
Bayesian Disease Progression Models that Capture Health Disparities

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Abstract

1 Disease progression models, in which a patient’s latent severity is modeled as
2 progressing over time and producing observed symptoms, have developed great po-
3 tential to help with disease detection, prediction, and drug development. However,
4 a significant limitation of existing models is that they do not typically account for
5 healthcare disparities that can bias the observed data. We draw attention to three
6 key disparities: certain patient populations may (1) start receiving care only when
7 their disease is more severe, (2) experience faster disease progression even while
8 receiving care, or (3) receive care less frequently conditional on disease severity.
9 To address this, we develop an interpretable Bayesian disease progression model
10 that captures these three disparities. We show theoretically and empirically that
11 our model correctly estimates disparities and severity from observed data, and that
12 failing to account for these disparities produces biased estimates of severity.

13 1 Introduction

14 Using observed data to model the progression of a latent variable over time is useful for making
15 predictions in many settings. Models of infrastructure deterioration use physical observations and
16 inspection results to model a system’s overall health changing over time [1]; models of human
17 aging use a person’s observed physical and biological characteristics to learn the progression of their
18 underlying “biological age” [2]; and disease progression models, the setting we focus on in this paper,
19 use observed symptoms to learn a patient’s evolving latent disease severity [3]. Disease progression
20 models provide insight on both individual-level disease trajectories and general representations
21 of disease dynamics. Accurately modeling disease progression offers great promise in enabling
22 healthcare providers to better personalize care and predict a patient’s disease trajectory, detect diseases
23 at earlier stages, and study interventions such as drug development [4, 5].

24 In order for the benefits of these models to apply to all patients equitably, it is crucial that they
25 make accurate predictions for all populations of patients. However, disease progression models have
26 typically failed to account for systemic disparities in the healthcare process. Disparities have been
27 shown to exist along many demographic features including socioeconomic status [6, 7], proximity to
28 care [8, 9], and race [10] — intuitively, we expect that models not accounting for these disparities will
29 make predictions that are consistently inaccurate for some patient groups. In this paper, we define
30 three main axes along which we observe and analyze disparities:

- 31 1. Certain patient groups may start receiving care only when their disease is more severe
32 (leaving more of their disease trajectory unobserved).
- 33 2. Certain patient groups may experience faster disease progression even while receiving care
34 (indicating consistent differences in the efficacy or quality of treatment).

35 3. Certain patient groups may receive care less frequently conditional on disease severity
36 (decreasing the frequency with which they are observed in the data).

37 As such, our key contributions are: (1) we propose an interpretable Bayesian model that learns disease
38 progression while accounting for disparities along all three of these axes, (2) we show theoretically
39 and empirically that failing to account for any of these disparities will lead to biased severity estimates,
40 and (3) we outline the beginning of a heart failure case study. We anticipate that the results from this
41 case study, which we are working on in close collaboration with the New York-Presbyterian hospital
42 system, will have two main applications: descriptions of healthcare disparities across demographic
43 groups can help to target future interventions, and validating the model in a real healthcare setting
44 will demonstrate that it is possible to make predictions without bias from these disparities.

45 2 Related Work

46 **Disease progression modeling.** Disease progression models have been developed for many chronic
47 diseases, including Parkinson’s disease [3], Alzheimer’s disease [11], diabetes [12], and cancer [13].
48 A key feature of the progression models we consider is that a latent severity Z_t progresses over time
49 and gives rise to the observed symptoms X_t . Models in this family include variants of hidden markov
50 models (HMM) [14, 15, 16, 17, 18] and recurrent neural networks (RNN) [19, 20, 21, 22, 23, 24, 25].

51 **Healthcare disparities.** Disparities have been documented in many parts of the healthcare process.
52 Factors such as distance from hospitals [8, 9], distrust of the healthcare system [26], or lack of
53 insurance [27] can result in underutilization of health services. Biases in the judgements of healthcare
54 providers can lead to minority groups receiving later screening [28], fewer referrals [29], or generally
55 worse care [30]. And issues such as limited health literacy or trust in healthcare can create disparities
56 in follow-through for appointments or effectiveness of at-home care [31, 32].

57 These disparities have been shown to emerge along the three axes that we identify: (1) how severe a
58 patient’s disease gets before they start to receive care [33, 34, 35]; (2) how quickly their latent severity
59 Z_t progresses even while receiving care [36, 37]; and (3) how likely they are to visit a clinician
60 at a given disease severity level [38]. Despite thorough literature showing the existence of these
61 disparities and their impact on healthcare, disease progression models have not (to the best of our
62 knowledge) accounted for disparities when making predictions.

63 3 Model

64 We build on a standard setup for disease progression modeling, in which each patient i has an
65 underlying latent disease severity $Z_t^{(i)}$ that progresses over time and gives rise to a set of observed
66 features $X_t^{(i)}$ [39, 40]. For notational convenience, we will omit the (i) superscript from here on.

67 We characterize a patient’s severity $Z_t \in \mathbb{R}$ at timestep t by their *initial severity* Z_0 at their first
68 observation (which we denote as $t = 0$) and their *rate of progression* R after that point:

$$Z_t = Z_0 + R \cdot t$$

69 While we expect our approach to extend naturally to non-linear models of progression, estimating
70 the slope of a potentially non-linear progression still provides valuable insight on a patient’s general
71 disease trajectory relative to others. The assumption of linear progression over time to capture
72 long-term disease trajectory is a common approach in existing models [11, 2].

73 Whether a patient actually visits a healthcare provider at time t is captured by an observed binary
74 indicator $D_t \in \{0, 1\}$. If a patient does visit at time t , we will observe some recorded set of disease-
75 relevant features $X_t \in \mathbb{R}^d$ (e.g., lab results, imaging, and symptoms). At any given timestep, a
76 clinician will not necessarily observe or record all features — we model the features that *are* observed
77 as a noisy function of latent severity Z_t :

$$X_t = f(Z_t) + \epsilon_t$$

78 where diagonal covariance matrix $\sigma_\epsilon \in \mathbb{R}^{d \times d}$ parameterizes feature-specific noise $\epsilon_t \sim N(0, \sigma_\epsilon)$
79 (accounting for both measurement error and variation in how the patient’s physical state can fluctuate
80 day-to-day). We specifically instantiate f as a linear function $f(Z_t) = F \cdot Z_t + F_{int}$, where

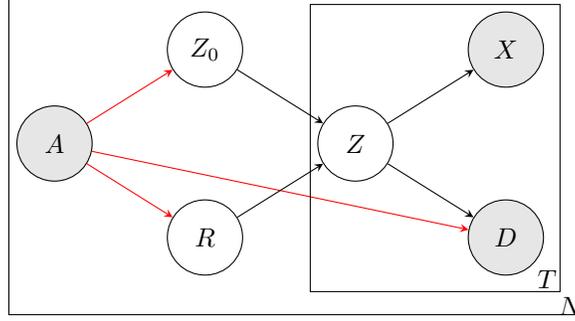


Figure 1: Plate diagram of generative model, capturing N patients over T timesteps. Shaded nodes indicate observed features, and red arrows indicate dependencies capturing health disparities.

81 $F_{int} \in \mathbb{R}^d$ is a feature-specific intercept and $F \in \mathbb{R}^d$ has its first element constrained to be positive
 82 for identifiability; we leave extending this to non-linear functions for future work.

