E-Risk Study Concept Paper Form

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1. Collaborating researchers

Please note:

Once approved, a formal data use agreement will be required between King's College London and the university or research organisation that employs any collaborator having access to the data if they are not a member of staff, a student or affiliate of King's College London. This needs to be signed by both universities/organisations before data access can be granted.

For projects carried out by a student (e.g., MSc/MA, MPhil/PhD, clinical doctorate), the lead applicant should be the student's supervisor at the same university, and the student should be named as the student collaborator requiring access to the data.

If you have additional collaborators, please name them below and indicate whether they need to have access to the data. It would be common, for instance, for other researchers to see summary results of analyses and act as co-authors on your paper without having access to the data. You will not be permitted to share the dataset except with those indicated in the table as requiring access.

Applicable?	**Category**	**Name**	**Email address**	**University/organisation**	**Needs access to data for analysis?**
	Applicant **(lead researcher)**	Alvin Junus	ajunus@hku.hk	The University of Hong Kong	◉ Yes ○ No
○ Applicable ◉ Not applicable	**Student collaborator** ***(if data is for their dissertation/thesis)***				
◉ Applicable ○ Not applicable	**E-Risk Sponsor** ***(if applicant is not an E-Risk investigator)***	Helen Fisher	helen.2.fisher@kcl.ac.uk	King's College London	○ Yes ◉ No

Are there additional collaborators to add?	◉ Yes ○ No
If yes, how many additional collaborators would you like to add?	1

Category	**Name**	**Email address**	**University/organisation**	**Needs access to data for analysis?**
Other collaborator #1	Massimiliano Orri	massimiliano.orri@mcgill.ca	McGill University	○ Yes ◉ No

Applicants: If you would like to continue your application later, please press the "Save and return later" button below. Please copy or write down the Return code provided.

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2. The project proposal

Note: *Please provide sufficient detail to enable the committee to review your proposal. Please be as specific as possible about the project aims and analysis methods as once approved this concept paper will be posted publicly and thus will act as a form of pre-registration of your project. Expand boxes as required.*

