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Research Report

Dynamic Bayesian network analysis of the social determinants of mental health

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Abstract

Mental disorders contribute substantially to the global burden of disease, accounting for up to 16.5% of all years of healthy life lost due to disability and premature mortality. Epidemiological evidence indicates that mental health problems are associated with a diverse range of demographic, social, and economic factors, referred to collectively as social determinants; however, the causal mechanisms underlying these associations are widely recognized to be complex and are only incompletely understood. Here, we use recently developed structure learning methods for Bayesian networks and high-quality panel data from Australia to construct a provisional dynamic network model of the causal dependencies connecting a broad selection of social determinants and mental health. This provisional causal model identifies a heterogeneous set of proximate risk-modifying factors (direct causes), including subjective financial well-being, community connectedness, loneliness, and general health, that mediate the individual-level mental health effects of all remaining variables included in our analyses. Simulation analyses indicate that ideal preventive interventions targeting people's sense of financial security, local community engagement, and loneliness have the greatest capacity to improve population mental health outcomes, while significant reductions in the prevalence of mental health problems may also be achieved by promoting physical well-being and participation in volunteer or charity work and paid employment. We conclude that policies such as a Job Guarantee that are capable of simultaneously altering multiple adverse (or protective) social and economic exposures are likely to be critical in effectively addressing the substantial personal and societal costs of mental health-related disability.

Keywords: causal inference, mental disorder, population health, risk factor, simulation

Significance Statement

Preventive interventions designed to restrict the incidence and progression of mental disorders through modification of the social and economic environments in which people live have the potential to substantially reduce mental health-related disability and premature mortality. Using recently developed structure learning methods for Bayesian networks and high-quality panel data from Australia, we construct a dynamic network model of the causal dependencies connecting a diverse selection of social determinants and mental health. Simulation analyses based on this causal model enable us to identify a set of social and economic intervention targets with the greatest capacity to deliver improvements in population mental health, including subjective financial well-being, local community connectedness, loneliness, and participation in volunteer or charity work and paid employment.

Competing Interests: J.-A.O. is Head of Systems Modelling, Simulation and Data Science at the Brain and Mind Centre, University of Sydney and Managing Director of Computer Simulation and Advanced Research Technologies (CSART). I.B.H. was an inaugural Commissioner on Australia's National Mental Health Commission (2012–2018). He is Co-Director, Health and Policy at the Brain and Mind Centre, University of Sydney. The Brain and Mind Centre operates an early-intervention youth service at Camperdown under contract to headspace. He has previously led community-based and pharmaceutical industry-supported (Wyeth, Eli Lily, Servier, Pfizer, AstraZeneca) projects focused on the identification and better management of anxiety and depression. He was a member of the Medical Advisory Panel for Medibank Private until October 2017, a Board Member of Psychosis Australia Trust, and a member of the Veterans Mental Health Clinical Reference group. He is the Chief Scientific Advisor to, and a 3.2% equity shareholder in, InnoWell Pty Ltd. InnoWell was formed by the University of Sydney (45% equity) and PwC (Australia; 45% equity) to deliver the $30 M Australian Government-funded Project Synergy (2017–2020, a 3-year program for the transformation of mental health services) and to lead transformation of mental health services internationally through the use of innovative technologies. The remaining authors declare no competing interests.
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Introduction

According to generally accepted etiological models (e.g. diathesis–stress models), mental disorders ordinarily develop as the product of dynamic interactions among predisposing biological and psychological factors (genotype, personality, etc.) and risk-modifying environmental exposures (1, 2). Epidemiological research has identified a diverse range of demographic, social, and economic factors that are consistently associated with mental health outcomes, including (among other factors) gender, ethnicity, education, employment status, financial hardship and poverty, income inequality, and social participation and support (3–5). Although substantial evidence from prospective cohort studies, randomized controlled trials, and natural experiments indicates that these potential risk-modifying factors, referred to collectively as social determinants, may contribute significantly to the emergence of psychopathology, the causal pathways connecting adverse (or protective) social and economic exposures and mental health are widely recognized to be complex and are only incompletely understood (6). Preventive interventions designed to restrict the incidence and progression of mental disorders through modification of the social and economic environments in which people live have the potential to significantly reduce mental health-related disability and pronounced inequalities in mental disorder prevalence, at least in principle (7); however, the development of effective prevention strategies will depend substantially on our ability to identify those social and economic interventions that are most capable of effecting population-level change, which in turn depends on the availability of sufficiently realistic causal models capturing the role of social determinants in generating mental health problems (5).

Dynamic Bayesian network (DBN) analysis provides a general approach for representing and (assuming specific conditions are satisfied) inferring complex causal networks that has only relatively recently been applied in mental health research (8–10). Formally, DBNs are directed acyclic graphical models that specify a joint probability distribution over a set of nT random variables corresponding to the observed values of n distinct features (i.e. measurable properties) of a stochastic dynamic system at each of T discrete time points. Assuming the observed system states (i.e. the values of the n features at each time point) are generated via a homogeneous Markov process (the simplest case), the dynamics of the system can be described using a two-time-slice DBN, comprising an initial Bayesian network, which specifies a joint probability distribution over all possible initial states, and a transition Bayesian network, specifying the conditional probability distribution $P_{\rightarrow} = P(X^{t+1} \mid X^t)$, where the random vector X^t corresponds to the system state at time point t and $P_{\rightarrow}$ applies at all time points (11–13). More generally, DBNs may be nonhomogeneous, allowing $P_{\rightarrow}$ to vary across time points, and can permit dependencies between noncontiguous system states where the Markov property cannot reasonably be assumed to apply (e.g. X^{t+1} may depend on X^{t-1} as well as X^t). Under specific conditions (including that no unmeasured confounding variables are present), the directed edges in a Bayesian network are interpretable as direct causal effects, so that DBNs inferred from observational data may be treated as provisional causal hypotheses that can be used to derive testable predictions about the effects of potential interventions on system behavior (14). Such predictions provide a means of assessing and, if necessary, refining a hypothesized causal network (via comparisons with new data) and may contribute directly to evidence-based policy development and selection.

