CHAPTER FOUR

Social cognition and problematic alcohol use: An organizing theoretical framework and suggestions for future work☆

Lakshmi Kumar[a], Shirley Y. Hill[b], and Kasey G. Creswell[a,*]
[a]Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, United States
[b]Department of Psychiatry, Psychology, and Human Genetics, University of Pittsburgh, Pittsburgh, PA, United States
*Corresponding author. e-mail address: kasey@andrew.cmu.edu

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Abstract

Alcohol is a social drug. Most alcohol use takes place in social settings, social factors play a key role in driving problematic alcohol use, and alcohol has profound effects on social behaviors, both acutely and in the long-term. In this chapter, we offer an organizing theoretical framework highlighting the importance of one particular social factor (i.e., social cognition) that both affects and is affected by alcohol use. Social cognition is conceptualized as being a set of mental abilities that permit successful social interactions. Here, we focus on three specific social cognitive abilities that have been commonly examined in the alcohol literature (i.e., empathy, theory of mind, and emotion recognition). After briefly describing theories put forth to explain the etiology of social cognition deficits, we then define each social cognition ability and describe the measures that are commonly used to assess them in the alcohol literature, ending with a critical appraisal of how well these abilities have been conceptualized and assessed by alcohol researchers. Next, we review growing literatures suggesting that deficits in social cognition may be a risk factor for problematic alcohol use, and that individuals with AUD show deficits in social cognition compared to healthy controls, and we describe mechanisms put forth to explain how social cognition is both a predictor and consequence of problematic alcohol use. We end by making recommendations for more rigorous future studies to address unanswered questions, and we discuss the potential methodological, conceptual, and clinical implications of this theoretical framework.

Alcohol is one of the mostly widely consumed psychoactive substances in the world (Ritchie & Roser, 2018; World Health Organization, 2022). In the United States, about 65% of individuals 18 years and older consumed alcohol in the past year (National Institute of Health, 2023; Substance Abuse & Mental Health Services Administration, 2021). While most people do not experience problems related to their alcohol use, approximately 11.3% of the United States population, and 5.1% of the global population, develop an alcohol use disorder (AUD) (Rehm & Shield, 2019; SAMHSA, 2021; WHO, 2022), defined as a "problematic pattern of alcohol use leading to clinically significant impairment or distress" in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013, p. 490). Excessive alcohol use is

one of the leading risk factors for population health worldwide (Griswold et al., 2018), contributing to 3 million deaths globally each year and accounting for 7.1% and 2.2% of the global burden of disease and injury for males and females, respectively (WHO, 2022). It is essential to identify and respond to early risk factors for problematic alcohol use in order to reduce the prevalence and severity of AUD, as well as to develop effective targeted treatments for AUD and its associated impairments (Hawkins, Catalano, & Miller, 1992; Levy et al., 2016; Witkiewitz, Litten, & Leggio, 2019).

Alcohol is best described as being a social drug (Fairbairn & Sayette, 2014; Kirkpatrick & de Wit, 2013; Steele & Southwick, 1985), as the vast majority of alcohol use takes place in social settings (McCabe, West, Veliz, Frank, & Boyd, 2014; Skrzynski & Creswell, 2020, 2021). Social factors are important in understanding why people drink alcohol and why some individuals go on to develop alcohol problems (Cooper, Kuntsche, Levitt, Barber, & Wolf, 2016; Creswell, 2021; Lindgren, Neighbors, Westgate, & Salemink, 2014; Patrick, Schulenberg, O'malley, Johnston, & Bachman, 2011; Venerable & Fairbairn, 2020). Indeed, models of AUD risk increasingly conceptualize social factors (e.g., interpersonal conflict, marital dissatisfaction, peer pressure, social norms) as being critical to the understanding of problem drinking (Cranford & Fairbairn, 2018; Fairbairn & Sayette, 2014; Leach & Kranzler, 2013; Leonard & Eiden, 2007; Schuckit, Smith, Anderson, & Brown, 2004; Sher, Grekin, & Williams, 2005). Alcohol, in turn, has profound effects on social behavior—both acutely (e.g., alcohol's ability to enhance sociality, affective empathy, and social bonding; Dolder et al., 2017; Fairbairn et al., 2018; Goodwin & Sayette, 2022; Sayette et al., 2012) and long-term (i.e., impairments in social functioning as seen in AUD; see Bora & Zorlu, 2017; Castellano et al., 2015; Kumar, Skrzynski, & Creswell, 2022a; Massey, Newmark, & Wakschlag, 2018; Onuoha, Quintana, Lyvers, & Guastella, 2016; Winters, Brandon-Friedman, Yepes, & Hinckley, 2021). In this chapter, we offer a novel theoretical framework highlighting the importance of one particular social factor (i.e., social cognition) that both affects and is affected by alcohol use (see Fig. 1).

Social cognition is conceptualized as being a set of mental abilities that permit successful social interactions, including perceiving, processing, interpreting, and responding to social stimuli (Arioli, Crespi, & Canessa, 2018; Frith, 2008; Higgins & Bargh, 1987). We focus here on three specific social cognition abilities that have been commonly examined in the alcohol literature (i.e., empathy, theory of mind, and emotion

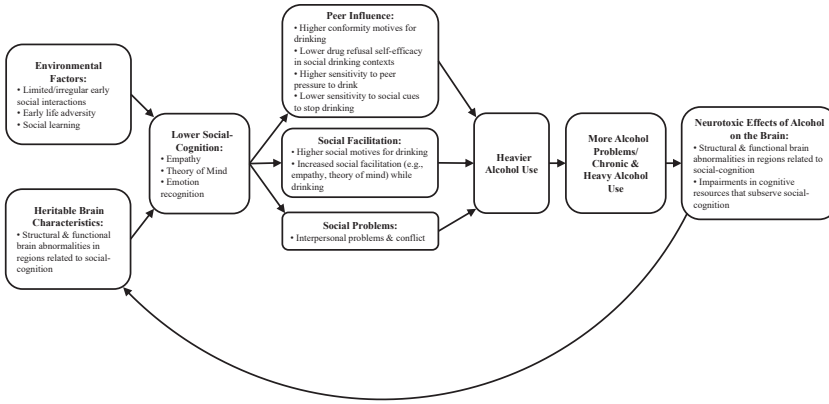

Fig. 1 Conceptual model illustrating how deficits in social cognition are both a risk factor for and consequence of problematic alcohol use.

recognition). We begin by briefly describing theories put forth to explain the etiology of social cognition deficits. We then define each social cognition ability and describe the measures that are commonly used to assess them in the alcohol literature, ending with a critical appraisal of how well these abilities have been conceptualized and assessed by alcohol researchers. Next, we review a growing literature suggesting that lower social cognition may lead to the development of alcohol problems, and we offer mechanisms to explain why lower social cognition is a risk factor for problematic alcohol use. We then review the literature documenting social cognition deficits in individuals with AUD compared to healthy controls, and we discuss the mechanisms put forth in the literature to explain why individuals with AUD might experience these social cognition impairments. We end with some broader considerations (e.g., recommendations for more rigorous future research to address unanswered questions, potential methodological, conceptual, and clinical implications of this theoretical framework).

1. Social cognition abilities: Etiology, definitions, and measures

In the broadest sense, social cognition is an umbrella term that refers to any cognitive process that involves other people (Beer & Ochsner, 2006; Frith, 2008). More specifically, social cognition has been defined as a set of mental abilities used to successfully interact with others, including social

inference (the ability to make accurate inferences about the thoughts, feelings, and intentions of others based on social cues; Arioli et al., 2018; Beer & Ochsner, 2006), social perception (the ability to interpret and understand social cues, such as body language, facial expressions, tone of voice, and other nonverbal signals; Beer & Ochsner, 2006), and social decision making (the ability to make sound decisions in social situations, such as choosing the appropriate response to social cues or choosing between competing social goals; Arioli et al., 2018; Frith & Singer, 2008). Here, we focus our review on the three most commonly studied social cognitive abilities in the alcohol literature—empathy, theory of mind, and emotion recognition. These social cognitive abilities are fundamental to the development of social competence and for successful interpersonal navigation (Beer & Ochsner, 2006; Frith, 2008) and, as we discuss in more detail below, this network of skills is associated with several fronto-limbic brain areas, namely the dorsolateral prefrontal cortex, orbitofrontal cortices, amygdala, and anterior cingulate cortex (Arioli et al., 2018; Saxe, 2006).[1] In this section, we first briefly describe research on the causes of social cognition deficits. We then define each social cognitive ability and summarize the most commonly used measures for each.[2] We end this section with a critical appraisal of how well each social cognition ability has been conceptualized and assessed in the alcohol literature.

1.1 Etiology

As shown in Fig. 1, deficits in social cognition are thought to be influenced by both heritable brain characteristics (i.e., structural and functional brain abnormalities in regions related to social cognition), some of which are thought to be passed down from parents with AUD to their offspring (as we describe in more detail below), and certain environmental factors

[1] Social cognition also includes callous unemotional (CU) traits, which are characterized by a severe lack of empathy, disregard for others' feelings, and a tendency to engage in behavior that is cruel, aggressive, and manipulative (Frick & White, 2008; Frick, 2004). Individuals with high levels of CU traits are at increased risk for developing conduct disorder, antisocial personality disorder, and other externalizing pathology (Frick & Viding, 2009; Frick & White, 2008), including alcohol and substance use disorders (Hyde & Dotterer, 2022; Winters et al., 2021; Wymbs et al., 2012). In this chapter, we focus on normative variation in social cognitive abilities rather than clinically low levels of social cognition as seen with CU traits.

[2] Recent meta-analytic and systematic reviews of social cognition measures in the alcohol literature and broader literature provide exhaustive lists of such measures for interested readers (see Baltariu, Enea, Kaffenberger, Duiverman, & aan het Rot, 2023; Kittel, Olderbak, & Wilhelm, 2022; Pabst, Gautier, & Maurage, 2022; Sanov et al., 2023).

(e.g., early life adversity). Infants as young as a few months old can engage in social referencing, using others' facial and emotional expressions to guide their own behavior (Striano & Reid, 2006). Social cognitive abilities continue to develop throughout childhood and adolescence, but limited or irregular social interactions and early life adversity, such as neglect or abuse, can have a negative impact on social cognition development (Rokita, Dauvermann, & Donohoe, 2018). On the other hand, regular exposure to a wide range of adaptive social situations and interactions can positively impact social cognition development in infancy and beyond, primarily through social learning (De Jaegher, Di Paolo, & Gallagher, 2010; Striano & Reid, 2006). While a full review of this literature is beyond the scope of this chapter, several reviews on the development of social cognition, as well as impairments in such development, are available for the interested reader (Kilford, Garrett, & Blakemore, 2016; Rokita et al., 2018).

