Possible Kefir Biological Effects: 4: Effect of Kefir Beverage on The Histopathological and Macroscopical Changes in Adipose Tissue of High Fat-Fed STZ- Induced Diabetic Male Wistar Rat

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ABSTRACT
This study was designed to investigate the effect of kefir consumption as a common dairy fermented product on keep the bodyweight balanced on high streptozotocin-induced diabetic wistar rats which fed with a daily kefir gavage also to examine any effects regards the abdominal circumference (AC) and body mass; observed results compared to normal male rats; Experiments were carried out on 60 albino male rats, with age 8 weeks, weighing about 220-250 g, after the adaptation period, male rats were divided randomly into two experiments by six groups: experiment I included 3 non-diabetic ones and experiment II included three induced diabetes groups. The groups were fed as follows: group 1 received a standard diet and served as control. Group 2 was fed on a standard diet and kefir (0.7 ml/animal/day by gavage). Group 3 received a high-fat diet and kefir (0.7 ml/animal/day by gavage). The diabetic males of groups A, B and C were fed a high-fat diet. Group B received in addition kefir (0.7 ml/animal/day by gavage). Group C was injected additionally with insulin (0.76 UI/200 mg BW/day); all the groups have the access to the drinking water all the time.; the bodyweight of i and diabetic rat males was determined on 1st treatment day, and every week until the end the experiment plan (5 weeks) then all the groups were sacrificed with measuring the AC and weighting the fat mass; Summarizing it could be said; through all the work plan (5 weeks), all the six animal groups showed a significant increase in the mean body weight with a normal level in experiment I unless in group 3 which fed only kefir, while in experiment II kefir helps the diabetic treated group to not weight compared to the untreated one; similarity kefir neither affect AC nor relative fat weight.

INTRODUCTION
The Middle East and North Africa region have the highest prevalence of DM in adults. Egypt has a prevalence range from 7.2 to 11% (Sherif and Sumpio, 2015). Now, Egypt is among the 10 "leading" countries in the world in terms of the number of people with DM.

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It is estimated that in 2025, DM prevalence will be 13.4%. The urban population within Egypt has a higher prevalence of DM (20%) compared to the rural population (4.9%) with no significant difference between genders (Bos and Agyemang, 2013).

Several studies suggest that measuring islet autoantibodies in relatives of those with type 1 diabetes may identify individuals who are at risk for developing type 1 diabetes. There is evidence to suggest that early diagnosis may limit acute complications (Ziegler et al., 2013).

The intestinal microbiota is a relevant therapeutic source for the treatment of different diseases. Although there have been proposed different strategies including pre/probiotics and fecal microbiota transplantation interventions (Ejtahed et al., 2016).

Kefir has been considered a probiotic due to its antioxidant and anti-inflammatory properties (Guven and Gulmez, 2003; Kwon et al., 2008).

In STZ-induced DM, it has been shown that daily administration of kefir caused an improvement in the increased levels of glycemia and glucose tolerance compared to conventional fermented milk (Yadav et al., 2008; Hadisaputro et al., 2012; Giovana et al., 2014). Besides drug treatment for diabetes; in recent years, many efforts have been made on traditional medicines as a complementary therapy in the treatment of diabetes. In this regard, probiotics have been considered in diabetic patients. (Guarner et al., 2005).

MATERAILS AND METHODS

1- Experimental Animals:

White male albino rats (Wistar rat) (Rattus norvegicus) from order Rodentia and family Muridae were used in the present study. Experiments were carried out on 60 albino rats, aged 8 weeks and weighing 220-250 gm. The animals were obtained from the ENVIGO Company, USA. IACUC Protocol Number (ORA use only): 2017-17.

Rats were kept in the Lab of Animal Research Facility (LARF) building, University of Idaho, USA, for 1 week under observation before experimentation to exclude any intercurrent infection and to acclimatize the animals to the new conditions. The selected animals were housed (3-4) in polycarbonate cages with softwood chips as bedding at a temperature of 23 ± 2°C, relative humidity of 50 ± 5% with good ventilation constant light/dark periods of 12 hours (hr.) each. Rats were either fed on standard rodent pellet diet (for groups 1,2) or fed with a high-fat diet (Sirrivasan et al., 2004), (groups (3, A, B, C), drinking tap water was provided at libitum for all groups.

