Assessing the relationship between nasal decongestant use and hypertension in UK Biobank: A cross-sectional study

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ABSTRACT

INTRODUCTION Hypertension is a leading global health issue, with growing prevalence and significant contributions from medication-induced causes. This study investigates the association between nasal decongestant use and hypertension in the UK Biobank population, evaluating potential hypertensive effects of adrenergic agonists and intranasal corticosteroids.

METHODS This cross-sectional study of 500295 UK Biobank participants examined the association between use of adrenergic agonist nasal decongestants and intranasal steroids, self-reported hypertension, and systolic/diastolic blood pressure (SBP/DBP). Multiple logistic regression models were fitted to estimate odds ratios (ORs) for nasal decongestant use, SBP and DBP, adjusting for sociodemographic and lifestyle covariates.

RESULTS This study identified 8918 adult participants who self-reported hypertension and were using nasal decongestants that are considered unsuitable for individuals with arterial hypertension. Adrenergic agonist use was not significantly associated with elevated SBP (AOR=1.25; 95% CI: 0.69–2.26) or DBP (AOR=1.02; 95% CI: 0.53–1.94), while steroid use was not significantly associated with elevated SBP (AOR=1.03; 95% CI: 0.98–1.07) but was associated with a statistically significant increase in the odds of high DBP (AOR=1.09; 95% CI: 1.04–1.15).

CONCLUSIONS In this large cross-sectional analysis of UK Biobank participants, use of adrenergic agonist nasal decongestants was not significantly associated with elevated SBP or DBP, while intranasal corticosteroid use was linked to a modest but statistically significant increase in the odds of high DBP. These findings underline the need to consider potential cardiovascular effects of commonly used nasal medications, particularly in individuals with hypertension. Further longitudinal and mechanistic studies are required to clarify causality, explore dose response relationships, and guide safer prescribing and self-medication practices.

INTRODUCTION

Hypertension is considered one of the most pressing public health challenges globally. The World Health Organization (WHO), in its inaugural Global Report on Hypertension published in 2023, expressed concern regarding the increasing global burden of hypertension and highlighted the need to address this issue[1]. The report estimated that more than a billion people worldwide have high blood pressure, with a diagnosis established in approximately 54% of the adult population globally. Furthermore, the report stated that of this 54%, approximately 42% received some form of treatment to control it, and hypertension is effectively controlled in only about 20% of those who received treatment[1]. The findings of the WHO report align with the findings of two separate systematic reviews conducted by Stanaway et al.[2] and Mills et al.[3], which reported disparities in the prevalence of hypertension worldwide, with high-income countries experiencing decreasing prevalence, and increasing prevalence observed in low- and middle-income countries.

In the modern practice, drug-induced secondary hypertension is a neglected issue despite contributing significantly to the increasing hypertension burden on populations[4-6]. Currently, there are approximately 50 drugs available commercially that are linked to secondary hypertension[7-10]. These medications span several distinct categories, have diverse mechanisms of action, and some are readily accessible as over-the-counter (OTC) or prescription medicines[10,11]. Among these, adrenergic agonist nasal decongestants and intranasal corticosteroids represent a particularly relevant group due to their widespread use and accessibility[11]. Although most nasal decongestants and intranasal corticosteroids are widely regarded as safe and routinely used across clinical settings, disparities in safety evidence have emerged, with clinical trials frequently reporting minimal risk and case reports documenting severe adverse outcomes in individuals[12-16]. These conflicting findings are particularly relevant to the target drugs (oxymetazoline, xylometazoline, and intranasal corticosteroids) examined in this study.

Buysschaert et al.[13] reported cases of hypertension, cardiomyopathy, and end-organ failure in a 34-year-old male patient who had been using xylometazoline daily for extended periods[13]. Russo et al.[14] found that individuals aged 18–30 years and 60–75 years were the most frequent users of topical decongestants, with approximately 32% using them for longer than five days. In one of the contrasting experimental studies, Bellew et al.[15] found no statistically significant increase in blood pressure related to nasal decongestant use. Although the result was insignificant, the authors reported their failure to recruit enough participants, which may have resulted in an underpowered study. Klas et al.[16], in their study, discussed potential mechanisms leading to adverse reactions from long-term high-frequency use of nasal decongestants and called for further research to fully understand the underlying mechanisms behind nasal decongestant use and hypertension.