83 **Capturing disparities.** We next specify a demographic feature vector A for each patient. A can
 84 capture multiple social determinants of health (each element of A can encode any continuous or
 85 categorical feature), but for simplicity in exposition, we assume A encodes a single categorical label
 86 (e.g., a patient’s race group). By modeling dependence between A and other aspects of the model,
 87 depicted in Figure 1, we can capture health disparities along three interpretable axes; as we discuss in
 88 §2, the existence of these disparities has been well-documented in past studies:

- 89 1. **Underserved patients may start receiving care only when their disease is more severe.**
 90 We capture this by learning group-specific distributions of Z_0 , a patient’s disease severity at
 91 first visit. We pin Z_0 for one group ($A = a_0$) to be drawn from a unit normal distribution (as
 92 is standard because it fixes the scale of Z_t). For other groups $A = a$, $Z_0 \sim N\left(\mu_{Z_0}^{(a)}, \sigma_{Z_0}^{(a)}\right)$,
 93 where $\mu_{Z_0}^{(a)}$ and $\sigma_{Z_0}^{(a)}$ are learned group-specific parameters for group a .
- 94 2. **Underserved patients may experience faster disease progression even while receiving
 95 care.** This we capture by learning group-specific distributions of progression rate $R \sim$
 96 $N\left(\mu_R^{(a)}, \sigma_R^{(a)}\right)$, where $\mu_R^{(a)}$ and $\sigma_R^{(a)}$ are learned group-specific parameters for group a .
- 97 3. **Underserved patients may receive care frequently conditional on disease severity.** This
 98 we capture by modeling patient visits as generated by an inhomogeneous Poisson process
 99 parameterized by a non-negative, time-varying rate parameter λ_t that depends on both Z_t
 100 and A for all groups a : $\log(\lambda_t) = \beta_0 + (\beta_Z \cdot Z_t) + \beta_A^{(a)}$, where β_Z and β_0 are learned
 101 parameters for the entire population and $\beta_A^{(a)}$ is a learned group-specific parameter for group
 102 a . We pin $\beta_A^{(a_0)}$ at 0 as a reference for all other groups.

103 Overall, our model parameters (on which we place weakly informative priors) are F , F_{int} , σ_ϵ , $\{\mu_{Z_0}^{(a)}\}$,
 104 $\{\sigma_{Z_0}^{(a)}\}$, $\{\mu_R^{(a)}\}$, $\{\sigma_R^{(a)}\}$, β_0 , β_Z , and $\{\beta_A^{(a)}\}$ for all demographic groups a . We learn these values
 105 from our observed data X_t , D_t , and A . Figure 1 summarizes the data generating process.

106 4 Theoretical analysis

107 4.1 Identifiability

108 As we show in §A.1, our model is identifiable, meaning different sets of parameters yield different
 109 observed data distributions [41, 42]:

110 **Theorem 4.1.** *All parameters of the model are identified by $P(X_t, D_t | A)$.*

111 We confirm our theoretical identifiability results experimentally in §5, showing that the model does
 112 indeed recover the true parameters in synthetic data.

113 **4.2 Bias in models that do not account for disparities**

114 Next we show that disease progression models will produce biased estimates of severity if they fail
115 to account for any of the three disparity types we capture. We use the strict Monotone Likelihood
116 Ratio Property (MLRP) to characterize the existence of disparities between two populations [43].
117 Our results apply to any setting in which data is generated according to the relationships depicted in
118 Figure 1 and disparities exist, not relying on the parametric assumptions of our implemented model.

119 First, we prove that any model failing to account for disparity 1 will produce biased severity estimates:

120 **Theorem 4.2.** *A model that does not take into account demographic disparities in initial disease*
121 *severity Z_0 will underestimate the disease severity of groups with higher values of initial severity and*
122 *overestimate that of groups with lower values of initial severity.*

123 That is (for the underestimation case), if $P(Z_0 = z_0 | A = a)$ strictly MLRPs $P(Z_0 = z_0)$ for some
124 group a , then $\mathbb{E}[Z_t | X_t = x_t] < \mathbb{E}[Z_t | X_t = x_t, A = a]$. A full proof is provided in §B.1. We then
125 prove that failing to account for disparity 2 or disparity 3 will also lead to biased estimates of severity
126 (full proofs in §B.2 and §B.3, respectively):

127 **Theorem 4.3.** *A model that does not take into account demographic disparities in rate of progression*
128 *R will underestimate the disease severity of groups with higher progression rates and overestimate*
129 *that of groups with lower progression rates.*

130 **Theorem 4.4.** *A model that does not take into account demographic disparities in visit frequency λ_t*
131 *will underestimate the disease severity of groups with lower visit frequency and overestimate that of*
132 *groups with higher visit frequency.*

133 **5 Synthetic experiments**

134 We implement our model in Stan, a Bayesian inference package [44], to validate our theoretical
135 results in simulations with synthetic data.

136 **5.1 Identifiability**

137 We first verify Theorem 4.1 in simulations, showing our model can accurately recover the true
138 data-generating parameters for synthetic data. Across 50 runs, we find high correlation between
139 the true parameters and the posterior mean estimates (mean Pearson’s r 0.98 across all parameters;
140 median 0.98), and good calibration (mean linear regression slope 0.97; median 0.98). We provide
141 scatterplots of all parameter recovery in Appendix C.

142 **5.2 Bias in models that do not account for disparities**

143 We now verify in simulation that failing to account for disparities can lead to biased severity estimates.
144 We generate simulated data for two groups, $A = 0$ and $A = 1$, where group 1 is underserved with
145 respect to each of the three disparities we capture (i.e., $\mu_{Z_0}^{(1)} > \mu_{Z_0}^{(0)}$, $\mu_R^{(1)} > \mu_R^{(0)}$, and $\beta_A^{(0)} > \beta_A^{(1)}$).
146 We then fit our main model, which accounts for all disparities, alongside three models that each fail
147 to account for one of the disparities, on the same set of data to compare their recovery of individual
148 patient severity values. As seen in Figure 2, the models that do not account for disparities all
149 underestimate severity for the underserved group 1 and overestimates severity for the other group —
150 these simulations empirically support Theorems 4.2, 4.3, and 4.4. While our main model achieves
151 average error (mean inferred estimate minus mean true value for a single run) -0.004 and -0.02
152 for groups 0 and 1 respectively, the other models have error 1.03, 0.01, and 0.42 for group 0 (all
153 overestimated) and error -0.78 , -0.24 , and -0.88 for group 1 (all underestimated).

154 **References**

155 [1] Samer Madanat, Rabi Mishalani, and Wan Hashim Wan Ibrahim. Estimation of Infrastructure
156 Transition Probabilities from Condition Rating Data. *Journal of Infrastructure Systems*, 1(2):120–
157 125, June 1995.

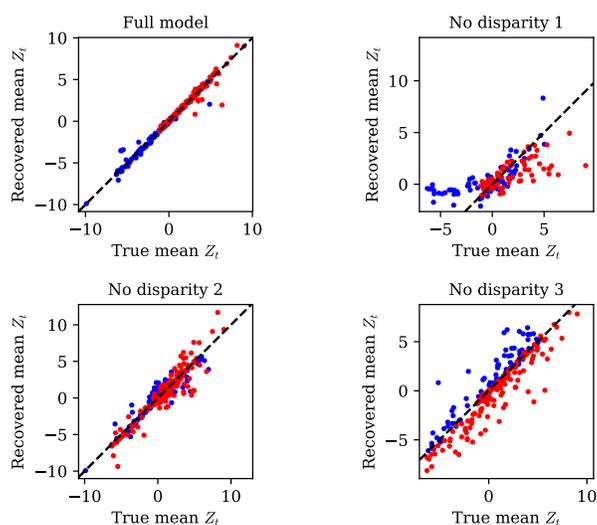


Figure 2: Failing to account for disparities produces biased estimates of severity Z_t . We compare severity estimates from four models: our full model (upper left), which accounts for all disparities, and three models that each fail to account for one axis of disparity. Each model is fit on the same simulated data, in which members of group 1 (red) tend to be underserved. While our main model produces accurate and well-calibrated severity estimates (estimates lie near dotted line indicating equality), the other models overestimate severity for group 0 and underestimate it for group 1.