1.2 Definitions

1.2.1 Empathy

Empathy lacks a universally accepted definition, but it is typically thought to encompass a wide range of social abilities and experiences that allow individuals to connect with others, including feeling concern for others, sharing in others' emotions, feeling personal distress in response to others' emotional states, understanding others' thoughts and feelings, and merging the self and others' perspectives (e.g., Davis, 1980; Decety & Ickes, 2011; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004; see Cuff, Brown, Taylor, & Howat, 2016 for a review). Typically, empathy is conceptualized as being multidimensional with a cognitive component (i.e., the capacity to understand another's emotional perspective or state), and an affective component (i.e., the capacity to respond emotionally to or share another's emotional state) (Cuff et al., 2016; Hoffman, 1982; Riggio, Tucker, & Coffaro, 1989). Empathy has most often been construed as being a dispositional or trait level (i.e., between-person) construct in alcohol studies but, outside of this literature, there is growing awareness that empathy is also a state level (i.e., within-person) construct (Depow, Francis, & Inzlicht, 2021; Nezlek, Feist, Wilson, & Plesko, 2001; Nezlek, Schütz, Lopes, & Smith, 2007; Stellar & Duong, 2023; Van der Graaff et al., 2016). Trait empathy can be thought of as a general tendency for a person to show empathy across situations and contexts, whereas state empathy can be thought of as an individual's fluctuations in empathy in response to particular interpersonal

interactions (Nezlek et al., 2001; Van der Graaff et al., 2016) or features of the social context (Stellar & Duong, 2023; Zaki, 2014). The motivated model of empathy suggests that people are either driven to employ empathy or to avoid it (Zaki, 2014). For example, positive affect, affiliation, and social desirability typically motivate people to approach empathy, whereas competition, material costs, and cognitive costs typically motivate people to avoid empathy (Cameron et al., 2019; Stellar & Duong, 2023; Zaki, 2014). Thus, while people show individual differences in average empathy (trait empathy), they can also fluctuate and shift meaningfully in their empathy across situations and social contexts (state empathy; Depow et al., 2021; Nezlek et al., 2001; Ringwald & Wright, 2021), the latter of which may have important implications for understanding alcohol use beyond trait empathy (Kumar, Ringwald, Wright, & Creswell, 2023), as we discuss in more detail below.

1.2.2 Theory of mind

A functional theory of mind (ToM) helps people judge, analyze, and infer others' behaviors and make good decisions in social environments (Apperly & Butterfill, 2009). ToM is typically defined as the capacity to understand other people by ascribing mental states (e.g., thoughts, intentions, desires, beliefs) to them (Apperly & Butterfill, 2009; Frith & Frith, 2005). ToM is also thought to be a multidimensional construct and, like empathy, consists of a cognitive (or reasoning) facet and an affective (or decoding) facet (Shamay-Tsoory & Aharon-Peretz, 2007). Cognitive ToM refers to the ability to understand and make inferences about other's mental states, such as beliefs, desires, intentions, and knowledge (Shamay-Tsoory & Aharon-Peretz, 2007). Affective ToM refers to the ability to understand and make inferences about others' emotions (Shamay-Tsoory & Aharon-Peretz, 2007).

In addition to the distinction between cognitive and affective ToM, when considering the development of ToM abilities in young children, it has been useful to also consider first-order vs. second-order ToM (Miller, 2009, 2012; Perner & Wimmer, 1985). First-order ToM usually develops around the ages of 3–5 and involves an individual's ability to understand and make inferences about the mental states of others based on others' behavior and the situational context (Miller, 2009, 2012). It involves the recognition that others can have different beliefs, desires, or emotions than oneself. Second-order ToM is considered to be a higher-level cognitive ability, which usually emerges around the ages of 5–7, and involves an

individual's ability to understand and make inferences about the mental states of others based on one's own beliefs about the mental states of others (Miller, 2009, 2012; Perner & Wimmer, 1985). In other words, second-order ToM involves predicting what one person thinks or feels about what another person is thinking or feeling. As we mention below, the distinction between first-order and second-order ToM may be useful to consider when examining the offspring of individuals with AUD, who may have inherited brain characteristics associated with social cognition deficits themselves.

1.2.3 Emotion recognition

Emotion recognition is typically defined as the ability to accurately identify emotions in others (Castellano et al., 2015; De Gelder, 2009), usually conveyed through facial expressions, vocal tones, and other nonverbal cues (Rosenberg & Ekman, 2020; Schlegel, Grandjean, & Scherer, 2012).

1.3 Measures

The assessment of empathy and ToM in the alcohol literature has been approached using self-report questionnaires, performance-based measures, and neuroimaging methods (e.g., fMRI). Emotion recognition has been assessed with performance-based measures. Tables 1 and 2 describes the most commonly used assessments for empathy, ToM, and emotion recognition in alcohol research. We describe findings from neuroimaging methods in social cognition research in a section below, to contextualize our discussion of mechanisms for why individuals with AUD may have social cognition deficits compared to healthy controls.

1.4 Critical appraisal of the conceptualization and measurement of social cognitive abilities in alcohol research

Before reviewing work showing how deficits in empathy, ToM, and emotion recognition may both predict problematic alcohol use and result from it, we first consider three limitations related to how these abilities have been conceptualized and assessed in alcohol research (see also Creswell & Kumar, 2023; Kittel et al., 2022; Oakley, Brewer, Bird, & Catmur, 2016; Pabst & Maurage, 2023; Pabst et al., 2022; Sunahara et al., 2022). First, there is considerable overlap in how social cognitive abilities have been conceptualized, particularly with cognitive empathy, affective ToM, and emotion recognition, all of which share key similarities (i.e., they all involve the ability to perceive and comprehend another person's

Table 1 Empathy and theory of mind (ToM) measures commonly used in the alcohol literature.

Measure	Type	Subscales	Description	Response anchors	Scoring	Psychometrics
Empathy						
Interpersonal Reactivity Index (IRI; Davis, 1980)	Self-report questionnaire	Cognitive empathy, Affective empathy	28 Items assessing four components of empathy: **Perspective taking** (i.e., ability to understand other's thoughts, feelings, and experiences from their point of view); **Empathic concern** (i.e., feelings of concern and compassion for others); **Fantasy** (i.e., tendency to become emotionally involved in fictional characters and stories); **Personal distress** (i.e., anxiety and discomfort in response to others' emotional states)	5-point Likert scale (1 = Does not describe me well to 5 = Describes me very well)	Average scores on the four subscales and average total score, with larger scores reflecting more empathy	Cronbach's alpha of 0.68–0.79; Test–retest reliability of 0.61–0.81 (Davis, 1980)

(*continued*)

Table 1 Empathy and theory of mind (ToM) measures commonly used in the alcohol literature. (*cont'd*)

Measure	Type	Subscales	Description	Response anchors	Scoring	Psychometrics
Empathy Quotient (EQ; Baron-Cohen & Wheelwright, 2004)	Self-report questionnaire	Total empathy	60 Items assessing cognitive (e.g., "I am good at predicting how someone will feel") and affective (e.g., "I get upset if I see people suffering on news programs") empathy	4-point Likert scale (1 = Strongly agree to 4 = Strongly disagree)	Average total score, with larger scores reflecting more empathy	Cronbach's alpha ~0.92; Test–retest reliability ~0.97 (Baron-Cohen & Wheelwright, 2004; Lawrence et al., 2004)
Eysenck Impulsivity-Venturesomeness-Empathy Scale (EIVES; Eysenck & Eysenck, 1978)	Self-report questionnaire	Total empathy	63 Items assessing three personality traits: **Impulsivity** (i.e., doing and saying things without thinking); **Venturesomeness** (i.e., taking risks and thrill seeking); **Empathy** (i.e., understanding and sharing other's feelings)	Yes/No format	Summed yes score for each subscale	Cronbach's alpha of 0.637–0.654 (Eysenck & Eysenck, 1978)

Multifaceted Empathy Test (English-validated MET; Dziobek et al., 2008; Foell, Brislin, Drislane, Dziobek, & Patrick, 2018)	Behavioral measure	Cognitive empathy, Affective empathy	Computer-assisted test consisting of 40 photographs that show people in positive- or negative-emotionally charged situations	**Cognitive empathy:** Participants asked to select correct emotional state from list of four responses (e.g., sad, desperate, proud, bored); **Affective empathy:** 9-point Likert scale (1 = Not at all to 9 = Very strongly) of how much participants feel what the person in each scene is feeling	**Cognitive empathy:** Percent of correct responses relative to total responses **Affective empathy:** Average score for positive and negative emotion valenced items and average total score	**Cognitive empathy:** Cronbach's alpha of 0.49 **Affective empathy:** Cronbach's alpha of 0.93–0.94 (Foell et al., 2018)

(*continued*)

Table 1 Empathy and theory of mind (ToM) measures commonly used in the alcohol literature. (*cont'd*)

Measure	Type	Subscales	Description	Response anchors	Scoring	Psychometrics
Theory of mind (ToM)						
Reading the Mind in the Eyes Test (RMET; Baron-Cohen et al., 2001)	Behavioral measure	Affective ToM	Participants have to infer the emotional states from 36 images of the eye region alone. Stimuli represent positive (e.g., playful, happy) and negative (e.g., sad, hateful) emotions as well as neutral (e.g., serious, reflective) mental states.	Participants asked to choose best word that describes what the person in the picture is thinking or feeling among four options on the answer sheet	Summed total number of correct responses	Cronbach's alpha of 0.73; RMET more related with emotion perception ($r = 0.33$) than ToM measures ($r = 0.29$), suggesting poor construct validity (Kittel et al., 2022)

Faux Pas Test (Stone et al., 1998)	Behavioral measure	Cognitive ToM	Participants presented with short vignettes that depict social interactions between two characters with a positive or negative outcome. Task includes 10 Faux Pas vignettes and 10 control vignettes without a Faux pas Six subscales: Faux Pas detection, understanding inappropriateness, intentions, belief, empathy, and story comprehension.	Participants asked to identify faux pas in each scenario by answering the yes/no question "Did anyone say something they shouldn't have said or something awkward?"; If yes, asked seven additional yes/no questions about social norms and the mental states of characters in the scenario; If no, asked two more yes/no questions	Summed correct responses for each subscale and then divided by the number of questions in that subscale	Cronbach's alpha of 0.91 (Chen et al., 2017; Yeh, Hua, & Liu, 2009); Test–retest reliability of 0.76–0.83 (Chen et al., 2017; Zhu et al., 2007)

(*continued*)

Table 1 Empathy and theory of mind (ToM) measures commonly used in the alcohol literature. (*cont'd*)

Measure	Type	Subscales	Description	Response anchors	Scoring	Psychometrics
Yoni's Task (Shamay-Tsoory & Aharon-Peretz, 2007)	Behavioral measure	Cognitive ToM Affective ToM First-order ToM Second-order ToM	64- or 98-item computerized task showing a face named "Yoni" and four colored pictures placed around the face referring to different semantic categories (e.g., fruit, means of transport) or faces; Sentences include both cognitive (e.g., "thinking of") and affective (e.g., "loves") ToM, as well as first-order (e.g., "Yoni is thinking of/loves...") and second-order (e.g., "Yoni is thinking of/loves the fruit that...wants") ToM	Sentences appear on top of the screen in each trial and participants asked to correctly identify the picture/face to which Yoni is directed to; Participants then asked to answer questions about what the characters are thinking or feeling; Control condition requires physical judgements (e.g., "which object is closest to Yoni").	Summed correct scores on the subscales and summed correct total score, with higher scores reflecting more ToM; Average reaction time for each subscale and average total reaction time	Cronbach's alpha of 0.90 (Isernia et al., 2022) and $r = 0.32–0.63$ (Lannoy et al., 2020)

Table 2 Emotion Recognition Measures Commonly Used in the Alcohol Literature.