This study investigates drug-induced secondary hypertension by assessing the association between nasal decongestant (oxymetazoline, xylometazoline, and intranasal corticosteroids) use and hypertension within the UK Biobank. We therefore seek to accomplish two objectives: 1) to explore the prevalence of hypertension in UK Biobank participants; and 2) to assess the association between nasal decongestant use and hypertension within this population. These findings may have implications for the appropriate use of nasal decongestants, especially for individuals at risk of cardiovascular issues.

METHODS

Study design

A cross-sectional study was conducted using data obtained from the UK Biobank, a large-scale biomedical database comprising approximately 500000 participants aged 37–73 years from England, Wales, and Scotland[17]. UK Biobank data were collected between 2006 and 2010 across 22 assessment centers in the United Kingdom (UK)[17]. Study reporting adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement[18].

Exposure and covariates

Baseline data were collected at the assessment centers using standardized touchscreen questionnaires followed by a nurse-led, computer-assisted interview, while physical measurements were obtained by trained staff according to uniform operating procedures[19]. During the nurse-led, computer-assisted interview, participants were asked to report their use of adrenergic nasal decongestants (oxymetazoline, xylometazoline) and intranasal corticosteroid use. Medication was coded as: 1=adrenergic agonists, 2=intranasal steroids, and 0=others. Sociodemographic variables were coded as: sex (0=female, 1=male), age (categorized as 37–49, 50–59, and ≥60 years), the Townsend deprivation index (transformed into quintiles; 1=least deprived, 5=most deprived), and ethnicity[20]. Ethnicity was grouped as White, Asian, Black, Chinese, Mixed, or Other. Behavioral factors such as smoking and alcohol consumption status were categorized as current, previous, or never[19]. During the nurse-led, computer-assisted interview, participants were asked to report physician-diagnosed chronic respiratory conditions, including asthma, bronchitis, chronic obstructive pulmonary disease (COPD), and sinusitis. Chronic respiratory disease status was coded as: Present=1, Absent=0. Body mass index (BMI) was recorded and classified using WHO criteria (healthy weight, obese, overweight, underweight)[17].

Outcomes

The outcomes of this study were arterial hypertension, determined from self-reports and systolic and diastolic blood pressure measurements. Self-reported hypertension status was obtained during a nurse-led interview. Arterial blood pressure was measured during the baseline assessment (2006–2010) using an automated electronic blood pressure monitor (Omron 705 IT)[17,21]. Two consecutive readings were taken with a minimum 1-minute interval between measurements[17,21]. The average of the two readings was recorded in the UK Biobank and was used in the analysis[17,21]. Arterial blood pressure values recorded in the UK Biobank were classified according to the thresholds described in the European Society of Hypertension 2023 guidelines[4]. Systolic blood pressure ≥140 mmHg was classified as hypertension (coded as: Present=1, Otherwise=0). Similarly, diastolic blood pressure ≥90 mmHg was classified as hypertension (coded as: Present=1, Otherwise=0)[4,21].

Inclusion and exclusion criteria

All adult participants with valid measurements were included in the study, while those with non-contributing exposure data were excluded due to missing values[17,19,21].

Missing data were assessed using plots, and records with missing values were excluded from the study. Age, sex, socioeconomic status, ethnicity, BMI, smoking status, alcohol consumption, and chronic respiratory disease were considered potential confounders and were adjusted for in regression models, where appropriate.

Statistical analysis

Descriptive statistics were used to evaluate the distribution of hypertension across demographic, behavioral, and clinical characteristics. Multiple logistic regression was used to model the probability of arterial hypertension. Each outcome (self-reported hypertension, elevated SBP, elevated DBP) had a separate model. Regression models included nasal decongestant use as the primary exposure variable, with age, sex, socioeconomic status, ethnicity, BMI, and smoking status as covariates in the self-reported hypertension model, and with the addition of chronic respiratory disease and alcohol consumption in the arterial blood pressure models (elevated SBP and elevated DBP). Regression models for self-reported hypertension, SBP, and DBP were refined through stepwise exclusion of non-significant variables. The Likelihood Ratio Test (LRT) was used to compare nested models to identify the best-fitting model[22]. To assess model performance, two techniques were used: 1) the LRT was used to evaluate statistical improvement in model fit when additional predictors were included; and 2) k-fold cross-validation (using partitioned training and testing datasets) assessed model robustness and generalizability, reporting predictive accuracy[23]. Data preparation and analysis were conducted using RStudio (v4.4.1)[24]. Statistical significance for this study was set at $p<0.05$.

