Evaluating buffering effects for social determinants of health and type 2 diabetes in the All of Us Research Program

Vincent Lam
National Institutes of Health

Shivam Sharma
Georgia Institute of Technology

Christian S. Alvarez
National Institutes of Health

I. King Jordan
Georgia Institute of Technology

Leonardo Mariño-Ramírez

marino@nih.gov

National Institutes of Health

Research Article

Keywords: Health disparities, diabetes, interactions, All of Us, social determinants of health

Additional Declarations: No competing interests reported.

Abstract

Introduction: Social determinants of health (SDOH) play a strong role in influencing type 2 diabetes (T2D) risk. Certain protective SDOH have been demonstrated to mitigate or buffer against the positive associations between risk-associated SDOH and poor health outcomes, though such buffering effects have only been evaluated among a limited range of SDOH and outside the context of T2D. This study aims to assess potential buffering effects among SDOH associated with T2D case status.

Methods: The study population was constructed from *All of Us* Research Program data. Survey data were used to derive composite metrics measuring 10 different SDOH. Logistic regression models modeling T2D case status as a function of each SDOH were used to designate each as either a T2D risk-associated or protective factor. Interaction models were used to measure potential buffering effects between T2D risk-associated and protective factors.

Results: The main study population consists of 215,270 participants. All ten SDOH were significantly associated with T2D. Social support and neighborhood cohesion emerged as T2D protective factors, with the rest being classified as risk-associated factors for T2D. A single buffering effect was observed between social support and stress ($\beta = 0.43$, $p < 0.05$) (95% CI, -0.84 to -0.01). Reverse buffering effects were observed between neighborhood cohesion and discrimination, food insecurity, and individual-level socioeconomic deprivation (SED). Reverse buffering effects were also observed between social support and food insecurity, individual-level SED, and spirituality.

Conclusions: The buffering effect observed between social support and stress on T2D is consistent with previous findings. The reverse buffering effects observed in interactions between certain SDOH factors suggest that some risk-associated SDOH may weaken the potential benefits of certain protective SDOH. Interventions aimed at alleviating adverse socioeconomic conditions may have a compounding effect on reducing T2D risk by enabling communities to benefit from T2D protective factors.

BACKGROUND

In public health research, social determinants of health (SDOH) have been recognized as key factors in shaping health outcomes [1, 2]. SDOH are defined by the World Health Organization as "the non-medical factors that influence health outcomes". SDOH encompass factors related to the conditions and environments in which people live and work. They can include but are not limited to one's employment status, education, housing, healthcare access, and social inclusion [3]. SDOH strongly influence health, with such factors being estimated to contribute to 30–60% of all health outcomes [3–5]. For instance, individuals who are more highly educated and who have higher incomes are at reduced risk for conditions such as diabetes and generally have better health and longer lifespans [6–9]. On the other hand, individuals with adverse SDOH exposures such as discrimination and stress often demonstrate worse health outcomes [10–13].

Different SDOH often act in concert to influence health outcomes. Individuals exposed to harmful working conditions are more likely to live in poorer environments due to the typically low wages associated with such jobs [14]. Furthermore, synergistic effects on poor health outcomes have been observed between SDOH such as economic hardship and social capital [15]. In other instances, certain SDOH have been shown to mitigate or buffer the effects of adverse SDOH on health. One of the most notable examples can be found in the stress-buffering hypothesis, whereby social support weakens the positive association between stress and negative health outcomes [16, 17]. Similar effects have been observed in other SDOH, with resilience having previously been shown to buffer the positive association between discrimination and poor mental health outcomes [18, 19].

Type 2 diabetes (T2D) is a potentially compelling subject disease for the study of how different SDOH buffer the detrimental effects of other SDOH on health outcomes. T2D is a complex common disease that comprises roughly 90% of the 38.4 million cases of diabetes in the US [20, 21]. Its etiology is associated with a combination of genetic and environmental factors, the latter including various SDOH [22–24]. Stress is among the SDOH shown to increase risk of T2D, potentially through the activation of biological responses commonly implicated in the disease [25] [26]. As social support has been shown to buffer the effects of stress on health outcomes, there is a possibility that social support and other similar SDOH protective factors may reduce T2D risk through the buffering of stress and other T2D risk-associated factors [16, 17]. Buffering effects on risk-associated factors for T2D-related symptoms have been previously explored in literature, though such explorations have mainly been in the context of T2D distress (i.e. the emotional burden of living with T2D) [27–29]. There have been few studies on how SDOH specifically buffer the effects of adverse SDOH on T2D risk. As such, the goal of this study is to explore the potential buffering role of protective SDOH by examining their modifying effect on the associations between risk-related SDOH and T2D.

