Salivary uric acid, total protein and periodontal health status variation in relation to the body mass index
(A Clinical and Biochemical study)

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ABSTRACT
Background: Obesity is the fastest growing health-related problem in the world. It plays an affecting role in the biochemistry of many serious systemic diseases like diabetes and CVD. Periodontitis appeared to have a reciprocal relationship with both, obesity on one hand side and the serious systemic diseases on other hand.

Aim of study: is to investigate the effect of obesity and periodontitis on the salivary flow and its uric acid and total protein.

Material and method: Eighty one male, aged 30-40 y, systemically healthy, no smokers, having chronic periodontopathic inflammation(gingivitis and / or periodontitis), grouped in three according to BMI, each of 27: GI; normal weight, G2; pre-obese, and G3; obese. PL.I, GI, BOP, PPD, CAL were clinically recorded at the same hour of unstimulated saliva collection. Probing depth and attachment levels were arranged in scales.

Result: Obese group showed significantly high scores of all periodontal data over other 2 groups. Pre-obese showed also significant high scores on the normal weighted. Uric acid was insignificantly low in obese than other 2 groups, while the total protein, in obese was significantly higher than other 2 groups, as the salivary flow did. The differences in uric acid and total proteins values were insignificant between normal weights and pre-obese. A significant positive correlation have been found among periodontal parameters, scale 1 PPD/CAL salivary flow, uric acid, total protein and obesity.

Conclusion: Obesity is not only enhancing the severity of periodontopathy, but also modifies the chemical Constituents of saliva.

Keywords: Salivary flow, total protein, uric acid, gingivitis.

INTRODUCTION
Periodontal disease is a bacterial origin, affecting the supporting structures and leading to gingival inflammation, which may progress to a more destructive condition characterized with bone loss and pocket formation (1). Bacterial challenge as it is the initiative factor, plays its role by the activation of the immune system against the bacterial antigenic invasion (2). More or less, the progression of the disease from a simple gingivitis to the complicated periodontitis could take years unless associated with a systemic risk factor or factors (3). Many serious risk factors have been demonstrated to be linked with periodontal disease in reciprocal relationship, like Diabetes, hypertension, arteriosclerosis, cardiovascular disease, hyperlipidimia, cholelithiasis and cardiovascular diseases (4). Obesity could play a central role in most of these diseases (5). Obesity has been highlighted as a predictor of the majority of these serious diseases and plays a role in blood biochemistry imbalance (6).

Plasma chemical constituents have been demonstrated to be transferred to the salivary flow in concentrations differs according to the systemic health conditions like uric acid, C reactive protein, immunoglobulin and total proteins (7).

The aim of this study is to investigate the influences of obesity on the unstimulated salivary flow, salivary uric acid and total protein values and also the influences of all on the periodontopathy of Iraqi subjects.

MATERIAL AND METHODS
Three groups of periodontopathic volunteers have been chosen (having either gingivitis and / or periodontitis), constituting 81 subjects, aged 30-40 years, male, non smokers and systemically healthy. Females have been excluded to avoid sexual hormone interferences. Each group of 27 subject, according to their body weight on BMI standard scale, first group (G1) as a normal weight(BMI,18.5-24.99), second group (G2) as pre-obese group (BMI,25-29.99), and third group (G3) as obese group (BMI, ≥ 30).The clinical parameters, Plaque index (PL.I), Gingival index (G.I), Bleeding on probing (BOP), Probing pocket depth (PPD) and Clinical attachment level (CAL) have been clinically recorded, PPD and CAL.
records have been distributed in scales; Scale one: 0-3\textsuperscript{mm}, Scale 2: 3-5\textsuperscript{mm} Scale 3: 5-7\textsuperscript{mm}. The unstimulated salivary flow samples have been collected at 9-12 AM\textsuperscript{(8)}, after careful mouth rinse with potable water. Subjects have to be fasting at least one hour before collection. Timing the collection process was 5-10 minutes. Collected samples have been centrifuged (4000\textsuperscript{RPM}) for 10 minutes, freeze at -20\textdegree C. Proteins analyzed as it react in acidic medium with pirogallol red to be molibdate to form a colored complex. The intensity of color is proportional to the protein concentration read on spectrophotometer at 598\textsuperscript{nm}. (Reagent supplier: Spinreact.co/Spain). Uric acid been oxidized by Uricase to allantoine and hydrogen peroxide, when 1\textmu L of reconstituted react with 20\textmu L of prepared saliva then incubated at 37\textdegree C for 5\textsuperscript{mm}. Oxidation read on spectrophotometer at 520\textsuperscript{nm}.(Reagent supply: Biomaghreb.co/Tunisia) Results were statistically analyzed with t-test, F-Fisher test, chi-square, and spearman coefficient of correlation\textsuperscript{(9)}.\

RESULTS

Obese group showed a significant higher PL.I, G.I and BOP than that of pre-obese which is also significantly higher than that of normal weight,[Tables 1 and 4]. Obese subjects also showed an increased number and depth of periodontal pockets. It showed a significant increase in PPD than normal weight groups, while it is insignificant between pre-obese and normal weight, and same insignificance between obese and pre-obese. As same do the CAL scales and scores [Table 2 and 4]. Salivary uric acid showed an insignificant reduction of its value in the obese than in the other two groups, and that of pre-obese is also insignificantly lesser than that of normal weight. Total protein showed a significant high value in the obese than normal weight, while the pre-obese showed a medium value, insignificantly lesser than that of obese and insignificantly higher than that of normal weight. [Table 3]

