

Logistic Regression Model with Jumps for Depression

Author

Charles Mwangi ⁽¹⁾; Kennedy Nyongesa ⁽²⁾; Everlyne Odero ⁽³⁾

Author's email: cwmwangi@kabarak.ac.ke

(1) Kabarak University, Kenya; (2,3) Masinde Muliro University of Science and Technology, Kenya.

Cite this article in APA

Mwangi, C., Nyongesa, K., & Odero, E. (2026). Logistic regression model with jumps for depression. *Journal of physical and applied sciences*, 5(1), 22–36. <https://doi.org/10.51317/51neqd27>



A publication of Editon
Consortium Publishing (online)

Article history

Article history

Received: 2026-08-13

Accepted: 2026-09-09

Published: 2026-10-15

Scan this QR to read the paper
online



Copyright: ©2026 by the author(s).

This article is an Open Access article distributed under the terms and conditions of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International License (CC BY-NC-SA 4.0).



Abstract

This study develops a Bayesian logistic regression model incorporating treatment-like jumps to predict depression transitions among police officers. Six symptoms (insomnia, guilt, suicidal ideation, psychomotor retardation, anxiety and fatigue), each scored from 0 to 3, are linked to latent normal or depressed states through a logit function. Treatment is modelled as a jump that reduces one symptom score, with scores floored at zero, yielding a finite-mixture recovery probability. Data were simulated in R 4.4.5 for 100,000 officers, without using clinical records. Officers were classified as depressed if any symptom score exceeded zero, and recovery probabilities were generated for depressed officers using the treatment-adjusted model. Model parameters were estimated using Metropolis-within-Gibbs Markov chain Monte Carlo (MCMC), with 30,000 iterations and a 10,000-iteration burn-in. The estimates recovered the simulation's true parameter values, with convergence occurring within approximately 1,000–1,600 iterations. However, symptom coefficient estimates under treatment jumps of sizes one, two and three had posterior standard deviations comparable to their means, indicating substantial uncertainty and no clearly distinguishable effects from zero. Modelled transition probabilities ranged from 0.5001 to 0.5037 across the treatment scenarios. Probabilities decreased for officers with symptom scores above one but remained unchanged for symptom-free officers. Within the simulation, the jump model produced lower depression-transition probabilities than the treatment model, which in turn produced lower probabilities than the conventional logistic regression model. The findings demonstrate the model's potential for incorporating symptom-specific treatment effects while highlighting the need for validation using clinical data.

Keywords: Bayesian inference, depression, jump process, logistic regression, MCMC.

1.0 INTRODUCTION

Depressive disorders constitute a major contributor to the worldwide burden of disease. According to the Global Burden of Disease Study 2021, approximately 332 million people globally were living with depressive disorders, which ranked second among all causes of years lived with disability and represented nearly 6 per cent of the global total (GBD 2021 Diseases and Injuries Collaborators, 2024). Clinically, depression is marked by persistent low mood or loss of interest and pleasure, accompanied by disturbed sleep, appetite, concentration and energy, and it impairs social and occupational functioning (World Health Organization, 2023). Community prevalence depends heavily on how depression is measured. A meta-analysis of community surveys from 30 countries involving 1,112,573 adults estimated an aggregate point prevalence of 12.9 per cent (95% CI [11.1, 15.1]), a one-year prevalence of 7.2 per cent and a lifetime prevalence of 10.8 per cent. The pooled point prevalence was 17.3 per cent in studies using self-report screening instruments, compared with lower estimates from diagnostic interviews (Lim et al., 2018).

The Kenya National Commission on Human Rights (2011) estimated that 20–25 per cent of outpatients and up to 40 per cent of inpatients in Kenyan health facilities have some form of mental illness. That estimate refers to mental illness as a whole rather than depression, and to people already attending health facilities rather than the general population. It is reproduced, with the same scope, in the Ministry of Health's national clinical guidelines for common mental disorders (Ministry of Health, 2024). The underlying facility survey is more specific. Among adult inpatients and outpatients screened with the Beck Depression Inventory at ten general medical facilities in Kenya, 42 per cent had symptoms of mild to severe depression, while only 4.1 per cent had a psychiatric condition recorded in their files, indicating that the recorded detection rate was far below the symptom rate (Ndeti et al., 2009). Depression also appears at high rates in defined clinical groups. Kuria et al. (2012) reported a prevalence of 63.8 per cent at intake among 188 alcohol-dependent persons entering detoxification and rehabilitation, assessed using the Composite International Diagnostic Interview. This figure describes an alcohol-dependent, treatment-seeking sample and is not a population estimate for Kenya. Depressive symptoms have likewise been documented among adults living with HIV in rural Kilifi (Nyongesa et al., 2019) and among patients of traditional health practitioners (Musyimi et al., 2017), while poverty, unemployment and substance misuse recur as correlates in Kenyan research (Musyimi et al., 2017).

A systematic review and meta-analysis of 67 studies covering 272,463 serving police personnel in 24 countries estimated pooled point prevalences of 14.6 per cent for depression or depressive symptoms (95% CI [10.6, 18.6]), 14.2 per cent for post-traumatic stress disorder, 9.6 per cent for generalised anxiety disorder, 8.5 per cent for suicidal ideation, 25.7 per cent for hazardous drinking and 5.0 per cent for alcohol dependence (Syed et al., 2020). Depression estimates were higher in studies from Africa, Asia and Australia (24.9%) than in European (10.6%) or North American (15.0%) samples. Most contributing studies used self-report instruments, and between-study heterogeneity was high; therefore, these figures represent symptom prevalences rather than confirmed diagnoses. Syed et al. (2020) also found that occupational stress was the strongest measured risk factor for depression (OR = 3.74, 95% CI [2.70, 5.18]) and suicidal ideation (OR = 1.72, 95% CI [1.52, 1.93]). Avoidant coping and higher alcohol consumption were associated with post-traumatic stress disorder, while greater peer support was associated with fewer post-traumatic stress symptoms. These findings suggest that organisational conditions, rather than exposure to critical incidents alone, may account for much of the measured risk.

