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ORIGINAL RESEARCH

# Smoking-related Alterations in Serum Levels of Thyroid Hormones and Insulin in Female and Male Students

Ismail Meral, PhD; Ayse Arslan, PhD; Aydin Him, PhD; Harun Arslan, MD


## ABSTRACT

**Context** • Cigarette smoking has large-scale and complex effects on the endocrine system. Various studies related to cigarette smoking have provided differing results. Therefore, more research is needed to determine the effects on the body that are created by cigarette smoking.

**Objectives** • The study was designed to investigate the effects of cigarette smoking, primarily on thyroid hormones in serum, such as on levels of total triiodothyronine ($tT_3$), free triiodothyronine ($fT_3$), total thyroxine ($tT_4$), free thyroxine ($fT_4$), thyroid-stimulating hormone (TSH) (ie, thyrotropin), and insulin of young students aged 18-25 y.

**Design** • This study was a randomized, controlled trial.

**Setting** • The study was performed in the Department of Physiology, School of Medicine, Yuzuncu Yil University (Van, Turkey).

**Participants** • Eighty healthy students, 40 females and 40 males, were included in the study.

**Intervention** • Of the 40 female participants, 25 were smokers, and 15 were nonsmokers. Of the 40 male participants, 25 were smokers, and 15 were nonsmokers. The intervention (smoking) group, therefore, consisted of 50 participants, and the control (nonsmoking) group consisted of 30 participants.

**Outcome Measures** • Serum concentrations of thyroid hormones and insulin were determined by enzyme-linked immunesorbent assays (ELISAs), using monoclonal antibodies; and by measurement of blood glucose, using a glucometer.

**Results** • The study found that both female and male smokers had higher levels of serum $tT_3$ and insulin hormone than nonsmokers had. A positive correlation was found between age and insulin resistance in male smokers. The study also found that male smokers had higher levels of serum $tT_3$ and $fT_4$ hormone than female smokers had.

**Conclusions** • Smoking may be associated with an increased secretion of thyroid hormones and the development of insulin resistance. With aging, insulin resistance may increase more in male smokers than in female smokers. (*Altern Ther Health Med.* 2015;21(5):24-29.)



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According to the World Health Organization (WHO), the tobacco epidemic has led to the death of 6 million people worldwide each year and has become one of the most important public health problems the world faces today. Every 6 seconds, 1 person in the world dies from diseases related to cigarette smoking.[1] Cigarettes and tobacco contain more than 4000 chemicals. Among them, at least 60 substances have been identified as toxic. Polycyclic aromatic hydrocarbons, nitro compounds, and aromatic amines are only a few of them.[2]

Cigarette smoking has large-scale and complex effects on the endocrine system. It affects the functions of the pituitary, thyroid, and adrenal glands and of the testis and ovaries. Calcium metabolism and insulin activity are also affected by smoking. Cigarette smoking contributes to the

**Table 1.** Descriptive Characteristics of the Participants Included in the Study

| Parameters | Smoking | | Nonsmoking | |
|---|---|---|---|---|
| | Male | Female | Male | Female |
| Gender, n | 25 | 25 | 15 | 15 |
| Age, y | 22.60 | 22.60 | 21.13 | 21.93 |
| Height, cm | 174.24 | 165.12 | 172.86 | 165.60 |
| Weight, kg | 68.24 | 55.96 | 66.66 | 56.66 |
| BMI, kg/m$^2$ | 22.43 | 20.48 | 22.28 | 20.66 |
| Daily cigarette consumption | 1 pack | 1 pack | — | — |
| Duration of smoking, y | 5-6 | 5-6 | — | — |

Abbreviation: BMI, body mass index.

development of type 2 diabetes because it contributes to the development of insulin resistance.[3] It also has been shown to have variable effects on thyroid functions due to an increase or decrease in the concentrations of thyroid hormones, such as thyroxine ($T_4$) and triiodothyronine ($T_3$).[4,5]

