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Predictors of progression of arterial hypertension in patients with type 2 diabetes

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Abstract

Background: Arterial hypertension (aHT) is a common comorbidity in patients with type 2 diabetes (T2D). The aim of the present analysis was to determine which predictors are associated with the progression of aHT in participants with T2D.
Methods: We analyzed data from the sleep-disordered breathing (SDB) sub-study of the DIACORE study, a prospective cohort study of participants with T2D. Blood pressure values were determined at baseline and after a mean follow-up of 2.7 years in a standardized manner with three repeated measurements at rest. Arterial hypertension was defined as blood pressure ≥140/80 mm Hg. Progression of aHT was defined as systolic blood pressure ≥140 mm Hg at follow-up with a concomitant increase of at least 10 mm Hg.
Results: Of 1122 participants (41% female, age 66 ± 9 years, body mass index 30.7 ± 5.3 kg/m^2), 925 had pre-existing aHT at baseline. At follow-up, 280 had aHT with additional progression. Multivariate regression analysis revealed that systolic blood pressure at baseline (odds ratio [95% confidence interval]: 0.984 [0.976;0.993]; $p < 0.001$) and age (OR [95%CI]: 1.024 [1.002;1.047]; $p = 0.015$) were associated with progression of aHT, independently of known modulators. Neither SDB nor its treatment were associated with progression of aHT.
Conclusion: In participants with T2D, lower systolic blood pressure at baseline and age but not SDB were associated with progression of aHT.

Keywords
Sleep apnea syndromes · Cardiovascular diseases · Blood pressure · Body mass index · Heart disease risk factors

Supplementary Information

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In the past three decades, the worldwide prevalence of arterial hypertension (aHT) among adults aged 30–79 years has risen from 650 million to 1.28 billion [1]. This surge in patients with aHT has increased the importance of associated health complications and sequelae [2]. Poorly controlled aHT is the leading risk factor for cardiovascular diseases such as stroke, myocardial infarction, and chronic heart failure [3, 4]. Globally, an estimated 10.4 million deaths per year are linked to aHT [5].

AHT is closely linked to type 2 diabetes (T2D) [6, 7]. More than 50% of patients with T2D also suffer from aHT [8]. Guidelines from the European Society of Cardiology, the European Society of Hypertension, and the International Society of Hypertension recommend a systolic blood pressure <140 mm Hg and a diastolic blood pressure <80 mm Hg for patients with T2D above 65 years. It is advised to initiate antihypertensive drug therapy if a patient with T2D exhibits a blood pressure of or exceeding 140/90 mm Hg [9, 10].

Sleep-disordered breathing (SDB) is a common disorder characterized by repetitive nocturnal apneas and hypo-

pneas and arousals from sleep. These episodes can lead to increased sympathetic tone and endothelial dysfunction, resulting in aHT [11, 12]. There is a large overlap between patients with aHT and those with SDB: approximately 30–40% of patients with aHT are also diagnosed with SDB [13, 14]. Cross-sectional studies have revealed a prevalence of aHT in SDB patients ranging from 35 to 80% [15]. Furthermore, there is a close relation between patients with SDB and T2D [16].

The present study aimed to investigate risk factors for the progression of aHT in a cohort of T2D participants. Particular attention was paid to the potential predictive value of SDB.

Methods

Study design

The DIACORE (DIAbetes COhoRtE) study is a prospective cohort study of 3000 participants with T2D conducted at two university hospital centers in Germany [17, 18]. Its design and protocol have been described in detail previously [17]. From 2010 to 2014, recruitment of participants took place through a variety of methods [17]. T2D was defined as a fasting plasma glucose level of ≥126 mg/dl in at least two measurements or a 2-hour glucose value in the oral glucose tolerance test of >200 mg/dl and the need for blood-glucose-lowering medication [17, 19]. During the study, participants were interviewed using a standardized online questionnaire and underwent blood sampling and physical examination [17]. Anthropometric parameters (height, weight, waist-to-hip ratio) were measured in light clothing without shoes [17]. Obesity was defined as body mass index (BMI) ≥30 kg/m^2 [20].

