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Modelling aspects of the effect of community stigma on the prevalence of anxiety and/or depression

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Abstract: Mental health is an important component of overall well-being, but over two in five Australians will experience a mental disorder in their lifetime. Anxiety and depression compose a large proportion of the mental disorders in Australia, and can substantially affect the lives of those affected. Stigma about these disorders is thought to adversely affect many aspects of treatment, including delaying treatment seeking behaviours, the duration required for treatment to take effect, and withdrawal from treatment.

There have been findings showing strong social clustering of anxiety and/or depression. One such postulated reason for this is that contact with people suffering from anxiety and/or depression can increase the risk of otherwise unaffected people, which is a direct analogue to "transmission". As such, we use a transmission model framework to investigate the changes in long-term prevalence of anxiety and/or depression as a result of stigma in a community affecting model pathways to and from treatment, using strata for those affected by stigma and those unaffected (neutral). The population is divided into states for those unaffected (U), affected by anxiety and/or depression (A), undergoing treatment (T), and with managed anxiety and/or depression (M). Those in the A and T states are considered to be experiencing acute affects of anxiety and/or depression and are able to affect others, whilst those in the M state are considered to still be receiving treatment but not longer able to affect others, and may be re-affected.

We first calibrate our model, showing a strong linear relationship between our "transmission" rate (β) and the rate of spontaneously experiencing the disorders (υ) to capture the reported prevalence of anxiety and/or depression. We explore the effect of stigma on model pathways related to treatment parameters on this prevalence, using univariate and bivariate sweeps. Finally, we conduct a sensitivity analysis to gain insights on how parameter estimates and ranges will affect future prevalence estimates.

We found that increasing levels of stigma in a community nonlinearly increased the burden of anxiety and/or depression. This result was consistent for all calibrated parameter combinations explored. We also showed that, as expected, modelled burden was most sensitive to the transmission rate (β), and next most sensitive to the average periods of time spent being actively treated (ω, σ_n). We further explored the impact of the most sensitive combinations of the effects of stigma on the model parameters. Surprisingly, we found a strong relationship between the calibrated values of the spontaneous rate of experiencing the disorder (υ), and the transmission rate (β). This relationship suggested transmission was always larger, and is further evidence of a transmission framework being appropriate to explore anxiety and/or depression in this framework.

It is important to emphasise that the progression of anxiety and depression are nuanced, with a complex array of underlying drivers and risk factors. We have taken a simplified approach, and focus on likely effects of parameter combinations on long-term population prevalence of anxiety and/or depression to mitigate the limitations of our approach. Overall, this helps provide information on the most important parameters needed to better understand how policies might affect the overall mental health of a population with regards to anxiety and/or depression, in the presence of stigma affecting treatment-related model pathways.

Keywords: *Mental health, stigma, anxiety, depression, transmission model*

1 INTRODUCTION

Mental health is an important part of health and wellbeing. The recent National Study of Mental Health and Wellbeing (ABS 2022*b*) found that over two in five Australians have experienced a mental disorder at some time in their life, with one in five having experienced a disorder in the 12 months prior to the survey. Of the mental disorders, anxiety and/or depression (AD) are relatively common, with the study having identified that in the 12 months prior to the survey, 16.8% of the population experienced symptoms of anxiety disorder(s) and 4.6% had experienced depressive episode(s) (ABS 2022*b*). AD can negatively impact the way people live, through withdrawal of social activities or avoidance of situations, to the point it becomes difficult to function (ABS 2022*b*). As a reflection of the mental health burden in Australia and its impact, 7.6% of total government health expenditure, $11.0 billion total or $431 per person, was spent on mental health related services in Australia in 2019-2020, and 32.5% of mental-health related subsidised prescriptions were for antidepressants in 2019-2020 (AIHW 2022*b*).