- 158 [2] Emma Pierson, Pang Wei Koh, Tatsunori Hashimoto, Daphne Koller, Jure Leskovec, Nicholas
 159 Eriksson, and Percy Liang. Inferring Multidimensional Rates of Aging from Cross-Sectional
 160 Data, March 2019.
- 161 [3] Teun M. Post, Jan I. Freijer, Joost DeJongh, and Meindert Danhof. Disease System Analy-
 162 sis: Basic Disease Progression Models in Degenerative Disease. *Pharmaceutical Research*,
 163 22(7):1038–1049, July 2005.
- 164 [4] D R Mould, N G Denman, and S Duffull. Using Disease Progression Models as a Tool to Detect
 165 Drug Effect. *Clinical Pharmacology & Therapeutics*, 82(1):81–86, July 2007.
- 166 [5] K Romero, K Ito, Ja Rogers, D Polhamus, R Qiu, D Stephenson, R Mohs, R Lalonde, V Sinha,
 167 Y Wang, D Brown, M Isaac, S Vamvakas, R Hemmings, L Pani, Lj Bain, B Corrigan, and
 168 Alzheimer’s Disease Neuroimaging Initiative* for the Coalition Against Major Diseases**. The
 169 future is now: Model-based clinical trial design for Alzheimer’s disease. *Clinical Pharmacology
 170 & Therapeutics*, 97(3):210–214, March 2015.
- 171 [6] Kathryn E. Weaver, Julia H. Rowland, Keith M. Bellizzi, and Noreen M. Aziz. Forgoing medical
 172 care because of cost: Assessing disparities in healthcare access among cancer survivors living
 173 in the United States. *Cancer*, 116(14):3493–3504, July 2010.
- 174 [7] Sarah Miller and Laura R. Wherry. Health and Access to Care during the First 2 Years of the
 175 ACA Medicaid Expansions. *New England Journal of Medicine*, 376(10):947–956, March 2017.
- 176 [8] Leighton Chan, L. Gary Hart, and David C. Goodman. Geographic Access to Health Care for
 177 Rural Medicare Beneficiaries. *The Journal of Rural Health*, 22(2):140–146, April 2006.
- 178 [9] Megan Reilly. Health Disparities and Access to Healthcare in Rural vs. Urban Areas. *Theory in
 179 Action*, 14(2):6–27, April 2021.
- 180 [10] Ruqaiyah Yearby. Racial Disparities in Health Status and Access to Healthcare: The Contin-
 181 uation of Inequality in the United States Due to Structural Racism. *The American Journal of
 182 Economics and Sociology*, 77(3-4):1113–1152, May 2018.

- 183 [11] N H Holford and K E Peace. Methodologic aspects of a population pharmacodynamic model
184 for cognitive effects in Alzheimer patients treated with tacrine. *Proceedings of the National*
185 *Academy of Sciences*, 89(23):11466–11470, December 1992.
- 186 [12] Sajida Perveen, Muhammad Shahbaz, Muhammad Sajjad Ansari, Karim Keshavjee, and Aziz
187 Guergachi. A Hybrid Approach for Modeling Type 2 Diabetes Mellitus Progression. *Frontiers*
188 *in Genetics*, 10:1076, January 2020.
- 189 [13] A. Gupta and Z. Bar-Joseph. Extracting Dynamics from Static Cancer Expression Data.
190 *IEEE/ACM Transactions on Computational Biology and Bioinformatics*, 5(2):172–182, April
191 2008.
- 192 [14] Xiang Wang, David Sontag, and Fei Wang. Unsupervised learning of disease progression
193 models. In *Proceedings of the 20th ACM SIGKDD international conference on Knowledge*
194 *discovery and data mining*, KDD 2014, pages 85–94, New York New York USA, August 2014.
195 ACM.
- 196 [15] Yu-Ying Liu, Shuang Li, Fuxin Li, Le Song, and James M. Rehg. Efficient Learning of
197 Continuous-Time Hidden Markov Models for Disease Progression. *Advances in Neural Infor-*
198 *mation Processing Systems*, 28:3599–3607, 2015.
- 199 [16] Ahmed M Alaa and Scott Hu. Learning from Clinical Judgments: Semi-Markov-Modulated
200 Marked Hawkes Processes for Risk Prognosis. 2017.
- 201 [17] R. Sukkar, E. Katz, Yanwei Zhang, D. Raunig, and B. T. Wyman. Disease progression mod-
202 eling using Hidden Markov Models. In *2012 Annual International Conference of the IEEE*
203 *Engineering in Medicine and Biology Society*, pages 2845–2848, San Diego, CA, August 2012.
204 IEEE.
- 205 [18] Christopher H. Jackson, Linda D. Sharples, Simon G. Thompson, Stephen W. Duffy, and
206 Elisabeth Couto. Multistate Markov models for disease progression with classification error.
207 *Journal of the Royal Statistical Society: Series D (The Statistician)*, 52(2):193–209, July 2003.
- 208 [19] Edward Choi, Mohammad Taha Bahadori, Andy Schuetz, Walter F. Stewart, and Jimeng Sun.
209 Doctor AI: Predicting Clinical Events via Recurrent Neural Networks. *JMLR workshop and*
210 *conference proceedings*, 56:301–318, August 2016.
- 211 [20] Zachary C. Lipton, David C. Kale, Charles Elkan, and Randall Wetzell. Learning to Diagnose
212 with LSTM Recurrent Neural Networks, March 2017.
- 213 [21] Bryan Lim and Mihaela van der Schaar. Disease-Atlas: Navigating Disease Trajectories with
214 Deep Learning, July 2018.
- 215 [22] Edward Choi, Mohammad Taha Bahadori, Joshua A. Kulas, Andy Schuetz, Walter F. Stewart,
216 and Jimeng Sun. RETAIN: An Interpretable Predictive Model for Healthcare using Reverse
217 Time Attention Mechanism. 2016.
- 218 [23] Fenglong Ma, Radha Chitta, Jing Zhou, Quanzeng You, Tong Sun, and Jing Gao. Dipole: Diag-
219 nosis Prediction in Healthcare via Attention-based Bidirectional Recurrent Neural Networks.
220 2017.
- 221 [24] Bum Chul Kwon, Min-Je Choi, Joanne Taery Kim, Edward Choi, Young Bin Kim, Soonwook
222 Kwon, Jimeng Sun, and Jaegul Choo. RetainVis: Visual Analytics with Interpretable and
223 Interactive Recurrent Neural Networks on Electronic Medical Records. *IEEE Transactions on*
224 *Visualization and Computer Graphics*, 25(1):299–309, January 2019.
- 225 [25] Ahmed M. Alaa and Mihaela van der Schaar. Attentive State-Space Modeling of Disease
226 Progression. In H. Wallach, H. Larochelle, A. Beygelzimer, F. d’ Alché-Buc, E. Fox, and
227 R. Garnett, editors, *Advances in Neural Information Processing Systems*, volume 32. Curran
228 Associates, Inc., 2019.
- 229 [26] Thomas A. LaVeist, Lydia A. Isaac, and Karen Patricia Williams. Mistrust of Health Care
230 Organizations Is Associated with Underutilization of Health Services. *Health Services Research*,
231 44(6):2093–2105, December 2009.