In a meta-analysis of studies involving first responders, 33.1 per cent (95% CI [26.7, 40.1]) endorsed stigma-related items concerning mental health care, and 9.3 per cent (95% CI [7.0, 12.3]) endorsed explicit barriers to care. Fear concerning confidentiality and potential damage to career prospects were the most frequently reported concerns (Haugen et al., 2017). A review of 44 studies of law enforcement suicide identified lack of organisational support, traumatic exposure, shift work, stigma associated with help-seeking, domestic difficulties and alcohol use as recurrent correlates of suicide and suicidal ideation. The review also noted that estimated suicide rates vary according to the comparison population used (Violanti et al., 2019). Using national occupational mortality surveillance data, Violanti and Steege (2021) found a proportionate mortality ratio for suicide of 154 (95% CI [147, 162]) among law enforcement workers relative to all employed decedents. Alcohol misuse among officers has been associated with critical-incident exposure and trauma symptoms (Ménard & Arter, 2013), as well as officer-perpetrated domestic violence (Oehme et al., 2012). These findings derive from high-income settings and predominantly male samples, limiting their direct applicability to Kenya.

The Taskforce on Mental Health in Kenya recommended a mental health literacy curriculum for the National Police Service, alongside the Prisons Service and the Kenya Defence Forces (Ministry of Health, 2020). The National Police Service Commission reports administrative figures of approximately 12,000 officers with work-related mental health problems and annual averages of 13 homicides and 11 suicides within the service. These are service records rather than estimates from a prevalence survey, and no published prevalence study of depression in the Kenyan police service was identified (Leley, 2025). The Commission launched a Policy on Counselling and Welfare in 2018 and has since deployed counsellors and social workers across its regions. The National Police Service Commission (Amendment) Act, 2025, gives that policy statutory force and provides for psychosocial support centres in every county (National Police Service Commission, 2025).

Few studies provide statistical models that predict depression among police officers from observable symptoms while also representing treatment within the model. The main objective of this study is to model depression among police officers using logistic regression and extend the approach to models incorporating jumps.

2.0 LITERATURE REVIEW

Symptoms and Prediction of Depressed State

The model developed here treats six graded symptoms as observable indicators of a latent state that is not directly observed. Therefore, the relevant issue in the symptom literature is not whether each symptom is associated with depression, but how the symptoms differ in predictive strength, direction of effect and measurement reliability. Regarding predictive strength, insomnia is a well-established prospective predictor. In a meta-analysis of longitudinal epidemiological studies involving individuals who were not depressed at baseline, Baglioni et al. (2011) reported an odds ratio of 2.60 (95% CI [1.98, 3.42]) for subsequent depression onset. Batterham et al. (2012) found a similar association in a prospective cohort after accounting for personality. Geoffroy et al. (2018) described the converse relationship in a population-based sample, in which sleep disturbance was highly prevalent during major depressive episodes. Taken together, these studies suggest that sleep symptoms provide information about depressive status both before and during an episode. This property is relevant to a model that uses the same symptom vector to estimate transitions from the normal to the depressed state and persistence in the depressed state.

Ionescu et al. (2013) characterised anxious depression as a presentation associated with greater severity, poorer treatment response and higher suicide risk. Anxiety may therefore provide information about both entry into and persistence in the depressed state. With this model formulation, the coefficients in both the β and α vectors are expected to be positive, which agrees with the estimates provided in Tables 1 and 3. Suicidal ideation occupies an ambiguous position because it is included as a symptom in this model but is also treated as an outcome in occupational research. Syed et al. (2020) reported a pooled prevalence of 8.5 per cent for suicidal ideation among serving police personnel. Including suicidal ideation as a predictor may improve sensitivity to depressive severity while also conflating symptom severity with suicide risk. Psychomotor retardation has long been regarded as a relatively specific sign of depression (Sobin & Sackeim, 1997). Wüthrich et al. (2022) demonstrated that psychomotor slowing can be measured objectively using actigraphy rather than relying exclusively on clinical ratings. Retardation and insomnia can be assessed using objective or psychometrically validated measures, including actigraphy and the Insomnia Severity Index (Morin et al., 2011). By contrast, Tilghman-Osborne et al. (2010) found substantial variation in the definitions and measurement of guilt across studies and age groups, with limited agreement on what constitutes excessive guilt.

The model presupposes that each of the six symptoms can be assessed consistently using a shared ordinal scale comprising four categories, scored from 0 to 3. This assumption implies that the symptom scores are sufficiently comparable for use within the same model, although equal numerical distances between ordinal categories do not necessarily represent equal clinical differences. The reviewed evidence supports this measurement assumption unevenly, and the model specification does not explicitly account for differences in measurement reliability across symptoms. Such differences may contribute to unmodelled heterogeneity and complicate the interpretation of the posterior standard deviations associated with the guilt and anxiety coefficients, which indicate considerable uncertainty in the estimated effects.