Cigarette smoking is a strong risk factor for the development of thyroid disease and may inhibit or stimulate thyroid functions. Graves' disease, Graves' ophthalmopathy, goiter, and abnormalities in thyroid hormones may occur in relation to cigarette smoking.[3] Although the exact mechanism is not understood, it is believed that nicotine leads to activation of the sympathetic nervous system and, thus, increases the total secretion of thyroid hormones. Further, some substances in cigarettes, such as thiocyanate and 2,3-hidroxypridine, may affect the normal physiology of the thyroid.[6,7]

Cigarette smoking can have multiple effects on thyroid function. In various studies, $T_4$ levels in serum remained unchanged[8] or were slightly elevated,[9] whereas $T_3$ levels increased[10] or remained unchanged.[8] Levels of thyrotropin-stimulating hormone (TSH) have been found to have decreased[11] or remained unaltered.[12]

Those variable results may be due to the fact that the various studies are not directly comparable. Although they were conducted among smokers and nonsmokers, significant differences existed in ages, genders, body weights, cigarette smoking habits, and time of abstinence from smoking.

Therefore, to investigate the effects of cigarette smoking, the current study was designed to measure the levels of thyroid hormones, insulin, and fasting blood glucose in male and female university students, aged 18 to 25 years, who smoked cigarettes and to compare them with the levels in healthy, nonsmoking students in the same age group. Several aspects of the study differed from previous investigations: (1) participants were younger (ie, aged 18-25 y), (2) the effects of smoking on the 2 genders were determined, (3) the effects on participants due to the daily quantity of cigarettes (ie, 20 cigarettes/d) and the duration of use (ie, for at least 5 y) were addressed, and (4) the correlation coefficiency in the subgroups were determined to assess the relationships between the variables.

## METHODS

### Participants

A total of 80 students, aged 18 to 25 years, were included in the study. Forty of the participants were females, whereas 40 of them were male. The smoking group included 50 smokers, 25 males and 25 females, whereas the control group included 30 nonsmokers, 15 males and 15 females. The characteristics of the nonsmoking controls and the cigarette smokers are given in Table 1.

The participants were students of the current second author, Ayse Arslan. Smokers and nonsmokers were chosen based on the listed exclusion criteria and matched for age, gender, body mass index (BMI), and health status; both groups included healthy individuals.

Students were excluded from the study if they (1) were dieting; (2) were using medicine for any disease; (3) had hypertension, diabetes mellitus, or respiratory disease; or (4) had nodules in their thyroid glands, as determined by ultrasonographic examination. All participants were single (ie, not married) and none of the females were pregnant.

Ethical approval was obtained from the Science and Ethics Committee of Yüzüncü Yıl University (Van, Turkey). The study was conducted only after voluntary consent was obtained from the participants. A full explanation of the study's protocol was provided to the students before their consent was requested.

### Procedures

For 1 month, blood samples were drawn from the students after an overnight fasting period. An average of 5 mL of blood was taken from each participant. The blood was placed in biochemical tubes and allowed to clot for serum analysis. Serum samples were stored at -80°C until analysis.

### Outcome Measures

The levels of total triiodothyronine ($tT_3$), free triiodothyronine ($fT_3$), total thyroxine ($tT_4$), free thyroxine ($fT_4$), TSH, insulin, and fasting blood glucose were measured. The hormone analyses were made using monoclonal antibodies in an enzyme-linked immunosorbent assay (ELISA-CHB ST-360 automatic, Shanghai Kehua Laboratory System Co, Shanghai, China). Blood-glucose levels were measured using a glucometer (Optium Xceed, Medisense, Abingdon, Oxon, UK) and blood-glucose testing strips (Optium plus, Medisense).

### Statistical Analyses

Because the data were normally distributed according to the Kolmogorov-Smirnov test, an analysis of variance (ANOVA) was performed to determine the differences in the parameters measured in the study between the smoking and nonsmoking groups and also between female and male participants. The results were expressed as mean ± standard error, and $P < .05$ was

considered statistically significant. In addition, Pearson's correlation coefficients were calculated in the subgroups to determine whether a relationship existed between the variables. The SPSS statistical software package (SPSS for Windows version v13.0, SPSS Inc, Armonk, NY, USA) was used for the statistical analyses.