In 2011, the DIACORE sleep-disordered breathing (DIACORE-SDB) sub-study was initiated [18]. As part of this, participants underwent ambulatory polygraphy (Apnea-Link®; ResMed, Australia, Sydney) [17].

The protocol, data protection policy, and study procedures were approved by the ethics committees of the participating institutions and comply with the Declaration of Helsinki. Participation in the DIACORE study was confirmed by written informed consent of the participants [17]. The study is registered at the German Clinical Trials Register (DRKS00010498).

Study population

Inclusion and exclusion criteria of the DIACORE study have been described in detail previously [17]. For the DIACORE-SDB sub-study, participants had to consent for SDB monitoring and were not allowed to have existing treatment with nocturnal positive airway pressure (PAP) therapy [18].

High alcohol intake (≥3 drinks per week), smoking status, coronary heart disease (defined as coronary intervention, coronary bypass surgery, or myocardial infarction), physical activity (defined as light activity at least three times per week), and pre-existing aHT (defined as pre-diagnosed aHT or established antihypertensive medication) were evaluated by standardized questionnaires. Socioeconomic status was classified according to the German Robert Koch Institute [21] and subdivided into four groups ranging from 1 (lowest) to 4 (highest), taking into account educational level, professional qualification, and income.

Assessment of blood pressure and progression of arterial hypertension

Blood pressure was measured using a GE Dinamap Vital Signs Monitor, model V100 (Carescape, Germany) [17]. The cuff size was selected according to the participant's upper arm circumference (either 23–33 cm or 31–40 cm) and placed at the heart level of the dominant arm [17]. After resting for at least 5 min in a sitting position, three blood pressure measurements were taken 2 min apart and the mean of the second and third values was used for the analysis [17].

Pathologic blood pressure values greater than 180/110 mm Hg or less than 90/50 mm Hg were reported to participants at the study visit [17]. Progression of aHT was defined as a systolic blood pressure of 140 mm Hg or higher at follow-up, accompanied by an increase of at least 10 mm Hg in comparison to the baseline visit.

Assessment of antihypertensive treatment and coronary artery disease

The participants' medication intake was assessed through medical records and questionnaires. Antihypertensive therapy was determined according to whether the participant was taking any medication belonging to the following groups: diuretics, beta blockers, calcium antagonists, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or renin antagonists. Coronary heart disease was assessed based on participants' reports and validated by medical records.

Assessment of sleep-disordered breathing

Ambulatory polygraphy with a validated monitoring device (ApneaLink®; ResMed, Sydney, Australia) was performed to record airflow via nasal cannulae and pulse oximetry using a finger clip. This method has undergone validation in numerous studies concerning monitoring of SDB, as detailed previously [22–24].

The ApneaLink® device settings were used to define apnea, hypopnea, and desaturation [25]. Apnea was defined as a drop in nasal airflow of at least 80% for ≥10 s, hypopnea as a drop in airflow of 50–80% compared with baseline for ≥10 s followed by a drop in oxygen saturation of ≥4%, and desaturation as a drop in oxygen saturation of at least 4%. The results included the apnea–hypopnea index (AHI), the oxygen desaturation index (ODI), and the TSat90% (percentage of peripheral oxygen saturation below 90% during the entire recording period). Since chest straps were not used, it was not possible to distinguish between obstructive and central apneas. All participants were informed about the monitoring results, though no further diagnostics or treatment were part of the DIACORE study protocol [17]. Newly initiated continuous positive airway pressure (CPAP) therapy during the course of the study was documented in standardized questionnaires during follow-up examinations [17].

