Phalanx Labs

Method paper

Research paper · Version 1.0

How to Stop Drinking Alcohol

Why drinking is hard to stop for people who are in control everywhere else, how to stop safely, and what a staged 90-day method targets, day by day

Phalanx Labs

31 October 2026

phalanxlabs.io

Where each section sits in the 90 days

Days 1 to 30	Days 31 to 60	Days 61 to 90
Brain chemistry restabilization	Impulse control	Identity rewiring

1 How do you stop drinking? The short answer — Background

2 Is it safe to stop drinking suddenly? — Days 1 to 15

3 Why can't I stop drinking when I'm in control everywhere else? — Days 1 to 51

4 Why do I want a drink at the end of every day? — Days 7 to 47

5 Does alcohol help you sleep, and why can't I sleep without it? — Days 3 to 5

6 What triggers the urge to drink? — Days 18 to 52

7 How long do alcohol cravings last? — Days 35 to 60

8 Can I just cut down instead of stopping completely? — Days 11 to 43

9 How do I stop drinking when everyone around me drinks? — Days 38 to 80

10 What happens to your body and brain when you stop drinking? — Days 6 to 25

11 What should I do if I drink again? — Day 15 onward

12 What does the Phalanx program target for someone stopping drinking, and when?

Days 81 to 90

13 What do our results show, and what can't this paper tell you?

Background

14 When should you see a doctor, and what is this program not?

Background

Sections	Figures	Tables	References	Reading time
14	15	5	88	80 min

phalanxlabs.io

Abstract

What works to stop drinking is changing the things that make you drink, not trying harder when you want one. Alcohol changes how your brain handles reward, stress and sleep. It also ties the urge to drink to the times, places, people and feelings where you've had a drink before, so willpower is weakest at the exact moments the urge is strongest. People who stop for good usually fix their sleep and stress first. Then they get rid of what they can, plan what they'll do about every trigger that's left, and keep support in place for the months when the risk of drinking again is highest. If you drink heavily every day, talk to a doctor before you stop, because alcohol withdrawal can be dangerous.

This paper explains each part of that answer and the research behind it. It covers why you can be in control at work and still lose to the evening drink, whether it's safe to stop suddenly, why you want a drink at the end of every day, why you can't sleep without it, what sets off the urge, how long cravings last, whether cutting down works, how to stop when everyone around you drinks, what happens to your body and brain once you stop, and what to do if you drink again. Each section starts with a short, direct answer. The detail and the evidence follow.

The last part of the paper describes what the Behavior Control System, the 90-day method Phalanx Labs runs with every client, targets for someone stopping drinking and on which days. Of the 290+ clients we have worked with across 12 countries over 7 years, 98.4% had already tried and failed to stop before they came to us. We report a 97% success rate, defined as the share of clients who ran the program and stayed off the habit through the program and the six months of aftercare that follow it, and we explain how it is tracked and what it cannot show. It comes from client follow-up, not from a controlled trial. The program is not therapy, clinical care or medical treatment, and it does not replace a doctor.

Keywords

how to stop drinking · alcohol withdrawal · alcohol cravings · GABA · glutamate · dopamine · endogenous opioids · tolerance · stress and relapse · alcohol and sleep

REM sleep | trigger reactivity | habit | relapse prevention | social networks | recovery identity

Section 01

How do you stop drinking? The short answer

In brief

You stop drinking for good by changing what makes you drink: your sleep, your stress, the triggers around you and the plan you have for the moments you want a drink. Trying harder in the moment is the approach that fails most often. If you drink heavily every day, see a doctor first.

Medical safety note

Alcohol withdrawal can be dangerous. If you drink heavily every day, talk to a doctor before you stop. Withdrawal can begin within hours of the last drink, and in severe cases it causes seizures or delirium tremens, a state of confusion that can be fatal. A doctor can tell you whether you need medical help to stop safely and can plan the stop with you. If you take benzodiazepines or other sleeping or anxiety medication, don't stop them suddenly or mix them with alcohol. If someone in withdrawal has a seizure, becomes confused or starts seeing things, call emergency services. Section 2 explains the risk in full.

Most people who want to stop drinking have already tried. Of the 290+ clients we have worked with across 12 countries over 7 years, 98.4% had already tried and failed to stop before they came to us, and alcohol is one of the habits they bring most often. The attempts were sincere: a dry month, a rule about weeknights, a switch to beer only, a promise made the morning after. The published record shows how common it is for those attempts to fail. Across studies of people stopping alcohol and tobacco, the curve that describes relapse has the same shape: losses are steepest soon after

stopping and slow down as sober time builds up[1]. In a 16-year study of 461 people who first sought help for a drinking problem, those who got no help were less likely to reach three years of remission and, if they did, more likely to relapse later[2].

The usual explanation for those failures is a lack of willpower. That explanation is wrong in a specific and useful way. Willpower, in the sense of deliberate control by the prefrontal cortex, is real, but it is the faculty most easily taken offline by the conditions in which most drinking happens: stress, fatigue, poor sleep and the presence of something that has been linked to drinking before[3-5]. Alcohol adds a problem of its own. Even at doses that don't impair thinking in general, alcohol weakens inhibitory control and makes the next drink more appealing[6]. So the decision not to drink is made by a rested, sober person in the morning and has to be carried out by a tired, stressed person in the evening, sometimes after a first drink has already lowered the brake. People are poor at predicting how different they will feel in that second state[7].

Modern addiction research explains what is happening underneath. Koob and Volkow describe addiction as a cycle of three stages, each run by different brain systems: drinking and its reward, in which dopamine and the brain's own opioids teach the habit and turn it into routine; withdrawal and low mood, in which the reward system responds less and the brain's stress systems take a bigger role; and preoccupation, in which craving rises and the prefrontal cortex can't hold it back[8]. Alcohol has its own version of each stage. It changes the balance between the brain's main calming and exciting chemicals, which produces tolerance and withdrawal. It disrupts sleep. It becomes the way a hard day ends. And it is legal, social and everywhere, so the triggers for it are hard to avoid.

phalanxlabs.io

Table 1. The ways people most often try to stop or control drinking, what each one changes, and what each one leaves in place. These are the attempts our clients most often describe before they came to us.

Attempt	What it changes	What it leaves in place
Stopping cold turkey on resolve	The decision, at the moment of temptation	Poor sleep, stress, the triggers of past drinking and the evening hours drinking used to fill. Relies on self-control at the time it is weakest, and is unsafe for heavy daily drinkers without a doctor.
Rules: weekends only, two glasses, no spirits	How much and how often	Each permitted drink weakens self-control and wakes up the learned urge, so the rule is applied by the person least able to keep it.
A dry month	Drinking, for a set period	An end date. The triggers, the stress and the way the day ends are waiting on the first day after it.
Switching drinks or switching days	What is drunk, or when	The same reward loop, running through a different bottle or a different night.
Reading, apps and podcasts	Understanding	Knowing why the urge happens does not provide a prepared response for the moment it starts.
Starting again on Monday	Motivation	Nothing structural. The conditions that led to the last drink are still in place for the next one.

phalanxlabs.io

Fig. 1 Two ways to stop drinking — Comparison

	— The usual attempt	✓ The approach in this paper
Safety	Stop today, whatever the amount	Heavy daily drinkers see a doctor first, and the stop date follows medical advice
Starting point	Stop on resolve, the morning after	Fix sleep, daylight, exercise and daily structure first; stop on Day 15
Main tool	Willpower in the evening	Conditions changed in advance, and a rehearsed response for each trigger that's left
Stress	Something to push through	The most common route to a drink, with its own set of responses
Sleep	Expected to sort itself out	Treated as a defence and rebuilt before the stop date
Social drinking	Faced on the night	Planned before each event; time with people who support not drinking increased
A drink after stopping	Proof of failure; the attempt ends	An event with a cause; the plan is changed the same week
Identity	Someone trying to cut down	Someone who doesn't drink, built through action and rehearsal

Figure 1. The usual attempt to stop drinking compared with the approach this paper describes, on the points that decide whether stopping lasts. The right-hand column summarizes the method set out in Sections 2 to 12.

The rest of this paper takes the questions people ask about stopping drinking one at a time. Section 2 answers whether it is safe to stop suddenly. Sections 3 to 7 explain why drinking is hard to stop: what alcohol does to the reward and habit systems, why the urge comes at the end of the day, what it does to sleep, how triggers are learned and how long cravings last. Sections 8 and 9 deal with cutting down and with stopping when everyone around you drinks. Section 10 describes what happens to the body and brain after stopping, and Section 11 what to do after drinking again. Section 12 sets out what the Behavior Control System targets for someone stopping drinking, day by day, and Section 13 reports our results and the limits of this paper. Where the program is described, we say what each day targets and why. We do not publish how.

Section 02

Is it safe to stop drinking suddenly?

Days 1 to 15, Brain chemistry restabilization

In brief

Not for everyone. If your body has adapted to heavy daily drinking, stopping suddenly can bring on withdrawal, and severe withdrawal can cause seizures and delirium tremens. Talk to a doctor before you stop. They can tell you whether you need medical support and plan the stop with you.

It depends on how much and how often you drink, and only a doctor can judge that for you. Withdrawal happens when a brain that has adapted to regular heavy drinking suddenly has no alcohol. Heavy drinkers who stop or cut down sharply can develop symptoms that range from tremors, irritability, anxiety and agitation to hallucinations, seizures and delirium tremens[9]. Symptoms can start within 6 to 24 hours of the last drink[10]. That is why the standing rule in this paper, and in our program, is simple: anyone who drinks heavily every day talks to a doctor before stopping.

2.1 The brake and the accelerator

The brain balances two main signalling chemicals. GABA is the main inhibitory transmitter: it calms nerve cells down and acts as the brain's brake. Glutamate is the main excitatory transmitter: it acts as the accelerator. Alcohol works on both. It acts largely through the GABA-A receptor,

increasing the brake's effect, which is a large part of why the first drink takes the edge off so quickly[11]. It also damps down glutamate signalling. The result is the familiar feeling of the first drink: shoulders drop, the noise in the head goes quiet and the day stops pressing.

When alcohol is present most days, the brain adapts to keep working. Davies's review describes both short-term and long-term changes in GABA-A receptors with continued alcohol exposure[11], and the glutamate system shifts the other way. This adaptation is what tolerance feels like: the same amount of alcohol does less. It also means that when the alcohol is suddenly gone, the brain is left with its brake turned down and its accelerator turned up. Saitz describes withdrawal as the result of alcohol-induced imbalances in brain chemistry that cause excessive nerve activity once alcohol is withheld[9]. The imbalance has been measured directly. In a magnetic resonance spectroscopy study of 47 alcohol-dependent patients in detoxification and 57 healthy controls, glutamate levels in prefrontal regions were significantly raised during acute withdrawal, and the changes returned to normal within a few weeks of abstinence; the same pattern was found in alcohol-dependent rats[12].

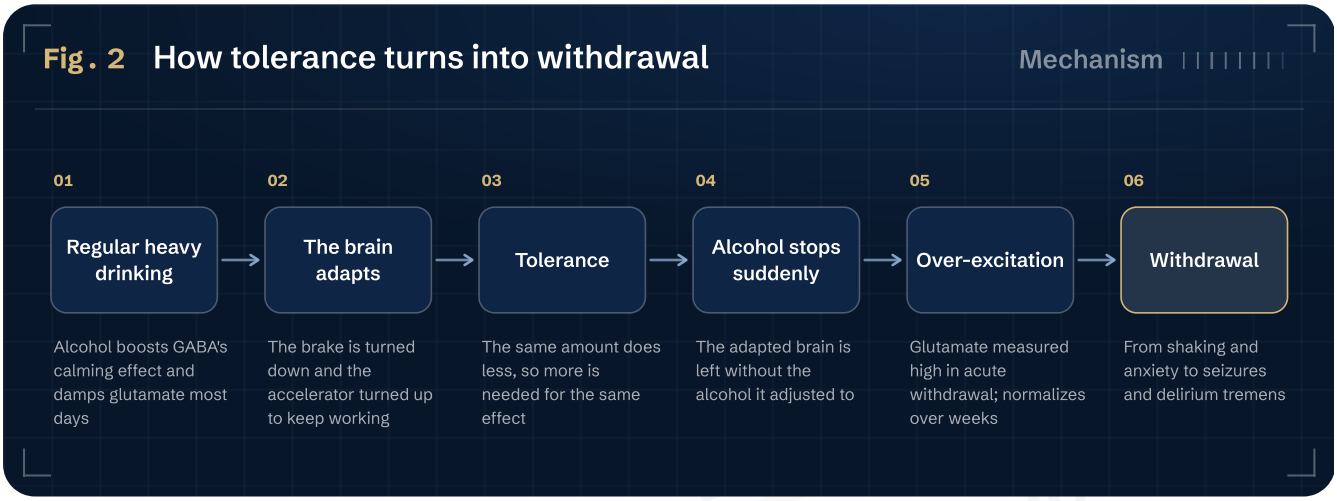


Figure 2. The chain from regular heavy drinking to withdrawal, assembled from the reviews and studies cited in Section 2.1. Each link is documented separately; the chain as drawn is our summary. It is why a heavy daily drinker's stop is planned with a doctor.

2.2 What withdrawal looks like, and when

Mirijello and colleagues' clinical review gives the time course that doctors work from[10]. Symptoms can develop within 6 to 24 hours after drinking stops or drops sharply. Many cases are mild and pass within two to seven days of the last drink. Up to half of patients with alcohol use disorder have withdrawal symptoms when they stop. Of patients who show withdrawal symptoms, almost one in ten develops withdrawal seizures, usually 24 to 48 hours after the last drink. Delirium tremens occurs in about 5% of patients with withdrawal, usually 48 to 72 hours after the last drink but sometimes up to ten days later, and it can be fatal. The review lists previous withdrawal episodes, previous withdrawal seizures or delirium tremens, severe dependence, older age and other illness among the factors that raise the risk.

Table 2. The usual time course of alcohol withdrawal, as described in the clinical review by Mirijello and colleagues (2015), with early symptoms as listed by Saitz (1998). Timings vary between people. This table explains why heavy daily drinkers need a doctor before they stop. It is not a guide to managing withdrawal at home.

Time since the last drink	What can happen
6 to 24 hours	Symptoms can begin: tremors, irritability, anxiety, agitation, signs of an overactive nervous system
24 to 48 hours	Withdrawal seizures, in almost 10% of patients who show withdrawal symptoms
48 to 72 hours, and up to 10 days	Delirium tremens, in about 5% of patients with withdrawal; it is fatal in 1% to 5% of those patients
2 to 7 days	Milder withdrawal usually passes
Weeks to months	Some symptoms can persist after the acute period (see Sections 5 and 7)

Withdrawal can also get worse with each repetition. Becker's review describes a kindling process in alcohol withdrawal: in many people with alcohol dependence, withdrawal symptoms become more severe after repeated withdrawal episodes, and both clinical and experimental evidence support the mechanism[13]. Becker concludes that kindling may also add to relapse risk and to alcohol-related brain damage. For someone who has stopped and started many times on their own, this is a second reason to involve a doctor. The previous attempts may have made the next withdrawal harder, not easier.

2.3 What a doctor can do

Withdrawal has effective medical treatment. In the evidence-based guideline by Mayo-Smith for the American Society of Addiction Medicine, benzodiazepines reduced the severity of withdrawal, reduced delirium by 4.9 cases per 100 patients and reduced seizures by 7.7 per 100 patients, with dosing best guided by withdrawal scales[14]. Mirijello and colleagues describe benzodiazepines as the gold-standard treatment, with severe cases sometimes needing intensive care[10]. Heavy drinking can also leave people short of thiamine. Thiamine deficiency causes Wernicke's encephalopathy, a serious brain disorder that is still often missed, and Sechi and Serra emphasize thiamine for its prevention and for preventing Korsakoff's syndrome in people who misuse alcohol[15]. Decisions about either belong to a doctor.

A doctor can also prescribe medication that helps people stay stopped. In a systematic review and meta-analysis of 122 randomized trials by Jonas and colleagues, acamprosate and oral naltrexone were both associated with fewer people returning to drinking: about 12 people needed to be treated with acamprosate, and about 20 with naltrexone, to prevent one return to any drinking, with no significant difference between the two when compared directly[16]. Imaging work suggests one reason naltrexone helps: drinking alcohol releases the brain's own opioids in the nucleus accumbens and orbitofrontal cortex, and naltrexone blocks the receptors they act on[17]. Schuckit's review of alcohol use disorders lists detoxification, cognitive-behavioral approaches to prevent relapse and the judicious use of medication to reduce craving among the established treatments[18]. Our program does not prescribe or recommend medication. It works alongside whatever medical care a person has, and it never replaces it.

In the program · Phase 1, Brain chemistry restabilization — **Days 1 to 15**

Before Day 1	**Medical safety** The Root-Cause Diagnostic asks how much and how often the person drinks, and about any previous withdrawal. Anyone who drinks heavily every day is told to see a doctor before they begin. Their stop on Day 15 follows medical advice: the doctor sets the date and the method, and the program builds the rest of the 90 days around it.
Day 15	**Medical safety** On the day abstinence begins, the client is given general information about supplements and told to consult a doctor before taking any of them. The program does not prescribe or recommend medication or supplements.

What this means in practice

If you drink every day, and especially if you drink in the morning, shake or sweat when you go without, or have had withdrawal before, don't stop on your own. See a doctor first. If you drink less than that and a doctor has told you stopping is safe for you, the rest of this paper applies to you directly.