- 232 [27] Arjun K. Venkatesh, Shih-Chuan Chou, Shu-Xia Li, Jennie Choi, Joseph S. Ross, Gail
233 D’Onofrio, Harlan M. Krumholz, and Kumar Dharmarajan. Association Between Insurance
234 Status and Access to Hospital Care in Emergency Department Disposition. *JAMA Internal*
235 *Medicine*, 179(5):686, May 2019.
- 236 [28] Richard J. Lee, Ravi A. Madan, Jayoung Kim, Edwin M. Posadas, and Evan Y. Yu. Disparities
237 in Cancer Care and the Asian American Population. *The Oncologist*, 26(6):453–460, June 2021.
- 238 [29] Bruce E. Landon, Jukka-Pekka Onnela, Laurie Meneades, A. James O’Malley, and Nancy L.
239 Keating. Assessment of Racial Disparities in Primary Care Physician Specialty Referrals. *JAMA*
240 *Network Open*, 4(1):e2029238, January 2021.
- 241 [30] Gráinne Schäfer, Kenneth M. Prkachin, Kimberley A. Kaseweter, and Amanda C. De C Williams.
242 Health care providers’ judgments in chronic pain: the influence of gender and trustworthiness.
243 *Pain*, 157(8):1618–1625, August 2016.
- 244 [31] Milton S. Davis. Physiologic, Psychological and Demographic Factors in Patient Compliance
245 with Doctors’ Orders. *Medical Care*, 6(2):115–122, 1968.
- 246 [32] Dwayne T. Brandon, Lydia A. Isaac, and Thomas A. LaVeist. The legacy of Tuskegee and
247 trust in medical care: is Tuskegee responsible for race differences in mistrust of medical care?
248 *Journal of the National Medical Association*, 97(7):951–956, July 2005.
- 249 [33] Irene Y. Chen, Rahul G. Krishnan, and David Sontag. Clustering Interval-Censored Time-Series
250 for Disease Phenotyping, December 2021.
- 251 [34] Javaid Iqbal, Ophira Ginsburg, Paula A. Rochon, Ping Sun, and Steven A. Narod. Differences
252 in Breast Cancer Stage at Diagnosis and Cancer-Specific Survival by Race and Ethnicity in the
253 United States. *JAMA*, 313(2):165, January 2015.
- 254 [35] Xiao Hu, John W Melson, Stacey S Pan, Yana V Salei, and Yu Cao. Screening, Diagnosis, and
255 Initial Care of Asian and White Patients With Lung Cancer. *The Oncologist*, 29(4):332–341,
256 April 2024.
- 257 [36] Clarissa Jonas Diamantidis, Lindsay Zepel, Virginia Wang, Valerie A. Smith, Sarah Hud-
258 son Scholle, Loida Tamayo, and Matthew L. Maciejewski. Disparities in Chronic Kidney
259 Disease Progression by Medicare Advantage Enrollees. *American Journal of Nephrology*,
260 52(12):949–957, 2021.
- 261 [37] Jonathan Suarez, Jordana B. Cohen, Vishnu Potluri, Wei Yang, David E. Kaplan, Marina Serper,
262 Siddharth P. Shah, and Peter Philip Reese. Racial Disparities in Nephrology Consultation and
263 Disease Progression among Veterans with CKD: An Observational Cohort Study. *Journal of*
264 *the American Society of Nephrology*, 29(10):2563–2573, October 2018.
- 265 [38] Sarah Nouri, Courtney R. Lyles, Elizabeth B. Sherwin, Magdalene Kuznia, Anna D. Rubinsky,
266 Kathryn E. Kemper, Oanh K. Nguyen, Urmimala Sarkar, Dean Schillinger, and Elaine C.
267 Khoong. Visit and Between-Visit Interaction Frequency Before and After COVID-19 Telehealth
268 Implementation. *JAMA Network Open*, 6(9):e2333944, September 2023.
- 269 [39] Petr Klemra and Stanislav Doubal. A new approach to the concept and computation of
270 biological age. *Mechanisms of Ageing and Development*, 127(3):240–248, March 2006.
- 271 [40] M. E. Levine. Modeling the Rate of Senescence: Can Estimated Biological Age Predict
272 Mortality More Accurately Than Chronological Age? *The Journals of Gerontology Series A:*
273 *Biological Sciences and Medical Sciences*, 68(6):667–674, June 2013.
- 274 [41] R. Bellman and K.J. Åström. On structural identifiability. *Mathematical Biosciences*, 7(3-
275 4):329–339, April 1970.
- 276 [42] C. Cobelli and J. J. DiStefano. Parameter and structural identifiability concepts and ambiguities:
277 a critical review and analysis. *American Journal of Physiology-Regulatory, Integrative and*
278 *Comparative Physiology*, 239(1):R7–R24, July 1980.

279 [43] Ben Klemens. When Do Ordered Prior Distributions Induce Ordered Posterior Distributions?
 280 *SSRN Electronic Journal*, 2007.

281 [44] Bob Carpenter, Andrew Gelman, Matthew D. Hoffman, Daniel Lee, Ben Goodrich, Michael
 282 Betancourt, Marcus Brubaker, Jiqiang Guo, Peter Li, and Allen Riddell. *Stan*: A Probabilistic
 283 Programming Language. *Journal of Statistical Software*, 76(1), 2017.

284 A Identifiability Proofs

285 A.1 Proof of Theorem 4.1

286 *Proof.* We want to show that each set of parameter assignments leads to a different distribution over
 287 the observed data. To do this, we divide our argument into four lemmas:

288 **Lemma A.1.** *Parameters $F, F_{int}, \sigma_\epsilon$ are identified by $P(X_t | A = a_0)$.*

289 *Proof.* would probably cut the restatement of model definitions here and throughout
 290 the proofs. First we restate relevant details of the generative model for group a_0 :

$$\begin{aligned} 291 & Z_0 \sim N(0, 1) \\ 292 & Z_t = Z_0 + R \cdot t \\ & X_t = F \cdot Z_t + F_{int} + \epsilon_t, \text{ where } \epsilon_t \sim N(0, \sigma_\epsilon) \end{aligned} \quad (1)$$

293 We first note that at $t = 0$ we have $Z_t = Z_0$ and thus $Z_t \sim N(0, 1)$. Then equation
 294 (1) captures a factor analysis model we should cite the source of the expression with
 295 factor loading matrix F and diagonal covariance matrix σ_ϵ . So at $t = 0$, we have
 296 for group a_0 that

$$X_0 \sim N(F_{int}, FF^T + \sigma_\epsilon).$$

297 my guess is that it should be σ^2 not σ . In general, let's use a different variable
 298 besides sigma to refer to the covariance matrix - capital sigma I think could be
 299 fine. I think the following sentence should come first and be less conversational.
 300 We want to show that each set of assignments to $F, F_{int}, \sigma_\epsilon$ leads to a different
 301 distribution of X_0 for group a_0 , i.e. we can uniquely determine the values of these
 302 three parameters by observing $P(X_0 | A = a_0)$. To do this, we show that the
 303 mapping from the parameter values to observed distribution $P(X_0 | A = a_0)$ is an
 304 injective function — we assume there are two sets of parameters $\{F, F_{int}, \sigma_\epsilon\}$ and
 305 $\{F', F_{int}', \sigma_\epsilon'\}$ that lead to the same observed distribution of X_0 and show that
 306 the parameter values must be equal.

307 Assuming the two sets of parameters map to distributions of X_0 with the same
 308 mean, it must hold that $F_{int} = F_{int}'$. Thus, parameter F_{int} is identified by data
 309 distribution $P(X_0 | A = a_0)$.

310 Further, the covariance matrix of X_0 induced by each set of parameters must be
 311 the same: $F(F)^T + \sigma_\epsilon = F'(F')^T + \sigma_\epsilon'$. Element-wise equality of the covariance
 312 matrix gives us the following, where subscripts i refer to the i -th element of each
 313 parameter vector:

$$314 F_i F_j = F'_i F'_j \quad \forall i, j, i \neq j \quad (2)$$

$$(F_i)^2 + \sigma_{\epsilon_i} = (F'_i)^2 + \sigma_{\epsilon_i}' \quad (3)$$

315 Combining equality constraint (2) for multiple pairs of indices, we have that for all
 316 assignments of distinct indices i, j, k :

$$(F_i F_j = F'_i F'_j) \wedge (F_i F_k = F'_i F'_k) \implies \frac{F'_j}{F_j} = \frac{F'_k}{F_k}$$

317

$$(F_j F_k = F'_j F'_k) \wedge \left(\frac{F'_j}{F_j} = \frac{F'_k}{F_k} \right) \implies (F_j = \alpha F'_j) \wedge (F_k = \alpha F'_k),$$

318
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where $\alpha \in \{-1, +1\}$ not exactly sure how second line follows, is there some way to better-explain the argument?. Since we have fixed $F_0 > 0$ for all factor loading matrices F , we have:

$$F_0 = \alpha F'_0 \implies \alpha = 1 \implies F_i = F'_i \quad \forall i \in [0, d), \quad (4)$$

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meaning we have identified F .