Anxiety and depression are complex, nuanced conditions, with many studies exploring the complex interactions of drivers and risk factors (see, for example, (ABS 2022*b*, AIHW 1999, 2022*a*, Liu et al. 2016, Simanek & Meier 2015)). Here we make many simplifying assumptions on the progressive structure of AD to explore some of the potential effects of stigma. Stigma is also complex and nuanced, with multiple aspects interacting with mental health in different ways (Corrigan & Watson 2002, Harman & Heath 2017, Torales et al. 2023). Here, we focus on negative effects on model pathways to and from treatment. Depending on the precise definition of stigma, the estimated proportions of the population affected varies greatly (Harman & Heath 2017), thus, to offset our possible overestimate of the prevalence of AD, we use the lower end of the range, 15%, as the default value.

Based on the well-established concept of "Emotional Contagion" (see, for example, Fowler & Christakis (2008), Kramer et al. (2014), Rosenquist et al. (2011)), we use a transmission model framework to explore the levels of AD in a community. Rosenquist et al. explored the hypothesis that depressive symptoms may spread from person to person in social networks, based on data from the Framingham Heart Study (Rosenquist et al. 2011). They find people are 95% (CI 59–135%) more likely to be depressed if they are directly connected to someone who is depressed, and 43% (21–70%) for people at two degrees of separation.

Using a simplified progression of AD in a transmission model framework, we explore how model pathways to and from treatment being negatively affected by stigma changes the population-level mental health burden.

2 METHODS

2.1 Model of progression through states of anxiety and/or depression

To model the population level effects of stigma about anxiety and/or depression (AD) in communities, we first consider a simplified version of progression through states of AD. We assume that people start by being unaffected by anxiety or depression (U). At some point, a person might become acutely affected by AD (A), and after a delay will receive treatment for AD (T) in the form of counselling and/or pharmaceutical interventions. After a period of receiving treatment, people may then be considered to have "managed" AD (M), by which we mean not suffering from acute effects but may still be receiving ongoing treatment. The full progression between these states is depicted in Figure 1, noting one can progress through the four states in order, or become re-affected by AD (due to relapse or new exposure) from the treatment or managed states, and that returning to an original "unaffected" state (i.e. "recovering") is possible after time in the "managed" state.

As per the classic compartmental epidemiological modelling approach, we assume the population is well mixed (so equally likely to come into contact with any other individual in the population, regardless of their current state or stigma status), and homogeneity within compartments/states (everyone in a state has identical properties, with respect to AD). Due to the latter assumption, we have stratified the population into those affected by stigma (subscript s), and those unaffected by stigma (subscript n), as shown in Figure 1.

Unlike a traditional compartmental model, it is possible for people to "spontaneously" (in a transmission sense) become (re-) affected by AD. Subsequently, progression through the stigmatised or neutral strata of the simplified AD states at the population level is governed by a nonlinear system of ordinary different equations (ODEs), where t denotes time, dot derivative with respect to time, and most parameters are defined in Table 1,

$$\dot{U}_n = (1-p)\mu N - (\lambda_n(t) + \nu_n + \mu)U_n(t) + \omega_n M_n(t)\,, \tag{1}$$

$$\dot{A}_n = (\lambda_n(t) + \nu_n)U_n(t) - (\gamma_n + \mu)A_n(t) + \psi_n T_n(t) + (c\lambda_n(t) + \eta_n)M_n(t)\,, \tag{2}$$

