THE EFFECT OF HIGH-INTENSITY AEROBIC TRAINING ON SERUM LEVELS OF Adiponectin, Preptin AND INSULIN RESISTANCE IN PATIENTS WITH TYPE 2 DIABETES

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ABSTRACT

Background and purpose: Adiponectin is an adipocyte derived from adipose tissue that is involved in regulating glucose metabolism. The present study aimed to investigate the impact of high-intensity aerobic training on serum levels of adiponectin, preptin and insulin resistance in men with type 2 diabetes.

Methods: 24 men with type 2 diabetes who were able to participate in regular physical activity were voluntarily selected and randomly divided into two groups of intense aerobic exercise (n=12) and control (n=12). For 8 weeks, the experimental group performed 3 sessions of the training protocol each week. Changes in adiponectin, preptin, glucose, insulin, and insulin resistance were analyzed before and after the training via a covariance statistical test (P <0.05).

Results: An inter-group comparison showed that, unlike insulin levels (P=0.001) and insulin resistance (P=0.005) that significantly decreased, the values of adiponectin (P=0.067), preptin (P=0.097) and glucose (P=0.077) remained unchanged. However, the intra-group comparisons revealed a significant increase in adiponectin values (P=0.044) and a significant decrease in glucose (P=0.015), insulin (P=0.002) and insulin resistance (P=0.003) in the experimental group.

Conclusion: It appears that employing intense aerobic activity can reduce the effects of type 2 diabetes in the target tissue through expressing the adiponectin hormone and decreasing levels of preptin, glucose, insulin, and insulin resistance.

Keywords: Exercise Physiology, SportNutrition, Diabetes.

I. INTRODUCTION

Diabetes mellitus, simply known as diabetes, is a chronic metabolic disease characterized by high blood glucose levels, insufficient secretion or impaired insulin function (Farzanegi, 2017). Changes in lifestyle and insufficient physical activity may lead to diabetes. Of the main types of this disease, insulin-dependent diabetes, also known as type 2 diabetes, is the most common (Rezaeimanesh and Amiri Farsani, 2019). The main indicator of type 2 diabetes is hyperglycemia caused by insulin resistance, which eventually leads to vascular complications (Fizeľová et al., 2014).

Adiponectin is one of the adipocains closely related to obesity and insulin resistance. The average plasma concentration of this protein in individuals is 10 micrograms per milliliter (Xu et al., 2004) and about 0.01% of total plasma proteins. Mainly secreted by adipocytes, adiponectin is also produced to some extent by bone marrow, osteoblasts, fetal tissue, myocytes, cardiomyocytes, and salivary gland epithelial cells (Tumminia et al., 2019).

Adiponectin mediates its biological functions via three known receptors, AdipoR1, AdipoR2, and T-cadherin, which are distributed throughout the body. Biological functions of adiponectin are multifold ranging from anti-
diabetic, anti-atherogenic, anti-inflammatory to anti-cancer (Parida et al., 2019). Adiponectin appears to protect skeletal muscles from inflammatory damage induced by oxidative stress. It has been suggested that decreased adiponectin levels could be associated with pathologic conditions, including obesity and diabetes (Jiménez-Maldonado et al., 2019) as its low serum levels indicate the spread of diabetes (Bluher et al., 2006).

Exercise has been broadly recommended as an effective therapeutic strategy to prevent many obesity related metabolic disturbances (Arena et al. 2017). Numerous studies have been conducted on adiponectin concentrations and serum lipid levels, in most of which an inverse relationship has been reported between triglycerides, cholesterol, LDL and a positive relationship between HDL and serum adiponectin concentration (Kimmet al., 2010).

In most studies, the effect of moderate aerobic activity on adiponectin concentration has been considered to be minimal (Polak et al., 2006), while the effect of strenuous aerobic exercises has been reported to be positive in humans (Fatouros et al., 2005) and mice (Zenget al., 2007). However, weight loss, induced by caloric restriction alone or the combination of caloric restriction and exercise has showed inconsistent effects on circulating adiponectin concentrations (Wang et al., 2015).

