**Association between Obstructive Sleep Apnea and Cardiovascular Risk Factors among Patients with Type 2 Diabetes Mellitus.**

## Introduction

Sleep is an essential component of our health and well-being. Obstructive sleep apnea (OSA) is one of the most common sleep disorders and is estimated that it affects approximately 24% of men and 9% of women and the prevalence of OSA have been estimated as 2%–10% worldwide.[1,2] OSA is a sleep disorder in which there is a repetitive complete obstruction (apnea) or partial obstruction (hypopnea) of the collapsible part of the upper airway during sleep.[3] Symptoms of OSA is associated with excessive daytime sleepiness or chronic fatigue. Independent of obesity, OSA can lead to cardiovascular diseases (CVDs)—including hypertension, coronary heart disease, and heart failure. Also, it can have a deleterious impact on renal function and has been linked to diabetes mellitus (DM) and insülin resistance.[4–8] The prevalence of OSA syndrome is high among patients with type 2 DM.[9,10] Kendzerska et al. reported that 11% of patients with OSA who were followed up for 67 months developed DM and that patients with an apnea-hypopnea index (AHI) greater than 30 had a 30% higher risk of developing diabetes than those with an AHI score of less than 5.[11] Clinical data suggest that this effect is most likely mediated via intermittent oxygen desaturation.[12]

Several mechanisms have been proposed to explain this very high cardiovascular and type 2 diabetes risk among people with OSA—including, activation of the sympathetic nervous system, increased levels of endothelin, oxidative stress, changes in adipokine profiles, and inflammatory activation. OSA, CVDs and type 2 diabetes often coexist and interact, sharing multiple pathophysiological mechanisms—including, activation of the sympathetic nervous system, increased levels of endothelin, oxidative stress, genetics, HPA axis

alterations, changes in adipokine profiles, and inflammatory activation. OSA has negative synergistic effects on the cardiovascular and endocrine system through multiple mechanisms (e.g., activation of the sympathetic nervous system, increased levels of endothelin, oxidative stress, HPA axis alterations, changes in adipokine profiles, and inflammatory activation.[12–15]

Also, type 2 diabetes contributes to the risk of developing CVD. Shah et al. reported in their study that type 2 diabetes was positively associated with peripheral arterial disease, ischaemic stroke, stable angina, heart failure, and nonfatal myocardial infarction.[15]According to the World Health Organisation, the prevalence of CVD and type 2 diabetes is increasing rapidly—17.5 million deaths (31% of all deaths worldwide) that occurred in the world in 2012 were due to CVD, while diabetes caused 1.5 million (2.7%) deaths in the same year.[16] Constanzo et al. reported that OSA may be present in 30%–50% of patients with heart failure. Multiple studies using continuous positive airway pressure have shown improvements in the clinical state as well as a retardation of CVD progression.[17] OSA is a treatable condition; patients with CVD should be proactively screened for OSA to decrease both endocrine and cardiovascular outcomes of OSA.[4] This study used a cross-sectional analytical study design to determine the risk of sleep apnea and cardiovascular risk in adult patients with type 2 diabetes.

## Methods

The study is a cross-sectional analytical study and was conducted between March 3, 2016 and July 15, 2016. The study population consisted of adult patients with type 2 diabetes who applied to the diabetes polyclinic of a public hospital located in the west of Turkey during the study period.

### *Subjects*

The sample of the study consisted of 228 patients who accepted to participate in the study and were 18 years or older, cognitively competent, literate, had standing height and weight measurements and were being monitored for type 2 diabetes in the a diabetes policlinic of public hospital between March 3, 2016 and July 15, 2016. Since no study has examined the relationship between type 2 diabetes and OSA markers, the effect size calculation could not be carried out in this study. At the end of the study, a power analysis was made with the g power program. The research was completed with a medium effect size, 0.05 margin of error, and 95% power.

Routine tests were performed in the hospital. None of these patients used insulin injections or medications that would affect sleep and none had severe painful peripheral neuropathy and alcohol dependency. Smoking status was defined as smoking 10 cigarettes daily. The degree of obesity was classified based on the results of the 2015 Turkish Nutrition Guide: patients with $< 25$ kg/m$^2$ body mass index (BMI) were classified as nonobese and $\geq 25$ kg/m$^2$ as obese.[18] None of the patients had low weight.

