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Aspects of the Aetiology of Mental Health Problems among University Students

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ASPECTS OF THE AETIOLOGY OF MENTAL HEALTH PROBLEMS AMONG UNIVERSITY STUDENTS

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Till mamma

Populärvetenskaplig sammanfattning

Psykisk ohälsa är en av de vanligaste formerna av funktionsnedsättande ohälsa bland unga vuxna. Den självrapporterade förekomsten av psykisk ohälsa har ökat dramatiskt de senaste decennierna, inte minst hos universitetsstudenter. Uppemot en femtedel av alla studenter uppskattas årligen uppfylla kriterierna för en psykiatrisk diagnos, men få av dessa får vård. Förutom det lidande som är förknippat med psykisk ohälsa så är dessa problem också kopplade till sämre studieresultat och sämre framtida arbetsprestation.

Vad som ligger bakom utvecklandet av psykisk ohälsa och psykiatriska tillstånd är långt ifrån klarlagt, men det antas finnas av en mängd olika orsaker. Allt från genetiska till psykologiska och sociala faktorer har visats kopplade till risken att utveckla psykisk ohälsa. Tillsammans utgör dessa faktorer ett komplext etiologiskt nätverk av möjliga orsaker. Komplexiteten och den stora mängden möjliga orsaker leder till utmaningar i försöken att förstå etiologin bakom psykisk ohälsa. I den här avhandlingen tar jag avstamp i moderna ramverk för kausal inferens för att resonera kring möjliga orsaker till psykisk ohälsa hos studenter. Sökandet efter orsaker till de höga, och till synes ökande, nivåerna av psykisk ohälsa hos studenter ses i den här avhandlingen i ljuset av förändringar i studenters beteenden och miljö under de senaste decennierna.

Avhandlingen bygger på kohorten Sustainable University Life, som mellan 2019 och 2021 följde över 4000 universitetsstudenter med webenkäter var tredje månad under ett år, i syfte att identifiera faktorer av betydelse för psykisk och muskuloskeletal hälsa.

COVID-19-pandemin (eng. coronavirus disease) och de restriktioner som pandemin medförde ledde till abrupta förändringar av svenska universitetsstudenters vardag under våren 2020, framför allt i den mycket snabba övergången från campus- till distansundervisning. I början av pandemin fanns en påtaglig oro för hur den fysiska distanseringen skulle påverka studenters psykiska hälsa. I Studie I följde vi studenter från hösten 2019 och under det första halvåret av COVID-19-pandemin. Resultaten visade på små förändringar i medelvärden av depression-, ångest- och stressymptom under de första månaderna av pandemin jämfört med månaderna innan pandemin bröt ut. Våra resultat pekar mot

att COVID-19-pandemin hade relativt liten påverkan på svenska studenters psykiska hälsa på gruppnivå, även om en begränsning är att vi inte med säkerhet vet hur studenternas symptomnivåer hade utvecklats i frånvaro av pandemin.

En annan, mindre abrupt, förändring är att nivåerna av sömnstörningar har ökat påtagligt bland universitetsstudenter de senaste 20 åren. Sömnstörningar förekommer vid så gott som alla former av psykisk ohälsa, och är en väletablerad riskfaktor för en rad psykiatriska tillstånd. Det finns flera hypoteser kring vilka mekanismer som kopplar samman sömnstörningar och psykisk ohälsa. En hypotes är att sömnstörningar försämrar förmågan till emotionsreglering, vilket i sin tur tros vara kopplat till utvecklandet av psykiska symptom. Om denna hypotes stämmer finns möjligheten att sömnstörningar också skulle kunna öka sårbarheten för andra riskfaktorer. Om emotionsregleringsförmågan blir nedsatt skulle det kunna leda till att andra riskfaktorer, som t.ex. ensamhet, inte kan hanteras på ett adekvat sätt och därför får större negativ påverkan på den psykiska hälsan. I Studie II undersökte vi om kopplingen mellan depressionsymptom och ensamhet, perfektionistisk oro, riskbruk av alkohol och fysisk inaktivitet varierade beroende på sömnkvalitet i ett tvärsnitt av universitetsstudenter. Vi fann att alla de undersökta faktorerna hade tydliga kopplingar till depressionsymptom, men att styrkan i dessa samband var relativt opåverkade av sömnkvalitet. För perfektionistisk oro fann vi visserligen att kopplingen till depressionsymptom var något starkare hos dem som sov sämre, men detta förklarade endast en mycket liten del av variationen i depressionsymptom hos studenterna. Våra resultat tyder på att sömnkvalitet inte verkar påverka hur starkt ensamhet, riskbruk av alkohol och fysisk inaktivitet är kopplade till depressionssymptom.

Uppskjutandebeteende, även kallat prokrastinering, är vanligt förekommande hos universitetsstudenter och tros av vissa forskare ha ökat under de senaste decennierna. Ungefär hälften av universitetsstudenterna uppger att de prokrastinerar i sådan utsträckning att det hindrar dem att nå viktiga personliga mål. Det har föreslagits att prokrastinering skulle kunna öka risken för både fysisk och psykisk ohälsa genom att leda till högre stress, sämre hälsobeteenden och uppskjutande av att söka vård. Flera tvärsnittsstudier har funnit samband mellan prokrastinering och symptom på psykisk ohälsa. Vad som är hönan och ägget är dock fortfarande oklart utifrån dessa studier, då

prokrastinering även skulle kunna vara en följd av psykisk ohälsa. I Studie III undersökte vi därför sambanden mellan prokrastinering och en rad hälsoutfall nio månader senare, däribland symptom på depression, ångest och stress. Genom att statistiskt justera för tidigare nivåer av psykisk ohälsa och andra störfaktorer försökte vi komma närmare den kausala effekten av prokrastinering på olika hälsoutfall än vad som varit möjligt i tidigare tvärsnittsstudier. Vi fann att prokrastinering var kopplat till sämre nivåer på en rad hälsoutfall nio månader senare hos universitetsstudenter, även efter justering för tidigare hälsonivåer och andra möjliga störfaktorer. Dessa resultat stärker evidensen för att prokrastinering skulle kunna ha negativa hälsoeffekter hos studenter, även om vi inte med säkerhet kan utesluta att det kan finnas andra förklaringar till de observerade sambanden. Sambanden var också relativt svaga, så även om prokrastinering skulle kunna öka risken för ohälsa, så verkar den potentiella effekten på specifika hälsoutfall vara relativt svag.

Ytterligare en möjlig orsak till psykisk ohälsa är sexuella trakasserier och sexuellt våld. Sedan #Metoo-rörelsens start 2017 har medvetenheten om förekomsten av dessa problem ökat, även om det är oklart om detta har påverkat deras faktiska förekomst. Inte desto mindre rapporterar en stor andel av universitetsstudenter, särskilt kvinnor och personer från sexuella minoriteter, att de utsatts för sexuella trakasserier och sexuellt våld under sina universitetsstudier. Både sexuella trakasserier och sexuellt våld ses ofta som del av ett kontinuum av negativa sexuella erfarenheter av olika allvarlighetsgrad som spänner från sexuell jargong till sexuellt tvång och våldtäkt. Ett flertal studier visar på samband mellan sexuella trakasserier och sexuellt våld och psykisk ohälsa. En begränsning i den tillgängliga forskningen är att olika typer av sexuella trakasserier och sexuellt våld ofta klumpats ihop. Det har gjort att det finns relativt lite kunskap om hur olika typer av sexuella trakasserier och sexuellt våld skiljer sig åt avseende möjlig påverkan på psykisk hälsa. Det finns även begränsad kunskap kring hur dessa möjliga effekter förändras över tid. I Studie IV undersökte vi därför sambanden mellan att nyligen ha utsatts för olika typer av sexuella trakasserier och sexuellt våld och symptom på depression och ångest tre, sex och nio månader senare, hos kvinnliga och manliga universitetsstudenter. Vi fann att utsatthet för tre former av sexuella trakasserier och sexuellt våld var kopplade till senare nivåer av depression- och/eller ångestsymtom hos kvinnor: 1) en bred definition av sexuella trakasserier, 2) oönskad sexuell uppmärksamhet och 3) sex mot ens vilja. Den generella tendensen var att symptomnivåer hos utsatta

kvinnor var förhöjda efter tre månader, men att dessa sedan avtog vid sex- och niomånadersuppföljningarna för de flesta former av utsatthet. Ett undantag var de kvinnor som utsatts för sex mot sin vilja, där de förhöjda symptomnivåerna kvarstod under hela uppföljningstiden. Dessa tidstrender är dock osäkra och kan vara resultatet av slumpmässiga variationer. Vi såg också tendenser till skillnader i hur starkt olika former av sexuella trakasserier och sexuellt våld var kopplade till ångest- och depressionsymptom bland kvinnor, men även här är våra uppskattningar osäkra. När det gäller männen var estimaten osäkra överlag, varför vi avstår från att tolka dessa resultat. Överlag tyder resultaten på att utsatthet för sexuella trakasserier och sexuellt våld kan leda till förhöjda nivåer av ångest- och depressionsymptom hos kvinnor, även om vi inte kan utesluta andra förklaringar till de observerade sambanden. Det finns i resultaten visst stöd för hypotesen att effekten på psykisk ohälsa kan skilja sig mellan olika former av sexuella trakasserier och sexuellt våld, även om våra resultat innehåller en stor grad av osäkerhet.

I relation till de stora ökningar av psykisk ohälsa bland studenter som rapporterats var de potentiella effekterna på psykisk ohälsa som framkom i dessa studier relativt små. Symptomnivåer av depression, ångest och stress höll sig relativt stabila under de första månaderna av COVID-19-pandemin. Interaktioner mellan sömnkvalitet och andra potentiella riskfaktorer för depression svarade för en liten del av variansen i depressionsymptom. Prokrastinering samt att nyligen blivit utsatt för sexuella trakasserier eller sexuellt våld hade samband med högre nivåer av psykisk ohälsa, men sambanden var relativt svaga. De svaga sambanden är i sig inte förvånande, givet att det antas finnas en stor mängd faktorer kopplade till psykisk hälsa, vilket gör starka effekter hos vanligt förekommande riskfaktorer osannolika på gruppnivå.

Sammanfattningsvis stärker dessa resultat evidensen för att prokrastinering samt sexuella trakasserier och sexuellt våld skulle kunna orsaka psykisk ohälsa hos universitetsstudenter. Ett lika viktigt fynd var de relativt stabila nivåerna av psykisk ohälsa under COVID-19-pandemin. Även om dessa resultat är långt ifrån att förklara den höga förekomsten av psykisk ohälsa, så har avhandlingen spritt ljus över några aspekter av etiologin bakom psykisk ohälsa bland studenter.

Abstract

Mental disorders are among the leading causes of years lost due to disability in young people globally. Students are no exception, both mental disorders and mental health problems are common among university students with dramatic increases reported in recent years. The aetiology of mental disorders and mental health problems is generally assumed to be multicausal, with factors at different levels contributing to their development. Given the complexity of the causal network underlying mental health problems, it has been argued that a clear causal framework is needed when studying the aetiology of mental health problems. This thesis aimed to investigate some aspects of the potential aetiology of mental health problems among university students. Specifically, it focuses on four exposures at the psychosocial level presented in four studies: 1) the coronavirus disease (COVID-19) pandemic, 2) poor sleep quality, 3) procrastination and 4) sexual harassment and sexual violence. In addition to the discussion provided in each respective paper, this thesis discusses limitations and possible interpretations of our results from a modern causal inference perspective.

The four studies of this thesis are based on The Sustainable University Life (SUN) cohort. The SUN cohort followed 4262 university students from eight universities in and around Stockholm, Sweden, with web-surveys at five time-points over one year.

In Study I, we aimed to determine the mean trajectories of depression, anxiety, and stress symptoms among university students in Stockholm before and during the first months of the COVID-19 pandemic. For this, we included a subsample of 1836 university students that entered the SUN cohort before the outbreak of the COVID-19 pandemic, and were followed during the months before the pandemic, during the first wave of the pandemic and in the summer, months following the first wave of the pandemic. We found that mean depression, anxiety, and stress symptom levels were largely stable during the first wave compared to the months before the pandemic and decreased slightly during the following summer months. Our results indicate that mean levels of mental health symptoms did not change much during the early phase of the pandemic compared to before the pandemic.

In Study II, we aimed to determine whether sleep quality statistically interacts cross-sectionally with loneliness, risky alcohol use, perfectionistic concerns and/or physical inactivity in relation to depressive symptoms in university students. We conducted a cross-sectional study using baseline-data from all 4262 participants in the SUN cohort. We found that while all factors were associated with depressive symptoms, only perfectionistic concerns interacted with sleep quality in its relation to depression. This interaction was quite weak and explained only a small proportion of the variance in depressive symptoms. Overall, we did not find support for our hypothesis that poor sleep quality could interact with several different potential risk factors for depressive symptoms.

In Study III, we aimed to evaluate the associations between procrastination and sixteen subsequent health outcomes (including mental health symptoms, disabling pain, lifestyle behaviours and psychosocial health factors), measured 9 months later, among university students. We used data from all participants responding to the first follow-up in the SUN cohort (n=3525) and found that procrastination was related to several subsequent health outcomes, including symptoms of depression, anxiety and stress, while controlling for multiple potential confounders. Although we cannot rule out non-causal explanations for these associations, the results indicate that procrastination could have an effect on health outcomes among students, but that it is likely to be rather small for any specific health outcome.

In Study IV, we investigated the impact of recent exposure to different forms of sexual harassment and sexual violence; 1) unwanted sexual attention, 2) offensive sexual remarks, 3) presentation or distribution of sexist material, 4) uncomfortable touching, 5) being offered benefits for sex and 6) sex against ones will, along with a wide definition of sexual harassment: sexual harassment (wide subjective definition) on levels of depression and anxiety symptoms three, six and nine months later, for women and men, respectively. We conducted a cohort study using data from all women and men responding to the first follow-up in the SUN cohort (n= 3503). Our results showed that women recently exposed to 1) sexual harassment (wide definition), 2) unwanted sexual attention and 3) sex against ones will showed higher subsequent levels of depression and/or anxiety symptoms. The general trend was that all exposures were related to higher symptom levels at three months, but that this difference

between exposed and unexposed diminished over time, although these trends are uncertain with wide confidence intervals. The exception was exposure to sex against ones will, where exposed showed elevated symptom levels throughout the follow-up period. For men, the estimates were uncertain overall, and we refrain from interpreting these results. Our results indicate that recent exposure to different forms of sexual harassment and sexual violence may impact later depression and anxiety symptoms among women, and that there could be differences in the strength and long-term impact on mental health between different forms of sexual harassment and sexual violence. Again, we cannot, with certainty rule out non-causal reasons for these associations.

Interpreting any of these results as causal effects rests on multiple assumptions, which are discussed in the thesis. Determination of causal effects preferably relies on triangulation of results from different studies with different methodology. Overall, however, I believe that this thesis has strengthened the evidence that procrastination and sexual harassment and sexual violence may be causes of mental health problems among university students. An equally important finding, is that mental health seemed rather stable during COVID-19 pandemic, indicating that the pandemic may not have caused increased mental health problems on the group level.