Treatment and Effect on Symptom Severity

The treatment literature answers a different question from the prediction literature, and the mismatch between the two is what the jump formulation is intended to bridge. Kamenov et al. (2017) pool trials of psychotherapy, pharmacotherapy and their combination and find combined treatment most effective for functioning and quality of life, but the outcome in that synthesis, as in the trials it summarises, is a response rate or a mean change in a total score. Treatment enters as a single average effect on an aggregate, so nothing in the result indicates which symptom moved or by how much. First, treatment can be aimed at a single symptom and still act on the depressive state: the TRIAD study of combined pharmacotherapy and cognitive behavioural therapy for insomnia in patients with comorbid depression and insomnia targets sleep specifically, and finds that response varies with a measurable baseline sleep characteristic (Edinger et al., 2023). This is the closest empirical analogue to a jump that lowers one symptom score. Second, response is heterogeneous rather than uniform. Thase (2011) reviews treatment-resistant depression and shows that a substantial minority of patients do not remit despite adequate trials. A treatment term that subtracts a fixed amount from every treated patient cannot represent that; a mixture in which a jump of size zero occurs with probability p and a jump of size one, two or three occurs with probability $q = 1 - p$ can. The graded ordinal scales used to measure response, of which the Insomnia Severity Index is a validated example with established cut-offs for detecting treatment response (Morin et al., 2011), make a downward move along a severity scale a faithful representation of how improvement is actually recorded.

The trials and meta-analyses measure change in symptoms; they do not re-estimate the coefficients linking symptoms to the state. The quantity targeted here therefore has no direct empirical counterpart in the literature, which is both the justification for examining it by simulation and the reason the resulting estimates describe a property of the model rather than a clinical effect.

Transition, Mixture and Depression Models

Oskooyee et al. (2011) used a Markov chain to predict the severity of major depression, while Demic and Cheng (2014) modelled transitions between disease states in depression. Both approaches yield interpretable transition probabilities across a small state space, and the two-state normal and depressed structure adopted in this study draws on this tradition. However, these approaches differ from the present model in their treatment of the observed state. In the present formulation, the state is latent and inferred through a logit link applied to six observed symptom scores. Consequently, the transition probabilities are functions of symptom profiles rather than directly observed state labels. Ali et al. (2024) and Nivetha et al. (2025) developed compartmental differential-equation models of depression dynamics, with the latter incorporating optimal control. These models represent treatment explicitly, unlike many individual-level prediction models, but treatment operates as a control applied to population compartments rather than as a change in an individual's symptom profile. Their parameters are also not estimated directly from individual symptom scores. Best et al. (2010), by contrast, modelled serotonin kinetics mechanistically at a level of biological detail that cannot ordinarily be inferred from clinical symptom records. The proposed jump model occupies an intermediate position: it operates at the individual level and uses symptom scores, as in Markov and logistic models, while representing treatment explicitly, as in control-theoretic models.

Cramer et al. (2016) conceptualised major depression as a dynamic system of interacting symptoms, in which symptoms may have causal significance and changes in one symptom may alter the overall configuration. This perspective provides a rationale for considering how treatment-induced changes in an individual symptom might influence the probability of a depressive state. However, Guloksuz et al. (2017) demonstrated the importance of model specification choices in symptom-network research. This comparison highlights a distinction between the expressive capacity of network models and the interpretability of logistic regression coefficients, which quantify changes in log odds under the specified model. Bzdok and Meyer-Lindenberg (2018) reviewed machine learning applications in precision psychiatry, highlighting the potential trade-off between predictive performance and interpretability. Because discrimination measures were not calculated in the present study, its predictive performance cannot be directly compared with that of machine learning approaches.

The proposed jump model changes one symptom score at a time, with the jump magnitude treated as a random variable. The recovery probability, π_{DN} is represented as a finite mixture with weights determined by the probabilities of different jump outcomes. Finite mixture theory provides a mathematical framework for constructing such likelihoods (McLachlan & Peel, 2000), while Markov chain Monte Carlo (MCMC) techniques enable the estimation of posterior distributions in situations where closed-form analytical solutions cannot be obtained or are computationally infeasible (Gamerman & Lopes, 2006). Grazian and Robert (2018) are relevant to the interpretation of the resulting estimates because weak identification in mixture models can complicate posterior inference. This consideration is pertinent to the near-zero posterior means and comparatively large posterior standard deviations obtained in the present study. Reviews of simulation modelling in mental health (Brailsford et al., 2009; Long & Meadows, 2018) support

the use of simulation to investigate model behaviour when individual-level empirical data are unavailable. However, simulation characterises the behaviour of the specified model under its assumptions rather than establishing its validity for real patients.

The literature indicates that police officers constitute a population with substantial mental health burdens and that occupational stress is an important correlate of depression. A systematic review and meta-analysis covering 272,463 serving police personnel estimated a pooled prevalence of depression or depressive symptoms of 14.6 per cent and identified occupational stress as the strongest measured risk factor for depression (OR = 3.74) (Syed et al., 2020). Research among first responders also indicates that stigma may impede help-seeking, with approximately one-third endorsing stigma-related barriers to mental health care (Haugen et al., 2017). Nevertheless, much of this literature is descriptive, reporting prevalence estimates and associations at the group level rather than providing a model that estimates an individual's probability of depression from their symptom profile.

The Kenyan literature has a complementary limitation. Existing studies have examined depressive symptoms among adults living with HIV (Nyongesa et al., 2019), patients of traditional health practitioners (Musyimi et al., 2017), adolescents and young adults, alcohol-dependent patients (Kuria et al., 2012), and general medical facility attendees (Ndeti et al., 2009). However, these studies do not establish the prevalence of depression among Kenyan police officers. Furthermore, the National Police Service Commission's reported figures are administrative counts rather than estimates derived from a population-based prevalence survey (Leley, 2025). Two gaps therefore remain: the need for a symptom-based predictive model relevant to police officers and the absence of identified Kenyan police-specific prevalence data on which to estimate and validate such a model. This study addresses the first gap through simulation but cannot resolve the second without empirical data from the target population.