We applied efficient structure learning methods for Bayesian networks (15, 16) to construct a provisional model of the causal dependencies underlying associations between mental health (measured via the 36-item Short Form [SF-36] Survey) and a broad range of social and economic variables (see Table S1), using panel data for a nationally representative sample of *c*. 25,000 Australian residents (aged 15 years and above) from 18 waves of the Household, Income and Labour Dynamics in Australia (HILDA) Survey (one wave per year from 2002 to 2019) (17). This provisional causal model enables us to identify a set of proximate risk-modifying factors that mediate the effects of all remaining variables on the probability of observing significant mental health problems (at the individual level) and provides a means of accommodating complex causal mechanisms —involving multiple causation, equifinality (i.e. distinct causal pathways producing equivalent outcomes), and feedback—in predicting the population mental health effects of preventive interventions targeting specific risk (or protective) factors.

Results

Two-time-slice DBN analysis

Figure 1 presents the structure of the maximum a posteriori (MAP) transition Bayesian network for a two-time-slice DBN estimated from the combined data for all HILDA Survey participants and waves via iterative Markov chain Monte Carlo (MCMC) simulation (15). Nodes in Fig. 1A correspond to the values of each variable at time points t and $t+1$, so that edges indicate direct probabilistic dependencies between variables at the same time point (contemporaneous dependencies, shown in red), consecutive time points (time-delayed or temporal dependencies, shown in black), or both the same time point and consecutive time points (blue edges). Dependencies between variables at the same time point may be considered to reflect (hypothetical) causal mechanisms operating on a temporal scale shorter than the interval between consecutive observation times (1 year in this case), with dependencies across time points reflecting mechanisms operating on a temporal scale similar to the interobservation interval (8). Mental health at time point $t+1$ depends directly on mental health, general health (including physical health and functioning), and loneliness at time point t, and participants' satisfaction with their local community and finances at time point $t+1$ (Fig. 1B). All remaining variables except alcohol consumption and caring for a disabled spouse or relative (which have no effects on other variables) influence mental health indirectly through one or more of these direct dependencies (e.g. income at time point t affects mental health at time point $t+1$ via its direct effect on participants' satisfaction with their financial situation at time point $t+1$; see Fig. 1A). Participants' satisfaction with their local community and finances at time point $t+1$ and general health, physical activity, social participation, and loneliness at time point t depend directly on mental health at time point t (Fig. 1B). As each of these variables influences mental health at later time points (directly or indirectly), the probability of a participant reporting mental health problems at any particular wave depends indirectly on past changes in mental health, so that the sequence of mental health states observed over consecutive waves may be partially attributed to feedback effects.

Estimates of the degree to which social, economic, and health-related states at time point $t+1$ are influenced by each variable at time point t are presented in Fig. 2, where the value in cell ij is equal to the probability of observing a value of 1 for the variable

