Simulating Social Mechanisms of Depression

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Abstract

Depression is a mental health disorder that affects a substantial proportion of the population and leads to individual suffering, increased mortality and economic damage. The risk of entering a depressive episode is affected by a person's history and their social environment, and can in turn affect the structure of their social network. Therefore, a systemic perspective might be helpful when trying to reduce prevalence of depression in the population. In this paper we implement a simple agent-based model in which individuals' social connections affect the likelihood that they enter a depressive episode, and calibrate it against empirical data on the prevalence, heritability and spread of depression. We found that the number of social contacts and their positive impact had a much greater effect on outcomes than the strength of the transmission of depression itself. Furthermore, none of the interventions we tested that targeted availability of therapy and negative effects of depression had a substantial impact.

Introduction

Approximately 30% men and 40% of women in the Western world will experience at least one depressive episode in their lifetime (Kruijshaar et al., 2005). Depression is a frequently recurring disorder; the risk of experiencing another episode after the first one is around 50% (Eaton et al., 2008) with the risk increasing after each episode (Solomon et al., 2000). Depression is also highly disabling (Duggan et al., 1999), with heightened risks for unemployment (Clayborne et al., 2019; Lerner et al., 2004), mortality (Angst et al., 1999; Cuijpers and Smit, 2002; Lawrence et al., 2010) and a high economic burden (Sobocki et al., 2006). Roughly half of people experiencing a depressive episode seek treatment (Bristow and Patten, 2002; Lubian et al., 2016). Among people going to therapy, approximately half experience a reduction of negative symptoms (Santoft et al., 2019). Policies for societal mental health improvement often focus on creating more mental health services (Garratt, 2023). Depression is therefore often treated only on an individual level. However, depression is strongly influenced by the social network of a person and can even affect the structure of the social network itself.

There is some evidence for a protective effect of social support on depression (Huang et al., 2020; Pfeiffer et al., 2011; Grey et al., 2020), with social isolation also predicting depression (Santini et al., 2020). On the other hand, having frequent contact with depressed individuals also heightens the risk of depression (Rosenquist et al., 2011; Andarlia and Gunawan, 2021). Studies show that the contagion risk of depression spreads to up to three degrees of separation (Rosenquist et al., 2011), similar to contagion effects found for happiness, smoking or obesity (Christakis and Fowler, 2013).

Emotional contagion theory explains contagion effects in two different ways (Paz et al., 2022). Firstly there is mood contagion: our mood is influenced by the mood of others in our environment (Joiner Jr. and Katz, 1999; Bhullar, 2012). Secondly there is imitation of behaviour: if negative emotion regulation behaviour is prevalent in our environment we are more likely to copy it, which may result in dysfunctional coping strategies and co-rumination (Rose et al., 2014).

Depression is not only influenced by the environment of a person, but also shapes and structures the social environment, since people tend to befriend people who are similar to them (McPherson et al., 2001; Block and Grund, 2014). This also extends to depression levels (Morita et al., 2022). Friendships also last longer when people are more similar (Noel and Nyhan, 2011). There is some disagreement as to the reasons behind depression homophily with some authors suggesting that these homophily effects of depression may be caused by the social withdrawal of depressed people, limiting their participation in normative network processes and leaving primarily other depressed people as their possible socialising partners (Schaefer et al., 2011). However, newer findings suggest that peer-rejection might cause social isolation in depressed people (Aronson and Bergh, 2021), thus leaving only other depressed individuals as potential sources for socialising.

Another influence on the clustering of depression in social networks is heritability. Children of depressed parents are three times more likely to develop depression than children of healthy parents (Weissman et al., 2016; Rasic et al.,

2014). Estimations for the variance of depression that is explained by genetic factors vary between 0.3-0.5 (Kendall et al., 2021; Sullivan et al., 2000).

These findings suggest that the prevalence of depression in a population is at least in part determined by feedback effects within the social network as well as within the lifetime of individuals. In order to identify the most important factors that lead to an increase in the prevalence of depression and ultimately to find an effective intervention regime it is necessary to take these systemic effects into account. Simulation modelling can be helpful in understanding the qualitative dynamics of complex systems like this one and to extrapolate the effect of interventions (Chattoe-Brown, 2013).