The validated Epworth Sleepiness Scale (ESS) was used to measure subjective daytime sleepiness. Participants were asked
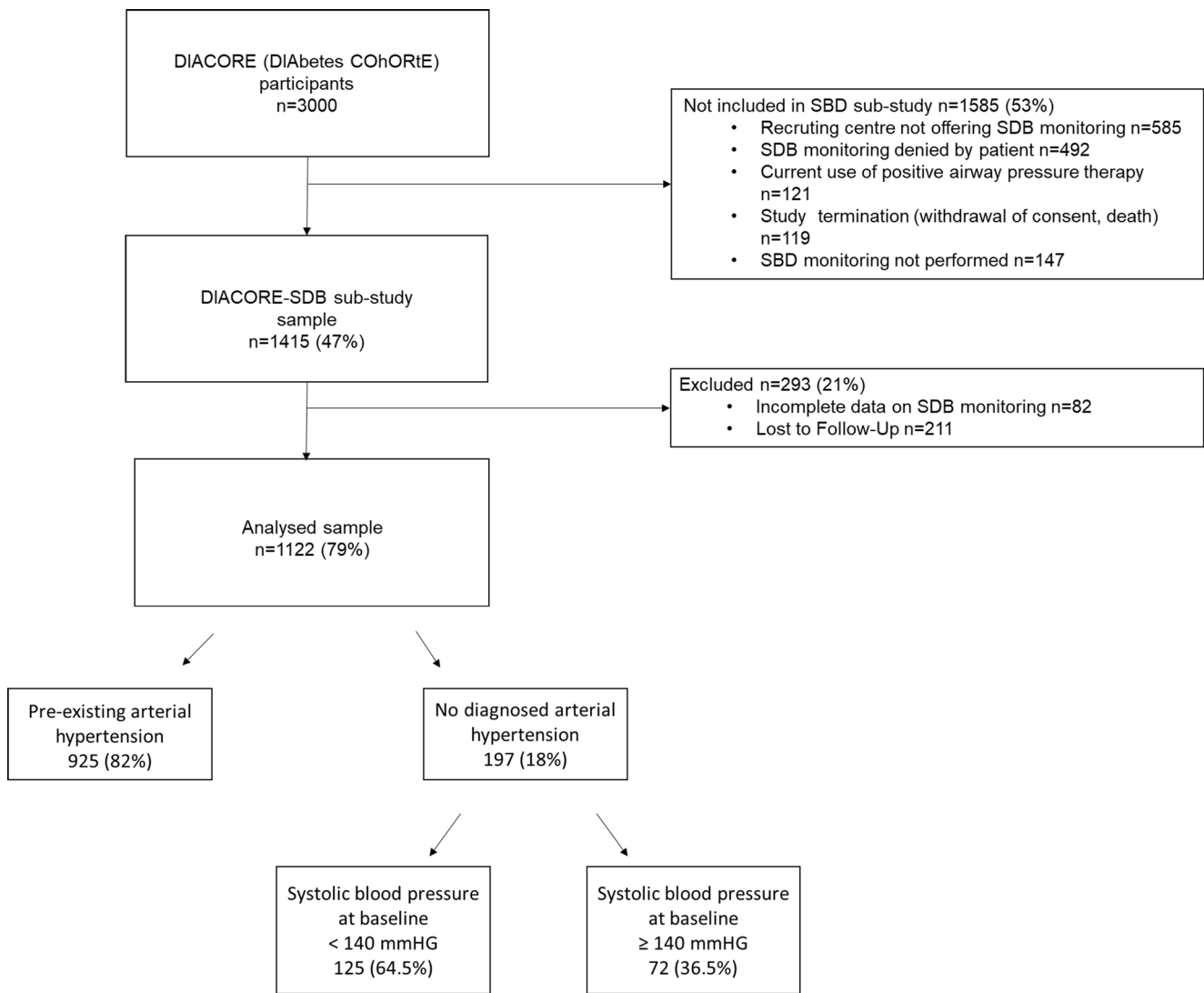
Fig. 1 ▲ Study flowchart

to rate the likelihood of falling asleep in various common situations (scores range from 0 as least sleepy to 24 as sleepiest) [18].

Statistical analysis

Statistical analysis of data was conducted with SPSS software (version 28.0.1.1; IBM SPSS Statistics, Armonk, New York, USA). Mean ± standard deviation (SD) was used to calculate descriptive data for normally distributed variables, while median and interquartile ranges were used for non-normally distributed variables. Categorical data were expressed in absolute numbers and percentages (%). The t-test was used for normally distributed continuous variables, Mann–Whitney U test for non-

normally distributed continuous variables, and chi-squared test for categorical variables. Univariate and multivariate binary logistic regressions were conducted to identify predictors of the progression of aHT. Risk factors for incident or progressive aHT, such as age, sex, BMI, T2D duration, systolic blood pressure, estimated glomerular filtration rate (eGFR), alcohol consumption, coronary heart disease, smoking status, physical activity, pre-existing aHT, and number of different antihypertensive agents were used as covariates. A p-value <0.05 was defined as statistically significant.

