Tomato (*Lycopersicon esculentum*) prevents lead-induced testicular toxicity

**ABSTRACT**

**BACKGROUND:** Lead, an example of heavy metals, has, for decades, been known for its adverse effects on various body organs and systems such that their functions are compromised. **AIM:** In the present study, the ability of lead to adversely affect the male reproductive system was investigated and tomato (*Lycopersicon esculentum*: Source of antioxidants) paste (TP) was administered orally to prevent the adverse effects of Pb. **MATERIALS AND METHODS:** Fifteen Sprague Dawley rats, randomised into three groups (n = 5), were used for this study. Animals in Group A served as the control and were drinking distilled water. Animals in Groups B and C were drinking 1% Pb (II) acetate (LA). Group C animals were, in addition to drinking LA, treated with 1.5 ml of TP/day. All treatments were for 8 weeks. **STATISTICAL ANALYSIS USED:** A Mann–Whitney U-test was used to analyse the results obtained. **RESULTS:** The obtained results showed that Pb caused a significant reduction in the testicular weight, sperm count, life–death ratio, sperm motility, normal sperm morphology, and plasma and tissue superoxide dismutase and catalase activity, but a significant increase in plasma and tissue malondialdehyde concentration. But, Pb did not cause any significant change in the serum testosterone level. TP however, significantly reduced these adverse effects of Pb. **CONCLUSION:** These findings lead to the conclusion that TP significantly lowered the adverse effects of Pb exposure on the kidney as well as Pb-induced oxidative stress.

**KEY WORDS:** Heavy metals, lead, reactive oxygen species, testicular parameters, tomato

**INTRODUCTION**

Lead, a dangerous heavy metal, is harmful even in small amounts. Nevertheless, humans get exposed to Pb through their environment and diet. The manifestations of Pb poisoning in humans are nonspecific. They may include weight loss, anaemia, memory loss, nephropathy, infertility, etc. However, oxidation accompanies lead toxicity. Tomato, on the contrary, is a source of antioxidants and is made up of components very appropriate for detoxification, illnesses prevention, attaining growth, helping the immunologic system, maintaining blood in good state, etc.

This research, therefore, focuses on whether oral administration of cooked tomatoes prevents Pb-induced testicular toxicity or not.

**MATERIALS AND METHODS**

Fifteen (15) adult male Sprague Dawley rats (180 g–220 g) were used for this study. They were inbred at the Animal House section of the Department of Physiology, Ladoke Akintola University of Technology, Ogbomoso. The animals were acclimatized over a period of 2 weeks.

**Preparation of tomato paste**

Tomato paste (TP) was prepared by grinding tomatoes and heating in a water bath for 45 min at 80°C.

**Grouping of animals and treatment**

The rats were grouped into three groups (Groups A, B, and C, n = 5). Animals in Group A served as the control and were drinking distilled water. Animals in Groups B and C were drinking 1% Pb (II) acetate (LA). Group C animals were, in addition to drinking LA, treated with 1.5 ml of TP/day. All treatments were for 8 weeks.

**Animal sacrifice and collection of samples**

Twenty-four hours after the last treatment, each animal was sacrificed by cervical dislocation and blood samples were collected via heart puncture. Blood sample obtained from each rat was divided into two: One half in a plain bottle and the other half in an ethylenediaminetetraacetic acid
bottle. Plasma and serum were obtained by centrifugation at 3000 rpm for 20 min (g = 9.78 m/s²). Testis and caudal epididymis were excised from each rat.

**Collection of data and statistical analysis**

One testis from each rat was homogenized for tissue superoxide dismutase (SOD), catalase (CAT) and malondialdehyde (MDA) and sperm count (SC) was carried out from the epididymis. Plasma and tissue SOD activities were determined using the method described by Fridovich (1986).

\[ \text{Plasma and tissue SOD activities} \]

Plasma and tissue CAT activities were determined using the method described by Sinha (1972). Plasma and tissue MDA concentrations were determined using the procedure described by Varshney and Kale (1990).