Preptin is a new hormone that regulates energy consumption (Aydin, 2014). It is a peptide with 34 amino acids and is secreted by pancreatic beta cells along with insulin and amylin. It is involved in glucose homeostasis (Ramezankhani et al., 2015). Preptin and amylin are peptide hormones that are associated with insulin resistance (Baskaya Dogan et al., 2016).

Although the main synthesis of preptin is in pancreatic beta cells, it is also produced in several tissues, including the salivary glands, breast tissue, and kidneys. Together with other appetizing peptides, preptin plays an important role in the development of obesity and energy balance regulation by affecting the centers of hunger and satiety in the parenchymal nuclei and arch nuclei (Ozkan et al, 2013).

The primary information about preptin has been obtained from animal experiments. In 2001, Buchanan et al. reported that preptin regulated insulin secretion in response to glucose. The intravenous preptin secretion in rats reduced blood glucose and insulin secretion during glucose loading (Buchanan et al., 2001). Cheng et al. reported that preptin affects the insulin-like growth factor (IGF-2R), kinase protein C, and phospholipids C by activating the growth factor, and stimulates insulin secretion when glucose concentration is high (Cheng et al., 2012).

In the first clinical study of humans, the results showed that levels of preptin in diabetic patients were higher than in normal individuals. Although levels of preptin have a positive relationship with levels of triglycerides, total cholesterol, and insulin resistance, they have no relation with body mass index (BMI) and insulin. Also, the results of high-protein plasma levels were higher in women (Yang et al., 2009). However, the results of other studies reported a positive association between serum preptin and BMI levels (Ozkan et al., 2013).

Not only does physical activity create a negative energy balance, it also changes the secretion of hormones and peptides that affect energy balance (Maleki et al., 2016). Moreover, it causes major biochemical and physiological adaptations in the human body. Physical activity is the basis for the treatment of type 2 diabetes. Studies show that active people with fasting blood sugar have lower triglycerides, cholesterol, LDL, and HOMA-IR (Meex et al., 2015).

As exercise plays an important role in physical and mental health via reducing or modifying health risk factors, it has always been the main concern for experts, so much so that the American Diabetes Association (ADA) recommended that people with diabetes do regular aerobic exercises with moderate to high intensity (American Diabetes Association, 2017).

In this context, high-intensity interval training (HIIT) has arisen as an effective method to improve body composition in overweight and obese adults (Wewege et al. 2017) where the described advantages over more classic regimens (constant-moderate intensity) are time efficiency and enjoyability (Gibala et al. 2012).

Aerobic exercise forces the large muscles of the body to function regularly and continuously for a while. It can increase insulin action in each muscle fiber by increasing muscle blood flow, the number of capillaries, as well as enhancing the activity and increasing the number of GLUT4, hexokinase, and glycogen synthase (Hwang et al., 2011). High-intensity aerobic exercise can serve as a suitable alternative to traditional aerobic exercise, making even more significant changes in physiological, functional, and health indicators in adults and patients.
(Meka et al., 2008). Research shows that endurance training lowers the adipose tissue, heart rate, and blood pressure and improves glucose metabolism and lipid profile (Kelley et al., 2012).

As adiponectin is inversely related to obesity, insulin resistance, type 2 diabetes, and lipid profile and preptin is secreted in response to glucose levels, which regulates insulin secretion and is also linked to type 2 diabetes, their study may be important with regard to the possible factors associated with insulin resistance (Yang et al., 2009). However, the current knowledge about adiponectin and preptin is limited in biological systems. In the present study, high-intensity aerobic exercises were used to investigate their potential impact on serum levels of adiponectin, preptin, and insulin resistance in men with type 2 diabetes. We used high-intensity aerobic exercise because it is more effective on various health-related factors (e.g., body composition, lipid profile, blood pressure), metabolic variables, and insulin resistance than the low-intensity aerobic exercise (Miller et al., 2014).