The individual-level prediction models discussed in the literature represent depressive states and symptoms but do not explicitly incorporate treatment-induced changes in individual symptom scores (Demic & Cheng, 2014; Oskooyee et al., 2011). Similarly, the treatment literature evaluates therapeutic outcomes primarily through aggregate score changes and response rates, leaving the symptom-to-state coefficients largely unexamined (Edinger et al., 2023; Kamenov et al., 2017; Thase, 2011). Models that explicitly represent treatment often apply it to population compartments rather than to individual symptom profiles (Ali et al., 2024; Nivetha et al., 2025). The reviewed literature therefore does not establish how the contribution of an individual symptom to the probability of a depressive state changes following treatment. The rationale for investigating this question comes from evidence that treatment can target a specific symptom (Edinger et al., 2023) and that treatment responses vary across patients (Thase, 2011). The proposed jump model brings these considerations together in a simulation framework, with its findings interpreted as properties of the model rather than demonstrated clinical effects.

3.0 METHODOLOGY

Symptoms and States

For each officer, a clinician records six symptoms on a four-level scale: 0 = none, 1 = mild, 2 = moderate, 3 = severe. This is the kind of graded scoring used in instruments such as the Insomnia Severity Index (Morin et al. 2011). The symptoms are x_1 = insomnia, x_2 = guilt, x_3 = suicidal ideation, x_4 = psychomotor retardation, x_5 = anxiety and x_6 = fatigue. The latent state is Normal (N) if all six scores are zero, and Depression (D) if at least one score is above zero. The transition probabilities π_{NN} , π_{ND} , π_{DD} and π_{DN} are functions of $X = (x_1, \dots, x_6)$ and form a 2×2 matrix.

Logistic Model

The probability of depression is modelled with a logistic link:

$$\log[\pi / (1 - \pi)] = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \beta_4 x_4 + \beta_5 x_5 + \beta_6 x_6$$

and the persistence probability in the depressed state is modelled in the same way:

$$\log[\pi_{DD} / (1 - \pi_{DD})] = \alpha_0 + \alpha_1 x_1 + \alpha_2 x_2 + \dots + \alpha_6 x_6$$

Jump (Treatment) Model

Treatment is represented by a jump of size j that lowers a symptom level:

$$X^*i = X_i - j, \text{ with } 0 \leq X^*i \leq 3$$

A jump of size 0 occurs with probability p , and a jump of size 1, 2 or 3 occurs with probability $q = 1 - p$.

When one symptom i is above zero, the recovery ($D \rightarrow N$) probability is the finite mixture.

$$\pi_{DN} = p\pi_{DD}(X_1, \dots, X_i, \dots, X_6) + q\pi_{DD}(X_1, \dots, X^*i, \dots, X_6)$$

When two symptoms are jumped, the mixture has components p^2 , pq , qp and q^2 . With all six symptoms, it has seven terms, weighted p^6 , p^5q , ..., q^6 , as the thesis writes them.

Likelihood and Posterior

Let y be the number of officers showing symptoms and $y' \leq y$ those who recover under jumps, so y' is binomial with parameter π_{DN} and size y . The joint likelihood of the $N \rightarrow D$ and $D \rightarrow N$ parts, multiplied by independent normal priors on α and β , gives the posterior used for estimation.

Estimation

Parameters α and β were estimated simultaneously with a Gibbs sampler. Each coefficient was updated in turn from its conditional posterior, starting from initial values of 1 for the base model. Estimates converged to the simulation's true values after about 1,000 iterations for a jump of size one and about 1,300 for a jump of size two. The thesis appendix reports a sampler with 30,000 iterations, a 10,000 burn-in and step size 0.2, with jump probability $p = 0.4$ for no jump. All simulations used R 4.4.5.

4.0 RESULTS AND DISCUSSION

This section presents and discusses the results of the study.

Jump of Size One

This section presents the results of parameter on the model with jump size one.

Table 1: Parameter Estimates, Jump of Size One

Coefficient	Mean	SD	Naive SE
β_0	0.0005199	0.000998	0.0000231
β_1 (insomnia)	0.0006827	0.0009474	0.0000212
β_2 (guilt)	0.0012048	0.0010121	0.0000226
β_3 (suicidal)	0.0011298	0.0010529	0.0000235
β_4 (retardation)	0.0011539	0.0010219	0.0000229
β_5 (anxiety)	0.0012028	0.0009739	0.0000218
β_6 (fatigue)	0.0012241	0.0010297	0.0002302
α_0	5.75E-04	0.001008	2.25E-05
α_1	6.48E-06	0.001033	2.31E-05
α_2	1.17E-03	0.000992	2.22E-05
α_3	1.18E-03	0.000984	2.20E-05
α_4	1.15E-03	0.001006	2.25E-05
α_5	1.20E-03	0.000984	2.20E-05
α_6	1.27E-03	0.001025	2.29E-05

All coefficients are positive, so every symptom raises the probability of depression. Insomnia has the smallest contribution and fatigue the largest. The transition probabilities for ten sample officers (Table 2) lie between 0.50014 (no symptoms) and 0.50226. They fell for officers with symptoms above level one and were unchanged for officers whose symptom level was zero. The cumulative probability curve was a step-shaped empirical distribution function over a narrow range of about 0.50 to 0.54.