Ethical approval

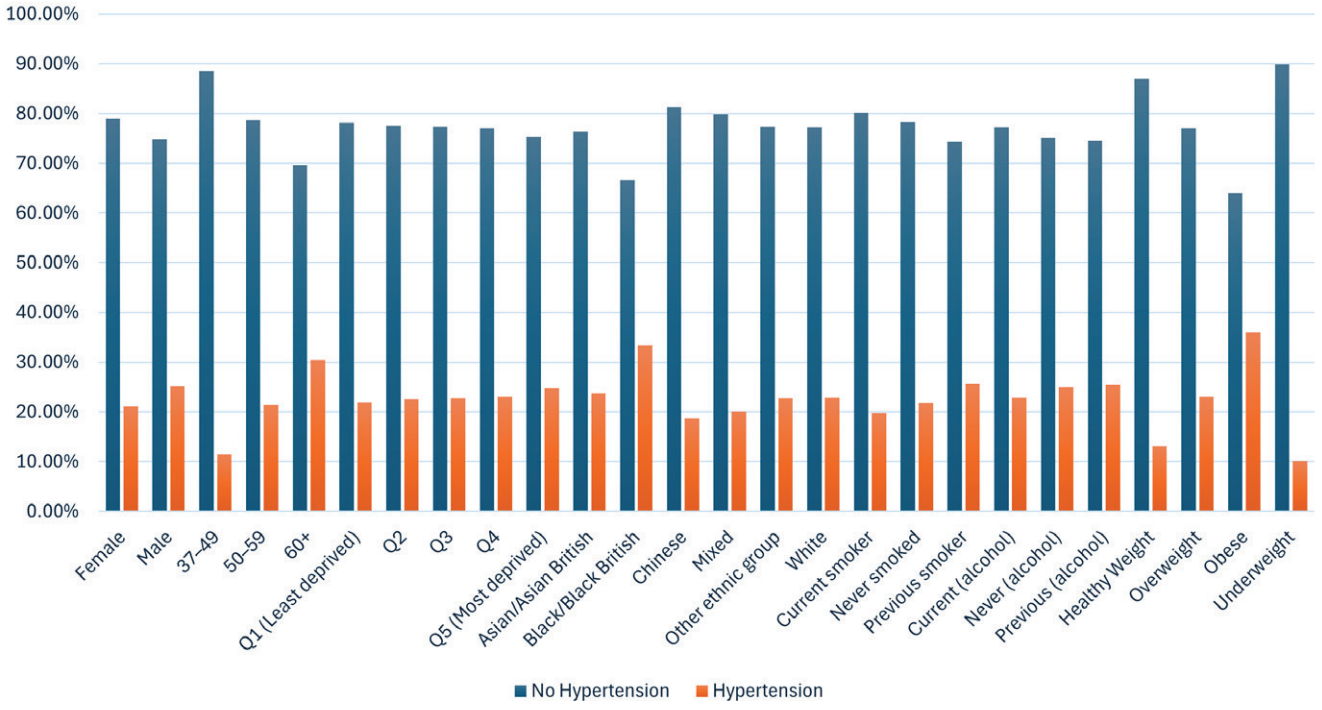
Separate ethical approval for this study was not required as UK Biobank has pre-existing approval from the Northwest Multi-centre Research Ethics Committee (REC reference: 16/NW/0274) as a Research Tissue Bank and is licensed by the Human Tissue Authority[17,21]. This study was conducted as part of UK Biobank application 71392. However, UK Biobank mandates that researchers comply with the General Data Protection Regulation (GDPR), ensuring data security and confidentiality[17,21]. These standards were rigorously followed in this study.

RESULTS

Prevalence of hypertension in the UK Biobank

The final dataset contained 486098 eligible individuals, of which 111619 (22.96%) reported having hypertension during the structured interview with a nurse. Descriptive statistics were used to evaluate the prevalence of hypertension across demographic, behavioral, and clinical

Figure 1. Prevalence of hypertension across demographic and health categories, UK Biobank: 2006–2010, United Kingdom (N=486098)



Bar chart showing the percentage of individuals with hypertension across demographic and health-related categories. Categories include sex (female, male), age groups (37–49, 50–59, ≥60 years), socioeconomic status (Townsend Q1–Q5), ethnicity, smoking status (current, never, previous), alcohol consumption status (current, never, previous), chronic respiratory disease (CRD), medication use (adrenergic agonist, intranasal steroid) and body mass index (BMI).

