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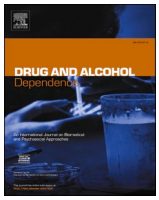

Review

Association between risk propensity and substance use: A multilevel meta-analysis

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ABSTRACT

Background: Substance use and its related disorders represent a significant global health concern. Risk propensity has been identified as a factor influencing substance use.
Methods: Multilevel meta-analysis was applied to quantitatively synthesise studies examining the associations between risk propensity and substance use and to identify potential factors that influence these relationships.
Results: A total of 323 effect sizes, 186 independent samples, and 148 studies were included in this meta-analysis. The results showed that risk propensity was statistically positively associated with substance use ($r = 0.116$), as well as with all three main types of substances, including tobacco ($r = 0.092$), alcohol ($r = 0.114$), and drugs ($r = 0.115$). Such a link was not significantly different across substance types. Moderator analyses showed that the type of risk propensity assessments and age of the study samples moderated the association between risk propensity and one or more types of substance use.
Conclusions: The findings underscore the importance of considering the role of risk propensity in the initiation of substance use and developing early interventions to prevent substance use. Future interventions could consider early detection and targeted intervention for young people with a heightened risk propensity.

1. Introduction

Risk propensity refers to the general tendency to respond more positively to uncertain situations that involve a trade-off between positive and negative outcomes (Figner and Weber, 2011). Psychoactive substances are defined as "a drug or other substance that affects how the brain works and causes changes in mood, awareness, thoughts, feelings, or behaviour" (National Cancer Institute, n.d.). Tobacco and alcohol are the most prevalent psychoactive substances, while various illicit drugs are being increasingly used (United Nations Office on Drugs and Crime, 2023).

Understanding the risk factors for substance use and disorders is crucial for the detection and intervention of substance use. Susceptibility to substance use and the development of substance use disorders involve a complex interplay of genetic and biological factors (Merikangas and McClair, 2012; Prom-Wormley et al., 2017) and social-environmental influences (Henneberger et al., 2021; Mennis et al., 2016).

While both genetic-biological and social-environmental factors can be difficult to assess, monitor, and alter, one mediating factor between the social/genetic/biological factors and substance use can be individuals' dispositions (e.g., personality). Individuals' dispositions involve specific patterns of responding and behaving in various situations and are shaped by genetic, biological, environmental, and social factors. They may play essential roles in substance use, its escalation, and the development of substance use disorders. Previous studies have identified various dispositional or attitudinal characteristics that can be linked to substance use (Hartman et al., 2013; Kotov et al., 2010). For example, a meta-analysis by Kotov et al. (2010) revealed significant associations between personality traits—e.g., high neuroticism, high disinhibition, low conscientiousness—and substance use disorders. A longitudinal study by Hartman et al. (2013) revealed that greater novelty-seeking and lower persistence are associated with greater cigarette use, alcohol consumption, and illicit substance use at an earlier age.

Most of the traits identified in the literature overlap with and point to

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an individual's risk propensity, which reflects one's attitudes towards risk. Risk attitudes involve cognitive (e.g., risk perception), emotional (e.g., risk tolerance), and behavioural (e.g., risky behaviours) responses to risk. The spectrum of risk attitudes ranges from "risk aversion" to "risk seeking" (Hillson and Murray-Webster, 2017). The literature has various definitions of risk attitudes and risk propensities (Highhouse et al., 2022).

The links between risk propensity and substance use have been explored in many empirical studies, but the findings are mixed. Some studies have reported a positive correlation between risk propensity and substance use such as smoking (Lejuez et al., 2005; Lejuez et al., 2003; Weiss et al., 2019), alcohol use (Edge and Oliver, 2019; Lejuez et al., 2002), cannabis use (Keyes et al., 2015), and polysubstance use (Hopko et al., 2006). However, some studies did not find a significant link (Dean et al., 2011) or even a negative link (Ashenhurst et al., 2011; Raymond et al., 2020). It is currently unclear how risk propensity is associated with substance use and its disorders and what factors can influence these associations. Understanding the role of risk propensity will benefit our understanding of the mechanisms and the potential interplay of biological/social factors on substance use. As risk propensity is more observable and assessable, it can be a useful construct in preventing, detecting early, monitoring, and intervening substance use.

Thus, the primary purpose of this study was to evaluate the associations between risk propensity and substance use as well as disorders using a meta-analysis. We also explored potential factors that can alter the associations to examine further potential mechanisms underlying the link between risk propensity and substance use.

1.1. Substance types

This review addresses three main types of substances: tobacco, alcohol, and drugs (including caffeine). Previous research suggests that the association between risk propensity and substance use may differ across substance types; however, the findings are inconclusive. For example, some studies (Aklin et al., 2005; Bornovalova et al., 2005) have reported a moderate correlation between risk propensity and drug use, contrasting with weaker associations observed with tobacco or alcohol use. In contrast, other research (Ayvasik and Sümer, 2010; de Water et al., 2017) has demonstrated similarly weak correlations between risk propensity and the use of all three substances. Given the inconclusive findings of prior research, we aimed to investigate whether the type of substance moderates the association between risk propensity and substance use.

Prior research has underscored that risk propensity in relation to substance use may differ across the levels of severity of substance use; however, the findings are mixed. For example, high alcohol and tobacco use, together with problem severity, has a negative correlation with risk propensity (Ashenhurst et al., 2011; Ryan et al., 2013). In contrast, some studies suggest that risk propensity may be more strongly associated with substance use than with misuse or dependence (Merline et al., 2008; Stamates and Lau-Barraco, 2017). In the current review, we investigate whether the severity of substance use moderates the relationship between risk propensity and substance use.

1.2. Assessments of substance use

Most clinical diagnoses of substance-related disorders are based on DSM and ICD classification, whereas a small number of studies have used chart reviews and clinical records to evaluate substance use. Self-reported assessments are also often used to assess substance use (Stewart et al., 2023). Examples are the Fagerstrom Test for Nicotine Dependence (Heatherton et al., 1991), the Modified Fagerstrom Tolerance Questionnaire (mFTQ; Prokhorov et al., 1996), the Alcohol Use Disorder Identification Test (AUDIT; Saunders et al., 1993), and the Michigan Alcohol Screening Test (Selzer, 1971). Some studies also assessed substance use with a single item (e.g., "Do you currently smoke?"). The different types of assessments involved different informants (clinician judgement vs. self-rating), settings (clinical practice vs. research), and assumptions of the measured construct (categorical vs. continuous) (Samuel et al., 2016). These differences could contribute to the heterogeneity in past studies assessing the relationship between risk propensity and substance use. Thus, we examined whether variations in the tools for assessing substance use moderate the relationship between risk propensity and substance use.

1.3. Assessment of risk propensity

Previous studies have employed diverse measurements to assess risk propensity. The assessments can be divided into two main paradigms: behavioural tasks and self-report questionnaires. Behavioural tasks are usually performed in laboratory settings and encompass abstract, gamified tasks in which respondents choose a specific action or given option. The commonly used tasks include Balloon Analogue Risk Task (BART; Lejuez et al., 2002), Iowa Gambling Task (IGT; Bechara et al., 1994), Cambridge Gambling Task (CGT; Rogers et al., 1999), and Columbia Card Task (CCT; Figner et al., 2009). Self-report questionnaires involve respondents self-reporting their responses to one or more statements that reflect risk propensity. Popular scales include the Domain-Specific Risk-Taking Scale (DOSPERT; Weber et al., 2002), and the Conformity to Masculine Norms Inventory-Risk-Taking subscale (Mahalik et al., 2003).