METHODS

Study Population

The study population was constructed using participant data available through the *All of Us* Researcher Workbench—a cloud computing platform through which registered users can access and interact with data from the *All of Us* Research Program. The *All of Us* participant body consists of adults aged 18 and older residing in the US or in a US territory. Volunteers can enroll in the program online via JoinAllofUs.org or in person through a participating healthcare provider. Individuals who are incarcerated or unable to provide consent are not eligible to enroll in the program.

Participant demographic, electronic health record (EHR), and survey data were drawn from the *All of Us* Registered Tier Dataset v7 (curated version R2022Q4R9). Enrollment began on May 31st, 2017, with data collected up to July 1st, 2022. Extracted demographic data consisted of participant date of birth and assigned sex at birth. International Classification of Diseases codes (ICD-9-CM and ICD-10-CM) were extracted from participant EHR data and mapped to phecode 250.2 to classify participants as T2D cases

and non-cases [30]. Patients with phenotypes corresponding to phecodes 249–250.99 were excluded from analyses following phecode exclusion criteria. The study sample was thus restricted to individuals who have EHR data available and who were classified as either T2D cases or non-cases. Furthermore, the study sample was restricted to individuals who were assigned either male or female at birth in order to facilitate the use of sex as a covariate in study models.

Measuring Social Determinants of Health

Social determinants of health were measured using data collected from the *All of Us* "Social Determinants of Health" survey, which includes questions from previously validated surveys and scales. Composite metrics for eight SDOH were constructed using responses to these surveys, with each metric representing a distinct social determinant, e.g.: (1) everyday discrimination, (2) discrimination in healthcare settings, (3) food insecurity, (4) neighborhood cohesion, (5) perceived neighborhood disorder, (6) social support, (7) spirituality, and (8) stress, with this terminology derived from the source material of each question set. Participants can provide responses ranging from "strongly disagree" to "strongly agree" or from "never" to "all the time" based on the question asked. These responses were coded as ordinal values starting from 0 and incrementing by 1, with greater values representing greater degrees of the SDOH.

Matrices consisting of participant ordinal responses to different questions were generated for each of the eight question sets. As they were comprised of ordinal values, these matrices were converted to eight polychoric correlation matrices using the polychoric function from version 2.4.1 of the psych package in R version 4.3.1. The *eigen* function was used to perform principal component analysis on each of the polychoric correlation matrices. This function generated eight new matrices composed of eigenvectors (principal components) arranged in decreasing order of their corresponding eigenvalues. The first principal component of each matrix was min-max normalized and used as a metric for each of the eight social determinants of health. These composite metrics had values ranging from 0 to 1, with greater values representing greater degrees of the social determinant of health they measure.

Socioeconomic deprivation (SED) was measured at both the individuals and zip code levels to capture the multifaceted nature of socioeconomic influences on health. An individual-level metric for SED was derived from participant responses to questions from the *All of Us* "The Basics" survey through methods described by Gupta et al [31]. Variables incorporated into this metric include education, employment, insurance status, home ownership status, and household income. Zip code-level SED was measured using a composite metric created by Brokamp et al [32]. Variables incorporated into this metric include the fraction of households below the poverty level, median household income, the fraction of adults older than 25 with at least a high school education, the fraction of the population without health insurance, the fraction of the population receiving public assistance, and the fraction of homes in the area that are vacant. This metric is available on the Researcher Workbench for most *All of Us* participants. Both measures of SED have values ranging from 0 to 1, with greater values representing more severe levels of SED.

Statistical Analyses

All statistical analyses were performed using R version 4.3.1. As participants varied in the types of SDOH information they provided, participant datasets used in statistical analyses represent subsets of the main study sample. To classify the ten SDOH as either factor associated with increased or decreased T2D, logistic regression models were used for each social determinant using the *glm* function from the *stats* package. In each model, T2D status (case = 1, non-case = 0) was modeled as a function of an individual SDOH. Participant age and sex were included as covariates to account for potential confounders due to the overrepresentation of older and female participants in the All of Us sample. SDOH with positive and significant association with T2D were classified as risk-associated SDOH, while those with negative and significant associations were classified as potential protective factors.

