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ARTICLE

Three-year change in glycemic state and the future risk of incident hypertension among Iranian adults: Tehran Lipid and Glucose Study

Seyyed Saeed Moazzeni [1], Soheil Dehghani [1], Mitra Hasheminia[1], Reyhane Hizomi Arani [1], Fereidoun Azizi[2] and Farzad Hadaegh [1]✉

The impact of 3-year change in glycemic state on the risk of hypertension among Tehranian adults aged ≥20 years was assessed. The study population included 1679 men and 2348 women who were non-diabetic normotensive at enrollment. The following categories were defined both at baseline visit and three years later (second visit): normoglycemia [normal fasting glucose (NFG) and normal glucose tolerance (NGT)] and prediabetes [impaired fasting glucose (IFG) or impaired glucose tolerance (IGT)]. Changes in the categories, i.e., regression to normoglycemia, remaining in previous status, and progression to diabetes were assessed. Changes in fasting plasma glucose (FPG) and 2-hour plasma glucose (2hPG) categories were also considered separately. We used the Cox models adjusted for traditional hypertension risk factors to estimate hazard ratios (HRs) with 95% confidence intervals (CIs). During a median follow-up of 9.4 years, 485 men and 589 women developed hypertension. Among men, considering both FPG and 2hPG, compared to individuals remaining normoglycemic, changing from prediabetes to normoglycemia had a HR of 1.30 (95% CI: 0.98–1.71; P-value: 0.064). Defining changes by 2hPG showed that compared to remaining NGT, incident IGT (progression from NGT to IGT) had a significant association with increased risk of hypertension development by a HR of 1.61 (1.13–2.30). Among women, on the other hand, change in glycemic state generally didn't show a significant association with incident hypertension. To sum up, change in glycemic state hadn't a significant association with hypertension development among women; however, compared to remaining NGT, men with incident IGT had a significant higher risk.

INTRODUCTION

A great number of the world population have been suffering from non-communicable diseases (NCDs), especially type 2 diabetes mellitus (T2DM) and hypertension [1]. T2DM (in general term dysglycemic state) has been identified as a major issue due to the high burden worldly [2]. Prediabetes is defined as an intermediate glucose regulation state that prone person to NCDs development [3, 4]; it is characterized by elevated blood glucose concentrations, including those with impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or elevated glycosylated hemoglobin A1C (HbA1C), but not in diabetic ranges [5, 6]. A national study has shown that about 11.4 and 14.6% of Iranian adults had T2DM and IFG, respectively, in 2011 [7]. Moreover, using oral glucose tolerance test and fasting plasma glucose (FPG) level, we reported the prevalence of prediabetes among Tehranian residents reached about 24.5% in 2008–2011 [8].

In addition to the high burden of dysglycemia, the World Health Organization (WHO) reported hypertension as being one of the main important risk factors for morbidity and mortality globally; more than one billion people are involved with hypertension, and it causes 9.4 million deaths yearly [9]. A systematic review and meta-analysis study has reported that the prevalence of hypertension in the Middle East and North Africa (MENA) region has been increasing in the past three decades [10]. Furthermore, nearly 2.7% of Tehranian adults (3% of men and 2.6% of women) develop hypertension annually [11].

Hypertension has a two-way relationship with dysglycemia [12] and is two times more common among diabetic patients [13]. Higher risk of hypertension was also found for adults with prediabetic state in some but not all studies; however, this higher risk may have been significant only for one of IFG, IGT, or elevated HbA1C or only for one gender [4, 11, 14–20]. In addition to the snapshot assessment of the glycemic state, change in glycemic state was shown to have associations with NCDs, especially cardiovascular disease (CVD) [21, 22]. To the best of our knowledge, there is only one cohort study that described the effect of change in glycemic state on hypertension development [18]; the researchers found the progression of glycemic state as a strong determinant of incident hypertension; however, different components of prediabetes were not considered [18]. Previously we reported that the presence of IFG was associated with more than 20% significantly higher risk of incident hypertension only for women but not men [4]. In the current study, we aim to extend our previous study by clarifying the association of 3-year change