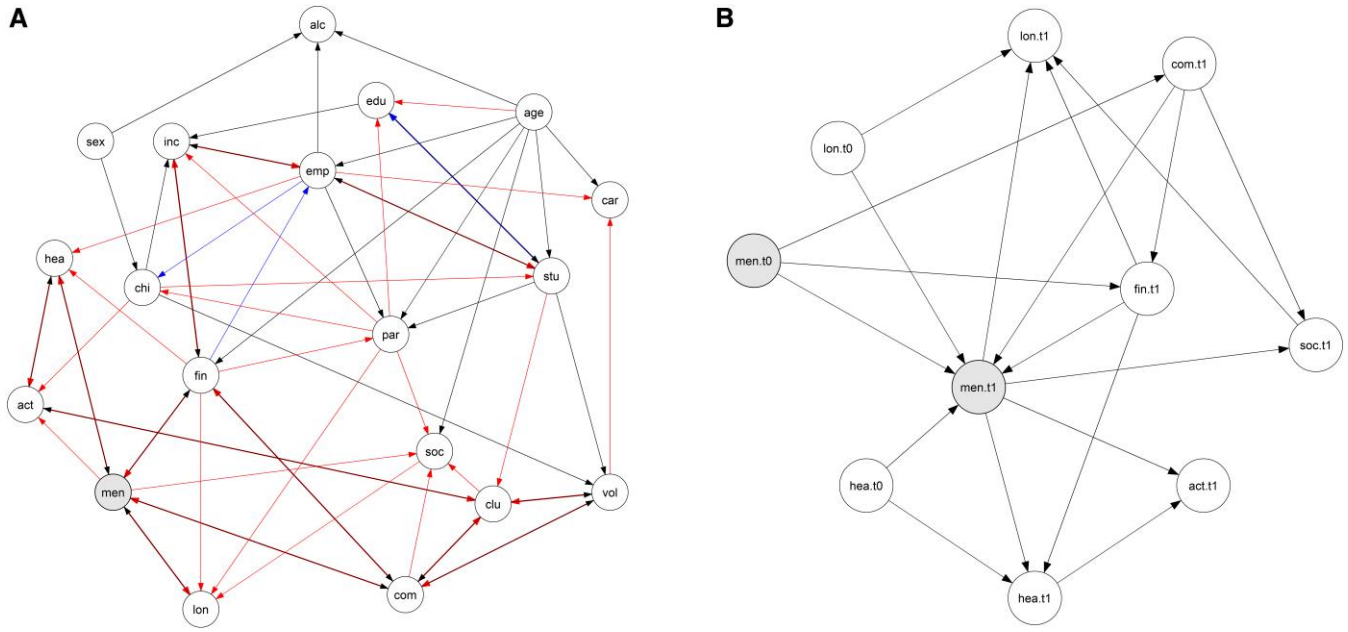

Fig. 1. MAP transition Bayesian network. A) Complete network. Nodes correspond to the values of each variable at time points t and $t+1$, so that edges indicate direct probabilistic dependencies between variables at the same time point (i.e. contemporaneous dependencies, shown in red), consecutive time points (time-delayed or temporal dependencies, shown in black), or both (shown in blue). Heavier edges indicate bidirectional relationships. Note that the value of each variable at time point $t+1$ also depends on its value at time point t (self-loops not shown). B) Partial network, showing nodes adjacent to mental health at time points t and $t+1$ (shaded gray). The suffixes ".t0" and ".t1" indicate time points t and $t+1$, respectively. Node labels: edu, postsecondary qualification; stu, studying; emp, employed; inc, household income; par, intimate partner; chi, ever had children; hea, general health; men, mental health; alc, alcohol consumption; act, physically active; car, caring for a disabled spouse or relative; vol, volunteer or charity work; clu, active member of a club or association; soc, socialize every week; fin, satisfied with finances; com, satisfied with local community; lon, loneliness.

in column j when the variable in row i is set to 1 (calculated assuming the transition Bayesian network in Fig. 1) divided by the corresponding probability when the variable in row i is set to 0 (all variables are binary, taking values of 0 or 1 only; Tables S1 and S2). Mental health problems at time point t (defined as SF-36 mental health and well-being scores <52; see Materials and methods and Table S1) have the most pronounced effect on participants' mental health in the following wave, increasing the probability of observing mental health problems at time point $t+1$ by a factor of 6.15 (515%) relative to that for participants with no mental health problems at time point t. Poor general health and loneliness at time point t increase the probability of mental health problems at time point $t+1$ by factors of 2.01 and 1.81 (101 and 81%), respectively, while participants satisfied with their financial situation and local community at time point t have a *c.* 20–25% lower risk of experiencing mental health problems at time point $t+1$ than those dissatisfied with their finances and local community at time point t (risk ratios 0.74 and 0.78, respectively). Participants reporting mental health problems at time point t are *c.* 50–60% more likely than those without mental health problems to experience loneliness and poor general health at time point $t+1$ (risk ratios 1.60 and 1.48, respectively) and *c.* 10–15% less likely to be satisfied with their financial situation and local community (risk ratios 0.86 and 0.88, respectively).

Preventive interventions: simulation analyses

The proportion of survey participants reporting mental health problems remains relatively stable over the period 2002–2012, fluctuating around a mean value of 9.38%, before increasing significantly, from 9.16% in 2012 to 12.03% in 2019 (Fig. 3A and B). Adjustment for general health, loneliness, and participants' satisfaction with their financial situation and local community (via regression analysis) has no appreciable effect on the increase in the odds of observing mental health problems after 2012, indicating that changing exposure to these proximate risk-modifying factors does not contribute significantly to the apparent deterioration in mental health over the second half of the study period (Fig. 3A). Moreover, as general health, loneliness, and participants' satisfaction with their financial situation and local community mediate the mental health effects of the remaining variables included in our DBN analysis, none of the potential risk-modifying factors considered here is capable of accounting for the observed increase in the prevalence of mental health problems. Not surprisingly then, simulated prevalence trajectories derived from the inferred two-time-slice DBN, which assumes the same conditional probability distribution $P_{\rightarrow}$ for all time points (see Introduction), fail to capture the increasing proportion of participants experiencing mental health problems post-2012 (see Fig. 3B). Estimating node-specific conditional probability tables separately for each wave allows the conditional probability of a participant reporting mental health problems at time point $t+1$ (i.e. for any particular system state at time point t) to vary across waves, substantially improving the fit of the simulated prevalence values to the empirical estimates (see Fig. 3B and Materials and methods).

Figure 3C and D presents the results of simulation analyses examining the potential effects of modifying general health, loneliness, and participants' satisfaction with their financial situation and local community on the prevalence of mental health problems across all waves (for these analyses, we used the nonhomogeneous DBN incorporating separately estimated conditional probability tables for each wave; see Fig. 3B). Modifying