To examine potential buffering effects, interaction terms between protective and risk-associated SDOH factors for T2D were added to the logistic regression models for all possible combinations of SDOH. Buffering effects were defined as negative and significant interactions, indicating that a protective factor mitigates the effect of a risk-associated factor on T2D. In contrast, positive interactions between SDOH that are protective and SDOH that increase risk indicate that the effects of risk-associated factors mitigate the effects of protective factors on T2D; positive interactions of this kind are referred to as reverse buffering [33]. These tests were supplemented with sensitivity analyses, in which the same interaction analyses were run with composite metric values converted to z-scores. Variance inflation factor analysis was used to assess collinearity between each risk-associated and protective factor pair. A p-value threshold of less than 0.05 was used to determine statistical significance in all analyses.

RESULTS

Participant Characteristics

The sample for this study consisted of 215,278 individuals classified as T2D cases or non-cases based on EHR data and assigned sex at birth (Table 1 and Figure S1). Participants were predominantly female and middle-aged or older. The overall prevalence of T2D in the sample was 24.26%.

Table 1
Characteristics of *All of Us* participants with EHR data available who were assigned either male or female at birth.

Characteristic	Full Cohort (n = 215,278) (100%)	Female (n = 133,996) (62.24%)	Male (n = 81,282) (37.76%)
Mean age—years (SD)	56.44 (16.96)	59.05 (16.73)	54.86 (16.91)
T2D Cases (Prevalence %)	52,232 (24.26)	29,992 (22.38)	22,240 (27.36)

SDOH Analyses

Ten SDOH as measured by their respective composite metrics, were tested for the extent to which they were associated with T2D (Table 2). All ten SDOH were found to be significantly associated with T2D ($p < 0.05$). Of these SDOH, neighborhood cohesion ($\beta = -1.21$, $p < 0.05$) and social support ($\beta = -0.80$, $p < 0.05$) were associated with decreased odds of T2D and were therefore classified as T2D "protective factors". The remaining eight SDOH were associated with increased odds of T2D and were therefore classified as "risk-associated factor". These include area and individual-level SED, food insecurity, perceived neighborhood disorder, discrimination, stress, healthcare discrimination, and spirituality. The most potent risk-associated factor for T2D by effect size was area-level SED ($\beta = 2.46$, $p < 0.05$) whereas the least potent factor was spirituality ($\beta = 0.85$, $p = 1.05e-107$). No significant collinearity was observed between any pair of risk-associated and protective factor (Table S1). When not controlling for age or sex, these classifications remain consistent except for stress, which is not significantly (Table S2).

Table 2
Associations between social determinants of health and T2D risk. T2D ~ SDOH + age + sex

SDOH	Estimate	Std. Error	z-value	p-value
Risk-associated factors				
SED (Area-level)	2.46	8.21e-2	29.92	1.10e-196
SED (Individual-level)	2.00	2.22e-2	90.19	0.00e + 00
Food Insecurity	1.65	5.02e-2	32.90	2.15e-237
Perceived Neighborhood Disorder	1.45	6.67e-2	21.76	5.47e-105
Discrimination	1.33	6.98e-2	19.04	8.20e-81
Stress	1.22	6.34e-2	19.23	1.99e-82
Healthcare Discrimination	0.93	6.85e-2	13.64	2.23e-42
Spirituality	0.85	3.85e-2	22.05	1.05e-107
Protective Factors				
Neighborhood Cohesion	-1.21	5.92e-2	-20.39	1.86e-92
Social Support	-0.80	4.07e-2	-19.80	3.13e-87

Interaction effects between all combinations of T2D, including protective and risk-associated factors were assessed to identify potential buffering effects (Table 2 and Fig. 1). The first protective factor, neighborhood cohesion, demonstrated positive interactions with food insecurity ($\beta = 1.90$, $p < 0.05$), individual-level SED ($\beta = 1.22$, $p < 0.05$), and everyday discrimination ($\beta = 0.90$, $p < 0.05$). The second protective factor, social support, demonstrated reverse-buffering effects with risk-associated factors such as food insecurity ($\beta = 1.14$, $p < 0.05$), individual-level SED ($\beta = 0.87$, $p < 4.04e-6$), and spirituality ($\beta = 0.37$, $p < 0.05$). Additionally, social support demonstrated a buffering effect with stress ($\beta = -0.43$, $p <$

0.05). When not controlling for age or sex, the reverse buffering effect between neighborhood cohesion and discrimination is no longer observed and a new reverse buffering effect between social support and healthcare discrimination is observed (Table S3 and Figure S2). These significant interactions and their directionality remained when composite metric values were replaced with their corresponding z-scores (Table S4).