Section 03

Why can't I stop drinking when I'm in control everywhere else?

Days 1 to 51

In brief

Because drinking isn't run by the part of you that makes decisions at work. Years of drinking train your reward and habit systems to want alcohol and to reach for it automatically, while the part of the brain that holds your plans is at its weakest in the evening, and weaker still after the first drink.

Being in control at work and losing to the evening drink is not a contradiction. The two happen in different brain systems and under different conditions. At work, decisions are made by goal-directed control, supported by the prefrontal cortex, in daylight, with a clear head and a reason to stay sharp. Drinking happens later, when the prefrontal cortex is tired and stressed, in settings the brain has linked to alcohol many times, and it is driven by reward and habit systems that respond to those settings before any decision is made[8, 19]. The person hasn't lost their discipline. They are using it in a place where it has little say.

phalanxlabs.io

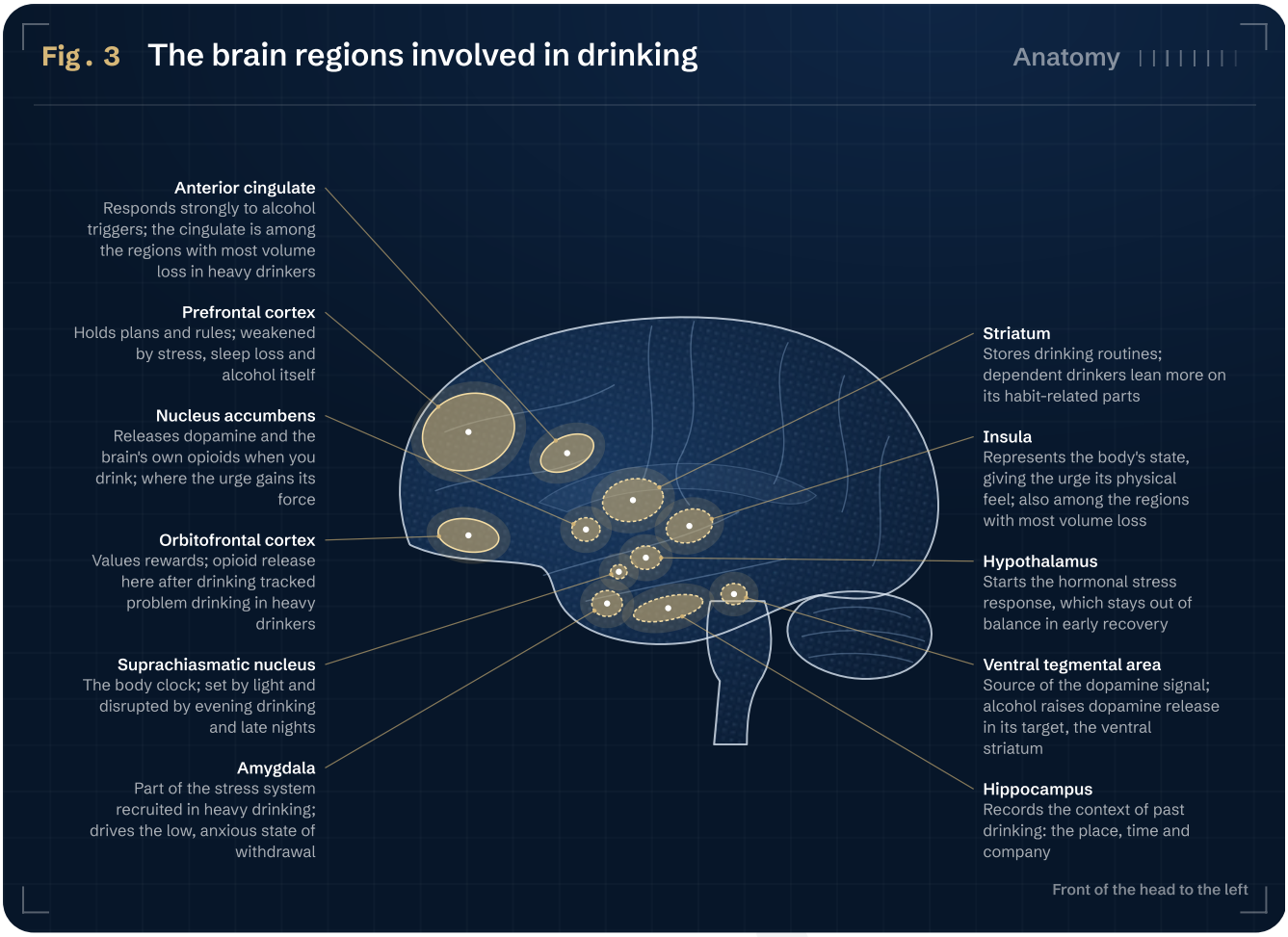


Figure 3. A simplified map of the regions this paper refers to, with the role each plays in drinking and in stopping. The roles summarize the studies cited in Sections 2 to 7. Real function is spread across circuits, and no region acts alone.

3.1 What a drink does to the reward system

Alcohol acts on the reward system directly. In a PET imaging study by Boileau and colleagues, six healthy volunteers drank either alcohol in orange juice or orange juice alone; after alcohol, the scans showed increased dopamine release in the ventral striatum, including the nucleus accumbens, and the size of the change tracked the stimulant effect of the drink on heart rate[20]. Alcohol also releases the brain's own opioids. Mitchell and colleagues scanned heavy drinkers and controls before and after a drink and found opioid release in the nucleus accumbens and the orbitofrontal cortex, both regions involved in valuing rewards; in the heavy drinkers, the change in the orbitofrontal cortex correlated with problem drinking and with how high they felt[17]. A drink therefore delivers two rewards at once: the calming effect on GABA described in Section 2 and a dopamine and opioid signal in the reward system.

Dopamine's main job in this system is teaching. In the work that defined the modern view, dopamine neurons responded to the difference between the reward received and the reward expected, and with experience the response moved from the reward itself to the earliest reliable sign that it was coming[21]. For a drinker, the earliest signs are often the end of the working day, the

walk into the kitchen, the sound of a bottle opening and the people they usually drink with. Those become the moments the reward system responds to. Berridge and Robinson add a distinction that explains why drinking can continue long after it stops being much fun. The brain's systems for wanting a reward are separate from its systems for liking it, and repeated use can strengthen wanting, especially wanting set off by associated triggers, without strengthening liking[22].

Definition: wanting and liking

Wanting is the motivational urge a trigger produces once the brain has learned that it predicts reward. Liking is the pleasure of the reward itself. The two run on different brain systems. With repeated drinking, wanting can grow while liking stays the same or falls, which is why a person can feel a strong urge for a drink they no longer much enjoy.

3.2 What years of drinking do to it

Over time, the reward system responds less to everything else. Volkow and colleagues gave 20 recently detoxified people with alcohol dependence and 20 controls a dose of a stimulant and measured the dopamine response with PET. In the ventral striatum, where the nucleus accumbens sits, and in the putamen, the dopamine increase was 70% and 50% lower than in controls; in controls but not in the alcohol-dependent group, activity in the orbitofrontal cortex was linked to the size of the dopamine response, which the authors suggest may underlie a reduced sensitivity to rewards[23]. Most of the people in that study also smoked, and a stimulant is not a natural reward, so the finding is a measure of a blunted system rather than a direct measure of how a sunset or a good meal feels. It fits what heavy drinkers describe: ordinary life feels flatter, and the drink becomes one of the few things that still lifts the evening.

phalanxlabs.io

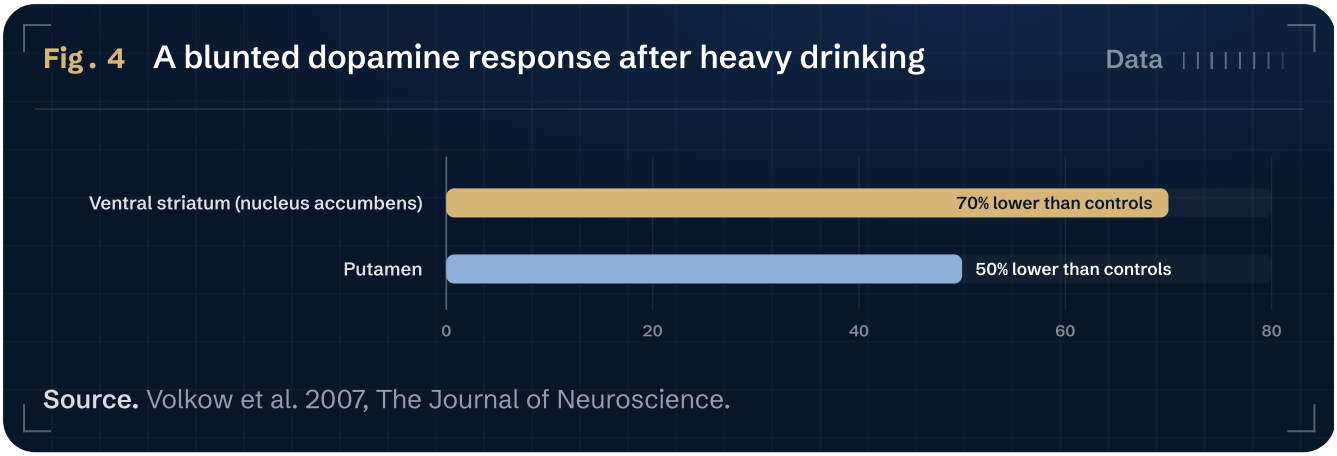


Figure 4. How much lower the dopamine increase to a stimulant dose was in 20 recently detoxified people with alcohol dependence than in 20 controls, by brain region, as reported by Volkow and colleagues (2007). Most of the alcohol-dependent participants also smoked. These are the study's figures. They describe a group, not any one person, and they were measured early in abstinence.

Koob and Volkow's review places this change in the second stage of their cycle. In the withdrawal and negative-mood stage, the dopamine part of the reward system works less well, while brain stress chemicals such as corticotropin-releasing factor and dynorphin are recruited in the extended amygdala[8]. The person drinks less to feel good and more to stop feeling bad, a shift Section 4 examines in detail.

3.3 From a choice to a habit

Repetition also changes who is in charge of the behavior. Everitt and Robbins describe addiction as a shift from goal-directed control, which weighs an action by its expected outcome, to habitual control, which runs a practiced response directly from the setting, with prefrontal control weakening along the way[19]. Habit research describes the same process in ordinary life: behavior repeated in a stable context comes to be triggered by that context with little reference to current goals[24]. In alcohol dependence this shift has been measured. Sjoerds and colleagues compared 31 abstinent alcohol-dependent patients with 19 matched controls on a learning task built to separate the two kinds of control. The patients relied more on stimulus-response habits, with less engagement of brain areas linked to goal-directed action, the ventromedial prefrontal cortex and anterior putamen, and more of an area linked to habit, the posterior putamen[25].

Stress pushes the balance further toward habit. In a laboratory study, people who were stressed before learning a task kept working for a food they had just eaten their fill of, while unstressed people stopped; stress made their behavior insensitive to what the outcome was now worth[4]. This is the experience of deciding at nine in the morning that tonight will be different and finding at seven in the evening that the decision has no grip. The morning decision came from goal-directed control. The evening is run by a habit triggered by the setting, after a day of stress, and the system that made the decision isn't the one in charge.

3.4 Why the first drink matters so much

Alcohol weakens the brake that would stop the next drink. Field and colleagues' review shows that alcohol impairs inhibitory control at doses that don't impair thinking more generally, that this effect is linked to alcohol's ability to prime further alcohol seeking, and that alcohol also changes how strongly alcohol-related things grab attention[6]. A plan to have one drink is therefore carried out by a person whose control has been lowered by that drink. This is the plainest reason why rules about quantity fail so often for people who already struggle to stop, a point Section 8 returns to.

The way the brain values time adds to it. Delay discounting is the tendency to value a reward less the further away it is. Petry compared people actively drinking heavily, people with alcohol dependence who were abstinent and controls, and found the steepest discounting in the active drinkers, intermediate rates in the abstinent group and the shallowest in controls; alcohol itself was discounted more steeply than money[26]. A meta-analysis confirmed greater discounting across addictive behaviors[27]. Bickel and colleagues describe this short time horizon, together with an excessive value placed on one reward, as the core of what they call reinforcer pathology, and argue that lengthening the horizon and building other rewards are both routes out[28]. Tonight's drink is immediate and certain. A better morning, a clearer month and a healthier year are far away. The program works on both sides of that comparison.

In the program — Days 1 to 51

Days 1 , 2 and 10	**Understanding the loop** The client learns how repeated high-intensity rewards produce tolerance, how the brain changes with repetition and what dopamine does in that process, and then the five-stage cycle the program uses to describe every habit, so they can start recognizing each stage in their own nightly log.
Days 17 and 51	**Reward system and delay discounting** On Day 17 the client takes up a demanding new pursuit with clear goals and room to improve, and gives it time every day, so that an earned reward begins to compete with the drink. On Day 51 they build a vivid picture of their future self, to call on when a craving makes tonight's drink feel bigger than everything that follows it.

Understanding comes first in the program because it changes how the rest of the work is experienced. A client who knows that the evening urge is a learned response, produced by systems that change with sustained abstinence, can watch it happen instead of treating it as proof of weakness. One of our clients, Bjorn, a senior operator in the Netherlands who came to us for alcohol, put it this way: “Now I know how my brain works and what it needs. That's the difference.” The demanding new pursuit on Day 17 rests on animal research in which an enriched environment, with novelty, exercise, social contact and challenge, both prevented the development of drug addiction and reduced relapse-like behavior[29], and on the case for alternative rewards set out by Bickel and colleagues[28].

Section 04

Why do I want a drink at the end of every day?

Days 7 to 47

In brief

Because the evening drink has become the way your day ends and your stress lets go. Over time it also relieves a tense, restless feeling that the drinking itself has made worse, so the urge gets stronger the more you rely on it.

The evening drink usually starts as relief and turns into need. At first it takes the edge off a hard day. With repetition, the brain's stress systems adjust to it, the hours without alcohol start to feel more tense than they used to, and the drink is increasingly used to get back to normal rather than to feel good. The end of the day is when stress has built up most, when self-control is most tired and when the setting, the time and the routine all point toward a drink. That's why the urge arrives at the same time most days.

4.1 Drinking for relief

Alcohol's effect on stress has been studied for decades. Sher and Levenson's laboratory work on what they called the stress-response-dampening effect of alcohol linked individual differences in that effect to risk for alcoholism[30]. Cooper and colleagues later showed that people drink for distinct reasons, and that two of them matter most: drinking to cope, to escape or reduce negative feelings, and drinking to enhance, to increase positive ones. In two random samples, one of adults and one of adolescents, these motives were the closest predictors of drinking and drinking problems, with different causes and different consequences[31]. Both are common among our clients. The evening drink after a hard day is coping. The drink after a good one, a deal closed or a week finished, is enhancement, and celebration can be as strong a trigger as stress.

Anxiety and drinking feed each other. Kushner, Abrams and Borchardt's review concluded that anxiety disorders and alcohol problems can each set off the other, and proposed a cycle in which alcohol's short-term relief of anxiety, combined with the longer-term anxiety produced by heavy drinking and withdrawal, drives both upward[32]. Many drinkers recognize the second half of that cycle as the anxious, on-edge morning after.

4.2 How relief turns into need

Koob and Le Moal proposed that addiction is best understood through allostasis: the brain keeps apparent stability by shifting its set point, and the processes that normally counter a drug's effect fail to return to their usual range, leaving the person in a lasting deviation from normal reward function[33]. Koob later described how brain stress systems, including corticotropin-releasing factor in the extended amygdala, are recruited as dependence develops and come to drive use through negative reinforcement: the drink is repeated to relieve a negative state that drinking itself has deepened[34]. Becker's review of alcohol dependence makes the same point in clinical terms: withdrawal-related anxiety, which reflects adaptive changes in the body's stress response system, is one factor contributing to relapse, and the fear of withdrawal can itself keep drinking going[35].