II. METHODOLOGY

The research method was quasi-experimental research involving pretest-posttest design and control group. Subjects were selected voluntarily and purposefully from among male patients with type 2 diabetes aged 30-45 years referred to the clinic. The objectives and conditions of the study were explained to the volunteers and consent forms were given to them, if they agreed. Finally, they referred to a specialist physician for permission to participate in the research. In total, 28 men with type 2 diabetes who were able to participate in regular exercise programs were selected based on inclusion criteria and were randomly divided into two groups of training (n=14) and control (n=14). Sample size was calculated based on Fleiss's formula taking into account a power of 0.8, alpha of 0.05, and average changes of 5. It should be noted that during the research period, two individuals from each training and control groups withdrew their cooperation, causing the numbers in each group to drop to 12. The training group performed high-intensity aerobic exercise for 8 weeks and 3 sessions per week. However, the subjects in the control group continued their sedentary lifestyle during the study period. The training group withdrew their cooperation, causing the numbers in each group to drop to 12. The training group performed high-intensity aerobic exercise for 8 weeks and 3 sessions per week. However, the subjects in the control group continued their sedentary lifestyle during the study period. The training group withdrew their cooperation, causing the numbers in each group to drop to 12. The training group performed high-intensity aerobic exercise for 8 weeks and 3 sessions per week. However, the subjects in the control group continued their sedentary lifestyle during the study period.

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III. TRAINING PROTOCOL

One week before the start of the training protocol, the participants came into the laboratory to learn how to implement the protocol. After dividing the participants into training and control groups, height, weight (using a stadiometer and a scale, respectively), and BMI (weight in kg dividing by height squared in m) were measured.

According to the ADA, the maximum heart rate occurred within 3 sessions of intense aerobic training per week with an intensity of 70 to 80% (American Diabetes Association, 2017). In the first session, the training time was 25 minutes, then one minute was added to every following session, reaching 42 minutes at the end of the sixth week. Finally, the training time reached a fixed time of 43 minutes during the seventh and eighth weeks. The intensity of the training started from 70% of the maximum heart rate and was increased by 5% every three weeks until it reached 80% of the maximum heart rate and maintained a steadystate (Table 1). After warming up, which consisted of 10 minutes of slow running and 5 minutes of active stretching, the subjects performed the main training, i.e. the aerobic exercise. At the end of each training session, the subjects walked for 10 minutes to cool down and performed soft and stretching exercises.

<table>
<thead>
<tr>
<th>Week</th>
<th>Saturday</th>
<th>Monday</th>
<th>Wednesday</th>
<th>Maximum heart rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>25</td>
<td>26</td>
<td>27</td>
<td>70</td>
</tr>
<tr>
<td>Second</td>
<td>28</td>
<td>29</td>
<td>30</td>
<td>70</td>
</tr>
<tr>
<td>Third</td>
<td>31</td>
<td>32</td>
<td>33</td>
<td>70</td>
</tr>
<tr>
<td>Fourth</td>
<td>34</td>
<td>35</td>
<td>36</td>
<td>75</td>
</tr>
<tr>
<td>Fifth</td>
<td>37</td>
<td>38</td>
<td>39</td>
<td>75</td>
</tr>
<tr>
<td>Sixth</td>
<td>40</td>
<td>41</td>
<td>42</td>
<td>75</td>
</tr>
<tr>
<td>Seventh</td>
<td>43</td>
<td>43</td>
<td>43</td>
<td>80</td>
</tr>
<tr>
<td>Eighth</td>
<td>43</td>
<td>43</td>
<td>43</td>
<td>80</td>
</tr>
</tbody>
</table>

Table 1. Training Protocol
Biochemical Analysis

Blood samples were taken 48 hours prior to the training and 48 hours after the last training session at the laboratory. 10 cc of blood was taken from each group by a specialist in accordance with the principles of hygiene. The samples were poured into special serum tubes and, after centrifugation and separation of the serum, were frozen at -80 °C and used to measure the research variables. We used the Czech Biovendor to measure the concentration of adiponectin by ELISA, the preptin kit of the German company Zell Bio with a sensitivity of 5 ng / ml to measure the preptin concentration. Fasting blood sugar was measured using Hitachi 902 Auto analyzer (Hitachi, Japan) and Glucose Pars kit (Iran). Plasma insulin was measured using ELISA method and Monobind Inc kit, with a sensitivity of 0.75 micro units per ml and a coefficient of intragroup changes of 6.3%. To calculate insulin resistance, we used the HOMA-IR homeostasis model evaluation method. The fasting glucose and insulin were calculated based on the following formula:

\[ \text{HOMA-IR} = \frac{[\text{Fasting insulin (µU/ml)} \times \text{Fasting glucose (m mol/ l)}]}{22.5} \]