	education.t1	study.t1	employed.t1	income.t1	partner.t1	children.t1	health.t1	mental health.t1	alcohol.t1	active.t1	carer.t1	volunteer.t1	club.t1	social.t1	finances.t1	community.t1	lonely.t1
age.t0	1.006	1.617	1.084	1.018	0.956	1	0.981	1.017	0.485	1	0.555	1	1	1.205	0.965	1	0.985
sex.t0	1	1.002	1	1	1	0.996	1	1	1.19	1	1	1	1	1	1	1	1
education.t0	23.908	0.971	1.006	0.822	1	1	0.998	1	1	1	0.999	1	1	1	1	1	1
study.t0	1.133	9.289	1.131	1.001	0.996	1	0.954	1	1	1.001	0.988	1.001	0.999	1	1	1	1.001
employed.t0	1.001	1.058	5.2	0.457	1.037	1.011	0.672	1	0.952	1.009	0.901	1	1.001	0.997	1	1	0.991
income.t0	1	0.998	0.92	5.649	0.997	1.002	1.058	1.048	1	0.998	1.007	1	1	1	0.901	1	1.031
partner.t0	1.003	0.993	1.008	0.802	12.891	1.037	0.997	1	1	0.998	0.999	1	1	0.882	1	1	0.721
children.t0	0.993	0.773	0.995	1.182	1	16.443	1.002	0.998	1	0.915	1.016	1.254	1.017	1.001	1.001	1.005	0.999
health.t0	1	1	1	1	1	1	13.376	2.014	1	0.841	1	1	0.986	0.987	1	1	1.146
mental health.t0	1	1	0.988	1.038	0.996	1	1.478	6.146	1	0.932	1.001	1	0.98	0.941	0.864	0.88	1.598
alcohol.t0	1	1	1	1	1	1	1	1	16.559	1	1	1	1	1	1	1	1
active.t0	1	1	1	1	1	1	0.736	1	1	2.76	1	1	1.082	1.004	1	1	0.999
carer.t0	1	1	1	1	1	1	1	1	1	1	14.823	1	1	1	1	1	1
volunteer.t0	1	1	1.001	0.998	1	1	0.994	0.969	1	1.001	1.205	7.609	1.341	1.022	1.009	1.066	0.989
club.t0	1	1	1.001	0.998	1	1	0.994	0.967	1	1.091	1.028	1.475	4.47	1.081	1.009	1.069	0.978
social.t0	1	1	1	1	1	1	1	1	1	1	1	1	1	2.301	1	1	0.87
finances.t0	1	0.998	0.977	0.856	1.018	1	0.86	0.741	1	1.009	1.002	1	1.013	1.012	2.056	1.111	0.836
community.t0	1	1	1.006	0.981	1.002	1	0.952	0.781	1	1.006	1.017	1.304	1.104	1.058	1.076	1.946	0.936
lonely.t0	1	1	1	1	1	1	1.088	1.806	1	0.986	1	1	0.999	0.99	1	1	3.463

Fig. 2. Estimates of the extent to which observed social, economic, and health-related states at time point $t + 1$ depend on the state of each variable at time point t. The value in cell ij is a risk ratio, equal to the probability of observing a value of 1 for the variable in column j when the variable in row i is set to 1 divided by the corresponding probability when the variable in row i is set to 0 (all variables are binary). The suffixes ".t0" and ".t1" indicate time points t and $t + 1$, respectively. Red and blue cell colors indicate risk ratios above 1 and below 1, respectively, with darker colors indicating stronger dependency.

participants' satisfaction with their financial situation such that all participants are satisfied with their finances at every wave leads to a 3.55 percentage point reduction in the prevalence of mental health problems in 2019 (7.99 vs. 11.54% for reference simulations derived from the DBN fitted to the empirical data), with a mean reduction across all waves of 3.04 percentage points. Maximally increasing participants' satisfaction with their local community similarly reduces the prevalence of mental health problems at each wave by a mean of 2.31 percentage points, while interventions eliminating loneliness and poor general health result in mean prevalence reductions of 2.11 percentage points and 1.88 percentage points, respectively. Predicted reductions in the prevalence of mental health problems for ideal interventions targeting more distant risk-modifying factors (those influencing mental health indirectly, via their effects on general health, loneliness, and participants' satisfaction with their finances and local community) are presented in Fig. 4. Other than increasing participation in volunteer or charity work and expanding paid employment, both of which reduce the proportion of participants reporting mental health problems at each wave by *c.* 1 percentage point, direct modification of these more remote risk (or protective) factors generally has only a comparatively modest effect on the prevalence of mental health problems over the study period.

Discussion

The results presented above provide support for several conclusions about the contributions of a broad range of social determinants to the onset and persistence of mental health problems that have significant implications for the development of effective prevention strategies aimed at reducing mental health-related disability and premature mortality. Participants' risk of experiencing mental health problems at any given time point depends directly on a heterogeneous array of moderately to highly influential social, economic, and health-related factors, including recent mental health, physical health and functioning, loneliness, a sense of financial security, and feeling part of a local community (see Figs. 1 and 2). Nearly all remaining variables included in our analyses directly or indirectly affect these proximate risk-modifying factors (alcohol consumption and caring for a disabled spouse or relative are the only exceptions; Fig. 1A), so that mental health problems may originate via multiple pathways, involving distinct (but potentially overlapping) sets of social and economic causes. Moreover, mental health at any specific time point has direct effects on general health, physical activity, social participation, loneliness, and the extent to which participants are satisfied with their finances and local community, and thereby indirectly influences the probability of observing mental health problems at future time points (Fig. 1). Collectively, these results emphasize