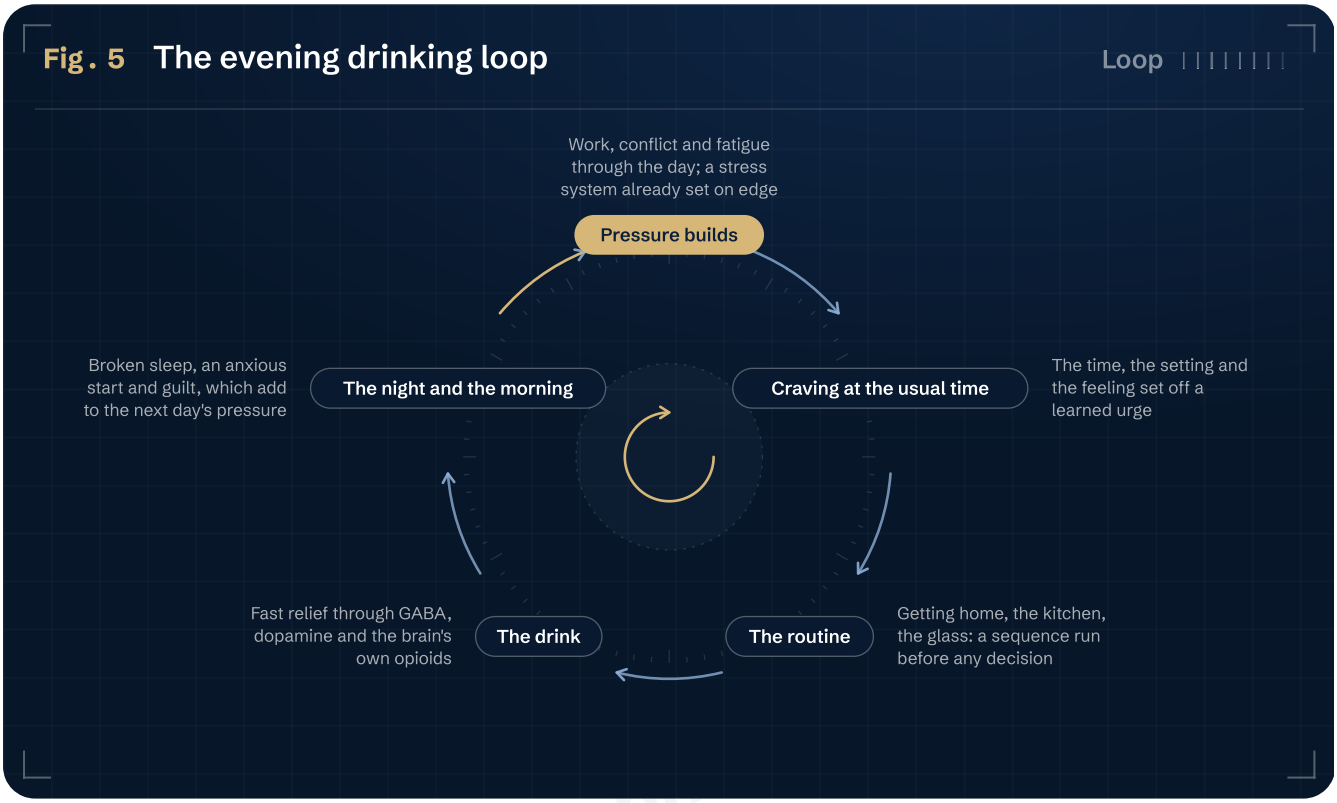


Figure 5. The cycle behind a daily evening drink, built on the five-stage loop the program teaches on Day 10 and on the studies cited in Sections 3 and 4. Each stage is a point where the program intervenes: Phase 1 lowers the stress and sleep debt that feed the first stage, Phase 2 builds responses for the craving and the routine, and Phase 3 and aftercare work on the guilt and self-judgment that restart the cycle.

The low mood and anxiety of early sobriety have a known course. Heilig, Egli, Crabbe and Becker's review notes that the depressed mood and raised anxiety of early withdrawal occur in the vast majority of alcohol-dependent patients and in most cases ease over three to six weeks of abstinence, but that the risk of relapse lasts much longer[36]. Animal work reviewed in the same paper shows that a long history of dependence leaves subtler changes that alter emotional processing and increase sensitivity to stress well into protracted abstinence, and emerging human data support the clinical

relevance of that negative emotionality for relapse. Explaining this in advance changes how a hard evening in the fifth week is read. It is an expected stage with a known cause and a known direction, not a sign that stopping isn't working.

4.3 Stress predicts drinking again

Stress is one of the most consistent predictors of relapse to alcohol. Sinha and colleagues studied treatment-engaged, alcohol-dependent patients after a month of abstinence. Compared with controls, the patients showed a dysregulated hormonal stress system, more anxiety and more craving when exposed to stress and to alcohol triggers in the laboratory. They were then followed for 90 days. High craving provoked by stress and by alcohol triggers, and a measure of adrenal sensitivity taken in a relaxed state, each predicted a shorter time to relapse[37]. Stress also acts on the faculty people use to resist. Even mild, uncontrollable acute stress can rapidly impair prefrontal function[3], and stress shifts control toward habit[4].

So the evening drink can't be treated as a question of willpower alone. The person needs ways to bring their stress down that work as quickly as a drink and that can be used at the moment the stress peaks. Naming the feeling is one of them: in an imaging study, putting a feeling into words reduced the amygdala's response to emotional images and increased activity in a region of the prefrontal cortex linked to inhibitory control[38]. Slow breathing is another. In a study of 40 healthy adults, eight weeks of diaphragmatic breathing practice improved sustained attention, reduced negative mood and lowered cortisol[39]. Those studies were of general populations, not of drinkers, and we cite them for the mechanism: short, learnable practices that lower arousal within minutes.

In the program — Days 7 to 47

Days 7 and 16	**Stress system and attention to internal states** A short daily meditation practice begins on Day 7. On Day 16, controlled heat and cold exposure is added within limits on duration and frequency, as a chosen, controllable physical stressor that gives a strong reset at the time of day drinking used to fill. Anyone with a heart condition, high blood pressure or another medical condition checks with their doctor first.
Days 44 to 47	**Stress system and emotion** The client learns to name their emotional states precisely, learns how the body's stress response works and why stress is the most common route back to a drink, and then learns a slow breathing practice and a muscle relaxation practice for lowering arousal on purpose when the pressure builds.

Section 05

Does alcohol help you sleep, and why can't I sleep without it?

Days 3 to 5, Brain chemistry restabilization

In brief

Alcohol helps you fall asleep faster, but it breaks up the second half of the night and cuts down dreaming sleep, so you wake up less rested. When you stop, sleep often gets worse before it gets better, and poor sleep is linked to drinking again. That's why sleep is fixed before the stop date.

Alcohol puts you to sleep and then ruins the sleep it gave you. It shortens the time it takes to fall asleep, but as the body breaks it down, the second half of the night becomes lighter and more broken, and dreaming (REM) sleep is delayed and, at higher doses, reduced[40]. After years of drinking, the brain has also learned to fall asleep with alcohol, so the first weeks without it often bring trouble falling asleep and waking in the night. Sleep improves slowly with time, and for some people it takes more than a year to settle fully[41]. Because poor sleep in early sobriety predicts drinking again[42], we treat sleep as one of the main defences, not as a side effect to wait out.

5.1 What a drink does to the night

Ebrahim and colleagues' review of alcohol and normal sleep found that at all doses studied, alcohol shortens the time taken to fall asleep and makes the first half of the night more consolidated, with more disrupted sleep in the second half. It delays the first period of REM sleep, and total REM sleep is reduced at moderate and high doses[40]. Colrain, Nicholas and Baker reach the same conclusion and add what happens with heavy, long-term drinking: alcohol dependence is associated with chronic sleep disturbance, less slow-wave sleep and more REM sleep than normal, changes that last long into abstinence and may play a part in relapse[43]. Their review also notes evidence of tolerance to alcohol's sedative effect with repeated use, which is part of why the nightcap tends to grow.

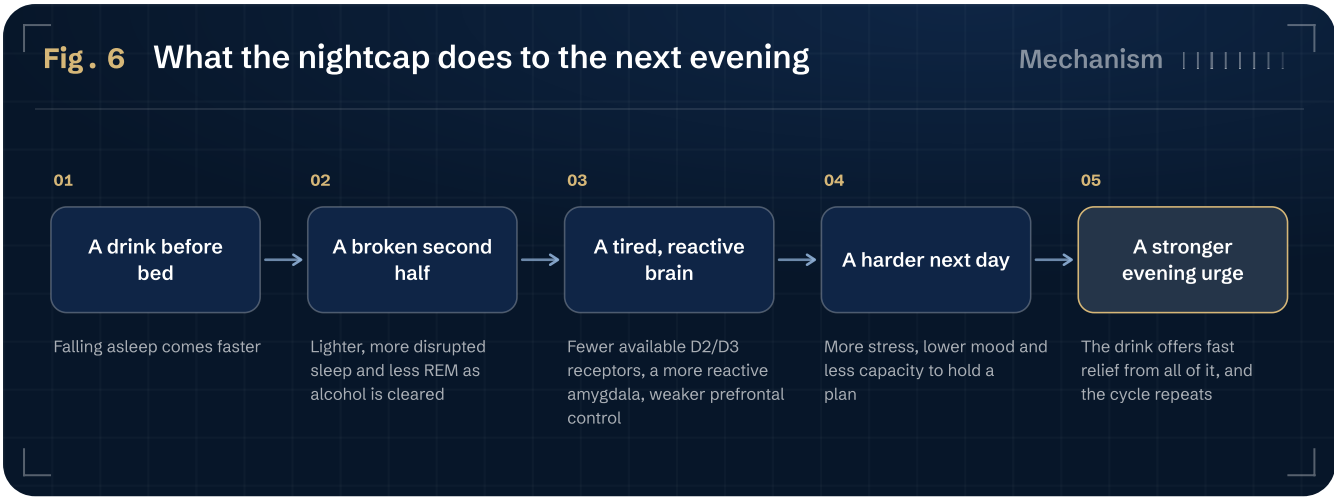


Figure 6. The chain by which drinking at night raises the urge to drink the following evening, assembled from the studies cited in Section 5. Each step is documented separately; the full chain is our synthesis. The program breaks it before the stop date by fixing sleep timing, light and the bedroom.

Sleep loss acts directly on the systems that drive drinking. One night without sleep reduced the availability of dopamine D2 and D3 receptors in the human ventral striatum[44]. In another study, a night without sleep made the amygdala respond more strongly to negative images and weakened its connection with the medial prefrontal cortex, which normally keeps it in check[45]. A poor night therefore produces, the next day, a reward system that responds less to ordinary things, an emotional system that reacts more to stress, and weaker control over both. A drink relieves all three for an hour, and makes the next night worse.

5.2 Sleep after stopping, and why it matters

Sleep recovers after stopping, but slowly. Drummond and colleagues followed men with alcohol dependence at about 16 days, 19 weeks, 14 months and 27 months of abstinence. Sleep was short, fragmented and shallow early on, improved slowly over at least the first year, and some aspects were still abnormal after 27 months; insomnia and broken sleep at around five months may have been related to relapse by 14 months[41]. The later follow-ups were small, with nine men at 14 months and four at 27 months, so the long-term figures are tentative. The direction is clear enough to plan around: the first weeks without alcohol are likely to bring worse sleep, not better, and the person needs other ways to fall and stay asleep.

The link to relapse is the reason sleep comes first in the program. Brower and colleagues studied 172 men and women in treatment for alcohol dependence. Of them, 61% had insomnia in the six months before treatment, and those with insomnia were twice as likely to report often drinking to help them sleep (55% against 28%). Among the 74 patients followed for an average of five months after treatment, 60% of those with insomnia at the start drank again, against 30% of those without, and insomnia remained a strong predictor after other factors were taken into account. A history of

drinking to sleep did not itself predict relapse[42]. The finding is an association, not proof that fixing sleep prevents relapse. But it shows that someone who stops drinking while their sleep is broken is starting with one of the strongest known risks still in place.

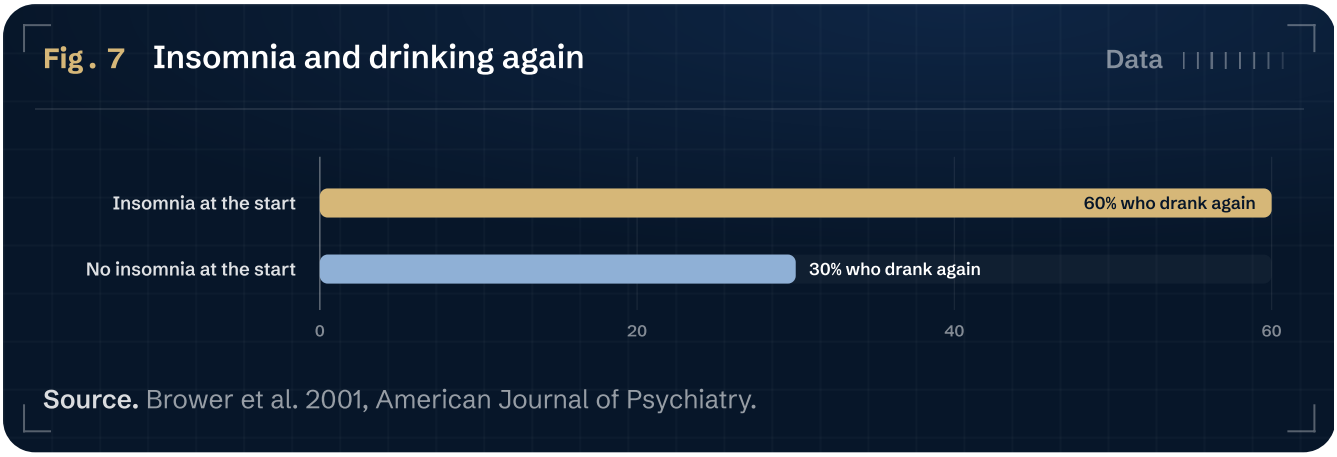


Figure 7. The share of patients treated for alcohol dependence who drank again during an average of five months of follow-up, by whether they had insomnia at the start of treatment, as reported by Brower and colleagues (2001) for the 74 patients followed. These are the study's figures. They show an association, not that insomnia caused the relapses.

5.3 The body clock

Sleep is timed by the body clock, and the clock is set mainly by light. Light acts on the master clock in the suprachiasmatic nucleus and on other regions to regulate circadian rhythms, sleep and mood[46]. The circadian system also regulates reward processing, which is one reason disrupted rhythms and substance use are so often found together[47]. Many of our clients who drink in the evening also keep late hours under artificial light and use screens in bed, so their clock is weakly anchored before alcohol is even considered. A fixed wake time anchors it from one end, daylight early in the day strengthens the anchor, and less light in the evening stops it being pushed later at the other end.

In the program · Phase 1, Brain chemistry restabilization **Days 3 to 5**

Body clock and sleep

On Day 3 the client fixes their sleep and wake times, seven days a week. On Day 4, morning daylight and a daily walk are added. On Day 5, evening light is reduced and the phone leaves the bedroom. By Day 15, when drinking stops, the client has had nearly two weeks of a steadier night to fall back on.

Why sleep comes before the stop date

Stopping drinking takes away the thing many people use to fall asleep. If the stop comes first, the person meets the worst nights of early sobriety with nothing else in place, and poor sleep is one of the strongest known predictors of drinking again. Rebuilding sleep timing, light and the bedroom in the two weeks before Day 15 means the stop lands on firmer ground.

Section 06

What triggers the urge to drink?

Days 18 to 52

In brief

The places, times, people, objects and feelings that were around when you drank before. Your brain has linked them to alcohol, so any one of them can start a craving before you've decided anything. Stopping doesn't erase those links, so each trigger needs a plan.

Triggers are the things your brain has learned to link with drinking. Every time you drink, the brain records what was around you: where you were, what time it was, who you were with, how you felt and what had just happened. Repeat that often enough and each of those things starts the urge on its own. That's why getting home, finishing a big piece of work, a particular friend, a bad phone call, Friday evening or a glass in the cupboard can bring on a craving out of nowhere. Good news can be as strong a trigger as bad news. And because stopping doesn't delete what the brain has learned, the triggers are still there after the drinking stops.

6.1 How triggers are learned

The learning is the dopamine prediction signal described in Section 3: the reward response moves from the drink to the earliest reliable sign that a drink is coming[21]. Researchers measure the result by showing people the sights, smells or objects linked to their habit and recording their reaction, a method they call cue reactivity, which this paper calls trigger reactivity. A meta-analysis across addictions found that exposure to habit-related triggers reliably increased reported craving, with

smaller effects on physical arousal[48]. For alcohol specifically, Schacht, Anton and Myrick pooled 28 brain imaging studies covering 679 people with alcohol use disorders and 174 controls. Alcohol triggers reliably activated limbic and prefrontal regions, including the ventral striatum, the anterior cingulate and the ventromedial prefrontal cortex, and activation of the ventral striatum was the response most often linked to behavior and most often reduced by treatment[49].

Trigger reactivity may also help predict who drinks again. In a pilot study by Grüsser and colleagues, ten abstinent alcohol-dependent patients and controls viewed alcohol and neutral images during brain scans, and the patients were then followed for three months. Alcohol images activated the putamen, anterior cingulate and medial prefrontal cortex in the patients, and the activation was most pronounced in the five who went on to drink again. The amount they later drank was linked to the strength of that brain response, not to how much craving they reported or how long they had been abstinent[50]. The study was small and the authors call it a pilot. Its point matters for anyone trying to stop: the strength of a trigger's hold can be greater than the person feels or reports.

6.2 Why stopping doesn't erase them

Bouton's review of extinction explains why triggers outlast the drinking. Stopping doesn't erase the original learning. It lays new learning over it, and the new learning depends more on context than the old. When the person returns to the setting, mood or company in which they used to drink, or has a drink again, the old response can come back[51]. Two practical conclusions follow. Triggers that can be removed should be removed, and the ones that can't need a new, prepared response, used and repeated in the settings where the old response was learned.

Removal comes first because it is the cheapest and most reliable form of control. Duckworth, Gendler and Gross's review argues that strategies which change the situation before an impulse begins are more effective than strategies for resisting an impulse already under way[52]. Verplanken and Wood describe how a change in the context where a habit runs disrupts its automatic triggering and opens a window in which new behavior is easier to establish[53]. If there is no alcohol in the house, the evening routine that ends at the fridge has nowhere to go.



Figure 8. Triggers grouped at the level the trigger-reactivity research uses, against the broad classes of response built in the program's second phase. A filled cell marks a response class commonly matched to that group. The actual match is made trigger by trigger in each client's plan on Day 52, and the specific responses are not published.