[1]Prevention of Metabolic Disorders Research Center, Research Institute for Endocrine Sciences, Shahid Beheshti University of Medical Sciences, Tehran, Iran. [2]Endocrine Research Center, Research Institute for Endocrine Sciences, Shahid Beheshti University of Medical Sciences, Tehran, Iran. ✉email: fzhadaegh@endocrine.ac.ir
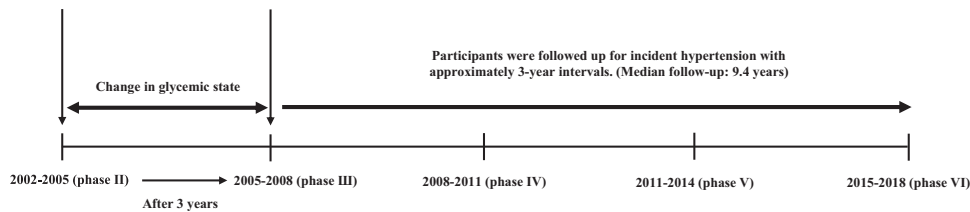
Fig. 1 Timeline of the study design: Tehran Lipid and Glucose Study, Iran, 2002–2018.

in glycemic state with the risk of incident hypertension among Tehranian men and women.

MATERIALS AND METHODS
Study design and population
The Tehran Lipid and Glucose Study (TLGS) is a large population-based cohort study conducted on the Tehranian civilization in district No 13. It has been designed to determine the prevalence and incidence of NCDs. For this study, as shown in Fig. 1, we considered the second phase of TLGS as baseline recruitment (2001–2005). After about 3 years, the glycemic state of participants was reassessed at phase III (2005–2008) again. Then, participants have been followed for incident hypertension with approximately 3-year intervals (i.e., phase IV: 2008–2011, phase V: 2011–2014, and phase VI: 2015–2018). The details of the TLGS protocol and design have been described elsewhere [23].

As presented in Supplementary Fig. 1, for this study, among a sum of 3891 male and 5036 female participants aged ≥20 years, 443 men and 623 women with prevalent T2DM at baseline were excluded. Moreover, we excluded 781 male and 861 female subjects because of prevalent hypertension at baseline or incident hypertension until the end of phase III. Among the remaining 2667 men and 3552 women, 929 male and 1015 female individuals were also eliminated, due to missing data on glycemic state or blood pressure at phase II or III. Other exclusion criterion was missing data on covariates (59 men and 189 women), leading to 1679 men and 2348 women remaining eligible for the analysis of the current study (response rate was 63 and 66% for men and women, respectively).

Clinical and laboratory measurements
At recruitment, a valid questionnaire was used to obtain the data on demographic identification, past medical/drugs history, family history, and smoking habits regarding the TLGS protocol [23]. Using the Modifiable Activity Questionnaire (MAQ), which measures all three forms of activities including leisure time, job and household activities over the past year, the level of physical activity was assessed [23, 24].

Weight was measured with a sensitive scale by 100 g error, and height was measured in a standing position without shoes. Then, body mass index (BMI) was calculated by weight in kilograms divided by the square of height in meters. An expert, who was not aware about the cardiometabiolic profiles of participants, used a standardized mercury sphygmomanometer (calibrated by the Iranian Institute of Standards and Industrial Research) to measure the blood pressure (BP) of participants. Subsequent to 15 min of rest, two measurements were done on the right arm with a 30-s interval; the mean of these two office BP measurements was considered as the subject's BP. Blood samples from all participants were taken after 12–14 h of fasting. Also, for those without a history of using glucose-lowering drugs, after taking 75 g glucose orally, 2-hour post-challenge plasma glucose (2hPG) was measured. Details for laboratory measurements, including FPG, 2hPG, high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG), are published elsewhere [23].