Table 2: Transition Probabilities, Jump of Size One

Y	Insomnia	Guilt	Suicidal	Retardation	Anxiety	Fatigue	π_{JND}
1	2	0	2	1	0	2	0.501688644
1	1	2	2	2	1	0	0.502209936
1	0	2	1	0	2	2	0.502258735
1	0	0	0	0	2	1	0.501061248
1	2	1	0	1	0	0	0.500756149
0	0	0	0	0	0	0	0.50014375
1	0	2	1	0	1	0	0.501323747
1	0	0	0	0	2	0	0.500743749
1	0	1	0	0	2	2	0.501671244
1	0	2	0	2	1	2	0.502238735

Jump of Size Two

Estimates converged at about 1,300 iterations (Table 3). Again, all coefficients are positive. Insomnia has the smallest value ($\alpha_1 = 0.000243$, $\beta_1 = 0.000207$) and guilt the largest among the α 's ($\alpha_2 = 0.001224$). The transition probabilities for the sample officers range from 0.50144 to 0.50235. They decreased for officers with symptoms above level one and were unchanged where symptoms were zero.

Table 3: Parameter Estimates, Jump of Size Two

Coefficient	Mean	SD	Naive SE
α_0	0.000576	0.001021	2.28E-05
α_1	0.000243	0.000992	2.22E-05
α_2	0.001224	0.001024	2.29E-05
α_3	0.001183	0.000992	2.22E-05
α_4	0.001225	0.001027	2.30E-05
α_5	0.001215	0.000986	2.21E-05
α_6	0.001184	0.001018	2.28E-05
β_0	0.0005894	0.0009672	0.00002561
β_1	0.0002073	0.0010066	0.0002862
β_2	0.0011806	0.0009891	0.0002885
β_3	0.0011047	0.0009986	0.0002824
β_4	0.0011581	0.0010095	0.00002775
β_5	0.0011783	0.0010041	0.0002870
β_6	0.001239	0.0010059	0.00002850

Comparison with the Treatment and Ordinary Logistic Models

For ten sample officers, Table 4 compares the depressed-to-normal probabilities across the three approaches, as reported in the thesis. In the jump model, π_{JDN} is closest to π_{NN} , so the thesis ranks the jump model first, the treatment model second and the ordinary logistic model third. A second comparison against the jump of size one gives the same ordering, with the jump model having the lowest π_{ND} , followed by the treatment model and then the ordinary logistic model.

Table 4: Comparison of the Models

π_{NN}	π_{DN} (logistic)	π_{TDN} (treatment)	π_{JDN} (jump)
0.500135725	0.946156383	0.502944191	0.501688644
0.500135725	0.955395047	0.503347700	0.502209936
0.500135725	0.945698091	0.503707057	0.502258735
0.500135725	0.804895321	0.501932590	0.501061248
0.500135725	0.838676042	0.501479996	0.500756149
0.500135725	0.841863218	0.500129975	0.500756149
0.500135725	0.893053521	0.502505879	0.501323747
0.500135725	0.803173480	0.501927765	0.500743749
0.500135725	0.870855485	0.502552528	0.501671244
0.500135725	0.945974221	0.503700857	0.502238735

Discussion

The jump formulation behaves as its specification requires. Lowering one symptom score in a randomly selected fraction of officers reduces the modelled probability of the depressed state for those officers, and officers whose scores are already zero are unaffected because the jump is floored at zero. Neither result is an empirical finding. Given positive symptom coefficients and a floor at zero, no other outcome is arithmetically possible, so these results confirm that the implementation matches the specification rather than telling us anything about treatment.

The ordering of the three models reported in Table 5 should be read in the same way. The jump model yields the depressed-to-normal probabilities closest to π_{NN} , followed by the treatment model and then the ordinary logistic model, but this ranking was not established by any formal model-comparison criterion. No information criterion, held-out predictive check, discrimination measure or calibration measure was computed, and the comparison rests on ten officers. What the ordering records is which of the three ways of parameterising treatment produces one-step probabilities nearest π_{NN} in this particular simulation, which is a statement about the parameterisation and not a demonstration that one model fits better than another.

The observed ordering does not establish that officers return from the depressed state to the normal state more quickly under the jump model than under the treatment model. All reported quantities are one-step transition probabilities conditional on a symptom profile. The model does not include an explicit time index, a sojourn-time distribution or simulations over repeated periods, and the time spent in each state is not modelled. Consequently, a transition probability that lies closer to the normal-to-normal transition probability at a single step provides no information about the number of steps or elapsed time required to return to the normal state. Assessing recovery speed would require calculating the expected sojourn time or expected number of steps under each specification, or conducting longitudinal simulations over repeated periods and comparing the distributions of time to first return to the normal state. Neither analysis was performed. The comparison is therefore limited to one-step transition probabilities, and any claim about the relative speed of recovery remains unverified. Similarly, the long-run behaviour of the transition matrix was not calculated, so the available results do not establish whether recovery is permanent or sustained.

The treatment can be directed at a single symptom and still act on the depressive state is documented empirically (Edinger et al. 2023), and the mixture weights p and q correspond to the heterogeneity of response documented by Thase (2011), in which a substantial minority of patients do not remit despite adequate treatment. Both findings are encoded in the model as structure. They are inputs to the specification rather than results obtained from it, and the simulation cannot confirm them because it was built to contain them. What would constitute a genuine result, namely an estimate of how much a given symptom's contribution to the probability of the depressed state changes once that symptom has been treated, is reported here only for data whose true coefficients were fixed by the analyst, so the estimates recover the simulation's own parameters and carry no information about treatment in patients. The contrast with Kamenov et al. (2017) is informative in a negative way: that meta-analysis reports precisely the aggregate effect this model sets out to disaggregate, and because no reviewed empirical study disaggregates treatment effects by symptom, there is at present no external quantity against which the symptom-specific jump effects could be validated.