Statistical Analysis

The Shapiro-Wilk test was used to determine whether or not the data distribution was normal, and the Levon test was used to investigate the equivalence of variance. After assuring that the data were normal, the statistical test of covariance (ANCOVA) and t-pair were used at \( P<0.05 \) level. Data analysis was performed via SPSS software version 23.

IV. RESULTS

Physical characteristics and body composition measurements of participants at baseline and post-intervention are included in Table 2. Participants in the training and the control groups were of a similar age and height. There were no group differences in body weight and BMI at baseline. Body weight \( (P=0.001) \) and BMI \( (P=0.001) \), there was a significant decrease in the training group in post-intervention.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group</th>
<th>Post-test Mean and SD</th>
<th>Pre-test Mean and SD</th>
<th>P In-group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Training</td>
<td>35.1±4.1</td>
<td>35.1±4.1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>34.7±5.5</td>
<td>34.7±5.5</td>
<td>-</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>Training</td>
<td>179±6.8</td>
<td>179±6.8</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>177±6.4</td>
<td>177±6.4</td>
<td>-</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>Training</td>
<td>81±6.1</td>
<td>77.9±5.3</td>
<td>0.001*</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>80.2±4.3</td>
<td>79.8±3.8</td>
<td>0.269</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>Training</td>
<td>25.4±1.6</td>
<td>24.4±1.5</td>
<td>0.001*</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>25.5±1.1</td>
<td>25.6±1.2</td>
<td>0.309</td>
</tr>
</tbody>
</table>

*In-group Statistical significance

Statistical analysis of the data showed that after 8 weeks of intense aerobic exercise at the resting levels of adiponectin \( (F_{1,21}=3.72, \ P=0.067) \), there was no significant difference (Fig.1). Intragroup changes showed that there was a significant increase \( (CV=10.7\%) \) between the mean before and after the training period in the training group \( (P=0.044) \). Also, the study of preptin-related data \( (F_{1,21}=3.02, \ P=0.097) \) showed that there was no significant difference between the changes of the two groups after the training period (Fig.2). A study of intragroup changes in the preptin index also showed that there was no significant difference between the groups \( (P>0.05) \).
In the glucose index, there was no significant difference between the changes in the two groups after the training period (\(F_{1,21}=3.47, \ P=0.077\)). Intragroup changes showed that there was a significant decrease (CV=8.5%) between the mean before and after the training period in the training group (P=0.015). However, in the insulin index (\(F_{1,21}=13.81, \ P=0.001\)) and insulin resistance (\(F_{1,21}=9.6, \ P=0.005\)), there was a significant difference between the two groups. Intragroup changes observed in the insulin(CV= 6.5%) (P=0.002) and insulin resistance (CV=15.4%) (P=0.003) showed that there was a significant decrease between the mean before and after the training period (Table 3).

