

Population Attributable Risk of Elevated Fasting Plasma Glucose for Hypertension among Native Rural and Tea Garden Communities in Northeast India

Prasanta Borah

pkborah.rmrncne@gov.in

ICMR-NIHR, Dibrugarh, Assam, India (formerly Regional Medical Research Centre, NE Region, Dibrugarh 786010, Assam, India) <https://orcid.org/0000-0002-5371-2787>

Trishnakhi Swargiari

Bhawanipur Anchalik College <https://orcid.org/0000-0003-0080-6568>

Devid Kardong

Dibrugarh University

Manoj Kalita

ICMR-NIHR, Dibrugarh, Assam, India (formerly Regional Medical Research Centre, NE Region, Dibrugarh 786010, Assam, India)

Article

Keywords: Population attributable risk, hypertension, fasting plasma glucose, rural population, Assam, HDSS

Posted Date: October 7th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10152079/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: There is **NO** conflict of interest to disclose.

Abstract

Aim: Elevated fasting plasma glucose (eFPG) is associated with increased risk of hypertension, contributing cardiovascular risks. However, its population-level impact on hypertension remains insufficiently measured. This study aimed to estimate the population attributable risk (PAR) of eFPG for hypertension among native rural and tea garden communities of Assam, Northeast India.

Methods: The participants of a cross-sectional study conducted in Dibrugarh Health and Demographic Surveillance Site, Assam, India were grouped into cases (hypertensive) and control (normotensive) based on European Society of Hypertension (ESH) guidelines 2023. A total of 1238 eligible adults (≥ 18 years) were consecutively recruited after obtaining informed consent. Sociodemographic, lifestyle, anthropometric, and biochemical data were collected using standardized protocols. eFPG was defined as FPG ≥ 100 mg/dL. Logistic regression used to estimate crude and adjusted odds ratios (ORs) with 95% confidence intervals (CIs). Population attributable risk (PAR) was estimated using adjusted ORs and the prevalence of eFPG. Two-sided $p < 0.05$ considered statistically significant.

Results: eFPG was significantly associated with hypertension [AOR: 1.63 (1.24–2.14)]. Estimated PAR of hypertension attributable to eFPG was 18.49% (95% CI: 7.83–29.10), indicating that nearly one in five hypertension cases could be attributed to elevated blood glucose levels.

Conclusion: eFPG contributes significantly to the burden of hypertension in these rural populations. Early detection and proper treatment of dysglycemia may decrease the risk of hypertension and associated risks.

Introduction

Hypertension has been identified as a major risk of cardiovascular disease, and premature mortality (1, 2); and a growing load in low, and middle-income countries (3). India has witnessed rapid increase in hypertension incidence, extending beyond the urban areas to rural and socioeconomically disadvantaged populations (4). Disturbances in glucose metabolism, including impaired fasting glucose and diabetes mellitus, become more prevalent and frequently coexist with hypertension, increasing the risk of cardiovascular disorders (5). There are several pathophysiological mechanisms identified as linking way of dysglycaemia and hypertension (6). They include insulin resistance, dysfunction of endothelial system, activation of renin–angiotensin–aldosterone system (RAAS), and chronic low-grade systemic inflammations etc (6).

A number of cohort studies demonstrated that the individuals with abnormal glucose homeostasis have higher risk of increased blood pressure and developing hypertension than to those with normal glucose levels (5, 7, 8). Some large cohort studies also reported that fasting plasma glucose predicted the incident of hypertension independent of traditional risk factors (9, 10). While numerous studies have demonstrated the association between elevated fasting plasma glucose (eFPG) and hypertension,

population-level impact of dysglycaemia on hypertension in rural and underprivileged populations with reportedly high (11, 12) prevalence of hypertension is limited.