Definition of the main exposures, confounding factors, and outcome
Based on the 2003 American Diabetes Association (ADA) guideline [25], we defined IFG as $5.6 \leq FPG < 7$ mmol/L and IGT as $7.8 \leq 2hPG < 11$ mmol/L. Prediabetes was defined as those with IFG and/or IGT. T2DM was also defined as FPG ≥ 7 mmol/L, or 2hPG ≥ 11.1 mmol/L, or using glucose-lowering drugs. Moreover, normal fasting glucose (NFG) was those with FPG < 5.6 mmol/L, and normal glucose tolerance (NGT) was those with 2hPG < 7.8 mmol/L. The normoglycemic participants were those with NFG and NGT.

Smoking status was divided into two groups; current smokers versus former/never smokers. Subjects with <1500 MET (metabolic equivalent

task-minutes per week) were also in the low physical active group [23]. Furthermore, a positive family history of premature CVD has been defined as prior diagnosed CVD in female first-degree blood relatives aged below 65 years or male first-degree blood relatives who were below 55 years.

Regarding the seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high BP (the JNC 7 report) [26], hypertension was defined as the existence of one of continued parameters: (a) having systolic blood pressure (SBP) ≥ 140 mm Hg, (b) having diastolic blood pressure (DBP) ≥ 90 mm Hg, or (c) initiation of using anti-hypertensive medications.

Statistical analysis
Descriptive statistics were used to describe the baseline characteristics of the participants. Normally distributed continuous variables were reported as mean ± standard deviation (SD), and the highly skewed variables were reported as median (interquartile range: IQR). Number (%) was also used for reporting categorical variables. We compared the baseline characteristics of the respondents versus non-respondents (those with missing data on main exposure or covariates) and also normoglycemic versus prediabetic groups, using chi-square, ANOVA, and Kruskal–Wallis tests as appropriate.

The time to event was described as the time of censoring or the outcome occurring, whichever firstly came. In the case of death, leaving the district, or being until the end of the study (April 2018) without any event, we censored participants. For individuals with incident hypertension, the event date was defined as the mid-time between the dates of the follow-up visit at which an individual developed hypertension and the last follow-up visit preceding incident hypertension.

We categorized participants into different glycemic change groups by three methods: (a) using both FPG and 2hPG [normoglycemia to normoglycemia (reference), normoglycemia to prediabetes, prediabetes to normoglycemia, prediabetes to prediabetes, and prediabetes to T2DM]. (b) using FPG [NFG to NFG (reference), NFG to IFG, IFG to NFG, IFG to IFG, and IFG to diabetic range of FPG]; (c) using 2hPG [NGT to NGT (reference), NGT to IGT, IGT to NGT, IGT to IGT, and IGT to diabetic range of 2hPG]. It should be noted that the frequency of normoglycemic participants that changed to diabetes was too low to include in our models.

The Cox proportional hazard models were applied to evaluate the sex-specific association of glycemic state change with incident hypertension. The hazard ratios (HRs) with 95% confidence intervals (CIs) were reported in three models: Model 1: adjusted for age; Model 2: adjusted for age, BMI, current smoking, low physical activity, history of CVD, and family history of premature CVD; Model 3: Model 2 + further adjusted for TG/HDL-C, as a surrogate of insulin resistance (IR) [27, 28].

Using the Schoenfeld residual test, the proportionality was assessed for the Cox models. All proportionality assumptions were appropriate in our analysis. STATA version 14 (StataCorp LP, College Station, TX, USA) statistical software was used for statistical analyses of this study, and a two-tailed p-value < 0.05 was considered significant.

RESULTS
The study population included 1679 men with a mean age of 40.8 years and 2348 women with a mean age of 38.8 years. Baseline characteristics of the respondent and non-respondents are compared in Supplementary Table 1. Among men, respondents had lower HDL-C and were more physically inactive, but they smoked less. Among women, respondents were older and had higher level of BMI, FPG, and 2hPG. Moreover, female respondents were less educated, but they were more physically active and smoked less.

Baseline characteristics of the participants are shown in Table 1. Among continuous variables, generally, the cardiometabolic

Table 1. Baseline characteristics of the participants based on their baseline glycemic state: Tehran Lipid and Glucose Study.