Goal of measure	Measures	Description	Typical outcomes	Psychometrics
Ability to identify specific emotion (e.g., sadness, fear) from facial expressions	Variant of: **Emotion Recognition Task** (e.g., Penn Emotion Recognition Test; Geneva Emotion Recognition Test; Kohler, Turner, Gur, & Gur, 2004; Schlegel, Grandjean, & Scherer, 2014) or **Alternate Forced Choice Task** (e.g., 2 Alternate Forced Choice Task; Attwood, Ohlson, Benton, Penton-Voak, & Munafò, 2009)	**Static images** of facial expressions; **Morphed facial expressions** from a neutral or ambiguous expression to a full emotional exemplar (e.g., anger, fear)	**Static images:** **Accuracy** (i.e., correct identification of an emotion); **Reaction times** (i.e., amount of time elapsed between facial stimulus presentation and emotion identification) **Morphed facial expressions:** **Accuracy**; **Reaction times**; **Threshold detection** (i.e., point at which an emotion is identified from facial stimuli containing varying amounts of emotional content); **False alarms** (i.e., number of incorrect identifications of an absent emotion); **Neutral-response errors** (i.e., incorrect identification of a specific emotion as neutral), **Response sensitivity** (i.e., ability to discriminate the presence of a specific emotion from a stimulus); **Response bias** (i.e., tendency to see a specific emotion even when emotion is absent); **Error ratios** (i.e., proportion of correct to incorrect responses for identifying a specific emotion from facial expressions)	Cronbach's alphas of 0.53–0.93 (Lyusin & Ovsyannikova, 2016; Pinkham, Penn, Green, & Harvey, 2016; Williams, Vehabovic, & Simms, 2023); Test–retest reliability of 0.68–0.86 (Lyusin & Ovsyannikova, 2016; Pinkham et al., 2016)[a]

(*continued*)

Table 2 Emotion Recognition Measures Commonly Used in the Alcohol Literature. (*cont'd*)

Goal of measure	Measures	Description	Typical outcomes	Psychometrics
Tendency to see a specific emotion (e.g., happiness) over another emotion (e.g., anger) when both are on a continuum	Variant of: **Alternate Forced Choice Task** (e.g., 2 or 6 Alternate Forced Choice Task; Attwood, Ataya, Benton, Penton-Voak, & Munafò, 2009; Eastwood, Penton-Voak, Munafò, & Attwood, 2020)	Facial expressions that gradually morph from one emotional endpoint (e.g., happiness) to another (e.g., anger)	**Balance point** (i.e., point along the continuum where a participant is equally likely to identify an image as either of the two emotional endpoints); Balance points close to one end of the spectrum (e.g., a threshold closer to happiness vs a threshold closer to anger) rather than the middle range (i.e., 50%) indicate a recognition bias for that specific emotion over the other	

[a]Note: Some have argued that the standard psychometric model, which attributes low internal consistency to random error, may not be applicable to nonverbal sensitivity tests (i.e., tests that measure abilities to recognize communication from nonverbal expressions; Hall, 2001). This argument centers on the fact that the emotions under investigation are distinct entities that share some characteristics but differ in many other aspects, making standard item consistency analysis infeasible for most emotion recognition tasks (Hall, 2001; Scherer & Scherer, 2011).

emotional or mental state; Castellano et al., 2015; Cuff et al., 2016; Shamay-Tsoory & Aharon-Peretz, 2007). While some researchers find evidence of them being independent constructs (e.g., Kanske, Böckler, Trautwein, & Singer, 2015), others describe ToM as being a facet of cognitive empathy (e.g., Stellar & Duong, 2023), cognitive empathy and ToM as being similar or the same constructs (e.g., Batson, 2009; Blair, 2005; Lawrence, Shaw, Baker, Baron-Cohen, & David, 2004) and/or emotion recognition as being a lower-level process that supports successful empathy and/or ToM (e.g., Kittel et al., 2022; Pabst et al., 2022). Further, while empathy and ToM have been construed as having both cognitive and affective components, which have been shown to have distinctive heritability (Abramson, Uzefovsky, Toccaceli, & Knafo-Noam, 2020) and differentially predict alcohol and other health outcomes (Hoffman, 1982; Kumar et al., 2022a; Riggio et al., 1989; Shamay-Tsoory & Aharon-Peretz, 2007), many studies in the alcohol literature use measures that do not separate out these components (see Bora & Zorlu, 2017; Kumar et al., 2022a; Onuoha et al., 2016), so it is difficult to draw conclusions about what exactly is driving associations between these two social cognition abilities and problematic alcohol use. Finally, with few exceptions, alcohol researchers have conceptualized empathy and ToM to be dispositional trait-like constructs, despite compelling evidence suggesting that they may in fact be state-like and vary depending on the context (e.g., Stellar & Duong, 2023), with potential differential associations between trait and state social cognition and alcohol use (Kumar et al., 2023). These issues with overlapping conceptualizations of social cognition abilities, failure to consider the multi-dimensional nature of empathy and ToM, and the relative neglect of potentially important within-person changes in social cognition abilities have likely hindered progress in understanding the role of these social cognition deficits in problematic alcohol use.

Second, common empathy, ToM, and emotion recognition assessments used in alcohol studies may have questionable validity (Kittel et al., 2022; Pabst et al., 2022). For instance, common ToM tasks (e.g., the Reading the Mind in the Eyes Test (RMET); Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001) often require successful emotion recognition in order to perform well on them (Kittel et al., 2022), blurring the lines between two social cognition constructs that are thought to be distinct by many (Oakley et al., 2016; Quesque & Rossetti, 2020). In fact, a recent meta-analysis that used random-effects models to compare the RMET (Baron-Cohen et al., 2001) to emotion perception, cognitive empathy, and affective empathy

measures demonstrated that the RMET (Baron-Cohen et al., 2001) shared 23% of variance with emotion recognition measures, 15% of variance with other measures of ToM (e.g., the Faux Pas test; Stone, Baron-Cohen, & Knight, 1998), 4% of variance with cognitive empathy measures, and 4% of variance with affective empathy measures (Kittel et al., 2022). These results suggest that the RMET (Baron-Cohen et al., 2001) at least partly overlaps with emotion recognition, and to a lesser degree with cognitive and affective empathy, which is problematic for conceptualizations that treat these social cognitive abilities as being distinct (e.g., Bora & Zorlu, 2017; Buitelaar, Van Der Wees, Swaab–Barneveld, & Van Der Gaag, 1999).

Researchers have also raised concerns about the construct validity of widely used empathy self-report measures, such as the Interpersoal Reactivity Index (IRI; Davis, 1980) and the Empathy Quotient (EQ; Baron-Cohen & Wheelwright, 2004), noting that these measures often do not contain items directly querying cognitive or affective empathy, as they are commonly defined (Murphy et al., 2020; Pabst & Maurage, 2023). Further, there are often weak associations between self-report measures of empathy and their behavioral counterparts (Murphy & Lilienfeld, 2019; Pabst & Maurage, 2023; Sunahara et al., 2022), which some have argued is evidence of poor construct validity of the empathy self-reports (e.g., Murphy & Lilienfeld, 2019; Pabst & Maurage, 2023). In fact, some researchers have suggested that individuals with AUD may be poor judges of their empathic abilities and have prioritized behavioral measures of empathy over self-reports to index "objective" empathic ability (Pabst & Maurage, 2023). However, there are theoretical and methodological explanations for weak associations between empathy self-report and behavioral measures beyond assuming a lack of insight about empathic abilities (see Creswell & Kumar, 2023), and more research is needed to substantiate this claim, especially given the questionable ecological validity of widely used empathy behavioral tasks (Creswell & Kumar, 2023; Sunahara et al., 2022), as we discuss in more detail in the next paragraph.

In addition to concerns about the construct validity of common ToM and empathy measures, widely used emotion recognition tasks that ask participants to categorize static facial expressions by choosing from a list of emotion labels have been criticized for lacking ecological validity and ignoring how language may impact emotion perception (Barrett, Lindquist, & Gendron, 2007; Barrett, Mesquita, & Gendron, 2011; Pabst et al., 2022). In general, social cognition tasks have poor ecological validity because they do not require participants to actually interact with other

people, but to rather make inferences based on pictures, videos, or vignettes of other people, which is not how social cognition works in the real world (Creswell & Kumar, 2023; Kittel et al., 2022; Pabst et al., 2022). More research is needed to establish the construct, ecological, and discriminant validity of tasks used to assess social cognitive abilities.

Third, empathy, ToM, and emotion recognition represent a restricted range of social cognition components (Pabst et al., 2022), and future studies should consider other core components of social cognition that are important for understanding adaptive social functioning (Couture, 2006; Fett et al., 2011; Klein Tuente, Bogaerts, & Veling, 2019) and may also be important for understanding problematic alcohol use, including social perception (the identification and use of information about social contexts and subtle interpersonal relationships), social knowledge (the knowledge of social rules or expectations that underlie social situations), and attributional biases (tendencies to endorse certain types of causal explanations for social events more than others) (Gautier, Pabst, & Maurage, 2021; Green et al., 2008; Pabst, Peyroux, Gautier, de Timary, & Maurage, 2021; Pabst et al., 2022; Pinkham, 2014). With these caveats in mind, we now turn to theoretical accounts and empirical studies suggesting that lower social cognition abilities may be a risk factor for the development of problematic alcohol use.