characteristics (Figure 1). Among 8918 decongestant users who reported hypertension, 49 were in the adrenergic agonist group, and 8869 were in the intranasal steroid group. We found a higher prevalence of hypertension in males (25.2%) compared to females (21.1%). Prevalence of hypertension increased with age, rising from 11.5% among those aged 37–49 years to 30.4% among those aged ≥60 years. Black participants had the highest prevalence (33.4%), followed by Asian participants (23.7%). A modest inverse trend in hypertension prevalence was observed across Townsend deprivation quintiles. Obesity was strongly associated with hypertension (36.0%), whereas individuals of healthy weight and those who were underweight had lower prevalence (13.1% and 10.1%, respectively). Former smokers and individuals with a history of alcohol consumption also had higher prevalence. All associations were statistically significant ($p<0.001$).

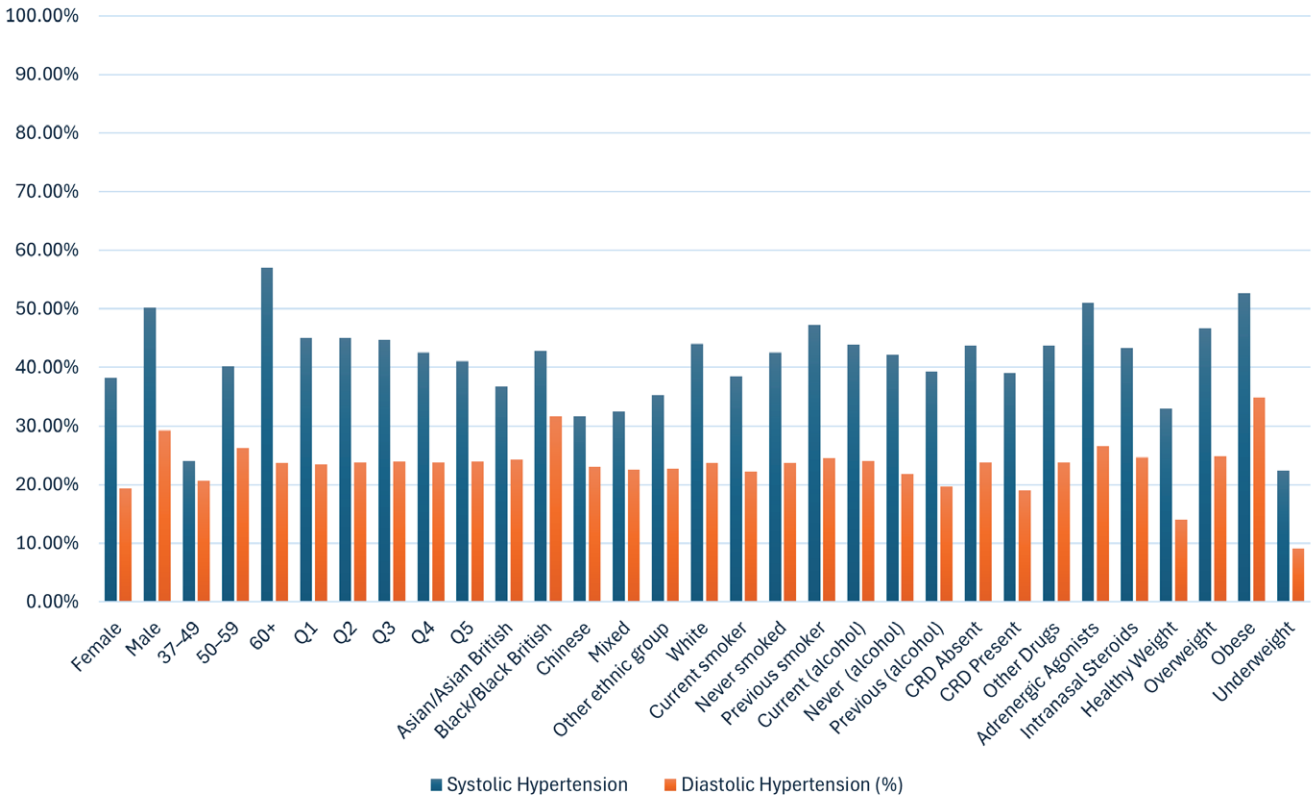
Descriptive statistics further demonstrated sex-based differences in hypertension outcome (Figure 2). Males exhibited higher prevalence of both systolic (50.2%) and diastolic (29.2%) hypertension compared with females (38.2% and 19.3%, respectively). Age was positively associated with systolic hypertension, increasing from 24.0% among individuals aged 37–49 years to 57.0% among those aged ≥60 years. A slight inverse association was again observed between hypertension prevalence and Townsend deprivation quintiles. Participants with chronic respiratory disease had a marginally higher prevalence of systolic (43.7%) and diastolic (23.8%) hypertension. Systolic hypertension was more prevalent among users of adrenergic agonists (51.0%) than among intranasal steroid users and non-users, with a similar pattern observed for diastolic blood pressure.