Population attributable risk (PAR) is the proportion of risk of the disease in a population that can be attributed to the causal effect of a specific risk factor or group of risk factors (13). Estimating PAR for hypertension attributable to elevated fasting plasma glucose can prioritize prevention strategies by early glycaemic control. The PAR (%) is based on both the strength of the exposure risk (eFPG)–outcome (hypertension) association, and on the distribution of exposure and outcome in the population (14). PAR has been widely applying in epidemiological tool to determine the contribution of obesity, environmental factors, lifestyle habits and dyslipidaemia to cardiovascular diseases (15, 16). A study based on rural and tea garden population from Northeastern region of India have also reported the incidence of hypertension attributable to some lifestyle factors such as alcohol and tobacco use (14). The present study is aimed to estimate the PAR of hypertension due to elevated fasting plasma glucose in a population covering native rural and tea garden communities of Assam, Northeast India.

Method

Study design and study area

A community based cross- sectional study was conducted in the Health and Demographic Surveillance Site, located in Dibrugarh, Assam (Dibrugarh- HDSS). The HDSS covers 60 contiguous villages and 20 tea gardens, representing both native rural and tea garden populations.

Sample size calculation and study population

Considering a prevalence of 26.3% (Impaired fasting glucose and Diabetic range FPG from Assam reported from ICMR- INDIAB study) (17) and a relative allowable error of 10% of the estimated prevalence at 95% confidence interval, the required sample size was calculated as 1077. After adjusting for a 10% non-response rate, the final sample size required for the study was 1196, which was rounded off to 1200 participants. We obtained approval from the Institutional Ethics Committee (Human) of ICMR-Regional Medical Research Centre, NE Region, Dibrugarh (No. RMRC/Dib/IEC (Human)/2021-22/963).

Sampling strategy and participant recruitment

Field clinics were established and community awareness activities were conducted at randomly chosen, community-accessible locations such as village community centres, residential 'lines' (clusters of households) of tea garden areas, schools, and other social gathering premises within the HDSS area. Participants who attended the camps, consented and met the eligibility criteria were consecutively

recruited until the desired sample size was achieved. A total of 1238 participants representing 10 villages and 1 tea garden within the HDSS area were enrolled.

Inclusion and Exclusion criteria:

Adults aged ≥ 18 years of either gender were eligible for the study. Subjects with previous history of cardiovascular disease, pregnant women or lactating mothers, women currently on oral contraceptives or hormone replacement therapy, unable to provide written informed consent or understanding the study procedure were excluded from the study.

Collection of socio demographic, lifestyle and anthropometric data

A pre tested and structured questionnaire based on the WHO STEPwise approach to noncommunicable disease risk factor surveillance (WHO STEPS) (18, 19) was used to collect the information on socio demographic characteristics and lifestyle behaviours. Anthropometric measurements included body weight, height, and waist circumference. Height was measured to the nearest 1 centimetre using a Seca 213 portable stadiometer placed on a flat surface. Body weight was measured to the nearest 100 g using a weighing scale (Krupps). Body mass index (BMI) was calculated as weight in kilograms divided by height in meter squared (kg/m^2). BMI was categorized as per World Health Organization guidelines as underweight ($< 18.5 \text{ kg}/\text{m}^2$), normal weight ($18.5\text{--}24.9 \text{ kg}/\text{m}^2$), pre-obese ($25.0\text{--}29.9 \text{ kg}/\text{m}^2$), and obese ($\geq 30.0 \text{ kg}/\text{m}^2$) (20). Waist circumference was measured to the nearest 1 cm using a non-stretchable metallic measuring tape, with the participant standing straight (21). All anthropometric measurements were performed according to standard protocols.

Measurement of blood pressure

Blood pressure of participant was measured in the sitting position in the right hand using a digital blood pressure monitor (Omron, Model: HEM-7124). We took appropriate cuff size, placing the device positioned at heart level. Three measurements were recorded at an interval of 5–10 minutes and then the average of the last two readings were taken for analysis. Hypertension was defined as systolic blood pressure $\geq 140 \text{ mmHg}$ and/or diastolic blood pressure $\geq 90 \text{ mmHg}$, or current use of antihypertensive medication (22).

