International Journal of Research and Review
www.ijrrjournal.com E-ISSN: 2349-9788; P-ISSN: 2454-2237

Research Paper

Effect of Smoking on Follicle-Stimulating Hormone, Luteinizing Hormone and Testosterone in Men

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ABSTRACT

Background: Smoking has a negative impact on the reproductive health. The aim of the study was determine effect of smoking on follicle-stimulating hormone (FSH), luteinizing hormone (LH) and testosterone in men.
Methods: The study was conducted using a cross sectional study design. The populations in this study were all active smoker and non smoker in Baiturrahmah University with sample size 50 respondents. Sampling technique with consecutive sampling. FSH, LH and testosterone examination in Biochemistry Laboratory of Andalas University. Data analysis used independent sample T test. A two-tailed *P*-value of <0.05 was considered statistically significant. All data were analysed using SPSS 21.0 program.
Results: The results of the study found there were mean differences in FSH level between active smoker and non-smoker were 3.857±1.493 mIU/ml and 4.977±2.083 mIU/ml ($p<0.05$). LH level showed were 4.984±2.237 mIU/ml and 5.500±2.623 mIU/ml between active smoker and non-smoker ($p>0.05$). While there were mean differences in testosterone level between active smoker and non-smoker were 15.393±2.782 nmol/l and 20.836±6.360 nmol/l ($p<0.05$).
Conclusion: This analysis confirmed there were effect of FSH and testoterone level between active smoker and non-smoker.

Keywords: FSH, LH, Smoking, Testosterone

INTRODUCTION

Smoking and complications are important social and health problems in all countries. [1] World Health Organization (WHO) estimates that in 2020 smoking-related diseases will become a major health problem in many countries. [2] According to WHO 2008 Indonesia has the third largest number of smokers in the world after China and India. [3]

Smoking has a negative impact on the reproductive health of men and women. Cigarettes contain a lot of chemicals. Nicotine is one of the main components of the particulate phase of tobacco combustion. It has been found to be very dangerous for the human body, its absorption through the respiratory tract, oral mucosa and skin, while 80-90% are metabolized by the liver, kidneys and lungs. [4] Nicotine and its metabolites (cotinine) have been found in serum, urine, saliva and milk, and have recently been found in semen plasma in smokers exposed to tobacco smoke. [5]

Various hormones including sex steroid hormones can change, due to the effects of nicotine including the axis of the hypothalamus-pituitary-gonad, and metabolism of steroid sex hormones. The prostate and seminal vesicle function can also be influenced by nicotine in cigarettes. [6] Nicotine can affect the work of the central nervous system. [7] Gonadotropin Releasing Hormone (GnRH) in the hypothalamus stimulates the Anterior Hipofise to produce Follicle Stimulating

Hormone (FSH) and Luteinizing hormone (LH). FSH stimulates Sertoli cells to produce Androgen Biding Protein (ABP), LH stimulates leydig cells to produce testosterone. [8]

Other cigarette content such as cadmium affects the male reproductive system. Cadmium level are found to be higher in the seminal plasma and blood of infertile men. Cadmium inhibits the concentration and motility of spermatozoa, because of its antisteroidogenic properties associated with exposure to cadmium in leydig cells. The effects of cadmium on leydig cells include: decreased cell survival, decreased testosterone secretion and an increase in malondialdehyde levels. [9]

Gas and particulates in cigarette smoke are free radicals that can cause an increase in Reactive Oxygen Species (ROS) in the body. [10] Free radicals are atoms or molecules (collections of atoms) that have unpaired electrons. Increased ROS will cause oxidative stress. Oxidative stress is the result of an imbalance between oxidants and antioxidants in the body, an increased ROS which is one of the mediators of male infertility. [11] ROS physiologically plays an important role in sperm function, but at a high level the effect is detrimental to sperm. High levels of ROS exceeding antioxidants cause damage to sperm membrane, also results in a decrease in leydig cell function. [12]

Previous study known LH levels in non smokers higher than active smoker. [13] FSH levels are higher in men who not smoking. [12] Another study showed that testosterone levels decreased in active smoker than non smoker. In accordance with these findings, it shows that there is an inhibiting effect of smoking on the male reproductive system. [6] The aim of the study was determine effect of smoking on follicle-stimulating hormone (FSH), luteinizing hormone (LH) and testosterone in.

MATERIALS & METHODS

Study Design and Research Sample

The study was conducted using a cross sectional study design. The populations in this study were all active smoker and non smoker in Baiturrahmah University with sample size 50 respondent (consist of two groups: 25 active smoker and 25 non smoker). Sampling technique with consecutive sampling. Inclusion criteria: men aged 20-49 years, smoking for 10 years and consuming cigarettes >20 cigarettes per day.

Operational Definitions

The variables of this study included independent variable were smoking. Dependent variables were FSH, LH and testosterone.

Data Collection Technique

Blood (~ 2 ml) was collected from the antecubital vein in vials without EDTA (clot vials) and serum isolated by centrifugation (1000 x rpm, 10 min). Follicle stimulating hormone (FSH), leutanising hormone (LH), and testosterone concentrations were determined in the serum using standard ELISA kits (Biorad Laboratories, USA) to evaluate the endocrine status of individuals involved in the study group consisting of heavy smoking-severeoligo/azoospermic patients while nonsmoking-normozoo-spermic group were kept as controls. This study was approved by the Ethical Committee, Faculty of Medicine, Universitas Andalas, West Sumatera Indonesia with registration number 355/KEP/FK/2014.

Data Analysis

The quantitative variables were recorded as mean and standard deviation. Data analysis used independent sample T test. A two-tailed *P*-value of <0.05 was considered statistically significant. All data were analysed using SPSS 21.0 program.