$$\dot{T}_n = \gamma_n A_n(t) - (\psi_n + \sigma_n + \mu)T_n(t)\,, \tag{3}$$

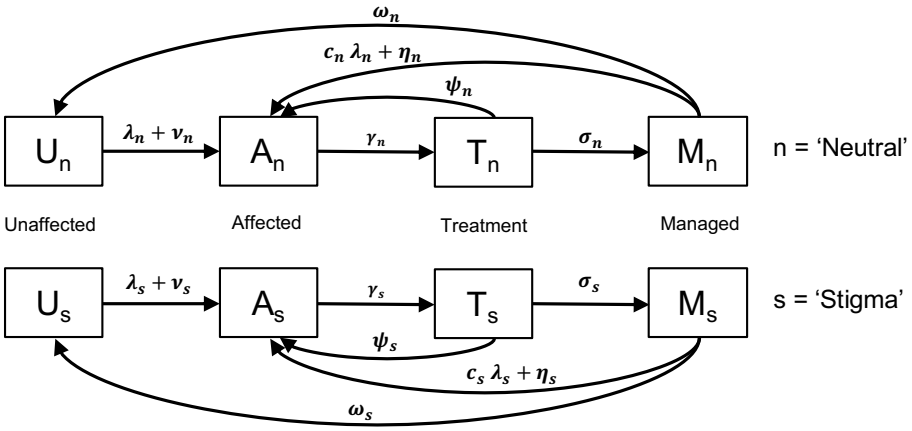

Figure 1. A simplified model of progression through states of anxiety and/or depression. The system of ODEs is fully described in (1)–(8), and parameters are fully described in Table1. Note demography and interactions (see (9)) between strata have not been depicted for visual clarity. Arrows show the direction of movement between the various states, with the parameters denoting the rate of movement.

$$\dot{M}_n = \sigma_n T_n(t) - (\omega_n + c\lambda_n(t) + \eta_n + \mu)M_n(t)\,, \tag{4}$$
$$\dot{U}_s = p\mu N - (\lambda_s(t) + \nu_s + \mu)U_s(t) + \omega_s M_s(t)\,, \tag{5}$$
$$\dot{A}_s = (\lambda_s(t) + \nu_s)U_s(t) - (\gamma_s + \mu)A_s(t) + \psi_s T_s(t) + (c\lambda_s(t) + \eta_s)M_s(t)\,, \tag{6}$$
$$\dot{T}_s = \gamma_s A_s(t) - (\psi_s + \sigma_s + \mu)T_s(t)\,, \tag{7}$$
$$\dot{M}_s = \sigma_s T_s(t) - (\omega_s + c\lambda_s(t) + \eta_s + \mu)M_s(t)\,, \tag{8}$$

and the total population $N = U_n + A_n + T_n + M_n + U_s + A_s + T_s + M_s = 1$. Since we have no data to suggest that those affected by stigma are more susceptible or more infectious, the associated rate parameters are assumed equal for the stigma and neutral classes, i.e. we let $\nu_n = \nu_s$, $\eta_n = \eta_s$, and $\lambda_n(t) = \lambda_s(t)$, so the equivalent of the transmission model "force of infection" is

$$\lambda_n(t) = \lambda_s(t) = \beta\left(A_n + A_s + aT_n + aT_s\right)/N\,, \tag{9}$$

where the transmission rate β incorporates the contact rate between people and the probability of being affected by AD given contact with someone affected by it. We have assumed a constant population size, including not considering deaths as a consequence of AD. The prevalence of AD is given by $N(t) - U_n(t) - U_s(t)$.

2.2 Model calibration and simulation scenarios

Due to the number of simplifying assumptions we have made, including a simplification of both the progression through AD and the drivers and risk factors, we focus on the steady state of this system of ODEs. That is, we do not consider this model sufficiently realistic that exploring the transient behaviour is of value. Due to the combination of two strata and nonlinearity, there is not an analytic solution for the steady state. We subsequently solve the system numerically, in Matlab R2022a with 'ode15s', where code is available from `https://github.com/rihickson/model-for-anxiety-depression`. We note the steady state is reached in approximately 5 years, but all simulations are run to 50 years to ensure stable values. This delay is important to note when considering implications for policy decisions.

We use a least squared error approach to calibrate the model. Our target prevalence, α, is based on the combined prevalences for any anxiety disorder or depressive episode from the National Study of Mental Health and Wellbeing (ABS 2022*b*). We note this may result in the double counting of some portion of the population, and that the α =21.4% reported were for the 12 month period preceding the survey date, not at a given point in time. However, since the average duration of a first major episode of depression is estimated to be 114-181 weeks (AIHW 1999), this approximation is sufficient for calibration for the purposes of this study, where we explore the effect of stigma on model pathways into and out of treatment. For this calibration, we had too many free parameters (β, η, and ν), so we calibrated the values for β and ν, and assumed $\eta = 2\nu$ based on the

Table 1. Detail about the model parameters for System (1)–(8). Note all rates are in units of per day.