Some aspects of the social mechanisms of depression have been modelled before. Abdelhamid et al. (2016) for example look at the development of depression in a social network of students, without, however, including homophily and contagion effects. Andarlia and Gunawan (2021) model contagion effects of depression and recovery processes in a randomly mixing population without network structure. Benny et al. (2022) model interventions for depression but focus on effects of income and social network size on maternal depression. Silverman et al. (2015) also analyse interventions for population well-being but focus on healthcare effects and do not model contagion and homophily processes. As they focus on specific aspects of the system in isolation, none of these studies can, however, show the consequences of feedback effects between network structure, contagion, homophily and family history.

In this paper we present a qualitative agent-based model of a socially connected population where vulnerability to depression is affected by genetics, individual life history and the social environment. We calibrate the model against available population level data on prevalence and risk of depression. Using the calibrated model we investigate the importance of social factors for the development of depression and extrapolate the effect of a number of different interventions on the prevalence of depression.

Methods

We implemented an agent-based model, simulating individuals' education, career and social networks and their interaction with the individuals' likelihood to have a depressive episode.

We model social support effects, contagion effects, homophily effects and heritability of depression. The model also includes a number of outcomes and variables that are known to be affected by or interact with depression such as education (Esch et al., 2014), socioeconomic state (Lorant et al., 2003), employment (Bartelink et al., 2020; Lerner et al., 2004; Kammerling and O'Connor, 1993; Perkins and Rinaldi, 2002) and relationship quality (Horn et al., 2017; Whisman et al., 2021).

We test the effect of policies against depression on a societal level in our model. These policies target the availability of therapeutic spaces, reducing the social isolation of depression, mitigating job losses caused by depression, and reducing the number of children dropping out of education due to depression.

While this is a purely qualitative model, where possible we aimed to base parameter values on empirical data. If no data was available we tried to find values that seemed plausible to all authors and produced model dynamics similar to observed populations in preliminary simulation runs.

The model source code in Julia and the scripts used to generate the results are available online at `https://github.com/johannasop/DepressionModel`

Model description

We simulate a fixed size population of 1000 agents over 200 time steps of one year.

Setup We initialise the population by generating 800 adults and 200 children. We assign the agents' age following the empirical age distribution of the United Kingdom in 2020.

We generate 280 couples (Office for National Statistics (ONS), 2023) by randomly picking two agents with a maximum age difference of 10 years. Each previously generated child is then assigned to a randomly selected couple.

Socio-economic status (SES) for all couples and single agents is drawn from a uniform distribution between 1 and 4 and the income determined accordingly (see below). For parents and single agents the susceptibility genotype is drawn from an exponential distribution with parameter λ. See below for a description of the inheritance and the calculation of the susceptibility phenotype. A social network is generated for each agent (see below).

Population dynamics For the sake of simplicity we decided to keep population size constant over the course of the simulation. Agents die as soon as they reach the age of 80. At the end of each time step enough new agents are created to reset the population size to 1000.

Newly created agents are assigned to a randomly selected eligible couple and a social network is generated for them (see below).

Heritability In order to model the heritability of depression we implemented simple genetics and a genotype-phenotype map. Each agent has a diploid genotype for susceptibility (c_1, c_2). The genotype of newly created agents is determined by picking a random gene from each parent. The agent's phenotype s is then calculated as:

$$s = h \cdot (c_1 + c_2)/2 + (1 - h) \cdot \eta \tag{1}$$

with heritability parameter h and $\eta \sim Exp(\lambda)$.

Social connections Agents' social connections are classified by type: parents, children, a partner, friends and acquaintances.

In each time step, single agents above the age of 18 try to find a spouse with a probability of 0.4, reflecting findings about dating attitudes among adults (Pew Research Center, 2020). Candidates are randomly selected from friends, acquaintances and the general population, with probabilities of 0.35, 0.35 and 0.3, respectively. Candidates have to be unrelated single adults and have to have the same SES and a similar age to the focal agent. Candidates with the same history of depression are always accepted, others with a probability of $1 - m_s$. 200 attempts are made to find a suitable candidate. The length of relationships is drawn from a Poisson distribution with the parameter randomly drawn from $\{3, 7, 15\}$.