6.3 Noticing a trigger in time

A response only helps if it starts before the drinking routine has run. Quinn and colleagues compared the strategies people used to control strong and weak habits, and found that vigilant monitoring, watching for the signs of the habit and telling oneself not to do it, was the most effective strategy for strong habits, and that it worked by strengthening inhibitory control[54]. In the program, this kind of awareness is trained weeks before the full response plan is written, so that noticing a trigger is routine by the time there is a prepared response to use.

6.4 Planning a response for each trigger

The research on implementation intentions shows why the plan has to be specific. These are plans that state what a person will do when a particular situation arises. Gollwitzer and Sheeran's meta-analysis found that forming them had a medium-to-large effect on reaching goals, in part because they make the critical situation easier to notice and trigger the planned response with little deliberation when it arrives[55]. The effect has been tested on drinking directly. In a randomized trial by Armitage with 248 adults from the general population, the two groups who formed implementation intentions reduced their alcohol consumption, while the two control groups did not; choosing a ready-made plan worked as well as writing one's own[56]. Those participants were not trying to stop and most were not heavy drinkers, so the trial shows the mechanism rather than the

size of the effect for someone stopping. A response that has to be chosen in the moment is chosen by the person least able to choose well. A response tied in advance to its trigger runs with far less effort.

In the program — Days 18 to 52

Day 18	**Noticing triggers** The client begins continuous awareness training: a brief, structured check of the situation, their thoughts and their emotional state at each change of setting through the day, so that a trigger is noticed before the drinking routine is under way.
Days 30 and 31	**Trigger learning** The client learns how each drinking episode records the setting, time, company, mood and sensations around it, and then builds a complete map of their own triggers from the nightly log they have kept since Day 1, not from memory alone.
Day 32	**Context and access** Every trigger on the map that can be removed is removed: alcohol in the house, the routes, shops, subscriptions, apps and arrangements that make drinking easy, and the routine that comes just before the first drink. Where a trigger location can't be avoided, the client places a reminder of their commitment there.
Days 37 to 39	**The moment of temptation** The client learns the sequence every later response follows, from noticing a trigger to recording how the episode went; plans how to position themselves in drinking situations they can't avoid, such as a work dinner or a family event; and learns ways to break the drinking routine once it has started.
Day 52	**A response for every trigger** The client writes their own response plan: every trigger left on the map is paired with a specific, prepared response drawn from the tools of the first two phases, stated as what they will do when that trigger appears.

Section 07

How long do alcohol cravings last?

Days 35 to 60, Impulse control

In brief

A single craving rises, peaks and fades if you don't act on it, though no good study puts a fixed number of minutes on it. Over weeks, the low mood of early sobriety usually eases, but craving set off by triggers can stay strong or even grow for the first couple of months, and the risk of drinking again falls slowly over years. So the plan has to last well beyond the first month.

There are two answers, because there are two time scales. A single craving builds, peaks and passes, and each time it ends in a drink, the link between that trigger and drinking is reinforced. We haven't found reliable research that gives a fixed number of minutes, and we don't quote one. Over weeks and months, the picture is more complicated than most people expect. The anxious, low feeling of early withdrawal usually eases over three to six weeks[36], but craving set off by alcohol triggers was higher at 60 days than at 7 days in one study[57], and the risk of relapse falls with time without disappearing[1,2].

7.1 What happens in a single craving

A craving is felt in the body: restlessness, tension, a sense that the evening needs something. The sense of the body's internal state is represented in the insula, and the anterior insula has been proposed as a basis for moment-to-moment awareness of how one feels[58]. Kavanagh, Andrade and May's elaborated intrusion theory describes how such a feeling becomes a full craving episode[59]. A thought about drinking arrives automatically, set off by a trigger or a bodily state. If it promises relief or pleasure, the mind elaborates it, often as vivid images of the drink, and that elaboration fills working memory and makes the person more aware of what they are missing. The theory gives two points to work on: the physical state that feeds the first thought, and the elaboration that turns a thought into an episode.

Observing an urge without acting on it changes what happens next. In an experiment with 123 people who smoked, a brief instruction in urge surfing, watching the urge rise and fall without acting on it, did not reduce the strength of the urges people reported, but it did lead to fewer cigarettes over the following week[60]. That result is modest and useful. The practice changes what the person does with an urge, not whether the urge comes. In people with substance use disorders, a randomized trial found that mindfulness-based relapse prevention weakened the link between low mood and craving, and that this predicted less substance use four months later[61]. A larger trial is described in Section 11.

7.2 Cravings over weeks

Craving set off by triggers does not simply fade with sober time. Li and colleagues assessed adult men in inpatient treatment for alcohol dependence at 7, 14, 30 and 60 days of abstinence, using four separate groups of 80 and a further group of 19 tested at every point. Craving in response to alcohol triggers was highest at 60 days, in both designs, though heart rate, blood pressure and skin responses did not differ[57]. The authors call this incubation of craving, a pattern first described in animals and in people who smoke and use methamphetamine. The study was of men in an inpatient setting, and it stopped at 60 days, so it does not show when the curve turns down. What it shows is that the second month can bring stronger trigger-set craving than the first, at the same time as the withdrawal-related low mood is easing[36] and the glutamate imbalance of acute withdrawal has settled[12].

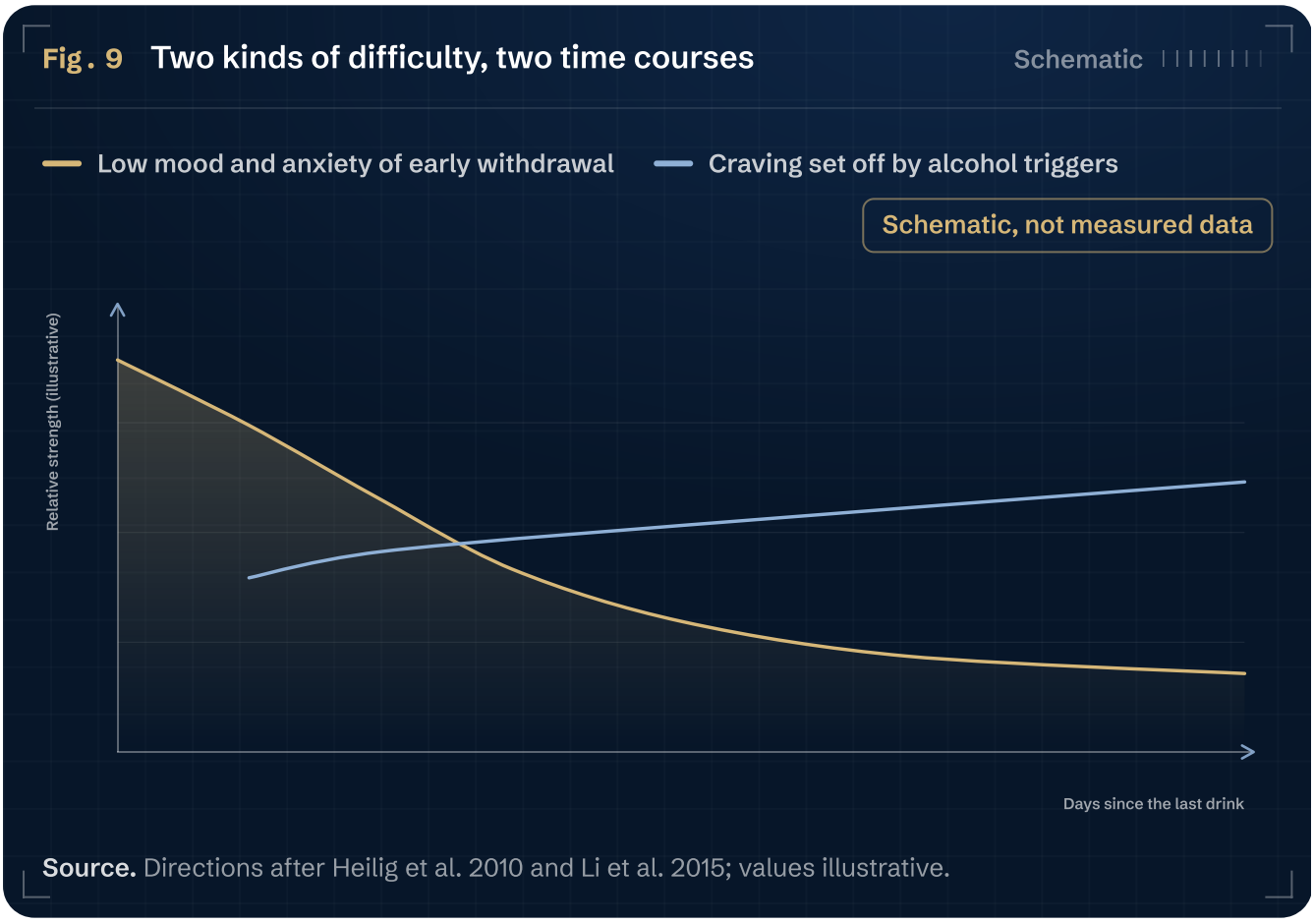


Figure 9. A schematic of two findings cited in Section 7.2. The low mood and anxiety of early withdrawal usually ease over three to six weeks (Heilig and colleagues 2010). Craving set off by alcohol triggers was highest at 60 days of the four points measured by Li and colleagues (2015), who did not test beyond 60 days. The values are illustrative and are not data from either study; they show only the direction each finding describes. The practical point shapes the program: the trigger work of Days 31 to 60 falls in the weeks when trigger-set craving may be strongest.

7.3 The risk over months and years

Kirshenbaum, Olsen and Bickel's quantitative review of the relapse curve across alcohol and tobacco found the same shape across studies: the proportion still abstinent falls fastest soon after stopping and then flattens as abstinent time accumulates[1]. Moos and Moos followed 461 people with drinking problems for 16 years from the point at which they first sought help. People who got no help were less likely to reach three years of remission and, if they did, more likely to relapse later. Among people who had remitted, those who drank more, saw their drinking as less of a problem, had less confidence in their ability to stay stopped and relied more on avoiding problems rather than dealing with them were more likely to relapse by 16 years[2]. The authors' conclusion is direct: people who cut down on their own may need help to keep it that way.

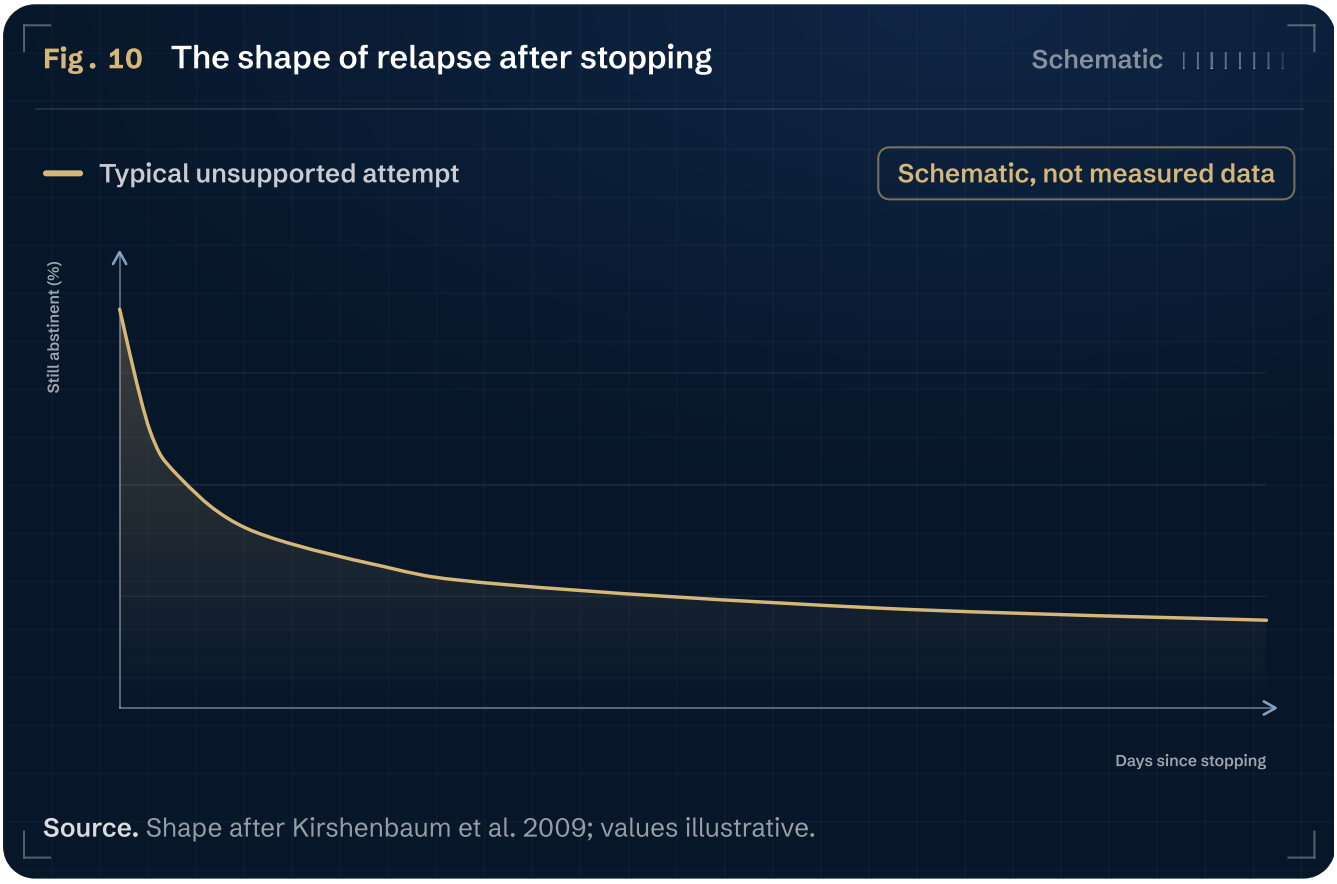


Figure 10. A schematic of the relapse curve described for alcohol and tobacco by Kirshenbaum and colleagues (2009): the share still abstinent falls fastest in the first weeks, then flattens without reaching a floor within months. The values are illustrative and show the shape only. The implication is that support has to be strongest at the start and has to continue after the first three months, which is why the program is followed by six months of aftercare.

In the program · Phase 2, Impulse control — **Days 35 to 60**

Day 35	**Why early sobriety is hard** The client learns why the early weeks without alcohol are hard: the brain has adapted to drinking and takes time to return toward balance. They learn why extra vigilance is needed in this period, so a hard evening is read as an expected stage rather than a sign that stopping isn't working.
Day 49	**The felt urge** The client learns to observe an urge as a physical event that rises, peaks and falls, without acting on it and without fighting it.
Days 53 to 60	**Practice under real conditions** The client uses their response plan every day, records every craving and how it was handled, and changes any response that didn't hold. The record lets them see how their cravings change in frequency and strength over the weeks, which is hard to see from inside a single evening.

Section 08

Can I just cut down instead of stopping completely?

Days 11 to 43

phalanxlabs.io

In brief

Some people can. But if you've set limits before and kept breaking them, stopping completely is usually the more reliable route, because the first drink weakens your self-control and wakes up the urge for the next. Our program asks for complete abstinence for that reason.

Some people who once had a serious drinking problem do go on to drink at low levels. But for someone who has repeatedly tried to cut down and failed, a limit is hard to keep for a specific reason: each drink lowers the self-control needed to stop at the limit and makes the next drink more appealing[6], and each drink also reactivates the learned link between drinking and its triggers[51]. A

limit has to be applied by the person a drink has just made less able to apply it. That's why our program asks for complete abstinence, and why we'd suggest the same to anyone whose own rules haven't held.

8.1 What the population data show

The honest starting point is that recovery without abstinence does happen. Dawson and colleagues analyzed a nationally representative US survey of 43,093 adults and looked at the 4,422 who had met the criteria for alcohol dependence before the past year. In the past year, 25.0% were still dependent, 27.3% were in partial remission, 11.8% drank without symptoms but in a pattern that put them at risk of relapse, 17.7% were drinking at low risk and 18.2% were abstaining. Only a quarter had ever received treatment. Greater severity of dependence made abstinent recovery more likely and recovery without abstinence less likely[62].