### Composition of HFD:

<table>
<thead>
<tr>
<th>Ingredients Diet</th>
<th>(g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Powdered NPD</td>
<td>365</td>
</tr>
<tr>
<td>Lard</td>
<td>310</td>
</tr>
<tr>
<td>Casein</td>
<td>250</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>10</td>
</tr>
<tr>
<td>Vitamin and mineral mix</td>
<td>60</td>
</tr>
<tr>
<td>dl-Methionine</td>
<td>03</td>
</tr>
<tr>
<td>Yeast powder</td>
<td>01</td>
</tr>
<tr>
<td>Sodium chloride</td>
<td>01</td>
</tr>
</tbody>
</table>

Generally, the protocol followed the general guidelines of animal care. All efforts were made to minimize the number used and their suffering.

2. Induction of Diabetes Mellitus:

Diabetes mellitus was experimentally
induced in overnight fasted male animals by an intraperitoneal (ip) injection of streptozotocin (STZ) at the dose of 45 mg/kg (Judiono et al., 2011; Suharyo et al., 2012; Giovana et al., 2014). Streptozotocin was dissolved in cold 0.01 M citrate buffer, pH 4.5 and always prepared freshly for immediate use within 5 minutes. The normal control group was given a citrate buffer without STZ. The development of diabetes was confirmed after 48 hours – 7 days of STZ injection. The animals with fasting blood glucose levels of more than 200 mg/dl were considered diabetic and included in this study.

3. Animal Grouping:

Male rats were divided into six groups 10 animals each, 3 non-diabetic, and 3 diabetic groups:

**Experiment I:**

**Group 1-** Control animals, (negative group) were fed a standard diet plus oral administration of distilled water at a dose of 0.7 ml/animal/day.

**Group 2-** animals were fed a standard diet and received oral administration of kefir (0.7 ml/animal/day).

**Group 3-** Animals received a high-fat diet (HFD) and additionally oral administration of kefir (0.7 ml/animal/day).

**Experiment II:**

**Group A-** The diabetic group (positive group), was fed HFD and received oral administration of distilled water (0.7 ml/animal/day).

**Group B-** Diabetic animals received HFD plus oral administration of kefir (0.7 ml/animal/day).

**Group C-** Diabetic group, fed HFD, was injected insulin (0.76 UI/200 mg BW/day).

By the end of the experimental time of 5 weeks, animals of all groups fasted 4-6 hours, weighted, anesthetized by isoflurane, and sacrificed. The collected blood was centrifuged, and the serum stored at -80 °C until use. Tissues for the histological investigations were excised immediately, fixed in Formal saline 4% for and embedded in paraffin.

3. Experimental Studies:

1-Gross Morphology of Adipose Tissue:

Adipose tissue was dissected out and dried on a filter paper. The absolute weight of the organ was determined, and its relative weight was calculated.

2-Histopathological Examination of Adipose Tissue:

Adipose tissue was immediately excised. Small tissue blocks were prepared and fixed in 4% neutral buffered formalin, then transferred to Washington State University, Veterinary School, Pathological lab, Pullman, WA, USA, for complete tissue process, 5 µm sections were stained in specific dyes such as Haematoxylin and eosin stain.

3-Macroscopic Examination (General Morphology of The Body):

a- Body weight (gm)

The body weight was determined on 1st treatment day, and every week until the end of the experiment plan.

b- Weight Gain (gm):

Weight gain = \[(Final - Initial) \div Initial\] \times 100

c- Abdominal Circumference (cm):

The abdominal width was measured at the widest abdominal area at the end of the experiment.

4. Statistical Analysis:

Variables with a normal distribution were expressed as mean ± standard deviation. Variables with no normal distribution were expressed as median (25th - 75th percentile). One-Way ANOVA test was used for comparing groups mean of normally distributed variables. Four multiple comparisons between different groups were done using Post Hoc Tukey test was used. For not normally distributed variables, Kruskal-Wallis 1-way ANOVA test was used. Data were analyzed using SPSS (Statistical Package for Social Science) version 24 software. P value < 0.05 was considered significant.