Symbol	Description	Values [min, max]	Source
μ	1/(average lifespan)	$3.2e^{-5}$	ABS (2022*a*)
β	Transmission rate, such that transitions are given by (9)	$6.6e^{-4}$ $[1.4e^{-9}, 4.1e^{-1}]$	Calibrated
a	Transmission modifier for those undergoing treatment	1	Assumed
c	Modifier for transition rate to the affected state from those with managed anxiety or depression	1	Assumed
ν	Rate of spontaneously becoming affected by anxiety or depression from the unaffected state	$1.5e^{-4}$ $[2.3e^{-13}, 6.1e^{-2}]$	Calibrated
η	Rate of spontaneously becoming re-affected by anxiety or depression from the managed state	2ν	AIHW (1999)
ω	1/(time after becoming managed that a person returns to the unaffected state)	$1.4e^{-3}$ $[2.7e^{-4}, 1.9e^{-2}]$	AIHW (1999)
p	Proportion of population experiencing stigma, i.e. in s	0.15 $[0, 1]$	Harman & Heath (2017)
γ_n	1/(time until someone affected by anxiety or depression seeks treatment)	$5.5e^{-3}$ $[1.4e^{-3}, 1.9e^{-2}]$	Wang *et al.* (2007)
h	Relative effect of stigma on the timing of treatment seeking behaviour, such that $\gamma_s = h\gamma_n$	0.17 $[0, 1]$	Assumed
ψ_n	Rate at which people withdraw from the treatment state T and return to acutely affected state A	$1e^{-4}$ $[1e^{-3}, 1e^{-5}]$	AIHW (1999)
k	Relative effect of stigma on the withdrawal rate from treatment, such that $\psi_s = k\psi_n$	1.1 $[1, 2]$	Assumed
σ_n	1/(time from treatment starting to becoming managed)	$5.5e^{-3}$ $[5.5e^{-4}, 3.3e^{-2}]$	AIHW (1999)
g	Relative effect of stigma on the rate people become managed, such that $\sigma_s = g\sigma_n$	0.9 $[0, 1]$	Assumed

finding that 50% of people who initially recover following treatment relapse in the short term (AIHW 1999). The calibration was implemented using 'fmincon' in Matlab R2022a, with specified lower bounds of 0.0 and upper bounds of 1.0, and 1000 uniformly randomised starting combinations of values (independently for β and ν) within this interval.

For simplicity, we also use α to calculate the initial conditions, $U_n(0) = (1-p)N - A_n(0)$, $A_n(0) = \alpha(1-p)N$, $U_s(0) = pN - A_s(0)$, and $A_s(0) = \alpha pN$. The initial conditions have no bearing on the results, as there is no chaotic behaviour and we study the steady state solutions.

Where there is no evidence that stigma affects parameters, we have assumed the neutral values (n strata), which leaves four scenarios to explore. **First**, *the effect of the proportion of the population (potentially) experiencing stigma about AD*, p. **Second**, *the effect of stigma delaying the treatment seeking behaviour* of those experiencing AD, by letting $\gamma_s = h\gamma_n$ and exploring the effect of $h \in [0, 1]$. $h = 1$ corresponds to no effect from stigma, while as $h \to 0$ the effects of stigma on treatment seeking becomes severe. **Third**, *the potential effect of stigma on the progression from treated to managed states* of AD, by letting $\sigma_s = g\sigma_n$, where $g \in [0, 1]$ has the same relationship with stigma as h (i.e. $\sigma_s \leq \sigma_n$). **Finally**, by considering *the effect of stigma on withdrawal from treatment*, by letting $\psi_s = k\psi_n$, $k \in [1, \infty)$, where unlike the previous parameters, as $k \to 1$ stigma is having no effect, but as $k \to \infty$ stigma is having more severe effects (i.e. $\psi_s \geq \psi_n$). For each of the single parameter sweeps, the default parameter values (expected values from Table 1) are used for all other parameters.