Title of project	Disentangling the web of interrelations between polygenic risk scores and psychosocial determinants that underlie youth suicide attempts
Background and rationale for project ***(approx. 300 - 1000 words)***	Suicide is a defining challenge for 21st century society, claiming over 720,000 lives annually [1], and is particularly pressing among the young population. Suicide has been the top cause of death for youths in places like the UK [2], Hong Kong [3], and Singapore [4], and was the third leading cause of death among 15-29-year-olds globally in 2021 [1]. Despite concerted research and prevention efforts, however, the latest studies indicate that youth suicide rates remain persistently high and in some contexts are even on an upward trajectory [5,6]. This signals a pressing need to resume stalled progress in lowering the burden of suicide on youths. Factors spanning biological, psychological, and social domains contribute to suicidality [7]. This notion is encapsulated in the stress-diathesis model of suicidal behavior [8], which operationalizes one's pathway to suicidal behavior as being influenced by two sets of factors - stressors, e.g., relationship or financial problems, major depressive episode, etc., and the individual's traits or innate vulnerability, e.g., predisposition to risky behaviors, pessimism, etc. The single putative factor which then differentiates suicidal behaviors from completed suicide is only the lethality of the means chosen by a suicide attempter [9]. Therefore, better understanding whom among youths are at greater risk of suicide attempt, in what way(s), and how to best intervene will be key in advancing youth suicide prevention efforts. A deeper, more comprehensive understanding of the biopsychosocial etiology of suicide attempt would represent a foundational step towards this cause. Findings from twin and family studies have shown that individuals' suicide-related traits were heritable, with heritability estimates ranging from 30% to 74% [10,11]. A landmark large-scale Genome-Wide Association Studies (GWAS) have identified up to 12 genome-wide significant (GWS) loci for suicide attempt [12]. Polygenic risk scores (PRS-SA) based on these loci may thus summarize a person's genetic predisposition to suicide attempt (SA), as validated in recent studies [13,14]. However, as heritability estimates are much less than 100% and the variance explained by PRS is reasonably small, these findings also indicate that other factors, e.g., the aforementioned stressors, must be involved in its etiology. Indeed, new empirical findings [15] lend support to the idea of genetic predisposition and a child's rearing environment both playing a systematic role in influencing their liability toward suicide attempt. Decades of research have accumulated an extensive evidence base for psychosocial determinants of youth suicide attempts, which Carballo et al. [16] summarized into three main clusters: (i) psychological factors, particularly depression, other psychiatric disorders, risk-taking behaviors such as substance use, and previous suicidality; (ii) adverse life events, particularly family situations such as low socioeconomic status (SES), (physical and sexual) abuse and neglect, interpersonal problems such as loneliness, bully victimization, and exposure to suicidal behaviors; and (iii) personality traits, particularly impulsivity. Nevertheless, a hallmark of suicidality is that it results from a complex interrelation between multiple factors [7,17]. For instance, indications of pleiotropy between genetic correlates of SA and major depressive disorder (MDD) have been recently observed [18], as well as pleiotropy between SA and attention-deficit hyperactivity disorder (ADHD) even after controlling for MDD [12,13]. Other determinants of SA, particularly risk-taking behaviors [12,18] and conduct problems [13,18], have also been suggested to have genetic overlaps with SA. Thus, the etiology of SA would likely involve a direct relationship between PRS-SA and SA that is partially mediated by risk factors such as MDD, ADHD, risk-taking behaviors, and conduct problems. New evidence further suggests that psychiatric disorders such as MDD and ADHD could also mediate the relationship between SA and childhood abuse and neglect [19]. Synthesizing these relationships together would conceptually give rise to a complex web of interrelations between SA and the aforementioned determinants, with still possible additional mediators that have been less well-documented. An empirical map of a biopsychosocial etiology of youth SA has been elusive, however, because studies on youth populations that comprehensively evaluate multifactorial determinants of SA have been scarce. Among studies that investigated established determinants of youth SA, the predominant focus has been on a few determinants' relationship with SA, but not their interrelations with one another [17]. Similarly, the newer studies that investigated links between PRS-SA and SA mostly did not consider other determinants beyond depression and distress, as alluded by Docherty et al. [12], thereby failing to account for the inherent complexity of interrelations between determinants that underlie incidence of youth SA. Moreover, there may be etiological heterogeneity across subgroups [7]. For example, poor family condition is a stronger risk factor for SA among girls [20], while risk-taking behaviors, particularly smoking, is a stronger risk factor among boys [21]. Furthermore, among those that longitudinally increase SA risk, factors such as depression are known to be more specific to girls and conduct disorder to boys [22]. These discrepancies therefore suggest that strengths of statistical interrelations between SA and its determinants may differ between sexes. Advances in statistical techniques in the past decade have provided researchers with a methodology to better delineate such webs of interrelations: psychometric network analysis (PNA) [23]. Due to its compatibility with survey and interview measurements, it has quickly gained prominence in data-driven psychopathological research [24]. PNA allows exploration into patterns of all possible pairwise conditional dependencies between a set of examined variables (conceptualized as nodes), in this case SA, its psychosocial determinants, and PRS-SA. Direct associations and mediations between nodes can then be teased apart and visualized as weighted edges in a network structure, where an edge's weight denotes the strength of statistical relationship between two connected nodes. Therefore, PNA combined with empirical data on youth samples covering the outlined variables will altogether have strong potential to map the biopsychosocial etiology of youth SA, providing fundamental insights to advance youth suicide prevention. Juxtaposing findings from independent but contextually similar cohorts will add to their robustness and significance. The E-Risk and Quebec longitudinal study of child development (QLSCD) [25] cohorts are thus ideally positioned, as both are longitudinal population-representative cohorts with similarly aged cohort members and measures of all the aforementioned variables.

