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Short report

Ethnic disparities in time to bipolar disorder diagnosis: retrospective cohort study

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Summary

Lithium is a first-line maintenance treatment for bipolar disorder yet delays in diagnosis and lithium initiation remain common. This retrospective cohort study examined whether time to bipolar disorder diagnosis and lithium initiation differ by ethnicity, gender or age among 88 patients at South London and Maudsley National Health Service Foundation Trust. Delay outcomes were non-normally distributed and log-transformed before analysis. No demographic characteristic was significantly associated with time to lithium initiation. Ethnicity was significantly associated with diagnostic delay, with individuals from minority ethnic groups experiencing longer delays than White individuals (median 556 *v.* 50.5 days; IQR [89.8, 1342] *v.* [6.25, 222]), which persisted after adjustment for age, gender, substance use and illness severity, and remained robust to outlier removal. Although social deprivation was not accounted for, these findings suggest possible systematic diagnostic inequities in bipolar disorder, calling for culturally informed diagnostic practices and further investigation in larger, multi-site samples.

Keywords

Bipolar type I or II disorders; lithium; ethnicity; diagnostic delay; treatment.

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Lithium is considered a first-line maintenance treatment for bipolar disorder, associated with substantial reductions in symptomatology, relapse, hospitalisation and suicide risk.[1–3] Despite its established efficacy, delays in lithium initiation are common, with a recent analysis of the South London and Maudsley (SLaM) data used in the present study identifying a delay of 1.8 years between first assessment and lithium initiation.[4] Diagnostic inaccuracy, such as misdiagnosing young women with borderline personality disorder rather than bipolar disorder or misinterpreting differential presentations of affective symptoms between ethnic groups,[5] is thought to contribute to bipolar disorder diagnosis delays, with estimates ranging from 6 to 10 years.[6,7] This study investigated whether time to bipolar disorder diagnosis and lithium initiation differ by gender, ethnicity or age.

Method

This sub-analysis of Perez-Rodriguez and colleagues[4] used equivalent methods and sample (see original paper). The study was approved by the Clinical Records Interactive Search (CRIS) Oversight Committee in November 2020. CRIS has ethical approval from the South Central – Oxford C Research Ethics Committee (REC) as a database for secondary research. The REC reference is: 23/SC/0257. Briefly, this is a retrospective observational cohort study utilising routinely collected data within SLaM. Delay was defined as time from first SLaM assessment to recorded bipolar disorder or lithium initiation.

Delay distributions were assessed using Shapiro–Wilk tests. As outcomes were non-normally distributed, Mann–Whitney *U* tests, Kruskal–Wallis tests and Spearman's correlations examined associations with gender, ethnicity and age, respectively. Ethnicity was subsequently collapsed into White versus non-White categories. Univariate linear regressions were performed using log-transformed delay outcomes, followed by adjusted models incorporating age, gender, in-patient initiation and substance use. Analyses were repeated after outlier removal, and *post hoc* power was assessed. All analyses were conducted in R (version 4.6.1 for macOS, Posit Software, PBC, Boston, USA, https://posit.co/downloads) with $\alpha = 0.05$.

Results

Time to diagnosis was the only outcome significantly associated with ethnicity (Kruskal–Wallis: $\chi^2 = 12.70$, $p = 0.026$, effect size = 0.094). Given small subgroup sizes (White $n = 38$, Black $n = 25$, Asian $n = 9$, Mixed $n = 5$), ethnicity was collapsed into a binary variable (White *v.* non-White). Although the authors are aware that research should avoid broad ethnic categories that minimise within-group heterogeneity, this collapsing was required to facilitate analyses.[8] Non-White patients were diagnosed significantly later (median 556 *v.* 50.5 days, IQR (89.8, 1342) *v.* (6.25, 222); $\beta = 1.81$, s.e. = 0.51, $t = 3.60$, $p < 0.001$).

In the fully adjusted model (covariates: age, gender, illicit substance use, in-patient/out-patient prescribing), ethnicity remained the sole significant predictor ($\beta = 2.04$, s.e. = 0.54, $t = 3.80$, $p < 0.001$). The model explained 16.7% of variance overall ($R^2 = 0.167$, adjusted $R^2 = 0.117$, $F(5, 82) = 3.30$, $p = 0.009$). A *post hoc* power calculation confirmed 90% power to detect the observed effect ($f^2 = 0.20$) at $\alpha = 0.05$.

Outlier removal ($n = 12$, identified via Cook's distance, leverage and standardised residuals) strengthened the ethnicity effect and the model explained substantially more variance ($\beta = 2.58$, $p < 0.001$; $R^2 = 0.367$). In-patient versus out-patient setting of lithium prescribing also emerged as a significant predictor in the sensitivity model ($\beta = 1.06$, $p = 0.022$), with out-patients experiencing longer diagnostic delays.

