 Does Cigarette Smoking Affect Serum Total Cortisol and Salivary Cortisol Levels?

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OBJECTIVES

In the current literature, there are some controversies about the influence of cigarette smoking on salivary cortisol levels (SaC). It is known that, total cortisol levels (TC) increase 20 minutes after cigarette smoking [1]. By contrast, a study reported that middle-aged Japanese male smokers had lower plasma cortisol level in the morning than non-smokers [2]. In a study SaC levels were found higher in smokers group than non-smokers [3]. In view of the known acute effects of smoking on TC, the question of whether SaC levels differ in smokers and non-smokers has not been satisfactorily answered yet. For this purpose, we measured TC and SaC levels of cigarette smokers and non-smokers in a group of healthy volunteers.

METHODS

Fifteen healthy smokers (5 females and 10 males) with the mean age of 39.5± 14.3, and 15 age and gender matched non-smoker healthy controls (mean:42.5± 13.46), (range:19-63 years) were recruited to the study. Cigarette smoking period was determined as packet/day x year per person.

Exclusion criteria were the presence of diabetes mellitus, malignancy, history of corticosteroid exposure, oral contraceptive use, and also presence of any disease that could affect HPA axis.

ACTH stimulation test: Tests were performed between 08.00- 09.00 a.m., after an overnight fast. Blood and saliva samples for TC and SaC were obtained before and after 30, 60, 90 minutes of administration of 250 µg synthetic ACTH.

Salivary samples: Sixty minutes before the test, individuals were not allowed to smoke, eat, drink liquids or brush their teeth. Saliva samples were collected by using oral swabs (Salimetrics®). SaC was measured by using high-sensitivity enzyme-immunassay kit (Salimetrics® Inc, State College, PA, USA) and TC levels were measured by radioimmunassay (RIA) method Immunotech (Prag, Czech Republic).

RESULTS

Demographic features and cigarette smoking period of the people are shown in Table 1 and Table 2. Basal, peak and delta TC, and SaC levels of the volunteers in cigarette smoker and non-smoker groups are also summarized in Table 3. Peak and delta TC and SaC levels after 250 µg ACTH stimulation test were significantly higher than their basal levels both in cigarette smoker and non-smoker groups. Basal TC levels were higher in non-smoker group than smoker’s, whereas peak and delta TC levels were not different in both groups. Basal, peak and delta SaC levels were different in neither cigarette smoker nor non-smoker groups (Table 3). Mean TC and SaC levels during test minutes in smoker and non-smoker groups are shown in Figure 1a and 1b. If we compare smoker and non-smoker groups according to gender, only peak TC levels after 250 µg ACTH stimulation test were found to be significantly higher in non-smokers than smokers in male group (Table 4). Basal and stimulated SaC levels were found to be similar among male, female and overall groups (Figure 2).

CONCLUSIONS

In contrast to some previous reports, TC levels were incompatible with smoking habits of the subjects included in our study. Being smoker does not affect SaC responses to ACTH test if the patient does not smoke during test. Our study does not exclude the possible acute effect of SaC levels in smokers.

References