Comparison with the symptom-based evidence reveals one feature of the estimates that is directionally consistent with prior research, although the results do not establish an empirical relationship. All symptom coefficients were positive under jumps of size one and two, consistent with the general association between these symptoms and depression. However, this consistency provides limited evidence because the true coefficients in the simulation were specified as positive. The comparatively small insomnia coefficient obtained under the different jump scenarios also differs from the evidence that insomnia is a strong prospective predictor of depression onset (Baglioni et al., 2011). Nevertheless, this discrepancy reflects the parameter values selected for the data-generating process rather than evidence about the relative importance of insomnia in clinical populations. The negative estimate for insomnia under a jump of size three ($\beta_1 = -0.00003$) does not demonstrate that treating insomnia was effective. Its posterior standard deviation of approximately 0.00098 is much larger than the magnitude of the estimate, indicating substantial uncertainty about the direction of the effect. This uncertainty is consistent with the identification challenges that can arise in mixture models (Grazian & Robert, 2018). Similar caution applies to the guilt and anxiety coefficients because their posterior uncertainty may also reflect differences in symptom measurement reliability, particularly given the documented variation in how guilt is defined and measured (Tilghman-Osborne et al., 2010).

Unlike the Markov formulations of Oskooyee et al. (2011) and Demic and Cheng (2014), the state here is latent, and the transition probabilities are functions of the observed symptom vector rather than of an observed state label, which is the intended advance and is realised in the results. But those formulations derive much of their value from repeated application of the transition matrix to characterise long-run behaviour, and that analysis is absent here, which is the same gap identified in the preceding paragraph on recovery speed. Relative to the compartmental models of Ali et al. (2024) and Nivetha et al. (2025), treatment acts on an individual officer's symptom profile rather than on population compartments, which is the intended difference. Relative to Cramer et al. (2016), the model adopts the premise that perturbing a single symptom can shift the wider configuration and operationalises it, but because the symptom coefficients are additive and independent and no interaction terms are included, it cannot represent the symptom-to-symptom propagation that gives the network account its explanatory content. The status of the results can therefore be stated explicitly. Established, but as consequences of the specification: that jumps lower modelled transition probabilities for symptomatic officers, and leave symptom-free officers unchanged. Established as a statement about the estimation procedure: that the sampler recovers the true coefficients of the data-generating process within roughly 1,000 to 1,600 iterations. Not established: that the ordering of the three models reflects anything beyond how treatment is parameterised; that recovery is reached more quickly under jumps

Limitations

Four limitations bound what can be claimed from these results. The first and most consequential is that every result comes from simulated data rather than from clinical records, so the estimates recover the parameters of a data-generating process chosen by the analyst and carry no information about depression in serving officers. The second concerns precision. Posterior means are on the order of 0.001, and their posterior standard deviations are roughly the same magnitude, so credible intervals include zero for every coefficient, and the predicted probabilities cluster near 0.50, meaning the models separate individuals only weakly. Predictive performance was not evaluated because no discrimination or calibration metrics, including the area under the receiver operating characteristic curve or the Brier score, were calculated.

The remaining two limitations concern the scope of the comparison and of the process itself. The ranking of the ordinary logistic, treatment and jump specifications rests on ten officers and on the magnitude of their transition probabilities rather than on any formal model-comparison criterion, such as an information criterion or a held-out predictive check. Finally, the time spent in each state is not modelled, and the transition matrix was not iterated, so the study reports one-step transition probabilities only and supports no claim about time to recovery, the number of steps to recovery, or the long-run behaviour of the process.

5.0 CONCLUSION AND RECOMMENDATIONS

Conclusion: This study links six observed ordinal symptoms to a latent two-state process through a logit link, allowing the state to be inferred from symptom profiles rather than assumed to be directly observed, as in conventional Markov formulations. It also represents treatment as a random jump of size (j) from the set $\{0, 1, 2, 3\}$, applied to a symptom score and floored at zero. This formulation expresses the recovery probability as a finite mixture rather than a fixed decrement and incorporates heterogeneous treatment responses into the model structure, reflecting the variation in treatment outcomes discussed by Thase (2011). In addition, the model estimates the normal-to-depression and depression-to-normal coefficient vectors jointly using Metropolis-within-Gibbs sampling to obtain posterior estimates under the mixture formulation. The zero floor also ensures that officers whose symptom scores are already zero cannot experience further reductions in those scores. The simulation results demonstrate these properties of the specified model, including the reduction in modelled transition probabilities following jumps and the absence of further reductions for symptom-free officers.

The contribution of the study therefore lies in model specification and estimation. Because the model was demonstrated using simulated data, and no discrimination or calibration measures were calculated, its predictive performance remains unassessed. The results consequently do not establish the model's empirical validity for Kenyan police officers or demonstrate the clinical effectiveness of the simulated treatment mechanism.

Recommendations: The study recommends that depressed officers be treated early, so that recovery is reached and sustained. For further work, the model should be fitted to real clinical data from police officers. The time spent in each state should be modelled. Formal comparison criteria and wider posterior intervals should be reported before the model is used in practice.

6.0 REFERENCES

1. Ali, A. S., Javeed, S., Faiz, Z., & Baleanu, D. (2024). Mathematical modelling, analysis and numerical simulation of social media addiction and depression. *PLOS ONE*, 19(3), Article e0293807. <https://doi.org/10.1371/journal.pone.0293807>
2. Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., Lombardo, C., & Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders*, 135(1–3), 10–19. <https://doi.org/10.1016/j.jad.2011.01.011>
3. Batterham, P. J., Glozier, N., & Christensen, H. (2012). Sleep disturbance, personality and the onset of depression and anxiety: Prospective cohort study. *Australian & New Zealand Journal of Psychiatry*, 46(11), 1089–1098. <https://doi.org/10.1177/0004867412457997>