	Men			Women		
	Normoglycemia	Prediabetes	*P*-value	Normoglycemia	Prediabetes	*P*-value
Number of participants	1369	310		1950	398	
Continuous variables, Mean ± SD						
Age (year)	39.24 ± 13.03	47.95 ± 13.50	<0.001	37.51 ± 11.50	44.90 ± 11.22	<0.001
BMI (kg/m^2)	25.46 ± 3.91	27.28 ± 3.76	<0.001	27.11 ± 4.58	30.05 ± 4.64	<0.001
SBP (mmHg)	111.67 ± 10.53	114.90 ± 11.01	<0.001	106.59 ± 11.26	113.07 ± 11.32	<0.001
DBP (mmHg)	72.05 ± 8.33	73.00 ± 8.23	0.067	70.60 ± 8.01	74.58 ± 7.46	<0.001
FPG (mmol/L)	4.87 ± 0.36	5.63 ± 0.48	<0.001	4.78 ± 0.35	5.49 ± 0.57	<0.001
2hPG (mmol/L)	5.22 ± 1.15	7.07 ± 1.90	<0.001	5.48 ± 1.02	7.80 ± 1.54	<0.001
HDL-C (mmol/L)	0.91 ± 0.22	0.89 ± 0.22	0.165	1.10 ± 0.28	1.03 ± 0.29	<0.001
Triglycerides (mmol/L)*	1.47 (1.05–2.15)	1.98 (1.37–2.70)	<0.001	1.20 (0.87–1.72)	1.76 (1.32–2.56)	<0.001
Categorical variables, number (%)						
Educational level, years			<0.001			<0.001
<6	210 (15.4%)	80 (25.9%)		411 (21.1%)	159 (40.0%)	
6–12	826 (60.3%)	178 (57.6%)		1225 (62.8%)	202 (50.9%)	
>12	333 (24.3%)	51 (16.5%)		313 (16.1%)	36 (9.1%)	
Current smoker, yes	380 (27.8%)	76 (24.5%)	0.247	50 (2.6%)	17 (4.3%)	0.062
Low physical activity, yes	877 (64.1%)	185 (59.7%)	0.145	1230 (63.1%)	229 (57.5)	0.039
History of CVD, yes	32 (2.3%)	20 (6.5%)	<0.001	19 (1.0%)	11 (2.8%)	0.004
Family history of premature CVD, yes	239 (17.5%)	53 (17.1%)	0.880	339 (17.4%)	74 (18.6%)	0.564

Values are shown as Mean ± SD and number (%) for continuous and categorical variables, respectively.
SD standard deviation, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *FPG* fasting plasma glucose, *2hPG* 2-hour post-challenge plasma glucose, *HDL-C* high-density lipoprotein cholesterol, *CVD* cardiovascular disease.
*For triglycerides, the values are reported as median (interquartile range) due to skewed distribution.

profile was worsened among prediabetic participants; they were older and had higher values for BMI, SBP, DBP (only among women), FPG, 2hPG, and triglycerides. Moreover, prediabetic women had lower HDL-C level. Among categorical variables, normoglycemic participants were more educated and with lower prevalent CVD, although they had a higher prevalence of low-physical activity.

During a median follow-up of 9.4 years (IQR: 8.4–10.2), 485 men and 589 women developed hypertension.

Multivariable HRs and 95% CI of incident hypertension according to glycemic state change are reported in Table 2 for male participants. Considering both FPG and 2hPG, compared to individuals remaining normoglycemic, those prediabetic individuals at baseline that changed to normoglycemia had a marginally significant higher risk of incident hypertension with a HR of 1.31 (95% CI: 0.99–1.72; *P*-value: 0.057) in model 2; the risk remained marginally significant even after adjustment for TG/HDL-C in model 3. Moreover, progression from normoglycemia to prediabetes and also prediabetes to T2DM had HRs of 1.25 (0.92–1.72) and 1.41 (0.70–2.86), respectively, in model 3. Considering FPG, different changes in glycemic state were not associated significantly with incident hypertension in model 3. On the other hand, defining changes by 2hPG showed that incident IGT (progression from NGT to IGT) had a significant association with increased risk of hypertension development by a HR of 1.61 (1.13–2.30) in model 2. It should be noted that the higher hypertension risk for newly-diagnosed IGT cases remained significant, even after adjustment for TG/HDL-C in model 3.