During the setup of the simulation and again for each newly created agent later on a social network is created from scratch. The initial number of friends and acquaintances is drawn from Poisson distributions with parameters $n_{f,0} = 4$ (see Neal, 2024; Wrzus et al., 2013) and $n_{a,0} = 15$, respectively. For friends a corresponding number of random agents that are unknown to the focal agent and whose age differs less than $5 + \text{age}/4$ is drawn from the general population. Acquaintances are assigned after friends; they are drawn from the agent's social circle (i.e. friends of friends) or the general population if the entire social circle already has become acquaintances.

Agents lose a number of acquaintances in each time step with numbers drawn from a Poisson distribution with parameter 1. They can also lose a friend with a yearly probability of 0.1. Acquaintances and friends are replaced by sampling random strangers from the focal agent's social circle. Candidates with the same depression history are always accepted, others with a probability of $1 - m_a$ or $1 - m_f$, respectively. If the social circle does not contain suitable candidates the general population is sampled instead.

From the second year of a depressive episode onward agents also have an additional chance of 0.1 and 0.3 of losing either a friend or an acquaintance, respectively, without replacement, so that social isolation effects due to depression (Aronson and Bergh, 2021; Rosenquist et al., 2011) are reflected in the model.

Income and SES An agent's SES is determined when they turn 15 and is set as a quarter of the maximum of the parents' income. The SES is further increased by 1 (with a maximum of 4) with a probability of 0.02.

Between the age of 15 and 25 depressed agents have a risk of 0.05 per year to lose a point of SES (with a minimum of 1), accounting for negative effects of depression on educational achievement (Esch et al., 2014).

An agent's income is calculated at age 25 and is drawn from a normal distribution with a mean $= 25\text{SES} - 12$ and a standard deviation of 7.5, then limited to the interval (0,100).

Depressed agents above the age of 25 have a risk of 0.05 per year to lose 10 points of their income (with a minimum of 10), accounting for negative effects of depression on employment (Lerner et al., 2004).

Depressive episodes In each time step healthy agents can enter a depressive episode. The risk of depression is the sum of various factors dependent on the agent's circumstances modified by the agent's susceptibility. We limit depression to ages 15 and above, reflecting empirical results that show that depression is rare in younger teenagers and children (Solmi et al., 2022).

Given a baseline depression rate r_0 we calculate the social effect on depression risk as:

$$r_{\text{social}} = r_0 + r_p + r_c + r_f p_f - r_{f,h} n_{f,h} + r_a p_a + r_s \quad (2)$$

The presence of at least one depressed parent increases the rate by r_p and r_c, respectively. The effect of depressed friends and acquaintances, r_f and r_a is modified by the proportion of depressed agents in the respective categories, p_f and p_a; however, each healthy friend decreases the rate by $r_{f,h}$. r_s is set as $r_{s,h}$ or $r_{s,d}$ dependent on the spouse's health status.

The final raw rate r is then calculated as a weighted mean of the social rate and the agent's raw rate at the previous time step:

$$r_t = w_{\text{hist}} r_{\text{social}} + (1 - w_{\text{hist}}) r_{t-1} \quad (3)$$

From this we obtain the probability to enter a depressive episode in the current year as:

$$P(\text{dep}) = 1 - e^{-r_t \cdot s} \quad (4)$$

where s denotes the agent's susceptibility and s_r its resilience.

Depressed agents become healthy with a fixed probability of 0.53 per year (Whiteford et al., 2013). Otherwise they can enter therapy. We assume that for reasons such as less service access or differing attitudes towards therapy, the ability and willingness to undergo therapy increases with SES (Leppänen et al., 2022; Niemeyer and Knaevelsrud, 2023), so we set the probability for therapy to 0.3 for SES 1, 0.5 for 2 and 3 and 0.8 at SES 4, averaging out to around 0.5 (Bristow and Patten, 2002; Lubian et al., 2016). Agents in therapy become healthy with a probability of 0.45. If therapy is unsuccessful an agent's probability to enter therapy in the future is reduced by 0.1 (see Hardy et al., 2019).

Calibration, sensitivity and interventions

We calibrated the model parameters that determine heritability, base risk of depression as well as the interactions between the social network and depression risk (see Table 1).

We used MCMC ABC with differential evolution (Amaya et al., 2021). As a target for the calibration we used empirical estimates of prevalence, increased risks, heritability and recurrence probability.