Table 3

Interactions between risk-associated factors and protective factors on T2D risk. T2D ~ RF*PF + age + sex, where RF = risk-associated factor and PF = protective factor.

Risk Associated-Factor	Estimate	Std. Error	z-value	p-value
Protective Factor: Neighborhood Cohesion				
Food Insecurity	1.90	0.23	8.19	2.53e-16
SED (Area-level)	1.35	0.94	1.43	1.52e-01
SED (Individual-level)	1.22	0.26	4.71	2.52e-06
Discrimination	0.90	0.31	2.92	3.56e-03
Healthcare Discrimination	3.20e-2	0.32	0.10	9.20e-01
Stress	-0.07	0.30	-0.25	8.00e-01
Spirituality	-0.11	0.21	-0.52	6.03e-01
Perceived Neighborhood Disorder	-0.22	0.27	-0.81	4.17e-01
Protective Factor: Social Support				
Food Insecurity	1.14	0.17	6.49	8.39e-11
SED (Individual-level)	0.87	0.19	4.61	4.04e-06
SED (Area-level)	0.85	0.67	1.27	2.03e-01
Spirituality	0.37	0.15	2.54	1.12e-02
Healthcare Discrimination	0.25	0.24	1.07	2.84e-01
Discrimination	0.23	0.23	1.02	3.10e-01
Perceived Neighborhood Disorder	0.16	0.23	0.67	5.05e-01
Stress	-0.43	0.21	-2.03	4.28e-02

DISCUSSION

In this study, we assessed the potential for various SDOH to exert buffering effects on risk-associated factors for T2D. Of the ten SDOH under investigation, eight emerged as factors associated with T2D and

two emerged as T2D protective factors. The classification of each of these SDOH as being either T2D risk-associated or protective factors is largely consistent with their classification in previous literature on T2D epidemiology [25, 26, 34–40]. However, the positive association observed between spirituality and T2D risk appears to be in contrast with previous findings, which suggest that religion and spirituality are generally associated with better health [41]. This association may be driven by individuals using religion or membership to religious communities to cope with the duress brought on by T2D [42, 43]. Furthermore, it may also be driven by unobserved confounding variables that are tightly associated with both spirituality and T2D.

Interaction effects between identified T2D protective and risk-associated factors were assessed to identify potential buffering effects. Several significant interaction effects involving both the neighborhood cohesion and social support protective factors were observed. Of these significant interactions, one buffering effect was observed, which involved social support and stress. This finding is consistent with the stress-buffering hypothesis, suggesting that the buffering effect that social support exerts on positive associations between stress and poor health outcomes may extend to T2D [16, 17]. The remaining six interaction effects observed represent reverse buffering or synergistic effects between the T2D protective factors and corresponding T2D risk-associated factors on T2D. Social support has previously been shown to exert similar reverse buffering effects on positive associations between stress and mental health and on positive associations between workload and job-related stress [44, 45]. Such effects may be explained by the more frequent occurrence of adverse social interactions that may accompany higher degrees of social support. Individuals with higher degrees of social support may also feel a weaker sense of autonomy, which may lead to worse health outcomes [46]. Furthermore, the reverse buffering effects observed may suggest a diminishing return of social support and neighborhood cohesion under adverse conditions. In environments characterized by high SED and food insecurity, social support and neighborhood cohesion may be insufficient to offer substantial protection against the risk of T2D.