We explore the effects of stigma on treatment-related model pathways by explicitly considering the steady state solutions with parameter sweeps, and through a sensitivity analysis. For the sensitivity analysis, we use a standard combination of Latin Hypercube Sampling (LHS) and the multivariate Partial Rank Correlation Coefficient (see, for example, (R. I. Hickson 2011)). Our focus output of interest is the steady state prevalence of AD in a community. That is, the proportion of the population experiencing any state of AD, namely $1 - U_n(\infty) - U_s(\infty)$. Since we are assuming a constant population size and ignoring overall effects of demography, we exclude μ from the sensitivity analysis. Since there is no evidence of difference in the ability of those undergoing treatment (a) or for changes in community effects by those with managed AD (c) we also

do not include them in the sensitivity analysis. The calibrated parameters and η were independently varied for the sensitivity analysis, η with expected, minimum, and maximum values of $2\times$ the respective values of ν.

3 RESULTS

For the calibration, we removed any values of the transmission rate β and spontaneous rate of becoming (re)-affected ν, resulting in prevalences of anxiety and/or depression (AD) of > 0.215 or < 0.115. We focussed on removing larger values of prevalence given our target α is likely an overestimate, resulting in 561 pairs. Due to a parameter identifiability issue, we explored the relationship between these two parameters and found $\nu = 2.051 \times 10^{-4} - 9.10645 \times 10^{-2}\beta$ with an $R^2 = 0.9996$. This gives a strong relationship between how much AD could be considered "spontaneous" versus related to the mental health status of close family and friends. We subsequently fit a Gamma distribution to the remaining values of ν, and found the values within $\pm$std$/2$ of the median, leaving 244 values. We used these and the corresponding values for β for simulations with multiple values. For simulations with single values, we randomly selected from the set of 244 and used that throughout (the expected values reported in Table 1 are these values).

We first show, Figure 2, how the proportion of the population in each of the AD states in the steady state changes with different proportions of the population (potentially) experiencing stigma. It is important to note that the y-axis represents the proportion of the total population N, not the stratified sub-populations. As such, this shows the population shifts from the n to the s strata with increasing values of p (proportion experiencing stigma). The interesting change is in the relative proportion of the population in each of the states, notably with a reduction in U_s compared with U_n, resulting in a net change in prevalence of AD as a direct consequence of the effect of stigma on treatment-related model pathways. These results suggest that the steady state prevalence of AD is a useful summary metric, which we therefore focus on in all subsequent figures.

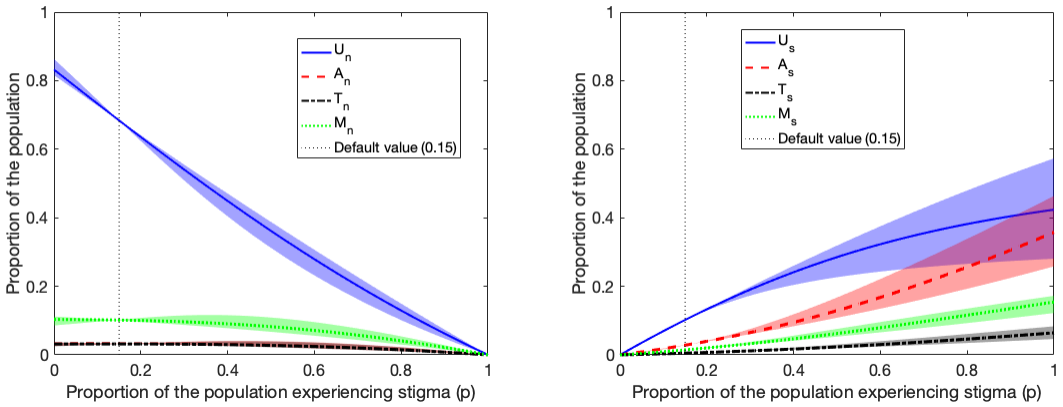

Figure 2. How the steady state values of the anxiety and/or depression states change with the proportion of the population (potentially) experiencing stigma (p), for the neutral strata (left) and the stigmatised strata (right). The shaded regions show the results for the feasible pairs of transmission rate β and rate of spontaneously becoming affected ν identified during calibration.