For the calibration against heritability we added twins to the model and estimated heritability measurements $\hat{h}_{\text{model}}$, $\hat{c}_{\text{model}}$ and $\hat{e}_{\text{model}}$ from the model results using Falconer's formula.

Increased risk dependent on contact type is computed by calculating the increase of the proportion of depressed contacts of an agent four years after a random year, in which that agent was depressed. The time interval of four years is based on Rosenquist et al. (2011) who argue that homophily effects are controlled by calculating increased risks. The increased risk for children of depressed parents is calculated by setting a random year where every person is listed as a depressed or non-depressed agent. Thirty years later the proportion of depressed children of depressed parents is divided by the proportion of depressed children of non-depressed parents (Weissman et al., 2016).

For a full list of measurements used, their values and the sources see Table 2. The distance function used in the calibration is the Euclidean distance between the two vectors comprised of empirical data and measurements in the model, respectively.

Using the parameter values found during calibration of the model we measured sensitivity of main population level results against changes in parameters that determine the shape of the social network as well as transmission of depression between individuals.

Finally, again using calibrated parameter values, we tested the effect of a number of different interventions in the model:

therapy for all All agents have a therapy probability of 1 irrespective of SES.

support for low SES Agents with low SES (1) have the same therapy probability as agents with medium SES (2,3).

job support Depressed agents do not lose income.

education support Depressed agents do not lose SES.

prevent isolation Long term depression does not lead to loss of friends or acquaintances.

Results

We find that for parents and children the transmission effect of depressed individuals in the social network had a moderate effect on population outcomes (Fig. 3). For all other social connections transmission appears to be largely irrelevant (Fig. 4, 5). Supporting effects of healthy friends on the other hand strongly improved current as well as lifetime

par.	explanation	prior	v	SD post.
r_0	base rate	$[0, 10]$	4.3	1.0
r_p	risk parent	$[0, 10]$	8.0	1.8
r_c	risk child	$[0, 10]$	0.8	0.6
r_s	risk spouse	$[0, 10]$	2.2	1.0
$r_{s,h}$	support spouse	$[0, 10]$	4.6	1.0
r_f	risk friends	$[0, 10]$	7.7	1.1
$r_{f,h}$	support friends	$[0, 10]$	1.5	0.3
r_a	risk acq.	$[0, 10]$	0.6	1.1
λ	distr. susceptibility	$[0, 1]$	0.8	0.1
w_{hist}	weight of prev. risk	$[0, 1]$	0.7	0.1
m_s	homophily spouse	$[0, 1]$	0.3	0.1
m_f	homophily friends	$[0, 1]$	0.3	0.2
m_a	homophily acq.	$[0, 1]$	0.2	0.2
h	heritability	$[0, 1]$	0.1	0.2

Table 1: Calibrated parameters, with use in the model, prior distribution (all uniform), calibrated value and standard deviation of posterior distribution

measurement	value	source
12-month prev.	0.075	Arias de la Torre et al. (2021)
$\geq$1 ep. (age 15–65)	0.25	Kruijshaar et al. (2005)
$\hat{h}$	0.37	Sullivan et al. (2000)
$\hat{c}$	0.0	ibid.
$\hat{e}$	0.63	ibid.
risk friends	4.95	Rosenquist et al. (2011)
risk acq.	2.18	ibid.
risk spouses	1.5	ibid.
risk children	3	Weissman et al. (2016)
recurr. after 1st	0.5	Eaton et al. (2008)
recurr. after 2nd	0.75	American Psychiatric Association (1995); Solomon et al. (2000)
recurr. after 3rd	0.9	American Psychiatric Association (1995); Solomon et al. (2000)

Table 2: Values and sources of empirical measurements used for the model calibration

prevalence (Fig. 4) as did the number of friends and acquaintances (Fig. 6).

None of the interventions we tested had a substantial effect on any of the outcomes we measured (Fig.7).

The distribution of a current depressive episode seems to indicate a higher prevalence for depressive symptoms in older agents (Fig. 1).

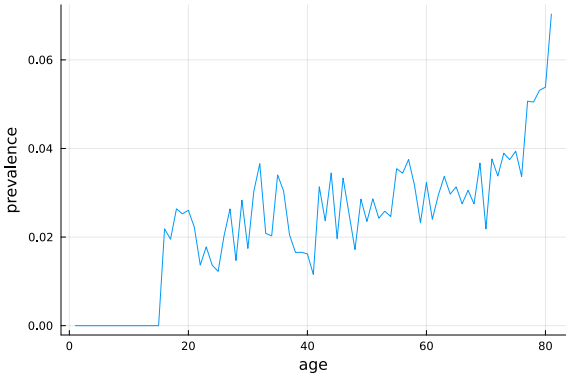

Figure 1: Prevalence of depression by age.