Results

Baseline characteristics

Among the 1415 participants initially enrolled in the DIACORE-SDB sub-study, 293 individuals (21%) were excluded from the final analysis due to incomplete polygraphy data or loss to follow-up (☐ **Fig. 1**). Study participants had an average age of 66 years; the majority were male (59%) and had visceral obesity, dyslipidemia, and a mildly decreased eGFR (☐ **Table 1**). More than half were either current or former smokers, and approximately a quarter had a history of coronary heart disease.

The participants' average systolic blood pressure at baseline was 139.0 ± 18.0 mm Hg, and the average diastolic

Table 1 Baseline characteristics of the 1122 analyzed subjects in total and divided by progression of arterial hypertension

Characteristic	Total sample	No progression	Progression	p-value[a]
n (%)	1122	842 (75)	280 (25)	–
Age (years)	66.2 ± 8.6	65.7 ± 8.8	67.8 ± 7.6	<0.001*
Female sex, n (%)	457 (41)	352 (42)	105 (38)	0.204
Body mass index (kg/m^2)	30.7 ± 5.3	30.9 ± 5.3	30.1 ± 5.2	0.041*
Obesity[b], n (%)	546 (49)	425 (50)	121 (43)	0.035*
Waist-to-hip ratio	0.95 ± 0.08	0.95 ± 0.08	0.95 ± 0.08	0.764
T2D duration, (years)	9.3 [5.4;15.1]	9.0 [5.2;14.9]	11.0 [6.3;17.7]	0.003*
Systolic blood pressure (mm Hg)	139.0 ± 18.0	139.8 ± 19.0	136.7 ± 14.5	0.004*
Diastolic blood pressure (mm Hg)	74.0 ± 9.9	74.6 ± 10.0	72.2 ± 9.3	<0.001*
Socioeconomic status[c]	3.0 [2.0;4.0]	3.0 [2.0;4.0]	3.0 [2.0;4.0]	0.453
Pre-existing arterial hypertension[d], n (%)	925 (82)	685 (81)	240 (86)	0.097
Never smoker, n (%)	500 (45)	375 (45)	125 (45)	0.975
High alcohol intake[e], n (%)	330 (39)	242 (29)	88 (31)	0.393
Coronary heart disease[f], n (%)	211 (19)	149 (18)	62 (22)	0.099
Physical inactivity[g], n (%)	624 (56)	482 (57)	142 (51)	0.057
AHI (/h)	10.0 [5.0;18.0]	10.0 [5.0;18.0]	9.0 [5.0;18.5]	0.647
ODI (/h)	9.0 [5.0;18.0]	9.0 [5.0;18.0]	9.0 [5.0;18.0]	0.816
TSat90 (%)	10.8 [2.5;33.7]	10.8 [2.6;33.0]	11.5 [1.9;35.8]	0.892
ESS	5.0 [3.0;7.0]	5.0 [3.0;7.0]	5.0 [3.0;7.0]	0.503
HbA1c (mmol/mol)	49.0 [43.0;55.0]	49.0 [43.0;55.0]	49.0 [44.0;55.0]	0.576
eGFR (ml/min/1.73 m^2)	77.2 ± 18.7	78.3 ± 18.4	74.1 ± 19.3	0.001*
Total cholesterol (mg/dl)	199 ± 42	199 ± 43	200 ± 42	0.668
HDL (mg/dl)	54 ± 16	54 ± 16	54 ± 15	0.777
LDL (mg/dl)	116 ± 36	115 ± 36	118 ± 36	0.379
Number of different antihypertensive agents ± SD	1.9 ± 1.4	1.8 ± 1.4	2.0 ± 1.3	0.088

Data are presented as mean ± standard deviation, median [interquartile range], or absolute and relative frequencies