Biochemical measurements

For biochemical analyses; venous blood sample of the subject was collected in appropriate vials after overnight fast for at least 8 hours. We estimated blood glucose, lipid profile (HDL, LDL, total cholesterol and triglycerides), blood urea, serum creatinine and uric acids. We used colorimetric method (except in LDL cholesterol) and followed manufacturer's instructions mentioned in different kit inserts (ERBA

Manheim reagents) (23–25). Friedewald formula was used to calculate LDL cholesterol: LDL cholesterol = total cholesterol – HDL cholesterol – (triglycerides/5) (26).

Exposure assessment

eFPG was defined as; FPG $\geq$ 100 mg/dL, combining IFG (100–125 mg/dL) and diabetic-range FPG ($\geq$ 126 mg/dL) as per ADA 2025 criteria (27).

Statistical analysis

All statistical analysis were carried out using IBM SPSS Statistics, version 26.0 (IBM Corporation, Armonk, New York, USA). For analytical purposes, participants were categorized into hypertensive and normotensive groups based on blood pressure measurements and treatment status. Participant characteristics were summarized using descriptive statistics; represented as N (%) and mean $\pm$ SD wherever applicable. Differences between the study groups were analysed using appropriate statistical tests. We used chi-square test for categorical variables. For continuous variables, Kolmogorov-Smirnov was performed for test of normality followed by non-parametric test for comparison. We used univariate and multivariable (adjusted for age, sex, physical activity, alcohol consumption, tobacco use, body mass index, waist circumference, systolic and diastolic blood pressure, biochemical parameters) analysis for crude and adjusted odds ratio of exposure variable (eFPG) and risk of hypertension. A two-sided p-value < 0.05 was considered as statistically significant.

Population attributable risk (PAR%) were calculated taking the prevalence of eFPG in the study population and the adjusted odds ratio derived from multivariable logistic regression analysis. The population attributable risk (PAR) was calculated using the formula; “PAR= $[P(OR-1)] / [P(OR-1) + 1] \times 100$ ” (13) where P represents the prevalence of the exposure in the population and OR is the adjusted odds ratio. Confidence limits were obtained by substituting the lower and upper bounds of the odds ratio into the PAR formula.

Results

Participant characteristics

Hypertension was observed in 37.40% of the participants. The study revealed that hypertensive individuals were older and had higher BMI and waist circumference compared to the normotensive ones. The prevalence of eFPG was higher among hypertensive (46.7%) than normotensive (29.3%) participants. Table 1 shows sociodemographic, anthropometric, biochemical, and lifestyle characteristics of study participants by hypertension status.

Association of elevated fasting plasma glucose with hypertension

Elevated fasting plasma glucose was found to have significant association with hypertension in both crude (OR = 2.11, 95% CI = 1.66–2.68) and adjusted analysis (OR = 1.63, 95% CI = 1.24–2.14, p value = 0.001. (Table.2)

Population attributable risk

Based on the adjusted odds ratio and the prevalence of elevated fasting plasma glucose in the study population, the estimated population attributable risk (PAR%) of hypertension due to eFPG was estimated as 18.49% (95% CI: 7.83–29.10). This finding suggests that nearly one-fifth of hypertension incidence in the study population may be attributable to elevated fasting plasma glucose (Table. 3)

Discussion

This study demonstrates a significant association between elevated fasting plasma glucose and hypertension among native rural and tea garden population of Assam, Northeast India. Importantly, the PAR analysis indicates that around 18.49% of hypertension cases, that is one fifth of the hypertensive participants in this population may be attributable to elevated fasting plasma glucose indicating public health relevance.

The estimated population attributable risk from this study population indicates the public health relevance of dysglycaemia as a modifiable risk cause for hypertension. Reducing the occurrence of elevated fasting plasma glucose even at moderate level can decrease the burden of hypertension (28). Given that our study population are inhabited in the areas with limited access to proper healthcare services, this finding is therefore particularly relevant for these communities.