Data collection was done through face-to-face interviews with patients diagnosed with type 2 diabetes in the diabetes polyclinic of the public hospital used for this study, which is located in the west of Turkey. The data collection was done between March 01, 2016 and July 15, 2016.

Dependent variable: berlin score; independent variables: sociodemographic characteristics, anthropometric measurements, framingham risk score in this study.

***Measurement tools***

In this study, the data were collected using the "Patient Information Form," "Berlin Questionnaire (BQ)," and "Framingham Risk Score (FRS)." The FRS was used to evaluate the risk for CVD among the patients, while the BQ was used to evaluate the risk for OSA.

*Patient information form:* This form is comprised of two parts. The first part collected information about DM among the patients and their sociodemographic characteristics. The second part contained questions about the BMI of the patients and their smoking/alcohol consumption status, which are considered to be among the risk factors for OSA, and other diseases that the patients might have.

*Berlin Questionnaire*: This questionnaire is used to screen for the risk of OSA at the community level through a consensus gathered in the "First Stage Sleep Conference" held in Berlin-Germany in 1996. The validity and reliability of the scale in the Turkish population was carried out by Acar et al.[19] and the sensitivity of BQ was found to be 87.9% in the study. The questionnaire contains 10 items in three categories. If the response to at least two items in the first two categories is 1, that category is considered positive (+). The third category is considered positive if the blood pressure level is answered as "yes" or if the BMI is ≥ 30. If two or three categories are positive, the patient is considered to be at high risk for OSA, and if only one category is positive, the patient is at low risk for OSA.[19,20]

*Framingham risk scoring*: Ten-year coronary disease risk was estimated separately for both sexes. The calculation includes six risk factors: gender, age, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), systolic (or diastolic) blood pressure, and smoking. Scores obtained according to values and categories were summed up, and the 10-year probability corresponding to an individual's risk was determined. With this score, only the coronary event risk (fatal and nonfatal sum) was calculated. Here, < 10% indicates low risk, 10%–20% moderate risk, and > 20% high risk.[21,22]

*Bodyweight measurement/height measurement and body mass index*: Bodyweight measurement was carried out in the morning hours using a calibrated and ± 0.5 kg sensitive weighing instrument, with the patients standing still and upright on their feet. The

measurements were carried out before the participants ate anything. The participants were asked to wear thin clothes and no shoes. The height and weight of each patient were measured, and the BMI was calculated as body weight in kilograms divided by the square of height in meters ($kg/m^2$). BMI values of less or greater than the normal values (18.50–24.99 $kg/m^2$) were considered as an indicator of increased risk of health.[18]

*Waist circumference measurement*: The waist circumference value gives an idea of the abdominal adipose tissue (i.e., organ fat). While the person to be measured was standing, the lowest rib on the right side was located and marked. On the hip, the hip bone protrusion (iliac) is located and marked. The midpoint between the two marks was located, and the waist circumference passing through this point was measured. A waist circumference of greater than 94–102 cm in males and 80–88 cm in females is a risk factor for lots of diseases. [18]

*Hip circumference measurement*: The researcher stood by the side of the patient and measured the circumference at the highest point. After measuring the waist and hip circumference, the waist/hip ratio was determined.

*Waist/hip circumference ratio*: The waist/hip circumference ratio is the dimensionless ratio of the circumference of the waist to that of the hips. This is calculated as waist measurement divided by hip measurement . Hip circumference was measured from the widest circumference of the hip while the patient was standing. According to WHO, the waist/hip ratio should be < 0.90 in males and < 0.85 in females. A waist/hip ratio of > 0.90 in males and > 0.85 in females is indicative of increased health risk.[18]

*Other measurements:* Blood pressure was measured with a mercury sphygmomanometer. Venous blood was drawn from each patient after an overnight fast to obtain baseline measurements of TC, while HDL-C was measured by enzymatic methods using a Hitachi 7150 autoanalyzer (Hitachi, Tokyo, Japan).

### *Statistical analysis*

In this descriptive study, the demographic characteristics of the participants and their responses to the scaled questionnaire were analyzed objectively. IBM SPSS 22.0 software package was used for the statistical analysis of the data. The demographic characteristics of those who volunteered to participate in the survey were analyzed, and the frequency distributions of the data were presented. The scale data were analyzed using the chi-square test in paired groups and the MWU (Mann whitney u) test in groups of three or more. Results were evaluated at a 95% confidence interval and a significance level $< 0.05$.