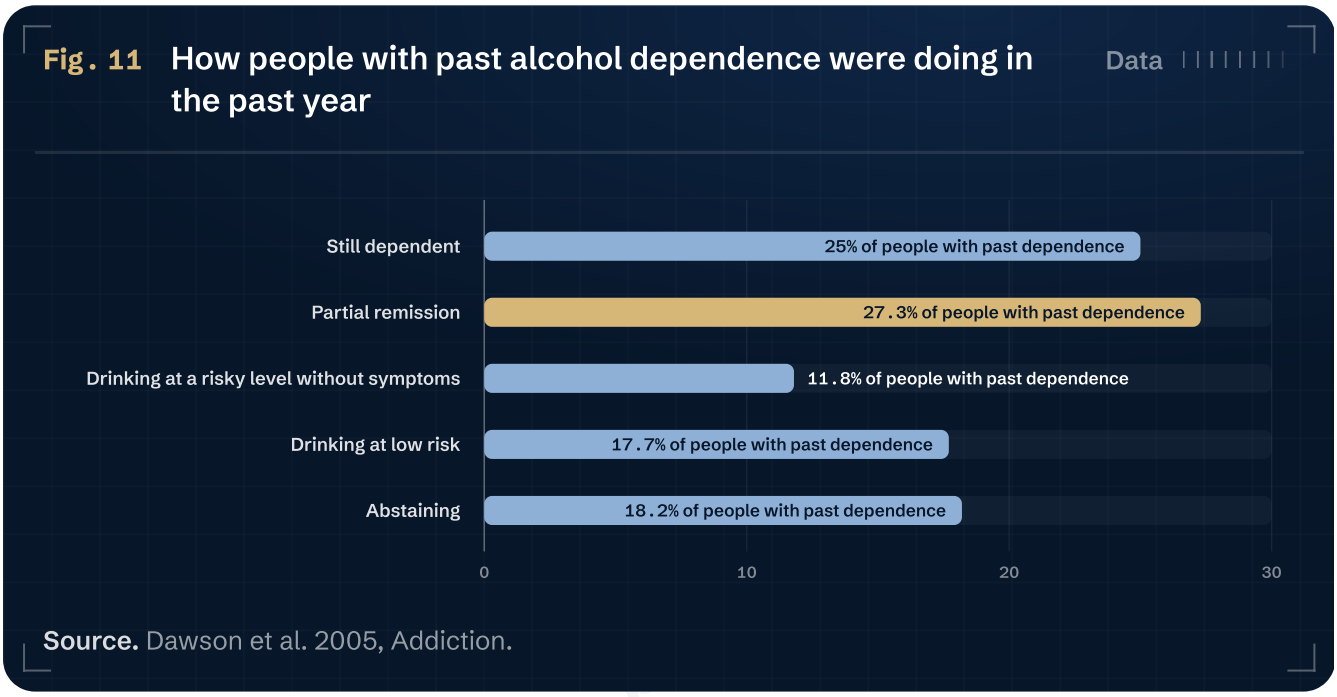


Figure 11. The past-year status of 4,422 US adults who had met the criteria for alcohol dependence before the past year, from a nationally representative survey reported by Dawson and colleagues (2005). These are the study's figures. Low-risk drinking was one of the outcomes, but the study also found that the more severe the dependence had been, the more likely recovery was to involve abstinence.

So the question for an individual is not whether anyone ever learns to drink at low risk, but whether they are likely to be one of the people who do. The people who come to us have usually answered that question already, through their own history. Of our 290+ clients, 98.4% had tried and failed to stop before. Many of those attempts were attempts to cut down. For them, another set of rules is a

repeat of what has already failed. The program's own position is plain: someone who has struggled to stay away from alcohol for more than a month without going back to it should stop completely, and moderation is unlikely to be the right path for them.

A set period off alcohol is a different thing again. De Visser, Robinson and Bond followed 857 British adults who took part in a month-long abstinence challenge. Taking part was linked to drinking less and to more confidence in refusing drinks six months later, even among those who didn't complete the month, and very few drank more afterwards[63]. A month off is useful and rarely harmful. But it has an end date, and for someone whose drinking is driven by stress, sleep and well-learned triggers, the conditions that drove it are waiting on the first day after it ends.

8.2 The thoughts that argue for one drink

Most drinking after a decision to stop is preceded by a thought that gives permission. One won't hurt. I've earned it. It's a special occasion. I'll start again on Monday. I'll get this under control and then drink like a normal person. These thoughts sound reasonable because they are produced in the hot state, when the value of the immediate reward is temporarily inflated and the person can't feel how differently they will see it tomorrow[7]. Arguing with them in the moment rarely works. Trying to push a thought away tends to make it return more often: in Wegner's experiments, people told not to think of a white bear later thought about it more than people who had been allowed to think about it all along[64].

Two approaches have better support. The first is to change one's relationship to the thought rather than its content: to notice it as a thought, not a fact or an instruction, a process acceptance and commitment therapy calls defusion. Hayes and colleagues' review found encouraging evidence for the approach while noting that the research base was still developing[65], and we use it with that caution. The second is to prepare a considered answer to the thought in advance, while calm, so the long-term view is ready when the thought arrives instead of having to be built under pressure. The program does both, for the specific permission thoughts each client has.

In the program — Days 11 to 43

Days 11 to 14	**The case for stopping completely** The client learns why the program asks for complete abstinence and why moderation keeps the learned urge active; takes an inventory of every source of fast, effortless reward in their life, alcohol included; and places their own drinking on a spectrum from habit through pattern and compulsion to addiction, using the evidence in their log.
Day 15	**Habits that travel together** Abstinence begins, from alcohol and from the other fast rewards on the inventory. The client identifies their core habit and the habits that travel with it, such as drinking with smoking, so that one doesn't keep waking the urge for the other.
Days 40 to 43	**Permission thoughts** The client identifies their own permission thoughts and writes a short statement of what is actually true for each; learns to notice a thought without being carried by it; and, for the thought that carries the most weight, conducts a structured written review of the evidence for and against it, prepared in advance for the moment it returns.

Section 09

How do I stop drinking when everyone around me drinks?

Days 38 to 80

In brief

Plan each social situation before you walk into it, spend more of your time with people who support you not drinking, and work on seeing yourself as someone who doesn't drink rather than someone trying

not to. Drinking spreads through friends and family, and so does not drinking.

You stop by deciding in advance how each drinking situation will go, and by changing the balance of who you spend time with. Research on a large community followed for decades found that people were more likely to drink heavily when the people they were connected to drank heavily, and more likely to abstain when those people abstained[66]. Treatment designed to shift a person's social circle toward support for not drinking has improved drinking outcomes for up to two years[67]. The other half of the work is identity. A person who still sees themselves as a drinker who is temporarily not drinking is exposed every time someone offers a glass. A person who sees themselves as someone who doesn't drink has a much simpler decision.

9.1 Drinking spreads through social ties

Rosenquist, Murabito, Fowler and Christakis analyzed 12,067 people in the Framingham Heart Study, assessed several times between 1971 and 2003, along with their friends, relatives, spouses, coworkers and neighbors. Clusters of drinkers and of abstainers were present at every point and extended to three degrees of separation. The clusters were not only explained by drinkers choosing each other: changes in the drinking of a person's network were followed by changes in their own. Relatives and friends had a significant effect; neighbors and coworkers did not[66]. In the full paper, a person was 50% more likely to drink heavily if someone they were directly connected to drank heavily, and 29% more likely to abstain if that person abstained.

phalanxlabs.io

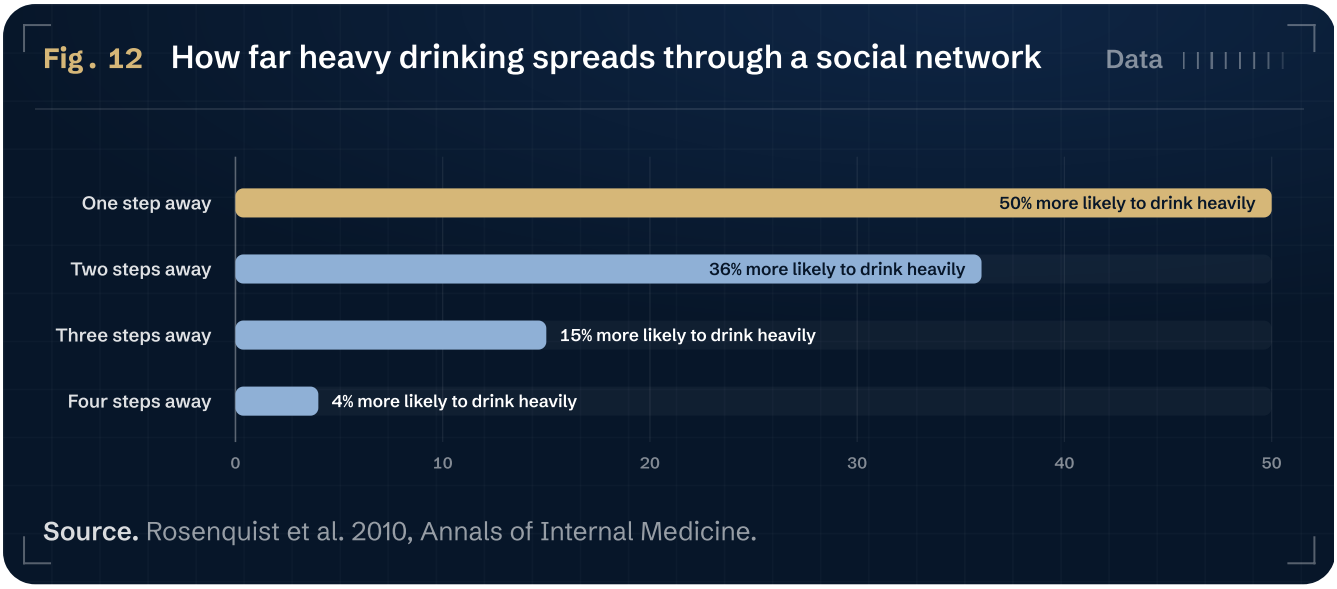


Figure 12. How much more likely a person was to drink heavily when someone in their network drank heavily, by how many social steps separated them, as reported by Rosenquist and colleagues (2010) from the Framingham Heart Study. One step is a direct contact such as a friend or relative; two steps is a friend of a friend. The effect disappeared at four steps. The same study found people 29% more likely to abstain when a direct contact abstained. These are the study's figures, from a community sample, not a prediction for any one person.

The influence can be turned to the person's advantage. In the Network Support Project, Litt and colleagues randomly assigned 210 alcohol-dependent men and women to a treatment designed to shift their social network toward support for abstinence, the same treatment with added incentives, or a case management control. The network support treatment changed participants' networks in that direction and improved drinking outcomes at the first follow-up[68]. At two years, it produced up to 20% more days abstinent than the other conditions, and the network changes were accompanied by gains in confidence and coping that strongly predicted long-term outcomes[67].

Mutual-help groups are one of the most direct ways to change a social network, and the evidence for them is strong. A Cochrane review by Kelly, Humphreys and Ferri covering 27 studies and 10,565 participants found high-certainty evidence that manualized Alcoholics Anonymous and twelve-step facilitation increased rates of continuous abstinence at 12 months compared with other established treatments such as cognitive behavioral therapy, an effect that held at 24 and 36 months[69]. Our program is not a twelve-step program, and many of our clients come to us after deciding that groups are not for them. Those who do attend a group can continue alongside the program.

9.2 From someone trying not to drink to someone who doesn't

Recovery research describes stopping as a change of social identity. Best and colleagues' social identity model of recovery proposes that people move away from groups whose identity is bound up with drinking or drug use and toward groups whose identity supports recovery, and that the new memberships support the new view of oneself[70]. Buckingham, Frings and Albery tested part of this

with members of Alcoholics Anonymous and Narcotics Anonymous and with ex-smokers. Seeing the recovery identity as distinct from the old one, and preferring it, was related to lower relapse, less appetitive behavior and higher self-efficacy, though the authors describe the results as exploratory[71]. Oyserman's identity-based motivation model explains the underlying process: people act in ways that feel consistent with the identities active for them in the moment, and read difficulty according to whether the goal feels like one that people like them pursue[72].

The program's self-image work makes the same point in one line: if you see yourself as someone trying to quit alcohol, you'll always remain trying. Identity work comes late in the program because a new self-description is only believable with evidence behind it, and by Day 61 the client has six or seven weeks of it.

9.3 Social situations, prepared in advance

Drinking situations that matter to someone's work or family life, such as a client dinner, a wedding or a holiday with heavy-drinking relatives, can't always be avoided, and many are rare enough that they can't be practiced often. Mental rehearsal fills that gap. Driskell, Copper and Moran's meta-analysis found that mental practice reliably improves performance, though less than physical practice[73]. Imagining a high-risk event in detail and rehearsing the response means meeting it the first time with a response that has already been used. Planning the situation itself, where to stand, what to hold, how to get home and when to leave, applies the situational strategies described in Section 6 to settings that can't be removed[52].

phalanxlabs.io

In the program — Days 38 to 80

Day 38	**Drinking situations that can't be avoided** The client plans how to position themselves in social situations where alcohol will be present: what they will drink instead, who they will be with, how they will get there and home, and when they will leave.
Days 61 to 66	**Identity** The client works on who they are becoming without alcohol: the stages people pass through when they change, their own change as a story with a before and an after, the person they are building in physical, mental and social terms, what most motivates them, and new goals that draw on it.
Days 67 to 70	**Social context** The client examines how they see themselves in relation to drinking, identifies the outside pressures that still draw them toward it, such as a drinking culture at work, family gatherings built around alcohol or friendships that are mostly about going out, and the beliefs that limit what they think they can do sober, and reframes each one against what has actually happened since Day 15.
Days 71 to 80	**Rehearsal** The client mentally rehearses handling their highest-risk drinking situations successfully, prepares how they will describe themselves in those situations, and practices observing an urge from a distance as something passing through rather than something that defines them.

phalanxlabs.io

Section 10

What happens to your body and brain when you stop drinking?

Days 6 to 25, Brain chemistry restabilization

In brief

In the first days, heavy drinkers may go through withdrawal. In the first weeks, sleep and mood are often rough before they settle. Within a month, studies have measured falls in blood pressure, weight and

insulin resistance. Over months, lost brain volume partly recovers and sleep keeps improving. Some changes take a year or more.

Your body starts to recover within days, and the recovery continues for months. Heavy daily drinkers can have withdrawal in the first few days, which is why they need a doctor (Section 2). Over the first weeks, the low mood and anxiety of early sobriety usually ease[36], although sleep often gets worse before it gets better[41]. In a study of moderate to heavy drinkers who stopped for a month, insulin resistance, blood pressure, weight and two cancer-related growth factors all fell[74]. In the brain, the glutamate imbalance of withdrawal settles within a few weeks[12], and grey matter begins to recover within the first two weeks[75], with further changes over the months that follow[76].

10.1 The first month

Mehta and colleagues recruited healthy people who drank more than about 64 grams of alcohol a week for men, or 48 grams for women, with known liver disease and alcohol dependence excluded. Ninety-four chose to stop drinking for a month and 47 chose to carry on. The abstinence group had been drinking an average of about 258 grams a week, roughly 32 standard UK units. After one month, the abstinence group showed significant falls in insulin resistance, blood pressure, weight and two growth factors linked to cancer, VEGF and EGF, none explained by changes in diet, exercise or smoking. The group who kept drinking showed no significant changes[74]. The participants chose their own group rather than being randomized, so the study shows an association. It is also one of the clearest measurements of what a month without alcohol does in people who were not dependent.

phalanxlabs.io

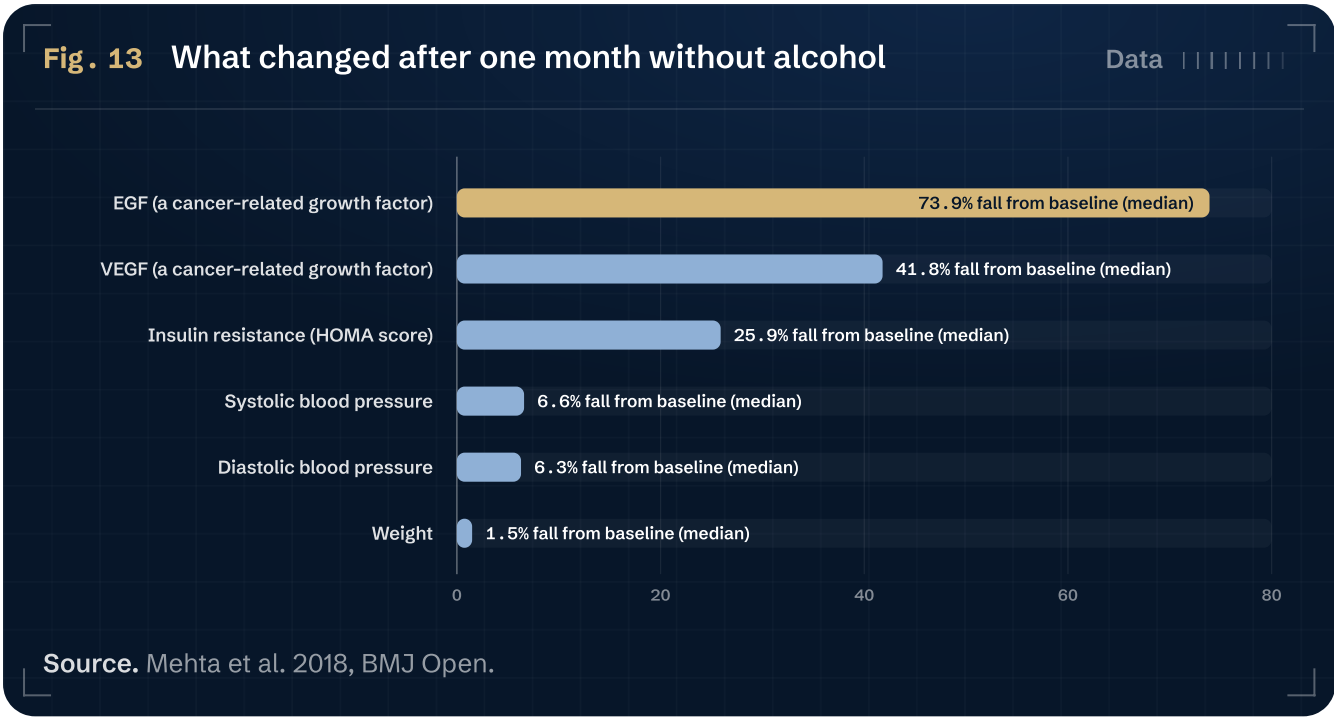


Figure 13. Median change from baseline after one month of abstinence in 94 moderate to heavy drinkers without alcohol dependence, as reported by Mehta and colleagues (2018). All changes were significant, and none was associated with changes in diet, exercise or smoking; the 47 people who kept drinking showed no significant change. These are the study's figures, shown as the size of the fall. Individual changes varied widely.