In our findings; elevated fasting plasma glucose independently predicts hypertension as an attributable risk. This finding is consistent with previously reported other cohort and cross-sectional studies that identified hypertension as an increased risk among the individuals with IFG and diabetes (5, 29). A Japanese cohort (7) and a Chinese cohort (9) reported that IFG independently predicted the occurrence of hypertension over a follow-up of at least 5 years, highlighting that fasting blood glucose as an important determinant of elevation of blood pressure. Other cohort studies have also reported similar findings showing more than a two-fold increased risk of incident hypertension over a mean follow-up of 3.7 ± 2.9 years (28). Our findings added the evidence and broadens the clinical understanding of metabolic health by showing that the public health consequences of dysglycaemia surpasses glucose metabolism, highlighted a significant contribution to hypertension suggesting that imbalances in glucose homeostasis are major, under recognized driver of the prevalence of hypertension within the study population.

In the present analysis we have considered lipid profile parameters and renal function markers simultaneously with the primary exposure. Dyslipidaemia, particularly elevated triglycerides and LDL cholesterol, are frequently associated with abnormal glucose metabolism and has been independently associated with hypertension as reported by previous studies (30, 31), while HDL cholesterol is often

inversely related to cardiovascular risk (32). The overall lipid profile markers were carefully adjusted for multivariable models in the analysis, thereby reducing the risk that the observed association between elevated fasting plasma glucose and hypertension was countered by underlying abnormalities in lipid metabolism.

From the perspectives of public health, our findings implies that a considerable proportion of the burden of hypertension in the population under study may be attributable to dysglycaemia. In practical terms, our study indicates that early detection, management and treatment of impaired fasting glucose through proper health care facilities, awareness interventions and lifestyle improvement could contribute relevantly to the prevention of hypertension. In the areas with limited resource and access to primitive healthcare facilities; the primary care level may offer a practical approach by addition of blood glucose screening with routine blood pressure monitoring. As per our knowledge, this is among the first studies to estimate the population attributable risk of elevated fasting plasma glucose for hypertension among native rural and tea garden communities of Assam, Northeast India

The strengths of this study lie in its community representation which was conducted within a well-defined Health and Demographic Surveillance Site, an adequate sample size, and the comprehensive adjustment for key sociodemographic, lifestyle habits, cardiometabolic, and renal markers. However, the observational nature of this study limits the ability to draw consequences, and dependence on single time measurements of fasting blood glucose were the limitations of the study. Hidden unmeasured factors including genetic markers cannot be ruled out from the consideration. In addition to these, the findings may not be directly generalizable to the populations beyond those with similar sociodemographic characteristics.

Conclusion

Elevated fasting plasma glucose is found to be an important contributor to hypertension among native rural and tea garden populations of Assam. A considerable proportion of incidence of hypertension could potentially be prevented through early detection, management and treatment of dysglycaemia.

Declarations

Data availability

The datasets generated during and/or analysed during the current study are not publicly available due to ethical restrictions from the institutional ethics committee upon governing the confidentiality of participant data, but are available from the corresponding author on reasonable request.

Declaration of AI Usage:

The authors used AI tools to assist with language editing and to enhance the clarity of this document. All content has been thoroughly reviewed by the authors, who take full responsibility for its

accuracy, integrity, and originality.

Acknowledgements

The authors sincerely acknowledge the Indian Council of Medical Research (ICMR) for financial support under the Junior Research Fellowship (JRF) Programme. The authors also thank the staff of the Health and Demographic Surveillance Site, Dibrugarh district, for their field support, and all study participants for their cooperation.

Author Contributions

Trishnakhi Swargiari: funding acquisition, conceptualization, data curation, methodology, analysis, writing- original draft, writing- review and editing.

Dr. Devid Kardong: conceptualization, supervision, methodology, validation, writing: review and editing.

Manoj Kalita: analysis, validation, writing- review and editing.

Dr. Prasanta Kumar Borah: supervision, conceptualization, methodology, analysis and writing- original draft, writing- review and editing.

Ethical consideration

The study has been permitted by the Institutional Ethics Committee (Human) of ICMR-RMRCNE (No. RMRC/Dib/IEC (Human)/2021-22/963).

Competing Interest

The authors declare they have no known competing interests associated with this publication.

Funding

This study was supported by the Indian Council of Medical Research (ICMR) under the Junior Research Fellowship (JRF) Programme (3/1/3/JRF- 2019/HRD- 082(31261)) (Trishnakhi Swargiari)

Role of the Funder

The funding agency had no role in the study design, data collection, analysis, interpretation of data, manuscript preparation, or the decision to submit the manuscript for publication.