The data obtained are presented as mean ± SD. The “Control Group” and the “Test Groups” were compared using the Mann–Whitney U-test. The significance level was set to a P-value <0.05.

**RESULTS**

The following results were obtained and are presented as mean ± SEM. Level of significance is taken at “P-value <0.05” (*) and/or “P-value <0.01” (**).

**Weight increase [g (Equivalent g is 9.87 m/s²)]**

Comparing their final and initial weights showed that there was significant weight gain (P-value <0.05) in all the groups over the 8 weeks of the research. There was, however, no significant (P-value >0.05) difference in weight gain across the three groups [Table 1].

**Testicular weight [g (Equivalent g is 9.87 m/s²)]**

The testicular weight of Group C showed no significant (P-value >0.05) difference from that of the control while the testicular weight of Group B was found to be significantly (P-value <0.05) lower than that of the control [Table 2].

**SC**

SC for Group B was significantly (P-value <0.01) lower than that of the control whereas SC for Group C showed no significant difference (P-value >0.05) from that of the control [Table 3].

**Life–death ratio of sperm cells**

Life–death ratio (LDR) of Group B was found to be significantly (P-value <0.01) lower than that of the control whereas Group C showed no significant (P-value >0.05) difference from the control [Table 4].

**Sperm motility (SM)**

Group B had SM that was significantly (P-value <0.01) lower than that of the control whereas SM for Group C was not significantly (P-value >0.05) different from that of the control [Table 5].

**Sperm morphology (SMP)**

Group B had SMP that was significantly (P-value <0.01) poor compared with that of the control. Group C, however, showed no significant (P-value >0.05) distortion in morphology [Table 6].

**Serum testosterone level (STL)**

There was no significant (P-value >0.05) difference between

| Table 3: Comparison of sperm count (million cells/mm³) across the groups |
|--------------------------|----------------|----------------|
|                        | Group A      | Group B      | Group C      |
| Sperm count (million cells/mm³) | 128.4 ± 0.349 | 14.00 ± 0.443** | 125.4 ± 0.631 |
| P-value (when compared with control) | 0.0040 | 0.3452 |

| Table 4: Comparison of life–death ratio of sperm cells across the three groups |
|--------------------------|----------------|----------------|
|                        | Group A      | Group B      | Group C      |
| Life–death ratio of sperm cells | 5.1 ± 0.161 | 1.12 ± 0.081** | 5.0 ± 0.178 |
| P-value (when compared with control) | 0.0040 | 0.4206 |

| Table 5: Comparison of sperm motility across the three groups |
|--------------------------|----------------|----------------|
|                        | Group A      | Group B      | Group C      |
| Sperm motility | 76.0 ± 0.409 | 11.0 ± 0.468** | 76.0 ± 0.511 |
| P-value (when compared with control) | 0.0039 | 0.5000 |
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Plasma SOD activity
Group B showed a significant (P-value <0.01) decrease in plasma SOD activity. Group C was, however, not significantly (P-value >0.05) different from the control in terms of the plasma SOD activity [Table 7].

Plasma CAT activity
Group B showed a significant (P-value <0.01) decrease in the plasma CAT activity. However, Group C showed no significant (P-value >0.05) difference in the CAT activity from the control [Table 8].

Plasma MDA concentration
Group B showed a significant (P-value <0.05) increase in the plasma MDA concentration whereas Group C showed no significant (P-value >0.05) difference from the control in terms of the plasma MDA activity [Table 9].

Testicular SOD activity
Group B showed a significant (P-value <0.01) decrease in testicular SOD activity. There was, however, no significant (P-value >0.05) difference in the testicular SOD activity of Group C from that of the control [Table 11].

Testicular CAT activity
Group B showed a significant (P-value <0.05) decrease in the testicular CAT activity. However, Group C showed no significant (P-value >0.05) difference from the control [Table 12].

Testicular MDA concentration
Group B showed a significant (P-value <0.01) increase in tissue MDA concentration whereas the MDA concentration in Group C was found to be significantly lower when compared with the control [Table 13].