List of scientific papers

I. **Johansson, F,** Côté, P, Hogg-Johnson, S, Rudman, A, Holm, LW, Grotle, M, Jensen, I, Sundberg, T, Edlund, K, & Skillgate, E. Depression, anxiety and stress among Swedish university students before and during six months of the COVID-19 pandemic: A cohort study. Scand. J. Public Health, 2021, 49(7), 741-749. doi: 10.1177/14034948211015814

II. **Johansson, F,** Côté, P, Onell, C, Källberg, H, Sundberg, T, Edlund, K, & Skillgate, E. Strengths of associations between depressive symptoms and loneliness, perfectionistic concerns, risky alcohol use and physical activity across levels of sleep quality in Swedish university students: A cross-sectional study. J, Sleep Res., 2023, 32(2), e13745. doi: 10.1111/jsr.13745

III. **Johansson F,** Rozental A, Edlund K, Côté, P, Sundberg, T, Onell, C, Rudman, A, & Skillgate, E. Associations Between Procrastination and Subsequent Health Outcomes Among University Students in Sweden. JAMA Netw Open. 2023;6(1):e2249346. doi: 10.1001/jamanetworkopen.2022.49346

IV. **Johansson, F,** Edlund, K, Sundgot-Borgen, J, Björklund, C, Côté, P, Onell, C, Sundberg, T, & Skillgate, E. Sexual Harassment, Sexual Violence and Subsequent Depression and Anxiety Symptoms among Swedish University Students: A Cohort Study. Manuscript.

Other relevant papers

1. Edlund, K, Sundberg, T, **Johansson, F,** Onell, C, Rudman, A, Holm, LW, Grotle, M, Jensen, I, Côté, P, & Skillgate, E. Sustainable UNiversity Life (SUN) study: protocol for a prospective cohort study of modifiable risk and prognostic factors for mental health problems and musculoskeletal pain among university students. BMJ Open, 2023, 12(4): e056489. doi: 10.1136/bmjopen-2021-056489
2. **Johansson, F,** Côté, P, Hogg-Johnson, S., & Skillgate, E. Depression, anxiety and stress among Swedish university students

during the second and third waves of COVID-19: A cohort study. Scand. J. Public Health, 2021, 49(7):750-754. doi:10.1177/14034948211031402

3. **Johansson F**, Magnusson K. Sexual Harassment, Sexual Violence, and Mental Health Outcomes: Causal Inference with Ambiguous Exposures [Internet]. OSF Preprints; 2023. Available from: osf.io/d2qvy. Reprinted in the Appendix under the CC BY 4.0 license.

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List of abbreviations

CI	Confidence interval
COVID-19	Coronavirus disease
DAG	Directed Acyclic Graph
DASS-21	Depression, Anxiety and Stress Scale 21 item version
DSM	Diagnostic and Statistical Manual of Mental Disorders
PSQI	Pittsburgh Sleep Quality Index
RDoC	Research Domain Criteria
RR	Risk ratio
SHV	Sexual Harassment and Sexual Violence
SUN	Sustainable University Life cohort

Introduction

> *And the disease called the Sacred arises from causes as the others, namely, those things which enter and quit the body, such as cold, the sun, and the winds, which are ever changing and are never at rest.* [1]
>
> - Hippocrates, 400 BCE

> *Research in psychiatry generates a bewildering diversity of causal claims. A reader of any of our major journals such as this one confronts efforts to prove that the risk for psychiatric disorders is influenced by economic, cultural, psychological, genetic, neural, brain structural and molecular influences. Underlying this broad range of casual claims is the fault-line of the mind–body problem. How do causes of psychiatric illness best understood as mental processes interrelate with those that reflect physical or biological events? Common sense suggests that causal pathways exist in both directions between the mind and the brain. However, despite much effort, the nature of such casual paths has remained frustratingly obscure. […] We argue that recent advances in the philosophy of science – particularly in the nature of causation and explanation –are of substantial relevance to our field. Specifically, interventionist causal models can provide a framework for the field of psychiatry that applies equally to the broad array of potential causal processes at work in psychiatric illness.* [2] (p.881)
>
> - Kenneth Kendler, 2009

Aetiology refers to the development of disease and is defined as 'the cause, set of causes or manner of causation of a disease or condition' [3]. For mental health and ill-health, the aetiologic search for causes has been ongoing for a long time. In 400 B.C.E., Hippocrates wrote the text *On the Sacred Disease* where he reacted against the then current understanding that mental illness was caused by the gods. Instead he suggested that mental illness, like all other illnesses, was the consequence of imbalanced

bodily humours, which in turn where affected by external forces like cold, sun and wind [1]. His contemporary Plato suggested other causes such as bad character, bad education and excess seed [4]. Later, medieval authors added causes such as alcohol intake, overwork, and grief [5]. The list of causes of mental ill-health continued to evolve and grow. By the beginning of the 19th century, medical texts contained long lists of both physical and moral causes of mental illness [6].

In the latter half of the 19th century, this multicausal perspective on mental and physical diseases began to decline. The emergence of germ theory shifted the focus from multicausal explanations of disease to a monocausal perspective that has been termed the 'doctrine of specific aetiology' [6]. This doctrine stated that diseases should be conceptualized by their specific causes, rather than by descriptions of their symptoms. The focus on specific causes of diseases led to major medical advances in the understanding and treatment of both infectious and deficiency diseases. In psychiatry too, this aetiologic paradigm led to a shift from multicausality to monocausality. Indeed, the focus on specific biological causes for mental disorders was seen as paramount to consolidate psychiatry's role within the field of medicine [7]. With a few exceptions, such as that discovery of the bacterium Treponema palladium as the cause of Syphilis, such specific causes remained elusive to the field of mental health.

With the rise of non-communicable diseases such as cardiovascular diseases and cancers as the major contributors to disease burden, the doctrine of specific aetiology was proving less and less useful, and from the 1950's, epidemiology again turned to models of multicausality [8] [1].

In 1977, psychiatrist George Engel reacted against the doctrine of specific aetiology, now in terms of the biomedical model, by introducing the biopsychosocial model [9]. Engel argued that not just psychiatry, but medicine overall, needed to move away from the reductionist biomedical model to consider psychological, social, and behavioural causes in the aetiology of illness and disease. The biopsychosocial model helped

[1] Still, the focus on specificity as a hallmark of aetiological discovery was echoed in the classic Bradford Hill criteria from the 1960's, where specificity was proposed as one of the criteria of causality, although nuanced by noting that many diseases had multiple determinants.

7. Hill AB. The Environment and Disease: Association or Causation? Proc R Soc Med. 1965;58(5):295-300.

reconcile different strands of aetiologic thinking within psychiatry, but its eclectic aetiological approach was also criticised for being non-directive, letting researchers freely choose which of the 'bio', 'psycho', or 'social' parts to focus on [10]. Coincidently, the American Psychiatric Association developed and published the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III). In contrast to earlier editions, DSM-III provided a descriptive nosology of mental disorders making claims about their aetiology [11]. In part, this has been viewed as a response to the biopsychosocial model's inability to clearly separate mental health from ill-health [12].

Today, mental health and ill-health are still, for practical purposes, categorized as discrete mental disorders based on their symptoms [13]. Most research, however, indicate that mental disorders are better understood as dimensional rather than categorical conditions [13]. Mental health can range from health to mild mental distress to severe mental disorders, without clear boundaries between health and illness [14]. There are also no clear-cut boundaries between most types of mental disorders [14-16] with comorbidity as the norm rather than the exception [17]. Given the lack of clear distinctions between health and ill-health and between different kinds of mental disorders, the term *mental health problems* is often used as an encompassing term including both milder and more severe forms of mental distress of different kinds [18].

Several approaches have been proposed for how to understand and deal with the fuzzy diagnostic borders of mental health in aetiologic research. In 2009, the American National Institute of Mental Health launched the Research Domain Criteria (RDoC) as a framework for investigating mental health [19]. RDoC advocates for shifting the focus from diagnostic categories to more basic dimensions of human functioning (such as attention, reinforcement, fear etc.). A focus on narrower psychological processes and their biological underpinnings is suggested as a promising way forward for understanding mental health [20]. This arguably reductionist approach is contrasted, or perhaps expanded, by the causal systems perspective. From this perspective, mental health problems are viewed as the product of a system of mutually reinforcing causal mechanisms, that can include both external causes and the symptoms themselves [21, 22]. From a casual systems perspective, the understanding of narrower psychological processes advocated by RDoC

is necessary, but not sufficient for understanding mental health and mental disorders. To be able to fully understand how the causal system works, one must also understand how the individual parts *work together* [23]. In relation to mental disorders, this means understanding how causes at different levels reinforce, sustain, and affect one another to give rise to what we recognize as mental health problems and mental disorders [23].

Although monocausal thinking still lingers [6], the aetiology of mental disorders and mental health problems is today generally accepted as multicausal, with risk factors at different levels (e.g. biological, psychological, and social) contributing to their development [6, 24, 25]. Unsurprisingly, given the high levels of comorbidity, risk factors are often shared across different mental disorders. At the biological level, many mental disorders share common genetic origins [26-28]. At the psychosocial levels, risk factors such as sleep disturbances [29] stressful life events, [30], adverse childhood experiences [31], neuroticism [32], perfectionism [33] and loneliness [34], among others, are shared across a range of common mental disorders (and often also among other adverse health outcomes). Further, many of these potential causes of mental health problems are interconnected with each other into what can be described as a 'web of causation' [35] or a 'causal network' [36].

Adding to the complexity of the aetiology of mental health problems is that both mental health problems and many of its potential causes are fluctuating and may bidirectionally influence each other over time, so that a variable may be both a cause and a consequence of mental health depending on the timeframe (cf. [37-40]). Imagine a student who after a night of poor sleep is tired and unable to concentrate. Not being able to focus raises his anxiety about his upcoming exam, and when night comes, he is unable to fall asleep as he lays in bed worrying that he might fail the course. Within a 24-hour cycle, sleep disturbances have been first a cause and then a consequence of anxiety. These kinds of feedback processes are common with behavioural, psychological, and psychosocial risk factors of mental health problems and are commonly described as vicious circles in psychological treatments [41, 42], such as the feedback process between fear and avoidance characterizing many anxiety disorders.

This diversity and complexity of both the potential influences on mental health, and the mental health concept itself, is bewildering, and may

invite to what has been described as an 'uncritical holistic approach' where everything is seen as important for mental health [2]. To avoid this uncritical holism and keep a clear head with regards to the pluralistic aetiology of mental health problems, it has been argued that a clear causal framework is needed [2, 24, 43].

Two of the dominating modern frameworks for causal reasoning are the Potential Outcomes Framework developed by Rubin [44] and Holland [45], and Structural Causal Models with its calculus of causation and causal graphical models developed by Pearl [36]. In both these frameworks, causation is defined as what would happen *under a certain intervention*. Causes are seen as difference-makers in the sense that interventions on the causative variable would make a difference for the outcomes, in this case mental health. These perspectives have the advantage of easily accommodating to causes at different levels regardless of disease pathways or mechanisms [24]. This is helpful with regards to mental health, where such knowledge is frustratingly scarce.

In this thesis, I adopt a dimensional view on mental health, viewing it as a continuum of mental distress with multicausal aetiology, a perspective akin to the causal systems perspective but also reflected in the biopsychosocial model. I further adopt the potential outcomes framework of causal inference, described in more detail below, to try to reason about some aspects of the aetiology of mental health problems. My aim is to reflect these perspectives both in the operationalization of the constructs and in the analytic strategies.

1 Literature review

1.1 MENTAL HEALTH AMONG UNIVERSITY STUDENTS

Mental disorders rank as one of the top reasons for non-fatal health loss [46], and are among the leading causes of years lost due to disability in young people globally [47]. Students are no exception, both mental disorders and mental health problems are common among university students [48-52]. Recent data from the World Health Organization show that one-fifth of university students met criteria for a mental disorder in the preceding year and few of these received treatment [51]. Both self-reported symptom levels and diagnosed mental disorders have increased considerably during the last decades among university students and young people overall [52-56]. From 2013 to 2021, the proportion scoring above screening thresholds increased by 135% for depression and 110% for anxiety in a large sample of US students [52]. Similar trends have been observed in Sweden. Population level data shows that the number of young persons (18-24 years) who had been diagnosed with a psychiatric disorder or received psychotropic medication increased from 10% to 16% for women and from 6% to 10% for men from 2006 to 2016, with most of these cases being depression or anxiety disorders [55]. Further, mental health has been shown to deteriorate during university studies [48, 57], and some studies indicate that mental health problems are more common among university students than in the general population as well as compared to age-matched peers [49, 58, 59].

How much of these increases that are due to rising levels of psychological suffering versus changes in other factors such as increased mental health awareness, reduced stigma, and better availability of care, is still unclear. Nonetheless, prevalence figures are high among university students, and besides the psychological suffering, mental health problems are also associated with several adverse educational outcomes, such as increased risk of attrition [51], academic impairment [60], reduced occupational preparedness and future work performance [61].

Regarding the causes of mental health problems among university students, systematic reviews focusing on young persons and undergraduate students have identified a wide range of potential risk factors at different levels (intrapersonal, interpersonal, and systemic) [50, 62]. They have also shown that the results and the quality of the studies

are mixed and pointed to the need for further high-quality longitudinal studies in order to draw robust conclusions [50]. With reports of increasing mental health problems among university students, the search for causes may be guided by parallel changes in cognitive, behavioural, and environmental factors in the student population.

The social restrictions imposed in response to the outbreak of the coronavirus disease pandemic (COVID-19 pandemic) in 2019, resulted in some of the most profound changes to students' environment in the last years, with considerable concerns about the potential adverse effects to students' mental health. Second, sleep disturbances have increased substantially among university students during the last decade [63]. A third factor shown to be highly prevalent and suggested to be increasing among university students is procrastination [64, 65]. Fourth, the increased awareness of sexual harassment and sexual violence (SHV) in the wake of the #MeToo movement in 2017 is yet another change, although one hopefully leading to decreased levels of these exposures.

This thesis focuses on the roles of the COVID-pandemic, sleep quality, procrastination and SHV as potential causes of depression and anxiety symptoms among university students.

1.2 COVID-19 PANDEMIC

When the COVID-19 pandemic hit in early 2020 it caused profound disruptions of the daily lives of citizens around the world. There were major concerns about how the pandemic, and the societal restrictions used to maintain the spread of the virus, would impact on mental health [66, 67]. For Swedish university students, the most noticeable restriction was the cancelling of all campus-based education between March 2020 and September 2021, forcing universities and students to rapidly adapt to online education.

At that time, our research group was in the middle of the data collection for the Sustainable UNiversity Life (SUN) cohort, that provides the basis for this thesis. The SUN cohort thus contain measurements on the same individuals both before and during the COVID-19 pandemic. This gave us an unique opportunity to investigate changes in mental health among Swedish students during the pandemic, compared to before the pandemic (Study I).

At that time, there were limited evidence of the mental health impact of the pandemic both among university students and in other populations.

Early cross-sectional data had indicated high levels of depression and anxiety, and that persons with pre-existing mental health problems were worse off during the early phase of the pandemic [68]. A recent systematic review, relying mainly on cross-sectional studies with matched control groups measured prior to the pandemic, similarly reported increased prevalences of mental health problems early in the COVID-19 pandemic [69]. Relying on cross-sectional studies to measure change over time has, however, received critique. Prevalence estimates from self-administered questionnaires have been argued to be highly variable, even among randomly drawn samples, and thus ill-suited to investigate changes over time [70].

In general, these initial cross-sectional findings were not supported by later systematic reviews of prospective studies. Three systematic reviews of prospective studies on mental health during the COVID-19 pandemic have compared mental health during the pandemic to pre-pandemic levels and found small or minimal changes in symptom levels [71-73]. One review found small increases in mental health symptoms during the first wave [72] and the two others reported small increases in mean depression symptoms during the first wave of the pandemic, with symptoms returning to pre-pandemic levels during mid-2020 [71, 73]. Similarly, in a majority of countries, including Sweden, suicide rates were not observed to increase during the pandemic, but instead more commonly decreased [74]. One systematic review stands out by reporting large increases in depression and anxiety symptoms during the first wave of the pandemic, with decreasing levels in the summer of 2020 [75]. However, this review included only longitudinal studies without pre-pandemic time-points.