References

1. Kannel WB. Blood Pressure as a Cardiovascular Risk Factor: Prevention and Treatment. JAMA1996; 275:1571–6. <https://doi.org/10.1001/JAMA.1996.03530440051036>.

2. Franklin SS, Wong ND. Hypertension and Cardiovascular Disease: Contributions of the Framingham Heart Study. *Glob Heart* 2013; 8:49–57. <https://doi.org/10.1016/J.GHEART.2012.12.004>.
3. Gheorghe A, Griffiths U, Murphy A, Legido-Quigley H, Lamptey P, Perel P. The economic burden of cardiovascular disease and hypertension in low- and middle-income countries: a systematic review. *BMC Public Health* 2018; 18(1):975. <https://doi.org/10.1186/S12889-018-5806-X>.
4. Gupta R, Gaur K, S Ram CV. Emerging trends in hypertension epidemiology in India. *J Hum Hypertens* 2018; 33(8):575–87. <https://doi.org/10.1038/s41371-018-0117-3>.
5. Zhao Y, Sun H, Wang B, Zhang M, Luo X, Ren Y, et al. Impaired fasting glucose predicts the development of hypertension over 6 years in female adults: Results from the rural Chinese cohort study. *J Diabetes Complications* 2017; 31(7):1090–95. <https://doi.org/10.1016/J.JDIACOMP.2017.04.006>.
6. Cheung BM, Li C. Diabetes and hypertension: Is there a common metabolic pathway? *Curr Atheroscler Rep*. Springer 2012; 14:160–66. <https://doi.org/10.1007/s11883-012-0227-2>.
7. Kuwabara M, Chintaluru Y, Kanbay M, Niwa K, Hisatome I, Andres-Hernando A, et al. Fasting blood glucose is predictive of hypertension in a general Japanese population. *J Hypertens* 2019; 37:167–74. <https://doi.org/10.1097/HJH.0000000000001895>.
8. Ahn SK, Lee JM, Ji SM, Kim KH, Park JH, Hyun MK. Incidence Hypertension and Fasting Blood Glucose from Real-World Data: Retrospective Cohort for 7-Years Follow-Up. *Int J Environ Res Public Health* 2021; 18: 2085. <https://doi.org/10.3390/IJERPH18042085>.
9. Luo B, Feng L, Bi Q, Shi R, Cao H, Zhang Y. Fasting Plasma Glucose and Glycated Hemoglobin Levels as Risk Factors for the Development of Hypertension: A Retrospective Cohort Study. *Diabetes Metab Syndr Obes* 2023;16:1791–8. <https://doi.org/10.2147/DMSO.S410670>.
10. Liu J, Cheng NN, Zhou ZY, Zhang Y, Yang J, Liu LS, et al. Effect of fasting blood glucose on risk of new-onset hypertension in rural Chinese population: a 15-year follow-up cohort. *BMC Cardiovasc Disord* 2021; 21:531. <https://doi.org/10.1186/S12872-021-02336-4>.
11. Hazarika NC, Biswas D, Narain K, Kalita HC, Mahanta J. Hypertension and its risk factors in tea garden workers of Assam. *Natl Med J India* 2002; 15(2):63–8. <http://www.ncbi.nlm.nih.gov/pubmed/12044117>.
12. Narain K, Biswas D, Mahanta J. Hypertension in the native rural population of Assam. *Natl Med J India* 2004;17(6):300–304. <http://www.ncbi.nlm.nih.gov/pubmed/15736549>.
13. Rockhill B, Newman B, Weinberg C. Use and misuse of population attributable fractions. *Am J Public Health* 1998;88(1):15–9. <https://doi.org/10.2105/ajph.88.1.15>.
14. Shankarishan P, Borah PK, Mohapatra PK, Ahmed G, Mahanta J. Population attributable risk estimates for risk factors associated with hypertension in an Indian population. *Eur J Prev Cardiol* 2013; 20(6):963–71. <https://doi.org/10.1177/2047487312462146>.
15. Global Burden of Metabolic Risk Factors for Chronic Diseases Collaboration. Cardiovascular disease, chronic kidney disease, and diabetes mortality burden of cardiometabolic risk factors from