RESULTS

1-Gross morphology of adipose tissue

The relative fat weight given in table 1 and figure 1 indicated non-significance change between the different groups in both experiments.
Table 1: Relative fat weight (gm) of two experiments (I and II) groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Groups</th>
<th>Experiment I</th>
<th>Experiment II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>1.51 ± 0.09</td>
<td>1.36 ± 0.11</td>
</tr>
<tr>
<td>Relative Fat weight (gm)</td>
<td>Group 2</td>
<td>1.40 ± 0.13</td>
<td>1.58 ± 0.19</td>
</tr>
<tr>
<td></td>
<td>Group 3</td>
<td>1.72 ± 0.10</td>
<td>1.65 ± 0.08</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>Group A</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Group B</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Group C</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are given as mean ± S.E.; means with the same superscript are not significantly different

** (p˂0.01): highly significant; * (p˂0.05): significant; NS: non-significant

**Group 1**: Control animals, (negative group) were fed a standard diet plus oral administration of distilled water

**Group 2**: Animals were fed a standard diet and received oral administration of kefir

**Group 3**: Animals received a high-fat diet (HFD) and additionally oral administration of kefir

**Group A**: Diabetic group (positive group), was fed HFD and received oral administration of distilled water

**Group B**: Diabetic animals received HFD plus oral administration of kefir

**Group C**: Diabetic group, fed HFD, was injected insulin

Fig. 1: Relative fat weight (gm) of two experiments (I and II) groups at the end of the experiment

2-Histopathological Examination of Adipose Tissue:

Figure 2 showed the pathological changes in the adipose tissue in the two different experiments.

**Experiment I**:

Adipose tissue of rat in C-N (group 1) revealed a fully-developed fat cell between pancreatic tissues, adipose tissue of rat in C-N + Kefir (group 2) and adipose tissue of rat CHFD+ Kefir (group 3) revealed full-developed fat cell.

**Experiment II**:

Similarly, there were no pathological lesions in the adipose tissues all over the experiment [I] (group A, B, C).
3-Macroscopic Examination (General Morphology of The Body):

a- Body weight (gm):

The bodyweight of normal and diabetic rat males was determined on 1st treatment day and every week until the end of the experiment plan (5 weeks) represented in table 2 and figure 3. In experiment I, the bodyweight for the three groups was in the same range from the first day of the experiment and didn’t appear any significance between them, through whole the experiment plan the bodyweight of the normal animals increased within the same level and reached 412 to 424 gram.

While in experiment II, the diabetic ones increased with low level and no significant between the groups unless in the last two weeks there was significance between group C and B, group C and A, group 3 and B; the bodyweight of the three groups reached 321 to 383 gram at the end of the experiment.

Summarizing, there was a marked increase in body weight in the six groups in both experiments at the end of the experiment (week 6) as compared to the initial body weight (week 0), the increased level was higher in the normal animals than the diabetic ones.

Fig. 2: Pathological changes in the adipose tissue in the different rat groups, H&E, bar= 200 µm.
Table (2): Bodyweight (gm) of two experiments (I and II) at different experimental periods.

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Experiments</th>
<th>Experiment I</th>
<th>Experiment II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
<td>Group 3</td>
</tr>
<tr>
<td>Body weight at 0 week</td>
<td>237.2 ± 3.27</td>
<td>246.5 ± 3.85</td>
<td>240.1 ± 4.23</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight at week 1</td>
<td>308.4 ± 8.75</td>
<td>336.9 ± 5.37</td>
<td>329.5 ± 5.91</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight at week 2</td>
<td>330.9 ± 11.01</td>
<td>333.0 ± 7.19</td>
<td>315.8 ± 5.70</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight at week 3</td>
<td>358.0 ± 12.75</td>
<td>360.4 ± 8.76</td>
<td>346.5 ± 7.56</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight at week 4</td>
<td>373.2 ± 12.75</td>
<td>386.5 ± 10.18</td>
<td>359.5 ± 8.12</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight at week 5</td>
<td>400.8 ± 12.34</td>
<td>410.3 ± 10.66</td>
<td>394.7 ± 9.82</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight at week 6</td>
<td>419.3 ± 14.72</td>
<td>424.3 ± 10.60</td>
<td>412.9 ± 10.57</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are given as mean ± S.E.; means with the same superscript are not significantly different:

** (p<0.01): highly significant; * (p<0.05): significant; NS: non-significant

Fig. 3: Bodyweight (gm) of two experiments (I and II) at different experimental periods.
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### b- Body Weight Gain (gm):

The body weight gain (gm) for each group was represented weekly throughout the experiment, data represented in table 3 and figures 4.