2. Social cognition and problematic alcohol use in non-clinical samples

Most previous studies have focused on deficits in social cognition that are present in individuals with AUD compared to healthy controls (which we review in the next section), and conceptualized these deficits as resulting from prolonged and heavy alcohol use (Kumar et al., 2022a; Massey et al., 2018; Winters et al., 2021). Recent theoretical models and empirical studies suggest that early deficits in social cognition may also predict future heavy alcohol use and the development of AUD (e.g., Hill et al., 2007; Kumar et al., 2022a; Kumar, Skrzynski, & Creswell, 2022b; Massey et al., 2018; Winters, Massey, & Sakai, 2023; Winters, Wu, & Fukui, 2020; Winters et al. 2021). Indeed, several aspects of social cognition have been shown to relate to the onset and course of AUD, such that poorer social cognition predicts early alcohol and drug use, greater severity of AUD, and poorer

treatment outcomes (Thoma, Friedmann, & Suchan, 2013; Thorberg, Young, Sullivan, & Lyvers, 2009; Uekermann & Daum, 2008). The aim of this section is to first summarize the existing literature on the associations between lower empathy, ToM, and emotion recognition abilities and problematic alcohol use in non-clinical samples (i.e., adolescents, college students, community adults). Rather than providing a narrative review of all individual studies, when relevant, we summarize existing meta-analyses and systematic reviews on these topics. We then discuss potential mechanisms explaining the link between lower social cognitive abilities and problematic alcohol use.

2.1 Empathy

Kumar et al. (2022a) meta-analyzed the associations between lower empathy, as assessed most commonly by self-report dispositional empathy questionnaires, and heavier alcohol consumption ($k = 11$) and more alcohol problems ($k = 7$) in non-clinical samples of adolescents, young adults, and adults and found small effect sizes of $r = -0.12$ and $r = -0.08$, respectively. There was not significant heterogeneity in effect sizes across studies, and thus moderators (e.g., cognitive vs affective empathy) were not tested. However, in several studies that examined affective and cognitive empathy separately (e.g., Laghi, Bianchi, Pompili, Lonigro, & Baiocco, 2019a; Lannoy et al., 2020; Lyvers, McCann, Coundouris, Edwards, & Thorberg, 2018), effects were stronger for affective (vs cognitive) empathy.

As noted above, the literature linking lower empathy to alcohol use and problems has focused almost exclusively on trait-level (vs state-level) empathy, but empathy has been construed as a state-like construct as well in the broader literature on empathy (Nezlek et al., 2001; Stellar & Duong, 2023). We are aware of only one prior study that tested whether state empathy is implicated in the within-person process of alcohol consumption (i.e., by determining whether individuals drank more or less alcohol on days that they reported lower or higher empathy). Using ecological momentary assessment (EMA) methodology, Kumar et al. (2023) examined associations between daily fluctuations in state empathy and daily alcohol use in a sample of adult alcohol drinkers ($n = 492$). Results showed that higher day-level state affective empathy was not associated with the likelihood of drinking on a particular day but, surprisingly, it was significantly associated with a greater number of drinks consumed on alcohol-consuming days, with the latter association remaining after controlling for day-level positive affect and negative affect (Kumar et al., 2023). These findings are in contrast to the

meta-analytic findings above linking lower trait empathy to heavier alcohol consumption and may be due to differences between trait vs state empathy in predicting outcomes. Nearly all of the studies included in these meta-analyses above had participants complete questionnaires about their typical engagement in empathy, while Kumar et al. (2023) asked participants to report on their empathy levels in real-time and with regard to specific social interactions in their daily lives. These findings suggest that there may be differential associations between state and trait empathy and alcohol use, but future research is needed to confirm this.

2.1.1 Theory of mind

Kumar et al. (2022b) meta-analyzed the association between lower ToM, as assessed almost exclusively by the RMET (Baron-Cohen et al., 2001), and more alcohol problems in non-clinical samples of adolescents, young adults, and adults ($k = 6$) and found a small effect size of $r = -0.16$. There was significant heterogeneity in effect sizes across studies, but this heterogeneity was not explained by several tested moderators (i.e., age, sex, study quality). It is notable that all but one of the studies included in this meta-analysis assessed decoding/affective ToM using the RMET (Baron-Cohen et al., 2001), and future studies are indicated to examine whether lower reasoning/cognitive ToM is also associated with alcohol problems in non-clinical samples. As noted above, researchers have questioned whether the RMET (Baron-Cohen et al., 2001) is a valid measure of ToM, suggesting that it may actually be assessing emotion recognition or, at the very least, that success on the task requires skillful emotion recognition (Kittel et al., 2022; Oakley et al., 2016; Pabst et al., 2022; Quesque & Rossetti, 2020). To the extent that the RMET (Baron-Cohen et al., 2001) is also capturing some elements of emotion recognition ability, then the meta-analysis results reported by Kumar et al. (2022b) suggest that individuals from non-clinical samples who report problematic alcohol use may also demonstrate poor emotion recognition. We discuss further evidence of the link between poor emotion recognition and problematic alcohol use in non-clinical samples below.

2.1.2 Emotion recognition

Lannoy et al. (2021) conducted a systematic review on emotional processes, including the ability to identify emotions in others, associated with binge drinking. They found that binge drinking was consistently linked to difficulties in recognizing fear in emotion identification tasks ($k = 10$), with some suggestion for impairments in the recognition of sadness, as well.

Furthermore, there was some evidence linking binge drinking to disrupted brain activity during the identification of emotional facial expressions. In addition to this systematic review, a longitudinal study found that a bias towards angry faces (i.e., fewer errors in recognizing angry expressions) during a baseline assessment predicted the initiation of tobacco and alcohol use during the subsequent four years in a sample of adolescents (Ernst et al., 2010).

2.1.3 Interim summary

As we mentioned above and discuss in more detail in the next section below, long-term heavy alcohol use is often thought to explain deficits in empathy, ToM, and emotion recognition in individuals with AUD (Bora & Zorlu, 2017; Kumar et al., 2022a; Onuoha et al., 2016), but the reviews and studies examined above demonstrate that the links between lower social cognition and heavier alcohol use and problems also exist in non-clinical samples of individuals, who likely do not have a chronic history of heavy alcohol consumption as seen in those with AUD. While studies included in these reviews were all cross-sectional in design (but see Ernst et al., 2010 for a longitudinal study), the results are at least consistent with the proposition that deficits in empathy, ToM, and emotion recognition may also precede the onset of heavy alcohol use/problems and serve as a risk factor for problematic alcohol use (Hill et al., 2007; Kumar et al., 2022a, 2022b; Lannoy et al., 2021; Massey et al., 2018; Winters et al., 2021).

3. Mechanisms for links between lower social cognition and problematic alcohol use

Mechanisms that have been proposed to explain the links between lower social cognition and heavier alcohol use and more alcohol problems center around motives for and experiences related to drinking in social situations, as well as increased interpersonal problems and conflict. These mechanisms generally fall into three broad categories (i.e., peer influence, social facilitation, and social problems; see Fig. 1). We review work supporting each of these three mechanisms below.

3.1 Peer influence

Researchers have proposed that individuals with lower social cognitive abilities may be prone to heavier alcohol use because they may be insensitive to social cues to stop drinking (Massey et al., 2018) and/or they may

misinterpret and over-value the attitudes and norms about the drinking of their peers and consider drinking a way to be accepted by their peer group (Cousijn, Luijten, & Ewing, 2018; Laghi, Bianchi, Pompili, Lonigro, & Baiocco, 2019b; Lannoy et al., 2020). Consistent with these hypotheses, in a sample of underage drinkers, Kumar, Zhou et al. (2022) showed that lower ToM (as assessed by the RMET; Baron-Cohen et al., 2001) had indirect effects on more frequent binge drinking and greater alcohol problems through higher conformity motives for drinking (i.e., the level of conforming and engaging in alcohol use in response to external social pressures). Similarly, another study showed that empathy was associated with alcohol and drug use indirectly through lower drug/alcohol refusal self-efficacy in social settings (Nguyen, Clark, & Belgrave, 2011). Finally, in one study that examined cognitive and affective empathy as moderator variables, there was a stronger association between lower drug/alcohol refusal self-efficacy in social settings and more binge drinking in the presence of low cognitive empathy (Laghi et al., 2019a), suggesting that individuals high in cognitive empathy may be better skilled in social interactions, allowing them to manage peer influence to drink. These findings are consistent with studies showing that individuals with lower social cognitive abilities may be more susceptible to peer pressure in general (Nguyen et al., 2011; Trinidad & Johnson, 2002; Trinidad, Unger, Chou, & Anderson Johnson, 2004). Taken together, studies provide initial support highlighting the role of peer influence in explaining why individuals with lower social cognitive abilities might escalate their drinking and develop alcohol problems.

3.2 Social facilitation

The second potential mechanism linking lower social cognition to problematic drinking centers on social facilitation. Individuals with social cognition deficits may have stronger drinking motives for social facilitation and actually experience increased social facilitation while drinking compared to those with higher social cognition abilities. Specifically, researchers have hypothesized that individuals who typically struggle with perceiving, interpreting, and responding to social stimuli may use alcohol as a coping mechanism in social situations (Cooper et al., 2016; Kuntsche, Knibbe, Gmel, & Engels, 2006a; 2006b), and they may gain particular benefit from alcohol's robust social affiliative effects in increasing affective empathy, social bonding, and other prosocial variables (Creswell et al., 2012; Dolder et al., 2017; Kirkpatrick & de Wit, 2013; Sayette et al., 2012)

and decreasing social tension (Fairbairn & Testa, 2017; Sayette, Smith, Breiner, & Wilson, 1992; Sayette et al., 2012; Yankofsky, Wilson, Adler, Hay, & Vrana, 1986). Put another way, individuals with lower (vs higher) social cognition may be more motivated to drink for alcohol's positively (e.g., increased social bonding) and negatively (e.g., decreased social tension) reinforcing social effects, and they may be more sensitive to these effects of alcohol, placing them at elevated risk to escalate their drinking and develop alcohol problems. Indeed, social motives for drinking (Cooper et al., 2016; Patrick et al., 2011; Smith, Goldman, Greenbaum, & Christiansen, 1995) and increased social reward from drinking (see Fairbairn & Sayette, 2014) have both been shown to predict alcohol problems in the broader alcohol literature.