10.2 The brain

Heavy drinking is associated with smaller volumes of grey and white matter, and some of that loss recovers with abstinence. Van Eijk and colleagues scanned 49 alcohol-dependent patients within the first 24 hours of detoxification and again after two weeks of supervised abstinence, and compared them with 55 healthy controls. On the first day, the patients had less grey matter, with the largest differences in the cingulate gyrus, frontal regions, cerebellum and insula. After two weeks, a significant though partial recovery of grey matter had already occurred in several regions, at different rates in different parts of the brain[75]. Pfefferbaum and colleagues followed 58 men with alcoholism with scans at about 12 and 32 days of sobriety and again two to twelve months later. Improvements in cortical grey matter and in the fluid spaces of the brain appeared early in abstinence, further changes appeared later in those who stayed abstinent, and white matter volume fell in those who went back to drinking[76].

The reward system recovers more slowly, and the evidence on its timing is thinner. The blunted dopamine response described in Section 3 was measured in people who had recently stopped[23], and the persistence of altered emotional processing into protracted abstinence is described mainly from animal work[36]. We tell clients what is known: recovery begins quickly, varies between people and between brain regions, and continues for months. We don't promise that any marker returns to normal by a given day, because the research doesn't support a date.

Table 3. What the studies cited in this paper found at different points after the last drink. Each finding comes from a specific group of people, described in the text, and timings vary between individuals. The table is a summary for orientation, not a forecast for any one person.

When	What the research found	Source
First hours to days	Withdrawal can begin within 6 to 24 hours in people who drank heavily; it needs a doctor	Mirijello et al. 2015
First two weeks	Partial recovery of grey matter volume has begun in several regions	van Eijk et al. 2013
First weeks	Sleep is short and broken early on; the glutamate imbalance of withdrawal normalizes	Drummond et al. 1998; Hermann et al. 2012
Three to six weeks	The low mood and anxiety of early withdrawal usually ease	Heilig et al. 2010
One month	In non-dependent drinkers: insulin resistance, blood pressure, weight and growth factors fall	Mehta et al. 2018
Around 60 days	Craving set off by triggers was higher than at 7 days in one study of inpatients	Li et al. 2015
Months to a year and more	Further brain volume changes with continued abstinence; sleep keeps improving slowly	Pfefferbaum et al. 1995; Drummond et al. 1998

10.3 Exercise, mood and fitness

Exercise is part of the first phase of the program, and the evidence for it needs stating carefully. Hallgren and colleagues' meta-analysis of 21 studies with 1,204 people with alcohol use disorders found that exercise did not reduce alcohol consumption on its own, but it did significantly reduce depressive symptoms and improve physical fitness[77]. Lynch and colleagues' review proposes that exercise acts on the dopamine and glutamate signalling that drug use alters, with effects that depend on the stage of addiction and the type of exercise[78]. We don't use exercise as a treatment for drinking. We use it because it lifts the low mood and restlessness of early sobriety, improves sleep and gives the evening a structure, and because visible progress in training is direct evidence to the client that they can change something about themselves[79].

In the program · Phase 1, Brain chemistry restabilization **Days 6 to 25**

Days 6 and 9	**Mood, fitness and the shape of the day** On Day 6, progressive resistance training and cardiovascular exercise become part of the daily practices, built up from wherever the client is. On Day 9, the client builds their day around fixed anchors, with a daily practice placed every few hours, so that the time drinking used to fill has something else in it.
Day 25	**Expecting recovery** The client reviews published imaging evidence that grey matter in control regions recovers with sustained abstinence, to replace the belief that the damage is permanent with an accurate, cautious expectation that function recovers over months.

Inge, a senior operator in Belgium who came to us for alcohol, described her first two months in one line: “I've been sober for 56 days today.” She has now been sober for over six months. We cite her not as evidence that the program works, which a single person can't provide, but because she describes the kind of craving the second phase aims for, a passing thought rather than a strong urge: “And even then, it wasn't a strong craving, just a thought about it. And I now know why it's important to not give in to the thoughts.”

Section 11

What should I do if I drink again?

Day 15 onward

In brief

Treat it as something that happened for a reason, not as proof that you can't do this. Don't carry on the next day, write down what led up to it, and fix the part of your plan that missed it. If it turns into several days of heavy drinking, talk to a doctor before you stop again.

A drink after stopping is not the end of the attempt unless you decide it is. What turns one drink into a full relapse is usually the story people tell themselves about it: that it proves they're weak, that they've always been like this, that the whole attempt has failed. People who explain a drink that way are more likely to keep drinking[80]. The better response is practical. Go back to your plan the next

day, work out what led to the drink, and change the part of the plan that didn't cover it. One extra point applies to alcohol: if a relapse becomes several days of heavy drinking, stopping again can bring withdrawal back, so talk to a doctor first[13].

The research on this is known as the abstinence violation effect. Curry, Marlatt and Gordon studied people who had lapsed after stopping smoking and found that those who went on to full relapse put their lapse down more to internal, stable and global causes, such as a lasting personal weakness, than those who lapsed and recovered[80]. Witkiewitz and Marlatt's reformulation of relapse prevention for alcohol and drug problems describes relapse as a dynamic process in which stress, craving, confidence, coping and mood interact, and in which small changes can produce sudden shifts in behavior[81]. In that model there is no single cause of relapse to remove and no point at which a person is permanently safe. What protects them is a set of coping responses that stay available as conditions change. Hendershot and colleagues' review of a decade of relapse prevention research describes the approach as part of most cognitive-behavioral treatments for addictive behaviors[82].

phalanxlabs.io

Fig. 14 Two ways to respond to a drink after stopping — Comparison

	– Abstinence violation response	✓ Planned response
What the drink means	Proof that I can't do this	An event with a specific cause
How it is explained	Internal, stable and global: who I am	Situational and specific: what happened, when and where
The next day	The attempt is over, so drinking continues	The daily practices and the plan continue as usual
What is learned	Nothing, apart from more guilt	Which trigger was missed and which response failed
Safety	Several days of heavy drinking, then another sudden stop	A doctor is consulted before stopping again after heavy drinking

Figure 14. The abstinence violation response, in which a drink is read as proof of personal failure, compared with the planned response the program prepares for. The left column follows the pattern of explanation Curry, Marlatt and Gordon (1987) found among people who relapsed. The right column describes the program's approach, which treats the drink as an event with a cause that can be found and corrected.

Confidence in one's ability to stay stopped matters for the long run. In Adamson, Sellman and Frampton's systematic review of what predicts the outcome of alcohol treatment, alcohol-related self-efficacy was among the most consistent predictors, along with dependence severity, psychiatric symptoms, motivation and the treatment goal[83]. Lower self-efficacy also predicted relapse over 16 years in Moos and Moos's study[2]. A drink read as proof of weakness lowers that confidence. A drink that is analyzed and answered with a better plan protects it.

The combination of awareness training and prepared coping responses that runs through the program has direct support in a randomized trial. Bowen and colleagues assigned 286 people who had completed initial treatment for substance use disorders, including alcohol, to mindfulness-based relapse prevention, standard relapse prevention or treatment as usual. Both relapse prevention approaches reduced the risk of relapse to substance use and heavy drinking compared with treatment as usual at six months, and at twelve months the mindfulness-based approach was

associated with fewer days of substance use and less heavy drinking than either[84]. Our program is not either protocol, and the trial doesn't test it. But the elements the trial found useful are the ones a client carries out of the 90 days.

In the program — Day 15 onward

Day 15	**Planning for a drink before it happens** On the day drinking stops, the program defines the difference between a single drink and a relapse, months before the definition is likely to be needed, so that a drink is treated as an event to record and analyze rather than an ending.
Days 91 to 100	**Relapse prevention** The first days of aftercare consolidate the change: the client writes to themselves about the past with forgiveness, including the things said and done while drinking, reinforces the identity built in the third phase and keeps a record of what they have gained.

If you drink again

Go back to your plan the next day. Write down what happened in the hours before: where you were, who with, how you felt and what you told yourself. Find the trigger your plan missed and add a response for it. If you are working with us, tell your specialist the same day. If one drink became several days of heavy drinking, talk to a doctor before you stop again.

phalanxlabs.io

Section 12

What does the Phalanx program target for someone stopping drinking, and when?

Days 81 to 90, Identity rewiring

In brief

Ninety days in three phases. The first month rebuilds sleep, stress, fitness and the shape of the day, and drinking stops on Day 15. The

second month maps every trigger and builds a response for each. The third works on identity and the people and places that still pull toward a drink. Six months of aftercare follow.

The Behavior Control System is the same 90-day program for every habit, applied to the loop beneath each person's drinking. Days 1 to 30 rebuild the ground drinking has been running on: sleep, the body clock, daylight, exercise, stress and a structured day. The client stops drinking on Day 15, after two weeks of that work, or on the date their doctor sets. Days 31 to 60 work on the moment a drink actually happens: every trigger is mapped, the ones that can be removed are removed, and each one left gets a prepared, rehearsed response, including the thoughts that argue for one drink and the stress that most often starts the evening. Days 61 to 90 work on identity and on the drinking culture, events and relationships that still pull the person back. Six months of aftercare follow. It asks for about thirty minutes of focused work a day: a short lesson each morning, a brief log each night, and a set of daily practices, which the program calls north stars, built into the routine the person already has.

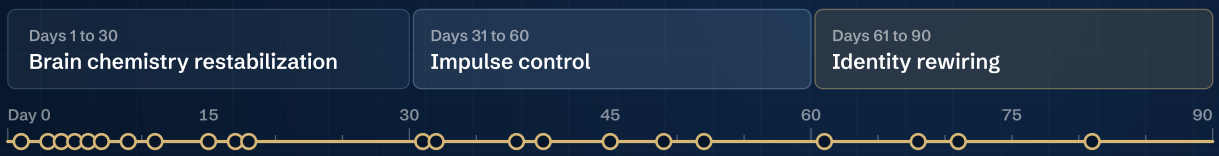
12.1 Before Day 1

Every program starts with a Root-Cause Diagnostic: an application describing the habits involved and what has already been tried, followed by a thirty-minute, one-on-one conversation with our team. For alcohol it has two jobs. The first is safety: it asks how much and how often the person drinks and about any previous withdrawal, and anyone who drinks heavily every day is told to see a doctor before they begin (Section 2). The second is to identify the loop beneath the drinking: what it does for the person, the earliest recurring trigger, the conditions that make it likely, why earlier attempts broke down and what is missing for change to hold. Two people who both drink every evening rarely have the same loop. One drinks to end a working day that never otherwise ends; another drinks only with a particular group; a third drinks alone after the household is asleep. The structure of the diagnostic parallels the three domains the Addictions Neuroclinical Assessment proposes for exactly this problem: the urge set off by triggers, negative emotion and executive function[85]. It is not a clinical assessment and takes no medical measurements.

A preparatory phase, Phase 0, follows before Day 1 and does not count toward the 90 days. It separates the decision to stop from the start. The client sets out in concrete terms the life they are building and what drinking has cost it, makes a commitment to someone who matters to them, chooses a start date that marks a real boundary in their life, and proves to themselves that they can hold one full day of control. For a heavy daily drinker, that day, like the stop on Day 15, follows the doctor's advice. The choice of start date rests on the fresh start effect: temporal landmarks such as the start of a week, a month or a meaningful date were followed by increases in aspirational behavior[86]. The day of control rests on Bandura's finding that direct experience of succeeding at a behavior is the most dependable source of belief that one can do it[79]. A commitment that is recorded and made to another person holds better than a private one[87].

Fig. 15 The 90 days for someone stopping drinking

Timeline



Homeostasis

Day 1 Nightly log and baseline begin
Self-monitoring

Day 3 Fixed sleep and wake times
Body clock and sleep

Day 4 Morning daylight and a daily walk
Body clock

Day 5 Evening light down; phone out of the bedroom
Sleep

Day 6 Progressive training and cardio
Mood and stress

Day 7 Daily meditation
Attention to internal states

Day 9 A day built on fixed anchors
Habit control

Day 11 The case for stopping completely
Reward system

Abstinence with systems

Day 31 Full trigger map
Trigger learning

Day 32 Alcohol and easy access removed
Context and access

Day 38 Social situations planned
Situational control

Day 40 Permission thoughts
Cognitive control

Day 45 Stress and drinking again
Stress system

Day 49 Urge observation
The felt urge

Day 52 A response for every trigger
Implementation intentions

Burn down the old subconscious and unconscious thinking models

Day 61 Identity work begins
Self-concept

Day 68 Drinking culture and outside pressures
Social context

Day 71 Rehearsing high-risk events
Mental rehearsal

Day 81 Replacing what drinking did
Habit control

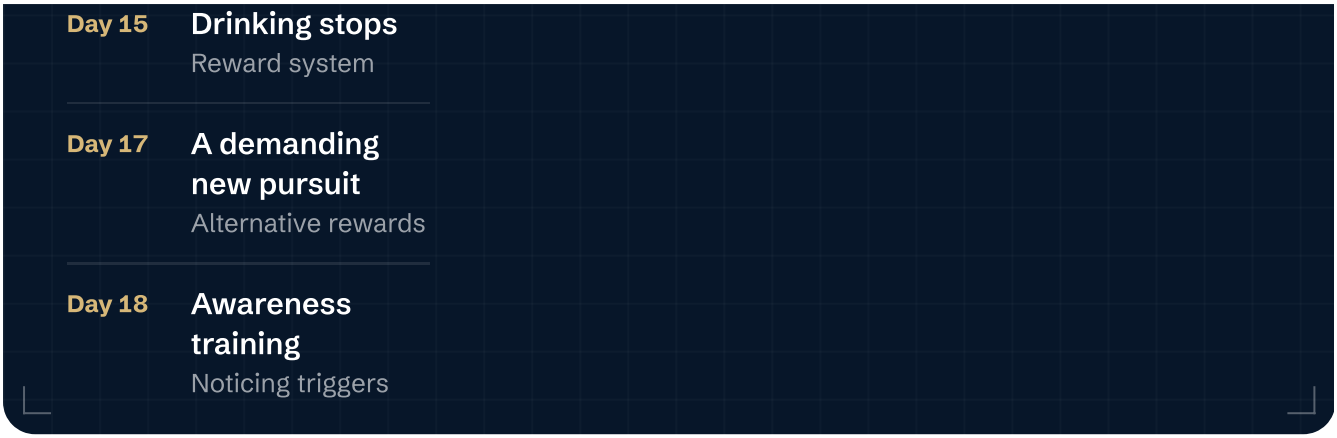


Figure 15. The three phases of the program, with the days on which targets that matter most for drinking are introduced. Phase 0 comes before Day 1. Drinking stops on Day 15, after two weeks of groundwork, or on the date a doctor sets for a heavy daily drinker. Six months of aftercare follow Day 90. The exercises themselves are not published.

phalanxlabs.io

Table 4. The day map for someone stopping drinking: what each stretch of the program targets, and the evidence behind the choice. Days refer to the program calendar. The table states the target only; the exercises themselves are not published.

Days	Target	Why it matters for stopping drinking
Before Day 1	Root-Cause Diagnostic: the loop beneath the drinking, and a medical check for heavy daily drinkers	Withdrawal can be dangerous[10]; the loop is read across trigger-set urges, negative emotion and executive function[85]
Before Day 1	Commitment, a meaningful start date, one day of control (on medical advice for heavy daily drinkers)	Fresh starts and direct experience of success raise follow-through[79, 86]
1 to 2	How the loop works; the nightly log	Recorded monitoring of progress improves goal attainment[87]
3 to 5	Sleep and wake times, daylight, evening light, phone out of the bedroom	Insomnia is linked to drinking again after treatment[42]
6	Progressive resistance training and cardio	Lifts mood and fitness in alcohol use disorders, though not drinking on its own[77]
7	Daily meditation	Groundwork for observing urges; mindfulness-based relapse prevention reduced heavy drinking[84]
8	Nutrition, at a general level	Heavy drinking can cause thiamine deficiency, which is a matter for a doctor[15]
9	A day built on fixed anchors	Stable routines fill the hours in which habits run[24]
10 to 14	The loop, the case for stopping completely, the reward inventory, self-assessment	Alcohol lowers inhibitory control and primes the next drink[6]
15	Drinking stops, on a doctor's plan where needed; habits that travel together	Each drink reactivates learned links to its triggers[51]

Days	Target	Why it matters for stopping drinking
16 to 23	Heat and cold exposure, a demanding new pursuit, awareness training, breathing, practice days	Earned rewards compete with the drink; vigilance controls strong habits[28, 54]
24 to 30	Perspective, evidence of recovery, how triggers are learned	Grey matter begins to recover within two weeks of stopping[75]
31 to 32	Full trigger map; alcohol and easy access removed	Changing the situation beats resisting an urge already under way[52]
33 to 39	Why early sobriety is hard, the response sequence, social situations, breaking the routine	Withdrawal-related low mood eases over weeks; relapse risk lasts longer[36]
40 to 43	Permission thoughts	Pushing a thought away makes it return[64]
44 to 51	Emotion, stress, breathing, relaxation, urge observation, an earned reward, the future self	Stress-provoked craving predicts a shorter time to relapse[37]
52 to 60	A response for every trigger; daily practice and a craving record	Implementation intentions reduced drinking in a randomized trial[56]
61 to 70	Identity, drinking culture, outside pressures, limiting beliefs	Drinking and abstaining both spread through social ties[66]
71 to 80	Rehearsal of high-risk drinking situations	Mental practice improves performance[73]
81 to 90	Replacing each function drinking served; review of the 90 days	A new routine takes weeks to months to become automatic[88]
After 90	Self-forgiveness, identity reinforcement, six months of aftercare	How a drink is explained predicts whether it becomes a relapse[80]

12.2 Why the order matters for alcohol

The order follows from the research in Sections 2 to 11. Drinking stops after sleep and stress have had two weeks of work, because poor sleep and stress-provoked craving are among the strongest known predictors of drinking again[37, 42], and because the hours the evening drink used to fill need something already in them on the first night without it. The trigger work comes in the second month, when the low mood of early withdrawal is easing but craving set off by triggers may be at its strongest[36, 57]. Identity and social context come last, because they change slowest and because a new self-description needs weeks of evidence behind it to be believed.