Relationship between nasal decongestant use and hypertension

Multiple logistic regression analysis, adjusting for age, sex, Townsend deprivation quintiles, ethnicity, BMI, and smoking status, demonstrated significant inverse associations between nasal decongestant use and self-reported hypertension (Table 1). Compared with non-users, individuals using adrenergic agonists had 67% lower odds of reporting hypertension (OR=0.38; 95% CI: 0.15–0.96; AOR=0.33; 95% CI: 0.13–0.86), while those using intranasal steroids had 28% lower odds (OR=0.70; 95% CI: 0.67–0.74; AOR=0.72; 95% CI: 0.68–0.77).

Regression analyses examining the association between

Figure 2. Prevalence of systolic and diastolic hypertension across demographic, clinical, and medication-use categories, UK Biobank: 2006–2010, United Kingdom (N=486098)



Bar chart presents the percentage of individuals with systolic (red bars) and diastolic (blue bars) hypertension across key population segments. Categories include sex (female, male), age groups (37–49, 50–59, ≥60 years), socioeconomic status (Townsend Q1–Q5), ethnicity, smoking status (current, never, previous), alcohol consumption status (current, never, previous), chronic respiratory disease (CRD), medication use (adrenergic agonist, intranasal steroid) and body mass index (BMI).

Table 1. Multiple logistic regression estimates for self-reported hypertension by medication use, UK Biobank: 2006-2010, United Kingdom (N=486098)

Medication use	No hypertension n	Self-reported hypertension n	OR (95% CI)	AOR (95% CI)
No decongestants (ref.)	367112	110068	1	1
Adrenergic agonists	44	5	0.38 (0.15–0.96)	0.33 (0.13–0.86)
Steroids	7323	1546	0.70 (0.67–0.74)	0.72 (0.68–0.77)

AOR: adjusted odds ratio; adjusted for age, sex, socioeconomic status, ethnicity, body mass index, and smoking status.

Table 2. Multiple logistic regression estimates for elevated systolic and diastolic blood pressure by medication use, UK Biobank: 2006–2010, United Kingdom (N=486098)

Medication use	Population n	High SBP		High DBP	
		OR (95% CI)	AOR (95% CI)	OR (95% CI)	AOR (95% CI)
No decongestants (ref.)	477180	1	1	1	1
Adrenergic agonists	49	1.34 (0.77–2.35)	1.25 (0.69–2.26)	1.16 (0.61–2.18)	1.02 (0.53–1.94)
Steroids	8869	0.99 (0.95–1.03)	1.03 (0.98–1.07)	1.05 (1.00–1.10)	1.09 (1.04–1.15)

SBP: systolic blood pressure. DBP: diastolic blood pressure. AOR: adjusted odds ratio; adjusted for age, sex, socioeconomic status, ethnicity, body mass index, chronic respiratory disease, alcohol consumption status, and smoking status.

nasal decongestant use and elevated blood pressure, are presented in Table 2. After adjusting for relevant prospective confounders, we found that use of adrenergic agonists was associated with a 25% increase in the odds of elevated systolic blood pressure (SBP >140 mmHg) (OR=1.34; 95% CI: 0.77–2.35; AOR=1.25; 95% CI: 0.69–2.26). Intranasal steroid use was associated with little change in the odds of elevated SBP (OR=0.99; 95% CI: 0.95–1.03; AOR=1.03; 95% CI: 0.98–1.03), and this association was also not statistically significant. For elevated diastolic blood pressure (DBP >90 mmHg), adrenergic agonist use was associated with a non-significant 2% increase in odds (OR=1.16; 95% CI: 0.61–2.18; AOR=1.02; 95% CI: 0.53–1.94). In contrast, intranasal steroid use was associated with a 9% increase in odds of high DBP (OR=1.05; 95% CI: 1.00–1.10; AOR=1.09; 95% CI: 1.04–1.15).