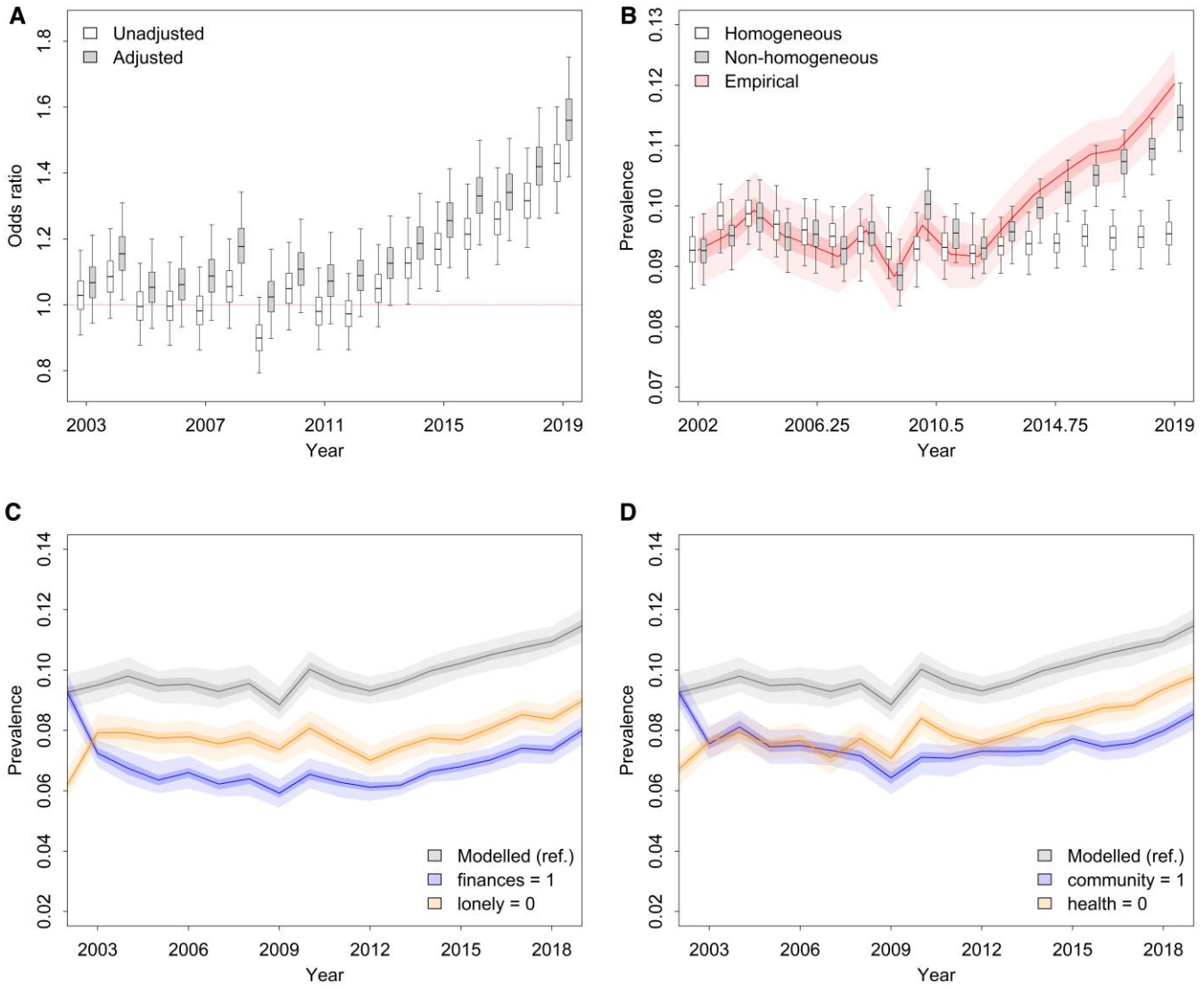

Fig. 3. Prevalence of mental health problems: regression modeling and simulation results. A) Mean odds ratios (with 50 and 95% credible intervals) derived from unadjusted and adjusted logistic regression models predicting the probability of mental health problems at each wave (wave 2, in 2002, is the reference category in both models). The adjusted model includes general health, loneliness, and participants' satisfaction with their finances and local community as covariates. B) Comparisons of empirical prevalence estimates with simulations based on homogeneous and nonhomogeneous DBN models (see Materials and methods and Results for details). C), D) Simulated prevalence trajectories for ideal preventive interventions targeting financial satisfaction and loneliness, and community connectedness and general health. The reference trajectory (in gray) is based on the nonhomogeneous DBN model. Pointwise 50 and 95% intervals derived from 200 simulation replicates are indicated with darker and lighter shading; the solid lines are mean values.

the potential complexity of the causal mechanisms underlying observed associations between mental health and a range of social determinants, providing evidence for multiple causation, equifinality (i.e. different causal pathways leading to equivalent endpoints) (18), and feedback. Allowing for this complexity (via simulation analysis), we find that preventive interventions targeting people's sense of financial security, local community connectedness, and loneliness have the greatest capacity to improve population mental health outcomes, while significant reductions in the prevalence of mental health problems may also be achieved by promoting physical well-being and participation in volunteer or charity work and paid employment.

Previous epidemiological and social research provides independent evidence supporting many (if not all) of the provisional causal dependencies identified in the transition Bayesian network in Fig. 1. Recent analyses of longitudinal data from the UK Biobank, for example, indicate that physical health (particularly multimorbidity) predicts future mental health outcomes in adults aged 40 years or more, with the incidence and persistence of depressive and anxiety symptoms increasing significantly as the number of cooccurring physical health conditions at baseline increases (19, 20). At the same time, there is substantial epidemiological evidence, including from retrospective cohort studies based on routinely collected health data for several million people, that the development of a mental disorder significantly increases the risk of subsequent physical health problems (21, 22). Mendelian randomization analysis (23) and population-based cohort studies (24, 25) similarly provide evidence for a bidirectional causal relationship between high-prevalence mental disorders and loneliness, while prospective cohort and intervention studies strongly indicate that financial stress (most often defined as a perceived inability to cover