AHI apnea–hypopnea index, *eGFR* estimated glomerular filtration rate, *ESS* Epworth Sleepiness Scale, *HbA1c* hemoglobin A1c, *HDL* high-density lipoproteins, *LDL* low-density lipoproteins, *ODI* oxygen desaturation index, *T2D* diabetes mellitus type 2

*Statistically significant p-value

[a]Differences between the two groups (t-test for continuous variables and chi-squared for categorical variables)

[b]Defined as body mass index ≥30 kg/m^2

[c]Subdivided into four groups ranging from 1 (lowest) to 4 (highest) according to the Robert Koch Institute (Germany) and including educational level, professional qualification, and income

[d]Defined as pre-diagnosed aHT or established antihypertensive medication

[e]≥ 3 drinks per week

[f]Defined as coronary intervention, coronary bypass surgery, or myocardial infarction

[g]Defined as light activity at least three times per week

blood pressure was 74.0 ± 9.9 mm Hg (◘ Table 1); 925 participants (82%) had pre-existing aHT. Of the remaining 197 participants without a diagnosis of aHT or established antihypertensive therapy, 72 presented with a baseline blood pressure of ≥140 mm Hg (◘ Fig. 1). Only 125 participants had neither antihypertensive therapy nor a diagnosis of aHT nor a systolic blood pressure ≥140 mm Hg at baseline, stressing the high prevalence of aHT amongst persons with T2D.

Progression of arterial hypertension

Of the 1122 analyzed participants, 280 (25%) had progression of aHT after a median follow-up time of 2.7 years. Within this group, the majority were male (62%) and older compared to participants without progression (◘ Table 1). Additionally, those with progression of aHT had a significantly lower BMI and had been diagnosed with T2D for a longer duration. Systolic and diastolic blood pressure were lower at baseline in those with progression of aHT. The only significant difference in medication between the two could be

observed for calcium channel blockers ($p = 0.037$; eTable 1 in the online supplement). No association was found between parameters of SDB and progression of aHT (◘ Table 1).

A multivariate regression model including significant variables from the univariate analysis (age, BMI, duration of T2D, systolic blood pressure, and eGFR) alongside recognized modulators of aHT (sex, history of coronary heart disease, high alcohol intake, HbA1c levels, AHI, pre-existing aHT, smoking status, physical inactivity, and antihypertensive medication use) revealed an independent association be-

Table 2 Association between progression of arterial hypertension (aHT) and risk factors in 1122 participants with diabetes mellitus type 2

Variable	Univariate analysis		Multivariate analysis	
10 mm Hg	OR [95% CI]	*p*-value	OR [95% CI]	*p*-value
Age	1.032 [1.014;1.049]	<0.001*	1.024 [1.002;1.047]	0.029*
Body mass index	0.972 [0.947;0.999]	0.042*	0.984 [0.953;1.016]	0.323
T2D duration	1.019 [1.003;1.035]	0.017*	1.011 [0.993;1.029]	0.239
Systolic blood pressure	0.990 [0.983;0.998]	0.013*	0.984 [0.976;0.993]	<0.001*
eGFR	0.989 [0.982;0.996]	0.002*	0.994 [0.985;1.003]	0.189
Female sex	0.835 [0.633;1.103]	0.204	0.836 [0.601;1.164]	0.290
Coronary heart disease[a]	1.323 [0.948;1.845]	0.100	1.112 [0.775;1.596]	0.565
High alcohol intake[b]	1.136 [0.848;1.523]	0.393	1.009 [0.730;1.396]	0.955
HbA1c	1.028 [0.905;1.167]	0.676	1.007 [0.994;1.020]	0.270
AHI	0.996 [0.986;1.007]	0.488	0.993 [0.982;1.005]	0.189
Pre-existing arterial hypertension[c]	1.375 [0.943;2.005]	0.097	1.218 [0.743;1.996]	0.434
Never smoker	1.004 [0.765;1.318]	0.975	1.065 [0.792;1.433]	0.676
Physical inactivity[d]	1.301 [0.992;1.706]	0.057	1.264 [0.952;1.679]	0.105
Number of different antihypertensive agents	1.090 [0.987;1.205]	0.088	1.019 [0.886;1.172]	0.790

Shown are the unstandardized odds ratio (OR) by regression analysis, 95% confidence intervals [95%CI], and *p*-values