In cross-validation, the model for self-reported hypertension achieved a mean accuracy of 77.04% (range: 76.92–77.16), the SBP model achieved 76.28% (range: 76.04–76.42), and the DBP model achieved 77.62% (range 77.20–77.89).

DISCUSSION

Descriptive statistics highlighted that hypertension prevalence increases with age and BMI and disproportionately affects males and Black participants. These patterns align with established epidemiological evidence and reinforce the importance of stratified risk assessment in hypertension research. The modest inverse relationship observed across Townsend deprivation quintiles requires further investigation, particularly considering socioeconomic gradients in healthcare access and medication use. Regression analysis showed significantly lower odds of self-reported hypertension among both adrenergic agonist and intranasal steroid decongestant user groups, suggesting a potential protective effect. Kartal et al.[25] suggested a possible explanation for the observed protective effect of intranasal corticosteroids on blood pressure. They proposed that the protective effect is unlikely to be direct. Instead, by relieving nasal obstruction and improving airflow, these medications may reduce hypoxia. Reduced hypoxia may lead to lower sympathetic nervous system activity, which could help limit increases in blood pressure[25]. However, the findings from the self-reported hypertension and decongestant use study contradicted severe adverse clinical cases reported by Buysschaert et al.[13] and Halder et al.[26].

Further analyses showed that while adrenergic agonists were associated with an increase in high SBP in the unadjusted model, and following adjustment of the model, both findings were statistically insignificant. Similarly, steroid use was associated with an increase in odds of high SBP in the adjusted model, but was also statistically insignificant. We found these results are in line with prior studies conducted by Bellew et al.[15] and Klas et al.[16]. For DBP, adrenergic agonists showed a statistically insignificant increase in odds of high DBP. At the same time, steroids showed a statistically significant increase in the odds of having high DBP. Cross-validation results indicated moderate predictive performance across all models[23,27].

Overall, although some variability across folds was observed, indicating sensitivity to data partitioning, overall performance suggests predictive ability above chance.

Strengths and limitations

A major strength of this study lies in its large and diverse sample size from the UK Biobank, including target participants from various socioeconomic backgrounds. Logistic regression models were refined based on potential confounders, contributing to methodological rigor. Additionally, this research represents one of the few observational studies investigating the effects of decongestants on arterial blood pressure in the UK setting. However, key limitations were present. The UK Biobank cohort lacks representation of the general UK population and is subject to 'healthy volunteer' bias[19]. Furthermore, reliance on self-reported data may have introduced recall and misclassification. Given the large sample size and the number of analyses conducted, we recognize that the association between intranasal steroid use and high DBP may reflect a chance finding rather than a clinically meaningful effect[27]. Thus, the possibility of a type I error could not be excluded. Additionally, residual confounding from unmeasured variables may have persisted, and the cross-sectional design of this study means that causal relationships cannot be established. Using UK Biobank data, this study offered interesting data, although limited by the nature of secondary data and the absence of an experimental design. It is recommended that future research should implement a target trial emulation framework to better control for confounding, and greater emphasis should be placed on capturing frequency, duration, and potential overuse of nasal decongestants. The UK Biobank records medication use at a single time point and does not provide detailed information on dose, treatment duration, or adherence. Alternatively, a purpose-designed prospective study collecting detailed data on frequency of use, duration, and dosage would allow more accurate evaluation of potential dose–response effects and reduce uncertainty around exposure measurement.

CONCLUSIONS

This study examined the association between nasal decongestant use, namely adrenergic agonists and intranasal corticosteroids, and arterial hypertension using the UK Biobank dataset. Using a cross-sectional design, we assessed whether these commonly used OTC medications are associated with elevated cardiovascular disease burden. Regression analysis indicated lower odds of hypertension among decongestant users. When SBP and DBP were evaluated independently, associations were largely non-significant, though intranasal steroids showed a modest association with DBP. Future investigations are warranted, preferably using prospective cohort designs or target trial emulation to better address residual confounding and measurement error.

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CONFLICTS OF INTEREST
The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none was reported.

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Ethical approval and informed consent were not required for this study as it is a secondary analysis of existing data.

DATA AVAILABILITY
The data supporting this research can be found within the article and Supplementary file.

AUTHORS' CONTRIBUTIONS
SB: search, data extraction and analysis, preparation of final manuscript, RH: search, design, comments on final manuscript, JC: search, design, data extraction and analysis, comments on final manuscript. All the authors have read and approved the final manuscript.

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