**Table 3.** Investigation of Inter- and Intra-group Changes in Variables of the Two Groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group</th>
<th>Pre-test Mean and SD</th>
<th>Post-test Mean and SD</th>
<th>In-group tP</th>
<th>Intergroup FP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adiponectin (ng/ml)</td>
<td>Training</td>
<td>11.2±1.7</td>
<td>12.4±2.2</td>
<td>2.27*0.044</td>
<td>3.720.067</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>11.3±1.7</td>
<td>11.4±1.8</td>
<td>0.420.68</td>
<td></td>
</tr>
<tr>
<td>Preptin (ng/L)</td>
<td>Training</td>
<td>1851±162</td>
<td>1791±176</td>
<td>2.130.057</td>
<td>3.020.097</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1848±139</td>
<td>1840±147</td>
<td>0.840.42</td>
<td></td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>Training</td>
<td>160.8±22</td>
<td>146.3±15.5</td>
<td>2.86*0.015</td>
<td>3.470.077</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>152.1±21.8</td>
<td>149.2±20.4</td>
<td>1.530.16</td>
<td></td>
</tr>
<tr>
<td>Insulin (IU/ml)</td>
<td>Training</td>
<td>21.5±5.7</td>
<td>20.1±4.7</td>
<td>3.95*0.002</td>
<td>13.81¥0.001</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>21.3±5.8</td>
<td>21.1±5.4</td>
<td>1.150.28</td>
<td></td>
</tr>
<tr>
<td>Insulin resistance (HOMA)</td>
<td>Training</td>
<td>8.39±2.1</td>
<td>7.1±1.4</td>
<td>3.79*0.003</td>
<td>9.6¥0.005</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>7.83±1.8</td>
<td>7.62±1.7</td>
<td>1.590.14</td>
<td></td>
</tr>
</tbody>
</table>

*In-group Statistical significance; ¥ intergroup Statistical significance

**V. DISCUSSION AND CONCLUSION**

High-intensity aerobic exercise is highly beneficialas it can help prevent or control type 2 diabetes. The findings of the present study showed that 8 weeks of high-intensity aerobic exercise did not lead to a significant change in the subjects' adiponectin, preptin, and glucose levels, whereas significant changes were observed in their insulin and insulin resistance indices.

Diabetes is among the most critical factors in serum adiponectin concentration, causing lower levels of serum adiponectin concentration indiabetic patients(Punyadeeraet al., 2005).
A study of adiponectin measurement data showed that the rate of intergroup changes, though increased by about 9%, was not significant after eight weeks of intense aerobic exercise. However, intragroup changes revealed a significant increase between the pre- and post-exercise mean in the exercise group. The findings of this study are consistent with the results of previous studies (Zarei et al., 2017; Johannsen et al., 2013; Aly, 2014; Wang et al., 2015).

The effects of exercise alone on adiponectin concentration are also inconsistent. Zarei et al. (2017) examined the effect of three exercise programs on adiponectin levels and reported the insufficiency of weight loss as one of the most important causes of insignificant adiponectin change (Zarei et al., 2017). However, Choi et al. (2013) found no critical changes in adiponectin levels in a 12-week study of combined exercise for obese women, despite weight loss and body mass index. Numa et al. (2012) reported adiponectin changes as consistent with high-density lipoprotein changes. They believed that adiponectin could regulate the high-density lipoprotein concentrations by secreting and refining apo-protein-1 in the liver (Numa et al., 2012).

Most studies suggested that adiponectin levels are reduced only in patients who have experienced significant weight loss. At least 10 percent weight loss is necessary to increase serum adiponectin concentration (Monzillo et al., 2003). Therefore, the amount of weight loss in the present study may not have been sufficient to alter adiponectin concentration. Some researchers believe the more muscle mass and volume are used in exercise, the stronger the likelihood of adiponectin serum changes will be. Adiponectin is believed to function as a signal between adipose tissue and muscle. By activating AMPK, adiponectin increases the oxidation of free fatty acids in muscle. AMPK activity, on the other hand, depends on the muscle mass involved (Jurimae et al., 2006).

Exercise and caloric restriction induced weight loss may function via different mechanisms to influence adiponectin concentration. One study showed that mRNA expression of adiponectin receptor 1 and 2 in skeletal muscle significantly increased in exercise trained individuals, but not in those undergoing a hypocaloric diet (Christiansen et al., 2010). Perhaps if a training program uses more muscle mass than the protocol used in this study (such as resistance training), it will have a greater effect on serum adiponectin concentration. In addition, lack of control over genetic factors and other factors affecting diabetes-independent research may have affected the results in the present study.

The study of preptin measurement data showed that the intergroup changes were not significant, despite a 2.9% decrease. Intra-group data did not show any significant changes, although its value in the exercise group decreased by 3.2%, compared to the pre-research period.

