The Effects of Watermelon Powder on Risk Factors of CVD and Inflammation in Atherogenic-Diet Fed Rats

Running Head: Effects of watermelon powder on CVD and inflammation

Authors:
Mee Young Hong, PhD
Alexis Beltran
Carly Sullivan

School of Exercise and Nutritional Sciences, San Diego State University, San Diego, CA 92182

Corresponding Author:
Mee Young Hong, PhD
Department of Exercise and Nutritional Sciences
San Diego State University
5500 Campanile Drive
San Diego, CA 92182-7251
(619) 594-2392
mhong@mail.sdsu.edu
Abstract

Objective
The objective of this study was to determine if watermelon improves the risk factors of CVD and inflammation in atherogenic diet-fed rats.

Methods
Fourth male Sprague Dawley rats were equally divided into four groups via the zigzag method. There were two diets and two treatments, which consisted of: control-no Dextran Sodium Sulfate (DSS), control-DSS, watermelon-no DSS, and watermelon-DSS. After four weeks rats were euthanized. Organs were collected for weight and blood was collected for triglyceride, total cholesterol, LDL-cholesterol, HDL-cholesterol, C-reactive protein (CRP), and total antioxidant capacity assessment.

Results
Watermelon groups had a significant decrease in LDL-cholesterol and total cholesterol. Watermelon groups also had an increase in HDL-cholesterol, decrease in CRP and LDH compared to the control groups not fed a watermelon diet (p<0.05). Groups treated with DSS had lower water and food intake than rats than consumed the watermelon powder diet. Spleen weights were higher for the DSS treated groups. Kidney weights were lower for the DSS treated groups and higher for the watermelon groups compared to the two control groups.

Conclusion
Watermelon powder reduces the risk of CVD by favorable changes of lipid profile, inflammation and antioxidant status.

Key Words: atherogenic; watermelon; inflammation; cardiovascular disease; cholesterol
Introduction

Cardiovascular Disease (CVD) is the number one cause of death in the United States (1). It was the cause of 1 out of every 6 deaths in the United States in 2007 and approximately every 25 seconds an American will have a coronary event (2). New research is constantly being conducted on new ways to help prevent and treat CVD and its risk factors including inflammation and serum lipid profile.

In vivo studies are being conducted on watermelon as a natural weapon for its ability to lower the risk factors against CVD (3). A pilot study at Florida State University found that watermelon is effective against prehypertension, one of the precursors of cardiovascular disease. This study found that when six grams of L-arginine/L-citrulline from watermelon extract were given daily to pre-hypertensive subjects for a period of six weeks, arterial function and lower aortic blood pressures were achieved (3).

Inflammation plays an important role in CVD. Similar to this study, the effects on green tea were studied on Sprague Dawley rats. They were administered an atherogenic diet and Dextran Sodium Sulfate (DSS) treatment. This study concluded that green tea supplementation decreased many of the risk factors for cardiovascular disease including inflammation and cholesterol. This study supports the use the green tea as a dietary component for cardiovascular health (4).

In addition to the cardiovascular benefits of provided by the citrulline, watermelon also provides powerful antioxidants including vitamin A, B6, C, and lycopene (3). A research study conducted in 2011 did a comparative evaluation of the antioxidant effects between orange juice and watermelon juice and their effect on serum lipid profiles Wister albino rats. This study concluded that watermelon caused a dose related decrease in superoxide dismutase activity (SOD). Both juices showed a dose related increase in HDL-cholesterol with no significant
difference (p>0.05) but only watermelon cause a dose related decrease in LDL-cholesterol. Orange juice even caused a dose related increase in LDL-cholesterol (5). This demonstrates the possible importance of watermelon consumption as a preventative measure for CVD.

Similar to the green tea study conducted the objective of this study was to determine if watermelon improves the risk factors of CVD and inflammation in atherogenic diet-fed rats. Our hypothesis was that watermelon powder reduces the risk factors of CVD and inflammation by increasing antioxidant and favorable changes of lipid profile and inflammation markers. Materials used in the study included a DSS treatment to induce inflammation and watermelon powder as the source of watermelon in the diet.