## RESULTS

The $fT_3$, $tT_3$, $fT_4$, $tT_4$, TSH, insulin, and glucose values of smoking and nonsmoking males and females are shown in Table 2. The $fT_3$, $fT_4$, $tT_4$, TSH, and glucose levels were not statistically different between the smoking and nonsmoking males and females. The $tT_3$ level was significantly higher for smoking males, at 125.52 ± 4.97 ($P < .05$) when compared with that for nonsmoking males, at 108.63 ± 6.22. However, no statistical difference existed between the $tT_3$ levels of the smoking females and nonsmoking females. The insulin levels for smoking males, at 17.94 ± 2.40, were significantly higher ($P < .05$) than those for nonsmoking males, at 9.80 ± 1.39. The insulin levels for smoking females, at 14.26 ± 2.17, were also significantly higher ($P < .05$) than those for nonsmoking females, at 10.56 ± 2.31.

The $fT_3$, $tT_3$, $fT_4$, $tT_4$, TSH, insulin, and glucose values of smoking and nonsmoking participants, without distinction between genders, are shown in Table 3. No statistically significant differences existed between the smoking and nonsmoking groups in terms of $fT_3$, $fT_4$, $tT_4$, TSH, and glucose values. The $tT_3$ and insulin values were statistically higher ($P < .05$) for the smoking group, at 116.88 ± 3.69 and 16.11 ± 1.62, respectively, versus the nonsmoking group, at 105.44 ± 3.61 and 10.19 ± 1.35, respectively.

When the Pearson's correlation coefficients were analyzed in male smokers, it was found that a positive correlation ($P < .05$) existed between the $tT_4$ and $tT_3$ levels (Table 4). When the $tT_4$ level increased in the male smokers, the $tT_3$ level also increased, by 77.5% ($P < .01$). In addition, a positive correlation ($P < .05$) was found between age and insulin level and also between the glucose and insulin levels in male smokers. As age increased in the male smokers, the insulin levels also increased by 44.5%, whereas the glucose level increased by 43.7%.

When the Pearson's correlation coefficients were analyzed in female smokers (Table 5), a positive correlation was found between (1) between age and glucose level, (2) $tT_4$ and $tT_3$, and (3) $fT_3$ and TSH levels. Glucose levels increased by 48.1% ($P < .05$) as age increased; $tT_3$ increased by 56.7% ($P < .01$) as $tT_4$ increased; and $fT_3$ increased by 42.6% ($P < .05$) as TSH increased. A negative correlation existed between $fT_3$ and age and between $fT_3$ and $fT_4$. As age increased in female smokers, the $fT_3$ level decreased by 41.8%, and as the $fT_4$ level increased, the $fT_3$ level decreased by 49.1%.

**Table 2.** Values of $fT_3$, $tT_3$, $fT_4$, $tT_4$, TSH, Insulin, and Glucose in Smoking and Nonsmoking Males and Females

| | Nonsmoking | | Smoking | |
|---|---|---|---|---|
| Parameters | Males Mean ± SE (n = 15) | Females Mean ± SE (n = 15) | Males Mean ± SE (n = 25) | Females Mean ± SE (n = 25) |
| $fT_3$ (pg/mL) | 3.22 ± 0.17 | 3.27 ± 0.14 | 3.22 ± 0.13 | 3.50 ± 0.12 |
| $tT_3$ (ng/dL) | 108.63 ± 6.22 | 102.25 ± 3.72 | 125.52 ± 4.97[a] | 108.23 ± 4.96 |
| $fT_4$ (ng/dL) | 1.48 ± 0.07 | 1.29 ± 0.04 | 1.56 ± 0.07 | 1.36 ± 0.04 |
| $tT_4$ (μg/dL) | 7.92 ± 0.44 | 7.50 ± 0.27 | 8.24 ± 0.28 | 7.46 ± 0.27 |
| TSH (μIU/mL) | 1.19 ± 0.21 | 1.53 ± 0.18 | 1.07 ± 0.13 | 1.09 ± 0.13 |
| Insulin (μIU/mL) | 9.80 ± 1.39 | 10.56 ± 2.31 | 17.94 ± 2.40[a] | 14.26 ± 2.17[b] |
| Glucose (mg/dL) | 83.07 ± 2.25 | 79.75 ± 1.55 | 87.31 ± 2.57 | 82.77 ± 2.49 |

Abbreviations: SE, standard error of the mean; $fT_3$, free triiodothyronine; $tT_3$, total triiodothyronine; $fT_4$, free thyroxine; $tT_4$, total thyroxine; TSH, thyrotropin stimulating hormone.