Frey et al. (2017)'s large-scale study demonstrated a weak association between the two different types of measures. The weak association can be either due to measurement errors or the types of risk propensity constructs that might be captured. Behavioural tasks capture participants' choice behaviours in settings where the reward and loss are usually points or hypothetical momentary incentives. However, statements in self-report scales may capture wider manifestations of risk attitudes, such as feelings, and can involve more complex real-life benefits and costs (e.g., social evaluation) (Frey et al., 2017). Thus, we examined whether the heterogeneity of the links between risk propensity and substance use could be due to the different types of assessments that were employed.

1.4. Sample characteristics

Previous studies had samples varying in the type of the target population (e.g., adolescents versus adults; clinical versus community samples). It is currently unclear whether the link between risk propensity and substance use remains stable across one's lifespan or is established early on. Adolescence is a crucial period associated with an increased risk of developing substance use behaviours. Given the characteristics of adolescents, including impulsivity and risky decision-making, individuals in these age groups are particularly vulnerable to substance use due to ongoing brain development, rapid increases in the consumption of substances, rapid recurrence after treatment, and a greater risk of lifetime substance use (Casey et al., 2011; Chassin et al., 2010; Lopes-Rosa et al., 2017; Volkow et al., 2021). On the other hand, older adults' substance use can be more complex in terms of being influenced by more social and environmental factors (such as being more informed about the social and legal risks of substance use). Older adults may also develop coping strategies that become more adaptive with maturation (Wingo et al., 2015). These strategies could potentially weaken the links between risk propensity and substance use. Therefore, we expect the relationship between risk propensity and substance use to be stronger in the youth samples than in the older or adult samples.

1.5. The current study

Although the link between risk propensity and substance use has been investigated for two decades, past studies have used diverse conceptualisation and measurements of both risk propensity and substance

use. There has not been a systematic understanding or synthesis of this link. The purpose of this study is to fill this gap by conducting the first meta-analysis to comprehensively examine the relationship between risk propensity and substance use and identify the factors that moderate this relationship.

This review first examined the overall association between risk propensity and substance use and quantified the magnitude of this association. Specifically, we hypothesised that a high-risk propensity is positively associated with substance use. Moreover, we hypothesised that a high-risk propensity is positively associated with each type of substance use (namely, tobacco, alcohol, or drug use). We then examined potential moderators of the relationship between risk propensity and substance use; these include the type of substance use, the severity of substance use, the tools used to assess substance use and risk propensity, and the characteristics of the studies and samples.

2. Methods

The protocol of this meta-analysis was registered on the Open Science Framework platform (OSF). This meta-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines (Page et al., 2021).

2.1. Information sources and search strategy

PubMed, PsycINFO (via Ovid), Embase, CINAHL, and Scopus databases were searched to identify relevant articles from January 2002 to February 9, 2023 (inclusive). There were no restrictions regarding target populations or cultural or geographical contexts. The full details of the search strategies are shown in Supplementary file 1.

2.2. Eligibility criteria

Articles were included if they assessed human subjects, were observational studies (cross-sectional, cohort, and longitudinal studies) published in peer-reviewed journals in the English language since 2002 and reported associations (or findings that can be converted to associations) between risk propensity and substance use. Articles must address both constructs to be included in the assessment. In this study, the risk propensity construct encompassed terms such as risk-taking, risk aversion, risk tolerance, risk-seeking, risk preference, and risk avoidance. Substance use was categorised according to DSM-5 into ten classes of drugs: alcohol, caffeine, cannabis, hallucinogens (including phencyclidine and other hallucinogens), inhalants, opioids, sedatives, hypnotics, anxiolytics, stimulants (including amphetamine-type substances, cocaine, and other stimulants), tobacco, and other (unknown) substances.

Articles were excluded if they: (1) were case reports, conference presentations, corrections, commentaries, book chapters or series, protocols/registration, qualitative studies, or systematic reviews/meta-analyses; (2) did not have full text available; (3) did not include measurements of both risk propensity and substance use; (4) did not investigate the variables of interest; (5) studies lacking sufficient effect size data. For studies that did not report effect sizes[1] or results that could enable the calculation of effect sizes, 3 attempts were made to contact the authors with three-week intervals between attempts. Articles were excluded if: (1) there was no response from the authors; (2) the requested information could not be obtained (e.g. due to lack of access to the raw data); (3) the authors' email addresses were invalid; or (4) the authors responded, but the information provided was unsuitable for final analysis.

We focused less on risk perceptions, as they are more related to one's knowledge and experience of the risk than to their tendency to respond to the risk.

2.3. Selection process

After removing duplicates, the titles and abstracts of 6013 articles were independently screened for eligibility by the first and second authors. The Systematic Review Accelerator's Screenatron tool (https://sr-accelerator.com/#/) was used to facilitate this process. Disagreements between the first and second authors were resolved by consulting a third author using the Disputatron on the Systematic Review Accelerator. Subsequently, the first and second authors independently screened the full texts of 442 potentially relevant articles for eligibility, employing the PICO portal (https://picoportal.org/). Any discrepancies in decisions were reviewed by the third author to reach a consensus. A total of 148 articles were selected for data extraction. Fig. 1 shows the PRISMA data selection flow chart.

2.4. Data collection process and data items

Data extraction was independently conducted by the first and second authors on the PICO portal. Any discrepancies between the reviewers were resolved by the third author. The following data were extracted from each included study: (1) variables related to substance use, including the types of substances, the severity of substance use (i.e., substance use, misuse, or dependence), and the methods of assessing substance use; (2) characteristics of risk propensity measures, such as assessment type (behavioural task vs. questionnaire), instruments employed for assessing risk propensity, scoring methods, and domains of risk propensity; (3) study details, including author(s), year of publication, study topic, country, and study design; (4) characteristics of the sampled population, including age (mean and range), the proportion of males, the types of samples, and sample settings; and (5) measures of effect size for the association between risk propensity and substance use. The extracted data were further classified and cleaned using the complete detailed data classification and coding manual shown in Supplementary file 2. In the event that an article reported multiple effect sizes from the same sample, we retained effect sizes if they represented (1) different substances to capture substance-specific effects or (2) different tasks or measures of risk propensity. The details about effect size retention can be found in Supplementary file 3.

Email contacts, including those sent to the corresponding authors of articles with missing data, were conducted with a maximum of three follow-ups to request the necessary information. For articles with missing data ($n = 8$), pairwise deletion was used in subsequent analyses.

2.5. Assessment of study risk of bias

The JBI Critical Appraisal Checklist for cross-sectional (Joanna Briggs Institute, 2020) was used to evaluate the risk of bias. Checklist items are selected and modified to match the purpose of this review.

Six items were used in comparison studies. These items included (1) the criteria for inclusion in the clearly defined sample (e.g., whether a clinical diagnosis was a criterion to recruit participants who use substances); (2) the study subjects and the detailed setting (e.g., whether participants were recruited from hospitals or communities); (3) the risk propensity measured in a valid and reliable way (e.g., whether the studies used validated questionnaires or behavioural tasks to assess risk propensity); (4) the substance use measured in a valid, reliable, or clear manner (e.g., whether the studies used validated questionnaires or clinical diagnoses to measure substance use); (5) the identification of confounding factors (e.g., whether the studies identified gender/age differences); and (6) strategies to address the stated confounding factors (e.g., whether the studies attempted to match or control for gender/age

[1] If articles lacked the total sample size, the mean age, or both—making it impossible to calculate the effect size—we contacted the authors to obtain the missing information. If the authors did not respond to our enquiry, we addressed the missing data using pairwise deletion (as detailed in the section on Addressing Missing Data).