Moreover, patients with an initial schizophrenia-spectrum disorder (SSD) diagnosis were disproportionately non-White: 9 of 11 (81.8%) were non-White compared with 41 of 77 (53.2%) without an initial SSD diagnosis. Given the small subgroup, findings are presented descriptively and should be interpreted cautiously.

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Table 1 Model statistics from unadjusted and adjusted regressions examining demographic and clinical characteristics associated with time to diagnosis

Variable	*N* (%)/mean (s.d.)	Univariate beta coefficient log, days (s.e.)	*p* value	Multivariable beta coefficient log, days (s.e.)	*p*-value
Age (years), mean (s.d.)	34.8 (12)	−0.01 (0.02)	0.68	0.02 (0.023)	0.49
Gender (ref: male), *n* (%)	27 (30.7)	0.56 (0.58)	0.34	0.90 (0.59)	0.13
Illicit substance use (ref: no substance use), *n* (%)	17 (19.3)	−0.29 (0.68)	0.67	0.04 (0.68)	0.95
Lithium initiation setting (ref: in-patient), *n* (%)	60 (68.2)	0.40 (0.58)	0.49	1.06	0.022
Ethnicity (ref: White), *n* (%)	38 (43.2)	1.81 (0.51)	<0.001	2.04 (0.54)	<0.001

Discussion

This study found that non-White individuals experienced significantly longer delays to bipolar disorder diagnosis than White individuals, an effect that remained after covariate adjustment and outlier removal (Table 1). The modest R^2 values are consistent with the multifactorial nature of diagnostic delay and noise in routinely collected clinical data, thus residual confounding from unmeasured factors cannot be excluded.

General barriers to bipolar disorder diagnosis, including misdiagnosis, healthcare access challenges, stigma and mistrust of services,[9] may be amplified in non-White individuals. Misinterpretation of affective symptoms within psychotic rather than bipolar frameworks may contribute, alongside documented overdiagnosis of schizophrenia and greater diagnostic instability in Black populations.[5,10,11] Supporting this possibility, non-White individuals disproportionately received SSD diagnoses before their bipolar disorder diagnosis, suggesting that diagnostic delay may represent a downstream consequence of misdiagnosis.

The absence of an ethnicity effect on time to lithium prescription may indicate that treatment decisions become more equitable once bipolar disorder is diagnosed. However, previous studies report less lithium and more antipsychotic prescribing among people of African ancestry.[12–14] This null finding may reflect sampling bias, as all participants received lithium. Therefore, future research should expand the sample to individuals who are not administered lithium at all.

Age and gender did not significantly influence delay outcomes. Females nevertheless had descriptively longer delays to bipolar disorder diagnosis ($M = 742.38$ days) compared with males ($M = 514.93$ days). The predominantly female sample may be limiting the detection of gender effects. Other limitations, including limited sample size, insufficient documentation and broad ethnic classifications, have been reported previously and may obscure within-group heterogeneity.

These findings align with the evidence of under-recognition of bipolar disorder in non-White populations.[5,10] Clinically, this emphasises the need for culturally informed diagnostic practices and reduced diagnostic overshadowing by psychotic disorder frameworks in non-White populations. Future studies should examine these findings in larger, representative samples while accounting for socioeconomic status.

Lived experience commentary (4th author)

I write as someone from a White British background who has lived with bipolar disorder type 1 throughout my adult life (first manic episode aged 18), with a lithium treatment delay of 14 years (first prescribed lithium aged 32). In the 5 years prior to commencing lithium, I had four manic episodes, but in the 5 years following lithium initiation, just one. My episodes have been devastating in their impact on my ability to function, maintain relationships and sustain employment. Happily, I am now functioning very well.

I was offered lithium aged 19 but declined due to reluctance to have blood tests. I feel my consultant psychiatrist should have made a stronger case for the drug. A later psychiatrist stated lithium was not a good fit because it would make me feel 'sluggish' (it doesn't). I was only started on lithium after a lengthy hospital admission, where I was given the options of either lithium or electroconvulsive therapy. I responded quickly to lithium and have only had one more minor manic episode since.

Lithium has been absolutely transformative for me and I only wish I had been prescribed it earlier. I'm sure it has also saved the National Health Service (NHS) significant costs. I commend this research into inequalities in lithium initiation, and while my story does not speak to the ethnic inequalities found in this paper, they are no doubt worthy of further exploration.

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Data availability

The data that support the findings of this study are available on request from the corresponding author (M.R.-B.).

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No artificial intelligence tools have been used in the research or writing process for this manuscript.