Consistent with this proposition that increased social facilitation at least partly explains the link between lower social cognition and risk for problematic drinking, one study showed that alcohol (target BAC = 0.4 g/L) vs placebo beverage consumption increased affective empathy for photographs with positive emotional valence, and this effect was stronger for social drinkers with lower trait empathy scores (Dolder et al., 2017). Two other studies found that alcohol (vs placebo or control beverages) increased social cognition, but neither tested whether the effects were stronger for participants with lower social cognitive abilities. Specifically, Johnson, Skromanis, Bruno, Mond, and Honan (2018) found that intoxicated participants (target BAC = 0.08%) responded significantly more negatively to Faux Pas items than placebo participants, indicating an enhancement effect for ToM ability in alcohol-intoxicated individuals. Additionally, Garrison, Yoon, Brown-Schmidt, Ariss, and Fairbairn (2022) found that alcohol (target BAC = 0.08%) increased common ground (i.e., shared language to describe ambiguous images to others, which they construed as a social-cognitive ability) compared to a no-alcohol control beverage. Beyond these studies, we are aware of only four other studies that tested whether alcohol increased empathy or ToM, reporting null or opposite effects, but these studies did not test the hypothesized interaction between lower social cognition and alcohol's effects (Francis, Gummerum, Ganis, Howard, & Terbeck, 2019; Hu, Cui, Fan, Pei, & Wang, 2018; Mitchell, Beck, Boyal, & Edwards, 2011; Thiel et al., 2018). Further, while several experimental studies have tested the acute effects of alcohol (vs placebo beverage) consumption on emotion recognition abilities in young adult social drinkers (see Baltariu et al., 2023; Sanov et al., 2023 for systematic reviews), with inconsistent results, none of these studies tested whether alcohol's effects on

emotion recognition differed based on lower (vs higher) social cognitive abilities. In general, more studies are needed to test whether stronger motives for and experiences related to increased social facilitation while drinking are driving the associations between lower social cognition and problematic drinking in non-clinical samples.

3.3 Social problems

Finally, individuals with deficits in social cognition may have difficulties in forming and maintaining adaptive interpersonal relationships and encounter social conflicts, which could increase the likelihood of problematic alcohol consumption and related consequences. Lower empathy is associated with aggression, antisocial behavior, and externalizing behavior, as well as giving or receiving abuse (Lovett & Sheffield, 2007; Miller & Eisenberg, 1988), while higher ToM has been linked to reduced aggressive or disruptive behavior in boys and increased prosocial behavior in girls (Walker, 2005). Additionally, skillful emotion recognition negatively associates with social problems in young boys and girls (Dede, Delk, & White, 2021), and positively associates with social adjustment in girls (Leppanen & Hietanen, 2001). Studies also show that lower social cognition may contribute to interpersonal problems and lead to reduced perceptions of social connectedness (Galinsky, Ku, & Wang, 2005; Hu et al., 2014; Mcwhirter, Besett-Alesch, Horibata, & Gat, 2002) and less engagement in prosocial behaviors (Fett et al., 2014; Imuta, Henry, Slaughter, Selcuk, & Ruffman, 2016; Tamnes et al., 2018), which in turn could lead individuals with lower social cognition to prioritize substance use over social connections (Winters et al., 2020, 2023). This is supported by a large literature linking social and interpersonal problems with increased alcohol use and problems in adolescents and adults (Armeli, Dehart, Tennen, Todd, & Affleck, 2007; Chaplin et al., 2012; Cranford & Fairbairn, 2018; Lemke, Schutte, Brennan, & Moos, 2008). Taken together, there is much support for social problems acting as a mediator in the link between lower social cognition and increased risk for problematic drinking.

4. Social cognition and alcohol use disorder (AUD)

Social impairments are a central feature of AUD (APA, 2013), and individuals with AUD often have difficulty maintaining personal relationships. This tendency has led to speculation that such individuals may have diminished social cognition that would make it more difficult to

maintain adaptive social connections (e.g., Uekermann & Daum, 2008; Valmas, Mosher Ruiz, Gansler, Sawyer, & Oscar-Berman, 2014). In this section, we first review work showing that individuals with AUD show deficits in empathy, ToM, and emotion recognition compared to healthy controls. Similar to the section above, we summarize existing meta-analyses and systematic reviews on these topics when appropriate, rather than providing a narrative review of all individual studies. We then review brain regions and networks associated with social cognition to inform our discussion of the proposed mechanisms underlying these social cognition deficits in individuals with AUD. We end this section by reviewing work suggesting that inherited differences in the neuroanatomical network that comprises the social brain may act as a risk factor for offspring of individuals with AUD to develop AUD themselves.

4.1 Empathy

Massey et al. (2018) conducted a narrative review on social cognition and substance use more generally. They found that adults with AUD often demonstrated impairments in various facets of empathy (e.g., empathic concern, perspective-taking), as assessed by self-report questionnaires and behavioral tasks (Dethier & Blairy, 2012; Maurage et al., 2011). The findings were mixed, however, as two studies found no empathy deficits in individuals with AUD compared to healthy controls (Schmidt et al., 2016; Thoma, Winter, Juckel, & Roser, 2013). Kumar et al. (2022a) estimated the aggregated effect size of empathy deficits (assessed nearly exclusively with self-report questionnaires) in adults with AUD ($n = 349$) compared to healthy controls ($n = 365$; $k = 9$) and found that individuals with AUD reported significantly lower empathy than healthy controls with a moderate effect size (Hedge's $g = -0.53$). Age was found to be a significant moderator, such that increases in age corresponded to an increase in effect size, suggesting that empathy deficits may be particularly pronounced for older individuals with AUD. However, because of the correlation between lifetime alcohol consumption and chronological age, it is unclear whether it is age or quantity of alcohol consumed that is driving this effect. Further, empathy deficits were stronger for cognitive empathy (Hedges' $g = -0.44$) compared to affective empathy (Hedges' $g = -0.19$) in the six studies that examined both of these empathy components. These results suggest that individuals with AUD may be able to emotionally respond to or share in other people's emotional states but may have difficulty understanding them. However, only six studies examined these subcomponents of empathy, all but one of which used self-

report questionnaires, and more research is needed to more clearly understand potential differences in cognitive vs affective empathy deficits in AUD. Indeed, some researchers suggest that there may be a specific deficit in affective empathy in AUD, supporting the proposition of an "affect processing system" impairment in alcoholism (Ferrari, Smeraldi, Bottero, & Politi, 2014; Maurage et al., 2011). Finally, effect sizes were generally stronger when the EQ (Baron-Cohen & Wheelwright, 2004) ($k = 3$; Hedges' $g = -0.62$) was administered vs the IRI (Davis, 1980) ($k = 3$; Hedges' $g = -0.12$). These comparisons were made in a small number of studies, but the findings suggest that the EQ (Baron-Cohen & Wheelwright, 2004) may be a more sensitive measure for capturing differences in empathy between individuals with AUD and healthy controls. Future studies are indicated to explore whether some measures of empathy, or some subscales (e.g., the IRI empathic concern subscale; Davis, 1980) are more sensitive to detecting empathy differences between individuals with AUD and healthy controls.

In addition to this meta-analysis on adults with AUD compared to healthy controls, two recent longitudinal studies investigated cognitive and affective empathy, as assessed by the IRI (Davis, 1980), in adolescents in relation to substance use (yes/no) and response to social consequences of use (defined as recognition of substance use causing a problem and motivation to change substance use) (Winters et al., 2020, 2023). The samples for both of these studies were drawn from the NIDA-funded Drug Abuse Treatment Outcome Studies for Adolescents (DATOS-A; Kristiansen & Hubbard, 2001), which followed adolescents admitted to outpatient substance use treatment programs in six large American cities between November 1993 and November 1995. In the first study (Winters et al., 2020), DATOS-A adolescents ($n = 826$) completed assessments throughout treatment (i.e., at 1 month, 3 months, and 6 months) and 6 months post-treatment. Cross-lagged effects showed that increases in affective empathy, but not cognitive empathy, predicted lower substance use over time, suggesting that lower levels of affective empathy may indicate a developmental vulnerability for substance using behavior (Winters et al., 2020). In the second study, Winters et al. (2023) followed DATOS-A adolescents ($n = 3382$) during treatment and up to 12-months post-treatment. Cross-lagged effects showed that increases in cognitive empathy, but not affective empathy, predicted greater response to social consequences of use, which in turn predicted less substance use over time (Winters et al., 2023). These results suggest that adolescents with higher levels of cognitive empathy may be more likely to recognize

the social consequences of substance use, which in turn motivates them to reduce their substance use. Taken together, findings from these two studies indicate that affective empathy may be directly associated with substance use in adolescents in treatment for substance use, whereas cognitive empathy may be indirectly related to substance use through social response to substance use consequences.

4.2 Theory of mind

In two prior meta-analyses (Bora & Zorlu, 2017; Onuoha et al., 2016), individuals with AUD, compared to healthy controls, showed impairments in ToM assessed using a range of measurement techniques (e.g., questionnaires, semi-structured interviews, behavioral tasks). Specifically, across 8 studies, Onuoha et al. (2016) found that compared to healthy controls ($n = 187$), individuals with AUD ($n = 187$) showed deficits in ToM, indicative of a large effect size (Hedges' $g = -1.62$). Similarly, across 12 studies (7 of which were included in the Onuoha et al., 2016 meta-analysis), Bora and Zorlu (2017) found that individuals with AUD ($n = 317$) showed impairments in both the decoding (i.e., affective, as assessed by the RMET; Baron-Cohen et al., 2001), and reasoning (i.e., cognitive, as assessed by asking participants to infer the thoughts of others based on vignettes or videos) facets of ToM compared to healthy controls ($n = 298$), with medium to large effect sizes ($d = 0.46$ and $d = 0.72$ respectively). Meta-regression analyses showed that studies with a greater proportion of males (vs females) showed more severe ToM deficits (Bora & Zorlu, 2017), suggesting that males with AUD may be more vulnerable to ToM impairments. Interestingly, these ToM deficits were observed not only in studies that included recently (i.e., less than 8 weeks) detoxified individuals, but also in those studies that included participants who had been abstinent for longer periods of time (i.e., 8 weeks or more). These findings suggest that difficulties in recognizing the mental states of others may contribute significantly to interpersonal challenges experienced by individuals with AUD, even during periods of prolonged abstinence (Bora & Zorlu, 2017). Overall, these two meta-analyses demonstrate that individuals with AUD show reliable deficits in ToM compared to healthy controls.

4.3 Emotion recognition

Two meta-analyses demonstrated that individuals with AUD show impairments in emotion recognition compared to healthy controls (Bora & Zorlu, 2017; Castellano et al., 2015). Specifically, across 10 studies, Castellano et al. (2015) showed that individuals with AUD ($n = 276$) had

significantly lower overall facial emotion recognition ability as assessed by a variety of tasks (e.g., identification of specific emotions, differentiation between emotional expression intensities), compared to healthy controls ($n = 226$) with a medium effect size ($d = -0.67$). Bora and Zorlu (2017) extended these findings by additionally testing deficits for specific emotions. They showed across 12 studies that individuals with AUD ($n = 435$) demonstrated a significant deficit in facial emotion recognition with a large effect size ($d = 0.65$), when compared to healthy controls ($n = 377$), particularly for disgust ($d = 0.62$) and anger ($d = 0.49$), which are emotions linked to social threat (e.g., Gutiérrez-García & Calvo, 2017) and may have particular importance for interpersonal functioning in severe AUD (Pabst, Heeren, & Maurage, 2020). The effect sizes for deficits in recognizing other emotions (i.e., happiness, fear, surprised, sad) were more modest ($d = 0.19$–0.33), and the difference between AUD and healthy controls was not significant for recognition of happiness (Bora & Zorlu, 2017). Additionally, facial emotion recognition deficits were more pronounced in individuals with AUD who had a longer duration of alcohol abuse, a larger amount of daily use, and more severe depressive symptoms compared to those who had less alcohol involvement and fewer depressive symptoms (Bora & Zorlu, 2017). Similar to the ToM deficits found in individuals with AUD (as discussed above), these facial emotion recognition deficits were evident not only in studies that included individuals who were recently detoxified, but also in studies that included individuals who were abstinent for longer periods of time. This suggests that facial emotion recognition difficulties may also be a persistent problem for individuals with AUD even during periods of prolonged abstinence.