All these systematic reviews note a large heterogeneity between studies, which may be the result of the COVID-19 pandemic having different impact in different parts of the world. Substantial differences between samples would not be surprising, given that the spread of the virus, as well as the societal restrictions, have varied considerably between countries. Thus, currently, the best knowledge suggests that on a global level, mental health remained rather stable during the COVID-19 pandemic. These results show, perhaps surprisingly, high levels of resilience to the effects of the COVID-19 pandemic in terms of mental health.

1.3 SLEEP DISTURBANCES

Sleep disturbances have been increasing among university students during the last decade [63], are known to co-occur with almost all mental disorders [76], and have been proposed as a transdiagnostic risk factor for various forms of psychopathology [77]. Several systematic reviews have identified sleep disturbances as a risk factor for depression as well as other mental disorders, both among students and in the general population [50, 78, 79].

Emotion regulation is one of the mechanisms suggested to mediate the effects of sleep disturbances on mental health [77, 80-82]. Emotion regulation is known to be affected by sleep [81], and has been suggested as an important process involved in the maintenance and development of various forms of psychopathology [83-85]. By affecting emotion regulation, sleep quality could potentially also interact with other risk factors of mental health problems (Figure 1). The reasoning is that if poor sleep quality impairs the ability to down-regulate negative emotions evoked by other risk factors, then sleep disturbances may amplify the effect of these other risk factors on mental health problems. If this would be the case, it would be of high clinical importance, as improving sleep could have the potential to increase resilience to a range of other risk factors.

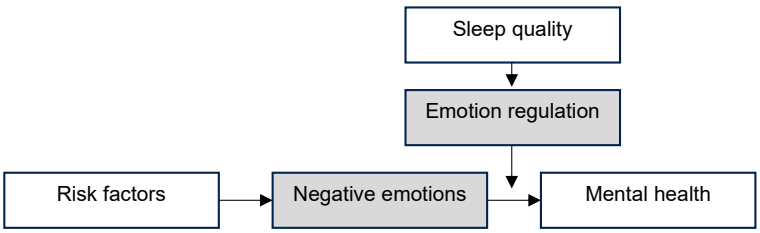

Figure 1. Theoretical model of sleep quality as an effect measure modifier of risk factors for mental health problems. Grey boxes represent constructs that were not measured in Study II.

There is some preliminary support for this hypothesis. Cohort studies have shown that the effect of negative life events, high job stress and parenting stress on later depression and mood is stronger among persons with poor sleep [86-88]. In cross-sectional studies, social isolation [89], chronic medical conditions [90], and problematic mobile telephone use [91], has been more strongly associated with depression symptoms among poor sleepers. Given the wide variety of potential risk factors in these studies, a wide focus, including different kinds of potential risk

factors (e.g. psychological, behavioural and social), is warranted when examining interaction effects between sleep quality and other potential risk factors.

Complex causal structures, such as the causal network underlying mental health, are likely to contain non-linear association and interactions between variables [23]. A limitation of most prior research on sleep quality and depression is that is has assumed linear associations and interactions, which may lead to misrepresentations of non-linear interactions and associations [92, 93]. Another problem with the current evidence is that interaction analyses often are secondary research questions. This increases the risk for publication bias, as it seems probable that secondary findings are often left out if non-significant, which puts their replicability into question.

In sum, sleep quality could potentially act as an amplifier, having synergistic effects with other risk factors in the causals system underlying mental health problems. Although much theorizing on emotion regulation and sleep points towards this possibility, few studies have tested this hypothesis.

1.4 PROCRASTINATION

Procrastination is defined as freely choosing to postpone intended actions despite expecting negative consequences [64]. Occasional procrastination seems like near universal human behaviour and may have few negative consequences in the long-term. For some, however, procrastination can become a pervasive behaviour pattern that may affect many different areas of life [64, 94]. Below, the term procrastination is used to refer to this more pervasive and trait-like kind of procrastination.

It has been estimated that roughly half of the university student population engages in repeated procrastination to an extent where procrastination strongly interferes with reaching personally relevant goals [65, 95]. Among these, almost all view their procrastination as problematic and about a third have considered seeking professional help for their procrastination [65]. The relatively free structure of university studies places high demands on students' capacity to self-regulate and this has been suggested as an explanation for the high frequency of procrastination among students. This free structure may also leave

students prone to procrastination more vulnerable to its potential negative effects [96].

Procrastination is associated with several adverse mental health outcomes, such as depression, anxiety, and stress [94, 97, 98], but longitudinal evidence on associations between procrastination and health outcomes is scarce [2]. This is problematic, since procrastination has been suggested as both a cause and a consequence of mental health problems [98], and the lack of longitudinal studies precludes conclusions on the causal direction between procrastination and various health outcomes.

On the one hand, procrastination has been suggested as a cause of mental and physical health problems. For instance, the procrastination health model suggests that procrastination leads to elevated stress levels, reduced health behaviours and delayed treatment seeking [100, 101], which in turns leads to poor overall health. Procrastination has also been suggested to work as a short-term emotion regulation strategy, where short-term mood repair is prioritized over long-term goals [102]. Avoiding or discontinuing aversive tasks may temporarily improve mood but is likely to have detrimental mental health effects in the long run. For instance by giving rise to negative self-evaluations [103], rumination [104], stress [101], and feelings of guilt and shame [105].

On the other hand, motivational problems and low energy levels are a well-known aspect of depression [106], that may make depressed people more likely to procrastinate. High anxiety levels could also increase procrastination by increasing the perceived task aversiveness and fear of failure, although the support for this hypothesis is mixed [64, 107].

Currently, longitudinal studies are needed to try to disentangle the potentially bidirectional effects between procrastination and health.

1.5 SEXUAL HARASSMENT AND SEXUAL VIOLENCE

SHV are often seen as part of a continuum of negative sexual experiences, ranging from sexual remarks to sexual assault and rape [108-110]. Unfortunately, many of these negative experiences are common among university students [109, 111, 112], especially among women and

[2] Following the publication of Study III, one more longitudinal study of has found evidence of associations between procrastination and subsequent health.
99. Sirois FM, Stride CB, Pychyl TA. Procrastination and health: A longitudinal test of the roles of stress and health behaviours. Br J Health Psychol. 2023;28(3):860-75.

sexual minorities [111]. Prevalence figures of SHV tend to vary widely between studies, partly due to difficulties defining these exposures consistently across studies [109]. In general, however, unwanted sexual attention and gender harassment are more prevalent, while the most severe forms of sexual coercion like sexual assault and rape are less common [111, 112]. In a recent large study of Norwegian students, about 22% of the women and 6% of the men reported having experienced at least some form of SHV during the past year. Among the women, 14% reported experiences of sexual remarks or suggestion, 11% unwanted touching, 0.4% reported experiencing rape and 0.5% attempted rape during the past year [113].

Several cross-sectional studies have shown that exposure to sexual harassment is associated with psychological distress [114-116] and reduced academic satisfaction [114] among university students. Sexual harassment is also associated with subsequent levels of symptoms of depression and anxiety as well as problematic drinking among university students [40, 117], and suicide and suicide attempts in working populations [118], even after controlling for baseline levels of mental health. Similarly, sexual violence experienced during the first semester of university studies is associated with later levels of depression and anxiety symptoms among women, even after adjustment for baseline mental health and prior exposure to sexual violence [119], and to lower subsequent academic performance [120].

Less is known, however, about how specific forms of SHV may impact on mental health. With SHV seen as a continuum of negative experiences of varying severity, it seems plausible that the mental health effects may differ depending on the severity of the offense. There are some cross-sectional and cohort studies indicating that sexual coercion has stronger associations with mental health problems, than does sexual harassment in general [40, 121, 122]. However, in most prior research, different forms of SHV have been combined into composite measures. Not only does this obscure differences in mental health impact between different forms of SHV, but it also complicates causal inference, as well as the interpretation of the estimate and the generalizability of the estimates, as is expanded upon in the Appendix.

Currently, there is therefore a need for research into the mental health effects of specific forms of SHV, both from a short- and a long-term perspective.

1.6 AN INTRODUCTION TO CAUSAL INFERENCE WITH MENTAL HEALTH OUTCOMES

These four exposures preside at different aetiologic levels: societal (COVID-19 pandemic), interpersonal (SHV) and psychological (procrastination and sleep disturbances). With a focus on aetiology, we are, by definition, required to view these exposures as potential causes. Traditionally, it has often been advised not to mention causality outside the realm of randomized controlled trials. This is changing, however, following rapid developments in the field of causal inference during the last decades [123]. These developments are not primarily ones of statistical methods, but rather the clarification of under which conditions causal effects can be estimated. Together with the advancement of conceptual tools such as directed acyclic graphs (DAGs), these developments have clarified under what circumstances causal effects can be estimated, even when randomization is not possible.

Modern frameworks for causal inference have been widely adopted in epidemiology, especially by epidemiological methodologists [123], together with vivid debates on how to incorporate these methodological advances into adjacent applied fields such as social epidemiology [124-127]. Still, these modern frameworks for causal inference is relatively uncommon in health care science and many social sciences like psychology. The lingering 'taboo' against explicitly stating causal research questions in observational research have been argued to obscure the actual research goals, impair study design and analysis, and to hamper scientific discussions [128, 129]. This is especially true regarding aetiologic research. Therefore, rather than trying to avoid causality, I devote some of this literature review to the theoretical framework underlying the use of words like 'effect', 'impact' and 'cause'.

1.6.1 Potential outcomes

The Potential Outcomes' Framework defines a causal effect as the contrast between two potential outcomes [44, 45, 123]. A potential outcome is the level of the outcome that would be observed if an individual's exposure status was, possibly contrary to fact, set to a certain exposure level. Let A denote an individual's exposure status, and $Y^{a=1}$ the potential outcome under exposure level a=1 (read as the potential outcome of setting exposure a to 1). We can then compare this potential outcome would observed under another exposure level, say a=0, ($Y^{a=0}$). The individual causal effect is defined as a contrast (e.g. difference or

ratio) between these two potential outcomes, $Y^{a=1} - Y^{a=0}$. This could be, for instance, a person's anxiety levels had she been exposed to a sexually offensive joke ($Y^{a=1}$), versus the anxiety levels she would have had she not been exposed to that joke ($Y^{a=0}$). The fundamental problem of causal inference is that only one of these potential outcomes can be observed [45]; the one corresponding to the individual's actual exposure status. For this reason, we generally cannot calculate individual causal effects. The central task for causal inference is to replace the potential outcomes with the observed outcomes. On group level, this can be done under three central conditions: exchangeability, positivity, and consistency [123]. [3]

1.6.2 Exchangeability and causal graphs

Exchangeability is the best known of the three conditions required for replacing the potential outcomes with our observed outcomes, commonly known as the assumptions of no unmeasured confounding and no selection bias. Formally, exchangeability means that the potential outcomes are independent of the actual exposure [123]. This means that the expected outcome among the unexposed is the same as that of the exposed, had they contrary to fact been exposed. In essence, this condition states that the groups under comparison, the exposed and the unexposed group, must be comparable on all characteristics of importance to the exposure – outcome relationship (i.e. no confounding or selection bias).

While the Potential Outcomes Framework offers an intuitive definition of a causal effect, it offers no straightforward way to determine when exchangeability is likely to hold. Here, causal graphs come in as a valuable tool [36]. One commonly used type of causal graphs are directed acyclic graphs (DAGs), that can be used to determine when exchangeability will hold and when it will not. A DAG is a non-parametric representation of a researcher's causal assumptions, with causal relations represented by arrows between variables. A series of variables that are connected by arrows are called a path. If a path connecting any two variables is open, the variables will be associated. A

[3] There are other assumptions underlying causal inference, such as that of no interference between units that is not discussed here. General statistical conditions for correct estimation such as no model misspecification and no measurement error (misclassification) apply as well but are not specific to causal inference.

path is open unless 1) there is a collider on the path, meaning that a variable on a path is a common effect of two other variables on the path (A → B ← C) or 2) we have conditioned on any variable on the path that is not a collider. Conditioning on a collider, on the other hand, will open a blocked path.

If the DAG is correct (and does not exclude variables of importance), it can be used to identify exchangeability by using the so-called *backdoor criterion* [36]. The backdoor criterion states that exchangeability is guaranteed in the absence of any open backdoor paths (paths other than the causal path of interest) between the exposure and the outcome. The DAG also helps identify variables that, if adjusted for, would block open backdoor paths to create *conditional exchangeability*.

The most commonly acknowledged threat to exchangeability is confounding: common causes of the exposure and the outcome. Another important threat is selection bias [130, 131], which is depicted in the DAG in Figure 2.

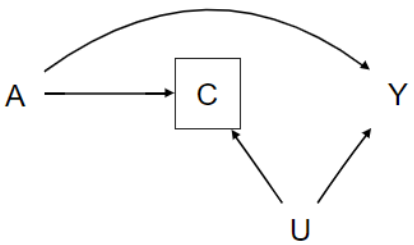

Figure 2. Directed acyclic graph showing selection bias due to conditioning on selection (C) that is a common effect of exposure (A) and risk factors (U) affecting both selection and the outcome (Y).

Here, the exposure A leads to loss to follow-up, denoted C for censoring. As does some other risk factors of Y, denoted by U. Since censoring, C, is a common effect of A and U, conditioning on C (which happens naturally with loss to follow-up and is represented by a box around the variable) opens the path A→ C ← U→ Y that will bias the estimate of the effect of A on Y. This can be handled by conditioning on the other factors (U) related to censoring which will again block the backdoor path, or by weighting techniques [132], or by performing multiple imputation, assuming data is available on all factors relating to censoring.

One of the benefits of DAGs is that they make explicit the assumptions needed for causal inference. This feature is what makes them such practical tools for identifying causal effects, but it also has a side-effect: a humbling reminder about potential lack of knowledge and the many assumptions you need to make for causal inferences. Especially when applied to multicausal conditions like mental health, the multitude of interrelated factors assumed to affect mental health makes the list of potential confounders very long, and any DAG may look like what a critic described as 'arrow salad' [133]. Since the causal knowledge necessary to design a credible DAG is often unavailable, with mental health as no exception, other practical criteria for confounder selection have been proposed [134]. Still, DAGs are fundamental for thinking and communicating clearly about causality, which is highly needed when approaching such complex causal structures as the aetiology of mental health problems.

1.6.3 Positivity

Perhaps less known among the conditions for causal inference is that of positivity, stating that for each covariate position in the data there must be a non-zero probability of exposure, such that all participants could possibly have been exposed. Violations of positivity will arise if participants in a certain covariate level will never be able to have the exposure. In a study on the mental health effects of childbirth, for instance, people without wombs could not possibly be exposed to giving birth to a child. This kind of positivity violation, when some participants cannot possibly have a certain exposure level, is referred to as a structural positivity violation [132] and is relatively easy to spot and avoid. A more subtle form of positivity violation may arise when there are so many covariate levels that at certain covariate positions (combinations of different covariate values) we find only one exposure level, or even no participants at all. Such situations are referred to as practical positivity violations [132]. Practical positivity violations are nearly certain to occur in many observational studies, when sample size is restricted and there are many covariates leading to highly stratified data. With continuous covariates, practical positivity violations are unavoidable given the infinite number of possible covariate values [132]. In such cases, parametric models are often used to smooth over covariate positions where some exposure levels are not present. This is done under the additional *no model misspecification assumption*. So, while

structural positivity may be seen as a identifiability problem, practical positivity is more of a modelling problem [135].