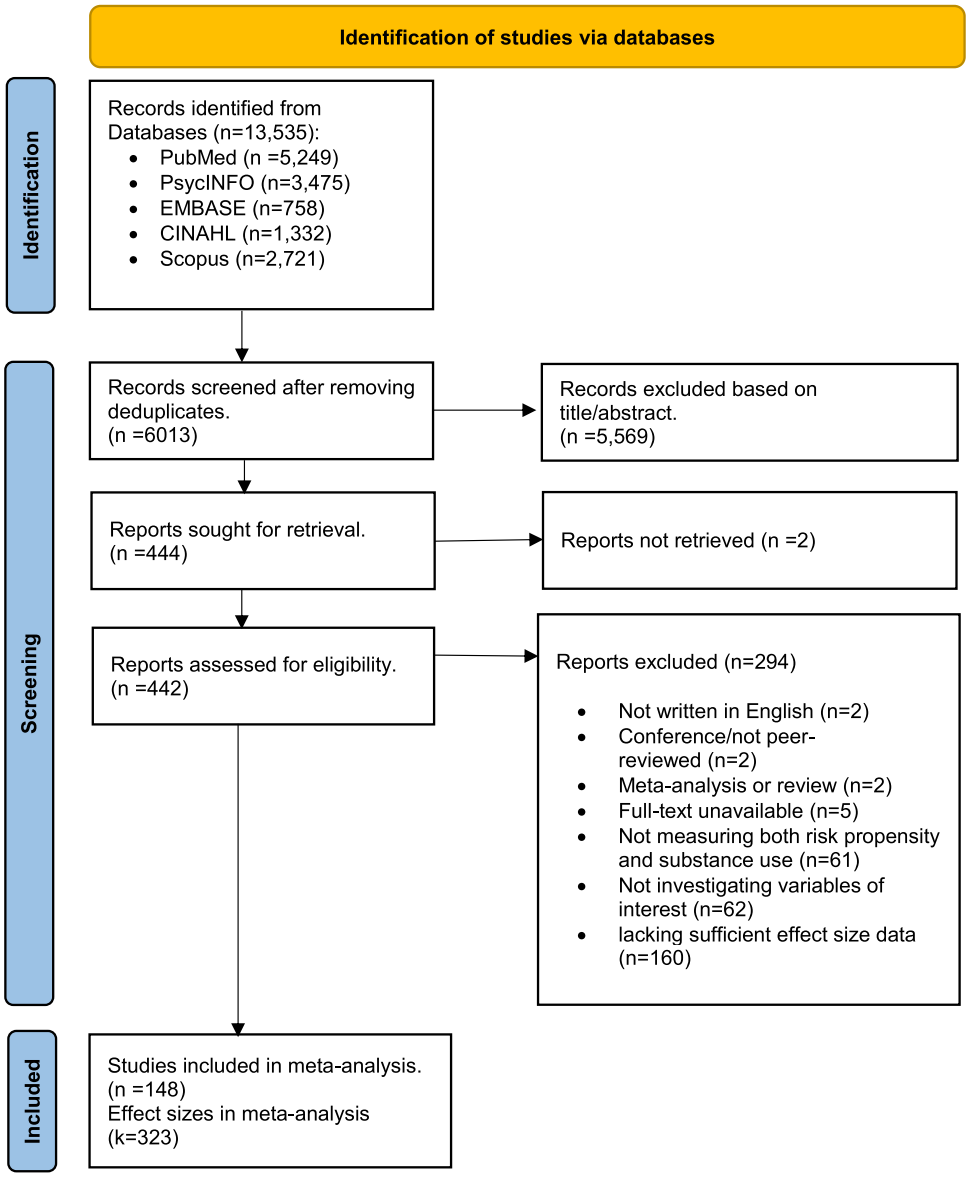

Fig. 1. PRISMA flow diagram showing study selection process.

differences). For association studies without a comparison between two groups, only items 1 through 4 were employed to assess the study quality.

The studies were independently assessed by the first and second authors. Each checklist item was scored as "0 =yes," "1 =no," "0.5 =unclear", or "not applicable." Disagreements were resolved by consulting the third author. Bias scores ranged from 0 to 6, with higher scores reflecting lower quality. Despite employing the JBI checklist, we did not exclude any studies from this meta-analysis.

2.6. Data processing

The primary effect size in the current meta-analysis was the correlation coefficient (r) representing the association between risk propensity and substance use. The direction of the correlation was adjusted such that a more positive value indicates a more positive association between risk propensity and substance use. Effect sizes of r = .10 were considered small,.30 were medium, and.50 were large (Cohen, 2013).

For studies that reported results in the form of means and standard deviations or other types of test statistics, transformation was applied (Fritz et al., 2012) to convert those results into *r* values for analysis.

For a detailed description of the data processing procedure, refer to Supplementary file 3.

2.7. Statistical analysis

A multilevel approach was adopted in this review to address the dependency of effect sizes. The multilevel meta-analysis was conducted in R (version 4.3.2) using the 'metafor' package (Viechtbauer, 2010).

A three-level meta-analytic model was employed to determine the overall effect size and perform the moderator analyses in accordance with the component guidance provided by Harrer et al. (Harrer et al., 2021). Three types of variances are considered: Level 1 accounts for the sampling variation in effect sizes, Level 2 accounts for the variance between effect sizes within the same research article, and Level 3 accounts for the variance between research articles (Assink and Wibbelink, 2016; Harrer et al., 2021).

Heterogeneity within and between samples was assessed using the I^2 index. An I^2 value of 25 %, 50 %, and 75 % corresponds to a small, medium, and large degree of heterogeneity, respectively (Huedo-Medina et al., 2006). As some articles also reported multiple independent samples, we also conducted the analysis using independent

samples as the higher-level unit, which resulted in a poorer model fit. Furthermore, adding independent samples as an additional level had similar results to the model without this additional level. Hence, we used research articles as Level 3 in this study. We first investigated the overall relationship between risk propensity and substance use and examined heterogeneity. Subsequently, we conducted a comparison between our three-level model and the two-level model to determine if the three-level model had significantly better model fit than the two-level model via likelihood ratio tests (Assink and Wibbelink, 2016; Harrer et al., 2021).

We performed subgroup analysis within the three-level model to investigate potential moderators influencing the overall effect. Considering the statistical power of subgroup analysis for detecting subgroup differences, groups with effect sizes of less than ten were not included (Harrer et al., 2021; Schwarzer et al., 2015). We first analysed each set of potential moderators separately. We then included all significant moderators in a multivariate model. Meta-analyses including moderator analyses were carried out to explore variations in risk propensity for each substance—tobacco, alcohol, and drugs separately. Potential outliers and influential cases were identified using Cook's distance, hat values, and DFBETAS. Effect sizes were considered to be influential or outliers based on the following criteria (Viechtbauer, 2010): (1) Cook's distance that exceeds the 50th percentile of the chi-square distribution with p degrees of freedom (p is the number of model coefficients); (2) a DFBETAS value greater than 1, or (3) a hat value greater than $3 \times (p/k)$ (k is the number of cases).

