibility and adoption.

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Biographies

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Data S1. Supporting Information.

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