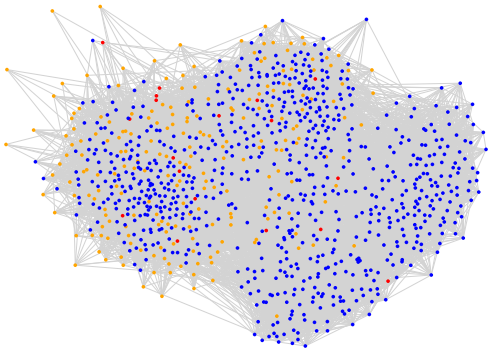

Figure 2: A snapshot of the social network at the end of a simulation using calibrated parameter values. Blue represents agents with no history of depression, orange those with a history of depression and red the ones that are currently depressed.

Discussion

Given the mounting evidence of effects of social networks on depression, we implemented a preliminary agent-based social network, in which social support effects, contagion effects, homophily effects and heritability effects impact depression outcomes.

We found a positive correlation in the age distribution for depression, which is also found by Fiske et al. (2003) and Stordal et al. (2003), validating our model regarding age

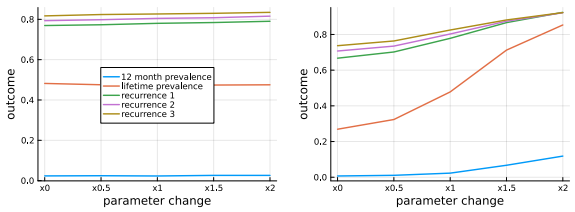

Figure 3: Sensitivity of simulation outcomes to friend homophily m_f (left) and transmission rate between parents and children r_p (right). Mean values of 100 replicates shown.

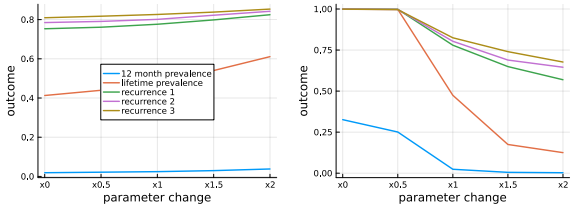

Figure 4: Sensitivity of simulation outcomes to the transmission rate between friends r_f (left) and the mitigating effect of healthy friends $r_{f,h}$ (right). Mean values of 100 replicates shown.

prevalence. The higher prevalence in older agents may be explainable with possible network changes due to mortality. Older agents are more likely to lose friends due to mortality and therefore have no buffer effect for depression left. Future research could implement mental strain due to grief and health problems to further analyse depression in old age. Furthermore, our model does not include changes in network size, except for changes due to death and depression. The social networks of people tend to decrease over their lifetime (Wrzus et al., 2013). Our model also does not include a number of social contacts that are relevant to people, such as neighbours (Rosenquist et al., 2011; Wrzus et al., 2013). Looking at these changes in the context of old people could be especially interesting.

We find that generally the supporting effect of friends and relatives as well as their number have a stronger effect on the outcome than transmission along the network. Furthermore interventions that target the accessibility of therapy or the impact of depression appear to have little effect.

In combination these results provide more insight. The parameters with the strongest influence on model outcomes (such as the rate with which healthy friends reduce symptoms) are not targeted by the tested interventions. Current interventions could therefore be focusing on the wrong influences on mental health. Since increasing the availability of therapy is expensive and does not seem to improve societal mental health overall, policy makers perhaps should target social network effects. For example, interventions could focus on strengthening the positive effects of social support

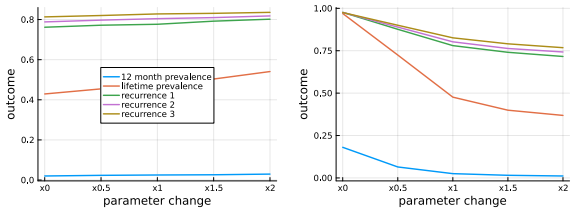

Figure 5: Sensitivity of simulation outcomes to the transmission rate between spouses r_s (left) and the mitigating effect of healthy spouses $r_{s,h}$ (right). Mean values of 100 replicates shown.