We used funnel plots to visually inspect whether the plot exhibited asymmetry to evaluate publication bias. Egger's regression test was used to assess asymmetry in the funnel plot. A significant result from Egger's test suggests the presence of potential publication bias (Egger et al., 1997).

3. Results

3.1. Study characteristics

The meta-analysis included 148 studies and 323 effect sizes (k) (Supplementary files 4 and 5). The study selection process is illustrated in Fig. 1. A total of 217,034 participants were included. The majority of the studies (approximately 76 %) were published between 2011 and 2023. Approximately 37.8 % of the articles focused on multiple substance use, 27 % focused on alcohol, 23 % focused on drugs, and 12.2 % focused on tobacco. The majority of the studies (86.5 %) adopted cross-sectional designs. Additionally, approximately 87 % of the studies were conducted in Western countries.

Out of a total of 186 independent samples, 55.9 % were classified as youth or adolescents, and 65.6 % were collected in community settings. The sample sizes ranged from 24 to 91,860, with a median of 113. The overall mean age of the participants was 26.2 years (SD = 11.3, range = 11.01–65.40), and the median proportion of male participants was 51.2 %.

Out of a total of 323 effect sizes, 206 (63.8 %) applied behavioural tasks to assess risk propensity, and 117 (36.2 %) used self-report questionnaires. Among the behavioural tasks, the BART (Lejuez et al., 2002) emerged as the most common instrument (46.6 %), followed by the IGT (12.6 %) (Bechara et al., 1994) and the CGT (7.3 %) (Rogers et al., 1999). On the other hand, the DOSPERT (Weber et al., 2002) was the most common self-report questionnaire instrument (23.9 %), followed by the CMNI (risk-taking subscale; 8.5 %) (Mahalik et al., 2003).

Approximately 38.1 % of effect sizes were for alcohol use, 32.8 % were for drug use, 18.3 % were for tobacco use, and 10.8 % were for multiple substance use. Self-report questionnaires were the most common tools for assessing substance use (68.7 %), followed by clinical diagnoses based on the DSM or the ICD (25.7 %). Regarding the severity of substance use, most studies focused on substance use (58.2 %), and approximately 13.9 % focused on substance dependence.

3.2. Risk of bias assessment

Of the 148 included studies, 68 had two groups (participants who use substances versus controls). Across the studies, the risk of bias was low, with 85.29 % of studies rated at or below 1. Only 2.9 % of studies had scores above the midpoint (sum score ≥ 3), reflecting a moderate level of bias. The most frequently observed sources of bias across studies were inadequate descriptions of the study subjects and settings, as well as invalid, unreliable, or unclear measurements of substance use. For the remaining 80 studies, the risk of bias was also low, with 91.25 % of the studies reporting a risk of bias less than 1. Approximately 1.3 % of these studies had scores above the midpoint (sum scores ≥ 2), suggesting a moderate level of bias. The most common sources of bias were invalid, unreliable, or unclear measurements of substance use.

3.3. Overall relationship between risk propensity and substance use

The results of estimating the overall correlation between risk propensity and substance use are displayed in Table 1. The results of a three-level random effects meta-analysis demonstrated a weak but statistically significant positive association between risk propensity and substance use ($k = 323$, $r = 0.116$, $p < 0.001$, 95 % CI = [0.087, 0.146]). Individuals with higher risk propensity are more likely to engage in substance use. The estimated variance component I^2_{Level3} was 78.52 %, and I^2_{Level2} was 19.38 %. The results indicate that over 78 % of the total variation is attributed to between-study differences, while over 19 % is attributed to within-study heterogeneity. The three-level model performed significantly better than the two-level model ($\chi^2 = 92.895$, $p < 0.001$).

Table 1 also summarises the results from the subgroup analysis. The results showed that the method used to assess substance use [$F(2, 320) = 3.083$, $p = 0.047$] significantly moderated the association between risk propensity and substance use. Smaller effect sizes were observed in clinical assessments than in self-reports.

The results also indicated that the type of risk propensity assessment significantly moderated the relationship between risk propensity and substance use [$F(1, 321) = 18.998$, $p < 0.001$]. Smaller effect sizes were found for behavioural tasks than for questionnaire-based assessments. We performed a follow-up moderator analysis by investigating different categories of risk propensity assessments and found that the effect size for the DOSPERT scale was significantly greater than that for all other types of measurements.

Moderator analysis suggests that the year of publication was a significant moderator [$F(1, 321) = 4.193$, $p = 0.041$], indicating that earlier studies were linked to larger effect sizes ($\beta_1 = -0.005$). Furthermore, younger participants and adolescents/youths exhibited larger effect sizes than their counterparts.

All the significant moderators in the bivariate models were subsequently entered into a multivariate model. The type of risk propensity assessment uniquely contributed to the explanation of variance while controlling for the effect of the other variables in the model (see Table 2). Larger effect sizes were found for the questionnaire type of risk propensity assessments compared to the behavioural tasks of risk propensity assessments.

No significant influential cases were detected. The funnel plot (Fig. 2) demonstrated a symmetrical distribution. Egger's regression test was then employed to evaluate publication bias. The result of Egger's regression test was non-significant ($p = 0.723$), suggesting that publication bias was unlikely.

3.4. The relationship between risk propensity and tobacco use

Based on 59 effect sizes obtained from 32 independent samples, the results of a three-level random effects meta-analysis demonstrated a small but statistically significant association between risk propensity and tobacco use ($r = 0.092$, $p = 0.003$, 95 % CI [0.033, 0.152])

Table 1
Results of the three-level meta-analysis for overall substance use.