Through the five weeks, each group appeared significant increase in the body weight gain from the beginning of the experiment until the end with variable values, the represented data in experiment I showed non-significance in the body weight gain between the three groups, and through whole the experiment plan the body weight gain of the normal animals increased within the same level and reached 172 to 182 gram.

While in experiment II, the diabetic ones increased with the low rate with no significant between the groups unless in the last week there was significance between group C and A, also group 3 and B through whole the experiment plan; the body weight gain of the three groups reached 91 to 142 gram at the end of the experiment.

Summarizing, the body weight gain rate of the normal animals was more than the diabetic ones, also the body weight gain in group B which treated with kefir was less than the other treated with insulin.

**Table 3:** Bodyweight gain (gm) of two experiments (I and II) at different experimental periods.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Experiment I</th>
<th>Experiment II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
</tr>
<tr>
<td>Body weight gain at Week 1</td>
<td>71.20 ± 7.64</td>
<td>90.40 ± 2.36</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Body weight gain at Week 2</td>
<td>93.70 ± 8.65</td>
<td>88.50 ± 4.43</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Body weight gain at Week 3</td>
<td>120.80 ±10.85</td>
<td>113.90 ± 6.46</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Body weight gain at Week 4</td>
<td>136.0 ±10.67</td>
<td>140.0 ± 7.85</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Body weight gain at Week 5</td>
<td>163.60 ±10.22</td>
<td>163.80 ± 8.35</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Total body weight gain</td>
<td>182.10 ±12.46</td>
<td>177.90 ± 8.41</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

Data are given as mean ± S.E.; means with the same superscript are not significantly different

"" (p<0.01): highly significant; ‘(p<0.05): significant; NS: non-significant
Fig. 4: Body weight gain (gm) of two experiments (I and II) at different experimental periods.

C- Effect on Abdominal Circumference:

The mean abdominal width (cm) was measured at the widest abdominal area for all the animals per each group at the end of the experiment as represented in table 4 and figure 5.

According to the P value, there was no significance between the groups in both experiments I and II, the only significance was between group C and A.

Table 4: Abdominal circumference (cm) of two experiments (I and II) at the end of the experiment.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Experiment I</th>
<th>Experiment II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
</tr>
<tr>
<td>Mean abdominal circumference (cm)</td>
<td>17.77 ± 0.31</td>
<td>18.13 ± 0.34</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are given as mean ± S.E.; means with the same superscript are not significantly different

** (p<0.01): highly significant; * (p<0.05): significant; NS: non-significant

Figure 5: Abdominal circumference (cm) of two experiments (I and II) at the end of the experiment.
DISCUSSION

There is increasing evidence that the gut microbiome has a causal role in obesity and obesity-related metabolic disorders because of its influence on genes that regulate lipid metabolism, energy utilization, and storage (Davis, 2016).

High-fat diet (HFD) was regarded as a major determinant of obesity until it was found that the gut microbiota had a significant role in this phenotype. Gut microbiota plays a vital role in human energy homeostasis and metabolism of fat and cholesterol (Baothman et al., 2016). The gut microbial symbiosis is considered as an underlying reason behind obesity and metabolic syndromes while the balanced and healthy gut microflora is linked with health (Shabana et al., 2018).

Regarding the morphology of the adipose tissue, absolute weight was determined and mean relative weight was calculated; represented data indicated non-sig- nificance between the different groups; and that was expected cause of kefir beverage feeding had no effects in increasing fat mass formed, also there were no histopathological changes between the normal animals and diabetic ones. The number of studies included in the meta-analyses of the effects of probiotics on body fat mass and the percentage was low, thus meaning that the latter results should be interpreted with caution. The agreements go with the overall of the seven studies reporting changes in fat mass showed a larger reduction in body fat mass and fat percentage in the intervention groups compared with the control groups, but the difference was non-significant because effect sizes were small (Borgeraas et al., 2018).

However, the studies by Agerholm-Larsen et al. (2000) and Lee et al., (2014) were the only studies reporting either increased amounts of fat mass or lower reduction in fat mass in the intervention group compared with the control group.

Urdaneta et al., (2007); Sahin and Yardimci, (2009) showed that using kefir supplemented diet had no significant differences in the weight of the body organs examined.