These findings are limited as a result of this work being an observational study. The results reported here may have been influenced by a number of unobserved confounding variables. Some such variables may include participant diet and lifestyle, BMI and other comorbidities, which are known to be associated with T2D risk but were not included as covariates in study models [47]. Such variables were not included as covariates due to the lower availability of this data and their potential collinearity with the T2D risk-associated and protective factors presented in the study. Additionally, the *All of Us* participant body is not representative of the US general population. *All of Us* participants are more likely to be female, older, and from underrepresented racial and ethnic groups. The prevalence for many common diseases including T2D is also higher among *All of Us* participants than in the general population [48]. Moreover, the validity of the results presented depend on the composite metrics used and the survey data they are based on, which may not capture all or the most relevant features of the SDOH they were intended to measure. Furthermore, the cross-sectional nature of this study limits the potential to infer causality from its results. Longitudinal studies are needed to validate the results described and to better understand the potential buffering effects of protective SDOH on T2D risk.

CONCLUSIONS

In conclusion, this study reinforces the known roles of several key SDOH as either T2D protective or risk-associated factors. The buffering effect that social support exhibited on the association between stress and T2D risk supports the stress-buffering hypothesis, suggesting that this hypothesis extends to conditions like T2D. This study also reveals several reverse buffering effects involving the social support and neighborhood cohesion protective factors. These findings may suggest that adverse conditions such as socioeconomic or food deprivation may mitigate the protective effects of social support and neighborhood cohesion on T2D risk. Thus, alleviating such adverse conditions may have a compounding effect on combating T2D risk by further enabling communities to benefit from protective factors.

Abbreviations

BMI
Body mass index
EHR
Electronic health record
SED
Socioeconomic deprivation
SDOH
Social determinants of health
T2D
Type 2 diabetes
US
United States

Declarations

Availability of data and materials

All of Us participant data can be accessed and analyzed from the Researcher Workbench by registered users: https://www.researchallofus.org/data-tools/workbench/.

Acknowledgements

The authors would like to thank all who participated in the *All of Us* Research Program, without whom this study would not be possible.

Funding

Author information

Authors and Affiliations

National Institute on Minority Health and Health Disparities, National Institutes of Health, 11545 Rockville Pike, Building 11545 Rockville Pike, Rockville, MD, 20818, USA

Vincent Lam,Christian S. Alvarez, & Leonardo Mariño-Ramírez

School of Biological Sciences, Georgia Institute of Technology, Atlanta, Georgia, USA

Shivam Sharma & I. King Jordan

Contributions

IKJ and LMR conceived of the study. LMR provided supervision and funding for the All of Us Researcher Workbench analysis. VL carried out most of the All of Us Researcher Workbench data analysis and prepared the figures and tables. SS contributed to the All of Us Researcher Workbench data analysis. CSA supported the statistical data analysis. VL, CSA and IKJ wrote and edited the manuscript. All authors read and approved the final manuscript.

Corresponding author

Correspondence to Leonardo Mariño-Ramírez.

Ethics approval and consent to participate

The All of Us operational protocol (#2016–05) is approved by the NIH Institutional Review Board. Written informed consent was obtained from all participants. All data available to researchers has had direct identifiers removed and has been further modified to minimize re-identification risks. Because the All of Us data were not collected specifically for this study and no one on the study team has access to the subject identifiers linked to the data, this study is not considered human subjects research according to the NIH Revised Common Rule for the Protection of Human Subjects: https://grants.nih.gov/policy/humansubjects/hs-decision.htm.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