Variables	s	k	β_0	r	t value				σ^2 level 2	σ^2 level 3	I^2_{Level2}	I^2_{Level3}
Random-effect model Overall Effect	148	323	0.117	0.116	7.836 * **				0.006	0.023	19.38 %	78.52 %
Subgroup analysis												
Moderator Variables	s	k	β_0	r	t_0	β_1	t_1	*F* (df1, df2)				
Substance type								*F* (3, 319) = 0.949	0.006	0.023	18.94 %	78.96 %
Multiple substances (RC)	24	35	0.146	0.145	3.906 * **							
Tobacco	28	59	0.093	0.093	4.194 * **	−0.052	−1.212					
Alcohol	59	123	0.123	0.122	6.655 * **	−0.023	−0.552					
Drugs	37	106	0.107	0.107	5.388 * **	−0.038	−0.916					
Substance use severity								*F* (3, 300) = 1.962	0.006	0.021	20.71 %	77.18 %
Substance use (RC)	78	188	0.138	0.137	7.435 * **							
Substance use disorders	16	22	0.050	0.050	1.063	−0.088	−1.747 +					
Mixed: survey	20	49	0.091	0.091	2.527 *	−0.047	−1.214					
Substance dependence	25	45	0.068	0.068	1.906 +	−0.070	−1.766 +					
Substance use assessment								*F* (2, 320) = 3.083 *	0.006	0.022	20.19 %	77.63 %
Self-report (RC)	91	222	0.138	0.137	7.645 * **							
Mixed or other	13	18	0.137	0.136	2.809 * *	−0.001	−0.018					
Clinical	44	83	0.061	0.061	2.239 *	−0.077	−2.367 *					
Types of risk propensity assessment								*F* (1, 321) = 18.998* **	0.006	0.019	22.70 %	74.86 %
Questionnaire/scale (RC)	46	117	0.188	0.186	8.771 * **							
Behavioural tasks	102	206	0.079	0.079	4.778 * **	−0.110	−4.359 * **					
The detailed types of risk propensity								*F* (6, 316) = 4.617 * **	0.006	0.018	23.78 %	73.72 %
DOSPERT (RC)	6	28	0.299	0.290	5.943 * **							
BART	58	96	0.092	0.092	4.228 * **	−0.208	−3.801 * **					
CGT	5	15	0.062	0.062	1.319	−0.237	−3.434 * **					
CMNI: risk-taking	4	10	0.208	0.205	2.627 * *	−0.091	−0.968					
IGT	15	26	0.037	0.037	1.014	−0.262	−4.224 * **					
Other scales	36	79	0.166	0.164	6.949 * **	−0.134	−2.499 *					
Other tasks	24	69	0.077	0.077	3.090 * *	−0.223	−4.135 * **					
Study characteristics												
Publication year (continuous)	148	323				−0.005	−2.048 *	*F* (1, 321) = 4.193*	0.006	0.022	19.87 %	77.98 %
Intercept						10.663	2.070 *					
Study design								*F* (1, 321) = 0.122	0.006	0.023	19.21 %	78.70 %
Cross-sectional design (RC)	128	275	0.119	0.118	7.344 * **							
Longitudinal design	20	48	0.104	0.104	2.669 * *	−0.015	−0.350					
Country								*F* (1, 317) = 0.025	0.006	0.023	19.56 %	78.35 %
Non-Western (RC)	17	31	0.120	0.119	2.988 * *							
Western	128	288	0.114	0.114	7.196 * **	−0.007	−0.159					
Sample characteristics												
Mean age (continuous)	144	316				−0.002	−2.379 *	*F* (1, 314) = 5.657 *	0.005	0.021	17.14 %	80.55 %
Intercept						0.178	5.876 * **					
Proportion of males (continuous)	145	308				−0.000	−0.959	*F* (1, 306) = 0.920	0.005	0.025	16.58 %	81.48 %
Intercept						0.128	5.661 * **					
Type of samples								*F* (1, 313) = 4.794 *	0.005	0.022	16.73 %	81.00 %
Youth or adolescents (RC)	81	190	0.136	0.135	7.358 * **							
Adults	62	125	0.079	0.079	3.734 * **	−0.057	−2.189 *					
Sample settings								*F* (2, 320) = 0.381	0.006	0.023	19.20 %	78.71 %
Other (RC)	13	31	0.153	0.152	3.203 * *							
Clinical	41	78	0.104	0.104	3.702 * **	−0.048	−0.873					
Community	94	214	0.116	0.115	6.352 * **	−0.037	−0.725					

s = number of independent studies, k = number of effect sizes, β_0= intercept of the model for each set/subset of the effect sizes, t_0 tests the intercept compared to zero, β_1 is the regression coefficient for models for continuous moderators, and is the difference between a level and the reference categorical of a categorical moderator, t_1 tests β_1 compared to zero, r Pearson correlation, *F* (df1, df2) omnibus test, RC reference category, σ^2 level 3 variance at the between-study level, σ^2 level 2 variance at the within-study level, I^2_{Level2} % of total variation within-study level, I^2_{Level3} % of total variation between-study level. *$p < 0.05$, * *$p < 0.01$, * **$p < 0.001$, +$p < 0.10$

(Table 3). These findings suggest that individuals with a propensity for risk-taking are more likely to engage in tobacco use. The results suggest that the mean age of the total sample was a significant moderator in the relationship between risk propensity and tobacco use, with younger participants demonstrating a larger effect size than older participants.

No significant outliers were detected for the three-level tobacco model. The funnel plot (Fig. 2) showed a symmetric distribution and Egger's regression test ($p = 0.780$) was also not significant. These findings suggested that there was no evidence of publication bias.

3.5. The relationship between risk propensity and alcohol use

Based on 123 effect sizes obtained from 74 independent samples, the results of a three-level random effects meta-analysis demonstrated a small but statistically significant association between risk propensity and alcohol use ($r = 0.114$, $p < 0.001$, 95 % CI [0.077, 0.154]). These findings suggest that individuals with a propensity for risk-taking are more likely to engage in alcohol use.

Table 4 summarises the results from the subgroup analysis on alcohol use. The results showed that the type of risk propensity assessment significantly moderated the association between risk propensity and alcohol consumption [F (1, 121) = 10.991, $p = 0.001$]. Larger effect

Table 2
Results of the multivariate model for substance use model.

	s	k	β (S.E)	t	95 % CI	*F* (df1, df2)	σ^2 level 3	σ^2 level 2
Intercept	142	312	9.853 (4.916)	2.004 *	[0.180, 19.526]	*F* (6, 305) = 4.687 * **	0.017	0.004
Substance use assessment								
Clinical vs. *Self-report*			−0.030 (0.038)	−0.787	[−0.104, 0.045]			
Mixed or other vs. *Self-report*			0.044 (0.050)	0.875	[−0.054, 0.141]			
Types of risk propensity assessment								
Behavioural tasks vs. *Questionnaire/scale*			−0.089 (0.026)	−3.415 * **	[−0.141, −0.038]			
Study characteristics								
Publication year			−0.005 (0.002)	−1.961 +	[−0.010, 0.000]			
Sample characteristics								
Mean age of total sample			−0.001 (0.002)	−0.351	[−0.004, 0.003]			
Adults vs. *Youth or adolescents*			−0.014 (0.043)	−0.319	[−0.098, 0.070]			

s = number of independent studies, k = number of effect sizes, *F* (df1, df2) omnibus test, σ^2 level 3 variance at the between-study level, σ^2 level 2 variance at the within-study level. *$p < 0.05$, * *$p < 0.01$, * **$p < 0.001$, +$p < 0.10$