AHI apnea–hypopnea index, *eGFR* estimated glomerular filtration rate, *HbA1c* hemoglobin A1c, *T2D* diabetes mellitus type 2

*Statistically significant *p*-value

[a]Defined as coronary intervention, coronary bypass surgery, or myocardial infarction

[b]≥ 3 drinks per week

[c]Defined as pre-diagnosed aHT or established antihypertensive medication

[d]Defined as light activity at least three times per week

tween progression of aHT and participants' age (odds ratio [95% confidence interval]: 1.02 [1.00;1.05]; $p = 0.029$) and baseline systolic blood pressure (OR [95%CI]: 0.98 [0.98;0.99]; $p < 0.001$; ◘ Table 2; eFigure 1).

In a sub-analysis, we stratified the cohort by sex (eTable 2; eTable 3). Among women, an association was observed between age (OR [95%CI]: 1.04 [1.00; 1.08]; $p = 0.029$), the presence of coronary heart disease (OR [95%CI]: 2.28 [1.14; 4.54]; $p = 0.019$), and high alcohol intake (OR [95%CI]: 0.36 [0.15; 0.87]; $p = 0.024$) with progression of aHT (◘ Table 3).

A greater progression of aHT by at least 20 mm Hg was independently associated with baseline systolic blood pressure (OR [95%CI]: 0.97 [0.96;0.98]; $p < 0.001$) and the duration of T2D (OR [95%CI]: 1.02 [1.00;1.05]; $p = 0.027$; eTable 4). Again, in women there was an association between greater progression of aHT and baseline systolic blood pressure (OR [95%CI]: 0.98 [0.97;0.99]; $p = 0.003$), high alcohol intake (OR [95%CI]: 0.17 [0.04;0.73]; $p = 0.017$), and age (OR [95%CI]: 1.05 [1.01;1.10]; $p = 0.017$; eTable 5).

Discussion

The present study in participants with T2D yielded the following novel findings: progression of aHT by at least 10 mm Hg was significantly associated with systolic blood pressure at baseline and participants' age. This association remained independent of other risk factors such as T2D duration, sex, BMI, renal function, history of coronary artery disease, high alcohol intake, HbA1c levels, AHI, pre-existing aHT, non-smoking status, physical inactivity, and use of antihypertensive medication. SDB was not associated with progression of aHT.

The 10 mm Hg threshold for progression of aHT is based on the assumption that a standard dose of a single antihypertensive agent typically results in a reduction in systolic blood pressure of approximately 10 mm Hg [26]. Moreover, meta-analyses of randomized controlled trials have consistently shown that a decrease in systolic blood pressure of 10 mm Hg is associated with a significant reduction in the risk of various cardiovascular events [27, 28]. These include a 20% decrease in overall cardiovascular events, a 10–15% decrease in all-cause mortality, a 35% decrease in the incidence of stroke, a 20% decrease in

coronary events, and a 40% decrease in the risk of heart failure [27, 28]. These relative risk reductions are consistent across a range of factors, such as baseline blood pressure in the hypertensive range, level of cardiovascular risk, comorbidities like diabetes mellitus and chronic kidney disease, age, gender, and ethnicity [9, 27, 29]. In contrast to this, an increase in systolic blood pressure of 20 mm Hg was associated with more than twice the mortality rate for stroke and twice the mortality rate from ischemic heart disease and other vascular causes [3, 30].

Previous studies have mainly explored predictors contributing to aHT or facilitating the transition from pre-aHT to manifest aHT. In the current study, 80% of participants had pre-existing aHT, and the aim was to investigate the progression of aHT.