Results for female participants are presented in Table 3. Generally, change in glycemic state did not show a significant association with incident hypertension. Moreover, most of the effect sizes were located in the trivial zone.

Finally, after further adjustment for BMI change from phase II (baseline) to phase III, among men, the association of incident IGT

with hypertension risk still remained significant with a HR of 1.68 (1.18–2.39; *P*-value: 0.004); moreover, changing from prediabetes to normoglycemia reached to a significant level [HR: 1.33 (1.00–1.75); *P*-value: 0.047]. Among women, on the other hand, results did not change.

DISCUSSION

In this population-based prospective study, we examined the association of 3–year changes in glycemic state with incident hypertension during over 9 years of follow-up. We found that change in glycemic state had not a significant association with hypertension development among women. For men, on the other hand, although most of the effect sizes were at the trivial zone, incident IGT showed a significantly higher risk for hypertension development, compared to remaining NGT; this higher risk remained significant even after adjustment for TG/HDL-C and BMI change.

Among Tehranian adults, we previously found the prediabetes at the baseline as a hypertension risk factor in women, not men [11]. Moreover, by considering different components of dysglycemia [4], compared to FPG of <5.6 mmol/L, Tehranian female adults with FPG of 5.6–6.9 (IFG) and ≥7.0 mmol/L were at higher risk of hypertension in a multivariable model; however, this higher risk was not found for men. Considering 2hPG, we also showed that only women with 2hPG ≥ 11.0 mmol/L were at higher risk of hypertension development, and IGT did not increase the risk in either gender [4]. These findings mean that in our population, the effect of impaired glycemic state at baseline on hypertension risk is more prominent among women than men. Now, we found that change in glycemic state (especially NGT to IGT) can have association with hypertension development only among men. It can be suggested that among Tehranian men, the development of hypertension can be more

Table 2. Multivariable hazard ratios (HR) and 95% confidence intervals (CI) of incident hypertension according to glycemic state change among men: Tehran Lipid and Glucose Study.

	E/N	Model 1		Model 2		Model 3	
		HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
FPG/2hPG							
Normal to Normal	313/1228	Reference		Reference		Reference	
Normal to Prediabetes	45/134	1.33 (0.97–1.83)	0.072	1.25 (0.91–1.72)	0.159	1.25 (0.92–1.72)	0.159
Prediabetes to Normal	63/151	**1.49 (1.14–1.96)**	**0.004**	1.31 (0.99–1.72)	0.057	1.30 (0.98–1.71)	0.064
Prediabetes to Prediabetes	55/141	1.30 (0.97–1.74)	0.078	1.09 (0.81–1.47)	0.566	1.08 (0.80–1.46)	0.598
Prediabetes to Diabetes	8/18	1.45 (0.72–2.94)	0.297	1.42 (0.70–2.87)	0.329	1.41 (0.70–2.86)	0.337
FPG							
NFG to NFG	359/1333	Reference		Reference		Reference	
NFF to IFG	42/122	1.23 (0.89–1.70)	0.202	1.15 (0.84–1.59)	0.383	1.15 (0.83–1.59)	0.395
IFG to NFG	52/128	**1.36 (1.01–1.82)**	**0.040**	1.21 (0.90–1.63)	0.195	1.21 (0.90–1.62)	0.211
IFG to IFG	29/83	1.15 (0.78–1.68)	0.484	1.06 (0.73–1.56)	0.751	1.06 (0.72–1.55)	0.774
IFG to Diabetes	3/8	1.38 (0.44–4.30)	0.578	1.10 (0.35–3.44)	0.866	1.09 (0.35–3.42)	0.877
2hPG							
NGT to NGT	386/1452	Reference		Reference		Reference	
NGT to IGT	35/74	**1.89 (1.33–2.68)**	**<0.001**	**1.61 (1.13–2.30)**	**0.008**	**1.61 (1.13–2.30)**	**0.009**
IGT to NGT	36/80	**1.50 (1.07–2.12)**	**0.020**	1.31 (0.93–1.86)	0.123	1.30 (0.92–1.85)	0.135
IGT to IGT	21/51	1.24 (0.79–1.94)	0.346	1.07 (0.68–1.67)	0.770	1.06 (0.68–1.66)	0.798
IGT to Diabetes	3/9	0.99 (0.32–3.09)	0.987	0.94 (0.30–2.93)	0.913	0.94 (0.30–2.92)	0.911