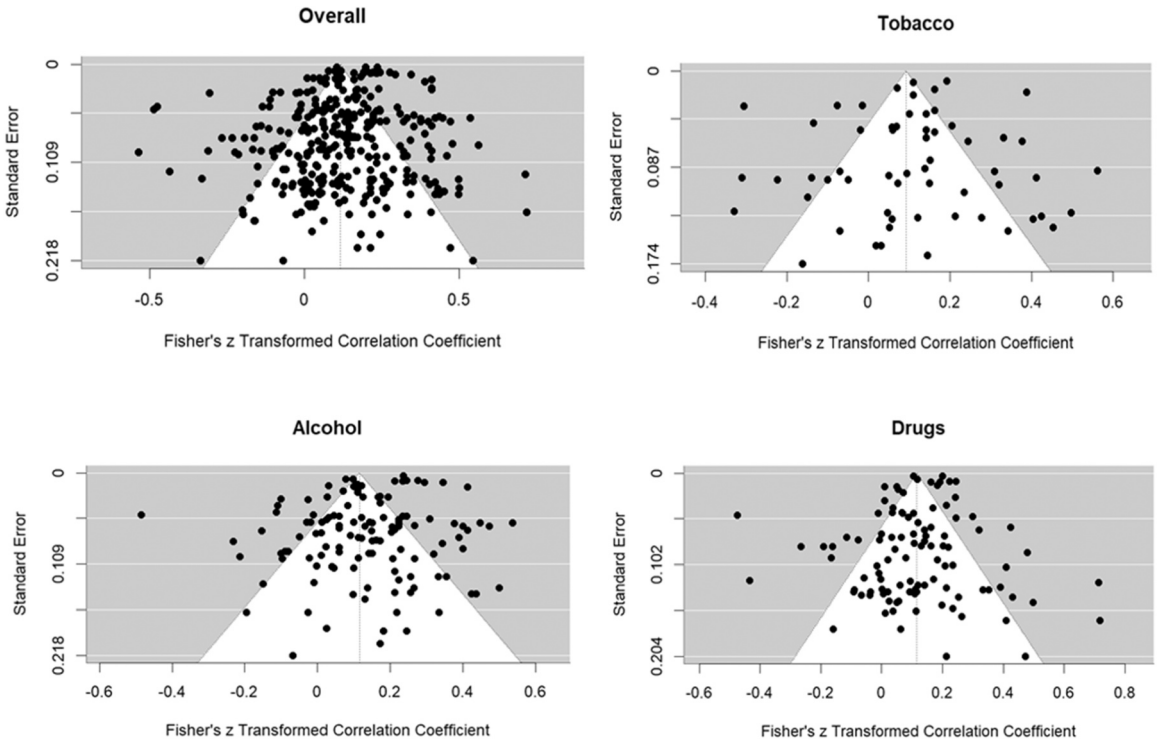

Fig. 2. Funnel plots of risk propensity and substance use: overall, tobacco, alcohol, and drugs.

sizes were observed in the questionnaire-based assessments than in the behavioural tasks. Follow-up investigations revealed that the effect size of the DOSPERT was significantly greater than that of the BART, other questionnaires, and other behavioural tasks. However, the association was not moderated by other moderators.

No significant outliers were detected for the three-level alcohol use model. The funnel plot (Fig. 2) exhibited symmetry, and the non-significant results of Egger's regression test (p = 0.870) further suggested the absence of publication bias.

3.6. The relationship between risk propensity and drug use

Based on 106 effect sizes obtained from 51 independent samples, the results of a three-level random effects meta-analysis demonstrated a statistically significant, albeit small, association between risk propensity and drug use (r = 0.115, p < 0.001, 95 % CI [0.070,0.162]). These findings suggest that individuals with a propensity for risk-taking are more likely to engage in drug use.

Table 5 presents a summary of the results from the subgroup analysis on drug use. The results indicated that the mean age of the total sample and the type of sample were significant moderators of the relationship between risk propensity and drug use. Younger participants and youth or adolescent participants exhibited stronger effects on the association between risk propensity and drug use.

No significant outliers were detected for the three-level drug model. The funnel plot (Fig. 2) showed a symmetric distribution, and Egger's regression test was also not significant (p = 0.495). These findings suggested there was no evidence of publication bias.

4. Discussion

This meta-analysis represents a comprehensive investigation that synthesised evidence from the previous two decades to support the link between risk propensity and substance use. Consistent with our hypothesis, the results showed a statistically significant association (r = 0.116) between risk propensity and substance use, and across all three main types of substances, including tobacco (r = 0.092), alcohol (r = 0.114) and drugs (r = 0.115). Individuals with greater risk-seeking tendencies are more prone to substance use overall than those who are risk-averse. Such a link was not significantly different across substance

Table 3
Results of the three-level meta-analysis for tobacco use.

Variables	s	k	β_0	r	t value				σ^2 level 2	σ^2 level 3	I^2_{Level2}	I^2_{Level3}
Random-effect model Tobacco	40	59	0.092	0.092	3.129 * *				0.006	0.024	18.02 %	77.36 %
Subgroup analysis Moderator Variables	s	k	β_0	r	t_0	β_1	t_1	F (df1, df2)				
Types of risk propensity assessment								F (1, 57) = 3.321 $^+$	0.006	0.022	20.99 %	74.04 %
Questionnaire/scale (RC)	17	26	0.141	0.140	3.641 * **							
Behavioural tasks	23	33	0.057	0.057	1.661	−0.083	−1.822 $^+$					
The detailed types of risk propensity								F (3, 51) = 1.664	0.006	0.023	20.70 %	74.51 %
DOSPERT (RC)	3	10	0.210	0.207	2.817 * *							
BART	13	17	0.104	0.104	1.929 $^+$	−0.106	−1.155					
Other scales	14	16	0.113	0.113	2.555 *	−0.097	−1.195					
Other tasks	7	12	0.035	0.035	0.721	−0.175	−2.114 *					
Study characteristics												
Publication year (continuous)	40	59				−0.008	−1.629	F (1, 57) = 2.654	0.006	0.024	18.73 %	76.53 %
Intercept						17.087	1.638					
Country								F (1, 56) = 0.027	0.005	0.024	17.96 %	76.97 %
Non-Western (RC)	7	10	0.074	0.074	1.079							
Western	32	48	0.086	0.086	2.632 *	0.012	0.164					
Sample characteristics												
Mean age (continuous)	38	57				−0.006	−2.633 *	F (1, 55) = 6.934 *	0.005	0.021	19.23 %	75.01 %
Intercept						0.243	3.914 * **					
Proportion of males (continuous)	38	53				−0.000	−0.335	F (1, 51) = 0.113	0.003	0.028	10.20 %	85.68 %
Intercept						0.096	2.283 *					
Type of samples								F (1, 55) = 1.406	0.005	0.021	19.73 %	74.46 %
Youth or adolescents (RC)	29	41	0.122	0.121	3.728 * **							
Adults	9	16	0.044	0.044	0.758	−0.079	−1.186					
Sample settings								F (1, 55) = 0.657	0.007	0.025	19.98 %	75.65 %
Other (RC)	6	11	0.146	0.145	1.972 $^+$							
Community	33	46	0.080	0.080	2.378 *	−0.066	−0.811					

s = number of independent studies, k = number of effect sizes, β_0= intercept of the model for each set/subset of the effect sizes, t_0 tests the intercept compared to zero, β_1 is the regression coefficient for models for continuous moderators, and is the difference between a level and the reference categorical of a categorical moderator, t_1 tests β_1 compared to zero, r Pearson correlation, F (df1, df2) omnibus test, RC reference category, σ^2 level 3 variance at the between-study level, σ^2 level 2 variance at the within-study level, I^2_{Level2} % of total variation within-study level, I^2_{Level3} % of total variation between-study level. *$p < 0.05$; * *$p < 0.01$; * **$p < 0.001$; $^+p < 0.10$

types. In addition, the link was not significantly different between substance use, misuse and dependence, suggesting that risk propensity was associated with both the uptake and the maintenance of substance use. Only the type of risk propensity assessments and age of the study samples moderated the link between risk propensity and one or more types of substance use.

Risk propensity entails increased tolerance of loss and threats, such as health consequences and social evaluation (e.g., perceived disapproval), which serve as barriers or factors deterring individuals from engaging in substance use. Individuals with an increased tolerance for the potential loss and threats are more likely to engage in substance use (Anderson and Mellor, 2008; Shou and Olney, 2022) as their increased intolerance diminishes the adverse effects associated with substance use, consequently fostering its initiation and sustained consumption. For example, Khwaja et al. (2006) showed that individuals who smoke were more risk-tolerant, and they may be more willing to bear health risks and trade-off current benefits against future health consequences (Khwaja et al., 2006). In contrast, perceived peer and parental disapproval of substance use can discourage individuals from engaging in such behaviour (Mrug and McCay, 2013).