References

1. Raphael D: Social determinants of health: present status, unanswered questions, and future directions. Int J Health Serv 2006, 36(4):651–677.
2. Braveman P, Egerter S, Williams DR: The social determinants of health: coming of age. Annu Rev Public Health 2011, 32:381–398.
3. Social Determinants of Health. [https://www.who.int/health-topics/social-determinants-of-health#tab=tab_1]
4. Whitman A, De Lew N, Chappel A, Aysola V, Zuckerman R: Addressing social determinants of health: examples of successful evidence-based strategies and current federal efforts. In: *ASPE report*. Washington, D.C.; 2022.
5. Marmot M, Allen JJ: Social determinants of health equity. Am J Public Health 2014, 104 Suppl 4(Suppl 4):S517-519.
6. Davies NM, Dickson M, Davey Smith G, van den Berg GJ, Windmeijer F: The Causal Effects of Education on Health Outcomes in the UK Biobank. Nat Hum Behav 2018, 2(2):117–125.
7. Raghupathi V, Raghupathi W: The influence of education on health: an empirical assessment of OECD countries for the period 1995–2015. Arch Public Health 2020, 78:20.
8. Ettner SL: New evidence on the relationship between income and health. J Health Econ 1996, 15(1):67–85.
9. Benzeval M, Judge K: Income and health: the time dimension. Soc Sci Med 2001, 52(9):1371–1390.
10. DeLilly CR, Flaskerud JH: Discrimination and health outcomes. Issues Ment Health Nurs 2012, 33(11):801–804.
11. Mays VM, Cochran SD, Barnes NW: Race, race-based discrimination, and health outcomes among African Americans. Annu Rev Psychol 2007, 58:201–225.
12. Barnes TL, MacLeod S, Tkatch R, Ahuja M, Albright L, Schaeffer JA, Yeh CS: Cumulative effect of loneliness and social isolation on health outcomes among older adults. Aging Ment Health 2022, 26(7):1327–1334.
13. Leigh-Hunt N, Bagguley D, Bash K, Turner V, Turnbull S, Valtorta N, Caan W: An overview of systematic reviews on the public health consequences of social isolation and loneliness. Public Health 2017, 152:157–171.
14. Egerter S, Dekker M, An J, Grossman-Kahn R, Braveman P: Work Matters for Health. In. Princeton, NJ: Robert Wood Johnson Foundation; 2008.
15. Ahnquist J, Wamala SP, Lindstrom M: Social determinants of health–a question of social or economic capital? Interaction effects of socioeconomic factors on health outcomes. Soc Sci Med 2012, 74(6):930–939.

16. Cohen S, Wills TA: Stress, social support, and the buffering hypothesis. Psychol Bull 1985, 98(2):310–357.

17. Gellert P, Hausler A, Suhr R, Gholami M, Rapp M, Kuhlmey A, Nordheim J: Testing the stress-buffering hypothesis of social support in couples coping with early-stage dementia. PLoS One 2018, 13(1):e0189849.

18. Yoon E, Coburn C, Spence SA: Perceived discrimination and mental health among older African Americans: the role of psychological well-being. Aging Ment Health 2019, 23(4):461–469.

19. Cook SH, Wood EP, Risner E, Weng CA, Xin Y: A national examination of discrimination, resilience, and depressive symptoms during the COVID-19 pandemic: the All of Us Research Program. Front Psychol 2023, 14:1175452.

20. Centers for Disease Control and Prevention: National diabetes statistics report. In. Atlanta, GA: Centers for Disease Control and Prevention; 2024.

21. Bullard KM, Cowie CC, Lessem SE, Saydah SH, Menke A, Geiss LS, Orchard TJ, Rolka DB, Imperatore G: Prevalence of Diagnosed Diabetes in Adults by Diabetes Type - United States, 2016. MMWR Morb Mortal Wkly Rep 2018, 67(12):359–361.

22. Beulens JWJ, Pinho MGM, Abreu TC, den Braver NR, Lam TM, Huss A, Vlaanderen J, Sonnenschein T, Siddiqui NZ, Yuan Z *et al*: Environmental risk factors of type 2 diabetes-an exposome approach. Diabetologia 2022, 65(2):263–274.

23. Murea M, Ma L, Freedman BI: Genetic and environmental factors associated with type 2 diabetes and diabetic vascular complications. Rev Diabet Stud 2012, 9(1):6–22.

24. Dendup T, Feng X, Clingan S, Astell-Burt T: Environmental Risk Factors for Developing Type 2 Diabetes Mellitus: A Systematic Review. Int J Environ Res Public Health 2018, 15(1).

25. Hackett RA, Steptoe A: Type 2 diabetes mellitus and psychological stress - a modifiable risk factor. Nat Rev Endocrinol 2017, 13(9):547–560.

26. Kelly SJ, Ismail M: Stress and type 2 diabetes: a review of how stress contributes to the development of type 2 diabetes. Annu Rev Public Health 2015, 36:441–462.

27. Baek RN, Tanenbaum ML, Gonzalez JS: Diabetes burden and diabetes distress: the buffering effect of social support. Ann Behav Med 2014, 48(2):145–155.

28. Beverly EA, Ritholz MD, Dhanyamraju K: The buffering effect of social support on diabetes distress and depressive symptoms in adults with Type 1 and Type 2 diabetes. Diabet Med 2021, 38(4):e14472.