[a] $P < .05$, statistically significant differences between smoking and nonsmoking male participants.

[b] $P < .05$, statistically significant differences between smoking and nonsmoking female participants.

**Table 3.** Values of $fT_3$, $tT_3$, $fT_4$, $tT_4$, TSH, Insulin, and Glucose of Smoking and Nonsmoking Participants, Without Distinction Between Males and Females

| Parameters | Nonsmoking Mean ± SE n = 30 | Smoking Mean ± SE n = 50 |
|---|---|---|
| $fT_3$ (pg/mL) | 3.25 ± 0.11 | 3.36 ± 0.09 |
| $tT_3$ (ng/dL) | 105.44 ± 3.61 | 116.88 ± 3.69[a] |
| $fT_4$ (ng/dL) | 1.39 ± 0.04 | 1.47 ± 0.04 |
| $tT_4$ (μg/dL) | 7.71 ± 0.26 | 7.85 ± 0.19 |
| TSH (μIU/mL) | 1.37 ± 0.14 | 1.08 ± 0.09 |
| Insulin (μIU/mL) | 10.19 ± 1.35 | 16.11 ± 1.62[a] |
| Glucose (mg/dL) | 81.42 ± 1.38 | 85.05 ± 1.80 |

Abbreviations: SE, standard error of the mean; $fT_3$, free triiodothyronine; $tT_3$, total triiodothyronine; $fT_4$, free thyroxine; $tT_4$, total thyroxine; TSH, thyrotropin stimulating hormone.

[a] $P < .05$, statistically significant differences between the 2 groups.

**Table 4.** Pearson's Correlation Coefficients, by Characteristic, in Male Smokers (n = 25)

| Parameters | $fT_3$ | $tT_3$ | $fT_4$ | $tT_4$ | TSH | Insulin | Glucose | Age | Height | Weight | BMI |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $fT_3$ (pg/mL) | 1 | | | | | | | | | | |
| $tT_3$ (ng/dL) | .213 | 1 | | | | | | | | | |
| $fT_4$ (ng/dL) | -.290 | .018 | 1 | | | | | | | | |
| $tT_4$ (µg/dL) | .088 | .775[a] | .181 | 1 | | | | | | | |
| TSH (µIU/mL) | -.243 | -.351 | .244 | -.307 | 1 | | | | | | |
| Insulin (µIU/mL) | -.031 | .156 | -.134 | .036 | -.339 | 1 | | | | | |
| Glucose (mg/dL) | .027 | .306 | -.032 | .112 | -.312 | .437[b] | 1 | | | | |
| Age | -.052 | .104 | .132 | .027 | -.395 | .445[b] | -.004 | 1 | | | |
| Height | .093 | .035 | .102 | -.171 | -.009 | .246 | -.109 | .238 | 1 | | |
| Weight | -.063 | .042 | .141 | -.175 | -.041 | .337 | -.068 | .308 | .917[a] | 1 | |
| BMI | -.347 | .029 | .151 | -.087 | -.099 | .334 | .074 | .296 | .265 | .626[a] | 1 |

Abbreviations: $fT_3$, free triiodothyronine; $tT_3$, total triiodothyronine; $fT_4$, free thyroxine; $tT_4$, total thyroxine; TSH, thyrotropin stimulating hormone; BMI, body mass index.

[a]$P < .01$.
[b]$P < .05$.