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Lastly, using equations (3) and (4) we get $F_i = F'_i \implies \sigma_{\epsilon_i} = \sigma_{\epsilon'_i}$. We have now shown that if two parameter sets induce the same distribution of X at time $t = 0$, they must have the same exact value assignments. Therefore $F, F_{int}, \sigma_\epsilon$ are identified by $P(X_t | A = a_0)$. \square

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Lemma A.2. *Parameters $\mu_{Z_0}^{(a)}, \sigma_{Z_0}^{(a)}, \mu_R^{(a)}, \sigma_R^{(a)}$ are identified by $P(X_t | A = a)$ for all groups a I might write this using the full set of parameters, including F etc (those covered in lemma 1). And I'm not sure I would say for all groups a ; I might just say $p(X_t|A)$.*

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Proof. Since we have shown that $F, F_{int}, \sigma_\epsilon$ are identified by themselves based on the observed data, we take their values as given in this argument let's say this more formally. Ideally I think we should just keep saying throughout "we show that if two parameter sets X and X' yield the same observed data distribution $p(\text{blar})$, they must be identical. By Lemma 1, we know that if subset X and subset X' yield same distribution subset Blar , they must be identical. [Rest of proof].. For each group a , we model the following:

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$$\begin{aligned} Z_0 &\sim N\left(\mu_{Z_0}^{(a)}, \sigma_{Z_0}^{(a)}\right) \\ R &\sim N\left(\mu_R^{(a)}, \sigma_R^{(a)}\right) \\ Z_t = Z_0 + R \cdot t &\implies Z_t \sim N\left(\mu_R^{(a)} \cdot t + \mu_{Z_0}^{(a)}, \sigma_R^{(a)} \cdot t^2 + \sigma_{Z_0}^{(a)}\right) \\ X_t = F \cdot Z_t + F_{int} + \epsilon_t, &\text{ where } \epsilon_t \sim N(0, \sigma_\epsilon) \end{aligned} \quad (5)$$

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lowercase sigma standardly refers to standard deviation, not covariance, so I think some of the entries above should be σ^2 probably also we should find a notation for the intercept term besides F_{int} , which is a bit clunky. one other notational thing - might be easier to use tilde for the alternate parameters not prime - e.g. $\tilde{\mu}^{(a)}$ takes up less space because the tilde just goes over the letter For convenience we will omit the (a) superscript for the rest of the proof. We see that equation (5) captures a factor analysis model with factor loading matrix F and diagonal covariance matrix σ_ϵ . So we have that

$$X_t \sim N(F_{int} + F(\mu_R \cdot t + \mu_{Z_0}), F(\sigma_R \cdot t^2 + \sigma_{Z_0})F^T + \sigma_\epsilon).$$

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We want to show that every set of assignments to $\mu_{Z_0}, \sigma_{Z_0}, \mu_R, \sigma_R$ leads to a different distribution of X_t at any time t , i.e. we can uniquely determine the values of these four parameters by observing $P(X_t | A = a)$. To do this, we show that the mapping from the parameter values to observed distribution $P(X_t | A = a)$ is an injective function — we assume there are two sets of parameters $\{\mu_{Z_0}, \sigma_{Z_0}, \mu_R, \sigma_R\}$ and $\{\mu_{Z_0}', \sigma_{Z_0}', \mu_R', \sigma_R'\}$ that lead to the same observed distribution of X_t at all t .

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We first consider $t = 0$, where $X_0 \sim N(F_{int} + F\mu_{Z_0}, F(\sigma_{Z_0})F^T + \sigma_\epsilon)$. For the two parameter sets to map to distributions of X_0 with the same mean, it must hold that

$$F_{int} + F\mu_{Z_0} = F_{int} + F\mu_{Z_0}' \implies \mu_{Z_0} = \mu_{Z_0}',$$

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and for the two parameter sets to map to distributions with the same covariance matrix, it must hold that

$$F(\sigma_{Z_0})F^T + \sigma_\epsilon = F(\sigma_{Z_0}')F^T + \sigma_\epsilon \implies \sigma_{Z_0} = \sigma_{Z_0}'.$$

359 So we have identified μ_{Z_0} and σ_{Z_0} . We next consider any time $t \neq 0$, where
 360 $X_t \sim N(F_{int} + F(\mu_R \cdot t + \mu_{Z_0}), F(\sigma_R \cdot t^2 + \sigma_{Z_0})F^T + \sigma_\epsilon)$. For the two
 361 parameter sets to map to distributions of X_t with the same mean, it must hold that

$$F_{int} + F(\mu_R \cdot t + \mu_{Z_0}) = F_{int} + F(\mu_{R'} \cdot t + \mu_{Z_0}') \implies \mu_R = \mu_{R'}$$

362 this looks right, but I might say explicitly it follows because we've already shown
 363 that μ_{Z_0} must equal μ_{Z_0}' , and similarly below.

364 and for the two parameter sets to map to distributions with the same covariance
 365 matrix, it must hold that

$$F(\sigma_R \cdot t^2 + \sigma_{Z_0})F^T + \sigma_\epsilon = F(\sigma_{R'} \cdot t^2 + \sigma_{Z_0}')F^T + \sigma_\epsilon \implies \sigma_R = \sigma_{R'}$$

366 So we have identified μ_R and σ_R . Thus we have shown that for any group a ,
 367 group-specific values of $\mu_{Z_0}, \sigma_{Z_0}, \mu_R, \sigma_R$ are identified by $P(X_t | A = a)$.

368 □

369 **Lemma A.3.** *Parameters β_0, β_Z are identified by $P(D_t | Z_t, A = a_0)$ this can't be quite the right*
 370 *theorem statement because we don't observe Z_t ; I think we want to say $p(D|A, t)$.*

371 *Proof.* Since we have shown that all group-specific distribution parameters
 372 $\mu_R^{(a)}, \sigma_R^{(a)}$ are identified by the observed data, we take their values as given in
 373 this argument. This means that we know the distributions of Z_0 (pinned) and R for
 374 group a_0 rewrite more formally as suggested above. In addition, we observe each
 375 event when a patient in group a_0 visits the hospital ($D_t = 1$), which means that the
 376 value λ_t can be recovered for all timepoints t . As described in §3, we model λ_t
 377 as a function of severity Z_t and demographic group. More specifically, we have
 378 $\log(\lambda_t) = \beta_0 + \beta_Z \cdot Z_t + \beta_A^{(a)}$. We define $\beta_A^{(a_0)}$ as 0 for reference, so for group
 379 a_0 we have $\log(\lambda_t) = \beta_0 + \beta_Z \cdot Z_t = \beta_0 + \beta_Z(Z_0 + R \cdot t)$.