With continuous and dimensional constructs like mental health, where the number of potential confounders is large, parametric models are needed to interpolate covariate positions where data is scarce. Adding to the complexity is that complex causal systems, like those likely to underly mental health problems, are likely to contain non-linear associations and interactions [23]. With the true causal model unknown, there is often necessity to select between different reasonable model specifications. This problem is approached in Study II, using cross-validation to select between model specifications, although in this study with the purpose of description rather than causal inference.

1.6.4 Consistency

The last, and perhaps least known, condition for causal inference is the *consistency* condition. This condition states that for each person, there can be only one potential outcome for each exposure level, and that the potential outcome matches the observed outcome corresponding to the observed exposure level. At first glance this may seem like a truism, but the consistency condition is near impossible to fulfil with psychosocial exposures. The reason is that psychosocial exposures, which are arguably of high interest in relation to mental health, are nearly impossible to operationalize with such precision that they hold the same thing for all respondents. If an exposure can have different versions, holding different meanings for different respondents, the potential outcomes may differ depending on what exposure-version we consider. Exposure to procrastination, for instance, may have different effects on mental health depending on whether we consider procrastination in relation to cleaning the house, in relation physical activity or in relation to contacting friends. I refrain from digging deeper into this issue here, and refer to the Appendix, where the consistency condition is discussed at length.

1.6.5 Time-varying exposures and outcomes

A further challenge for causal inference with mental health outcomes is that both mental health and many of its potential causes varies over time, creating even more complex causal structures [136]. One such example is the effect of SHV on symptoms of depression and anxiety. Both SHV and depression and anxiety symptom levels vary over time, and prior

research indicate that there may be bidirectional effects between them [40]. In causal inference jargon, there is potential feedback between the exposure and the outcome over time [136] (Figure 3).

At the same time, mental health problems and many of its potential risk factors show some degree of stability over time. Measurements taken at any single time-point will therefore serve as a proxy for prior levels as well. This kind of semi-stability over time is common for psychosocial exposures, where there may be state-like variability over time, but also stable, trait-like between-person differences. This forces us to think carefully about the temporality of exposure and outcome measurements. The DAG in Figure 3 depicts depression symptoms at baseline (Dep_0) as a mediator of the effect of prior SHV (SHV_{-1}) on the outcome (Dep_2). However, due to feedback between exposure and outcome, depression symptoms at baseline (Dep_0) are at the same time a confounder for the effect of SHV at follow-up 1 (SHV_1) on the outcome (Dep_2).

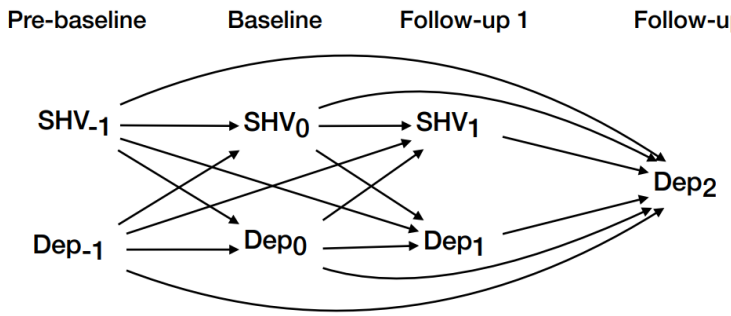

Figure 3. Directed acyclic graph showing a time-varying causal structure between sexual harassment and violence (SHV) and depression symptoms (Dep) at different timepoints, denoted by subscripts, before and during the follow-up period.

Followingly, if we are interested in the *accumulated* effect of SHV on mental health and use SHV at baseline as a proxy for the history of SHV, baseline mental health symptoms should not be controlled for, as they are on the causal path from earlier SHV to the outcome. However, if we are interested in the effect of *recent* exposure to SHV, baseline levels of mental health symptoms must be controlled for as they induce confounding. Which of these to effects that are of interest, the accumulated effect of exposure over time or the effect of recent exposure conditional on prior exposure and outcome levels, depends on the research question. If we are interested in the effect of prolonged

exposure to SHV, the former is of more interest. On the other hand, if we imagine that our results may be used to guide interventions on SHV here and now, the latter is arguably of more interest, since no interventions will be able to change prior levels of SHV or mental health.

Estimating accumulated effects has several challenges, including the need for very long follow-up times to accurately measure the exposure history. In practice, follow-up is often started at a later time-point when exposure may already be present. Then the exposure is generally referred to as a prevalent exposure (as it is already prevalent at the start of the study). This is contrasted by incident exposures, where follow-up begins before or at the time of exposure. Prevalent exposures are problematic, as they are subject to a wider range of biases than incident exposures [137].

One such bias is selection bias. In our case, where focus is on university students, we have conditioned on being a university student by design (S in Figure 4). It is likely that being at university (S) is a collider between many exposures (A) and prior mental health (Y_0). Procrastination, for example, is a behavioural pattern that is likely to be present before entering university and one that may affect the probability of entering university (A → S), since persons prone to procrastination may find academic activities more aversive or harder to complete. Mental health is also likely to influence the probability of entering university (Y_0 → S). This makes university attendance a collider opening a backdoor-path (A → S ← Y_0 →Y_1) biasing the estimate of the effect of A on Y_1, unless Y_0 is also conditioned on.

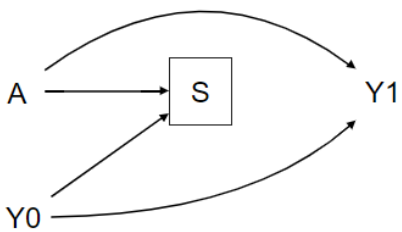

Figure 4. Directed acyclic graph showing selection bias where selection to the study sample (S) is affected by both exposure (A) and prior outcome levels (Y0).

For the reason described above, recent methodological papers have argued for the need for statistical control for prior exposure levels as well as prior outcome levels, in order to mimic the effect of incident or recent

exposure rather than prevalent exposure [136, 138, 139]. Especially in situations where the full exposure history is not available to us. This approach allows for the estimation of how changes in the exposure are related to the outcome [140]. Study III and IV of this thesis focus on the effect of recent exposure.

2 Research aims

2.1 STUDY I

First, to describe the trajectories of depression, anxiety, and stress symptoms among university students in Stockholm before and during the first six months of the COVID-19 pandemic. Second, to determine whether trajectories of depression, anxiety and stress symptoms were modified by pre-pandemic loneliness, poor sleep quality or mental health problems.

2.2 STUDY II

First, to determine whether sleep quality statistically interacts cross-sectionally with loneliness, risky alcohol use, perfectionistic concerns and/or physical inactivity in relation to depressive symptoms in Swedish university students. Second, to explore different functional forms to determine which best describes these potential associations and interactions.

2.3 STUDY III

To evaluate the associations between procrastination and 16 health outcomes at the end of a 9-month period among Swedish university students. The outcomes include mental health, disabling pain, and several unhealthy lifestyle behaviours and psychosocial health factors.

2.4 STUDY IV

To investigate the impact of recent exposure to different forms of sexual harassment and sexual violence; 1) unwanted sexual attention, 2) offensive sexual remarks, 3) presentation or distribution of sexist material, 4) uncomfortable touching, 5) being offered benefits for sex and 6) sex against ones will, along with a wide definition of sexual harassment: sexual harassment (wide subjective definition) on levels of depression and anxiety symptoms three, six and nine months later, for women and men, respectively.

3 Methods, material, and results

3.1 THE SUSTAINABLE UNIVERISTY LIFE COHORT

3.1.1 Study design and participants

The four studies of this thesis are based on the Sustainable University Life (SUN) cohort [141], that was collected aiming to determine factors of importance for mental and musculoskeletal health among university students. The SUN cohort followed 4262 university students at five time-points over one academic year using web surveys. The cohort enrolled full-time undergraduate or graduate students (up to masters' level) from selected education programmes at eight universities in the greater Stockholm area and Örebro. The cohort constitute a convenience sample aiming to represent a range of educational disciplines. Participants were recruited continuously from August 2019 to December 2020, with follow-up data collection ongoing until December 2021.

Table 1. Baseline characteristics of the Sustainable University Life Cohort

	n= 4262
Age, mean (SD)	24.6 (6.1)
Gender, n (%)	
Woman	2644 (62)
Man	1592 (37)
Other	26 (1)
Education type, n (%)	
Medical/Health	1965 (46)
Technical	1770 (42)
Social science/Humanities	344 (8)
Economic	119 (3)
Other	64 (2)
Year of studies, n (%)	
1st	1732 (41)
2nd	936 (22)
3rd	659 (16)
≥4th	935 (22)
Country of birth, n (%)	
Sweden	3337 (78)
The Nordic countries	138 (3)
In Europe (excluding the Nordic countries)	263 (6)
Outside Europe	524 (12)

In total, 18 973 students were invited and 4262 (22%) of these enrolled in the SUN cohort by responding to the baseline survey. Of those

enrolled, 3525 (83%) responded to the three-month follow-up, 3143 (74%) to the six-month follow-up, 2863 (67%) to the nine-month follow-up and 2702 (63%) to the twelve-month follow-up. The sample included mostly first-year (41%) and women students (62%) from medical or health education programmes (46%), with a mean (SD) age of 24.6 (6.1) years (Table 1).

3.1.2 Measures

The SUN cohort survey collected extensive information on demographic data and measures on a range of potential risk factors and health outcomes at five time-points (Table 2) [141].

Table 2. Overview of the measures collected in the Sustainable University Life cohort

	Baseline	3-month follow-up	6-month follow-up	9-month follow-up	12-month follow-up
Demographics	x				
DASS-21	x	x	x	x	x
NMQ	x	x	x	x	x
SED-GIH	x	x	x	x	x
ASSIST	x	x	x	x	x
BSQ-8C	x				
FMPS	x				
PGSI	x				
PSQI	x	x	x	x	x
CET	x				
COASI	x	x	x	x	x
SEQ	x	x	x	x	x
PPS		x	x	x	x
UCLA-lon	x	x	x	x	x

ASSIST: The Alcohol, Smoking and Substance Involvement Screening Test; BSQ-8: Brief form of the Body Shape Questionnaire; CET: Compulsive Exercise Test; COASI: Cyberbullying and Online Aggression Survey Instrument - victimization subscale; DASS-21: Depression, Anxiety and Stress Scale; FMPS: Frost Multidimensional Perfectionism Scale; NMQ: Nordic Musculoskeletal Questionnaire; PGSI: Problem Gambling Severity Index; PPS: Pure Procrastination Scale; PSQI: Pittsburg Sleep Quality Inventory; SED-GIH: GIH stationary single-item question; SEQ: Sexual Experiences Questionnaire; UCLA-lon: University of California, Los Angeles three item loneliness scale.

The main outcomes for this thesis were **symptoms of depression and anxiety,** measured using the short-form Depression, Anxiety and Stress Scale (DASS-21) subscales Depression and Anxiety, respectively. The DASS-21 consists of 21 items rated on a four-point scale from 0 ('Did not apply to me at all') to 3 ('Applied to me very much, or most of the time'). The items are divided into three subscales (Depression, Anxiety

and Stress) which comprises seven items each. Summing these items gives subscale scores ranging 0-21. DASS-21 has shown acceptable psychometric properties in Swedish university samples [142]. In the current sample Cronbach's α at baseline was 0.91 for the depression subscale and 0.79 for the anxiety subscale. Study I also used the Stress subscale from DASS-21 as an outcome and Study III used several other health outcomes as well, which are described in the respective articles [143, 144].

The four main exposures were: 1) the **COVID-19 pandemic** operationalized as any date after March 13, 2020 (Study I) and divided into two periods (March 13 to June 15 2020; June 16 to September 10, 2020), 2) **sleep quality** measured at baseline using the Pittsburgh Sleep Quality Index [145] (Study II), 3) **procrastination** measured at the three-month follow-up using five items from the Swedish version of the Pure Procrastination Scale [146] (Study III) and 4) **sexual harassment and sexual violence** measured at the three-month follow-up using six items from the Sexual Experiences Questionnaire [110] that were modified for the Swedish context (Study IV). Details about these measures and other variables used in the analyses are given in the respective studies.

3.2 STUDY I

3.2.1 Methods

Study I is a cohort study that used data from participants who entered the SUN cohort before the outbreak of the COVID-19 pandemic in Sweden, March 13, 2020 [147] to compare levels of depression, anxiety, and stress symptoms in the same individuals before and during the pandemic. The follow-up surveys of these participants were categorized into one pre-pandemic time-period (August 19, 2019- March 13, 2020) and two pandemic time-periods: March 14 to June 15, representing the first wave of the pandemic and June 16 to September 10, representing a time of relatively low spread (and also the summer holidays for most students). Responses provided outside of these dates were not included in the analyses, and follow-up rates therefore differs from the overall cohort. Mean levels of depression, anxiety and stress symptoms before the pandemic was then compared to mean levels during the two pandemic periods. The main analyses were conducted using all available data. We also conducted compete case analyses (including only participants with data in all three time-periods) to investigate if baseline

differences between responders and non-responders could have affected the results.

3.2.2 Main results

Of the 1836 participants who entered the cohort before the outbreak of the COVID-19 pandemic, 74% (n=1364) provided a follow-up measurement during the first wave of the pandemic and 60% (n=1095) during the following summer months. The demographics of this sample differed from the overall SUN cohort, since not all universities had yet been included in the SUN cohort. In this study, the majority of the sample were studying at medical or health education programmes (87%), 73% were women, 80% were born in Sweden, and the mean (SD) age in years was 26.5 (6.8) years.

Compared to before the COVID-19 pandemic, mean symptom levels during the first wave of the pandemic were 0.25 (95% CI: 0.04; 0.45) points higher for depression, -0.09 (95% CI: -0.24; 0.07) points lower for anxiety and -0.30 (95% CI: -0.52; -0.09) points lower for stress. Depression, anxiety, and stress symptoms were all lower during the summer months following the first wave, compared to before the pandemic (Figure 5). Further, students with pre-pandemic loneliness, sleep problems and mental health problems did not show worse trajectories of mental health symptoms during the pandemic [143].

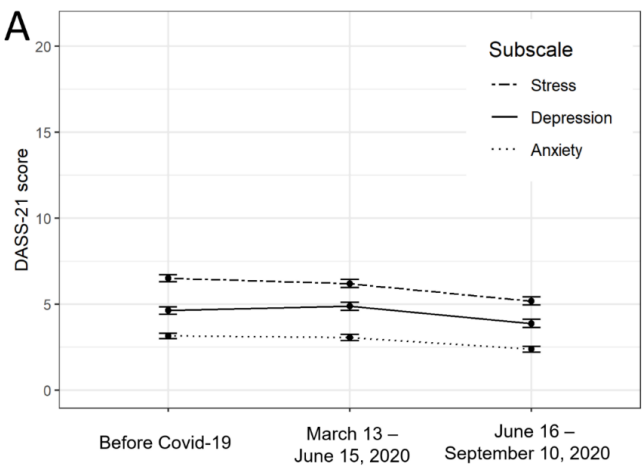

Figure 5. Mean levels of depression, anxiety, and stress symptoms at three time periods, before and during the COVID-19 pandemic. This figure is adapted from '[Depression, anxiety and stress among Swedish university students before and during six months of the COVID-19 pandemic: A cohort study]' by Fred Johansson et al., used under [CC BY 4.0].

3.3 STUDY II

3.3.1 Methods

Study II is a cross-sectional study using baseline survey data of the SUN cohort and aiming to 1) determine if sleep quality interacted with the potential risk factors loneliness, risky alcohol use, perfectionistic concerns and/or physical inactivity in relation to depression symptoms, and 2) explore the functional form of these associations and interactions. To this end, four regression models of increasing complexity were fitted for each of the potential risk factors (linear and non-linear with and without interaction terms between sleep quality and the potential risk factors). These models were compared on out-of-sample explained variance in depression symptoms (r^2) using repeated 10-fold cross validation. The models explaining the most variance were selected as the best fitting models, one for each potential risk factor. Cross validation helped us guard against overfitting, and also produced a measure of the simulated replicability to support the robustness of the findings.