Pearson correlation analysis was performed to reveal the differences in terms of age, BMI, waist circumference, waist/hip ratio, systolic blood pressure, diastolic blood pressure, TC, HDL, and FRS. The significance levels of correlations were accepted as 0.05.

### *Ethics Approval*

Approval was received from Adnan Menderes University Faculty of Medicine Non-Interventional Clinical Research Ethics Committee (approval no. 53043469/050.04-108). Written informed consent was obtained from all the participants.

## Results

According to our study, the distribution of patients with type 2 diabetes was 144 (63.1%) females and 84 (36.85%) males. A total of 22 subjects (20 males and 2 females) in the study stated that they consume alcohol. Of the participants who consume alcohol, 90.9% were males, while 9.1% were females. Smoking status was 65.5% in males and 34.5% in females. The effect of alcohol consumption ($p = 0.180$) and smoking ($p = 0.691$) on OSA syndrome was found to be insignificant (Tables 1 and 2). Also, 65.9% of the participants with a BMI value of greater than 30 $kg/m^2$ and 64.9% of those who were receiving hypertension

treatment were females. While 80.9% of the individuals with a high FRS were males, 89% of those without a Berlin risk were females (Table 1).

The mean total cholesterol of the participants was 54.46 ± 11.46, the mean systolic blood pressure was 78.80 ± 10.10, and the waist/hip ratio was 0.91 ± 0.087. When considered by gender, the mean total cholesterol of females was 193.41 ± 53.90, the mean systolic blood pressure of males was 136.05 ± 18.46, and the waist/hip ratio of males was 0.95 ± 0.09 (Table 2).

When the OSA risk level and the factors affecting the patients were evaluated, 54.2% of the females and 19% of the males in a total of 228 subjects were found to not carry risk, while 45.8% of the females and 81% of the males were found to be at risk. Age, FRS, BMI, waist/hip ratio, and waist circumference were found to have a statistically significant effect on the risk of OSA ($p>.05$)(Table 3).

There was a very weak positive correlation between age and BMI, while a moderate positive correlation was found between waist circumference and BMI. There was a very weak positive correlation between systolic blood pressure and age, while there was a moderate positive correlation between systolic and diastolic blood pressure. A very weak negative correlation was found between TC and systolic blood pressure (mean systolic blood pressure was within normal values). There was a very weak positive correlation between age and systolic blood pressure. There was a moderate correlation between Framingham (heart disease risk score) and age, and a weak positive correlation with systolic blood pressure. We found that the risk of heart disease and systolic blood pressure increased as the age increased.

**Discussion**

OSA syndrome is characterized by recurrent obstructions of the upper airway during sleep. Male gender, genetic characteristics, obesity (wide neck circumference and short thick neck), craniofacial anomalies (retrognathia, micrognathia), smoking, alcohol, and hypnotic

drug use have been reported to be the main predisposing factors that increase the tendency to develop OSA[9,20]. Among these, age, male gender, obesity, and smoking are well-known risk factors for CVD. OSA can also be accompanied by hypertension, DM, and coronary artery diseases.[23,24]

The present study aimed to investigate the relationship between OSA risk level and CVD risk level in patients with type 2 diabetes within the framework of FRS and Berlin form criteria.

The mean age of the patients with type 2 diabetes was 54.46 ± 11.46 years (18–80 years). The effect of age on the OSA risk level was found to be statistically insignificant ($p = 0.330$). This is consistent with previous literature where the effect of age on OSA risk has been reported as not clear [20,26]. It is estimated that the prevalence of OSA increases in advanced age (65 years and over). However, the relationship between age and OSA prevalence is not as simple as thought. In the comparisons made according to AHI, disorders are found more frequently in the elderly, but its relation with morbidity and mortality resulting from excessive daytime, sleepiness is not clearly known. Peppard et al. reported the prevalence of OSA as 3% and 9% in females aged 30–49 years and 50–70 years, respectively.[2] In another study, the frequency of normal and mild OSA was higher in adults, while the frequency of intermediate and advanced stage OSA was higher in the geriatric group.[25] However, it is not fully clear whether age alone increases the risk of OSA, as an increase in the disease with age has not been found as clear in the ages above 65 as under the age of 65.[26] This can be explained by the fact that mortality among patients with OSA is higher than those without OSA or that the disease decreases with age. However, there is no evidence that OSA causes death or regresses with age.