Author contributions

M.R.-B. contributed to conceptualisation, methodology, formal analysis, project administration, visualisation, writing – original draft preparation and writing – review and editing. V.P.R. contributed to conceptualisation, data curation, methodology, investigation, project administration, resources and writing – review and editing. E.B. contributed to conceptualisation, methodology, formal analysis and writing – review and editing. S.M.A. contributed to methodology and writing – original draft preparation. R.P. contributed to methodology, resources, validation, visualisation and writing – review and editing. R.S. contributed to methodology, validation and writing – review and editing. K.B. contributed to supervision and writing – review

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Declaration of interest

E.B. currently holds an unpaid secondment with Akrivia Health, but has no financial relationship with the company and the duties are distinct from the included work. R.P. has participated in scientific advisory boards for Boehringer Ingelheim and Teva, has received grant funding from Janssen and has received consulting fees from Akrivia Health, Boehringer Ingelheim, Bristol Myers Squibb, Clinilabs, Columbia Data Analytics, Eisai, Holmusk, Otsuka, Social Finance, Supernus and Teva. R.S. has received honoraria for educational lectures from Janssen. K.B. provides consultancy services to Bristol Myers Squibb. O.D.H. has received investigator-initiated research funding from and/or participated in advisory/speaker meetings organised by Alkermes, Angellini, Autifony, Biogen, Boehringer Ingelheim, Bristol Myers Squib, Eli Lilly, Elysium, Heptares, Global Medical Education, Invicro, Janssen, Karuna, Lundbeck, Merck, Minerva, Neurocrine, Ontrack/ Pangea, Otsuka, Ovid, Pasithea, QCell, Sunovion, Recordati, Roche, Rovi, Teva and Viatris/Mylan. He was previously a part-time employee of Lundbeck A/S. O.D.H. has a patent for the use of dopaminergic imaging. The views expressed are those of the author(s) and not necessarily those of H Lundbeck A/S, the NHS/NIHR or the Department of Health. A.H.Y. is employed by Imperial College London and is an honorary consultant at South London and Maudsley NHS Foundation Trust (NHS UK). A.H.Y. is the editor of *Journal of Psychopharmacology* and deputy editor of *BJPsych Open*. A.H.Y. gives paid lectures and serves on advisory boards for the following companies with drugs used in affective and related disorders: Flow Neuroscience, Novartis, Roche, Janssen, Takeda, Noema pharma, Compass, AstraZeneca, Boehringer Ingelheim, Eli Lilly, LivaNova, Lundbeck, Sunovion, Servier, Allegan, Bionomics, Sumitomo Dainippon Pharma, Sage, Neurocentrx and Otsuka. A.H.Y. is principal investigator in the Restore-Life Vagus Nerve Stimulation registry study funded by LivaNova. A.H.Y. is principal investigator on ESKETINTRD3004: 'An Open-label, Long-term, Safety and Efficacy Study of Intranasal Esketamine in Treatment-resistant Depression.' A.H.Y. is principal investigator on 'The Effects of Psilocybin on Cognitive Function in Healthy Participants'. A.H.Y. is principal investigator on 'The Safety and Efficacy of Psilocybin in Participants with Treatment-Resistant Depression (P-TRD)'. A.H.Y. is principal investigator on 'A Double-Blind, Randomized, Parallel-Group Study with Quetiapine Extended Release as Comparator to Evaluate the Efficacy and Safety of Seltorexant 20 mg as Adjunctive Therapy to Antidepressants in Adult and Elderly Patients with Major Depressive Disorder with Insomnia Symptoms Who Have Responded Inadequately to Antidepressant Therapy.' (Janssen). A.H.Y. is principal investigator on 'An Open-label, Long-term, Safety and Efficacy Study of Aticaprant as Adjunctive Therapy in Adult and Elderly Participants with Major Depressive Disorder (MDD).' (Janssen). A.H.Y. is principal investigator on 'A Randomized, Double-blind, Multicentre, Parallel-group, Placebo-controlled Study to Evaluate the Efficacy, Safety, and Tolerability of Aticaprant 10 mg as Adjunctive Therapy in Adult Participants with Major Depressive Disorder (MDD) with Moderate-to-severe Anhedonia and Inadequate Response to Current Antidepressant Therapy'. A.H.Y. is principal investigator on 'A Study of Disease Characteristics and Real-life Standard of Care Effectiveness in Patients with Major Depressive Disorder (MDD) With Anhedonia and Inadequate Response to Current Antidepressant Therapy Including an SSRI or SNR.' (Janssen). A.H.Y. is the UK chief investigator for Compass; COMP006 & COMP007 studies (Compass). A.H.Y. is the UK chief investigator for Novartis MDD study MIJ821A12201. A.H.Y. has no shareholdings in pharmaceutical companies. 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