The coefficient of correlation showed a –ve significant correlation between PL.I and uric acid, but a +ve significant correlation with total protein in all the three groups. No correlation has been found between G.I, uric Acid and total protein in normal weight group. In obese, G.I and BOP, both showed a significant negative correlation with uric acid, and +ve with total protein. Scale1 CAL and scale1 PPD, showed a positive correlation with the total protein and –ve with the uric acid [Table 5].

<table>
<thead>
<tr>
<th>BMI Groups</th>
<th>PL.I</th>
<th>G.I</th>
<th>BOP</th>
</tr>
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<tbody>
<tr>
<td>G1</td>
<td>0.929 ± 0.0813</td>
<td>0.798 ± 0.062</td>
<td>Total sites:2720 Bleeding: 195 % 7.169</td>
</tr>
<tr>
<td>SD 0.4228</td>
<td>SD 0.3223</td>
<td></td>
<td></td>
</tr>
<tr>
<td>G2</td>
<td>1.082 ± 0.0723</td>
<td>1.08 ± 0.054</td>
<td>Total sites:2692 Bleeding: 448 % 16.642</td>
</tr>
<tr>
<td>SD 0.3758</td>
<td>SD 0.2837</td>
<td></td>
<td></td>
</tr>
<tr>
<td>G3</td>
<td>1.366 ± 0.067</td>
<td>1.222 ± 0.056</td>
<td>Total sites:2800 Bleeding: 721 % 25.75</td>
</tr>
<tr>
<td>SD 0.3466</td>
<td>SD 0.295</td>
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<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scales</th>
<th>S1/PD No / %</th>
<th>S2/PD No / %</th>
<th>S3/PD No / %</th>
<th>S1/CAL No / %</th>
<th>S2/CAL No / %</th>
<th>S3/CAL No / %</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3\textsuperscript{mm}</td>
<td>98.67%</td>
<td>98.66%</td>
<td>95.64%</td>
<td>6.65%</td>
<td>7.35%</td>
<td>10.82%</td>
</tr>
<tr>
<td>3-5\textsuperscript{mm}</td>
<td>34</td>
<td>34</td>
<td>119</td>
<td>30</td>
<td>43</td>
<td>104</td>
</tr>
<tr>
<td>5-7\textsuperscript{mm}</td>
<td>2</td>
<td>2</td>
<td>11</td>
<td>7</td>
<td>6</td>
<td>14</td>
</tr>
</tbody>
</table>

Table 1: Records of PL.I and G.I mean, standard error and standard deviation, and BOP

Table 2: Number and Percent of PPD and CAL reading

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DISCUSSION
A cross-sectional tri-level study has been conducted to investigate the connection between the obesity as a systemic risk factor, with periodontopathy on one side and as a salivary chemicals modifying factor on other side, and to investigate the connection between salivary flow and some chemicals with periodontopathy. The reality of being the obesity as a conductive risk factor for many serious systemic diseases and to periodontopathy as well now is clear enough. An increasing prevalence of obesity is well documented in all ages and ethnicities worldwide. Obesity commonly accompanied by elements of the metabolic syndrome, including insulin resistance, hypertension, and dyslipidemia, is associated with increased risk of chronic inflammatory diseases such as periodontitis. Both periodontal disease and obesity are of multifactor etiology related to dietary habits but also closely correlated with socio-demographic background of the individuals. Most of the studies regarding association between obesity and periodontopathy are based on clinical data. Obesity is linked to chronic inflammation and a number of adipose-related pro-inflammatory cytokines, so called adipokines, are enhanced in

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plasma from obese subjects contributing to enhanced inflammatory response in many body organs. The immune system modulates central nervous system function particular by cytokines and the hypothalamic–pituitary–adrenal axis is reported to be dysregulated in subjects with obesity. The increased amount of adipokines from visceral fat may induce agglutination of blood in the microvasculature, decreasing blood flow to the Gingiva in obese people and facilitating the progression of gingival disease. Obese subjects in this study, showed increased PL.I, which is related to increase G.I, also related to increased severity of the disease shown by increased BOP. Obesity increases the host susceptibility by modulating the host immune and inflammatory system; this explain the increased PPD and CAL in obese group of our study, Wood et al 2003, reported that CAL and PPD, the indicator of periodontitis, were positively correlated with increased BMI. As well in the obese, the salivary flow showed significantly increased, more than that of other groups, Inoue et.al have explained that the salivary glands in obese subjects are more larger in size and may continuously stimulated by long term chewing process could lead to an increased flow. Salivary uric acid insignificantly reduced while the total protein was significantly elevated in value, this result could improve that obesity modify the proportional values of uric acid and proteins which are parts of plaque composites. The total protein was showed to be positively correlated with PL.I that is why the plaque of obese seemed to be softer and sticky.

REFERENCES
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