380 We want to show that our observations of patient visits identify the parameters
 381 β_0 and β_Z . First, we find it is more straightforward to reason about too informal
 382 $\log(\lambda_t)$, which has a one-to-one correspondence with λ_t since λ_t is positive and
 383 $\log(\cdot)$ is a bijection over \mathbb{R}^+ . Further, instead of the value $\log(\lambda_t)$ itself, which
 384 is dependent on each individual patient's value of Z_0 and R , we reason about
 385 the expectation of $\log(\lambda_t)$ over the known group-level distributions of Z_0 and R .
 386 Each set of observations $\mathbb{E}_{Z_0, R}[\log(\lambda_t)] \forall t$ uniquely defines the visit distribution
 387 of the group a_0 over time, so by showing that different parameters β_0, β_Z lead to
 388 different values of $\mathbb{E}_{Z_0, R}[\log(\lambda_t)]$ we complete the proof that unique parameters
 389 β_0, β_Z lead to a unique distribution of visit times over group a_0 . I think this is
 390 true, but we need to make the argument more succinct + precise. I think you're
 391 basically trying to say that if two distributions have unique $E[\log(\lambda)]$, they
 392 must have unique $p(D|t)$. So if we can show that different parameter sets yield
 393 unique $E[\log(\lambda)]$ they must have unique $p(D|t)$. And then we just show
 394 that different parameter sets yield unique $E[\log(\lambda)]$. But we need to make
 395 the first part of the claim more precise and actually show it's true. I think one way
 396 to do this is to argue that distributions with unique $E[\log(\lambda)]$ have unique $E[\lambda]$,
 397 and then use the definition of $p(D)$ in terms of λ to argue that if you have
 398 unique $E[\lambda]$ you have unique $p(D)$?.

399 We want to show that every set of assignments β_0, β_Z leads to a unique observation
 400 of $\mathbb{E}_{Z_0, R}[\log(\lambda_t)] = \mathbb{E}_{Z_0, R}[\beta_0 + \beta_Z(Z_0 + R \cdot t)]$ across time t . To do this, we
 401 show that the mapping from parameter values to the expected value of $\log(\lambda_t)$ over
 402 group a_0 is an injective function — we assume there are two sets of parameters
 403 $\{\beta_0, \beta_Z\}$ and $\{\beta_0', \beta_Z'\}$ that generate the same observed values $\mathbb{E}_{Z_0, R}[\log(\lambda_t)]$
 404 at all timesteps t . We want to show it must be the case that $\beta_0 = \beta_0'$ and $\beta_Z = \beta_Z'$.

405 We first consider some timestep t' such that we observe data at $t = t'$ and $t = t' + 1$.
 406 At timestep t' , we observe:

$$\mathbb{E}_{Z_0, R}[\beta_0 + \beta_Z \cdot Z_0 + \beta_Z \cdot R \cdot t'] = \mathbb{E}_{Z_0, R}[\beta_0' + \beta_Z' \cdot Z_0 + \beta_Z' \cdot R \cdot t']. \quad (6)$$

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At timestep $t' + 1$, we observe:

$$\mathbb{E}_{Z_0, R}[\beta_0 + \beta_Z \cdot Z_0 + \beta_Z \cdot R \cdot (t' + 1)] = \mathbb{E}_{Z_0, R}[\beta_0' + \beta_Z' \cdot Z_0 + \beta_Z' \cdot R \cdot (t' + 1)]. \quad (7)$$

408

Using linearity of expectation to combine results from (6) and (7), we have that

$$\mathbb{E}_{Z_0, R}[\beta_Z \cdot R] = \mathbb{E}_{Z_0, R}[\beta_Z' \cdot R] \implies \beta_Z \cdot \mathbb{E}_{Z_0, R}[R] = \beta_Z' \cdot \mathbb{E}_{Z_0, R}[R] \implies \beta_Z = \beta_Z'.$$

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hm, this doesn't follow if $\mathbb{E}[R]$ is 0?

410

So we have identified β_Z . We also note that at $t = 0$:

$$\begin{aligned} \mathbb{E}_{Z_0, R}[\beta_0 + \beta_Z \cdot Z_0] &= \mathbb{E}_{Z_0, R}[\beta_0' + \beta_Z' \cdot Z_0] \\ \implies \beta_0 + \beta_Z \cdot \mathbb{E}_{Z_0, R}[Z_0] &= \beta_0' + \beta_Z' \cdot \mathbb{E}_{Z_0, R}[Z_0] \\ \implies \beta_0 &= \beta_0' \end{aligned}$$

411

Thus we have shown that β_0, β_Z are identified by $P(D_t \mid Z_t, A = a_0)$.

412

□

413

Lemma A.4. *Parameters $\beta_A^{(a)}$ is identified by $P(D_t \mid Z_t, A = a)$ for all other groups a .*

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Proof. I'm willing to believe that similar reasoning works here if it works on the last part, but let's clean up the last part first. Since we have shown that all group-specific distribution parameters $\mu_{Z_0}^{(a)}, \sigma_{Z_0}^{(a)}, \mu_R^{(a)}, \sigma_R^{(a)}$ are identified by the observed data, as well as group-agnostic parameters of the poisson process β_0, β_Z , we take their values as given in this argument. We use an approach very similar to that for Lemma A.3. We let \mathcal{D}_a denote the distributions of Z_0 and R for group a (parameterized by $\mu_{Z_0}^{(a)}, \sigma_{Z_0}^{(a)}, \mu_R^{(a)}, \sigma_R^{(a)}$). Then, since each set of observations $\mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[\log(\lambda_t)] \forall t$ uniquely characterizes the distribution of visits for group a over time, we can prove identifiability by showing that different values of $\beta_A^{(a)}$ will induce different values of $\mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[\log(\lambda_t)]$. Note that we omit the (a) superscript for the rest of the proof, since we only reason about one group at a time.

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We want to show that every value of β_A leads to a unique observation of $\mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[\log(\lambda_t)]$ across time t . To do this, we show that the mapping from β_A to the expected value of $\log(\lambda_t)$ over group a is an injective function — we assume there are two values β_A and β_A' that generate the same observed values $\mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[\log(\lambda_t)]$ at all timesteps t . We want to show it must be the case that $\beta_A = \beta_A'$.

431

As described in §3, $\log(\lambda_t) = \beta_0 + \beta_Z \cdot Z_t + \beta_A = \beta_0 + \beta_Z(Z_0 + R \cdot t) + \beta_A$.

432

Considering an arbitrary time t , we have by assumption that

$$\begin{aligned} \mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[\beta_0 + \beta_Z(Z_0 + R \cdot t) + \beta_A] &= \mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[\beta_0 + \beta_Z(Z_0 + R \cdot t) + \beta_A'] \\ \implies \beta_0 + \beta_Z \cdot \mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[Z_0 + R \cdot t] + \beta_A &= \beta_0 + \beta_Z \cdot \mathbb{E}_{Z_0, R \sim \mathcal{D}_a}[Z_0 + R \cdot t] + \beta_A' \\ \implies \beta_A &= \beta_A' \end{aligned}$$

433

Thus we have shown that β_A is identified by $P(D_t \mid Z_t, A = a)$ for all other groups a .

434

□

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436

By showing that each parameter of the model is uniquely recovered from the observed data, we have proved that our model is identifiable.

437

□

438

B Proofs of Bias

439

In this section, we assume that all PDFs and conditional PDFs have positive support over their entire domain. We also assume that all PDFs are differentiable.

440

442 **B.1 Proof of Theorem 4.2**

443 We make the following assumptions about the existence of disparities in our setting:

444 **Assumption B.1.** A patient’s severity over time can be estimated by $Z_t = f(R, t) + Z_0$, where f is
 445 monotonically increasing in progression rate R .

446 **Assumption B.2.** There exists some underserved group a that tends to start receiving care at later,
 447 more severe stages of their disease: $P(Z_0 = z_0 | A = a)$ strictly MLRPs $P(Z_0 = z_0)$ with respect
 448 to Z_0 , i.e. $\frac{P(Z_0=z_0|A=a)}{P(Z_0=z_0)}$ is a strictly increasing function of Z_0 .

449 **Assumption B.3.** On average, this underserved group progresses no slower than the overall popula-
 450 tion: $\mathbb{E}[R | X_t = x_t, A = a] \geq \mathbb{E}[R | X_t = x_t]$.