In a retrospective cohort study involving 3416 participants with T2D, Lin et al. identified age, education level, physical activity, BMI, family history of aHT, diabetes treatment, systolic and diastolic blood pressure, fasting plasma glucose, and macroalbuminuria as pivotal risk factors for new onset of aHT [31]. This is consistent with the results of the current study and other studies that have found a con-

Table 3 Modulators for progression of arterial hypertension (aHT) in 665 men and in 457 women

Variable	Univariate analysis		Multivariate analysis	
10 mm Hg	OR [95% CI]	*p*-value	OR [95%CI]	*p*-value
Men (n = 665)				
Age	1.027 [1.005;1.050]	0.017*	1.015 [0.986;1.044]	0.313
Body mass index	0.951 [0.914;0.990]	0.013*	0.962 [0.917;1.008]	0.102
T2D duration	1.024 [1.004;1.043]	0.016*	1.020 [0.997;1.042]	0.083
Systolic blood pressure	0.987 [0.977;0.998]	0.015*	0.981 [0.970;0.992]	<0.001*
eGFR	0.991 [0.982;1.000]	0.045*	0.994 [0.983;1.005]	0.289
Coronary heart disease[a]	1.013 [0.680;1.509]	0.949	0.853 [0.553;1.315]	0.471
High alcohol intake[b]	1.357 [0.958;1.921]	0.086	1.268 [0.875;1.839]	0.210
HbA1c	1.005 [0.992;1.019]	0.436	1.013 [0.998;1.028]	0.095
AHI	0.993 [0.980;1.006]	0.278	0.994 [0.979;1.008]	0.398
Pre-existing arterial hypertension[c]	1.339 [0.821;2.185]	0.242	1.358 [0.716;2.575]	0.349
Never smoker	1.220 [0.848;1.756]	0.284	1.229 [0.838;1.803]	0.292
Physical inactivity[d]	1.399 [0.989;1.978]	0.058	1.326 [0.922;1.907]	0.128
Number of different antihypertensive agents	1.064 [0939;1.206]	0.332	1.036 [0.869;1.237]	0.691
Women (n = 457)				
Age	1.037 [1.010;1.065]	0.006*	1.040 [1.004;1.077]	0.029*
Body mass index	0.997 [0.960;1.035]	0.858	1.010 [0.965;1.057]	0.668
T2D duration	1.007 [0.979;1.036]	0.619	1.001 [0.969;1.033]	0.861
Systolic blood pressure	0.993 [0.981;1.005]	0.247	0.989 [0.976;1.002]	0.086
eGFR	0.985 [0.974;0.997]	0.013*	0.994 [0.979;1.009]	0.450
Coronary heart disease[a]	2.490 [1.311;4.731]	0.005*	2.279 [1.144;4.540]	0.019*
High alcohol intake[b]	0.375 [0.156;0.901]	0.028*	0.356 [0.145;0.873]	0.024*
HbA1c	0.993 [0.970;1.016]	0.549	0.992 [0.967;1.018]	0.545
AHI	1.000 [0.980;1.021]	1.000	0.996 [0.974;1.019]	0.731
Pre-existing arterial hypertension[c]	1.405 [0.777;2.542]	0.260	1.079 [0.484;2.405]	0.852
Never smoker	0.868 [0.555;1.356]	0.533	0.836 [0.519;1.345]	0.460
Physical inactivity[d]	1.115 [0.715;1.739]	0.632	1.164 [0.726;1.866]	0.528
Number of different antihypertensive agents	1.130 [0.959;1.332]	0.144	0.986 [0.779;1.250]	0.910

Shown are the unstandardized odds ratio (OR) by regression analysis, 95% confidence intervals [95%CI], and *p*-values

AHI apnea–hypopnea-index, *eGFR* estimated glomerular filtration rate, *HbA1c* hemoglobin A1c, *T2D* diabetes mellitus type 2

*Statistically significant *p*-value

[a] Defined as coronary intervention, coronary bypass surgery, or myocardial infarction

[b] ≥ 3 drinks per week

[c] Defined as pre-diagnosed aHT or established antihypertensive medication

[d] Defined as light activity at least three times per week

nection between older age and the occurrence of aHT in diabetes cohorts [32–34].

Given the study design constraints, we are limited to speculating on the potential protective effect of elevated systolic blood pressure. One plausible explanation could involve heightened awareness among either healthcare providers or patients themselves in response to the higher systolic blood pressure values, enabling prompt responses to escalating values.