5. Mechanisms for deficits in social cognition in individuals with AUD

Mechanisms that have been proposed to explain social cognition deficits in individuals with AUD generally fall under one broad category related to how chronic and heavy alcohol use negatively affects the brain and cognitive processes (i.e., structural and functional brain abnormalities in regions related to social cognition and impairments in cognitive processes that subserve social cognition; see Fig. 1). Before reviewing work supporting this proposed mechanism, we first discuss neuroimaging studies that delineate areas of the brain important for social cognition.

5.1 Brain regions and networks associated with social cognition

The specific brain regions and circuits considered to be part of the neural underpinnings of the social brain differ among researchers. It is generally agreed that the frontal cortex including the orbitofrontal cortex (OFC), anterior cingulate cortex, the temporal cortex that includes the amygdala and the superior temporal sulcus, and the temporal–parietal junction are involved in social cognition (Adolphs, 2009; Mars, Sallet, Neubert, & Rushworth, 2013). The insula and fusiform cortices are also included in a number of prior reports (Adolphs, 2009; Kennedy & Adolphs, 2012).

This review of brain morphological and functional characteristics focuses on behavioral functioning that is generally considered to fall under social cognition. We recognize that other areas of functioning such as regulation of affect, including the tendency for impulsive behavior, are often related to social cognitive functioning. The ideal set of studies for connecting risk to brain regional or network differences evaluates either those with AUD or their high-risk relatives, using paradigms that simultaneously measure social cognition functioning and brain morphology (volume, surface area, cortical thickness) or functioning using functional magnetic resonance imaging (fMRI) to measure blood oxygenation-level dependent (BOLD) response or connectivity between regions. With limited studies currently available, this chapter will attempt to include reference to studies that relate social cognitive functioning to specific brain regions or networks. It will also review studies of brain morphology and functioning in those with either an AUD or their high-risk relatives. With these data in hand, inferences about regions/networks that may be damaged as a result of prolonged and heavy alcohol use, as well as those that may have relevance to social cognition as an etiological factor in the development of AUD, can be made.

Adolphs (2009) was among the first to point out that cortical regions in the temporal lobe are involved in perception of social stimuli while the OFC is especially involved in linking these stimuli to motivation, emotion, and cognition. More recent studies continue to find a relationship between OFC gray matter volume and social cognition in prenatally alcohol exposed offspring (De Water et al., 2021). The role of the amygdala in prefrontal circuits supporting social cognition continue to be investigated (Gangopadhyay, Chawla, Dal Monte, & Chang, 2021), with substantial

structural and functional support for its role in social decision-making. The observations have been made across species to include rodents and non-human primates (Gangopadhyay et al., 2021).

The anterior cingulate has been identified as a key region in social cognition (Apps, Rushworth, & Chang, 2016; Lockwood, 2016). Individuals with psychopathy, characterized by a severe lack of empathy, display gray matter differences in a set of brain regions and circuits that consistently include the anterior and posterior cingulate along with the orbitofrontal cortex, amygdala and paralimbic structures (Anderson & Kiehl, 2012). Using data from lesion studies, neuroimaging, and single unit recording in humans and in non-human primates, researchers have proposed that a cytoarchitectonic distinction can be made in the dorsal/ventral dimension between the ACC sulcal and gyral regions (Apps et al., 2016). The gyral portion appears to be most influential in social cognition.

Individuals with autism, typically characterized by impairments in social cognition, who show developmental failure involving the amygdala also show alterations in the fusiform cortex that provide the neural substrate for social perception (Schultz, 2005). This lateral portion of the fusiform cortex in the ventral temporal lobe has been termed the "fusiform face area." It is hypothesized that social perception and social cognition that are normally linked in development are not linked in those who have autism, preventing them from acquiring social skills (Schultz, 2005). Finally, some have proposed a social network context model that includes the insula, known as the frontoinsular temporal network, to account for variation in human empathy (Melloni, Lopez, & Ibanez, 2014). The insula, located deep within the sylvian fissure, has multiple functions including vestibular and chemosensory functions such as emotional processing, and visceral sensations including pain (Uddin, Nomi, Hébert-Seropian, Ghaziri, & Boucher, 2017).

Now that we have reviewed areas and networks of the brain thought to be important for social cognition, we next turn to research suggesting neurotoxicity as a mechanism explaining why individuals with AUD show deficits in social cognition.

5.2 Effects of chronic alcohol use on the brain

Deficits in social cognition in individuals with AUD are often interpreted to be a result of neurotoxic effects of chronic and heavy alcohol use on the brain, particularly in areas related to social cognition (e.g., Bora & Zorlu, 2017; Le Berre, 2019; Massey et al., 2018). Individuals with AUD have

long been known to show variation from healthy controls in many brain regions. Indeed, chronic alcohol consumption is associated with severe and multiple neurocognitive problems (e.g., memory and executive functioning deficits), including abnormalities in prefrontal and limbic brain regions (Bosco, Capozzi, Colle, Marostica, & Tirassa, 2014; Durazzo, Gazdzinski, Yeh, & Meyerhoff, 2008; Oscar-Berman et al., 2014; Rupp et al., 2006; Volkow, Wang, Fowler, Tomasi, & Telang, 2011) important for social cognition (e.g., Grossmann, 2013; Gur & Gur, 2016; Rajmohan & Mohandas, 2007). Gray matter loss in both cortical and subcortical regions (Chye et al., 2020; Mackey et al., 2019), as well as volumetric reductions of the OFC and amygdala, all of which are important for social cognition (as reviewed above), have been reported in individuals with AUD compared to healthy controls (Makris et al., 2008; Zou, Durazzo, & Meyerhoff, 2018). Additionally, the Enhancing NeuroImaging Genetics through Meta-Analysis (ENIGMA) consortium on substance abuse analyzed 3905 individuals with alcohol, nicotine, methamphetamine, or cocaine dependence vs controls and found gray matter changes in many brain areas, but mainly in the hippocampus, thalamus, putamen, and amygdala (Chye et al., 2020). Many of these regions are critical for processing social information, including emotional and social cues, and their dysfunction can impair social cognition abilities. Importantly, these neuroanatomical abnormalities appear to contribute to persistent deficits in social cognition in individuals with AUD who are in remission (Bora & Zorlu, 2017). Taken together, a large body of work indicates differences in areas of the brain subserving social cognition across individuals with AUD and healthy controls, and these differences are thought to result from heavy and prolonged alcohol use. It is important to keep in mind, though, that alcohol related damage to the brain is widespread (i.e., not specific to areas linked to social cognition) and that the social cognition network encompasses regions with many other functional associations, such that damage to these regions has widespread consequences beyond social cognition.

5.3 Heritable brain characteristics

As shown in Fig. 1, in addition to environmental factors (e.g., social learning, early life adversity) that increase risk for lower social cognition (e.g., Rokita et al., 2018; Striano & Reid, 2006), inherited differences in the neuroanatomical network that comprises the social brain may act as a risk factor for offspring of individuals with AUD to develop AUD themselves. High-risk offspring from families with AUD show atypical neural

activation when viewing emotional faces (Glahn, Lovallo, & Fox, 2007; Hill et al., 2007; Hulvershorn et al., 2013; Peraza, Cservenka, Herting, & Nagel, 2015). In addition, neural activation to emotionally valenced words among high-risk offspring is related to problem drinking in adolescence (Heitzeg, Nigg, Yau, Zubieta, & Zucker, 2008). High risk offspring with a family history of AUD have been shown to have atypical structure and function of brain regions involved in executive processing, regulation of affect, decision making, and social cognition (Hill & O'Brien, 2015; Tessner & Hill, 2010). Compared to controls from low-risk families, adolescent and young adult offspring with a family history of AUD show volumetric reductions in the right hemisphere of the orbitofrontal cortex (OFC) (Hill et al., 2009) and the amygdala (Benegal, Antony, Venkatasubramanian, & Jayakumar, 2007; Dager et al., 2015; Hill et al., 2001, 2013), areas important for social cognition. These results have been observed in samples where either the majority of cases had not yet developed a substance use disorder (SUD) (Dager et al., 2015; Hill et al., 2001), were alcohol-naïve (Benegal et al., 2007), or the reduction in volume was seen even when cases with SUD were eliminated from data analysis (Hill et al., 2013). Interestingly, the effects of OFC volume on impulsivity appears to be influenced by the lateralization of volume with the Right/Left OFC volume predictive of SUD outcome (Hill et al., 2009). This finding was recently replicated in a study finding the Right/Left OFC ratio predictive of impulsivity (Elliott, Esmail, Weiner, & Johnson, 2023). In a longitudinal investigation, volume of the OFC in relation to amygdala volume was predictive of SUD outcome (O'Brien & Hill, 2017). Additionally, in a study of 98 young adults who were either from families with multiple cases of AUD or from control families with minimal problematic alcohol use, the high-risk offspring showed reduction of gray matter volume in the insula, fusiform cortex, and inferior temporal regions (Hill & Sharma, 2019), regions that are thought to be critical for social cognition (e.g., Campanella, West, Corradi-Dell'Acqua, & Skrap, 2022; Chen et al., 2022; Fan, Duncan, de Greck, & Northoff, 2011; Melloni et al., 2014; Schultz, 2005; Vucurovic et al., 2023).

Interestingly, the reduction of volume in the above-mentioned areas was associated with DRD2 methylation. In general, methylation is inversely related to gene expression so these results suggest that lesser DRD2 expression may be seen in those individuals with a family history of AUD. This epigenetic change may be the result of alcohol use in parents that can be transmitted across generations. Analysis of blood samples drawn

in offspring from densely affected families has also shown epigenetic alterations in an oncogene and a tumor suppressor gene (Hill, Rompala, Homanics, & Zezza, 2017). These changes appear to not be the direct effect of alcohol use in individuals whose samples were analyzed due to their young age and minimal exposure to alcohol and other drugs. The origin of epigenetic changes associated with a family history of AUD may be specific to genes directly associated with AUD susceptibility such as alcohol metabolizing enzymes (alcohol dehydrogenase), but epigenetic changes in other genes may result in alterations that may produce changes in neural circuitry that then predisposes the individual to greater susceptibility for problematic alcohol use and AUD. Based on observed differences in individuals with a family history of AUD who have not experienced substantial exposure to alcohol and drugs, it may be concluded that individuals can inherit neural alterations that may predispose them to developing problematic use of alcohol or other drugs through epigenetic changes transmitted across generations.