451 *Proof.* We want to show that $\mathbb{E}[Z_t | X_t = x_t, A = a] > \mathbb{E}[Z_t | X_t = x_t]$. We first show that
 452 $P(Z_0 = z_0 | X_t = x, A = a)$ strictly MLRPs $P(Z_0 = z_0 | X_t = x_t)$ with respect to Z_0 :

$$\begin{aligned} \frac{\partial}{\partial Z_0} \left(\frac{P(Z_0 = z_0 | X_t = x_t, A = a)}{P(Z_0 = z_0 | X_t = x_t)} \right) &= \frac{\partial}{\partial Z_0} \left(\frac{\frac{P(X_t=x_t|Z_0=z_0,A=a)P(Z_0=z_0|A=a)}{P(X_t=x_t|A=a)}}{\frac{P(X_t=x_t|Z_0=z_0)P(Z_0=z_0)}{P(X_t=x_t)}} \right) \\ &\quad \text{(Bayes Rule)} \\ &= \frac{\partial}{\partial Z_0} \left(\frac{\frac{P(Z_0=z_0|A=a)}{P(X_t=x_t|A=a)}}{\frac{P(Z_0=z_0)}{P(X_t=x_t)}} \right) \quad (X_t \perp A | Z_0, R) \\ &= \frac{P(X_t = x_t)}{P(X_t = x_t | A = a)} \cdot \frac{\partial}{\partial Z_0} \left(\frac{P(Z_0 = z_0 | A = a)}{P(Z_0 = z_0)} \right) \\ &> 0 \quad \text{(Assumption B.2)} \end{aligned}$$

453 Since MLRP implies FOSD [43], this also implies that $P(Z_0 = z_0 | X_t = x_t, A = a)$ strictly
 454 FOSDs $P(Z_0 = z_0 | X_t = x_t)$. It follows directly that $\mathbb{E}[Z_0 | X_t = x_t, A = a] > \mathbb{E}[Z_0 | X_t = x_t]$.

455 Furthermore,

$$\begin{aligned} \mathbb{E}[R | X_t = x_t, A = a] &\geq \mathbb{E}[R | X_t = x_t] \quad \text{(Assumption B.3)} \\ \implies \mathbb{E}[f(R, t) | X_t = x_t, A = a] &\geq \mathbb{E}[f(R, t) | X_t = x_t], \quad \forall t \geq 0 \quad \text{(Assumption B.1)} \\ \implies \mathbb{E}[f(R, t) | X_t = x_t, A = a] + \mathbb{E}[Z_0 | X_t = x_t, A = a] & \\ &> \mathbb{E}[f(R, t) | X_t = x_t] + \mathbb{E}[Z_0 | X_t = x_t], \quad \forall t \geq 0 \\ \implies \mathbb{E}[f(R, t) + Z_0 | X_t = x_t, A = a] &> \mathbb{E}[f(R, t) + Z_0 | X_t = x_t], \quad \forall t \geq 0 \\ \implies \mathbb{E}[Z_t | X_t = x_t, A = a] &> \mathbb{E}[Z_t | X_t = x_t] \end{aligned}$$

456 It is clear to see that this argument extends naturally to show that if a group is “overserved”, i.e. they
 457 tend to get care earlier than the rest of the population, that their severity will be overestimated: If
 458 there exists a group a' such that $P(Z_0 = z_0)$ strictly MLRPs $P(Z_0 = z_0 | A = a')$ with respect
 459 to Z_0 and $\mathbb{E}[R | X_t = x_t] \geq \mathbb{E}[R | X_t = x_t, A = a']$, then we will see that $\mathbb{E}[Z_t | X_t = x_t, A =$
 460 $a'] < \mathbb{E}[Z_t | X_t = x_t]$. Hence any model that does not take into account demographic disparities in
 461 initial disease severity levels at a patient’s first visit will lead to biased estimates of severity. \square

462 **B.2 Proof of Theorem 4.3**

463 We make the following assumptions about the existence of disparities in our setting:

464 **Assumption B.4.** A patient’s severity over time can be estimated by $Z_t = f(R, t) + Z_0$, where f is
 465 strictly monotonically increasing in progression rate R .

466 **Assumption B.5.** There exists some group a that tends to progress more quickly: $P(R = r | A = a)$
 467 strictly MLRPs $P(R = r)$ with respect to R , i.e. $\frac{P(R=r|A=a)}{P(R=r)}$ is a strictly increasing function of R .

468 **Assumption B.6.** On average, this underserved group is, on average, first observed no earlier than
 469 the overall population: $\mathbb{E}[Z_0 | X_t = x_t, A = a] \geq \mathbb{E}[Z_0 | X_t = x_t]$.

470 *Proof.* We want to show that $\mathbb{E}[Z_t | X_t = x_t, A = a] > \mathbb{E}[Z_t | X_t = x_t]$. We first show that
 471 $P(R = r | X_t = x_t, A = a)$ strictly MLRPs $P(R = r | X_t = x_t)$ with respect to R :

$$\begin{aligned}
 \frac{\partial}{\partial R} \left(\frac{P(R = r | X_t = x_t, A = a)}{P(R = r | X_t = x_t)} \right) &= \frac{\partial}{\partial R} \left(\frac{\frac{P(X_t = x_t | R = r, A = a) P(R = r | A = a)}{P(X_t = x_t | A = a)}}{\frac{P(X_t = x_t | R = r) P(Z_t = z_t)}{P(X_t = x_t)}} \right) \quad (\text{Bayes Rule}) \\
 &= \frac{\partial}{\partial R} \left(\frac{\frac{P(R = r | A = a)}{P(X_t = x_t | A = a)}}{\frac{P(R = r)}{P(X_t = x_t)}} \right) \quad (X \perp A | Z_0, R) \\
 &= \frac{P(X_t = x_t)}{P(X_t = x_t | A = a)} \cdot \frac{\partial}{\partial R} \left(\frac{P(R = r | A = a)}{P(R = r)} \right) \\
 &> 0 \quad (\text{Assumption B.5})
 \end{aligned}$$

472 Since MLRP implies FOSD [43], this also implies that $P(R = r | X_t = x_t, A = a)$ strictly FOSDs
 473 $P(R = r | X_t = x_t)$. It follows directly that:

$$\begin{aligned}
 &\mathbb{E}[R | X_t = x_t, A = a] > \mathbb{E}[R | X_t = x_t] \\
 &\implies \mathbb{E}[f(R, t) | X_t = x_t, A = a] > \mathbb{E}[f(R, t) | X_t = x_t], \quad \forall t > 0 \quad (\text{Assumption B.4}) \\
 &\implies \mathbb{E}[f(R, t) | X_t = x_t, A = a] + \mathbb{E}[Z_0 | X_t = x_t, A = a] \\
 &\quad > \mathbb{E}[f(R, t) | X_t = x_t] + \mathbb{E}[Z_0 | X_t = x_t], \quad \forall t > 0 \quad (\text{Assumption B.6}) \\
 &\implies \mathbb{E}[f(R, t) + Z_0 | X_t = x_t, A = a] > \mathbb{E}[f(R, t) + Z_0 | X_t = x_t], \quad \forall t > 0 \\
 &\implies \mathbb{E}[Z_t | X_t = x_t, A = a] > \mathbb{E}[Z_t | X_t = x_t]
 \end{aligned}$$

474 It is clear to see that this argument extends naturally to show that if a group is ‘‘overserved’’,
 475 i.e. they tend to progress more slowly than the rest of the population, that their severity will be
 476 overestimated: if there exists a group a' such that $P(R = r)$ strictly MLRPs $P(R = r | A = a')$
 477 with respect to R and $\mathbb{E}[Z_0 | X_t = x_t] \geq \mathbb{E}[Z_0 | X_t = x_t, A = a']$, then we will see that
 478 $\mathbb{E}[Z_t | X_t = x_t, A = a'] < \mathbb{E}[Z_t | X_t = x_t]$. Thus any model that does not take into account
 479 demographic disparities in patient progression rates will lead to biased estimates of severity. \square

480 B.3 Proof of Theorem 4.4

481 We make the following assumptions about the existence of disparities in our setting and patient visit
 482 rates:

483 **Assumption B.7.** A patient’s visit pattern can be estimated using an inhomogeneous poisson process
 484 characterized by visit rate Λ , such that $\log(\Lambda) = g(Z_t) + \beta_A^{(A)}$ for some function of severity $g(Z_t)$
 485 and group-specific adjustments $\beta_A^{(A)}$.