3.3.2 Main results

This study included all 4262 participants of the SUN cohort responding to the baseline survey, whose characteristics are presented in Table 1. We found that for loneliness, risky alcohol use and physical inactivity, the associations with depression symptoms were best described as non-linear functions not interacting with sleep quality (Figure 6, panels A, B and C). The association between perfectionistic concerns and depression symptoms was best described as a linear function that interacted with sleep quality, such that the association between perfectionistic concerns and depression symptoms was stronger when sleep quality was worse (Figure 6, panel D).

The mean difference in explained variance (r^2) between the final models, estimated on the full dataset, and the out-of-sample cross-validation of the models was very small (loneliness model -0.007; risk alcohol model -0.009; perfectionistic concerns model -0.006 and physical inactivity model -0.008), indicating that these models were not overfitted.

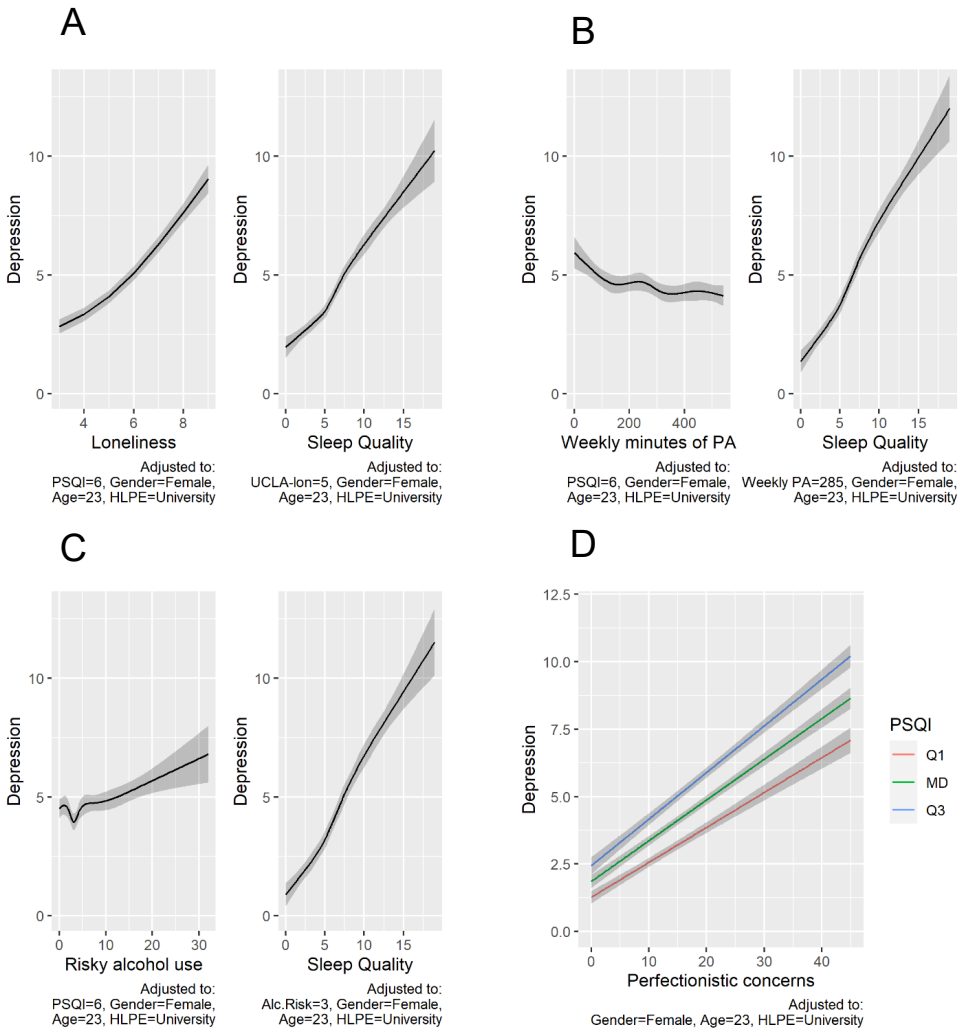

Figure 6. Estimated associations between depression symptoms and loneliness (panel A), physical activity (panel B), risky alcohol use (panel C), and perfectionistic concerns (panel D). This figure is adapted from 'Strengths of associations between depressive symptoms and loneliness, perfectionistic concerns, risky alcohol use and physical activity across levels of sleep quality in Swedish university students: A cross-sectional study' by Fred Johansson et al., used under CC BY 4.0.

3.4 STUDY III

3.4.1 Methods

Study III is a cohort study evaluating the associations between procrastination and a range of health outcomes nine months later. This study used data from all participants who responded to the three-month follow-up of the SUN cohort. There were two reasons to restrict the sample to participants responding to the three-month follow-up: 1) this was the first wave at which procrastination was measured, and 2) this enabled us to control for pre-exposure covariates (measured at baseline). Outcome measures were taken from the 12-month follow-up, i.e. nine months after the exposure measurement. The associations between procrastination and the subsequent health outcomes were evaluated using the newly developed outcome-wide framework [138]. Separate regression models were built for each outcome, linear regression to estimate standardised betas for continuous outcomes and modified Poisson regression to estimate risk ratios (RR) for binary outcomes. All models were adjusted for a common set of pre-exposure covariates, measured at baseline (three months before the exposure was measured). This covariate set included pre-exposure levels of all outcomes, and all covariates assumed to be a cause of either the exposure or an outcome [134]. E-values were calculated to assess the sensitivity of our results to unmeasured confounding [148]. We also calculated the point-biserial correlation between procrastination and missingness to evaluate the risk that selection bias could have affected our results.

3.4.2 Main results

This study included 3525 participants with a follow-up rate of 73% (n=2587) nine months later. A 1 SD increase in procrastination was associated with higher mean levels of depression, anxiety, and stress symptoms, as well as a higher risk of disabling pain in the upper extremities, poor sleep quality, physical inactivity, loneliness, and economic difficulties, even after adjustment for a large set of potential confounders (Figure 7).

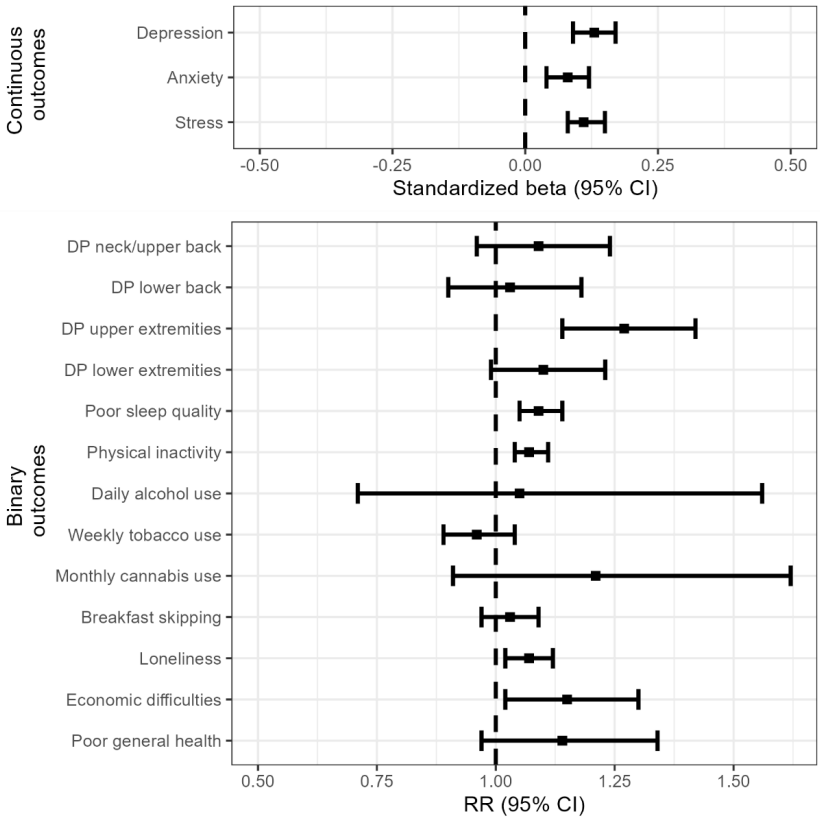

Figure 7. Forest plot of the associations between a one standard deviation increase in procrastination and the health outcomes nine months later. CI: Confidence interval; RR: Risk Ratio.

E-value analyses [148] indicated that unmeasured confounders would need to increase the risk of both procrastination and the outcomes by 35% to 86%, to move the point estimates to the null. We also found that procrastination was very weakly associated with loss to follow-up (r = 0.05), limiting the risk of selection bias [149].

3.5 STUDY IV

3.5.1 Methods

Study IV is a cohort study aiming to evaluate the impact of recent exposure to different forms of SHV on depression and anxiety symptoms three, six and nine months later, for women and men respectively. The sample in this study included participants of the SUN cohort who responded to the three-month follow-up and who identified as either women or men. The reasons for these restrictions to the sample

were again to be able to control for pre-exposure covariates, and to conduct separate analyses for women and men. Outcome measurements were taken from the 6-month, 9-month and 12-month follow-ups. Mean differences in the outcomes between exposed and unexposed were estimated for each follow-up and for each exposure. In order to estimate the impact of recent exposure and control for potential confounding, all analyses were adjusted for prior levels of all outcomes and all SHV exposures as well as a pre-exposure set of variables assumed to be potential confounders. The robustness of the results to potential selection bias was assessed by performing multiple imputation.

3.5.2 Main results

Study IV included 3503 participats, with follow-up response rates of 85% three months later, 78% six months later and 73% nine months later. The three-month exposure prevalence differed between the different forms of SHV, and all forms of SHV were more commonly experienced by women (Table 3).

Table 3. Three-month prevalence of exposure to different forms of SHV among women and men in the Sustainable University Life cohort.

	Women (n=2229)	Men (n=1274)	p
Sexual harassment (wide subjective definition), n (%)	203 (9.1)	15 (1.2)	<0.01
Offensive sexual remarks, n (%)	232 (10.4)	29 (2.3)	<0.01
Unwanted sexual attention, n (%)	548 (24.6)	69 (5.4)	<0.01
Presentation or distribution of sexist material, n (%)	53 (2.4)	22 (1.7)	0.25
Uncomfortable touching, n (%)	199 (8.9)	26 (2.0)	<0.01
Offered benefits for sex, n (%)	42 (1.9)	7 (0.5)	<0.01
Sex against ones will, n (%)	22 (1.0)	6 (0.5)	0.15

Among women, the estimated adjusted mean differences (MD) showed higher levels of depression and anxiety symptoms for women recently exposed to all forms of SHV three months following exposure (Figure 8). However, many of these estimates are uncertain, with confidence intervals not excluding null associations. Exposure to three forms of recent SHV were associated with higher subsequent levels of depression or anxiety symtpoms with confidence intervals excluding null associations: sexual harassment (wide subjective definition) was associated with higher depression and anxiety levels three months later,

unwanted sexual attention was associated with higher anxiety levels three and six months later and sex against ones will was associated with higher depression levels three and six months later (Figure 8). Trends indicate that the difference in symptom levels between exposed and unexposed decreased over time for all exposures except sex against ones will, but again, this trend is uncertain due to wide confidence intervals. These associations were similar when multiple imputation was used to account for potential selection bias.

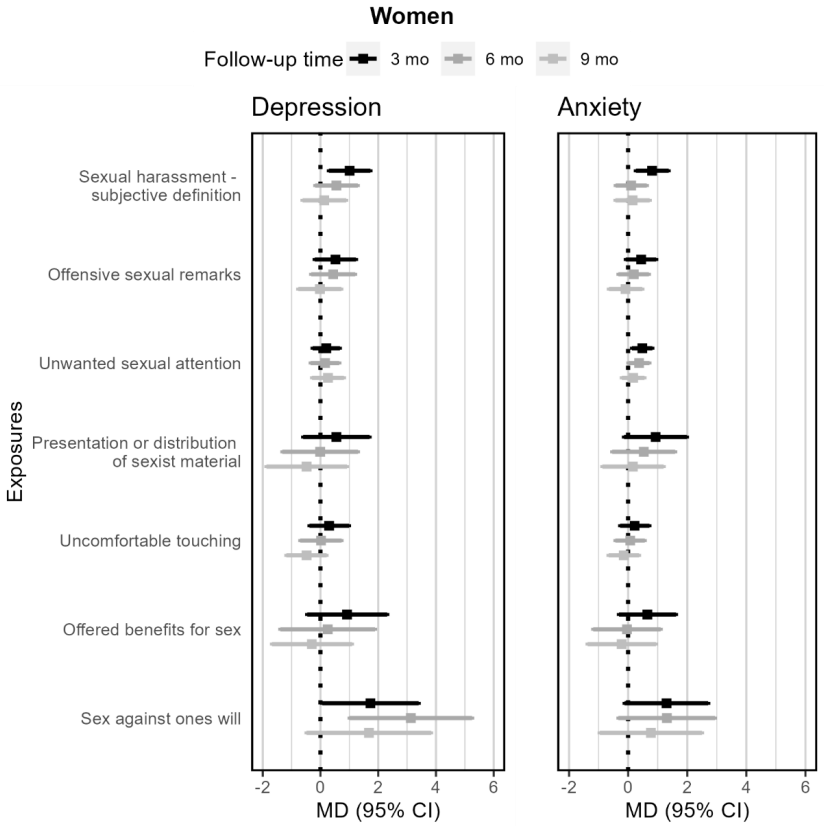

Figure 8. Forest plot of mean differences (MD) in depression and anxiety symptoms among women exposed versus unexposed to different forms of sexual harassment and sexual violence.

For men, the point estimates were scattered without a clear pattern (Figure 9). Recent exposure to several forms of SHV were associated with lower mean levels of depression and anxiety symptoms, but some were also associated with higher symptom levels. When multiple

imputation was used to account for possible selection bias, none of the confidence intervals excluded null associations.

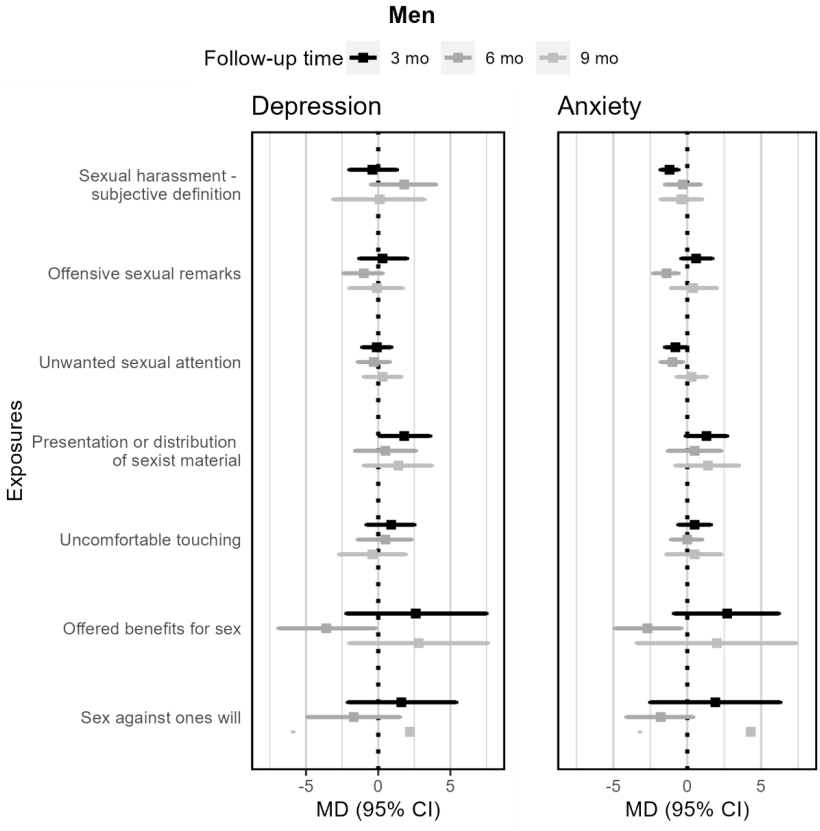

Figure 9 Forest plot of mean differences (MD) in depression and anxiety symptoms among men exposed versus unexposed to different forms of sexual harassment and sexual violence.