Taken together, the current evidence suggests that chronic and heavy alcohol use in individuals with AUD results in brain abnormalities that cause social cognition deficits. Additionally, many of these brain abnormalities may be passed down to offspring, increasing their risk to develop AUD themselves, providing further understanding of why AUD patterns may be shared in families. However, further research is necessary, particularly neuroimaging studies that focus on delineating specific brain regions associated with long term consequences of heavy alcohol use or that could aid in the identification of those at risk for developing AUD (as we discuss in more detail below).

6. Recommendations for future studies

In this chapter, we presented a theoretical model that organizes the literature on social cognition and problematic alcohol use, suggesting that deficits in social cognition may predispose individuals to alcohol problems and that chronic and heavy alcohol use may exacerbate these deficits. We also detailed several mechanisms to explain why lower social cognition may be a risk factor for problematic alcohol use and why prolonged heavy alcohol use may result in social cognition deficits. While promising, this budding area of research has several limitations that will need to be addressed in future studies. These limitations include issues in how social

cognitive abilities have been conceptualized and assessed, study design issues (namely, the heavy reliance on cross-sectional/observation studies), the need for more studies exploring underlying mechanisms linking social cognition to problematic alcohol use, limited external validity of existing studies leading to poor generalizability of findings, inadequate consideration of potential moderators, and comorbid psychopathology. In this section, we discuss these limitations and suggest directions for future research that will help to enhance our understanding of the interplay between social cognition and problematic alcohol use.

6.1 Definitions and measures of social cognition

As discussed above, inconsistent and overlapping definitions of social cognitive abilities, problems with measures commonly used to assess them (e.g., low construct, discriminant, and ecological validity, failure to consider the multi-dimensional nature of social cognitive abilities), and the relative neglect of social cognitive abilities beyond empathy, ToM, and emotion recognition have surely impeded progress in this area. We make the following three recommendations to help advance this literature. First, and most importantly, we believe more work is needed to demonstrate construct validity (the extent to which a measure accurately assesses what it's supposed to measure), convergent validity (how closely a measure is related to other measures that assess the same or similar constructs), and discriminant validity (the extent to which a test is not related to other measures that assess different constructs) of social cognition measures. The evidence that is available suggests concerns with these types of validity, at least for some social cognition measures. As we noted above, researchers have questioned the construct validity of a widely used task, the RMET (Baron-Cohen et al., 2001), to assess ToM (Kittel et al., 2022; Oakley et al., 2016; Pabst et al., 2022; Quesque & Rossetti, 2020), as well as two widely used self-report measures, the IRI (Davis, 1980) and EQ (Baron-Cohen & Wheelwright, 2004), to assess empathy (Murphy et al., 2020; Pabst & Maurage, 2023). Some researchers have suggested, and we agree, that until consensus on the conceptualization and measurement of empathy and related constructs are reached, it is better to bypass the terms empathy and ToM and instead focus on lower-level constructs (e.g., perspective-taking, feelings of concern towards other' distress, emotion recognition) that are clearer and more specific to the content of the assessment tools used (Hall & Schwartz, 2019; Pabst & Maurage, 2023).

In general, more studies that test for construct, discriminant, and convergent validity of social cognition measures are needed, particularly within the same sample of participants and with an expanded consideration of constructs/measures. Large studies are needed that administer several different measures of social cognitive abilities to individuals with varying levels of alcohol involvement, including ones that are not often administrated in alcohol research (e.g., social perception, social knowledge, and attributional biases), as well as measures of related constructs (e.g., social anxiety; Pittelkow, aan het Rot, Seidel, Feyel, & Roest, 2021), to firmly establish construct, discriminant, and convergent validity of social cognitive abilities within the same sample of individuals. We believe this work to be critical to advance our understanding of the role of social cognition in problematic alcohol use.

Second, we recommend that alcohol researchers consistently assess cognitive vs affective empathy, as well as cognitive vs affective ToM, using both trait and state measures, in order to clarify what may be driving associations with problematic alcohol use. When studies do assess empathy and ToM in a multidimensional way and relate these various social cognitive abilities to problematic alcohol use, differences are often found, suggesting that these components can and should be differentiated. Further, while we are aware of only one study that differentiated between trait vs state empathy when examining associations with alcohol use (Kumar et al., 2023), the broader literature on social cognition demonstrates that this distinction is critical (e.g., Stellar & Duong, 2023), and more alcohol research is needed that considers state vs trait measures of social cognitive abilities.

Finally, more ecologically valid measures and paradigms are needed to fully understand the role of social cognition in problematic alcohol use. In general, social cognition tasks lack ecological validity since they exclusively use third-person paradigms, in which participants are asked to merely observe others, who are displayed in pictures or videos or who are described in writing, when making inferences (Creswell & Kumar, 2023; Pabst & Maurage, 2023; Pabst et al., 2022). Paradigms that require participants to actually interact with others when making inferences about their emotions and mental states would go a long way in increasing ecological validity of such tasks (see also Creswell & Kumar, 2023; Pabst et al., 2022; Schilbach et al., 2013; Wu & Keysar, 2007). We make specific recommendations for how this might be achieved below.

6.2 Study designs and mechanisms

Meta-analyses consistently demonstrate that lower social cognition is linked to greater alcohol use and alcohol problems in non-clinical samples (i.e., Kumar et al., 2022a, 2022b) and that individuals with AUD show deficits in social cognition compared to healthy controls (i.e., Bora & Zorlu, 2017; Kumar et al., 2022a; Onuoha et al., 2016). One overarching limitation of this literature is that much of our knowledge comes from cross-sectional/correlational studies, which cannot address third variable problems, establish temporal precedence or causality, or provide strong support for underlying mechanisms. Given reliable associations, more rigorous study designs are now needed to clarify whether lower social cognition actually leads to increases in alcohol consumption and alcohol problems, and whether the social cognition deficits seen in individuals with AUD result from the neurotoxic effects of chronic and heavy alcohol use.

Several study designs would be helpful to test whether deficits in social cognition predict the subsequent development of alcohol problems. Prospective longitudinal studies would be useful to determine whether individuals with lower social cognition are more likely to escalate their drinking over time and develop alcohol problems compared to individuals with higher social cognition. EMA studies that examine social cognition, proposed mechanisms (i.e., peer pressure, social facilitation, interpersonal problems), and alcohol use and problems in individuals' daily lives can additionally help clarify the temporal ordering of these associations, identify mediating mechanisms, and speak to potential differential associations between state vs trait social cognition and alcohol outcomes (e.g., Kumar et al., 2023). These types of studies are critical to demonstrate the temporal precedence of lower social cognition in relation to problematic alcohol use.

In addition to longitudinal research, experimental studies that manipulate social cognition levels and measure alcohol consumption would provide particularly compelling evidence to establish a causal relationship between lower social cognition and heavier alcohol use. We are not aware of any prior studies that have attempted to do this, but we just began data collection in our lab in order to fill this critical knowledge gap. Specifically, in a 2 (low vs high empathy) × 2 (social drinking context vs drinking alone) between-subjects design with 152 young adult (ages 21–25) social drinkers, we aim to (1) test whether experimentally reducing (vs increasing) empathy using adaptations of validated empathy manipulations (Batson et al., 1997, 2003; Davis et al., 2004; Van Boven & Loewenstein, 2003) leads to an

increase in alcohol craving and alcohol consumption, particularly in social (vs alone) drinking contexts, and (2) determine whether two theoretically-relevant mechanisms (i.e., higher social and conformity drinking motives) explain these results, especially in social drinking contexts. This study will be the first to determine whether deficits in empathy are causally linked to alcohol use and why (i.e., due to increased social and conformity drinking motives). Importantly, we adapted commonly used empathy manipulations to increase their ecological validity, with participants believing the story they read and responded to was written by another participant in the lab with whom they would soon interact. Findings from this study will inform etiological theories of AUD that focus on lower social cognition and may suggest a novel target for intervention.

To identify whether enhanced social facilitation from alcohol at least partly explains why lower social cognition predicts heavier alcohol consumption and more alcohol problems, more experimental research is also needed to determine whether acute alcohol intoxication actually increases social cognitive abilities for those with lower social cognition. As reviewed above, some initial studies have demonstrated that acute alcohol intoxication has the ability to improve some social cognitive abilities (i.e., affective empathy, ToM, common ground; Dolder et al., 2017; Garrison et al., 2022; Johnson et al., 2018), and one prior study that tested for an alcohol beverage by trait empathy interaction found that alcohol's effects on increasing affective empathy were particularly strong for individuals with lower trait empathy (Dolder et al., 2017). More studies are needed to test this hypothesized interaction, though, as the Dolder et al. (2017) study is the only prior alcohol administration study that has done so, to our knowledge. More ecologically valid alcohol administration studies that use more ecologically valid social cognition measures are also needed to fully understand alcohol's effects on social cognition. With the exception of one study (Garrison et al., 2022), all prior studies on this topic required participants, the majority of whom were described as being social drinkers, to consume alcohol while alone in a laboratory room and then complete questionnaires or behavioral tasks assessing social cognition (also while alone) using third-person paradigms. Alcohol administration studies that utilize group drinking protocols and assess social cognition within this social drinking context using second-person tasks will likely permit a fuller understanding of alcohol's effects on social cognition, as well as whether the effects are stronger for those with lower trait social cognitive abilities. Indeed, in contrast to alcohol administration studies that test participants in

isolation, group alcohol administration studies have provided robust support for alcohol's rewarding social (e.g., increased social bonding) and emotional effects (e.g., increased positive affect; Fairbairn & Sayette, 2014; Fairbairn et al., 2018; Sayette et al., 2012) and have demonstrated that individuals with particular personality and genetic risk factors for AUD experience enhanced reward from alcohol in these social settings (Creswell et al., 2012; Fairbairn et al., 2015). These laboratory social drinking paradigms could similarly be used to test whether alcohol increases empathy, ToM, and emotion recognition using self-report and observational measures in order to capture social cognition abilities in real-time and in relation to other drinking participants in the lab (e.g., by coding language related to empathy and ToM while participants are drinking together and interacting). Given that most alcohol use is consumed in social settings, and social cognitive abilities are only relevant in relation to other individuals, studies that include social settings and use second-person social cognition tasks seem critical in order to fully understand alcohol's effects on social cognition and vice versa. These types of studies would address the concerns raised about poor ecological validity of current social cognition tasks (e.g., Creswell & Kumar, 2023; Kittel et al., 2022; Pabst et al., 2022; Sunahara et al., 2022), as well as alcohol administration paradigms requiring social drinking participants to drink alone (Creswell et al., 2012; Sayette et al., 2012).