**Methods**

*Animals and Diets*

In a research room at San Diego State University, forty male twenty-one day old Sprague-Dawley rats were housed individually in wired cages. Both temperature and humidity were controlled at approximately 20-24°C and 40-45% humidity. The San Diego State University animal subjects committee approved the procedures and training for the use of the rats in the study (6).

The forty rats were divided into four groups using the zigzag method. Each group contained ten rats, which were given diets and treatments with or without watermelon powder (Milne Fruit Products, Prosser, WA). The four groups included two diets and two treatments. The control, group 1, was given a treatment of no Dextran Sodium Sulfate (DSS) and atherogenic diet, which consisted of a placebo in place of the watermelon powder. The placebo was made up of a sucrose, glucose, and fructose ratio of 2:2:1. Group 2 received DSS, which was used to induce inflammation, and the atherogenic placebo diet. Group 3 was not given DSS and
was given the atherogenic diet with watermelon powder. Group 4 received the DSS treatment and the atherogenic diet with watermelon powder. The two experimental diets were composed of 33% sugar, 21% fat by wt, and 3% cholesterol by weight. The two sources of fat in the diet were corn oil (5 grams) and dairy butter (16 grams). The entire list of ingredients that made up the experimental diet can be found in Table 1. The atherogenic diet for the control group had a placebo, which was a maltodextrin used in place of watermelon powder to match the kilocalories of the watermelon powder. The placebo consisted of a 2:2:1 ratio of sucrose, glucose, and fructose. For the diet, which consisted of watermelon powder, 0.20 grams of watermelon powder were used. The watermelon powder used in the experimental diet consisted of sieved and freeze-dried watermelon solids.

Each rat was given 100.2 grams of food and every forty-eight hours the spillage was measured to account for the food the rats did not consume. The amount of water drank was also measured. The rats were given 2-3 days before the new diets were administered to the rats. For four weeks the rats were given their assigned diets and treatments. The weight of the rats, weight of food, and weight of water were all measured for four weeks. For forty-eight hours after the four experimental week was when the DSS treatment was given to two groups. Group 2, control, and Group 4, watermelon diet group were given a DSS treatment of 3% DSS. After those forty-eight hours the rats were given regular water and then were euthanized. Before euthanasia the rats were weighed to record their final wt and food and water intake was recorded.

Following euthanasia the rats were dissected and the spleen, kidneys, liver, and epididymal fat were removed and weighed for comparison. Blood samples were collected and from each rat and multiple tests were conducted. The tests included serum total cholesterol, HDL-cholesterol, triglycerides, serum LDL-cholesterol, CRP, LDH, and total antioxidant level.
Serum total cholesterol, HDL-cholesterol, triglycerides, glucose, and LDH were assessed using kits from Stanbio (Boerne, TX). Serum LDL-cholesterol was assessed by calculation, CRP from BD Biosciences (San Jose, CA), and total antioxidant levels test from Sigma (St. Louis, MO).

The Stanbio kit was used to measure the serum cholesterol, HDL-cholesterol, and triglycerides from the rats’ blood. LDL-cholesterol was determined by using the calculation of

\[
\text{Cholesterol} - \text{HDL} - \frac{\text{TG}}{5} = \text{LDL (mg/dL)}.
\]

Also, Serum Lactate Dehydrogenase (LDH) was measured. The increase of LDH can show signs of myocardial infarction, liver disease, pernicious and megaloblastic anemia, and muscular dystrophy. The LDH reagent composed of 75.0 mM L-lithium lactate, NAD 5.5 mM, 80.0 mM of buffer with a pH of 9.0±0.1, and non-reactive stabilizers and fillers. For each sample of blood, 1.0 mL of reconstituted reagent was added. 25 mL of serum was combined with 1.0 mL of the reagent and then incubated for one minute at 37°C. The spectrometer was zeroed at 340 nm and the results were read after 60 seconds.

The amount of glucose in the serum was measured by using the Stanbio Glucose LiquiColor test. For this assay, glucose oxidase is added to the serum, which causes the glucose to oxidize and forms hydrogen peroxide. The hydrogen peroxide, phenol, and 4-aminoantipyrine produced under the catalyst of peroxidase forms a red pigment after being incubated and put in the spectrometer at 500 nm. The darker red pigment produced results in a higher concentration of glucose in the serum. Therefore, a pungent color is directly proportional to the glucose concentration.