In the context of SDB, a cascade of physiological responses including awakening reactions, hypoxemia, intrathoracic pressure fluctuations, increased sympathetic tone, endothelial dysfunction, systemic inflammation, and hormonal responses has been implicated in the development of aHT [35–38]. Furthermore, the application of CPAP therapy or mandibular advancement splints in patients with pre-existing SDB is observed to elicit a reduction in blood pressure [39, 40]. In contrast to this, we found no association between SDB and the progression of aHT in this cohort. This discrepancy may stem from the high prevalence of participants already afflicted with aHT, along with their concurrent use of antihypertensive medications. Furthermore, a substantial portion of participants exhibit multiple components of metabolic syndrome, potentially diminishing the impact of SDB. Additionally, the relatively brief follow-up duration could have influenced the findings and underestimated the influence of SDB on the progression of aHT.

The strengths of this study include the large sample size and a standardized blood pressure examination protocol as well as a detailed survey of participants' medication, comorbidities, lifestyle, and socioeconomic factors.

However, certain limitations must be acknowledged. First, the absence of long-

term blood pressure measurements precludes statements on masked aHT or the presence of white coat hypertension. Second, with an average follow-up duration of approximately 2.7 years, the study's timeframe may be relatively short for a thorough assessment of aHT progression. Third, neither the dosage of the individual antihypertensives nor their type was considered. Fourth, by design, this study cannot establish a causal relationship between the progression of aHT and the various modulators.

In conclusion, the present study in individuals with T2D showed that lower systolic blood pressure at baseline and age were associated with progression of aHT, independently of known modulators. SDB was not associated with progression of aHT. Further research is needed to better understand the underlying mechanisms and causal relationships of progression of aHT in T2D.

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Declarations

For this article no studies with human participants or animals were performed by any of the authors. All studies mentioned were in accordance with the ethical standards indicated in each case.

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Zusammenfassung

Prädiktoren für das Fortschreiten arterieller Hypertonie bei Patienten mit Diabetes mellitus Typ 2

Hintergrund: Arterielle Hypertonie (aHT) ist eine weit verbreitete Erkrankung bei Patienten mit Typ-2-Diabetes (T2D). Ziel der vorliegenden Auswertung war es zu ermitteln, welche Prädiktoren mit dem Fortschreiten der aHT bei Studienteilnehmern mit T2D in Zusammenhang stehen.

Methoden: Dazu wurden Daten aus der Unterstudie zu schlafbezogenen Atmungsstörungen (SDB) der DIACORE-Studie ausgewertet, einer prospektiven Kohortenstudie an Teilnehmern mit T2D. Zu Beginn und nach einem durchschnittlichen Follow-up von 2,7 Jahren wurden Blutdruckwerte auf standardisierte Weise mit 3 wiederholten Messungen in Ruhe ermittelt. Arterielle Hypertonie wurde definiert als ein Blutdruck ≥140/80 mm Hg. Das Fortschreiten der aHT war definiert als systolischer Blutdruck ≥140 mm Hg beim Follow-up mit einer gleichzeitigen Erhöhung um mindestens 10 mm Hg.

Ergebnisse: Von 1122 Teilnehmern (41% Frauen, Alter: 66 ± 9 Jahre, Body-Mass-Index 30,7 ± 5,3 kg/m^2) wiesen 925 zu Studienbeginn eine vorbestehende aHT auf. Beim Follow-up zeigte sich bei 280 Teilnehmern eine aHT mit zusätzlichem Fortschreiten. Die multivariate Regressionsanalyse ergab, dass der systolische Blutdruck zu Beginn (Odds Ratio 0,984; 95%-Konfidenzintervall, 95%-KI: 0,976–0,993; $p < 0,001$) und das Alter (OR: 1,024; 95%-KI: 1,002–1,047; $p = 0,015$) mit dem Fortschreiten der aHT in Zusammenhang standen, unabhängig von bekannten Modulatoren. Weder eine SDB noch deren Behandlung war mit dem Fortschreiten der aHT verknüpft.

Schlussfolgerung: Bei Patienten mit T2D waren geringere systolische Blutdruckwerte zu Beginn der Studie und das Alter, nicht aber eine SDB mit dem Fortschreiten der aHT verknüpft.

Schlüsselwörter

Schlafapnoesyndrome · Herz-Kreislauf-Erkrankungen · Blutdruck · Body-Mass-Index · Risikofaktoren für Herzkrankheiten