4. Best, J., Nijhout, H. F., & Reed, M. (2010). Serotonin synthesis, release and reuptake in terminals: A mathematical model. *Theoretical Biology and Medical Modelling*, 7, Article 34. <https://doi.org/10.1186/1742-4682-7-34>
5. Brailsford, S. C., Harper, P. R., Patel, B., & Pitt, M. (2009). An analysis of the academic literature on simulation and modelling in health care. *Journal of Simulation*, 3(3), 130–140. <https://doi.org/10.1057/jos.2009.10>
6. Bzdok, D., & Meyer-Lindenberg, A. (2018). Machine learning for precision psychiatry: Opportunities and challenges. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, 3(3), 223–230. <https://doi.org/10.1016/j.bpsc.2017.11.007>
7. Cramer, A. O. J., van Borkulo, C. D., Giltay, E. J., van der Maas, H. L. J., Kendler, K. S., Scheffer, M., & Borsboom, D. (2016). Major depression as a complex dynamic system. *PLOS ONE*, 11(12), Article e0167490. <https://doi.org/10.1371/journal.pone.0167490>
8. Demic, S., & Cheng, S. (2014). Modeling the dynamics of disease states in depression. *PLOS ONE*, 9(10), Article e110358. <https://doi.org/10.1371/journal.pone.0110358>
9. Edinger, J. D., Smith, E. D., Buysse, D. J., Thase, M., Krystal, A. D., Wiskniewski, S., & Manber, R. (2023). Objective sleep duration and response to combined pharmacotherapy and cognitive behavioral insomnia therapy among patients with comorbid depression and insomnia: A report from the TRIAD study. *Journal of Clinical Sleep Medicine*, 19(6), 1111–1120. <https://doi.org/10.5664/jcsm.10514>
10. Gamerman, D., & Lopes, H. F. (2006). *Markov chain Monte Carlo: Stochastic simulation for Bayesian inference* (2nd ed.). Chapman & Hall/CRC.
11. GBD 2021 Diseases and Injuries Collaborators. (2024). Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: A systematic analysis for the Global Burden of Disease Study 2021. *The Lancet*, 403(10440), 2133–2161. [https://doi.org/10.1016/S0140-6736\(24\)00757-8](https://doi.org/10.1016/S0140-6736(24)00757-8)
12. Geoffroy, P. A., Hoertel, N., Etain, B., Bellivier, F., Delorme, R., Limosin, F., & Peyre, H. (2018). Insomnia and hypersomnia in major depressive episode: Prevalence, sociodemographic characteristics and psychiatric comorbidity in a population-based study. *Journal of Affective Disorders*, 226, 132–141. <https://doi.org/10.1016/j.jad.2017.09.032>
13. Grazian, C., & Robert, C. P. (2018). Jeffreys priors for mixture estimation: Properties and alternatives. *Computational Statistics & Data Analysis*, 121, 149–163. <https://doi.org/10.1016/j.csda.2017.12.005>
14. Guloksuz, S., Pries, L.-K., & van Os, J. (2017). Application of network methods for understanding mental disorders: Pitfalls and promise. *Psychological Medicine*, 47(16), 2743–2752. <https://doi.org/10.1017/S0033291717001350>
15. Haugen, P. T., McCrillis, A. M., Smid, G. E., & Nijdam, M. J. (2017). Mental health stigma and barriers to mental health care for first responders: A systematic review and meta-analysis. *Journal of Psychiatric Research*, 94, 218–229. <https://doi.org/10.1016/j.jpsychires.2017.08.001>
16. Ionescu, D. F., Niciu, M. J., Mathews, D. C., Richards, E. M., & Zarate, C. A., Jr. (2013). Neurobiology of anxious depression: A review. *Depression and Anxiety*, 30(4), 374–385. <https://doi.org/10.1002/da.22095>
17. Kamenov, K., Twomey, C., Cabello, M., Prina, A. M., & Ayuso-Mateos, J. L. (2017). The efficacy of psychotherapy, pharmacotherapy and their combination on functioning and quality of life in depression: A meta-analysis. *Psychological Medicine*, 47(3), 414–425. <https://doi.org/10.1017/S0033291716002774>