The course of OSA prevalence is between 3.1% and 7.5% in males and 2.1% and 4.5% in females. In this study, we found that the risk of OSA was high in the majority of the males. Similar to our study, Shim et al. reported that males had a higher risk for OSA than females[27]. Sleep Heart Health Study revealed that the risk of coronary artery disease in the 40–70 years age group increased by 68% in males with OSA.[27] The same study indicated that there was a significant relationship between OSA and all-cause mortality, especially coronary artery disease in males.[28] In another study, the prevalence of OSA was reported as 3% in females and 10% in males in the middle age group, while it was 9% and 17% in the advanced age group, respectively.[2] Yıldız et al. reported that severe OSA occurred more frequently in males.[29] Androgenic fat that often occurs in males causes central obesity and the abdominal fat that emerges in central obesity negatively affects the upper respiratory tract patency and breathing pattern. Besides, it is known that the risk of OSA is high in males due to differences in brain activity, upper airway anatomy, and hormonal differences,[2,30] which is consistent with our findings.

Among the patients with type 2 diabetes, females had a high systolic and diastolic blood pressure and were more often diagnosed with hypertension than males. However, these values were within the limits suggested in the literature. The effect of systolic/diastolic blood pressure values on OSA was found to be insignificant ($p = 0.779$). Studies have shown that there is a relationship between OSA and hypertension that is independent of obesity.[31] It has been reported that hypoxia experienced in OSA causes hypertension due to reasons such as stimulation of sympathetic activity, increased intrathoracic pressure, and vascular damage caused by increased free oxygen radicals.[32] This is in contrast to our findings. This difference was thought to be due to the mean age of the participants, which was under 65, and the mean systolic and diastolic blood pressure, which was within normal limits.

The BMI of the patients should be 20–25 kg/m$^2$, and waist circumference should be < 94 cm for males and < 80 cm for females.[33] Although the number of males and females was not equal, the BMI was distributed oppositely. Most of those with a BMI of less than 30 kg/cm$^2$ were males, and the majority of those with a BMI of greater than 30 kg/cm$^2$ were females. The mean BMI of the patients was determined to be qualified as obese. Its effect on the OSA risk level was found to be significant (p = 0.009). Concerning this, the mean waist/hip ratio of the majority of individuals with high risk in terms of waist/hip ratio risk assessment was 0.97. Also, 76.8% of the males were in the risk group, while 23.2% were not. On the other hand, 30.1% of the females were in the nonrisk distribution, while 69.9% were in the risky distribution. The effect of the waist/hip ratio on OSA was found to be significant (p = 0.000). Obesity has been reported as an important risk factor for OSA.[29] Obaseki et al. documented a high risk for OSA in patients with diabetes who had a BMI value of greater than 30 kg/m$^2$. Waist circumference and waist-hip ratio increase in obesity. In a study conducted on patients with diabetes, the waist circumference of individuals with high OSA risk was significantly higher than those with low waist circumference.[9] Another study reported that individuals with a high waist-hip ratio had a high risk for OSA.[10] Fat accumulation due to obesity, especially in the waist region, can increase the pressure on the diaphragm and cause difficulty breathing, and increased weight increases the respiratory load[20]. Accordingly, it is thought that the risk of OSA increases in patients with obesity and individuals with a high waist and waist/hip.