29. Johnson MD, Anderson JR, Walker A, Wilcox A, Lewis VL, Robbins DC: Spousal protective buffering and type 2 diabetes outcomes. Health Psychol 2014, 33(8):841–844.

30. Bastarache L: Using Phecodes for Research with the Electronic Health Record: From PheWAS to PheRS. Annu Rev Biomed Data Sci 2021, 4:1–19.

31. Gupta S, Lam V, Jordan IK, Mariño-Ramírez L: A composite socioeconomic deprivation index from All of Us survey data: associations with health outcomes and disparities. *medRxiv* 2024.

32. Brokamp C, Beck AF, Goyal NK, Ryan P, Greenberg JM, Hall ES: Material community deprivation and hospital utilization during the first year of life: an urban population-based cohort study. Ann Epidemiol 2019, 30:37–43.

33. Jenkins R, Elliott P: Stressors, burnout and social support: nurses in acute mental health settings. J Adv Nurs 2004, 48(6):622–631.

34. Grundmann N, Mielck A, Siegel M, Maier W: Area deprivation and the prevalence of type 2 diabetes and obesity: analysis at the municipality level in Germany. BMC Public Health 2014, 14:1264.

35. Tatulashvili S, Fagherazzi G, Dow C, Cohen R, Fosse S, Bihan H: Socioeconomic inequalities and type 2 diabetes complications: A systematic review. Diabetes Metab 2020, 46(2):89–99.

36. Fitzgerald N, Hromi-Fiedler A, Segura-Perez S, Perez-Escamilla R: Food insecurity is related to increased risk of type 2 diabetes among Latinas. Ethn Dis 2011, 21(3):328–334.

37. Yu MY, Velasquez AJ, Campos B, Robinette JW: Perceived neighborhood disorder and type 2 diabetes disparities in Hispanic, Black, and White Americans. Front Public Health 2024, 12:1258348.

38. Agbonlahor O, DeJarnett N, Hart JL, Bhatnagar A, McLeish AC, Walker KL: Racial/Ethnic Discrimination and Cardiometabolic Diseases: A Systematic Review. J Racial Ethn Health Disparities 2024, 11(2):783–807.

39. Bennett BL, Puhl RM: Diabetes stigma and weight stigma among physicians treating type 2 diabetes: Overlapping patterns of bias. Diabetes Res Clin Pract 2023, 202:110827.

40. Williams PC, Alhasan DM, Gaston SA, Henderson KL, Braxton Jackson W, 2nd, Jackson CL: Perceived neighborhood social cohesion and type 2 diabetes mellitus by age, sex/gender, and race/ethnicity in the United States. Prev Med 2023, 170:107477.

41. Koenig HG: Religion, spirituality, and health: the research and clinical implications. ISRN Psychiatry 2012, 2012:278730.

42. Satapathy S, Panda B, Panda J, panda S: Evaluation of maternal health services at session site during Health and Nutrition Day in Urban slums of Western Odisha. Indian Journal of Public Health Research and Development 2021, 12:466–474.

43. Duke N: Type 2 diabetes self-management: spirituality, coping and responsibility. Journal of Research in Nursing 2021, 26(8):743–760.

44. Kokoroko E, Sanda MA: Effect of Workload on Job Stress of Ghanaian OPD Nurses: The Role of Coworker Support. Saf Health Work 2019, 10(3):341–346.

45. Liu C, Huang N, Ahmed F, Shahid M, Wang X, Guo J: The reverse buffering effects of social support on the relationships between stresses and mental health: a survey of Chinese adults during the COVID-19 lockdown. Eur J Psychotraumatol 2021, 12(1):1952777.

46. Nahum-Shani I, Bamberger PA: Explaining the Variable Effects of Social Support on Work-Based Stressor-Strain Relations: The Role of Perceived Pattern of Support Exchange. Organ Behav Hum Decis Process 2011, 114(1):49–63.

47. Wu Y, Ding Y, Tanaka Y, Zhang W: Risk factors contributing to type 2 diabetes and recent advances in the treatment and prevention. Int J Med Sci 2014, 11(11):1185–1200.

48. Zeng C, Schlueter DJ, Tran TC, Babbar A, Cassini T, Bastarache LA, Denny JC: Comparison of phenomic profiles in the All of Us Research Program against the US general population and the UK Biobank. J Am Med Inform Assoc 2024, 31(4):846–854.