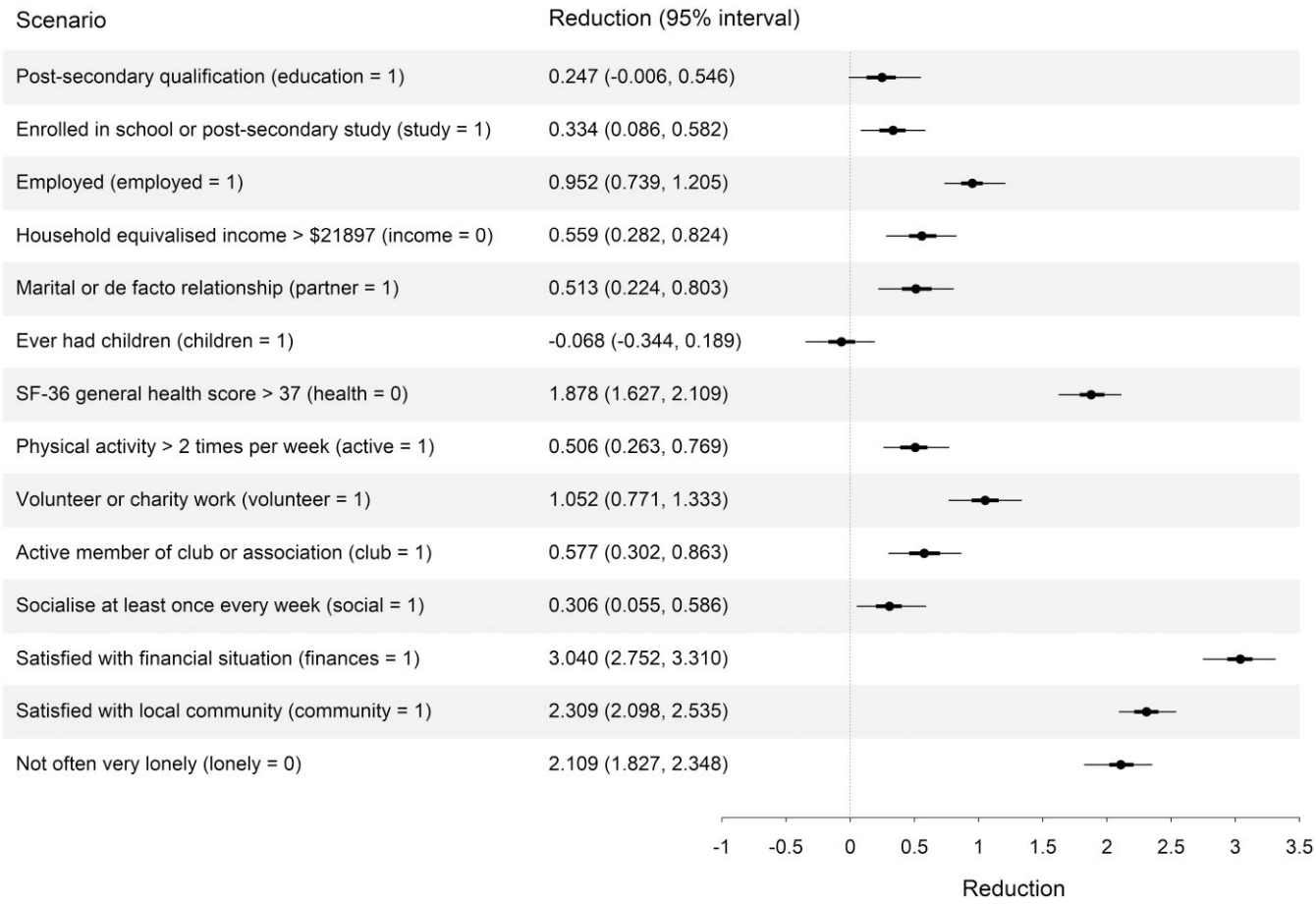

Fig. 4. Expected percentage point reductions in the prevalence of mental health problems for ideal intervention scenarios in which the value of a single risk-modifying factor is set to either 0 or 1 for all participants and waves. Mean estimates and 95% intervals derived from 200 simulation replicates are reported in the rightmost column and plotted on the far right (along with 50% intervals).

expenses with available financial assets) increases the risk of depression and other mental health problems (26, 27). Although evidence supporting an effect of local community connectedness on mental health is more limited than that for the other proximate risk-modifying factors identified in our analyses, recent prospective studies indicate that neighborhood social cohesion reduces the onset of depressive and anxiety symptoms in adolescents and young adults (28 , 29) and depression in adults aged 50 years or more (30).

Analysis of National Health Survey data for the period 2001 to 2017/2018 (six time points, 3–4 years apart) reveals that the age-standardized prevalence of very high psychological distress (Kessler 10 [K10] scores of 30 or more) for Australian adults aged 18–64 years remained relatively stable over the period prior to *c.* 2011, before increasing significantly between 2011 and 2017/2018 (31), consistent with the post-2012 increase in the proportion of HILDA Survey participants reporting mental health problems observed in our analyses (Fig. 3A and B). An increasing trend in the prevalence of clinically significant psychological distress (measured using the K10 scale) has also been inferred for the HILDA Survey sample (K10 data are available for every second wave from 2007 onward) (32) and can be attributed primarily to an increase in the incidence and recurrence of depressive and anxiety symptoms, rather than a decline in treatment-mediated recovery (such a decline could, in principle, result from an increase in treatment dropout and a corresponding reduction in the proportion of patients receiving minimally adequate care where help seeking for mental health problems is increasing more rapidly than the capacity of services to provide timely clinical interventions) (33). Although the causes of deteriorating population mental health in Australia and other high-income countries (34 , 35) are currently unclear, the regression modeling results presented in Fig. 3A suggest that the increasing proportion of HILDA Survey participants reporting mental health problems after 2012 cannot be explained by changing exposure to any of the risk-modifying factors included in our analyses, and, accordingly, must be ascribed to other causes (e.g. declining employment security and housing affordability, increasing upward social comparison related to social media use, decreasing national-level social cohesion) (36–38).