The subgroup analysis suggests that questionnaire-based risk propensity assessments yielded stronger effects compared to behavioural tasks. One possible explanation for this difference is the lower reliability observed in behavioural tasks compared to questionnaire-based measures (Frey et al., 2017), which may attenuate the link between risk propensity measured by behavioural tasks and substance use. The other possible reason is that behavioural tasks usually tap into risks that are about momentary losses or losses in points that differ from the core risks of substance use. Questionnaire measures, especially DOSPERT, often capture risk propensity more specifically to or cover health risks. Health risks entail physical harm, which is more strongly associated with bodily sensations that can be related to the biological mechanisms of substance use (Ditre et al., 2019; Egli et al., 2012; Patterson et al., 2012). Previous research has also shown that behavioural tasks, such as BART, inadequately predict health behaviours such as tobacco, alcohol, and drug use (Frey et al., 2017; Szrek et al., 2012). Further analyses indicated that assessment type had statistically significant moderation on the link between risk propensity and alcohol use. The subgroup analysis results for tobacco and drug use were not statistically significant. This result may be attributed to the relatively small number of effect sizes for questionnaire-based assessments.

We also found that the samples with younger age groups showed a stronger link between risk propensity and the use of tobacco and drugs. Adolescence represents a sensitive period of neurobiological maturation that significantly influences the development of reward processing and sensation seeking, subsequently impacting the likelihood of tobacco and drug use. The heightened risk propensity observed in adolescents results from the combined influence of two brain systems: the limbic and paralimbic areas and the lateral prefrontal and parietal cortices, along with parts of the anterior cingulate cortex (Glicksohn et al., 2018). Neuroimaging studies showed that sensation-seeking and risk propensity were all the products of the interaction of these brain systems (Crane et al., 2018; Glicksohn et al., 2018; Wan et al., 2020). A longitudinal study demonstrated a peak in sensation seeking as a sign of reward sensitivity (Steinberg, 2017) during adolescence and a decrease in sensation seeking into adulthood. Additionally, maladaptive coping strategies, such as anger and avoidance, are associated with substance use among adolescents (Lee-Winn et al., 2018). Furthermore, it is suggested that adolescent smoking and drug use might serve as a means of coping with psychological and somatic complaints (Botello-Harbaum et al., 2011;

Table 4
Results of the three-level meta-analysis for alcohol use.

Variables	s	k	β_0	r	t value				σ^2 level 2	σ^2 level 3	I^2_{Level2}	I^2_{Level3}
Random-effect model Alcohol	71	123	0.115	0.114	5.929 * **				0.004	0.019	17.99 %	79.39 %
Subgroup analysis Moderator Variables			β_0	r	t_0	β_1	t_1	*F* (df1, df2)				
Substance use severity								*F* (2, 112) = 0.544	0.004	0.018	19.34 %	78.07 %
Substance use (RC)	42	74	0.106	0.106	4.410 * **							
Mixed: survey	14	29	0.155	0.154	3.676 * **	0.049	0.999					
Substance dependence	8	12	0.132	0.131	2.443 *	0.026	0.453					
Substance use assessment								*F* (1, 116) = 1.809	0.004	0.018	19.07 %	78.44 %
Self-report (RC)	54	99	0.128	0.127	5.831 * **							
Clinical	14	19	0.058	0.058	1.225	−0.070	−1.345					
Types of risk propensity assessment								*F* (1, 121) = 10.991 * *	0.003	0.017	16.62 %	80.44 %
Questionnaire/scale (RC)	27	56	0.178	0.176	6.717 * **							
Behavioural tasks	44	67	0.067	0.067	2.878 * *	−0.111	−3.315 * *					
The detail types of risk propensity								*F* (3, 105) = 4.081 * *	0.004	0.017	17.41 %	79.99 %
DOSPERT (RC)	3	11	0.292	0.284	4.584 * **							
BART	31	41	0.073	0.073	2.404 *	−0.218	−3.103 * *					
Other scales	20	38	0.151	0.150	4.866 * **	−0.141	−2.077 *					
Other tasks	10	19	0.078	0.078	2.024 *	−0.214	−3.042 * *					
Study characteristics												
Publication year (continuous)	71	123				−0.002	−0.558	*F* (1, 121) = 0.311	0.004	0.019	17.88 %	79.53 %
Intercept						4.132	0.574					
Study design								*F* (1, 121) = 0.000	0.004	0.019	17.66 %	79.77 %
Cross-sectional design (RC)	60	102	0.115	0.114	5.327 * **							
Longitudinal design	11	21	0.115	0.114	2.486 *	0.000	0.001					
Country								*F* (1, 121) = 0.387	0.004	0.019	16.83 %	80.61 %
Non-Western (RC)	6	11	0.085	0.085	1.653							
Western	65	112	0.118	0.117	5.842 * **	0.033	0.622					
Sample characteristics												
Mean age (continuous)	69	121				−0.001	−0.553	*F* (1, 119) = 0.306	0.004	0.017	19.65 %	77.53 %
Intercept						0.130	3.682 * **					
Proportion of males (continuous)	70	121				−0.000	−0.505	*F* (1, 119) = 0.255	0.004	0.019	18.38 %	79.09 %
Intercept						0.128	4.235 * **					
Type of samples								*F* (1, 117) = 0.186	0.004	0.017	20.35 %	76.62 %
Youth or adolescents (RC)	43	81	0.114	0.114	5.017 * **							
Adults	25	38	0.099	0.099	3.338 * *	−0.015	−0.431					
Sample settings								*F* (2, 120) = 0.117	0.004	0.019	17.66 %	79.79 %
Other	9	12	0.137	0.136	2.491 *							
Clinical	11	17	0.122	0.121	2.594 *	−0.015	−0.203					
Community	51	94	0.110	0.110	4.891 * **	−0.027	−0.449					

s = number of independent studies, k = number of effect sizes, β_0= intercept of the model for each set/subset of the effect sizes, t_0 tests the intercept compared to zero, β_1 is the regression coefficient for models for continuous moderators, and is the difference between a level and the reference categorical of a categorical moderator, t_1 tests β_1 compared to zero, r Pearson correlation, *F* (df1, df2) omnibus test, RC reference category, σ^2 level 3 variance at the between-study level, σ^2 level 2 variance at the within-study level, I^2_{Level2} % of total variation within-study level, I^2_{Level3} % of total variation between-study level. *$p < 0.05$; * *$p < 0.01$; * **$p < 0.001$; $^{+}p < 0.10$

Groenewald et al., 2020). Thus, the increase in sensation seeking along with reward sensitivity, combined with maladaptive coping, may contribute to the increased strength of the link between risk propensity and the use of tobacco and drugs.

However, the age of the sample did not significantly moderate the relationship between risk propensity and alcohol use. The link is likely to be restricted by other social contextual factors, such as community access to alcohol or legal policy related to age restrictions on alcohol consumption. Dent et al. (2005) suggested that higher rates of illegal merchant sales in a community are associated with increased alcohol use and binge drinking, whereas communities with stricter law enforcement tend to have a lower prevalence of alcohol use and binge drinking. Furthermore, a community-based randomised trial highlighted that stricter implementation of underage drinking law enforcement can reduce underage drinking (Flewelling et al., 2013). Although tobacco use is also restricted by law based on age, self-report bias seems to be more pronounced for tobacco than for alcohol (Shillington and Clapp, 2000). This bias may be particularly evident when smoking is an infrequent behaviour and is likely to be forgotten among adolescents (Shillington and Clapp, 2000). Moreover, adolescents perceive tobacco use as less morally acceptable than alcohol use; therefore, the underreported bias related to smoking might be greater than that for alcohol use (Amonini and Donovan, 2006).