In the program · Phase 3, Identity rewiring **Days 81 to 90**

Replacing what drinking did

The client confirms a replacement for each function alcohol used to serve: the way they end a working day, let go of pressure, reward themselves or spend time with friends. They review the full 90 days against their log and record what they have gained.

Replacement is the last step because a habit that served a function leaves a gap when it stops, and a gap left empty tends to be filled by whatever is most available. A replacement placed at the same time and in the same setting as the old drink can take over its context[24]. Lally and colleagues found that the automaticity of a new daily behavior built gradually, reaching 95% of its plateau after a median of 66 days with wide variation between people, and that missing a single occasion did not materially affect the process[88]. By Day 90 the replacement has been in place long enough to begin running on its own.

12.3 In our clients' words

Two of our clients who came to us for alcohol have described their experience on the record. Inge, a senior operator in Belgium, had tried to stay sober before and held on by willpower each time. “I also understand now why my previous attempts at being sober didn't work,” she said, and of how it felt this time: “This time I feel different about sobriety. It doesn't feel like a punishment. It feels like a gift to myself and to my brain.” She has now been sober for over six months. Bjorn, a senior operator in the Netherlands, came to us having lost his motivation and joy in life. When he recorded his interview he said: “I've been sober for 71 days now and this time it was easier than previous attempts because of all the information and training exercises you get along with the program.” Their words are shared with consent. They are two people's experiences, not evidence of what will happen for anyone else.

What this paper does not publish

This paper states what each part of the program targets for someone stopping drinking, on which days and why, with the evidence for each choice. It does not publish the exercises, their steps, scripts, worksheets or wording. A reader can evaluate the reasoning from this paper, but cannot reproduce the program from it.

Section 13

What do our results show, and what can't this paper tell you?

In brief

We report a 97% success rate, meaning clients who ran the program and stayed off the habit through the program and six months of aftercare. It comes from our own client follow-up, not from a controlled trial, and the research in this paper supports each part of the method without testing the whole.

Phalanx Labs has worked with 290+ clients across 12 countries over 7 years. Of those clients, 98.4% had already tried and failed to stop before they came to us. Our success rate is 97%, defined as the share of clients who ran the program and stayed off the habit through the program and the six months of aftercare that follow it. That rate covers all of our clients and all of the habits they came with. We don't publish a separate rate for alcohol. Because the definition matters as much as the number, we set out below what each figure counts, how it is known and what it does not establish.

Table 5. The figures we report, what each one counts and how it is known. All figures are drawn from our own client records and cover every habit we work with, not alcohol alone.

Figure	What it counts	How it is known
290+ clients	People who have worked with Phalanx Labs over 7 years	Client records
12 countries	Countries in which those clients lived while working with us	Client records
98.4%	Share of clients who had already tried and failed to stop before they came to us	What clients reported about their history in their application and the diagnostic
97% success rate	Clients who ran the program and stayed off the habit through the program plus 6 months of aftercare	Client follow-up across the program and the six months of aftercare

13.1 How outcomes are tracked

Throughout the program, the client keeps a nightly log and completes a weekly progress update. These are written records of what happened, what was difficult and what was handled; they are not scored instruments. At the end of each of the first two phases the client has a progress call with our team, the first around Day 28 and the second after Day 60. During aftercare we follow up with clients on whether they have stayed off the habit. A client counts as a success when they have run the program and stayed off the habit through the program and the full six months of aftercare.

13.2 What our figures do not show

- Self-report. Whether a client has stayed off alcohol is known from what the client tells us, not from breath, blood or urine testing.
- Selection. Clients choose to apply, are accepted after a diagnostic in which we decline people we can't help, and are counted only if they ran the program. People who decide to work with us may differ in motivation or circumstance from people who don't.
- No comparison group. We can't say how many of the same people would have stopped through another route, such as medication, a mutual-help group or treatment, over the same period.
- Time window. The success rate covers the program and six months of aftercare. It does not describe what happens after that, and the relapse research in Section 7 shows that risk continues beyond it.

- All habits together. The rate is across every habit our clients bring. We don't report a rate for alcohol alone, and a single rate does not predict the outcome for any one person.

13.3 What the research in this paper does not show

The studies cited here have their own limits, and readers should weigh them. Much of the alcohol research was done with people in treatment for alcohol dependence, often in inpatient settings and often mostly or only men, such as the craving study by Li and colleagues and the sleep and imaging studies by Drummond and by Pfefferbaum and their colleagues[41, 57, 76]. Many of our clients drink heavily without ever having been in treatment, and the findings may apply to them with different strength. Several imaging studies were small, and some findings come from animals or from people who smoked rather than drank, such as the research on urge surfing and on how a lapse is explained[60, 80]; we use those for the mechanism they describe and say so where we cite them. Some of what the program does has weaker direct evidence for drinking than its reputation suggests: exercise, for example, improved mood and fitness in people with alcohol use disorders but did not reduce their drinking on its own[77].

Other approaches have stronger trial evidence than ours. Acamprosate and naltrexone have been tested in many randomized trials[16], and twelve-step facilitation has high-certainty evidence for continuous abstinence[69]. Our method has not been tested in a randomized or controlled trial, its outcomes have not been assessed independently, and it has not been published in a peer-reviewed journal. The research supports the reasoning behind each element. It does not show that the combination works, or how much each element contributes. The figures in Table 5 are our evidence for the combination, with the limits stated above. A controlled evaluation would need independent outcome assessment, a comparison condition and follow-up beyond six months. We would welcome one.

phalanxlabs.io

The definition, stated once more

97% success rate: clients who ran the program and stayed off the habit through the program plus 6 months of aftercare. Tracked through client follow-up, across all habits. Not a clinical trial, with no randomization and no control group.

Section 14

When should you see a doctor, and what is this program not?

In brief

See a doctor before you stop if you drink heavily every day, have had withdrawal before, or take sleeping or anxiety medication. Call emergency services for a seizure, confusion or hallucinations during withdrawal. The program is not therapy, clinical care or medical treatment, and it works alongside a doctor, never instead of one.

See a doctor before you stop drinking if you drink heavily every day, if you have had withdrawal symptoms such as shaking, sweating or anxiety when you go without, if you have ever had a withdrawal seizure or delirium tremens, if you take benzodiazepines or other sleeping or anxiety medication, or if you have a heart, liver or other medical condition. A doctor can assess the risk, plan the stop, treat withdrawal if it comes, and discuss medication that helps people stay stopped[10, 16]. If you or someone with you has a seizure, becomes confused or starts seeing things during withdrawal, call emergency services.

Medical safety note

Alcohol withdrawal can be dangerous. Anyone who drinks heavily every day talks to a doctor before stopping. The same applies to anyone taking benzodiazepines or other prescription drugs: stopping suddenly can be dangerous, they should not be combined with alcohol, and any change should be made with their doctor.

Phalanx Labs is a behavior and performance engineering firm. The Behavior Control System is a structured method for changing behavior. It is not therapy, clinical care or medical treatment, and it does not diagnose or treat any medical or psychiatric condition. Our team does not act as the client's doctor or therapist, and the program does not replace either. Where the diagnostic suggests withdrawal may be a risk, we tell the person to see a doctor before they begin, and the stop date follows medical advice rather than the other way round.

Some of the program's physical practices carry ordinary risks of their own. Cold and heat exposure and starting a training program are not suitable for everyone, and people with heart conditions, high blood pressure or other medical conditions should check with their doctor first. The program gives general information about supplements and tells clients to consult a doctor before taking any; it does not prescribe or recommend medication or supplements.

The program is also not designed for every situation. It is not a crisis service. It is not suitable as the only support for someone with a serious mental health condition, someone at risk of harming themselves or others, or someone whose drinking needs medically supervised treatment. The

diagnostic exists partly to identify these situations, and when we can't help, we say so and suggest the person seek appropriate care.

phalanxlabs.io

References

Numbered in the order the paper first cites them. Each links to the study itself.

1 **Kirshenbaum AP, Olsen DM, Bickel WK** (2009). A quantitative review of the ubiquitous relapse curve. Journal of Substance Abuse Treatment, 36(1), 8-17.

https://doi.org/10.1016/j.jsat.2008.04.001 Cited in Section 1, Section 7

2 **Moos RH, Moos BS** (2006). Rates and predictors of relapse after natural and treated remission from alcohol use disorders. Addiction, 101(2), 212-222.

https://doi.org/10.1111/j.1360-0443.2006.01310.x Cited in Section 1, Section 7, Section 11

3 **Arnsten AFT** (2009). Stress signalling pathways that impair prefrontal cortex structure and function. Nature Reviews Neuroscience, 10(6), 410-422.

https://doi.org/10.1038/nrn2648 Cited in Section 1, Section 4

4 **Schwabe L, Wolf OT** (2009). Stress Prompts Habit Behavior in Humans. The Journal of Neuroscience, 29(22), 7191-7198.

https://doi.org/10.1523/JNEUROSCI.0979-09.2009 Cited in Section 1, Section 3, Section 4

5 **Diamond A** (2013). Executive Functions. Annual Review of Psychology, 64(1), 135-168.

https://doi.org/10.1146/annurev-psych-113011-143750 Cited in Section 1

6 **Field M, Wiers RW, Christiansen P, Fillmore MT, Verster JC** (2010). Acute Alcohol Effects on Inhibitory Control and Implicit Cognition: Implications for Loss of Control Over Drinking. Alcoholism: Clinical and Experimental Research, 34(8), 1346-1352.

https://doi.org/10.1111/j.1530-0277.2010.01218.x

Cited in Section 1, Section 3, Section 8, Section 12

7 **Loewenstein G** (2005). Hot-cold empathy gaps and medical decision making. Health Psychology, 24(4 Suppl), S49-S56.

https://doi.org/10.1037/0278-6133.24.4.S49 Cited in Section 1, Section 8

8 **Koob GF, Volkow ND** (2016). Neurobiology of addiction: a neurocircuitry analysis. The Lancet Psychiatry, 3(8), 760-773.

https://doi.org/10.1016/S2215-0366(16)00104-8 Cited in Section 1, Section 3

9 **Saitz R** (1998). Introduction to alcohol withdrawal. Alcohol Health and Research World, 22(1), 5-12.

PubMed Cited in Section 2

10 **Mirijello A, D'Angelo C, Ferrulli A, Vassallo G, Antonelli M, Caputo F, et al.** (2015). Identification and Management of Alcohol Withdrawal Syndrome. Drugs, 75(4), 353-365.

https://doi.org/10.1007/s40265-015-0358-1 Cited in Section 2, Section 12, Section 14

11 **Davies M** (2003). The role of GABAA receptors in mediating the effects of alcohol in the central nervous system. Journal of Psychiatry & Neuroscience, 28(4), 263-274.

PubMed Cited in Section 2

12 **Hermann D, Weber-Fahr W, Sartorius A, Hoerst M, Frischknecht U, Tunc-Skarka N, et al.** (2012). Translational Magnetic Resonance Spectroscopy Reveals Excessive Central Glutamate Levels During Alcohol Withdrawal in Humans and Rats. Biological Psychiatry, 71(11), 1015-1021.

https://doi.org/10.1016/j.biopsych.2011.07.034 Cited in Section 2, Section 7, Section 10

13 **Becker HC** (1998). Kindling in alcohol withdrawal. Alcohol Health and Research World, 22(1), 25-33.

PubMed Cited in Section 2, Section 11

14 **Mayo-Smith MF** (1997). Pharmacological Management of Alcohol Withdrawal: A Meta-analysis and Evidence-Based Practice Guideline. JAMA, 278(2), 144-151.
https://doi.org/10.1001/jama.1997.03550020076042 Cited in Section 2

15 **Sechi G, Serra A** (2007). Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. The Lancet Neurology, 6(5), 442-455.
https://doi.org/10.1016/S1474-4422(07)70104-7 Cited in Section 2, Section 12

16 **Jonas DE, Amick HR, Feltner C, Bobashev G, Thomas K, Wines R, et al.** (2014). Pharmacotherapy for Adults With Alcohol Use Disorders in Outpatient Settings: A Systematic Review and Meta-analysis. JAMA, 311(18), 1889-1900.
https://doi.org/10.1001/jama.2014.3628 Cited in Section 2, Section 13, Section 14

17 **Mitchell JM, O'Neil JP, Janabi M, Marks SM, Jagust WJ, Fields HL** (2012). Alcohol Consumption Induces Endogenous Opioid Release in the Human Orbitofrontal Cortex and Nucleus Accumbens. Science Translational Medicine, 4(116), 116ra6.
https://doi.org/10.1126/scitranslmed.3002902 Cited in Section 2, Section 3

18 **Schuckit MA** (2009). Alcohol-use disorders. The Lancet, 373(9662), 492-501.
https://doi.org/10.1016/S0140-6736(09)60009-X Cited in Section 2

19 **Everitt BJ, Robbins TW** (2016). Drug Addiction: Updating Actions to Habits to Compulsions Ten Years On. Annual Review of Psychology, 67(1), 23-50.
https://doi.org/10.1146/annurev-psych-122414-033457 Cited in Section 3

20 **Boileau I, Assaad JM, Pihl RO, Benkelfat C, Leyton M, Diksic M, et al.** (2003). Alcohol promotes dopamine release in the human nucleus accumbens. Synapse, 49(4), 226-231.
https://doi.org/10.1002/syn.10226 Cited in Section 3

21 **Schultz W, Dayan P, Montague PR** (1997). A Neural Substrate of Prediction and Reward. Science, 275(5306), 1593-1599.

https://doi.org/10.1126/science.275.5306.1593 Cited in Section 3, Section 6

22 **Berridge KC, Robinson TE** (2016). Liking, wanting, and the incentive-sensitization theory of addiction. American Psychologist, 71(8), 670-679.

https://doi.org/10.1037/amp0000059 Cited in Section 3

23 **Volkow ND, Wang GJ, Telang F, Fowler JS, Logan J, Jayne M, et al.** (2007). Profound Decreases in Dopamine Release in Striatum in Detoxified Alcoholics: Possible Orbitofrontal Involvement. The Journal of Neuroscience, 27(46), 12700-12706.

https://doi.org/10.1523/JNEUROSCI.3371-07.2007 Cited in Section 3, Section 10

24 **Wood W, Rünger D** (2016). Psychology of Habit. Annual Review of Psychology, 67(1), 289-314.

https://doi.org/10.1146/annurev-psych-122414-033417 Cited in Section 3, Section 12

25 **Sjoerds Z, de Wit S, van den Brink W, Robbins TW, Beekman ATF, Penninx BWJH, et al.** (2013). Behavioral and neuroimaging evidence for overreliance on habit learning in alcohol-dependent patients. Translational Psychiatry, 3(12), e337.

https://doi.org/10.1038/tp.2013.107 Cited in Section 3

26 **Petry NM** (2001). Delay discounting of money and alcohol in actively using alcoholics, currently abstinent alcoholics, and controls. Psychopharmacology, 154(3), 243-250.

https://doi.org/10.1007/s002130000638 Cited in Section 3

27 **MacKillop J, Amlung MT, Few LR, Ray LA, Sweet LH, Munafò MR** (2011). Delayed reward discounting and addictive behavior: a meta-analysis. Psychopharmacology, 216(3), 305-321.

https://doi.org/10.1007/s00213-011-2229-0 Cited in Section 3

28 **Bickel WK, Johnson MW, Koffarnus MN, MacKillop J, Murphy JG** (2014). The Behavioral Economics of Substance Use Disorders: Reinforcement Pathologies and Their Repair. Annual Review of Clinical Psychology, 10(1), 641-677.

https://doi.org/10.1146/annurev-clinpsy-032813-153724 Cited in Section 3, Section 12

29 **Solinas M, Thiriet N, Chauvet C, Jaber M** (2010). Prevention and treatment of drug addiction by environmental enrichment. Progress in Neurobiology, 92(4), 572-592.

https://doi.org/10.1016/j.pneurobio.2010.08.002 Cited in Section 3

30 **Sher KJ, Levenson RW** (1982). Risk for alcoholism and individual differences in the stress-response-dampening effect of alcohol. Journal of Abnormal Psychology, 91(5), 350-367.

https://doi.org/10.1037/0021-843X.91.5.350 Cited in Section 4

31 **Cooper ML, Frone MR, Russell M, Mudar P** (1995). Drinking to regulate positive and negative emotions: A motivational model of alcohol use. Journal of Personality and Social Psychology, 69(5), 990-1005.

https://doi.org/10.1037/0022-3514.69.5.990 Cited in Section 4

32 **Kushner MG, Abrams K, Borchardt C** (2000). The relationship between anxiety disorders and alcohol use disorders: A review of major perspectives and findings. Clinical Psychology Review, 20(2), 149-171.

https://doi.org/10.1016/S0272-7358(99)00027-6 Cited in Section 4

33 **Koob GF, Le Moal M** (2001). Drug Addiction, Dysregulation of Reward, and Allostasis. Neuropsychopharmacology, 24(2), 97-129.

https://doi.org/10.1016/S0893-133X(00)00195-0 Cited in Section 4

34 **Koob GF** (2008). A Role for Brain Stress Systems in Addiction. Neuron, 59(1), 11-34.

https://doi.org/10.1016/j.neuron.2008.06.012 Cited in Section 4

35 **Becker HC** (2008). Alcohol dependence, withdrawal, and relapse. Alcohol Research & Health, 31(4), 348-361.