Limitations

Principal limitations of the analyses presented here include our dependence on observational data for a restricted (although still relatively broad) selection of social and economic variables, which entails that all inferences about causal effects can only be considered as provisional (14), and our use of SF-36 Survey responses rather than information from diagnostic interviews to identify participants experiencing clinically significant mental health problems. As indicated above, the set of variables represented in our DBN model (see Fig. 1 and Table S1) excludes a number of

potentially significant social and economic determinants of mental health, including employment and housing insecurity, childhood physical and/or sexual abuse, intimate partner violence, ethnic and racial discrimination, problematic social media use, and economic inequality and other factors affecting national-level (as opposed to local community) social cohesion (3, 5, 36–40). Aside from leaving our analyses open to confounding (in which inferred probabilistic dependencies between observed variables resulting only from their shared causal dependence on an excluded variable are wrongly ascribed to direct causal effects), the omission of several major social determinants of mental health restricts our ability to identify those preventive interventions that have the greatest capacity to reduce mental health-related disability at the population level. Similarly, our reliance on SF-36 Survey responses in identifying participants with relatively poor mental health, although itself not problematic, may limit the utility of our results in evaluating the potential effectiveness of alternative approaches to preventing mental disorder onset and recurrence, as participants with SF-36 mental health and well-being scores <52 (the threshold below which we considered participants to have a mental health problem; see Materials and methods) will not necessarily have a diagnosable mental health condition (and vice versa) (41).

Conclusion

Results from our DBN analyses suggest that the mental health effects of a broad array of social determinants are ultimately mediated by a heterogeneous set of proximate causes that includes subjective financial well-being, local community connectedness, loneliness, and physical health and functioning. Any of these mediating factors (or their upstream causes) may contribute significantly to the emergence of mental health problems, so that proposed policy interventions capable of simultaneously altering multiple social and economic risk-modifying factors may reasonably be assumed to hold the greatest promise for improving population mental health outcomes. Perhaps the best example of such a policy proposal is a permanent, nationally funded Job Guarantee program, providing all working-age citizens with access to adequately remunerated employment in public service roles created to support a variety of community development initiatives (environmental and community rehabilitation projects, transport and meal delivery services for the elderly, afterschool programs, etc.) (42). Aside from increasing participation in paid employment (by providing the unemployed with employment opportunities), a Job Guarantee would establish a minimum wage sufficient to support a decent standard of living, substantially improving the financial situation of many economically deprived households and promoting a general increase in people's sense of financial security (since the possibility of future unemployment is eliminated). Moreover, actively involving local communities in developing and prioritizing those initiatives receiving public support would help to ensure that a Job Guarantee program not only contributes maximally to enhancing community facilities and services, but also promotes increased community engagement, strengthening social connectedness and reducing loneliness. Along with health services delivery reforms aimed at increasing early access to effective mental health care (7), a Job Guarantee and/or other similarly far-reaching social and economic policies will likely be critical for achieving meaningful progress in reducing the vast personal and societal costs of depressive disorders, anxiety disorders, and other disabling mental health problems.

Materials and methods

Data

The HILDA Survey is a nationally representative panel survey of Australian households designed and managed by the Melbourne Institute of Applied Economic and Social Research and funded by the Australian Government Department of Social Services. Details of the survey methodology are provided in ref. (17). Briefly, data on a large number of demographic, social, economic, and health-related variables are collected for the same participants annually via interviewer- and self-administered questionnaires, including a Household Questionnaire, completed by a single household member, and a Person Questionnaire, completed by all household members aged 15 years and above. Responses were obtained for 7,682 of 11,693 randomly sampled households in the first survey wave (completed in 2001; response rate 65.7%), and a further 2,153 households were incorporated into the panel in 2011 (wave 11). Children aged under 15 years in responding households enter the survey sample as they turn 15 (at which point they start completing the Person Questionnaire), and new households are added to the panel when responding households "split" (e.g. as children leave home or couples separate), so that the sample remains representative of the national population across waves. For the DBN analyses presented here, we used data on participants' mental health, measured via the SF-36 Survey, and a broad selection of social, economic, and health-related risk-modifying factors for all waves from 2002 to 2019 (see Table S1). Numbers of participants with complete data at each wave are presented in Table S3. The HILDA Survey data collection protocols and survey instruments have been approved by the University of Melbourne Human Ethics Committee (17).

DBN analysis

Iterative MCMC simulation (15), implemented in BiDAG (16), was used to estimate the MAP transition Bayesian network in Fig. 1 and the MAP initial Bayesian network in Fig. S1. We used the Bayesian Dirichlet equivalent (BDe) score function with default parameter values (prior pseudocounts 0.5, edge penalization factor 2) and specified an initial search space defined by the skeleton of a directed acyclic graph obtained via a greedy hill-climbing search (performed with bnlearn) (43) in estimating both networks. All nodes corresponding to observed states at time point t in the transition Bayesian network were identified as root nodes (i.e. nodes with no parents), and we excluded all edges directed into age and sex at time point $t + 1$ (except edges from age and sex at time point t) using the blacklist option (these restrictions prevent effects of later states on earlier states and ensure that age and sex do not depend on other variables). For the initial Bayesian network analysis, in which we used data for a single time point only (wave 2), we specified age and sex as root nodes. Partition MCMC analyses (44) indicate that nearly all edges in the inferred MAP Bayesian networks have posterior probabilities above 0.99 (93.0% of edges in the transition Bayesian network and 87.8% of edges in the initial Bayesian network), while posterior probabilities for sampled edges that are not present in the MAP estimates are mostly <0.1 (see Figs. S1 and S2). Probabilities used in calculating the risk ratios presented in Fig. 2 were obtained from the MAP transition Bayesian network using the query function in gRain (45).