Figure 3 shows how the prevalence of AD changes with univariate changes in the relative effects of stigma on model pathways to and from treatment. As k (effect of stigma on treatment withdrawal) had no visible effect on the prevalence, it is excluded for visual clarity. This lack of impact on the prevalence is likely due to the small proportion of the population undergoing treatment in the s strata ($\approx 0.5\%$ of the population for expected parameter values). The ranges shown are for the calibrated value pairs of the transmission rate β and rate of spontaneously becoming (re-) affected ν, showing small variance around the expected values (that is, a reduced height in the shaded areas, such as that seen for $p \approx 0.15$ and $g > 0.5$).

Figure 4 depicts the sensitivity of the steady state prevalence of AD to model parameters. As expected, the transmission rate β has the most impact on prevalence. The relative ordering of the four key parameters we explore, p, h, g, and k (from most to least impact) reflects the relationships with prevalence shown in Figure 3. The most important use of this result is in highlighting the more valuable areas to improve our understanding of how stigma affects treatment-related pathways for those affected by AD in communities.

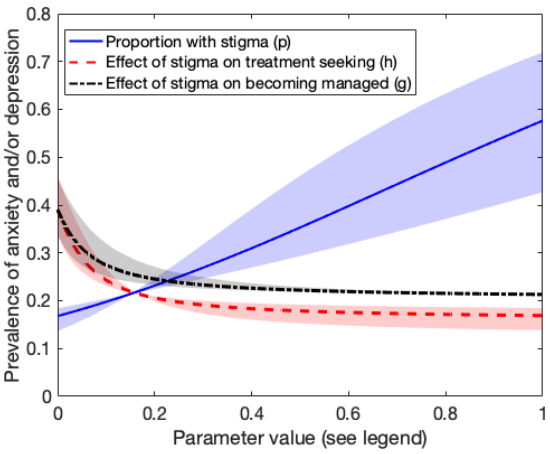

Figure 3. Change in the steady state prevalence of AD under different scenarios of stigma impacts. The parameters p, g, and h are explored individually, with all other parameters held constant (Table 1) for each sweep.

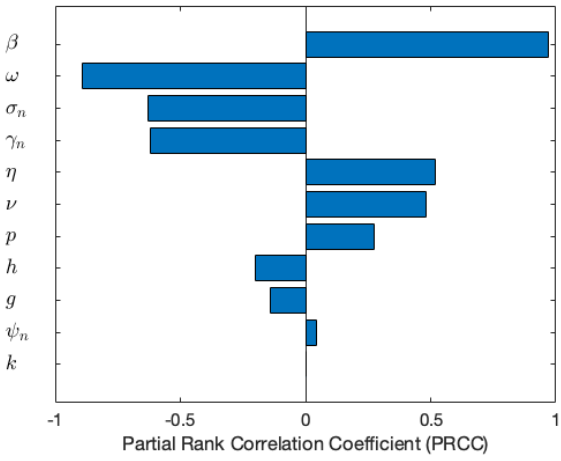

Figure 4. Sensitivity analysis of the steady state prevalence of AD to model parameters. The sign indicates whether prevalence is increased (positive) or decreased, and the PRCC magnitude the impact on prevalence (larger magnitude more sensitive).