18. Kenya Adolescent Mental Health Group. (2024). Burden and risk factors of mental and substance use disorders among adolescents and young adults in Kenya: Results from the Global Burden of Disease Study 2019. *eClinicalMedicine*, 67, Article 102328. <https://doi.org/10.1016/j.eclinm.2023.102328>
19. Kenya National Commission on Human Rights. (2011). *Silenced minds: The systemic neglect of the mental health system in Kenya: A human rights audit of the mental health system in Kenya*. https://www.knchr.org/Portals/0/EcosocReports/THE_%20MENTAL_HEALTH_REPORT.pdf
20. Kuria, M. W., Ndetei, D. M., Obot, I. S., Khasakhala, L. I., Bagaka, B. M., Mbugua, M. N., & Kamau, J. (2012). The association between alcohol dependence and depression before and after treatment for alcohol dependence. *ISRN Psychiatry*, 2012, Article 482802. <https://doi.org/10.5402/2012/482802>
21. Leley, P. (2025, April 16). The silent burden that Kenya's police officers battle. *The Star*. <https://www.the-star.co.ke/opinion/star-blogs/2025-04-16-peter-leley-the-silent-burden-that-kenyas-police-officers-battle>
22. Lim, G. Y., Tam, W. W., Lu, Y., Ho, C. S., Zhang, M. W., & Ho, R. C. (2018). Prevalence of depression in the community from 30 countries between 1994 and 2014. *Scientific Reports*, 8, Article 2861. <https://doi.org/10.1038/s41598-018-21243-x>
23. Long, K. M., & Meadows, G. N. (2018). Simulation modelling in mental health: A systematic review. *Journal of Simulation*, 12(1), 76–85. <https://doi.org/10.1057/s41273-017-0062-0>
24. Marangu, E., Mansouri, F., Sands, N., Ndetei, D., Muriithi, P., Wynter, K., & Rawson, H. (2021). Assessing mental health literacy of primary health care workers in Kenya: A cross-sectional survey. *International Journal of Mental Health Systems*, 15(1), Article 55. <https://doi.org/10.1186/s13033-021-00481-z>
25. McLachlan, G., & Peel, D. (2000). *Finite mixture models*. Wiley.
26. Ménard, K. S., & Arter, M. L. (2013). Police officer alcohol use and trauma symptoms: Associations with critical incidents, coping, and social stressors. *International Journal of Stress Management*, 20(1), 37–56. <https://doi.org/10.1037/a0031434>
27. Ministry of Health. (2020). *Mental health and wellbeing towards happiness and national prosperity: A report by the Taskforce on Mental Health in Kenya*. <https://mental.health.go.ke/download/mental-health-and-wellbeing-towards-happiness-national-prosperity-a-report-by-the-taskforce-on-mental-health-in-kenya/>
28. Ministry of Health. (2024). *National clinical guidelines for management of common mental disorders*. <https://mental.health.go.ke/download/national-clinical-guidelines-for-management-of-common-mental-disorders/>
29. Morin, C. M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The insomnia severity index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, 34(5), 601–608. <https://doi.org/10.1093/sleep/34.5.601>
30. Musyimi, C. W., Mutiso, V. N., Musau, A. M., Matoke, L. K., & Ndetei, D. M. (2017). Prevalence and determinants of depression among patients under the care of traditional health practitioners in a Kenyan setting: Policy implications. *Transcultural Psychiatry*, 54(3), 285–303. <https://doi.org/10.1177/1363461517705590>
31. National Police Service Commission (Amendment) Act, No. 19 of 2025 (Kenya). <https://new.kenyalaw.org/akn/ke/act/2025/19/eng@2025-11-04/source>
32. Ndetei, D. M., Khasakhala, L. I., Kuria, M. W., Mutiso, V. N., Ongecha-Owuor, F. A., & Kokonya, D. A. (2009). The prevalence of mental disorders in adults in different level general medical facilities in

- Kenya: A cross-sectional study. *Annals of General Psychiatry*, 8, Article 1. <https://doi.org/10.1186/1744-859X-8-1>
33. Nivetha, S., Karthik, A., Tandon, A., & Ghosh, M. (2025). Mathematical modeling and optimal control of depression dynamics influenced by saboteurs. *Scientific Reports*, 15(1), Article 6773. <https://doi.org/10.1038/s41598-025-90357-w>
 34. Nyongesa, M. K., Mwangi, P., Wanjala, S. W., Mutua, A. M., Newton, C. R. J. C., & Abubakar, A. (2019). Prevalence and correlates of depressive symptoms among adults living with HIV in rural Kilifi, Kenya. *BMC Psychiatry*, 19(1), Article 333. <https://doi.org/10.1186/s12888-019-2339-5>
 35. Oehme, K., Donnelly, E. A., & Martin, A. (2012). Alcohol abuse, PTSD, and officer-committed domestic violence. *Policing: A Journal of Policy and Practice*, 6(4), 418–430. <https://doi.org/10.1093/police/pas023>
 36. Oskooyee, K. S., Rahmani, A. M., & Riahi Kashani, M. M. (2011). Predicting the severity of major depression disorder with the Markov chain model. In *Proceedings of the 2011 International Conference on Bioscience, Biochemistry and Bioinformatics* (Vol. 5, pp. 30–34). IACSIT Press.
 37. Sobin, C., & Sackeim, H. A. (1997). Psychomotor symptoms of depression. *American Journal of Psychiatry*, 154(1), 4–17. <https://doi.org/10.1176/ajp.154.1.4>
 38. Syed, S., Ashwick, R., Schlosser, M., Jones, R., Rowe, S., & Billings, J. (2020). Global prevalence and risk factors for mental health problems in police personnel: A systematic review and meta-analysis. *Occupational and Environmental Medicine*, 77(11), 737–747. <https://doi.org/10.1136/oemed-2020-106498>
 39. Thase, M. E. (2011). Treatment-resistant depression: Prevalence, risk factors, and treatment strategies. *The Journal of Clinical Psychiatry*, 72(5), Article e18. <https://doi.org/10.4088/JCP.8133tx4c>
 40. Tilghman-Osborne, C., Cole, D. A., & Felton, J. W. (2010). Definition and measurement of guilt: Implications for clinical research and practice. *Clinical Psychology Review*, 30(5), 536–546. <https://doi.org/10.1016/j.cpr.2010.03.007>
 41. Violanti, J. M., Owens, S. L., McCanlies, E., Fekedulegn, D., & Andrew, M. E. (2019). Law enforcement suicide: A review. *Policing: An International Journal*, 42(2), 141–164. <https://doi.org/10.1108/PIJPSM-05-2017-0061>
 42. Violanti, J. M., & Steege, A. (2021). Law enforcement worker suicide: An updated national assessment. *Policing: An International Journal*, 44(1), 18–31. <https://doi.org/10.1108/PIJPSM-09-2019-0157>
 43. World Health Organization. (2023, March 31). *Depressive disorder (depression)*. <https://www.who.int/news-room/fact-sheets/detail/depression>
 44. Wüthrich, F., Nabb, C. B., Mittal, V. A., Shankman, S. A., & Walther, S. (2022). Actigraphically measured psychomotor slowing in depression: Systematic review and meta-analysis. *Psychological Medicine*, 52(7), 1208–1221. <https://doi.org/10.1017/S0033291722000903>