5. Implications and future research

Despite prior research exploring this association, inconsistent results pose challenges in establishing conclusive evidence. Our findings provided further support for the role of risk propensity as one robust predictor of substance use. Early detection and screening of individuals with high-risk propensity such as youths could be essential for early intervention and prevention of substance use. While most assessments of risk propensity are for research purposes, it can be useful to develop screening tools tailored for the target population with the integration of information from clinical records and other informant reports. These efforts would enable the tailoring of intervention programs, such as focused training in social resistance skills, life skills, or emotional regulation (Brumback et al., 2021; Griffin and Botvin, 2010).

While this study underscores that risk propensity plays a partially influential role in substance use, albeit a minor one, it highlights the need for further research to explore additional factors and mechanisms that contribute to substance use and to develop more comprehensive

Table 5
Results of the three-level meta-analysis for drug use.

Variables	s	k	β_0	r	t value				σ^2 level 2	σ^2 level 3	I^2_{Level2}	I^2_{Level3}
Random-effect model Drugs	57	106	0.116	0.115	4.998 * **				0.004	0.021	16.73 %	81.40 %
Subgroup analysis Moderator Variables			β_0	r	t_0	β_1	t_1	*F* (df1, df2)				
Substance use severity												
Substance use (RC)	29	47	0.132	0.131	4.960 * **			*F* (2, 85) = 1.529	0.005	0.013	27.85 %	69.98 %
Mixed: survey	2	11	−0.008	−0.008	−0.092	−0.140	−1.532					
Substance dependence	14	30	0.078	0.078	1.800 $^{+}$	−0.054	−1.068					
Substance use assessment								*F* (1, 95) = 1.002	0.005	0.018	21.04 %	77.04 %
Self-report (RC)	27	52	0.126	0.125	4.171 * **							
Clinical	24	45	0.080	0.080	2.237 *	−0.047	−1.001					
Types of risk propensity assessment								*F* (1, 104) = 1.751	0.005	0.019	20.94 %	77.07 %
Questionnaire/scale (RC)	16	27	0.156	0.155	4.133 * **							
Behavioural tasks	41	79	0.097	0.097	3.618 * **	−0.059	−1.323					
The detailed types of risk propensity								*F* (4, 93) = 0.185	0.004	0.019	17.56 %	80.50 %
Other scales (RC)	13	19	0.120	0.119	2.970 * *							
BART	17	27	0.098	0.098	2.714 * *	−0.022	−0.406					
CGT	2	10	0.093	0.093	1.731 $^{+}$	−0.027	−0.404					
IGT	8	13	0.120	0.119	2.455 *	−0.001	−0.011					
Other tasks	14	29	0.087	0.087	2.313 *	−0.034	−0.651					
Study characteristics												
Publication year (continuous)	57	106				0.002	0.586	*F* (1, 104) = 0.343	0.004	0.022	16.08 %	82.09 %
Intercept						−4.880	−0.572					
Study design								*F* (1, 104) = 0.026	0.004	0.022	16.15 %	82.03 %
Cross-sectional design (RC)	47	91	0.118	0.117	4.522 * **							
Longitudinal design	10	15	0.108	0.108	1.987 *	−0.010	−0.160					
Sample characteristics												
Mean age (continuous)	56	105				−0.004	−2.442 *	*F* (1, 103) = 5.963 *	0.003	0.014	18.8 %	78.46 %
Intercept						0.233	4.899 * **					
Proportion of males (continuous)	57	100				−0.000	−1.001	*F* (1, 98) = 1.002	0.002	0.026	8.31 %	90.09 %
Intercept						0.137	4.008 * **					
Type of samples								*F* (1, 104) = 5.309 *	0.003	0.025	11.18 %	87.11 %
Youth or adolescents (RC)	28	49	0.163	0.162	5.131 * **							
Adults	29	57	0.068	0.068	2.118 *	−0.095	−2.304 *					
Sample settings								*F* (1, 97) = 0.024	0.005	0.025	15.22 %	83.24 %
Community (RC)	27	52	0.114	0.114	3.316 * *							
Clinical	24	47	0.106	0.106	2.779 * *	−0.008	−0.155					

s = number of independent studies, k = number of effect sizes, β_0 = intercept of the model for each set/subset of the effect sizes, t0 tests the intercept compared to zero, β_1 is the regression coefficient for models for continuous moderators, and is the difference between a level and the reference categorical of a categorical moderator, t_1 tests β_1 compared to zero, r Pearson correlation, *F* (df1, df2) omnibus test, RC reference category, σ^2 level 3 variance at the between-study level, σ^2 level 2 variance at the within-study level, I^2_{Level2} % of total variation within-study level, I^2_{Level3} % of total variation between-study level. *$p < 0.05$; * *$p < 0.01$; $^{***}p < 0.001$; $^{+}p < 0.10$

prevention strategies. It is likely that other factors, such as peer level of substance use, levels of parent-adolescent conflict, community influences, or legal policy (Allen et al., 2021; Dent et al., 2005), also contribute to the interplay between risk propensity and substance use. Hence, future research should employ longitudinal designs to explore the causal relationships between risk propensity and substance use, providing deeper insights into the onset and persistence of substance use and related disorders. Additionally, cross-cultural studies are needed to examine variations in risk propensity and substance use across diverse cultural contexts, offering a broader understanding of these dynamics beyond Western settings.

6. Conclusion and limitations

Some limitations should be acknowledged. This review only included published, peer-reviewed journal articles with available or calculatable effect sizes. Therefore, there are unpublished studies that have not contributed to our understanding of the relationship between risk propensity and substance use. Second, most of the reviewed studies were cross-sectional, whereas a few were longitudinal. The purpose of this review was to focus on the association rather than its directionality; thus, we cannot speculate on the directionality of the examined relationships. Third, although this meta-analysis did not restrict the included studies by country, the majority were conducted in Western countries. More research is needed to understand the homogeneity of the link across cultural contexts. Fourth, most of the studies relied on self-reported measures for risk propensity and substance use. More research is needed with the integration of multiple sources of information including clinical records and other informant reports.

In conclusion, the current study provides evidence to support the link between risk propensity and substance use as well as across tobacco, alcohol, and drugs. The results of the moderator analysis vary across overall substance use as well as tobacco, alcohol, and drug use. The findings underscore the importance of considering the role of risk propensity in the initiation of substance use and developing early interventions to prevent substance use. Future interventions tailored to an individual's risk propensity could be designed to prevent or mitigate substance use.

Ethics approval

This study did not require ethical approval. The systematic review

used publicly accessible data as evidence. No personal or sensitive information from primary data sources was included in this study.

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CRediT authorship contribution statement

Yeo Joey Elizabeth: Writing – review & editing, Project administration, Investigation. **Wang Chia-Wen:** Writing – review & editing, Writing – original draft, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Shou Yiyun:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization.

Declaration of Competing Interest

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

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.drugalcdep.2025.112640.

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