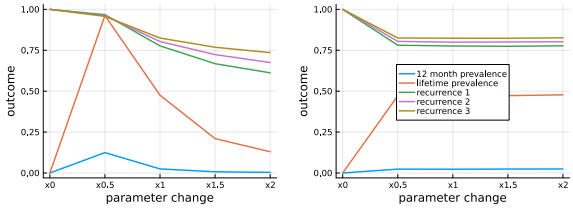

Figure 6: Sensitivity of simulation outcomes to the number of friends $n_{f,0}$ (left) and acquaintances $n_{a,0}$ (right). Mean values of 100 replicates shown.

by training positive support behaviour in schools, or offering training for social support for people in high risk groups. Such interventions could be particularly effective, since positive affect seems to be more contagious than negative affect (Bhullar, 2012).

Another important finding of the sensitivity analysis is the sensitivity of the outcome measures to the average number of friends. A higher average number of friends seem to have a strong effect on lowering depression prevalence. This is also found in a study by Thompson et al. (2022). However, they also describe a limit to the benefit of number of friends, emphasising that not only the number of friends but also the quality of the friendships are important for mental health. In the last decade worries about a 'loneliness epidemic' arose with some studies showing increased rates of loneliness (Buecker et al., 2021; Twenge et al., 2021) and depression (Wilson and Dumornay, 2022), especially in young people. Some authors suppose that one reason for rising depression rates in youth might be linked to the rising use of technology and consequently less face-to-face contact (Twenge, 2020; Twenge et al., 2019). Interventions could therefore perhaps target the fostering of close friendships by creating more spaces for face-to-face socialisation. Interestingly, the effects are not replicated for acquaintances, further emphasising the importance of friendship quality for mental health outcomes (Demır and Weitekamp, 2007).

Policies focusing only on preventing social isolation of depressed people does not seem to have an effect on outcome levels in the model. This may be because depressed

Figure 7: Effect of different types of interventions on prevalence and recurrence probabilities (10 replicates).

people tend to have more depressed friends and therefore maintaining these friendships does not have a buffering effect and may instead have a harmful impact. However, depressed people making and keeping friendships with non-depressed people is important for their mental health outcomes. Findings suggest that depressed people do not prefer to socialise with fellow depressed people (Schaefer et al., 2011), but that their social isolation from normative network processes only leaves other depressed people available for socialisation, or that peer-rejection might be the cause of social isolation (Aronson and Bergh, 2021).

To increase socialising success for depressed people, reducing the negative effects of depression on others may be beneficial. This might be possible with therapeutic interventions training positive coping mechanisms and interruption of negative coping behaviour. In these interventions negative coping behaviour that might occur in social contexts (such as co-rumination) should be especially emphasised. Furthermore, interventions should focus on informing non-depressed people on how to best support depressed people. If interventions focus on creating more understanding for the struggles of people with depression, peer-rejection could be reduced.

We note that the outcome variables analysed here might not be outcome variables that interest policy makers currently. For example, the cost and efficacy of interventions are not yet considered since these are preliminary results. Our model also does not yet include negative effects of unemployment on depression. The considerations of costs could be especially interesting regarding the analysis of prevention of job loss caused by depression. Since unemployment and mental health negatively effect each other (Lerner et al., 2004; Perkins and Rinaldi, 2002), and depression (Sobocki et al., 2006) causes a high economic burden, inter-

ventions targeting this aspect of mental health could prevent a downward spiral and in the long run prove to be especially cost-effective.

Further research and modelling effort on downward spirals could also focus on mortality and suicide. Depression is associated with a higher mortality risk (Cuijpers and Smit, 2002; Lawrence et al., 2010), at least partly due to suicides (Angst et al., 1999). Suicide bereavement in turn is also associated with a drastic negative effects on mental health and a higher risk of suicide, suggesting downward spiral effects of suicide in social networks (McDonnell et al., 2022), that perhaps interact with other discussed feedback effects. Therefore these effects and possible interventions could also be an interesting topic for future research.

Though these results are preliminary, they show interesting trends that should be considered as a starting point for more in-depth research about interventions for improving mental health on a social network basis.

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