C-Reactive Protein (CRP) Elisa kit was used to test the CRP in the rat serum. CRP is a protein that is produced by the liver if there is inflammation, tissue trauma, or bacterial infection. Also, CRP in the serum is a sign of cardiovascular disease. The serum samples were added to a
96 well plate which was coated with antibodies. CRP present in the serum would bind to the immobilized antibodies on the well plate. After incubating the serum and CRP standard for 30 minutes at room temperature, well plates were washed and a horseradish peroxidase conjugated anti-rat CRP, which was used to create a “sandwich” between the antibodies and antigens. Lastly, after another incubation the well plate was washed again and substrate was added which would produce a color for the spectrometer to read at 450 nm. A blue color produced would indicate presence of CRP. The darker the blue the more CRP present and if the sample is colorless the sample does not have any presence of CRP.

Antioxidant assay (total antioxidant capacity) test was used to measure the total concentration of antioxidants present in the serum. Free radicals and reactive oxygen are produced during many reactions in the body, which can cause serious physiological damage, which makes antioxidant concentration very important. Antioxidants are present in order to provide an electron in order to remove the free radical. A Trolox Standard, ABTS Substrate, hydrogen peroxide, and serum were added to the well plates. The addition of hydrogen peroxide and metmyoglobin is used to produce ferryl myoglobin, which is a free radical. The ferryl myoglobin is used to oxidize the ABTS to make a ABTS radical cation. The well plates were incubated for 5 minutes at room temperature, then a stop solution was added to each sample and samples were read at 405 nm. If there was a sufficient concentration of antioxidant in the serum, the level of ABTS cation radical would be eliminated or removed and the color intensity read by the spectrometer would be decreased.
Statistical Analysis

The data was analyzed by ANOVA procedure using SPSS (IBM, Armonk, New York). The effects of diets and treatment on weight, food intake, water intake, lipid profiles, CRP, LDH, and total antioxidant were analyzed using SPSS. Data is presented by Mean±SE and an alpha level of p<0.05 will show significance (6).

Results

Initial body weights of all 40 Sprague Dawley rats were recorded and all groups had a similar starting weight value. Final body weights right after the treatment for the control-no DSS and watermelon-no DSS had a lower body weight than those of those treated with DSS (p=0.003). On the last day final body weights were lower for the control-DSS and the Watermelon-DSS treated rats (p=0.007). There was not a significant difference in weight gain between the groups prior to DSS treatment. During treatment control-DSS and watermelon-DSS had lower body weights than non-DSS treated groups (p=0.001). Overall, DSS treated groups had lower body weights (p=0.03) (Table 2).

Before treatment all groups had similar food intake. During treatment, DSS treated groups had a reduced food intake before treatment and a lower amount of food intake in comparison to non-DSS treated groups (p<0.01). After treatment, all groups increased food intake but intake was not statistically different among groups.

Before treatment water intake was the same for the control and the watermelon groups. During treatment water intake was lower for DSS treated groups than the non-DSS treated groups (p<0.001). After treatment DSS treated groups had a higher water intake than non-DSS treated groups (p=0.020) (Table 3).
Spleen, kidney, and epididymal fat were measured after euthanasia. Liver and epididymal fat were similar among all groups and were not statistically different. Spleen weights for non-DSS treated were greater than DSS treated (P=0.049) (Figure 1). Watermelon group has a higher kidney weight than control groups (p=0.024). DSS treated rats had lower kidney weights than non-DSS treated rats (p=0.019) (Figure 2).

Glucose serums among all diets and treatments were similar and therefore not significant. Watermelon diet fed rats had lower triglyceride levels than the control groups (p=0.031). The watermelon groups had lower total cholesterol values compared to the control groups (p<0.001). DSS treatment increased total cholesterol in both control and watermelon treated groups (p=0.011).

Watermelon and control groups treated with DSS had lower HDL cholesterol (p=0.041). LDL cholesterol was lower in watermelon diet fed rats (p<0.001) and DSS treated had a higher LDL than rats not treated with DSS (p=0.008) (Figure 3).