Figures
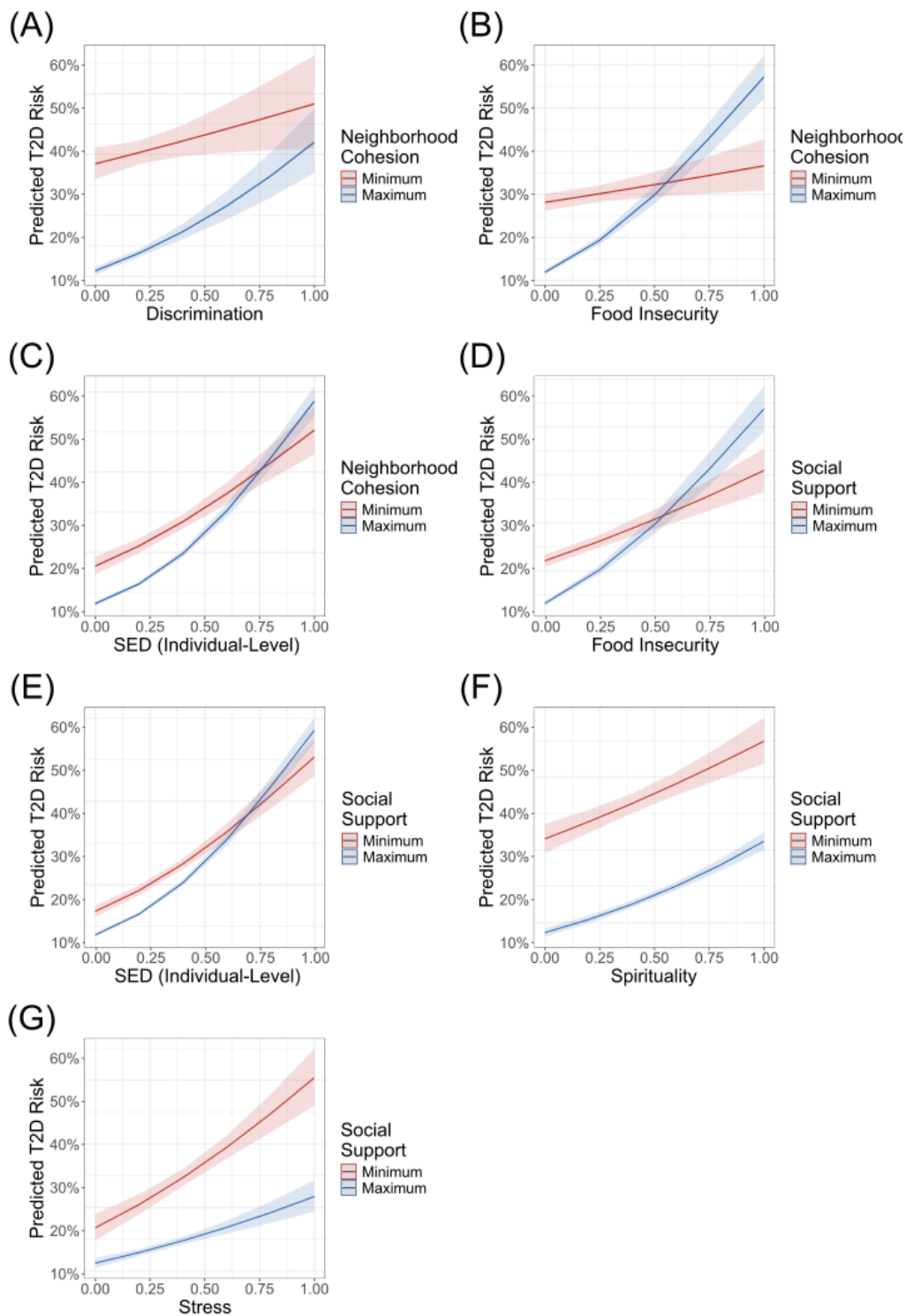
Figure 1

Interactions between T2D risk-associated and protective factors on T2D risk, adjusted for age and sex. Interaction effects between a selected T2D risk-associated factor (x-axis) and protective factor on predicted T2D risk (y-axis) based on the model T2D ~ RF*PF + age + sex, where RF=risk-associated factor and PF=protective factor.