486 **Assumption B.8.** There exists some group a that tends to receive care less frequently than other
 487 groups, conditional on disease severity: $\beta_A^{(a)} < \beta_A^{(A)}$ for all $A \neq a$.

488 **Assumption B.9.** Visit rate increases with disease severity: $g(Z_t)$ is a strictly monotonically increas-
 489 ing function of severity.

490 *Proof.* We want to show that $\mathbb{E}[Z_t | \Lambda = \lambda, A = a] > \mathbb{E}[Z_t | \Lambda = \lambda]$. We do this by calculating
 491 each term separately.

492

493 We first consider $\mathbb{E}[Z_t | \Lambda = \lambda, A = a]$. The strictly monotone assumption in B.9 ensures g is
 494 invertible, and the fact that all visit rates Λ are characterized by $\log(\Lambda) = g(Z_t) + \beta_A^{(A)}$ ensures
 495 that this holds over the entire range of Λ values. This gives us:

$$\begin{aligned}
 \mathbb{E}[Z_t | \Lambda = \lambda, A = a] &= \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(A)} \right) \mid \Lambda = \lambda, A = a \right] \\
 &= g^{-1} \left(\log(\lambda) - \beta_A^{(a)} \right)
 \end{aligned}$$

496 We next consider the case where a model infers severity without taking into account disparities in
 497 visit rate conditional on severity. Estimating severity Z_t based solely on visit observations gives:

$$\begin{aligned}
 \mathbb{E}[Z_t \mid \Lambda = \lambda] &= P(A = a) \cdot \mathbb{E}[Z_t \mid \Lambda = \lambda, A = a] + P(A \neq a) \cdot \mathbb{E}[Z_t \mid \Lambda = \lambda, A \neq a] \\
 &= P(A = a) \cdot \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(A)} \right) \mid \Lambda = \lambda, A = a \right] \\
 &\quad + P(A \neq a) \cdot \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(A)} \right) \mid \Lambda = \lambda, A \neq a \right] \\
 &< P(A = a) \cdot \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(A)} \right) \mid \Lambda = \lambda, A = a \right] \\
 &\quad + P(A \neq a) \cdot \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(a)} \right) \mid \Lambda = \lambda, A = a \right] \quad (*) \\
 &= P(A = a) \cdot \left(g^{-1} \left(\log(\lambda) - \beta_A^{(a)} \right) \right) + P(A \neq a) \cdot \left(g^{-1} \left(\log(\lambda) - \beta_A^{(a)} \right) \right) \\
 &= g^{-1} \left(\log(\lambda) - \beta_A^{(a)} \right) \\
 &= \mathbb{E}[Z_t \mid \Lambda = \lambda, A = a]
 \end{aligned}$$

498 As justification for (*):

$$\begin{aligned}
 &\beta_A^{(a)} < \beta_A^{(A)}, \quad \forall A \neq a, \forall \Lambda \quad (\text{Assumption B.8}) \\
 &\implies \log(\Lambda) - \beta_A^{(a)} > \log(\Lambda) - \beta_A^{(A)}, \quad \forall A \neq a, \forall \Lambda \\
 &\implies g^{-1} \left(\log(\Lambda) - \beta_A^{(a)} \right) > g^{-1} \left(\log(\Lambda) - \beta_A^{(A)} \right), \quad \forall A \neq a, \forall \Lambda \\
 &\quad (\text{Assumption B.9} \implies g^{-1}(Z_t) \text{ strictly monotonically increasing}) \\
 &\implies \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(a)} \right) \mid \Lambda = \lambda, A = a \right] > \mathbb{E} \left[g^{-1} \left(\log(\Lambda) - \beta_A^{(A)} \right) \mid \Lambda = \lambda, A \neq a \right]
 \end{aligned}$$

499 It is clear to see that this argument extends naturally to show that if a group is “overserved”, i.e.
 500 they tend to visit the hospital more frequently conditional on severity, that their severity will be
 501 overestimated: if there exists a group a' such that $\beta_A^{(a')} > \beta_A^{(A)}$ for all $A \neq a'$, then we will see
 502 that $\mathbb{E}[Z_t \mid \Lambda = \lambda, A = a'] < \mathbb{E}[Z_t \mid \Lambda = \lambda]$. Thus any model that does not take into account
 503 demographic disparities in patient visit rates given their severity will lead to biased estimates of
 504 severity. \square

505 C Simulations

506 Figure 3 shows the results of 50 simulation runs, where we randomly instantiate the parameters of
 507 our model and then generate data to fit on. We visualize the recovery of each parameter by plotting
 508 true parameter values versus recovered posterior mean values, with one dot per run.

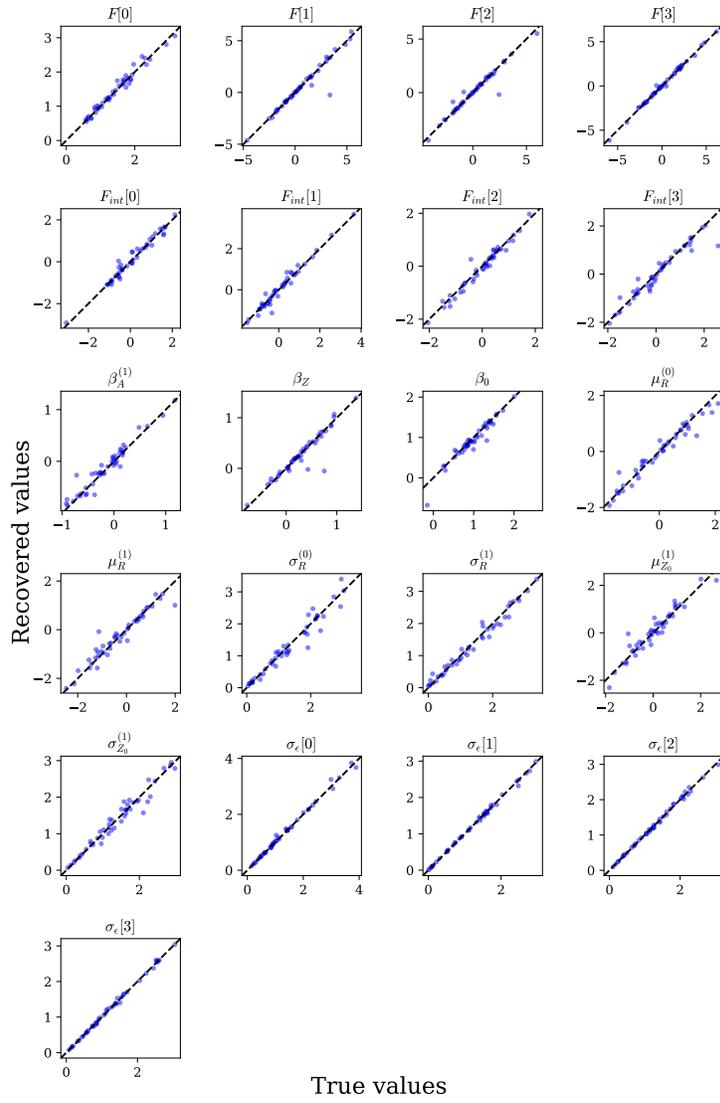


Figure 3: Parameter recovery on 50 runs of fitting our model to synthetic data.