RESULT

Characteristic of respondents (Table 1).

Table 1: Characteristic of respondents

Characteristic	Active smoker (Mean±SD)	Non-smoker (Mean±SD)	p-value
Age (years)	33.44±7.16	30.08±5.42	0.670
Bassal mass index (kg/m^2)	21.76±2.32	21.53±2.28	0.715

Table 1 known there were mean difference of age and BMI between active smoker and non-smoker (p>0.05).

Table 2: Mean difference of FSH level between active smoker and non-smoker

Group	n	FSH level (mIU/ml) Mean ± SD	p-value
Active smoker	25	3.857 ± 1.493	0.034
Non-smoker	25	4.977 ± 2.083	

Table 2 known there were mean differences in FSH level between active smoker and non-smoker were 3.857±1.493 mIU/ml and 4.977±2.083 mIU/ml (p<0.05).

Table 3: Mean difference of LH level between active smoker and non-smoker

Group	n	LH level (mIU/ml) Mean ± SD	p-value
Active smoker	25	4.984±2.237	0.458
Non-smoker	25	5.500±2.623	

Table 3 found LH level showed were 4.984±2.237 mIU/ml and 5.500±2.623 mIU/ml between active smoker and non-smoker (p>0.05).

Table 4: Mean difference of testosterone level between active smoker and non-smoker

Group	n	Testosterone level (nmol/l) Mean ± SD	p-value
Active smoker	25	15.393 ± 2.782	<0.001
Non-smoker	25	20.836 ± 6.360	

Table 4 found there were mean differences in testosterone level between active smoker and non-smoker were 15.393±2.782 nmol/l and 20.836±6.360 nmol/l (p<0.05).

DISCUSSION

The results of the study found there were mean differences in FSH level between active smoker and non-smoker were 3.857±1.493 mIU/ml and 4.977±2.083 mIU/ml (p<0.05). LH level showed were 4.984±2.237 mIU/ml and 5.500±2.623 mIU/ml between active smoker and non-smoker (p>0.05). While there were mean differences in testosterone level between active smoker and non-smoker were 15.393±2.782 nmol/l and 20.836±6.360 nmol/l (p<0.05).

Someone who continues to smoke for years, his blood will be contaminated by nicotine which through blood vessels will spread throughout the body, including to the reproductive organs. Smoking has a negative impact on the reproductive health of men and women. Cigarettes contain many chemicals. [3] When cigarettes are burned, there is a combustion and pyrolysis reaction. [14] The results of combustion into the lungs then through blood circulation to the brain and into other tissues. [9]

Previous study found that smoking more than 10 cigarettes per day caused a decrease in FSH 37% compared to those who smoked less than 10 cigarettes per day. [15] In contrast to another study, it was found that FSH levels were lower in nonsmokers compared to smokers and former smokers. [13] Research conducted by Heidary, *et al*, on animals that were affected by cigarettes and hookah showed the results of serum FSH levels were not significantly increased in mice treated with hookah, and significantly increased in mice treated with cigarettes compared with controls. [6]

In this study, it was found that the mean value of LH and standard deviation in heavy smokers was lower when compared to nonsmokers because it is in accordance with the theory that cigarette content can affect the central nervous system, so that stimulation to the hypothalamus in the central nervous system decreases which causes stimulation to GnRH also decreases, consequently the stimulation to the anterior pituitary also decreases, so the LH produced also decreases. When LH decreases the stimulus to leydig cells will also decrease. LH stimulates leydig cells to produce testosterone. Testosterone is secreted by Leydig Interstitial cells in the testis, but only occurs when Interstitial cells are transplanted by LH from the Anterior Pituitary gland. Furthermore the amount of testosterone secreted increases in proportion to the amount of LH available. [8]

In theory, in the hypothalamus-pituitary-gonad system, an increase in LH will cause testosterone to rise, if testosterone rises will cause negative feedback so that LH decreases. According to previous study, cigarette smoke can

interfere with the normal functioning of this system. [16]

The results of this study differ from another study, that LH levels increase significantly in smokers compared to nonsmokers and ex-smokers. [13] Smoking from time to time causes degeneration of leydig cells, this is also supported by results of animal studies found that from the results of histological examination of rat testicles showed less and experienced degeneration of leydig cells. [1]

This study also showed the results that in heavy smokers lower testosterone levels compared with nonsmokers. This could be due to the effects of smoking which can affect the work of the central nervous system. The hypothalamus in the central nervous system will stimulate GnRH, GnRH stimulates the Anterior Pituitary to produce FSH and LH. Nicotine contained in cigarettes will affect GnRH resulting in a decrease in FSH and LH. Decreasing LH will cause the number of leydig cells to decrease so that the testosterone produced will also decrease.

Cigarette smoke contains free radicals that can cause an increase in Reactive Oxygen Species (ROS) in the body. [10] Free radicals are atoms or molecules (collections of atoms) that have unpaired electrons. Increased ROS will cause oxidative stress. Oxidative stress is the result of an imbalance between oxidants and antioxidants in the body, an increased ROS which is one of the mediators of male infertility. [11] ROS physiologically plays an important role in sperm function, but at a high level the effect is detrimental to sperm. High levels of ROS exceeding antioxidants cause damage to sperm membrane, also results in a decrease in leydig cell function. [12]

CONCLUSION

The conclusion of this study confirmed there were effect of FSH and testoterone level between active smoker and non-smoker.

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How to cite this article: Devita H. Effect of smoking on follicle-stimulating hormone, luteinizing hormone and testosterone in men. International Journal of Research and Review. 2019; 6(8):448-452.

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