E/N number of event/number of participants, *FPG* fasting plasma glucose, *2hPG* 2-hour post-challenge plasma glucose, *NFG* normal fasting glucose, *IFG* impaired fasting glucose, *NGT* normal glucose tolerance, *IGT* impaired glucose tolerance.
Model 1: Adjusted for age.
Model 2: Adjusted for age, body mass index, current smoking, low physical activity, history of cardiovascular disease (CVD), and family history of premature CVD.
Model 3: Model 2 + further adjusted for Triglycerides/High-density lipoprotein cholesterol (TG/HDL-C).
The bold values are statistically significant.

influenced by the progression of glycemic state through 2hPG than the snapshot measurements of glycemic state at baseline. Similar to our results for men, among Korean adults, it was found that prediabetic participants were not significantly at higher risk of hypertension, in comparison with normoglycemia; however, compared to the maintaining glycemic state, the progression of normoglycemia to prediabetes, normoglycemia to T2DM, and prediabetes to T2DM significantly increased the risk of incident hypertension [18]; the authors suggested that the aggravation of IR may be an underlying mechanism. In our results, on the other hand, adjustment for TG/HDL-C, as a IR surrogate [27, 28], did not change the effect sizes prominently. It was suggested that the role of lipids (TG and HDL-C) in IR was more prominent in IFG than IGT [29]; therefore, it was expectable that adjustment for TG/HDL-C could not change the effect of incident IGT tangibly. Furthermore, the site of IR and type of beta-cell dysfunction differ between IFG and IGT [29]. Importantly, it is now clear that mainly due to the steroid hormones, many aspects of energy balance and glucose metabolism are regulated differently in men and women and influence their predisposition to cardiometabolic disorders. Premenopausal women are at lower risk of cardiometabolic disorders than men with the same age and BMI. This cardiometabolic advantage in women disappears after the menopause or when DM develops [30, 31]. About the higher

risk for our male participants with reversion from prediabetes to normoglycemia, it could be a chance association.

The strengths of this study consist of standardized measurement techniques and assessment of sex-specific association by considering components of dysglycemia (IFG and IGT) separately.

A number of important limitations need to be considered. First, our sample size was limited, resulting in wide confidence intervals and insignificant *P*-values. In addition to our exploratory study, this relationship definitely should be reexamined in future confirmatory studies with larger sample sizes. However, most of our insignificant effect sizes were located in trivial zones (0.8–1.2), which were independent of sample size [32]. Second, more than 30% of our recruited participants were missed due to missing data. Although there were statistically significant differences between respondents and non-respondents, the values of differences were not clinically significant. Third, our study population included only urban residents of Tehran, and our findings may not be generalizable to rural areas. Fourth, TG/HDL-C was found as a predictor of IR among Tehranian residents [28]; however, TG/HDL-C has not been validated as a surrogate of IR among Iranian adults. Finally, data were not available for some potential hypertension predictors such as air pollution, dietary factors, and family history of hypertension.

Table 3. Multivariable hazard ratios (HR) and 95% confidence intervals (CI) of incident hypertension according to glycemic state change among women: Tehran Lipid and Glucose Study.