4 Ethical considerations

The conduct of the SUN cohort was ethically approved by the Swedish Ethical Review Authority (refence no. 2019-03276, 2020-01449, 2022-01435-02). Participants received information about the study and provided written informed consent electronically before enrolling in the study. All participants were informed about their right to resign from the study at any time. Participants received a voucher giving a one-month access to a gym chain for each survey they filled out. Ethically, large compensations may be deemed as undue influence of the potential participants. We deemed that this compensation was not so large that it would unduly influence students to participate, as did the Swedish Ethical Review Authority. One other important ethical aspect is the safeguarding of personal information about the respondents. Respondents provided sensitive personal information such as health status and ethnic origin. To avoid the risk of any unauthorized access, data was handled with great care by a restricted number of persons, according to the GDPR and Sophiahemmet University guidelines. Being asked questions about health and related risk factors also carries the risk of upsetting participants. Therefore, student health offices at the included universities were informed about the study, and respondents were recommended to seek contact with them if the survey raised questions about their health. Yet another ethical aspect that warrants attention is how participants were prompted to respond to the surveys. Participants not answering their follow-up surveys received four automatic reminders, as well as manual reminders first by text message and then by telephone call. Some participants indicated that they found these reminders annoying, which is ethically problematic. On the other hand, as researchers we also have an ethical responsibility to maintain the scientific quality of the study. If the follow-up rate had dropped too low, we would not have been able to draw conclusions from the collected data. This would have caused another ethical problem, since scientific value of the contributions of all the participants who responded to our surveys would have diminished. This ethical dilemma has been extensively discussed in the research group, and it has been agreed that the benefits of reminding the participants to respond, in terms of response rate and quality of the studies, outweighs the potential adverse effects for the participants. We took care to promptly remove participants from the study if they communicated that they did not want to receive more reminders. In all, we believe that although participation

in the study may have caused discomfort for some of the participants, the benefits of performing the data collection, in terms of increased understanding of student mental and musculoskeletal health, outweighed the potential harm.

5 Discussion

This thesis set out to investigate some aspects of the aetiology of mental health problems among university students, focusing on the COVID-19 pandemic, sleep quality, procrastination and sexual harassment and sexual violence. Of note, only one of the research aims explicitly use causal language (Study IV). There are several reasons for this inconsistency. Partly, it is due to scientific convention, partly due to methodological limitations making causal inference speculative in combination with limited space to discuss the assumptions needed for causal inference in the articles. In this discussion, however, I will focus on the interpretation of these results from a modern causal inference framework. Other aspects of these results are discussed in the respective articles accompanying this thesis.

5.1 COVID-19 AND MENTAL HEALTH

In Study I, we aimed to determine mean trajectories of depression, anxiety, and stress symptom levels among Swedish university students, before and during the first months of the COVID-19 pandemic.

To our surprise (at the time), the results of Study I showed small changes in mean symptoms of depression, anxiety, and stress before and during the first six months of the COVID-19 pandemic, even in groups we expected to be especially vulnerable such as students with pre-pandemic loneliness, poor sleep and mental health problems [143]. In a follow-up study, we found that symptom levels remained mostly stable during the second and third wave of the pandemic as well [150]. While our results contrasted cross-sectional data at the time [68], they are in line with later reviews of prospective cohort studies [71, 73]. Results vary considerably between studies, but it seems that on a group level, many populations showed a considerable mental health resilience during the COVID-19 pandemic.

To what extent can our estimates be interpreted as casual effects of the pandemic? We compared mean symptom levels before and during the pandemic in the same participants. To be able to interpret these contrasts as causal effects, we need to make several assumptions. First, we need to assume exchangeability between the pre-pandemic and the pandemic time-periods. In plain language, this means assuming that mental health levels would have remained stable over time in the absence of the pandemic. This seems uncertain, first because we know that

depression and anxiety levels vary over the year with higher levels in the winter and lower levels in the summer [151, 152]. It is therefore likely that, rather than remaining stable, depression, anxiety, and stress symptom levels would have declined during March-September 2020 had the COVID-19 pandemic not happened. Second, during the post-pandemic time-points the students had spent more time at university and were at a different phase in the academic year, which could affect mental health symptoms. It is therefore possible that the pandemic increased symptom levels compared to what had been observed had the pandemic not happened. In any case, the effects of seasonality on mental health are rather small [152]. So even if potential outcomes under the different time-periods were not completely exchangeable, it seems that any potential negative effect of the pandemic on mental health could not have been very large on a group level.

Another threat to exchangeability is selection bias. Loss to follow-up could have been affected by both the pandemic and by mental health levels. We conducted sensitivity analyses including only complete cases, and the results were very similar to the main analyses. There is still the possibility, however, that those lost to follow-up had different trajectories of mental health, in which case our estimates would be biased. Positivity is not an issue in these analyses, given the low number of covariates. Consistency is not guaranteed, however, since we measured participants at slightly different phases of the pandemic, where the effects of the pandemic may have differed. The restrictions also differed slightly between students. For instance, medical students were allowed to do some of their clinical training at hospitals, rather than having all their education online. This may lessen the generalizability of these results to other student groups in Sweden.

Overall, the spread of the COVID-19 pandemic as well as the public health response to the pandemic varied substantially between countries. There is also variation between populations regarding potential moderators of effect of the pandemic on mental health, such as social and welfare systems, the level of digitalization, and systems for financing higher education. If these results are to be generalized, differences between populations must be taken into careful consideration.

To sum up, I believe our results indicate that, on average, the COVID-19 pandemic does not seem to have had any large impact on the mental health of Swedish university students, at least not on the students in our

sample. Importantly, this applies to average levels of mental health, and there may still be subgroups that have fared unwell during the pandemic.

5.2 SLEEP QUALITY AND INTERACTIONS WITH OTHER RISK FACTORS IN RELATION TO DEPRESSION SYMPTOMS

In Study II, we set out to explore the hypothesis that sleep quality may interact with other risk factors of depression. This study was based on the idea that by decreasing the ability to regulate emotions, sleep disturbances could amplify the negative effects of other risk factors on depression. We chose to focus on four potential risk factors for depression symptoms, representing different levels of risk factors for depression: loneliness (psychosocial), perfectionistic concerns (psychological) and risky alcohol use and physical inactivity (behavioural).

We found that while all variables had clear associations with depression symptoms, it was only perfectionistic concerns that interacted with sleep quality in relation to depression symptoms. Further, the interaction between sleep quality and perfectionistic concerns accounted for only a very small proportion of the variance in depression symptoms.

Of course, these results cannot be interpreted as causal effects, which was also not the explicit aim of this article. The use of cross-sectional data generally precludes any causal conclusions, mainly because with this design we cannot disentangle the temporal relationship between the variables. Also, we controlled only for a small number of potential confounders, so exchangeability seems unlikely to hold. Lastly, sleep quality is a very broad construct, and it is likely that there are many different versions of 'poor sleep quality', given the structure of the PSQI. Participants could have been defined as poor sleepers due to frequent awakenings, problems falling asleep, waking too early, tiredness and difficulty staying awake etc. [145]. Since these types of sleep problems may have different impacts on depression symptoms, the consistency condition is unlikely to hold. Still, our analyses may provide some insights into potential structures of the causal network of mental health problems. Causal effects between variables are unlikely, although not impossible, in the absence of associations. Therefore, the absence of statistical interactions between loneliness, risky alcohol use and physical inactivity and sleep quality in Study II makes causal interactions between these factors and sleep quality unlikely, at least when considering sleep quality as such a broad construct as in our analyses. One of the strengths

is that we allowed for different functional forms of the relationship between sleep quality, the potential risk factors, and depression symptoms. This flexible modelling approach reduces the risk of model misspecification, which is related to the positivity assumption as described above.

So overall, our results did not support the hypothesis that sleep quality could act as an effect modifier for risk factors of depression in general. However, the four variables we included as potential risk factors of depression constitutes only a very small portion of the potential risk factors of depression. It is still possible that other risk factors, not included in our study, may interact with sleep quality in relation to depression, as indicated by some previous research [86-91]. If this is the case, small interactive effects across a number of risk factors could add up to large effects [153]. Future research could explore this possibility by including a larger number of potential risk factors, preferably with longitudinal data and with more specific sleep measures, to overcome some of the limitations of this study.

5.3 PROCRASTIANTION, MENTAL HEALTH AND OTHER HEALTH OUTCOMES

Inspired by the procrastination health model [100, 101], we set out to determine the associations between procrastination on subsequent mental health and other health-related outcomes. We found that procrastination was associated subsequent depression and anxiety symptoms measured nine months later, but also to a range of other health outcomes.

Our results are largely in the same direction as much of the prior cross-sectional research on associations between procrastination and depression and anxiety symptoms [64, 97], and provides new evidence on potential effects of procrastination on other health-related outcomes that have not previously been studied. In my view, our results provide stronger evidence for causal effects of procrastination on various health outcomes since, unlike much prior research, we controlled for a wide range of potential confounders.

Regarding effect sizes, our crude estimates for depression and anxiety were of similar strength as estimates reported in prior research [64, 97], but the adjusted estimates were considerably weaker, about a third of the crude estimates(see eTable 1 in the Supplemental material for [144]). A

reduction of associational strength is expected when adjusting for potential confounders, after all the goal is to reduce non-causal association. Interestingly, though, is that adjustment for prior outcome levels accounted for almost all of this reduction in associational strength. There may be at least three reasons for this. First, prior depression and anxiety (Y_{-1}) may have effects on both procrastination (A_0) and later outcome levels (Y_1), causing confounding ($A_0 \leftarrow Y_{-1} \rightarrow Y_1$, in Figure 10), which was adjusted for in our analyses. Second, adjustment for prior outcome levels (Y_{-1}) may have removed confounding by other factors (C) that was mediated by prior outcome levels ($A_0 \leftarrow C \rightarrow Y_{-1} \rightarrow Y_1$, in Figure 10) [154]. Third, by adjusting for prior outcome levels (Y_{-1}), we remove the effect of prior procrastination mediated by prior outcome levels ($A_0 \leftarrow A_{-1} \rightarrow Y_{-1} \rightarrow Y_1$, in Figure 10).

This third reason is of particular importance for the interpretation of the estimate. By removing part of the effects of prior procrastination, the estimates can no longer be thought of as a proxy for any accumulated effect of procrastination on depression and anxiety symptoms. Rather, our estimates may reflect the effect of current procrastination on the outcomes. The focus on current procrastination levels have some desirable properties: it is conceptually closer to the effect of an intervention to change procrastination here and now (which would not change prior procrastination or outcomes), and it is less subject to some biases such as reverse causation [136], unmeasured confounding from stable factors (which are likely to mediate their effect through prior outcome levels) and selection bias (as detailed in the literature review).

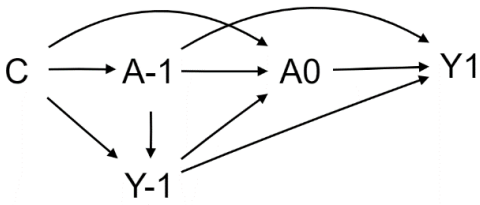

Figure 10. Directed acyclic graph of the effect of current procrastination, A0, on subsequent outcomes, Y1. C represents a set of time-invariant confounders, A-1 prior procrastination levels and Y-1 prior outcome levels.

With this estimand in focus, a limitation of our main analyses is that we did not control for prior procrastination levels which may also confound the effect of current exposure ($A_0 \leftarrow A_{-1} \rightarrow Y_1$, in Figure 10). This was

however done in a sensitivity analysis (see eTable 3 in [144]), which showed results similar to those of the main analyses. We also showed that unmeasured confounders would need to be at least moderately associated with both depression and anxiety, as well as with procrastination, to move the point estimates to the null [144]. Finally, we showed that procrastination was very weakly associated with loss to follow-up ($r = 0.05$), indicating small risk of selection bias in terms of internal validity [149, 155]. We still have the possibility of selection bias in relation to the external validity, which could be the results of different distributions of effect modifiers between our sample and the population to which we want to generalize [149, 155]. For instance, it is possible that procrastination has less detrimental effects on mental health among university students than among their peers not at university. After all, student procrastinators have made it to university despite their tendency to procrastinate. So, it may be that our sample has selected persons less severely affected by their procrastination. This does not affect the internal validity of our results, but it may affect or ability to generalize to non-student populations.

So, we have estimates intended to capture average effect of intervening to change current procrastination levels (by 1 SD) on later levels of depression and anxiety. There are of course several limitations, such as unmeasured and residual confounding and measurement error that could bias these estimates. To fulfil the positivity condition, we used parametric models to interpolate covariate positions, and therefore relied on the assumption of no model misspecification. Given the large number of covariates, it is unlikely that out model is fully without misspecification, which may have led to residual confounding. Also, in terms of consistency, there may be different 'versions' of procrastination, that may differ in their effect on health outcomes. For instance, the procrastination could relate predominantly to different aspects of life for different respondents (e.g., exercise, paying bills, studying for exams). This will likely affect our external validity, as is discussed in the Appendix.

If we assume that our estimates are a roughly accurate estimation of the average causal effect, the size of our estimates indicates that such an intervention may be somewhat beneficial across a range of health outcomes, but that the health benefits for any specific outcome would be rather small. For instance, the adjusted association between

procrastination and later depression indicated that a 1 SD increase of procrastination was associated to a 0.13 SD increase in depression, and many associations were weaker than this. It should be noted, however, that these are estimates of the average causal effect (i.e., at the population level), and there are likely some individuals who would benefit more of reduced procrastination than others. Identifying individuals who may benefit more from procrastination interventions is a potential focus for future research.

5.4 SEXUAL HARASSMENT, SEXUAL VIOLENCE AND MENTAL HEALTH

In Study IV, the aim was to determine the effect of recent exposure to different forms of SHV on symptoms of depression and anxiety three, six and nine months later.

Exposure to three forms of SHV showed clear associations to higher subsequent depression or anxiety levels among women. Estimates for the other forms of SHV were uncertain due to wide confidence intervals. The general trend, however, was that depression and anxiety levels were higher among those exposed to SHV at the three-month follow-up, regardless of the form of SHV, and that the difference between exposed and unexposed declined over time for all exposures except sex against ones will. There were also trends showing differences in associational strength between different forms of SHV, again with wide and overlapping confidence intervals precluding firm conclusions.

For the men, the estimates showed no clear pattern, were highly variable, and were not robust to sensitivity analyses using multiple imputation to account for selection bias of the internal validity. Given their high levels of uncertainty, I refrain from interpreting and discussing these results.

Our estimates indicate that the effect of recent exposure to any specific forms of SHV on depression and anxiety symptoms three, six and nine months later among women is rather small, with the exception of exposure to sex against ones will. It should be noted that we have tried to filter out the effect of only recent exposure, by controlling for both prior outcome and exposure levels. We have also attempted to estimate the effects of specific forms of SHV rather than SHV overall, for reasons that are discussed as length in the Appendix.