PubMed Cited in Section 4

36 **Heilig M, Egli M, Crabbe JC, Becker HC** (2010). Acute withdrawal, protracted abstinence and negative affect in alcoholism: are they linked?. Addiction Biology, 15(2), 169-184.

https://doi.org/10.1111/j.1369-1600.2009.00194.x

Cited in Section 4, Section 7, Section 10, Section 12

37 **Sinha R, Fox HC, Hong KI, Hansen J, Tuit K, Kreek MJ** (2011). Effects of Adrenal Sensitivity, Stress- and Cue-Induced Craving, and Anxiety on Subsequent Alcohol Relapse and Treatment Outcomes. Archives of General Psychiatry, 68(9), 942-952.

https://doi.org/10.1001/archgenpsychiatry.2011.49 Cited in Section 4, Section 12

38 **Lieberman MD, Eisenberger NI, Crockett MJ, Tom SM, Pfeifer JH, Way BM** (2007). Putting feelings into words: affect labeling disrupts amygdala activity in response to affective stimuli. Psychological Science, 18(5), 421-428.

https://doi.org/10.1111/j.1467-9280.2007.01916.x Cited in Section 4

39 **Ma X, Yue ZQ, Gong ZQ, Zhang H, Duan NY, Shi YT, et al.** (2017). The Effect of Diaphragmatic Breathing on Attention, Negative Affect and Stress in Healthy Adults. Frontiers in Psychology, 8, 874.

https://doi.org/10.3389/fpsyg.2017.00874 Cited in Section 4

40 **Ebrahim IO, Shapiro CM, Williams AJ, Fenwick PB** (2013). Alcohol and Sleep I: Effects on Normal Sleep. Alcoholism: Clinical and Experimental Research, 37(4), 539-549.

https://doi.org/10.1111/acer.12006 Cited in Section 5

41 **Drummond SPA, Gillin JC, Smith TL, DeModena A** (1998). The Sleep of Abstinent Pure Primary Alcoholic Patients: Natural Course and Relationship to Relapse. Alcoholism: Clinical and Experimental Research, 22(8), 1796-1802.

https://doi.org/10.1111/j.1530-0277.1998.tb03983.x Cited in Section 5, Section 10, Section 13

42 **Brower KJ, Aldrich MS, Robinson EAR, Zucker RA, Greden JF** (2001). Insomnia, Self-Medication, and Relapse to Alcoholism. American Journal of Psychiatry, 158(3), 399-404.

https://doi.org/10.1176/appi.ajp.158.3.399 Cited in Section 5, Section 12

43 **Colrain IM, Nicholas CL, Baker FC** (2014). Alcohol and the sleeping brain. Handbook of Clinical Neurology, 125, 415-431.

https://doi.org/10.1016/B978-0-444-62619-6.00024-0 Cited in Section 5

44 **Volkow ND, Tomasi D, Wang GJ, Telang F, Fowler JS, Logan J, et al.** (2012). Evidence That Sleep Deprivation Downregulates Dopamine D2R in Ventral Striatum in the Human Brain. The Journal of Neuroscience, 32(19), 6711-6717.

https://doi.org/10.1523/JNEUROSCI.0045-12.2012 Cited in Section 5

45 **Yoo SS, Gujar N, Hu P, Jolesz FA, Walker MP** (2007). The human emotional brain without sleep: a prefrontal amygdala disconnect. Current Biology, 17(20), R877-R878.

https://doi.org/10.1016/j.cub.2007.08.007 Cited in Section 5

46 **LeGates TA, Fernandez DC, Hattar S** (2014). Light as a central modulator of circadian rhythms, sleep and affect. Nature Reviews Neuroscience, 15(7), 443-454.

https://doi.org/10.1038/nrn3743 Cited in Section 5

47 **Hasler BP, Smith LJ, Cousins JC, Bootzin RR** (2012). Circadian rhythms, sleep, and substance abuse. Sleep Medicine Reviews, 16(1), 67-81.

https://doi.org/10.1016/j.smrv.2011.03.004 Cited in Section 5

48 **Carter BL, Tiffany ST** (1999). Meta-analysis of cue-reactivity in addiction research. Addiction, 94(3), 327-340.
https://doi.org/10.1046/j.1360-0443.1999.9433273.x Cited in Section 6

49 **Schacht JP, Anton RF, Myrick H** (2013). Functional neuroimaging studies of alcohol cue reactivity: a quantitative meta-analysis and systematic review. Addiction Biology, 18(1), 121-133.
https://doi.org/10.1111/j.1369-1600.2012.00464.x Cited in Section 6

50 **Grüsser SM, Wrase J, Klein S, Hermann D, Smolka MN, Ruf M, et al.** (2004). Cue-induced activation of the striatum and medial prefrontal cortex is associated with subsequent relapse in abstinent alcoholics. Psychopharmacology, 175(3), 296-302.
https://doi.org/10.1007/s00213-004-1828-4 Cited in Section 6

51 **Bouton ME** (2002). Context, ambiguity, and unlearning: sources of relapse after behavioral extinction. Biological Psychiatry, 52(10), 976-986.
https://doi.org/10.1016/S0006-3223(02)01546-9 Cited in Section 6, Section 8, Section 12

52 **Duckworth AL, Gendler TS, Gross JJ** (2016). Situational Strategies for Self-Control. Perspectives on Psychological Science, 11(1), 35-55.
https://doi.org/10.1177/1745691615623247 Cited in Section 6, Section 9, Section 12

53 **Verplanken B, Wood W** (2006). Interventions to Break and Create Consumer Habits. Journal of Public Policy & Marketing, 25(1), 90-103.
https://doi.org/10.1509/jppm.25.1.90 Cited in Section 6

54 **Quinn JM, Pascoe A, Wood W, Neal DT** (2010). Can't Control Yourself? Monitor Those Bad Habits. Personality and Social Psychology Bulletin, 36(4), 499-511.
https://doi.org/10.1177/0146167209360665 Cited in Section 6, Section 12

55 **Gollwitzer PM, Sheeran P** (2006). Implementation Intentions and Goal Achievement: A Meta-analysis of Effects and Processes. Advances in Experimental Social Psychology, 38, 69-119.

https://doi.org/10.1016/S0065-2601(06)38002-1 Cited in Section 6

56 **Armitage CJ** (2009). Effectiveness of experimenter-provided and self-generated implementation intentions to reduce alcohol consumption in a sample of the general population: A randomized exploratory trial. Health Psychology, 28(5), 545-553.

https://doi.org/10.1037/a0015984 Cited in Section 6, Section 12

57 **Li P, Wu P, Xin X, Fan YL, Wang GB, Wang F, et al.** (2015). Incubation of alcohol craving during abstinence in patients with alcohol dependence. Addiction Biology, 20(3), 513-522.

https://doi.org/10.1111/adb.12140 Cited in Section 7, Section 12, Section 13

58 **Craig AD** (2009). How do you feel--now? The anterior insula and human awareness. Nature Reviews Neuroscience, 10(1), 59-70.

https://doi.org/10.1038/nrn2555 Cited in Section 7

59 **Kavanagh DJ, Andrade J, May J** (2005). Imaginary Relish and Exquisite Torture: The Elaborated Intrusion Theory of Desire. Psychological Review, 112(2), 446-467.

https://doi.org/10.1037/0033-295X.112.2.446 Cited in Section 7

60 **Bowen S, Marlatt A** (2009). Surfing the urge: Brief mindfulness-based intervention for college student smokers. Psychology of Addictive Behaviors, 23(4), 666-671.

https://doi.org/10.1037/a0017127 Cited in Section 7, Section 13

61 **Witkiewitz K, Bowen S** (2010). Depression, craving, and substance use following a randomized trial of mindfulness-based relapse prevention. Journal of Consulting and Clinical Psychology, 78(3), 362-374.

https://doi.org/10.1037/a0019172 Cited in Section 7

62 **Dawson DA, Grant BF, Stinson FS, Chou PS, Huang B, Ruan WJ** (2005). Recovery from DSM-IV alcohol dependence: United States, 2001-2002. Addiction, 100(3), 281-292.

https://doi.org/10.1111/j.1360-0443.2004.00964.x Cited in Section 8

63 **de Visser RO, Robinson E, Bond R** (2016). Voluntary temporary abstinence from alcohol during "Dry January" and subsequent alcohol use. Health Psychology, 35(3), 281-289.

https://doi.org/10.1037/hea0000297 Cited in Section 8

64 **Wegner DM, Schneider DJ, Carter SR, White TL** (1987). Paradoxical effects of thought suppression. Journal of Personality and Social Psychology, 53(1), 5-13.

https://doi.org/10.1037/0022-3514.53.1.5 Cited in Section 8, Section 12

65 **Hayes SC, Luoma JB, Bond FW, Masuda A, Lillis J** (2006). Acceptance and Commitment Therapy: Model, processes and outcomes. Behaviour Research and Therapy, 44(1), 1-25.

https://doi.org/10.1016/j.brat.2005.06.006 Cited in Section 8

66 **Rosenquist JN, Murabito J, Fowler JH, Christakis NA** (2010). The Spread of Alcohol Consumption Behavior in a Large Social Network. Annals of Internal Medicine, 152(7), 426-433.

https://doi.org/10.7326/0003-4819-152-7-201004060-00007 Cited in Section 9, Section 12

67 **Litt MD, Kadden RM, Kabela-Cormier E, Petry NM** (2009). Changing network support for drinking: Network Support Project 2-year follow-up. Journal of Consulting and Clinical Psychology, 77(2), 229-242.

https://doi.org/10.1037/a0015252 Cited in Section 9

68 **Litt MD, Kadden RM, Kabela-Cormier E, Petry N** (2007). Changing network support for drinking: Initial findings from the Network Support Project. Journal of Consulting and Clinical Psychology, 75(4), 542-555.

https://doi.org/10.1037/0022-006X.75.4.542 Cited in Section 9

69 **Kelly JF, Humphreys K, Ferri M** (2020). Alcoholics Anonymous and other 12-step programs for alcohol use disorder. Cochrane Database of Systematic Reviews, 2020(3), CD012880.

https://doi.org/10.1002/14651858.CD012880.pub2 Cited in Section 9, Section 13

70 **Best D, Beckwith M, Haslam C, Haslam SA, Jetten J, Mawson E, et al.** (2016). Overcoming alcohol and other drug addiction as a process of social identity transition: the social identity model of recovery (SIMOR). Addiction Research & Theory, 24(2), 111-123.

https://doi.org/10.3109/16066359.2015.1075980 Cited in Section 9

71 **Buckingham SA, Frings D, Albery IP** (2013). Group membership and social identity in addiction recovery. Psychology of Addictive Behaviors, 27(4), 1132-1140.

https://doi.org/10.1037/a0032480 Cited in Section 9

72 **Oyserman D, Fryberg SA, Yoder N** (2007). Identity-based motivation and health. Journal of Personality and Social Psychology, 93(6), 1011-1027.

https://doi.org/10.1037/0022-3514.93.6.1011 Cited in Section 9

73 **Driskell JE, Copper C, Moran A** (1994). Does mental practice enhance performance?. Journal of Applied Psychology, 79(4), 481-492.

https://doi.org/10.1037/0021-9010.79.4.481 Cited in Section 9, Section 12

74 **Mehta G, Macdonald S, Cronberg A, Rosselli M, Khera-Butler T, Sumpter C, et al.** (2018). Short-term abstinence from alcohol and changes in cardiovascular risk factors, liver function tests and cancer-related growth factors: a prospective observational study. BMJ Open, 8(5), e020673.

https://doi.org/10.1136/bmjopen-2017-020673 Cited in Section 10

75 **van Eijk J, Demirakca T, Frischknecht U, Hermann D, Mann K, Ende G** (2013). Rapid Partial Regeneration of Brain Volume During the First 14 Days of Abstinence from Alcohol. Alcoholism: Clinical and Experimental Research, 37(1), 67-74.

https://doi.org/10.1111/j.1530-0277.2012.01853.x Cited in Section 10, Section 12

76 **Pfefferbaum A, Sullivan EV, Mathalon DH, Shear PK, Rosenbloom MJ, Lim KO** (1995). Longitudinal Changes in Magnetic Resonance Imaging Brain Volumes in Abstinent and Relapsed Alcoholics. Alcoholism: Clinical and Experimental Research, 19(5), 1177-1191.

https://doi.org/10.1111/j.1530-0277.1995.tb01598.x Cited in Section 10, Section 13

77 **Hallgren M, Vancampfort D, Giesen ES, Lundin A, Stubbs B** (2017). Exercise as treatment for alcohol use disorders: systematic review and meta-analysis. British Journal of Sports Medicine, 51(14), 1058-1064.

https://doi.org/10.1136/bjsports-2016-096814 Cited in Section 10, Section 12, Section 13

78 **Lynch WJ, Peterson AB, Sanchez V, Abel J, Smith MA** (2013). Exercise as a novel treatment for drug addiction: A neurobiological and stage-dependent hypothesis. Neuroscience & Biobehavioral Reviews, 37(8), 1622-1644.

https://doi.org/10.1016/j.neubiorev.2013.06.011 Cited in Section 10

79 **Bandura A** (1977). Self-efficacy: Toward a unifying theory of behavioral change. Psychological Review, 84(2), 191-215.

https://doi.org/10.1037/0033-295X.84.2.191 Cited in Section 10, Section 12

80 **Curry S, Marlatt GA, Gordon JR** (1987). Abstinence violation effect: Validation of an attributional construct with smoking cessation. Journal of Consulting and Clinical Psychology, 55(2), 145-149.

https://doi.org/10.1037/0022-006X.55.2.145 Cited in Section 11, Section 12, Section 13

81 **Witkiewitz K, Marlatt GA** (2004). Relapse Prevention for Alcohol and Drug Problems: That Was Zen, This Is Tao. American Psychologist, 59(4), 224-235.

https://doi.org/10.1037/0003-066X.59.4.224 Cited in Section 11

82 **Hendershot CS, Witkiewitz K, George WH, Marlatt GA** (2011). Relapse prevention for addictive behaviors. Substance Abuse Treatment, Prevention, and Policy, 6(1), 17.

https://doi.org/10.1186/1747-597X-6-17 Cited in Section 11

83 **Adamson SJ, Sellman JD, Frampton CMA** (2009). Patient predictors of alcohol treatment outcome: A systematic review. Journal of Substance Abuse Treatment, 36(1), 75-86.

https://doi.org/10.1016/j.jsat.2008.05.007 Cited in Section 11

84 **Bowen S, Witkiewitz K, Clifasefi SL, Grow J, Chawla N, Hsu SH, et al.** (2014). Relative Efficacy of Mindfulness-Based Relapse Prevention, Standard Relapse Prevention, and Treatment as Usual for Substance Use Disorders. JAMA Psychiatry, 71(5), 547-556.

https://doi.org/10.1001/jamapsychiatry.2013.4546 Cited in Section 11, Section 12

85 **Kwako LE, Momenan R, Litten RZ, Koob GF, Goldman D** (2016). Addictions Neuroclinical Assessment: A Neuroscience-Based Framework for Addictive Disorders. Biological Psychiatry, 80(3), 179-189.

https://doi.org/10.1016/j.biopsych.2015.10.024 Cited in Section 12

86 **Dai H, Milkman KL, Riis J** (2014). The Fresh Start Effect: Temporal Landmarks Motivate Aspirational Behavior. Management Science, 60(10), 2563-2582.

https://doi.org/10.1287/mnsc.2014.1901 Cited in Section 12

87 **Harkin B, Webb TL, Chang BPI, Prestwich A, Conner M, Kellar I, et al.** (2016). Does monitoring goal progress promote goal attainment? A meta-analysis of the experimental evidence. Psychological Bulletin, 142(2), 198-229.

https://doi.org/10.1037/bul0000025 Cited in Section 12

88 **Lally P, van Jaarsveld CHM, Potts HWW, Wardle J** (2010). How are habits formed: Modelling habit formation in the real world. European Journal of Social Psychology, 40(6), 998-1009.

https://doi.org/10.1002/ejsp.674 Cited in Section 12