	E/N	Model 1		Model 2		Model 3	
		HR (95% CI)	*P*-value	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
FPG/2hPG							
Normal to Normal	396/1799	Reference		Reference		Reference	
Normal to Prediabetes	42/139	0.95 (0.69–1.31)	0.747	0.94 (0.68–1.29)	0.692	0.92 (0.67–1.28)	0.634
Prediabetes to Normal	69/203	1.25 (0.97–1.62)	0.086	1.06 (0.82–1.38)	0.660	1.02 (0.79–1.33)	0.870
Prediabetes to Prediabetes	72/167	**1.35 (1.04–1.74)**	0.024	1.10 (0.84–1.42)	0.490	1.05 (0.81–1.36)	0.726
Prediabetes to Diabetes	10/28	1.08 (0.58–2.03)	0.808	0.92 (0.49–1.73)	0.794	0.85 (0.45–1.60)	0.615
FPG							
NFG to NFG	465/1997	Reference		Reference		Reference	
NFF to IFG	37/103	1.32 (0.94–1.84)	0.108	1.19 (0.85–1.66)	0.318	1.17 (0.84–1.64)	0.362
IFG to NFG	35/117	1.01 (0.72–1.42)	0.957	0.84 (0.59–1.19)	0.323	0.81 (0.57–1.15)	0.237
IFG to IFG	46/107	1.22 (0.90–.67)	0.199	0.95 (0.70–1.30)	0.749	0.93 (0.68–1.27)	0.640
IFG to Diabetes	6/14	2.04 (0.91–4.56)	0.083	1.61 (0.72–3.62)	0.247	1.65 (0.73–3.70)	0.227
2hPG							
NGT to NGT	452/1978	Reference		Reference		Reference	
NGT to IGT	38/110	0.99 (0.71–1.39)	0.950	1.01 (0.72–1.41)	0.956	0.99 (0.71–1.39)	0.954
IGT to NGT	60/146	**1.40 (1.07–1.84)**	**0.015**	1.17 (0.89–1.54)	0.265	1.12 (0.84–1.47)	0.439
IGT to IGT	29/75	1.10 (0.75–1.60)	0.640	0.92 (0.63–1.36)	0.691	0.87 (0.59–1.28)	0.474
IGT to Diabetes	10/26	1.14 (0.61–2.14)	0.681	0.98 (0.52–1.84)	0.940	0.90 (0.47–1.69)	0.735

E/N number of event/number of participants, *FPG* fasting plasma glucose, *2hPG* 2-hour post-challenge plasma glucose, *NFG* normal fasting glucose, *IFG* impaired fasting glucose, *NGT* normal glucose tolerance, *IGT* impaired glucose tolerance.
Model 1: Adjusted for age.
Model 2: Adjusted for age, body mass index, current smoking, low physical activity, history of cardiovascular disease (CVD), and family history of premature CVD.
Model 3: Model 2 + further adjusted for Triglycerides/ High-density lipoprotein cholesterol (TG/HDL-C).

CONCLUSION
To sum up, among Tehranian men, hypertension development was more influenced by the progression of dysglycemia (especially incident IGT) than snapshot assessment of glycemic state.

SUMMARY

What is known about topic

- It was suggested that prediabetes and diabetes increased the risk of incident hypertension.
- However, the relation of change in glycemic state was not clearly investigated.
- There is only one cohort study about this issue that found the progression of glycemic state as a strong determinant of hypertension.

What this study adds

- In this population-based prospective study, we examined the association between 3–year changes in glycemic state with incident hypertension.
- During over 9 years of follow-up, we found that change in glycemic state had not a significant association with hypertension development among women.

- For men, incident IGT showed a significant higher risk, compared to remaining NGT.

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COMPETING INTERESTS
The authors declare no competing interests.

ETHICS APPROVAL
The ethics committee of the Research Institute for Endocrine Sciences of the Shahid Beheshti University of Medical Sciences approved the study proposal (IR.SBMU.ENDOCRINE.REC.1400.138).

INFORMED CONSENT
Written informed consent was also obtained from all participants.

ADDITIONAL INFORMATION
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Correspondence and requests for materials should be addressed to Farzad Hadaegh.

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