Analyzing these studies, it could be argued that the changes in the exercise group may reflect the changes caused by weight loss and BMI, because previous research reported a positive relationship between preptin, BMI, and obesity. There have been limited investigations on the effect of physical activity on preptin. Elevated serum preptin has been reported in overweight and obese adults (El-Eshmawy et al., 2015), but we have not found any published data on changes in preptin synthesis following body mass reduction. In a study by Ramazan Khani et al. (2015), in line with the results of the present study, a decrease in preptin was reported after 16 weeks of exercise (Ramezankhani et al., 2015). Nazar Ali et al. (2019) examined the effect of high-intensity circular training on preptin and insulin resistance, concluding that six weeks of intense circular exercise affected the serum protein levels in women (Nazarali et al., 2019).

Accordingly, high-intensity exercise improves the skeletal muscle metabolism and anaerobic capacity, increasing muscle glycogen and glucose-4 membrane transport (GLUT4). Part of the adaptation to skeletal muscle is due to the intensity of the training because the intensity of physical activity is the main factor in increasing PGC1-α, the main enzyme of mitochondrial degradation. On the other hand, hormones produced from adipose tissue play a vital role in regulating and consuming energy, metabolism of fats, and carbohydrates through the production and secretion of adipocytes (Golizadeh et al., 2017). Preptin, as one of these peptides, appears to play a compensatory role in the process of maintaining blood glucose homeostasis along with insulin. Therefore, by increasing the capacity and sensitivity of muscle cells to insulin due to high-intensity exercise, this compensatory need is decreased and the production and secretion of preptin are reduced (Nazarali et al., 2019).

Another important effect of intense aerobic exercise on glucose homeostasis and blood glycemic factors is lowering fasting blood glucose, and insulin as well as improving insulin resistance index. In the present study, all three variables were reduced following the aerobic exercise training, although this reduction was merely significant for insulin and insulin resistance. Physical activity increases insulin sensitivity and glucose tolerance.
and thus lowers blood sugar (Rezaeimanesh and Amiri Farsani, 2019). Physical activity also increases glucose uptake by active muscles. This mechanism is mediated by stimulation and transfer of GLUT-4 to the cell membrane and rapid removal of glucose by active muscles through protein carriers (Arora et al., 2009).

Several mechanisms have been suggested to reduce insulin resistance after exercise. These mechanisms include increased insulin messaging and available receptors (Henriksen, 2002), increased glycogen synthase and hexokinase activity (Gilardiniet al., 2006), decreased release of free fatty acids and increased clearance (Henriksen, 2002), and finally increased glucose delivery to muscle and changes in its composition. Insulin resistance is the insufficient response of insulin-sensitive tissues (e.g. the liver, adipose tissue, and muscles) to insulin levels. The increased production of cytokines and fatty acids activates the inflammatory pathways in immune and metabolic cells. Activation of inflammatory pathways interferes with the insulin signaling pathway leading to insulin resistance (Rezaeimanesh and Amiri Farsani, 2019).

Physical activity can affect glucose homeostasis and reduce insulin resistance by increasing insulin signaling function, glucose transporters from within the cell membrane, glucose uptake rate, capillary density, gene expression or protein activity involved in insulin signaling, glycogen synthase activity, and ultimately increasing the glycogen storage (Arora et al., 2009). Also, this type of exercise leads to the reduction of body mass index (BMI) and body fat percentage, which can be considered as an important indicator in improving health.

In general, it is possible to control diabetes and the associated risks in a variety of ways. Today, experts believe that diet is not merely enough to control the disease, and hence physical activity must be added to the daily routine of people with type 2 diabetes. A regular, high-intensity physical activity can play a major role in reducing the effects of type 2 diabetes and increasing insulin sensitivity in target tissue by expressing adiponectin and reducing protein, glucose, insulin, and insulin resistance.

### VI. ACKNOWLEDGMENTS

The authors thank the participants for their enthusiastic contribution in the research and training sessions.

This study has been conducted as part of a research project under contract No. 176, dated 2020/3/11, funded by Khorramshahr University of Marine Science and Technology.

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