**Table 5.** Pearson's Correlation Coefficients, by Characteristic, in Female Smokers (n = 25)

| Parameters | $fT_3$ | $tT_3$ | $fT_4$ | $tT_4$ | TSH | Insulin | Glucose | Age | Height | Weight | BMI |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $fT_3$ (pg/mL) | 1 | | | | | | | | | | |
| $tT_3$ (ng/dL) | .209 | 1 | | | | | | | | | |
| $fT_4$ (ng/dL) | -.491[a] | .066 | 1 | | | | | | | | |
| $tT_4$ (µg/dL) | .221 | .567[b] | .260 | 1 | | | | | | | |
| TSH (µIU/mL) | .426[a] | .120 | -.298 | -.042 | 1 | | | | | | |
| Insulin (µIU/mL) | .013 | .256 | -.127 | -.137 | -.316 | 1 | | | | | |
| Glucose (mg/dL) | .176 | .178 | .031 | .130 | .134 | .313 | 1 | | | | |
| Age | -.418[a] | .092 | .305 | -.162 | .026 | .131 | .481[a] | 1 | | | |
| Height | .034 | -.204 | -.124 | -.243 | .327 | -.194 | -.116 | -.032 | 1 | | |
| Weight | .115 | .005 | -.162 | -.108 | .202 | -.072 | .144 | .086 | .769[b] | 1 | |
| BMI | .132 | .173 | -.129 | .030 | .041 | .041 | .268 | .142 | .373 | .879[b] | 1 |

Abbreviations: $fT_3$, free triiodothyronine; $tT_3$, total triiodothyronine; $fT_4$, free thyroxine; $tT_4$, total thyroxine; TSH, thyrotropin stimulating hormone.

[a]$P < .05$
[b]$P < .01$

## DISCUSSION

The current study was designed to investigate smoking-related alterations on levels of $tT_3$, $fT_3$, $tT_4$, $fT_4$, TSH, insulin, and fasting blood glucose for young male and female students aged 18 to 25 years. The study found that no differences existed between the smoking and nonsmoking groups regarding values of $fT_3$, $fT_4$, $tT_4$, TSH, and glucose, but the insulin and $tT_3$ values were significantly higher in the smoking group.

In a previous study, Soldin et al[13] found that active and passive smoke exposure produced a mild inhibitory effect on serum levels of $tT_3$, $tT_4$, and TSH in women of reproductive age (ie, from 18-44 y). That result is not consistent with the current study's results because the research team found a significant increase in $tT_3$ values for smokers. That finding may be due to the fact that the participants in the current study, compared with those in the prior study, were younger (ie, 18-25 y) and had a higher amount of daily cigarette inhalation (ie, 20 cigarettes/d).

In a study by Gulcu et al[14] that was conducted to determine the relationship between cigarette smoking and hypothyroidism or hyperthyroidism, 31 males who smoked 12 to 18 cigarettes per day and 32 males who had never been smokers were included in the study. The researchers in that study observed that the $tT_3$, $tT_4$, and TSH levels of the smoking groups decreased significantly. They suggested that excessive smoking might have caused hypothyroidism. In a study by Asvold et al,[15] the researchers suggested that smoking had an association with hyperthyroidism but not with hypothyroidism. The finding of a high level of serum $tT_3$ in smokers in the current study is similar to the finding in the Asvold et al study.

It has been suggested that nicotine stimulates the sympathetic nervous system and ultimately increases the secretion of thyroid hormones.[6,7] Two studies have found that nicotine can lead to the release of norepinephrine by binding to nicotinic cholinergic receptors, which are commonly found in the brain, and can stimulate the sympathetic nervous system.[16,17]

The current study also found that the $tT_3$ level was significantly higher in smoking males than nonsmoking males. However, no difference existed between the smoking and nonsmoking females' levels of $tT_3$. Zeman et al[18] have suggested that cigarettes preferred by females have lower levels of nicotine. In that study, the nicotine levels in the plasma and urine of females were lower than those of the males, and their nicotine metabolism was faster.