Project aims / objectives	1) To empirically chart a biopsychosocial etiology of youth suicide attempt; and 2) To elucidate possible etiological nuances across sexes
Brief statement of your hypothesis	H1: Variability of PRS-SA will have direct, longitudinal positive association with SA outcome and be partially mediated by psychosocial determinants, including but may not be limited to depression, ADHD, risk-taking behaviors, and conduct problems. H2: Statistical interrelations between SA outcome and its biopsychosocial determinants will differ between boys and girls.
Data analysis methods to be used ***(approx. 100 - 500 words)***	PRS-SA will be computed based on summary statistics from the latest GWAS meta-analysis by Docherty et al. [12]. No significance threshold will be applied to select single nucleotide polymorphisms (SNPs) for inclusion in PRS analyses (i.e., all matched SNPs will be included). The primary analytical method to be used will be psychometric network analysis . Analyses are planned to be conducted in R, where relevant packages have been established [26]. SA outcomes and psychosocial determinants at two time points - index age and outcome age - will be accounted separately to better isolate longitudinal associations from potential cross-sectional associations. For the E-Risk cohort, index and outcome age will be 12 and 18 years, respectively. Choice of variables is informed by three factors: 1) identified potential mediators between PRS-SA and SA; 2) the most prominent psychosocial determinants of SA as identified by Carballo et al. [16]; and 3) availability of measurements in both the E-Risk and QLSCD cohorts. The following variables will each be conceptualized as a node: (i) PRS-SA; (ii) Exposure to psychosocial determinants of SA before study samples were at 12 years of age - SES, childhood abuse and neglect, peer victimization, loneliness, risk-taking behaviors, conduct problems, psychiatric disorders, and family history of psychiatric disorders; (iii) Psychosocial determinants assessed for the period between the ages of 12 to 18 - victimization, loneliness, risk-taking behaviors, conduct problems, and psychiatric disorders; and (iv) SA outcome assessed at study samples' ages of 12 and 18. Due to the nodes being a mix of continuous, categorical, and binary in nature, the mixed graphical model (MGM) will be best placed to estimate their patterns of interrelations [26]. In MGM, for every possible pair of nodes, a node-wise penalized generalized linear regression will estimate their association after accounting for all other nodes in the network. Estimated regression weights are then combined and averaged. In the resulting network structure, edge weights will thus denote regression coefficients. Analysis will first be conducted for the total sample; subgroup analyses based on male and female sexes will then follow. Recommended guidelines will be followed to obtain estimates with optimal statistical rigor [26]. Penalization will utilize the Graphical least absolute shrinkage and selection operator (gLASSO) algorithm which will set small edge weights that are likely due to noise to exactly zero so as to result in a sparse / high-specificity network structure. Final model selection will triangulate between two possible optimization approaches: (i) a model that optimizes the extended Bayesian Information Criterion (EBIC); and (ii) a model that cross-validation (CV) prediction accuracy. Tuning hyperparameter γ for EBIC will be calibrated between 0.5 (resulting in estimations with maximum specificity) and 0.25 (striking a balance between sensitivity and specificity). Both total sample and subgroup analyses for both cohorts will follow the above procedures. In case of a relatively low ratio between any subgroup's sample size and the number of nodes, model selection through EBIC will be preferred. Sensitivity and specificity of edges' estimation will be balanced by calibrating the EBIC tuning hyperparameter γ between 0.5 and 0 (for maximum sensitivity). Subgroups' network estimation will adopt the same parameter choices to enable fair comparison. Finally, an established framework, the network comparison test (NCT) [26], will compare the overall connectivity, connection strength, and specific edges of interest before a cohort's male and female subgroup networks. In particular, statistically significant differences in the two networks' edge weights will indicate specific sex differences in the biopsychosocial etiology of youth SA. Further sensitivity analyses will be conducted to ascertain effects of non-independence of twin observations. Adapting prior procedures done by Kendler et al. [27], a network structure will be estimated using a random subsample of twins from each twin pair, and using NCT, be subsequently compared with the remaining subsample's network. Absence of statistically significant differences in the two subsample networks' edge weights would indicate that findings from the total sample are robust enough for safe interpretation. Edges that have significantly different weights in the two subsample networks will either be interpreted with high caution should there be plausible explanations (e.g., potential outliers), and not interpreted otherwise.
Significance for theory, research methods, or clinical practice	Evidence arising from this project could yield novel insights towards a more comprehensive understanding of the etiology of suicidality, particularly by clarifying through real-world data possible direct and indirect influences of genetic vulnerability on young males' and females' tendency to attempt suicide. In the longer term, findings here may be a pioneering contributor to an evidence base for genetic vulnerabilities to be weighed / debated in the (re)formulation of national suicide prevention strategies - an aspect which has been missing hitherto [28,29,30].
References cited	1. World Health Organization (2025). Suicide. https://www.who.int/news-room/fact-sheets/detail/suicide (Date accessed: 18 August 2025). 2. Office for National Statistics (2020). Leading causes of death, UK: 2001 to 2018. https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/causesofdeath/articles/leadingcausesofdeathuk/2001to2018 (Date accessed: 18 August 2025). 3. HKJC Centre for Suicide Research and Prevention (2024). Suicide rates by age group in Hong Kong. https://csrp.hku.hk/statistics/ (Date accessed: 11 August 2025). 4. Samaritans of Singapore (2023). Highest Recorded Suicide Numbers in Singapore Since 2000. https://www.sos.org.sg/pressroom/highest-recorded-suicide-numbers-in-singapore-since-2000 (Date accessed: 18 August 2025). 5. Bertuccio, P., Amerio, A., Grande, E., La Vecchia, C., Costanza, A., Aguglia, A., ... & Odone, A. (2024). Global trends in youth suicide from 1990 to 2020: an analysis of data from the WHO mortality database. EClinicalMedicine, 70, 102506.