Animals who were fed watermelon had a much lower LDH than the control groups who were not given watermelon as part of their diet (p<0.001)(Figure 4). Among the four different group, creatine kinase (CK) was similar and did not have a significant difference. Total antioxidant concentration was higher in the watermelon fed rats than the control group rats (p=0.049)(Figure 4). DSS treatment lowered the antioxidant capacity compared to the none DSS treatment (p=0.015)(Figure 5). Watermelon groups had a lower C-reactive protein than the control groups. While the DSS treatment elevated the C-reactive protein in the groups (Figure 6).

**Discussion**

Based on the results of this study there is a direct correlation between adding watermelon powder to an atherogenic diet and reducing the risk of CVD. The rats given watermelon powder...
along with an atherogenic diet had favorable changes of lipid profiles, inflammation and antioxidant status. Specifically, the rats who consumed watermelon powder had lower triglycerides, lower total cholesterol and LDL-cholesterol. In addition the groups that were fed watermelon powder had higher HDL-cholesterol, lower LDH, and higher antioxidant capacity, and much lower levels of C-reactive protein.

These results yield reduce risk factors for CVD. By having higher HDL cholesterol in the watermelon groups, the HDL cholesterol removes LDL cholesterol from the arteries and helps prevent atherosclerosis, which decreases the risk of CVD. The control groups also had a significantly higher amount of CRP, which suggests that an atherogenic diet elevates inflammation levels when compared to the diets with watermelon powder. Also, LDH was significantly higher in the two control groups, which suggests that the atherogenic diet could have increased risks for myocardial infarction or inflammation of certain organs.

In a similar study, the effects of green tea were studied when Sprague Dawley rats were administered an atherogenic diet and DSS treatment in their water. This study tested the green tea polyphenol’s possible antioxidative and anti-inflammatory effects. Markers including CRP, LDL-cholesterol, HDL-cholesterol, and total cholesterol were measured. This study found that the DSS treated had higher LDL cholesterol and total cholesterol. Our experiment yielded the same results in the DSS treated groups. Our results also matched up with the DSS treatment in that HDL cholesterol was decreased when inflammation was induced through the DSS treatment (4).

Proving the powerful capabilities of the compounds found in green tea and watermelon, the green tea study found that adding green tea polyphenols to the atherogenic diet lowered total and LDL cholesterol and raised HDL cholesterol. The green tea diet was also associated with
lowered CRP concentration and increased antioxidant capacity (4). Proving the same results in our study, the DSS treated groups also had higher total and LDL cholesterol and a decrease in HDL cholesterol. In addition, the watermelon groups had an increase in HDL cholesterol, antioxidant capacity, and CRP concentration and a decrease in total and LDL cholesterol.

Both green tea and watermelon possess powerful antioxidants. Each food component possess different antioxidants but overall any fruit or drink that contains a sufficient amount of these reducing agents can contribute to reducing the risk factors of CVD and inflammation. It can be concluded that the green tea lowers total and LDL cholesterol, raised HDL cholesterol, and was associated with decreased serum CRP concentrations similar to our results with watermelon.

In a similar study, the influence of the ingestion of L-arginine, L-citrulline, and antioxidants was studied in rabbits fed a high cholesterol diet. At the end of the 12-week study, after being fed L-arginine plus L-citrulline, alone or in combination with antioxidants, results showed an improvement in vasorelaxation and blood flow and a decrease in superoxide production. This study concluded that certain Nitric Oxide (NO)-boosting substances including L-arginine, L-citrulline, and antioxidants can get rid of oxidative stress and reverse progression of atherosclerosis (7).

In a recent review, NO research was done and evaluated the effects of NO on different diseases. Nitric Oxide Synthase produces NO, which then induces vasodilation of the arteries. NO is an important component of the conversion of Arginine to Citrulline cycle. It has been shown that high saturated fat content induces high plasma fatty acid levels, which decreases NO production. Also, studies have shown that giving hypertensive people arginine supplementation can increase NO production and relieve symptoms. Therefore, watermelon powder, which
contains citrulline, may speed up the Arginine-Citrulline Cycle, which then can increase NO production and reduce the risks for CVD and inflammation (8).