By the infusion of nicotine or cotinine, which is a metabolite of nicotine, another study has demonstrated that the plasma half-life of nicotine in females is shorter than in males.[19] Benowitz et al[20] have suggested that the clearance of nicotine or cotinine is slower in males compared with females. Those data suggest males are more affected by cigarettes and metabolites than females are, and, as a result, metabolic changes might emerge more significantly in males.

Cigarette smoking is considered a major risk factor for cardiovascular disease.[22] Some studies have shown that cigarette smoking decreases insulin sensitivity and leads to the development of insulin resistance.[21,21] It has also been shown that cigarette smoking is associated with metabolic disorders and that the risk of the occurrence of metabolic syndrome increases in smokers.[23,24] Eliasson[25] has revealed that cigarette smoking increases the risk of developing diabetes, by 50% in females and males.

In the current study, insulin levels were found to be higher in smoking males and females compared with those who did not smoke. Despite that significant increase in the level of insulin in smokers, a significant difference was not found between smokers and nonsmokers in terms of the glucose level. That result indicates that insulin resistance can occur in smokers. That finding is consistent with the finding of a previous study that indicated that cigarette smoking contributed to the development of type 2 diabetes due to the development of insulin resistance.[3] In the current study, it was also found that a positive correlation existed between age and insulin resistance in male smokers, but the same situation did not occur in female smokers. That finding showed development of insulin resistance may increase more with age in male smokers compared with female smokers.

It is not fully understood how cigarette smoking contributes to the development of insulin resistance. However, nicotine, carbon monoxide, and other toxic substances in cigarettes are believed to have a direct effect on the tissue formation of insulin resistance. According to other studies, continuous smoking can lead to vascular changes (eg, arterial spasm and atherosclerosis), thereby decreasing blood flow to skeletal muscles and decreasing the distribution of insulin-dependent glucose by impairing the endothelial function.[26,27]

Nicotine also contributes to the development of insulin resistance through increasing the release of corticosteroids and growth hormone, which are anti-insulin hormones. Levels of free fatty acids and triglycerides are high in smokers and are associated with insulin resistance. Some studies have shown that increased plasma levels of free fatty acids can cause insulin resistance and impair the distribution of insulin-dependent glucose.[26,28] In those studies, both insulin resistance and lipid intolerance have been found.

In another study,[29] smokers were separated into 3 groups, according to their members' annual cigarette consumption in terms of packages, as light (25), medium (20-39), and heavy (≥40) smokers. It was observed that metabolic disorders intensified when cigarette consumption increased (eg, high blood sugar, high triglycerides, and low high-density lipoprotein [HDL] cholesterol). Accordingly, it was concluded that the risk of the occurrence of metabolic syndrome was higher in smokers.

In another study,[30] male and female smokers aged 18 to 92 years were investigated regarding metabolic disorders. The researchers found that males have higher risk factors for metabolic disorders; the mean ages and glucose concentrations were similar in both genders. Males tended

to have a higher mean blood pressure (systolic and diastolic), a higher level of triglycerides, and a lower level of HDL cholesterol. The researchers also found that no significant relationship existed between smoking and metabolic syndrome in female participants.

In the current study, the research team concluded that the $tT_3$ levels in male smokers and the insulin levels in male and female smokers increased, indicating that smoking was associated with the metabolism of thyroid hormones and insulin in healthy participants. Increased insulin secretion, without changes in glucose levels, indicated an insulin resistance in the participants.

In addition, $tT_3$ values in males were found in the current study to be higher than those of females, which revealed the fact that cigarette smoking caused different effects in females and males and that the effects in males might be more severe. Further, the presence of a positive correlation between age and insulin resistance in male smokers and an absence of the same relationship in female smokers showed that insulin resistance may increase more with age in males compared with females.

The emergence of the effects of cigarette smoking in such an apparent way, even after 5 years of use, in a younger age group (18-25 y) of the current study, is remarkable. Insulin resistance, particularly when observed in young individuals who smoke, may increase the risk of developing type 2 diabetes in the future in those persons. Therefore, quitting smoking may have immediate benefits to health at any age. For reductions in smoking among adults, effective interventions need to be augmented, such as smoke-free laws, tobacco price increases, and antitobacco media campaigns.