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Are there any files you would like to upload to support your concept paper?	○ Yes ◉ No

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3. Expected project outcomes

Please note:

The stated end date must be within 24 months of the date when this form is submitted. This end date will form part of the formal data use agreement and on this date you should delete the dataset. Therefore, it must be a realistic date for completion of the project including all analysis, writing a manuscript, review of the manuscript by all collaborators, submission, revisions, and acceptance of a paper for publication.

If you require an extension to the end date of the project, then you should contact Prof Fisher (helen.2.fisher@kcl.ac.uk) to discuss this. If you have signed a formal data use agreement, you will need to complete a form to request a licence extension. In some cases, we may also ask you to complete a new concept paper form if there have been substantial changes to the project or a long period of time has elapsed (e.g., greater than a year since the end date of the original project).

If the objective of the project is not a journal publication, please suggest an end date within 12 months instead of 24 months, and state a measurable, concrete outcome. If the objective of the project is a student dissertation, then the expected end date should be the deadline for submission of the dissertation; dissertation projects will only be accepted on agreement that they are strictly not for publication.

Date form submitted	04-09-2025 *DD-MM-YYYY*
End date for the project	03-09-2027 D-M-Y *DD-MM-YYYY*
Do you expect to publish your results in a journal?	(•) Yes ( ) No
If yes, please provide a provisional list of author names	**Alvin Junus, Helen L. Fisher, Massimiliano Orri, Louise Arseneault, Terrie Moffitt, Avshalom Caspi, Karen Sugden, Benjamin Williams**
If yes, please provide a provisional list of journals	**Molecular Psychiatry, Biological Psychiatry, British Journal of Psychiatry, Psychological Medicine, Journal of Child Psychology and Psychiatry, Epidemiology and Psychiatric Sciences, Journal of Affective Disorders**

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4. List of variables required

Please note:

When specifying variables, please be unambiguous. For each variable, specify the name of the measure, twin age, informant, and if you want specific subscales/derived categories (e.g., Depression from interview with twin at age 18; both number of symptoms and DSM-IV diagnosis). Alternatively, for maximum clarity, give actual variable names (e.g., MDESXE18 - MDE Symptom scale - P18 - Elder; DXMDEE18 - Major depressive episode, dsm4 - P18 - Elder).

By default, the dataset will usually include twin and family IDs, the "random" and "true" twin order variables, the cohort the twin is from (1994 or 1995), twin sex, ethnicity and zygosity variables, and family socioeconomic status at age 5. These routine background variables are listed in the table below. If you require further background variables, please specify them in your list.

Access to some parts of the dataset are restricted, namely identifiable data (e.g., postcodes, video recordings, individual-level genotypic and epigenetic data) which will not be shared outside King's College London, and linked administrative data which is only accessible via the UK Longitudinal Linkage Collaboration's Trusted Research Environment (this requires a separate formal data access agreement).