Regression modeling

We used multilevel logistic regression modeling to examine temporal patterns in the wave-specific proportions of participants

experiencing mental health problems, defined here as SF-36 mental health and well-being scores <52 (the least upper bound of the lowest 10% of scores recorded for wave 2; see Table S1). Bayesian MCMC sampling, performed with Stan version 2.32.2 (46), was used to separately fit two models to the data for all participants and waves (unadjusted and adjusted; see Fig. 3A), both having the form $\theta_{ij} = \text{logit}^{-1}(\alpha + X_{ij}\beta + \gamma_i)$, where θ_{ij} is the probability that participant i has a mental health problem at wave j, α is an intercept term, X_{ij} is a row vector of time-varying predictors for participant i, β is a column vector of regression coefficients, and $\gamma_i \sim \text{Normal}(0, \sigma^2)$ is a participant-specific random effect. The predictors in X_{ij} included indicator variables for each wave (except wave 2, which served as the reference category; see Fig. 3A) and, for the adjusted model, general health, loneliness, and participants' satisfaction with their finances and local community. We specified zero-centered normal prior distributions with SDs of 10^4 for the regression coefficients and a noninformative uniform prior distribution on the interval $[0, 10^4]$ for the SD of the random effects (i.e. σ). Four Markov chains, each initialized with parameter values sampled from the joint prior distribution, were run in parallel for 4,000 iterations, and we discarded the initial half of each chain as warmup. Potential scale reduction factors ($\hat{R}$) for all parameters were <1.01, indicating approximate convergence of the Markov chains to the posterior distribution (47). Parameter estimates (means and 95% credible intervals) for both models are presented in Table S4.

Simulation analyses

Bayesian estimates of the prevalence of mental health problems at each survey wave (mean values and 50 and 95% credible intervals, plotted in red in Fig. 3B) were derived from the empirical data assuming a binomial sampling model with a Beta(1, 1) prior distribution (47). Figure S4 illustrates the procedure used to simulate each of the 200 replicate prevalence trajectories for the homogeneous and nonhomogeneous DBN models summarized in Fig. 3B. Starting with a population of N_i simulated state vectors for wave i (each containing a single value for each variable), we generated a population of state vectors for the subsequent wave j as follows: (i) for each of the N_i state vectors, we used a regression model predicting loss to follow-up (Table S5) to calculate the probability of a participant with the simulated states appearing in one or more future survey waves, then sampled $N_j - m_j$ vectors without replacement (i.e. from the population of N_i vectors) using the calculated probabilities as sampling weights; here, m_j is the number of participants first appearing in the survey data at wave j; (ii) using either the MAP transition Bayesian network in Fig. 1 or (for the nonhomogeneous DBN model simulations) a transition Bayesian network structurally identical to the MAP network in Fig. 1 but with node-specific conditional probability tables estimated using the survey data for waves i and j only, the $N_j - m_j$ state vectors for wave i sampled in step 1 were simulated forward one time step to produce $N_j - m_j$ state vectors for wave j; and (iii) we simulated m_j state vectors using the MAP initial Bayesian network in Fig. S1 or (for the nonhomogeneous DBN model simulations) a cross-sectional MAP Bayesian network inferred from the survey data for wave j and combined these with the $N_j - m_j$ state vectors simulated in step 2. Prevalence values for wave j were derived from a sample of n_j state vectors drawn from the population of N_j vectors created in step 3, where n_j is the number of participants with complete data for wave j.

The potential effectiveness of preventive interventions targeting specific risk (or protective) factors in reducing the proportion of participants with mental health problems was assessed by simulating prevalence trajectories for ideal intervention scenarios in which the value of a single (targeted) risk-modifying factor was set to either 0 or 1 for all participants and waves (see Figs. 3C and D and 4). Prevalence trajectories were simulated following the procedure described above (see also Fig. S4), except that in steps 2 and 3 we used modified versions of the wave-specific (nonhomogeneous) transition and cross-sectional Bayesian networks, in which all edges directed into the target node (i.e. for the focal risk-modifying factor) were removed (13) and the target node state was set to the appropriate intervention value (0 for risk factors, 1 for protective factors; Fig. 4). For each intervention scenario, we simulated 200 replicate prevalence trajectories, each of which was compared with a reference simulation based on the unmodified Bayesian networks (those used to produce the nonhomogeneous DBN model simulations presented in Fig. 3B; see Fig. 3C and D). Mean percentage point differences between the wave-specific prevalence values for the reference and intervention simulations were calculated for each simulation replicate, yielding a distribution of expected differences that we summarized using simple descriptive statistics (means and 50 and 95% intervals; see Fig. 4).

Supplementary Material

Supplementary material is available at *PNAS Nexus* online.

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Author Contributions

All authors were involved in conceptualization. A.S. was involved in formal analysis and writing—original draft. I.L., M.V., F.I., J.-A.O., Y.J.C.S., M.K.C., and I.B.H. were involved in writing—review and editing.

Data Availability

The HILDA Survey data used in our analyses are available from the Australian Data Archive Dataverse (see https://dataverse.ada.edu.au/dataverse/hilda).

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