This study was similar to the one done in the SDSU laboratory. Instead of rabbits being used as the subjects, the watermelon powder study used rats as subjects. The study fed the rats an atherogenic diet just like the rabbits were fed, but the rats were not directly fed L-Citrulline or L-Arginine supplements. Two groups of rats received watermelon powder, which consists of L-Citrulline, as part of the experimental diet. The results of the rabbit study were that both citrulline and arginine increases NO production, which then reduces the risk of atherosclerosis, CVD, and possibly other diseases. One of the main ingredients in watermelon is L-Citrulline, therefore, watermelon powder decreases the risk of CVD and inflammation.

A study was conducted on the effects of a tomato-based diet on LDL, lowering risk of CVD, and atheroprotective properties were analyzed. The participants of the study were healthy individuals and were split up into two groups. The participants consumed a low tomato diet for the first part of the study, and then switched to a consumption of a high tomato diet. This study observed the effects of the lycopene in the tomatoes. The study found that consuming tomatoes, which have a high concentration of lycopene, lowered cholesterol levels and decrease risk for CVD. Although, the design of the Cambridge study was different it still measured LDL and lowering cholesterol while consuming a fruit with lycopene and antioxidants (9).

This study done at SDSU also concluded that consuming watermelon had an effect on lowering LDL and lowering risk of CVD. This is because lycopene resists oxidation, which would reduce free radicals and prevent damage done to the arteries. Also, the study done at Cambridge found that there are other nutrients in the tomatoes that may have had an effect on lowering LDL, like flavonoids and polyphenols. It may be concluded that any fruit with those
nutrients, such as watermelon, can have this effect.

The limitation and possible further research for this study includes adding another variable group of 20 Sprague Dawley rats with a higher percent watermelon powder diet to find out if a higher percent of watermelon powder yields higher antioxidant capacity and further lowers the risk of CVD and inflammation.

In conclusion, watermelon powder had beneficial effects on reducing the risk factors of CVD and inflammation through an increase in antioxidant capacity, decrease in CRP, and decrease in LDH. Also, it was shown to increase HDL-cholesterol, decrease in LDL-cholesterol and total cholesterol.
References


Table 1. Composition of Experimental Diets

<table>
<thead>
<tr>
<th>Ingredients (g)</th>
<th>Control (placebo)</th>
<th>Watermelon Powder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cornstarch</td>
<td>12.30</td>
<td>12.30</td>
</tr>
<tr>
<td>Sucrose</td>
<td>33.00</td>
<td>33.00</td>
</tr>
<tr>
<td>Cellulose</td>
<td>5.00</td>
<td>5.00</td>
</tr>
<tr>
<td>Casein</td>
<td>20.00</td>
<td>20.00</td>
</tr>
<tr>
<td>Corn Oil</td>
<td>5.00</td>
<td>5.00</td>
</tr>
<tr>
<td>Dairy Butter</td>
<td>16.00</td>
<td>16.00</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>3.00</td>
<td>3.00</td>
</tr>
<tr>
<td>Salt mix, AIN-93G</td>
<td>3.50</td>
<td>3.50</td>
</tr>
<tr>
<td>Vitamin mix, AIN-93-G</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Methionine</td>
<td>0.30</td>
<td>0.30</td>
</tr>
<tr>
<td>Sodium cholate</td>
<td>0.50</td>
<td>0.50</td>
</tr>
<tr>
<td>Choline chloride</td>
<td>0.40</td>
<td>0.40</td>
</tr>
<tr>
<td>Placebo</td>
<td>0.20</td>
<td>0.20</td>
</tr>
<tr>
<td>Watermelon powder</td>
<td>0.00</td>
<td>0.20</td>
</tr>
<tr>
<td>Total (g)</td>
<td>100.2</td>
<td>100.2</td>
</tr>
</tbody>
</table>

Table 2. Initial body weights and final body weight and weight gain before and after treatment.