To better understand the relationship between the effects of stigma on model pathways to and from treatment and the steady state prevalence of AD, we conducted bivariate parameter sweeps. The most impactful combinations are shown in Figure 5. The combinations of proportion of the community experiencing stigma (p) and either effect of stigma on how long it takes to seek treatment (h; left) or on time it takes to become managed (g; right) show resulting AD prevalences with ranges between 0.18–0.96. We also explored the effect of the combination of p (proportion experiencing stigma) and k (effect of stigma on treatment withdrawal), and as expected given the low sensitivity of the prevalence to k, the vertical contour lines showed variations in prevalence were almost entirely due to p, with values in the range 0.18–0.48 (not shown). Similarly, a consideration of the effect of g and h on prevalence results in fairly equally effected changes from $\approx$0.18–0.35 (not shown).

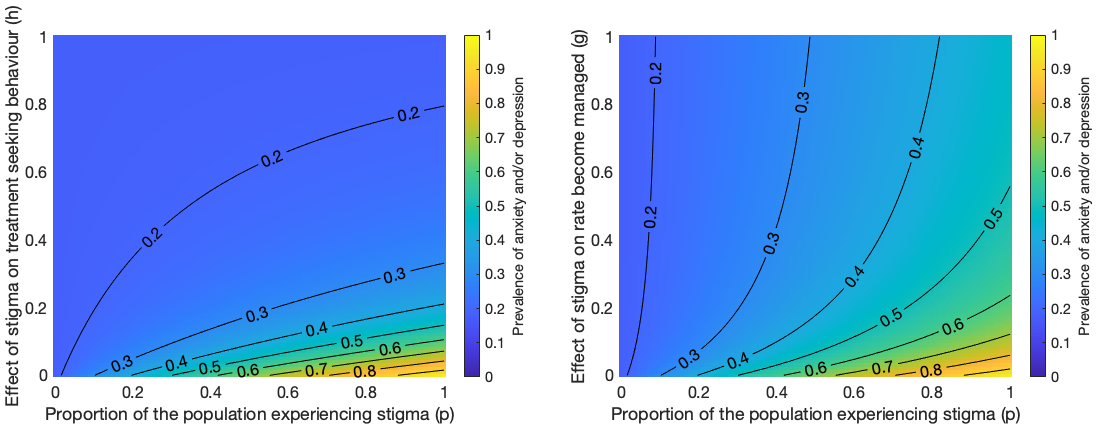

Figure 5. Change in the steady state prevalence of AD due to interactions of the proportion of the population experiencing stigma p (x-axis) and the strength of the effect of stigma on (left) delaying treatment seeking behaviour h ($h = 0$ strong effect, $h = 1$ weak effect); or (right) rate of progression to a managed state g ($g = 0$ strong effect, $g = 1$ weak effect). The prevalance of AD is shown by the heat map, with yellower colours indicating higher values.

4 DISCUSSION AND CONCLUSIONS

This is a first simple model to explore an epidemiological modelling approach to understanding the potential effects of stigma on model flow in and out of treatment for those affected by anxiety and/or depression (AD). Through this modelling process, we have identified data relevant to understanding population level dynamics of AD. Given the relationship identified between the transmission rate (β) and rate of spontaneously becoming affected (ν) through the calibration, we believe this indicates an infectious diseases framework for modelling AD prevalence in a community is appropriate, though care must be taken in the interpretation of the results.

Our simplified progression structure introduces a number of limitations. As such, it is important that the results of this simple model are interpreted in a more qualitative way, improving our understanding of relative effects. Another important consideration is that constructs such as stigma are difficult to quantify but have known qualitative effects. An example appropriate use of our findings would be to use this to prioritise further studies to understand the relationship between the clusters of AD, understanding how community levels of stigma affect delays in treatment seeking, and the time needed for treatment to allow progression to the managed state. That is, when considering the long-term population level prevalence of AD, our model suggests the effect of stigma on the withdrawal rate from treatment is a lower priority.

There are many possible enhancements to this modelling approach that follow this further evidence a transmission modelling framework is qualitatively appropriate. For example, including known seasonal effects (seasonal affective disorder AIHW (1999)), enabling movement between or differences in interactions between the stratified populations, include known age group and gender differences AIHW (1999), allowing prior episodes of AD to influence the rates of progression ($A \to T$, $M \to A$), and not assuming everyone (eventually) receives treatment.

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