Background variables that will be included by default:

Variable name	Description
FAMILYID	**Unique family identifier**
ATWINID	**Twin A ID (ex chkdg)**
BTWINID	**Twin B ID (ex chkdg)**
RORDERP5	**Random Twin Order**
TORDER	**True Twin Order**
RISKS	**Sample Groups**
COHORT	**Cohort**
SAMPSEX	**Sex of Twins**
ZYGOSITY	**Zygosity**
SETHNIC	**Ethnicity of Twins**
SESWQ35	**Social Class Composite**

Please select the variables that will be requested	☐ Age 5 variables ☐ Age 7 variables ☐ Age 10 variables ☑ Age 12 variables ☑ Age 18 variables ☐ Age 26 variables

	☐ Age 30* variables
Age 12 variables	**CDICATE12 - Clinically significant depression - P12 - Elder** **ANYCDDX_EMT512 - Any CD dx from 5 to 12, reported by mum/teacher, Elder** **ADHDANYE512 - Any ADHD dx [incl meds] - P5-12 - Elder** **FHPSYHSUIC12 - Family History of psychiatric hospitalisation or suicide attempt** **sub03ec12 - Have you ever tried drinking alcohol like beer, wine, vodka or cider?** **sub04ec12 - How often do you drink alcohol like beer, wine, vodka or cider?** **sub07ec12 - Have you ever tried smoking a cigarette?** **sub08ec12 - How often do you smoke cigarettes?** **sub15ec12 - Have you ever tried any has or cannabis?** **sub16ec12 - Have you ever tried any pills to get high?** **sub17ec12 - Have you ever tried any glue or gas to sniff?** **EX_SVE12 - Exposed to any severe victimization (0/1), 5-12, E-Twin** **LONELYE12 - Loneliness scale - P12 - Elder** **SHARMSUICE12 - Self-Harm/Suicidal Behaviour - P12 - Elder**
Age 18 variables	**DXMDEE18 - Major depressive episode, dsm4 - P18 - Elder** **CDMODE18 - Moderate Conduct Disorder (>=5 count) - P18 - Elder** **DXADHD5X_18E - DSM-5 ADHD Dx (based on >=5 Symp) [incl 4 NEET & meds] - P18 - ET** **DXAUDE18 - Alcohol use disorder, dsm5 - P18 - Elder** **ALCVOL20E18 - Heavy alcohol drinking (age 18) - Elder** **SMKCURE18 - Smoking daily - current - P18 - Elder** **SMKDLYE18 - Ever a daily smoker - P18 - Elder** **SMKDXFTNDE18 - Fagerstrom Dx for nicotine dependence - P18 - Elder** **DXDRG5E18 - Substance use disorder, dsm5 - P18 - Elder** **DXMARJ5E18 - Marijuana use disorder, dsm5 - P18 - Elder** **ANYSEVVCTZE18 - Any severe victimization between 12-18 - P18 - Elder** **LONELYE18 - Loneliness scale - P18 - Elder** **SUICATE18 Suicide attempted - P18 - Elder** **Polygenic score for suicide attempt based on Docherty et al. (2023). GWAS meta-analysis of suicide attempt. American Journal of Psychiatry, 180(10), 723-738, + Covariates for PGS (e.g., first 10 PCs, batch number etc.)**
Are you requesting access to identifiable or linked data?	○ Yes ◉ No
Which format(s) do you require the data in?	☑ CSV ☐ Excel ☐ SPSS ☐ STATA ☐ Other

Applicants: If you would like to continue your application later, please press the "Save and return later" button below. Please copy or write down the Return code provided.

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5. Data security agreement and signature

Please click in each box to indicate that you will adhere to each of the points listed below.

◉ I adhere	**I am current on Human Subjects Training (CITI (www.citiprogram.org) or equivalent)**
◉ I adhere	**My project is covered by the King's ethics committee OR I have /will obtain ethical approval from my home institution.**
◉ I adhere	**I will treat all data as "restricted" and store in a secure fashion.** **My computer or laptop is:** **a) encrypted (recommended programmes are FileVault2 for Macs, and Bitlocker for Windows machines)** **b) password-protected** **c) configured to lock-out after 15 minutes of inactivity AND**