Lipid levels are generally recommended as TC < 200 mg/dl, HDL > 40 (males) or > 48 (female). It has been found that increased TC level and decreasing HDL level increases the risk for CVD.[34] In the study, the patients' TC and HDL levels were within the desired range. The effect of TC (p = 0.330) and HDL (p = 0.514) levels on OSA was found to be insignificant. Simiakakis et al. reported that patients with OSA had low HDL and higher Low-

density lipoprotein (LDL) levels compared to the control group.[35] According to the American Sleep Health Study, an inverse relationship was found between AHI and HDL cholesterol in young males and females independent of obesity, whereas this relationship was not found in older males.[36] Daytime sleepiness and inactivity associated with OSA are expected to be effective in reduced HDL. Besides, due to hypoxia, increased free oxygen radicals and a change in lipid profile occur. Similar to our results, Cintra et al. did not detect a significant difference in total cholesterol and other fractions in patients with OSA.[8] Meszaros et al. reported that inherited factors played an important role in the pathogenesis of dyslipidemia in OSA.[14] The lack of effect of lipid levels on OSA in our study can be explained by the low mean age, which is an important risk factor for dyslipidemia in patients with diabetes who participated in our study. However, our patients might not have had a genetic predisposition. This factor was not examined in our study.

In the Berlin form evaluation, 58.8% of the participants were found to have a high risk for OSA. In a cross-sectional study, One hundred and sixty-two (49.5%) patients had a high risk for OSA[10]. In another study, approximately half of the patients with diabetes (49.5%) were found to be in the high-risk group.[10] Kendzerska et al. found in their cohort study that OSA was an important risk factor for the development of diabetes.[11] Hypoxia significantly increases sympathetic nervous system activity. Intermittent hypoxia affects insulin-target organs such as adipose tissue, liver, and skeletal muscle, as well as pancreatic insulin production and secretion. Hyperglycemia resulting from this leads to an increase in hepatic gluconeogenesis and a decrease in glucose reuptake in skeletal muscles. It also seriously stimulates insulin resistance.[13] Accordingly, the risk of OSA is high in people with diabetes. This is consistent with our findings.

The majority of patients with diabetes participating in the present study appeared to have a low FRS. When evaluated in terms of OSA, the majority of patients with high FRS

also had a high risk of OSA. Cintra et al. reported that high FRS was associated with poor sleep efficiency.[8] However, Özdemir et al. reported that the FRS scale was insufficient in determining cardiovascular risk in patients with OSA.[37] The hypoxia that occurs in OSA causes a change in cholesterol metabolism as well as an increase in epinephrine secretion and cardiovascular risk. Therefore, patients with high OSA risk included in our study may have had higher FRS. However, FRS reveals a 10-year cardiovascular risk. The reason why participants with diabetes had low FRS may be related to the year when the diabetes developed. In our study, the year diabetes was diagnosed among the participants was not determined. The risk of CVD due to diabetes will increase as the year increases. The results obtained seem to be consistent with the literature.

In conclusion, our data revealed that sleep disturbance is common among patients with type 2 diabetes and that individuals at high risk of OSA are also at higher risk of CVD. OSA appears to increase the risk of CVD among patients with type 2 diabetes. It is known that CVD and OSA can be both a cause and a result of each other and often accompany each other. Therefore, early detection and treatment of sleep disorders in patients with type 2 diabetes is recommended. This intervention is important for the reduction and prognosis of the CVD risk resulting from sleep disorders.

Our study has some limitations. Confirmation tests of night polysomnography were not used in this study. However, the Berlin questionnaire has been shown to be sensitive in detecting positive cases. However, one of the strengths of this study is that it highlights more sleep research among patients with diabetes in developing countries, with a particular focus on the contribution of sleep disordersand CVD to diabetes control. It is important for caregivers to understand the role of sleep apnea and cvd in diabetes-related morbidity in order to take a holistic approach to their care.

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**Tables**

**Table 1. Descriptive findings by gender**

| | | Male (n=84) | | Female (144) | |
|---|---|---|---|---|---|
| | | N | % | N | % |
| Alcohol consumption | Yes | 20 | 90.9* | 2 | 9.1* |
| | No | 64 | 31.1* | 142 | 68.9* |
| Smoking status | Yes, more than 10 a day | 19 | 65.5* | 10 | 34.5* |
| | No | 65 | 32.7* | 134 | 67.3* |
| BMI | <30 kg/cm$^2$ | 13 | 65.0* | 7 | 35* |
| | >30 kg/cm$^2$ | 71 | 34.1* | 137 | 65.9* |