In experiment I, feeding the normal animals with kefir in group 2 didn’t affect the bodyweight of the animals, it remains in the same level as in group 1, however, it was expected changes in the body weight in group 3 which fed on HFD and kefir, so kefir avoided any metabolic disorders.

While in experiment II, the three diabetic ones started with the same bodyweight but through the experiment, there wasn’t noticeable gaining weight cause of diabetic complications but we could notice the significance between group C and B; at the end of the experiment, group C treated with insulin showed more bodyweight than group B which treated with kefir and that means kefir had desirable consequences regarding the body weight in the diabetic animals; also it keeps animals accepting weight even their illness symptoms.

The weight gain in the trial groups was an expected result when previous studies considered it (Akbarzadeh et al., 2007; İşbilen et al., 2007); the bodyweight accepted by the normal male rats feed with beverage kefir were much higher than the treated diabetic ones.

After calculated the total body weight gain and the percentage, it explained as well the increase of the total BW gain and its percentage in the normal males treated with kefir more than the diabetic group which taking kefir. Some researchers showed the potential effects of the components of dairy products like kefir for decreasing the body weight (Zemel et al., 2000; Zemel, 2003; Zemel et al., 2004; Zemel et al., 2005; Teegarden, 2005; Mirmiran et al., 2005; Major et al., 2007; Shahar et al., 2007; Vergnaud et al., 2008; Van Loan, 2009; Sanders, 2012). A diet rich in dairy calcium intake enhances weight reduction in type 2 diabetic patients (Danit et al., 2007). Akbarzadeh. et al., (2007) explained that the bodyweight of the diabetic rats

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which induced experimentally by streptozotocin decreased comparing with the normal rats. Yasamin et al., (2016) observed in their study that Kefir drink leads to a similar weight loss, compared with milk in overweight or obese premenopausal women. A total of 13 studies revealed the effects of probiotic supplementation consumption and found that the administration of probiotics was associated with a significantly larger reduction in BMI and weight loss (Borgeraas et al., 2018). Zemel et al., (2004) hypothesized that a dairy-rich diet containing kefir drink would lead to a greater weight loss, as kefir drink might have the antiobesity properties of dairy products and probiotics in combination. Chen et al., (2012) did not support the beneficial effects of increasing dairy consumption on body weight and fat loss in studies without energy restriction. Previous reviews have found probiotics to reduce body weight in adults, and the reported effect level was small (Zhang et al., 2015; Dror et al., 2017).

Other studies found probiotics to increase weight in infants and children while having the opposite effect among adults (Dror et al., 2017). A different study observed that Kefir's treatment resulted in a significant increase in body mass gain (Fabiane et al., 2016).

Majority of randomized controlled trials (RCTs) have failed to show the potential weight-reducing effects of dairy products in the absence of energy restriction (Baran et al., 1990; Zemel et al., 2005; Palacios et al., 2011; Van Meijl and Mensink, 2010; Chen et al., 2012).

However, some studies observed that rat body weight gain was similar in both groups (control and kefir); No significant differences were found (Elena et al., 2007; Sahin and Yardimci, 2009; Atasoglu et al., 2010; Kizak and Celik, 2012; Aliakbarpour et al., 2013; Salaj et al., 2013; Piccolo et al., 2015).

Oral administration of viable strains of bacteria (probiotics) has been proposed as a way of enhancement the gut ecosystem to favour weight reduction or decrease weight gain; however, the mechanisms may influence gut microbiota are largely unknown (Sanders, 2016). In the meantime, several recent studies have, however, found probiotic supplementation to promote both weight gain and weight loss (Zhang et al., 2015; Drissi et al., 2016). Probiotics which provided in the form of fermented dairy products had a highly growing evidence regarding the significant contribution of gut microbiota to energy homeostasis and weight control (Arora and Sharma, 2011).

We showed that the mean length of the abdominal circumference which measured during the sacrifice process was nearly in the same range for all the rat males groups, there were no differences between the normal and diabetic ones; by another way no significance in the waist circumference (WC) in the untreated and treated groups.

Some studies involving adults have shown negative relationships between dairy intake and a variety of anthropometric indicators for general and/or central obesity; which were suggestive of inverse associations of total dairy intake with weight and WC (Wang et al., 2014).

In the other study, receiving an adequate-dairy in overweight or obese adults significantly reduced WC and body fat but had no effect on body weight (Stancliffe et al., 2011; Chang et al., 2011; Kadooka et al., 2013).

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