In reality, exposure to SHV is often repeated over time [156], and exposure to different forms of SHV tend to co-occur [110], so that

exposure to one form of SHV is associated with a higher probability of exposure to other forms as well. For any persons exposed to SHV, effects of repeated exposures to different forms of SHV may accumulate over time, potentially leading to a significantly higher burden on mental health than is reflected by our estimates. Our estimates would not capture any such accumulated negative impact of repeated exposure to different kinds of SHV on mental health.

However, by narrowing our focus to the effects of recent exposure to specific forms of SHV at given time-points, I believe that we have increased the plausibility of exchangeability and consistency, that is, for our estimates to be viewed as causal. I also believe that a narrower focus may provide more nuanced knowledge that could be useful to guide interventions. Our estimates indicate, for instance, that exposure to most forms of SHV have short-term negative effects on mental health for women that decline over time. In contrast, the negative impact of exposure to sex against ones will does not seem to decline, at least not within nine months. Although all forms of SHV are unwanted, it would seem that interventions to reduce the most severe forms of SHV are perhaps of highest importance in relation to student mental health.

There are limitations to these results. As always with observational data, we have the risk of residual and unmeasured confounding, for which we performed no sensitivity analyses in Study IV. Our multiple imputation analyses indicated that selection bias does not seem to have affected the internal validity of the results among women, although it may have among men. This conclusion rests, however, on the assumption that the imputation model was well-specified and included all relevant predictors for loss to follow-up, which is not possible to verify. As with the other studies, there is also the possibility that selection bias may have affected the external validity. Further, many of the SHV exposures were rare in the male sample, which may have caused practical positivity violations that could explain the high variability of the estimates. We also have the possibility of measurement error, especially for the exposure measurement of SHV, since these measures were rather crude. This may have biased our estimates, most likely resulting in an attenuation of the estimated effects. A strength of this study is that by using relatively specific exposures, we reduced consistency issues, as expanded on in the Appendix.

To sum up, there seems to be negative effects of SHV on mean levels depression and anxiety symptoms among women, and that these effects may differ in strength and in duration between different forms of SHV.

5.5 GENERAL DISCUSSION

Self-reported mental health problems among university students have increased rapidly during the last years [52-56]. Data from the US show that the proportion of students scoring above the cut-off for depression and anxiety on self-rated measures has more than doubled in the last decade [52]. In relation to these large increases in self-rated mental health problems among university students [52-56], the potential mental health effects found in these four studies were small. This is not surprising, since small population level effects are to be expected for individual causes of a multicausal construct like mental health, that is assumed to be caused by a multitude of other factors as well. Further, as discussed above, Study III and Study IV aimed to provide estimates of the effects or recent exposure, which may accumulate if exposures are repeated over time [153].

Although far from explaining the rapid increases in student mental health problems, I believe that these results help shed light on at least some aspects of the aetiology of student mental health problems.

6 Conclusions

Mental health was largely stable during the COVID-19 pandemic. Interaction between sleep quality and perfectionistic concerns accounted for a small proportion of the variance in depression symptoms, and loneliness, risky alcohol use and physical inactivity were not found to interact with sleep quality in their associations to depression symptoms. Procrastination and recent exposure to SHV were associated with later levels of depression and anxiety symptoms, which could reflect causal effects, but the associations were mostly rather weak.

Interpreting any of these results as causal effects rests on several assumptions, some of them unverifiable. Still, I believe that this thesis has strengthened the evidence that procrastination and SHV are potential causes of mental health problems among university students. An equally important finding is that mental health among the students seemed rather stable during the COVID-19 pandemic, indicating that the

pandemic may not have caused increased mental health problems on the group level.

7 Clinical implications

In situations such as the COVID-19 pandemic, public health officials must consider not only what measures might be effective to reduce the spread of the virus but also potential side-effects of these measures. During the COVID-19 pandemic, little information was available on the potential mental health effects of measures like physical distancing and switching from campus- to online-based education. The finding that mental health among Swedish university students remained relatively stable during the public health measures taken during the COVID-19 pandemic may be important when deciding on a public health strategy in similar situations in the future.

Our second study showed that the associations between loneliness, perfectionistic concerns, risky alcohol use or physical inactivity and depression symptoms did not differ in any substantial way across levels of sleep quality. From this, it seems that interventions on sleep quality are unlikely to alter the impact of these risk factors on depression. Still, sleep disturbance is an important risk factor for mental health problems in and of itself, and needs further public health and clinical consideration in relation to student mental health.

The third study showed that procrastination may have small effects on a range of health outcomes. If the associations are interpreted causally, this would imply that broad interventions to reduce procrastination in student population in general are unlikely to have any large health effects on specific outcomes but may have smaller effects over a range of health outcomes. Whether such interventions are worthwhile is of course a matter of cost versus benefit. Some of the interventions that have been suggested to potentially reduce procrastination among students are relatively cheap and easy to implement. These include, for instance, offering courses in study skills and self-regulation or implementing shorter deadlines [96]. Given the low cost, these interventions could be worthwhile even if they produce only small average effects on health outcomes. I therefore believe that interventions supporting students not to procrastinate should be explored further as a mean to reduce both mental and physical health problems among students.

The fourth study showed that several forms of SHV was associated with subsequent depression and anxiety symptoms among women students, and that exposure to sex against ones will was associated with prolonged

elevation of symptoms. From my perspective, this indicates that interventions to reduce severe forms of SHV may be prioritized if the focus is to improve mental health. Still, all forms of SHV are undesirable and, ideally, interventions, at for instance organizational culture, could reduce all forms of SHV. This is an important topic for future research.

8 Future research

Although we found small changes in mean mental health symptom levels during the COVID-19 pandemic, some of the potential mental health effects of the pandemic may still lay in the future. The COVID-19 pandemic accelerated the transition into digital learning environments in higher education, arguably changing the social landscape of university students. Further research is needed on the long-term effects of these changes, and how to intervene to create a supportive and healthy learning environment for students, both physically and digitally. For instance, social connectedness among young people has decreased during the last two decades and this development was exacerbated during the pandemic [157]. With loneliness and social isolation as two of the top risk factors for mental and physical ill-health, there is an urgent need for research on how to support social interaction and implement 'pro-connection technologies', both for students as well as the population in general [158].

Overall research on contextual interventions, focusing on changing student environment to support student mental health, are scarce. Most intervention research have evaluated interventions targeting individual behaviours, emotions, and cognitions, like CBT for depression and anxiety among students [159]. Individual interventions are highly needed and should be developed further. However, I believe that the high and increasing prevalence of mental health problems among students [52] points to the need to consider contextual interventions to the student environment to support students' mental health. Large-scale studies on contextual interventions are admittedly hard to perform, which may explain the dearth of studies focusing on contextual changes.

There are exceptions, however. For instance, delaying school start by 30 minutes, in order to better match natural sleep rhythms, has shown positive associations to students mood, alertness and health [160]. Social support groups have shown promising results for reducing loneliness among students [161]. For procrastination, several contextual changes to the student environment have been suggested, such as shorter deadlines and decreasing distractions in the study environment [96], but large-scale studies evaluating the effect of such interventions are lacking. Similarly, preventive efforts related to sexual harassment and violence have been suggested. These include, for instance, creating organizational structures

promoting egalitarianism and an active leadership demonstrating that SHV will not be tolerated [109]. However, a recent review concludes that prevention and intervention efforts have largely focused on the victims, and that large-scale organizational interventions targeting SHV remain to be evaluated in research [109]. I believe that a promising avenue for future research would be to focus more on such contextual interventions.

As for the aetiology, although mental health problems are commonly viewed as multicausal [162], the predominant approach to studying causal effects is to investigate bivariate associations (e.g. the effect of insomnia on depression), while adjusting for selected confounders [24]. With a strong design, this approach may give reasonably well-grounded claims about causality [123]. This approach has identified a wide range of risk factors [21, 25], but information on individual risk factors is not easily integrated to a more general understanding of joint effects of these risk factors on the development of mental health problems. Some promising attempts to integrate causal knowledge into a larger system of interacting causes have been made, for instance based on the relatively newly developed Network theory of mental disorders [22], but there are still considerable challenges with regards to causal interpretation of these networks. Study II of this thesis was an attempt to look at potential interactions between sleep quality and other risk factors in relation to depression symptoms. However, further theoretical, and empirical work in this area is highly needed for a better understanding of joint effects of multiple risk factors and psychological processes in the development of mental health problems, both among students and the population in general.

It is possible that these systems of interacting risk factors are too complex to model in any reliable way, and that we must resort to studying bivariate associations between exposures and mental health (as has been the case for three of the studies in this thesis). In such cases, I believe that much could be gained by trying to speak more openly about causality. By avoiding the term causal, the true aims of much observational research remains obscure [129]. By explicitly stating the research goal, future research would also need to be more specific about the causal questions in focus. As detailed in the Literature review and in the Appendix, this entails specifying an unambiguous exposure, providing a clear and transparent reasoning about the assumed causal

structure, and a clear specification about the causal contrast of interest. This would hopefully lead to a more open discussion of the potential strengths and weaknesses of different approaches. One suggested approach to achieve more rigorous design for causal inference with observational data is to first specify the randomized controlled trial that you would perform, if it had been possible, and then use observational data to try to emulate this 'target trial' [123]. This approach can help avoid many of the pitfalls in causal inference with observational data, such as ill-defined exposures and inappropriate start of follow-up time [163]. However, the approach has thus far mainly been applied in medical settings, so further developments may be needed to apply the framework with psychosocial exposures.

9 The human perspective in illness, care and health

"The area is based on an individual perspective on health care issues. Here, human experiences of care are studied, as well as experiences of and reactions to illness, ill health and the path to regained health. The field of knowledge also includes human experiences when healing is not possible. Furthermore, interventions aiming to prevent and treat diseases and contribute to the development of patient-safe and person-centered care, are included."

Sophiahemmet University conducts research and provides doctoral education in care science with the research area specified as "the human perspective in illness, care and health", as defined above. This doctoral thesis ties to the last sentence of the definition, as the end goal is to advance prevention and treatment strategies for mental health problems. As has been touched upon already, effective prevention and treatment require knowledge about the nature of the targeted condition as well as its causes. This thesis has a group level, rather than an individual, perspective on mental health. Still, my hope is that this doctoral project can in some ways contribute with knowledge that can guide "interventions aiming to prevent and treat diseases" both for groups and individuals.

10 Agenda 2030

The 2030 Agenda for Sustainable Development consist of 17 goals adopted by all member states of the United Nations in 2015. Of these goals, this thesis relates most closely to the third goal: "Good health and wellbeing", but also to the fourth goal: "Quality education".

The third sustainability goal defines several specific targets. One of these is target 3.4 aiming to "By 2030, reduce by one third premature mortality from non-communicable diseases through prevention and treatment and promote mental health and well-being". One of the indicators of goal attainment for this target is suicide mortality rates. Suicide is the second leading cause of death in persons between 20-40 years, and most often related to mental disorders [164]. Advancing the knowledge on the causes behind mental health problems among university students is central to design effective prevention and treatment programs. This is key to prevent suicides and fulfilling the third of the global sustainability goals.

Mental health problems among students are also related to adverse educational outcomes [51]. Addressing mental health problems among university students is of importance to meet the fourth global sustainability goal of ensuring "inclusive and equitable quality education". If this is not done effectively, students with mental health problems may be at an educational disadvantage. This may be particularly important in terms of gender equality, as women generally show higher levels of mental health problems than men. Effective prevention and treatment may not only lead to higher well-being and fewer suicides but could also provide more equal opportunities in higher education.

To summarise, advancing the knowledge of causes underlying mental health problems among university students is highly relevant both to promote mental health and provide more equal opportunities in higher education.

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13 Appendix

13.1 SEXUAL HARASSMENT, SEXUAL VIOLENCE AND MENTAL HEALTH OUTCOMES: CAUSAL INFERENCE WITH AN AMBIGUOUS EXPOSURE

Sexual Harassment, Sexual Violence, and Mental Health Outcomes: Causal Inference with Ambiguous Exposures

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Abstract

Social exposures and their impact on mental health has proven hard to capture, partly owing to the complex and multifaceted nature of the social reality. Sexual harassment and sexual violence (SHV) are no exceptions. SHV can be conceptualized as a continuum of negative sexual experiences whose severity varies depending on multiple determinants (type of SHV, frequency, power relations, etc.). Further, exposure to SHV may be conceptualized either as discrete exposures (i.e., experiences of specific SHV events) or as exposure to a sexually hostile environment, often represented by a latent variable reflected by patters of SHV events. With any of these conceptualizations, SHV constitutes a broad construct containing many kinds of negative experiences. The ambiguity of SHV pose a challenge when attempting to determine its mental health consequences, as different forms of SHV may vary in terms of their mental health impact. In this article we use the potential outcomes framework to discuss the conceptualization of SHV in relation to mental health outcomes, focusing on the consistency condition. We discuss limitations and possible interpretations of estimates of the causal effect of SHV on mental health, both when SHV are conceptualized as discrete exposures and when conceptualized as one or several latent variables. We present the recently developed multiple versions of treatment theory and show how it can provide a formal interpretation of causal estimates under ambiguous exposures. Lastly, we provide suggestions on how the increase the clarity and interpretability of estimates of the effects of SHV on mental health, by increasing the precision of the causal questions and the use of more specific definitions of SHV exposures.

Introduction

An important question within research on sexual harassment and sexual violence (SHV) is: what are the effects of SHV on mental health? To be able to try to answer to this question, we need to be clear about what we mean with SHV [1]. A clarity that has been elusive to sexual harassment research. First, because there is no broadly accepted definition of sexual harassment [2]. Second, even with a clear definition, sexual harassment experiences come in many forms and their impact on mental health is likely to vary depending on specific circumstances of the SHV exposure.

Most definitions of sexual harassment focus on acts of sexual nature that are "unwanted or unwelcome, and which has the purpose or effect of being intimidating, hostile, degrading, humiliating or offensive" [3]. This broad definition contains a range of different behaviours and behaviour patterns, and it is commonly accepted that sexual harassment and sexual violence constitute a continuum of negative sexual exposures of varying severity [4-6]. Severity is hard to grade, however, as it may be determined by both objective criteria such as the moral appal of the act and subjective criteria such as the negative consequences for the victim [6]. Table 1 presents a list of some potential determinants of SHV severity [7-9], but the list is by no means exhaustive. This complex nature of SHV makes the concept difficult to measure and define SHV in an unambiguous way, such that SHV means the same thing for all persons. While the need to distinguish between different forms of SHV has been highlighted in relation to determining prevalence [10-12], less focus has been directed towards distinguishing between forms of SHV in terms of mental health consequences.

Table 1. Potential determinants of SHV severity

	Examples
Type of SHV	Being coerced into sex may be seen as more severe than offensive sexual remarks.
Frequency	Repeated forms of SHV may have more severe impacts on the victim than one-time events.
Context	Experiencing SHV at work may have different effects than experiencing SHV at home, in an educational setting or in the street.
Power relations	The power relations between the victim and the perpetrator could determine the severity. Being exposed to SHV by one's boss may have more detrimental consequences than exposure to SHV from a peer.

Causal inference with ambiguous exposures

Several recent papers have focused on the limitations of causal inference with ambiguous or ill-defined exposures in epidemiology in general [13-16], and in social epidemiology in particular [1]. An ambiguous exposure is one where each level of the exposure may contain several different exposure-versions, with potentially differential associations to the outcome. This situation is likely to occur in SHV research, where "exposure to SHV" could represent many different kinds of experiences, like sexist comments by a person passing on the stress, repeated groping in the workplace, sexual assault etc., and where the mental health impact, or severity, of different exposure-versions could be expected to differ.