Finally, longitudinal studies that include neuroimaging methods and retest the same group of individuals with AUD, in comparison to healthy controls, across time would be particularly informative for assessing within-individual changes in social cognitive abilities that might occur with prolonged abstinence or continued heavy use. These studies would help identify the specific brain regions associated with impairments in particular subparts of social cognition. Additionally, they would allow researchers to identify the trajectories of social cognition abilities in individuals with AUD and determine the factors that may influence these trajectories, including potential improvements with abstinence or further deterioration with continued heavy drinking. Such evidence would provide compelling support for the proposition that chronic and heavy alcohol use results in social cognition deficits due to the neurotoxic effects of alcohol, as well as delineating the potential time course for improvements after continued abstinence, which might be a potent motivator for individuals in AUD treatment. Future research with an expanded range of ecologically valid experimental paradigms, and age-appropriate measures of social cognitive

abilities (e.g., first-order and second-order ToM), are also needed in order to determine the extent of social cognition deficits in high-risk offspring. As discussed above, results to date suggest that structural alterations in regions involved in networks associated with social cognition may promote impairment in social cognition, potentially setting the stage for problematic drinking (e.g., Hill & O'Brien, 2015; Tessner & Hill, 2010). Neuroimaging studies would be particularly valuable here to help identify brain precursors for the development of AUD. Integration of both genetic and epigenetic factors with alterations in brain regions involved in the circuits of social cognition are needed to provide opportunities for intervention and prevention of problematic drinking across generations. In general, results from longitudinal, EMA, and experimental studies that use ecologically valid testing paradigms will help to clarify the strength, direction, and underlying mechanisms of the associations between social cognition deficits and alcohol problems. These types of more rigorous study designs (vs cross-sectional/observational studies) are now needed to move this area forward.

6.3 Generalizability

Another important area for future research is to expand the participant pools to include individuals from more diverse backgrounds. The vast majority of studies exploring the link between social cognition and problematic alcohol use have primarily included white participants (see Bora & Zorlu, 2017; Castellano et al., 2015; Kumar, et al., 2022a, 2022b). This may limit the generalizability of findings to people of color. Further, the relationship between social cognition and alcohol use may vary across cultures, as cultural norms and values can influence both social cognition and alcohol consumption patterns. For example, some cultures prioritize collectivism and social harmony over individualism, which may affect how social-cognitive processes are valued and expressed (Hong & Chiu, 2001; Vogeley & Roepstorff, 2009). Additionally, cultural differences in alcohol consumption patterns, such as drinking in moderation vs binge drinking (Castro, Barrera, Mena, & Aguirre, 2014), may also impact the relationship between social cognition and problematic alcohol use. We are not aware of any studies that have compared associations between social cognition and problematic alcohol use across various cultures. In general, more research is needed on diverse populations, as these studies can provide insight into whether and how social cognition deficits are associated with problematic alcohol use across different races/ethnicities, cultures, and groups.

6.4 Moderating variables

More studies are also needed to explore the potential moderating influence of sex and age on the link between social cognition deficits and problematic alcohol use.

Bora and Zorlu (2017) reported sex differences in ToM deficits in individuals with AUD compared to healthy controls, such that deficits were more pronounced in males than females, but study-level moderation by sex does not necessarily reflect within-study moderation by sex (see Berlin, Santanna, Schmid, Szczech, & Feldman, 2002; Reade, Delaney, Bailey, & Angus, 2008). Research suggests that males and females differ in their social cognition abilities, with females typically showing higher social cognitive abilities than males in adolescence (e.g., Devine & Hughes, 2013; Hall, 1978; Hanson & Mullis, 1985; McClure, 2000; Thompson & Voyer, 2014) and adulthood (e.g., Adenzato et al., 2017; Baron-Cohen & Wheelwright, 2004; Davis, 1983; Thompson & Voyer, 2014), and differ in their alcohol consumption, with males typically consuming more alcohol on average than females (Centers for Disease Control & Prevention, 2022). Future research that tests for within-study sex differences are needed to provide more conclusive evidence about potential sex differences in the association between social cognition deficits and problematic alcohol use. Such evidence might help inform targeted interventions and prevention strategies to address social cognition deficits and problematic drinking behaviors in a sex-specific manner.

Studies are also needed to explore potential age-related differences in the link between social cognition and problematic alcohol use. A meta-analysis found a stronger deficit in empathy in individuals with AUD vs healthy controls as age increased (Kumar et al., 2022a), but it is unclear whether it was age or quantity of alcohol consumed across the lifetime that was driving this effect. Interestingly, another meta-analysis found less emotion recognition impairment in older adults (i.e., mean age > 40) compared to younger adults with AUD (Castellano et al., 2015). But, again, study-level moderation by age in these two meta-analyses may not reflect within-study moderation by age, and more studies investigating the latter are needed. Additionally, studies are needed to investigate social cognitive abilities in adolescents and young adults with AUD compared to age-matched healthy controls, as existing studies have almost exclusively focused on middle and older adults. In general, more research is needed to fully understand potential moderating variables, such as sex and age, in the association between social cognitive deficits and problematic alcohol use.

6.5 Comorbid psychopathology

Social cognition deficits are associated with mental health conditions that also co-occur with AUD, including internalizing disorders (e.g., depression and anxiety disorders; Plana, Lavoie, Battaglia, & Achim, 2014; Weightman, Air, & Baune, 2014) and externalizing disorders (e.g., antisocial and borderline personality disorders, attention-deficit hyperactivity disorder; Bora & Pantelis, 2016; Harari, Shamay-Tsoory, Ravid, & Levkovitz, 2010; McKinley, Patrick, & Verona, 2018), some of which are associated with clinically low levels of empathy (e.g., callous–unemotional traits present in conduct disorder; Frick & Myers, 2018). For instance, depression has been associated with deficits and differences in emotion recognition, empathy, and ToM abilities (Ladegaard, Larsen, Videbech, & Lysaker, 2014; Weightman et al., 2014). Depression can lead to a negative bias in social cognition processing, making it difficult for individuals to accurately perceive social cues and make appropriate social judgments (Weightman et al., 2014). Similarly, anxiety disorders, such as social anxiety disorder, have been linked to deficits in empathy, ToM, and social perception (Alvi, Kumar, & Tabak, 2022; Gkika, Wittkowski, & Wells, 2018). Individuals with depression and social anxiety may avoid social situations or rely on alcohol to cope with their symptoms of depression and social anxiety, which can lead to problems with social cognition and alcohol misuse (APA, 2013; Gkika et al., 2018; Trew, 2011). Notably, in a study that controlled for depression and anxiety symptoms, individuals with severe AUD did not demonstrate a general emotion decoding deficit on the Facial Emotion Recognition Test (Gaudelus et al., 2014), but rather only showed a specific deficit for disgust/contempt (Maurage et al., 2021).

Taken together, these findings suggest that comorbid mental health conditions are important to consider when examining associations between social cognition definitions and problematic alcohol use. Generally, though, studies investigating social cognition deficits in individuals with AUD compared to healthy controls exclude individuals with comorbid substance use disorders and psychiatric disorders, such as psychosis and severe cognitive impairment (see Bora & Zorlu, 2017; Castellano et al., 2015; Kumar et al., 2022a). These studies additionally fail to provide information on other significant comorbidities (e.g., depression, social anxiety disorders), which could aid in enhancing knowledge about the relationship between social cognition and AUD. Investigating the impact of comorbid disorders is an essential area for future research on social

cognition and problematic alcohol use, especially for developing effective prevention and intervention strategies. For instance, comorbid conditions could worsen social cognition deficits in individuals with AUD, or individuals with comorbidities could show distinct patterns of social cognition deficits compared to those with AUD alone (e.g., Maurage et al., 2021). If such evidence exists, these findings could help in creating targeted interventions that address the specific social cognition deficits in individuals with AUD and co-occurring mental health conditions, thus enhancing treatment outcomes and quality of life for these individuals.

7. Conclusions and implications

In this chapter, we provided an organizing theoretical framework suggesting that deficits in social cognition are both a risk factor for and consequence of AUD. We reviewed several meta-analyses showing reliable associations between lower social cognitive abilities and problematic alcohol use in non-clinical samples, and reliable social cognition deficits in individuals with AUD compared to healthy controls, and we suggested potential underlying mechanisms explaining these associations. We detailed several methodological implications of this framework and made suggestions for future work. Specifically, prospective and experimental studies that use valid social cognition measures are needed to clarify the temporal ordering of these effects (i.e., whether deficits in social cognition predict the emergence of problematic alcohol use and whether chronic and heavy alcohol use seen in AUD exacerbates these deficits) and to delineate underlying mechanisms. If more rigorous studies demonstrate that impairments in social cognition are a risk factor for AUD, this would have both conceptual implications (informing etiological theories of AUD that focus on social processes) and clinical implications (suggesting that early detection and intervention with individuals who show deficits in social cognition may be an effective approach for preventing or reducing problematic alcohol use). In fact, research suggests that substance use prevention programs that target the improvement of social skills, which heavily rely on social cognitive abilities, are more effective in reducing substance use in young people compared to other school-based substance use prevention programs (Midford, 2010). Similarly, if impairments in social cognition result from chronic and heavy alcohol use and underlie some of the social and interpersonal dysfunctions found in AUD, this

would also suggest a target for treatment. Indeed, empathy, ToM, and emotion recognition are modifiable social cognitive abilities (Abramson et al., 2020; Batt-Rawden, Chisolm, Anton, & Flickinger, 2013; Vass, Fekete, Simon, & Simon, 2018) and interventions that improve empathy have been shown to reduce a range of problematic behavior, including intimate partner violence (Romero-Martínez, Lila, Martínez, Pedrón-Rico, & Moya-Albiol, 2016), bullying (Şahin, 2012) and aggression (Feshbach & Feshbach, 1982). Interventions aimed at ToM and emotion recognition typically focus on individuals with certain mental health disorders, showing improvements in those with schizophrenia and autism (Fletcher-Watson, McConnell, Manola, & McConachie, 2014; Vass et al., 2018). Notably, a recent study found that an intervention that increased empathy resulted in better alcohol abstinence self-efficacy in individuals with AUD (Yang & Kim, 2021). Therefore, treatments that aim to improve empathic abilities in individuals with AUD may result in better short- and long-term outcomes related to both interpersonal problems and alcohol use. In other words, interventions targeting empathy and other social cognitive abilities may potentially be helpful to improve the quality of life of individuals with AUD. In summary, this chapter highlights the importance of considering social cognition in the study of problematic alcohol use and suggests that targeting social cognition abilities may be a promising approach to prevent and treat AUD.

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