## CONCLUSIONS

Smoking may be associated with an increased secretion of thyroid hormones and the development of insulin resistance. With aging, insulin resistance may increase more in male smokers than in female smokers.


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### AUTHOR DISCLOSURE STATEMENT

The authors report no conflicts of interest.

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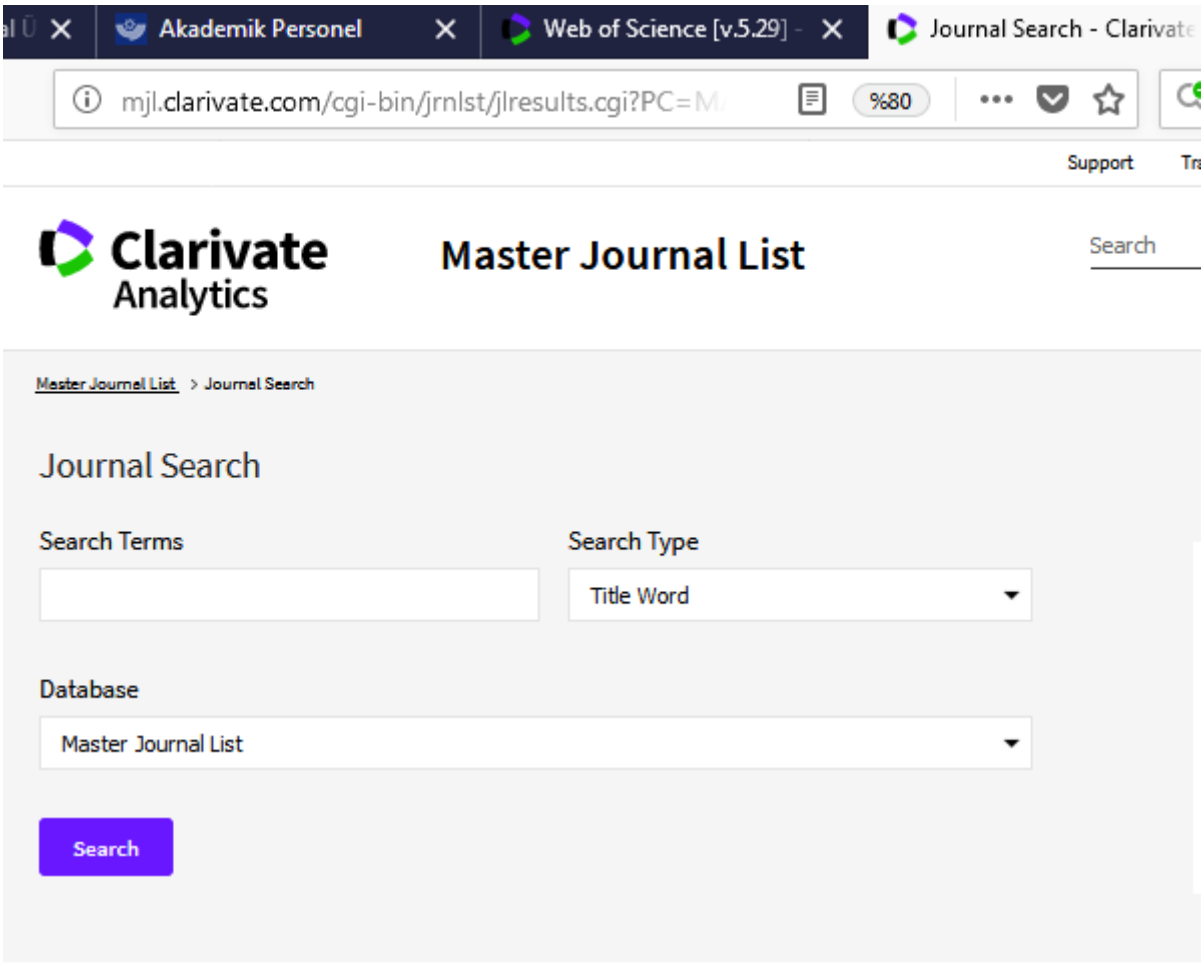


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