To be able to estimate a causal effect we generally need a well-defined causal contrast, that is a contrast between potential outcomes under two clearly defined exposure levels [13]. For the effect of SHV on depression, the causal contrast for any single person *i* can be defined as the depression levels (Y) we would observe had the person been exposed to SHV (Y_i^{SHV}) versus the depression level that would be observed had this same person not been exposed to SHV ($Y_i^{No\ SHV}$). The difference (or ratio) of these two potentials outcomes is referred to as the individual treatment (exposure) effect (ITE),

$$ITE_i = Y_i^{SHV} - Y_i^{No\ SHV}.$$

For a causal contrast to be well-defined, the exposure must be sufficiently well-specified so that each person has only one potential outcome for each value of the exposure, a condition known as *consistency* in the causal inference literature [17] [1]. This requires that there are no different exposure-versions within the same exposure level so that the potential outcome could differ depending on exposure-version [1]. If consistency holds, the average treatment effect (ATE) is the average difference of potential outcomes under exposure vs under non-exposure (i.e., the average of the ITEs) [18]. With well-defined causal contrasts, the ATE can be interpreted as the effect we would observe if we could hypothetically intervene to shift the exposure status from exposed to unexposed for the entire population, or a random subset of the population,

[1] More formally consistency requires that, for each individual, the observed outcome Y equals the potential outcome for that particular exposure level so that when exposure A = a, $Y_i^{obs} = Y_i^a$, which provides a formal link between observed and potential outcomes.

$$ATE = \frac{1}{N}\sum_{i=1}^{N}(Y_i^{SHV} - Y_i^{No\ SHV})$$

In reality, we cannot observe an individual simultaneously under both exposure and non-exposure. In practice the population ATE is estimated by comparing the observed outcomes of the exposed to those of the unexposed under the assumptions of exchangeability, positivity, and consistency [15].

In situations where exposure-levels, such as "exposed to SHV" and "unexposed to SHV", contains different exposure-versions for different persons, the exposure is said to ambiguous, and the consistency condition is likely to be violated. In such situations it is not straightforward how to interpret causal estimates such as the ATE [19], since it represents a mix of different causal contrasts.

For SHV it is difficult, if not impossible, to operationalize measurement in such a way that exposure (and non-exposure) means the same thing for all respondents, at least in observational settings. One relatively common approach to measuring SHV is to simply ask respondents whether they have been exposed to any form of SHV during a given time-period [11]. With such a crude measure, the exposure-levels (both exposed and unexposed) will inevitably contain a range of negative sexual experiences of different severity. For one person exposure to SHV may represent sexual coercion by her boss, while for another exposure may represent repeated experiences of sexual jokes. In such situations, the ATE represents the mean difference of the potential outcomes between two exposure-levels containing several different exposure-versions. This makes ATE vaguely defined and hard to interpret, as we are not clear about which potential outcomes we are trying to contrast, and what kind of group level intervention on SHV that would correspond to the estimated effect [19]. This is troublesome, as there is little evidence of the effectiveness of interventions trying to reduce SHV [5], and it has been argued that knowledge on specific forms of SHV victimization could help guide the development interventions for various forms of victimization [12].

One solution to this unclarity would be to assume that all exposure-variants (i.e., all different forms of SHV experiences) have the same effects on the outcome, an assumption sometimes referred to as the "treatment-variance irrelevance assumption" [14]. If this assumption holds, the ATE will be the same regardless of which exposure-versions we are imagining that we may intervene upon. For the effect of SHV on depression, this assumption entails that an intervention to reduce sexual jokes would have the same average effect on depression as an intervention that reduces sexual coercion. We believe this assumption to be unrealistic for the effects of SHV on mental health. One reason

why SHV is often conceptualized a continuum of experiences of *varying severity* is that their effects on the victim are assumed to vary.

Another solution to this unclarity is to improve the precision of our causal contrast by increasing the specificity of our causal questions [13]. Our initial question "what are the effects of SHV on mental health?" seems too vague given that we think that different forms of SHV may have different effects on different kinds of mental health. In a coming article by Johansson et. al. [20], the authors attempted to specify the causal questions by focusing on the mental health effects of specific forms of SHV. A more specific causal question, and one implicitly asked in this article was: "what is the average effect of having experienced sex against ones will at least once during the prior three months on levels of depression symptoms three months later among women?".

Although this question provides somewhat more clarity regarding the causal contrast, there is still a lot of ambiguity left. The question does not specify the power relations between the victim and the perpetrator or any specifics about the experience, like if physical violence was involved or the frequency of the experience. Judging whether a causal question is specific enough to allow for meaningful causal contrasts (i.e., for the treatment-variance irrelevance assumption to hold) is a matter of expertise judgement [13], but it seems safe to say that the above question still contains plenty of ambiguity.

With social phenomena, like SHV, the consistency condition will never strictly hold, there will always be variation in social exposures. However, by increasing the specificity of our causal questions and the definition of the exposure, we can increase the clarity of the meaning of our causal estimates [1].

Latent variable approach

In the discussion above, SHV was seen as discrete negative sexual experiences. An alternative conceptualization, and one often used in SHV research, is to view SHV as behaviour patterns reflecting a sexually hostile environment emanating from higher level social constructs such as sexist norms and culture [6]. These norms and cultures are conceptualized as one or several latent variables that are reflected by different forms of SHV experiences. From this perspective, the focus is not on specific SHV exposures per se, but rather on exposure to a sexually hostile environment, measured as one or more latent variables.

A latent variable is one that cannot not usually be observed, except through its reflections in thoughts or behaviour [21]. Consequently, a latent variable cannot be measured directly, but must be inferred through observable indicators, like SHV experiences. Often this is done by constructing a measurement scale, where item responses are assumed to be caused by the construct and thus can be viewed as reflections of the underlying latent variable. The latent variable framework has been used for the measurement of SHV, most notably in the widely used Sexual Experiences Questionnaire (SEQ) [6]. The questionnaire contains a list of items assessing the frequency of exposure to different forms of SHV experiences (e.g., sexist comments, sexual touching, sexual threats, etc.), which are assumed to reflect a sexually hostile environment. In the SEQ, the items are related to three correlated but, arguably, distinct latent variables: gender harassment, unwanted sexual attention, and sexual coercion.

The SEQ, like most other psychometric scales, was developed using classical test theory, where each item $(x_1 \ldots x_i)$ is assumed to be determined by 1) the latent variable η by a linear function denoted λ_i, and 2) an error term ε_i that is unique for each item and contains all other causes of x_i [21]. (Fig 1, panel A). After the development phase, scales, like the SEQ are often used, in practice, by summing the items, with the sum considered as an imprecise measure of the underlying latent variable. When such a scale is used to determine causal relation between an exposure and an outcome it is implicitly assumed that it is the latent variable, rather than the indicators themselves, that is causing the outcome [19] (Fig 1, panel B). Thus, with a latent variable approach, specific SHV experiences are implicitly assumed to be of little importance for mental health, except as reflections of a sexually hostile environment and sexist norms. Adequate psychometric properties of a scale, when evaluated using for instance factor analysis, it is no guarantee that this assumption holds [19, 22]. It could well be that some or all, of the indicators (the specific SHV experiences) are casually efficacious rather than the latent variable (sexually hostile environment) (Fig 1, panel C), or that both the latent variable and the indicators are causally efficacious (combination of panel B and C in Fig 1). More advanced latent variable approaches, like structural equation modelling, can detect violations of the assumed causal structure when data does not fit the assumed model [23-25], but this is not the case when sum scores are used.

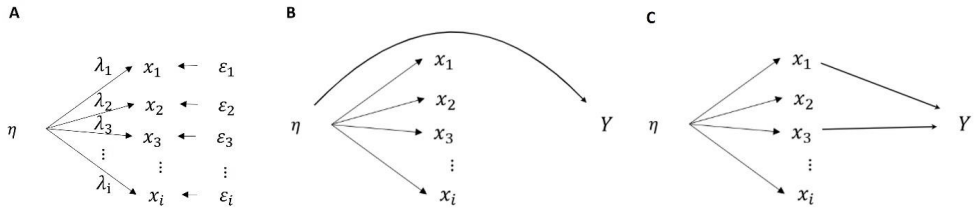

Fig 1. Panel A shows a classic measurement model. Panel B shows a structural model where only η is causally efficacious and Panel C shows a structural model where some of the indicators are causally efficacious.

We believe the assumption that only the latent variable has a causal effect on the outcomes (Fig 1, panel B) to be unrealistic when it comes to the effect of SHV exposure on mental health. For an individual experiencing SHV, exposure to a hostile sexual environment is likely part of what may cause mental health problems. However, the specific SHV experiences, for instance the specific event of unwanted touching or sexual coercion, are likely to affect the individual's mental health over and above what can be explained by experiencing sexist norms and culture.

An alternative interpretation of the association between a scale sum score and an outcome, without reference to an underlying latent variable, is to view the estimate as the *average* effect of different exposure-versions (represented by different combinations of item responses) on the outcome. Thus, we have gone full circle form where we started, and are back into the situation where we have an average effect of an ambiguous and ill-defined exposure.

Multiple versions of treatment

The multiple versions of treatment theory (MVT) provides a framework for identifying and interpreting the effects of exposures with multiple versions [26], and has recently been extended to situations involving exposures measured by psychometric scales [19].

MVT proposes that the effect of a composite exposure (containing different exposure-versions) on an outcome can be interpreted as the effect of randomly assigning people to exposure-versions in proportion to their distribution with-in levels of the exposure [19, 26], given that all exposure versions are unconfounded. In other words, the effect of a composite exposure may be viewed as the average effect of different exposure-versions weighted by their probability (i.e., prevalence among exposed) in the sample (see Fig 2 and Table 2 for illustration with hypothetical data). From this

follows that the ATE of a composite exposure will differ depending on the distribution of exposure-versions in the target population.

To exemplify, consider a hypothetical situation where different forms of SHV (exposure-versions) have different effects on depression. Such that the ATE of exposure to sexual assault is a 10-point increase in depression, the average effect of uncomfortable touching is a 3-point increase, and the average effect sexualized jokes is a 1-point increase in depression. Suppose we are not interested, or do not have data, on the specific exposure-versions but use the composite exposure "any SHV", indicating if any of the three exposure-versions were present (1) or not (0). Table 2 contains data from two hypothetical populations where the exposure-version effects are identical, but where the distribution of exposure-versions differ (sexual assault is more common in the first population and sexualized jokes are more common in the second population, Table 2). In this hypothetical example, due to the higher prevalence of the more severe exposure-version (sexual assault) in the first population, the ATE of the composite exposure "any SHV" is more than twice as large in the first population, compared to the second (see VanderWeele and Hernan [26] for formulas for calculating the ATE with composite exposures). One way to understand the ATE of a composite exposure is to view is as the balance point for the effects of different exposure-versions, with the position of each exposure-version is determined by its effect and the weight is determined by its prevalence (Fig 2).

Table 2. Hypothetical example of average treatment effects of three forms of SHV (exposure-versions) and a composite exposure in two populations

Exposure	True effect on dep.	Prevalence pop 1	Prevalence pop 2	Effect in pop 1	Effect in pop 2
Sexualized jokes	1	5%	20%	1	1
Unwelcome touching	3	15%	15%	3	3
Sexual assault	10	20%	5%	10	10
Any SHV (any of the above)	*	40%	40%	**6.3**	**2.9**

*this true effect is undefined as it depends on the probability of different exposure-versions in the target population. Dep = depression, Pop = population, SHV = sexual harassment and violence.

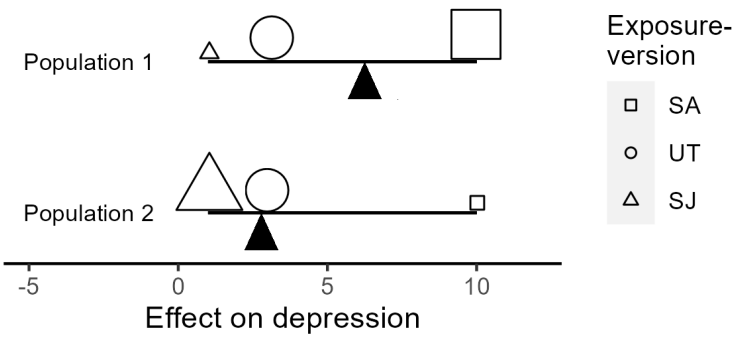

Fig 2. Illustration of how the average treatment effect of a composite exposure (filled triangle) is the average, or balancing point, of the average effects of different exposure-versions weighted by their prevalence in the population (size of the shapes) using the hypothetical example data from Table 2. ATE = average treatment effect, SA = sexual assault, UT = unwelcome touching, SJ = sexualized jokes.

The point of this example is to illustrate that if the effect of exposure-versions differs, that is if the treatment-variance irrelevance assumption does not hold, the effect of a composite exposure on an outcome will depend on the distribution of exposure-versions in the population [16]. So, even if a causal effect of a composite exposure may be estimated, it is limited in terms of transportability and interpretability [16], especially when the distribution of exposure-versions is unknown. Conceptualizations of SHV have been shown to vary considerably across sociocultural contexts [27]. With varying conceptualizations of SHV, the distribution of exposure-versions is likely to differ as well, making estimates of the mental health impact of SHV hard to generalize across contextual and cultural settings. And as mentioned above, with an unknown distribution of exposure-variants we do not know what hypothetical changes to SHV exposure that the ATE refers to, limiting its usefulness for guiding the design of interventions, as we do not know which forms of SHV are most deleterious.

Further, the effect of severe but rare exposure-variants will contribute relatively little to the ATE of a composite exposure since the effects of each exposure-version is weighted by its prevalence (Fig 2). For SHV, the prevalence of exposure differs considerably between different forms of SHV [10-12]. For instance, a coming paper by Johansson et. al. [20] found that exposure to offensive sexual remarks during the past three months was ten times more common than exposure to sex against ones will among female university students. When different forms of SHV are combined into composite exposures, this could have the unintended consequence of concealing part of the negative effects of more severe but rare forms of SHV on mental health.

A final limitation of composite exposures is that if do not know which the different exposure-versions are, it becomes difficult to determine which factors we need to control for to reduce confounding [19].

Summary and suggestions

With-in social epidemiology in general, and SHV research in particular, it will often not be possible to define exposures in an unambiguous way such that they hold the same meaning for all respondents. The social world is simply too complex. MVT can help us towards formal interpretations of causal estimates under ambiguous exposures, but it comes with limitations to interpretability and transportability.

We believe that promising way forward is to try to formulate causal questions with as little ambiguity as possible. In practice, this means estimating effects of more narrowly defined exposures. To further advance our understanding of the mental health effects of SHV, other different aspects of severity might need to be incorporated into measurement to reduce ambiguity (Table 1). When scales are used, the association between a sum score and the outcome can be supplemented by analyses of the associations of each item to the outcome, or possibly by collections of similar items to the outcome [19]. This could help generate a more nuanced and distinct understanding of the mental health effects of different forms of SHV, which may help guide prevention and intervention efforts targeting the mental health impact of SHV. While unambiguous exposures may seem like a mirage with-in social epidemiology, striving towards them will hopefully at least lessen ambiguity, and provide knowledge that may be more useful in attempts to decrease the negative impact of social exposures, such as SHV, on mental health.

Declarations

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Ethics approval

None.

Author contributions

Conception and writing of the first draft were performed by Fred Johansson. Kristoffer Magnusson provided intellectual revisions and both authors approved the final version of the manuscript.

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