- 1980 to 2010: A comparative risk assessment. *Lancet Diabetes Endocrinol* 2014; 2(8):634–47. [https://doi.org/10.1016/S2213-8587\(14\)70102-0](https://doi.org/10.1016/S2213-8587(14)70102-0).
16. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; 380(9859):2224–60. [https://doi.org/10.1016/S0140-6736\(12\)61766-8](https://doi.org/10.1016/S0140-6736(12)61766-8).
 17. Anjana RM, Deepa M, Pradeepa R, Mahanta J, Narain K, Das HK, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR–INDIAB population-based cross-sectional study. *Lancet Diabetes Endocrinol* 2017; 5(8):585–96. [https://doi.org/10.1016/S2213-8587\(17\)30174-2](https://doi.org/10.1016/S2213-8587(17)30174-2).
 18. World Health Organization. Noncommunicable Diseases and Mental Health Cluster (2005). WHO STEPS surveillance manual: the WHO STEPwise approach to chronic disease risk factor surveillance / Noncommunicable Diseases and Mental Health, World Health Organization. World Health Organization. World Health Organization. <https://iris.who.int/handle/10665/43376>.
 19. World Health Organization. Noncommunicable Diseases and Mental Health Cluster. Surveillance Team (2001). STEPS instruments for NCD risk factors (core and expanded version 1.4): the WHO STEPwise approach to Surveillance of noncommunicable diseases (STEPS). World Health Organization. World Health Organization. <https://iris.who.int/handle/10665/68346>.
 20. World Health Organization. Body mass index (BMI), <https://www.who.int/data/gho/data/themes/topics/topic-details/GHO/body-mass-index> [accessed 6 January, 2026].
 21. World Health Organization (2011). Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8–11 December 2008. <https://iris.who.int/handle/10665/44583>.
 22. Mancia G, Kreutz R, Brunström M, Burnier M, Grassi G, Januszewicz A, et al. 2023 ESH Guidelines for the management of arterial hypertension the Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association. *J Hypertens* 2023; 41(12):1874–2071. <https://doi.org/10.1097/HJH.0000000000003480>.
 23. Trinder P. Determination of Glucose in Blood Using Glucose Oxidase with an Alternative Oxygen Acceptor. *Ann Clin Biochem* 1969; 6(1):24–7. <https://doi.org/10.1177/000456326900600108>.
 24. Fawcett JK, Scott JE. A rapid and precise method for the determination of urea. *J Clin Pathol* 1960; 13:156–9. <https://doi.org/10.1136/JCP.13.2.156>.
 25. Owen JA, Iggo B, Scandrett FJ, Stewart CP. The determination of creatinine in plasma or serum, and in urine; a critical examination. *Biochem J* 1954; 58(3):426–37. <https://doi.org/10.1042/BJ0580426>.
 26. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972; 18(6):499–502. <http://www.ncbi.nlm.nih.gov/pubmed/4337382>.

27. American Diabetes Association Professional Practice Committee. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2025. *Diabetes Care* 2025;48(1 Suppl 1):S27–49. <https://doi.org/10.2337/dc25-S002>.
28. Geva M, Shlomai G, Berkovich A, Maor E, Leibowitz A, Tenenbaum A, et al. The association between fasting plasma glucose and glycated hemoglobin in the prediabetes range and future development of hypertension. *Cardiovasc Diabetol* 2019; 18(1): 53. <https://doi.org/10.1186/S12933-019-0859-4>.
29. Moazzeni SS, Dehghani S, Hasheminia M, Hizomi Arani R, Azizi F, Hadaegh F. Three-year change in glycemic state and the future risk of incident hypertension among Iranian adults: Tehran Lipid and Glucose Study. *J Hum Hypertens* 2023; 37(10):944–9. <https://doi.org/10.1038/s41371-022-00788-5>.
30. Grundy SM. Metabolic syndrome pandemic. *Arterioscler Thromb Vasc Biol* 2008;28(4):629–36. <https://doi.org/10.1161/ATVBAHA.107.151092>.
31. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet* 2005; 365(9468):1415–28. [https://doi.org/10.1016/S0140-6736\(05\)66378-7](https://doi.org/10.1016/S0140-6736(05)66378-7).
32. Duffy D, Rader DJ. Update on strategies to increase HDL quantity and function. *Nat Rev Cardiol* 2009; 6(7):455–63. <https://doi.org/10.1038/NRCARDIO.2009.94>.