| | | | | | |
|---|---|---|---|---|---|
| Receiving treatment for hypertension | Yes | 34 | 35.1* | 63 | 64.9* |
| | No | 50 | 38.2* | 81 | 61.8* |
| Systolic blood pressure | <130 | 25 | 30.5* | 57 | 69.5* |
| | >130 | 59 | 40.7* | 86 | 59.3* |
| Diastolic blood pressure | <85 | 61 | 35.9* | 109 | 64.1* |
| | >85 | 23 | 40.4* | 34 | 59.6* |
| Framingham risk score | Equal to or less than 10 | 39 | 24.2* | 122 | 75.8* |
| | 10-20 | 38 | 80.9* | 9 | 19.1* |
| Waist / hip ratio risk assessment | Non-risk | 21 | 33.9* | 41 | 66.1* |
| | Risky | 61 | 37.7* | 101 | 62.3* |
| Berlin | Non-risk | 16 | 17* | 78 | 83* |
| | Risky | 68 | 50.7* | 66 | 49.3* |

*Line percentage was taken.

**Table 2. Descriptive findings for parametric variables**

| | **Female** | | **Male** | | **Total** | |
|---|---|---|---|---|---|---|
| | **Min. – Max.** | **Mean ± std dev.** | **Min. – Max.** | **Mean ± std dev.** | **Min. – Max.** | **Mean ± std dev.** |
| Age | 26-78 | 54.36±11.12 | 18- 80 | 54.62±12.09 | 18-80 | 54,46 ±11.46 |
| Total cholesterol | 82-404 | 193.41±53.90 | 87-302 | 166.48±55.64 | 82-404 | 183,29 ±55.98 |
| HDL | 12-97 | 55.49±14.87 | 25-88 | 56.98±14.63 | 12-97 | 56,06 ±14.77 |
| Systolic blood pressure | 100-200 | 131.51±18.47 | 90-180 | 136.05±18.46 | 90-200 | 133.20 ±18.56 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Diastolic blood pressure | 50-114 | 78.39±10.26 | 60-104 | 79.48±9.86 | 50-114 | 78.80 ±10.10 |
| Waist / hip | 0.61-1.1 | 0.89±0.07 | 0.68-1.25 | 0.95±0.09 | 0,61-1.25 | 0.91 ±0.087 |
| Waist circumference | 67-140 | 108.49±12.53 | 65-149 | 109.15±15.14 | 65-149 | 108,74 ±13.51 |

**Table 3. OSA risk level in patients and factors affecting it**

| | | **Risk** | **Non-risk** | **P value** |
|---|---|---|---|---|
| **Age**[a] | | 54,45±11,4 | | P=0.330** |
| | | n (%) | n (%) | |
| **Gender** | Male | 16 (19) | 68 (81,0) | P=0.000* |
| | Female | 78 (54.2) | 66 (45,8) | |
| **Alcohol** | Yes | 6 (27,3) | 16 (72,7) | P=0.180* |
| | No | 88 (42,7) | 118 (57,3) | |
| **Smoking** | Yes | 13 (44.5) | 16 (55.2) | P=0.691* |

| | | | | |
|---|---|---|---|---|
| | No | 81 (40.7) | 118 (59.3) | |
| **BMI** | <30kg/m$^2$ | 10 (50) | 10 (50) | P=0.478* |
| | >30kg/m$^2$ | 84 (40.4) | 124 (59.6) | |
| **Receiving treatment for hypertension** | Yes | 41 (42.3) | 56 (57.7) | P=0,630* |
| | No | 53 (40.5) | 78 (59.5) | |
| **Systolic blood pressure** | <130 | 35 (42.7) | 47 (57.3) | P=0.779* |
| | >130 | 58 (40) | 87 (60) | |
| **Diastolic blood pressure** | <85 | 66 (38.8) | 104 (61.2) | P=0.787* |
| | >85 | 27 (47.4) | 30 (52.6) | |
| **Framingham risk score** | Equal to or less than 10 | 70 (43.5) | 91 (56.5) | P=0.028* |
| | 10-20 | 12 (25.5) | 35 (75.5) | |
| **Waist /Hip Ratio**[a] | | 0,91±0,087 | | P=0.022** |
| **BMI** | | 32.4±6.08 | | P=0.000 |
| **Waist circumference** | | 108,74±13,51 | | P=0.000 |
| **TC** | | 183.28±55.9 | | P=0.330 |
| **HDL** | | 56±14.7 | | P=0.514 |

[a] Mean Std Deviation values are given , *Chi-square, **MWU,