<table>
<thead>
<tr>
<th></th>
<th>Cont-no DSS</th>
<th>Cont-DSS</th>
<th>Watermelon-noDSS</th>
<th>Watermelon-DSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial Body Weight (g, NS)</td>
<td>59.83±1.05</td>
<td>59.80±0.93</td>
<td>59.79±0.88</td>
<td>59.80±0.84</td>
</tr>
<tr>
<td>Final body weight (g, before DSS, NS)</td>
<td>238.70±4.30</td>
<td>239.15±3.48</td>
<td>238.65±5.64</td>
<td>239.93±3.69</td>
</tr>
<tr>
<td>Final Body Weight (g, right after treatment)</td>
<td>254.80±5.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>240.80±4.09&lt;sup&gt;b&lt;/sup&gt;</td>
<td>254.80±5.83&lt;sup&gt;a&lt;/sup&gt;</td>
<td>238.64±3.60&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Final Body Weight (g, last day)</td>
<td>258.56±5.34&lt;sup&gt;a&lt;/sup&gt;</td>
<td>242.41±4.19&lt;sup&gt;b&lt;/sup&gt;</td>
<td>258.43±5.97&lt;sup&gt;a&lt;/sup&gt;</td>
<td>245.41±4.49&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Weight gain (g, before DSS, NS)</td>
<td>179.12±3.69</td>
<td>179.35±3.03</td>
<td>178.86±5.19</td>
<td>180.13±3.41</td>
</tr>
<tr>
<td>Weight gain (g, during treatment)</td>
<td>195.22±4.17&lt;sup&gt;a&lt;/sup&gt;</td>
<td>181.00±3.48</td>
<td>195.01±5.40&lt;sup&gt;a&lt;/sup&gt;</td>
<td>178.84±3.31&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Weight gain (g, grand)</td>
<td>198.98±4.48&lt;sup&gt;a&lt;/sup&gt;</td>
<td>182.61±3.61&lt;sup&gt;b&lt;/sup&gt;</td>
<td>198.64±5.56&lt;sup&gt;a&lt;/sup&gt;</td>
<td>185.61±4.22&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
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*Data presented as means ± SE (standard error). Data in rows with different subscript letters are statistically different (p<0.05).**
Table 3. Water intake before, during, and after treatment

<table>
<thead>
<tr>
<th></th>
<th>Cont-no DSS</th>
<th>Cont-DSS</th>
<th>Watermelon-no DSS</th>
<th>Watermelon-DSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water Intake – Before Treatment</td>
<td>23.75±0.86</td>
<td>24.12±1.43</td>
<td>26.28±1.70</td>
<td>26.14±1.94</td>
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<tr>
<td>Water Intake- During Treatment</td>
<td>26.66±0.56&lt;sup&gt;a&lt;/sup&gt;</td>
<td>21.81±1.08&lt;sup&gt;b&lt;/sup&gt;</td>
<td>28.39±1.79&lt;sup&gt;a&lt;/sup&gt;</td>
<td>19.55±2.08&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Water Intake- After Treatment</td>
<td>29.71±1.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>36.97±3.58&lt;sup&gt;b&lt;/sup&gt;</td>
<td>31.55±1.71&lt;sup&gt;a&lt;/sup&gt;</td>
<td>46.65±8.24&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Figure 1. Effects of treatment on weight of spleen. Bars represent means±SE. Bars with different subscripts differ significantly at P<0.05.
Figure 2. Effects of DSS treatment and watermelon on weight of kidneys. Bars represent means±SE. Bars with different subscripts differ significantly at P<0.05.

Figure 3. Effects of watermelon and treatment on triglyceride, total cholesterol, HDL-cholesterol, and LDL-cholesterol. Bars represent means±SE. Bars with different subscripts differ significantly at P<0.05.
Figure 4. Effects of watermelon on lactate dehydrogenase (LDH). Bars represent means±SE. Bars with different subscripts differ significantly at \( P<0.05 \).

Figure 5. Effects of watermelon on antioxidant capacity. Bars represent means±SE. Bars with different subscripts differ significantly at \( P<0.05 \).
Figure 6. Effects of watermelon on C-reactive protein (CRP). Bars represent means±SE. Bars with different subscripts differ significantly at P<0.05.