Tables

Tables 1 to 3 are available in the supplementary files section

Figures

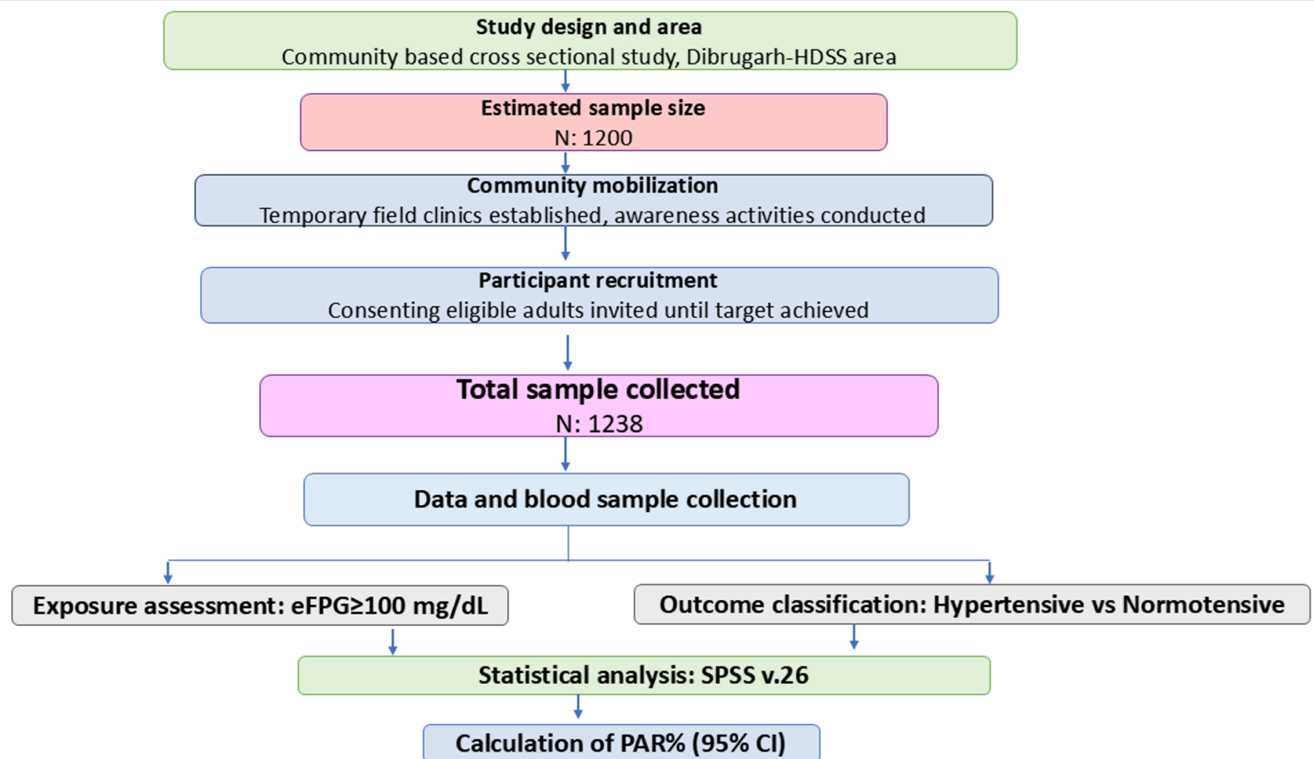


Figure 1. Overview of the methodology (Created with Microsoft PowerPoint)

Figure 1

See image above for figure legend.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Table1.xlsx](#)
- [Table2.xlsx](#)
- [Table3.xlsx](#)