DISCUSSION
The results of this study shows that 8 weeks of exposure to Pb does not significantly (P-value >0.05) affect weight gain in rats, as against the findings of Suzan et al., 1999[16] that chronic (12 weeks or more) exposure to Pb significantly (P-value <0.05) reduces weight gain. Therefore, exposure to Pb for a longer period is expected to significantly reduce weight gain. On the contrary, a significant (P-value <0.05) decrease was noticed in the testicular weight even for the 8 weeks of exposure. This ability of Pb to reduce weight gain and organ weight can be linked to the less-efficient metabolic processes associated with Pb toxicity.[17] However, there was no significant (P-value >0.05) decrease in the

Table 6: Sperm morphology across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Sperm morphology | 75.0 ± 0.376 | 13.0 ± 0.477** | 72.0 ± 0.478 |
| P-value (when compared with control) | 0.0041 | 0.2103 |

**P-value <0.01**

Table 7: Serum testosterone level across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Serum testosterone level (ng/ml) | 24.8 ± 0.256 | 24.4 ± 0.288 | 24.6 ± 0.246 |
| P-value (when compared with control) | 0.3452 | 0.5542 |

Table 8: Plasma SOD activity across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Plasma SOD activity | 1.658 ± 0.060 | 1.255 ± 0.074** | 1.679 ± 0.065 |
| P-value (when compared with control) | 0.0023 | 0.4206 |

**P-value <0.01**

Table 9: Plasma CAT activity across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Plasma CAT activity | 0.3954 ± 0.036 | 0.2670 ± 0.041** | 0.3962 ± 0.045 |
| P-value (when compared with control) | 0.0033 | 0.3452 |

**P-value <0.01**

Table 10: Plasma MDA concentration across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Plasma MDA concentration (µg/g protein) | 1371.5 ± 3.013 | 1627.3 ± 1.87* | 1432.4 ± 2.20 |
| P-value (when compared with control) | 0.0453 | 0.1547 |

*P-value <0.05*

Table 11: Testicular SOD activity across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Testicular SOD activity | 1.529 ± 0.084 | 1.224 ± 0.060** | 1.383 ± 0.087 |
| P-value (when compared with control) | 0.0079 | 0.2103 |

**P-value <0.01**

Table 12: Testicular CAT activity across the groups

|          | Group A | Group B | Group C |
|----------|---------|---------|---------|
| Testicular CAT activity | 0.3725 ± 0.0267 | 0.2392 ± 0.056* | 0.3352 ± 0.029 |
| P-value (when compared with control) | 0.0278 | 0.3452 |

**P-value <0.05**
testicular weight of animals treated with TP along with Pb exposure (Group C). This means that oral administration of 1.5 ml TP/day annuls adverse effect of Pb on weight gain. This may be partly due to the fatty acid composition of TP[18] and, more importantly, due to the presence of health-protective antioxidants such as lycopene, vitamin C, and vitamin A in TP[8] despite its relatively low caloric value (21 Kcal/100 g) and low protein content (0.85% by weight).[19]

There was no significant (P-value >0.05) decrease in the SC of animals treated with TP even though they were well exposed to Pb. On the contrary, the SC of the lead-only group (Group B) was significantly (P-value <0.01) lower than that of the control. This is because Pb (as well as most other heavy metals) interferes with the male reproductive system,[16] specifically with the testicular functions,[20,21] which are the determinants of SC. The administered tomato would therefore be responsible for the prevention of these lowering effects of Pb on SC by preventing its adverse effects on testicular functions.

In a similar way, the LDR of sperm cells of animals treated with tomato along with Pb was not significantly different (P-value >0.05) from that of the control whereas animals treated with Pb only showed a significant (P-value <0.01) decrease in the LDR. Therefore, TP must have somehow prevented the adverse effects of Pb on LDR, such that there was no significant difference in LDR of the control and that of the Pb + TP group. This is most likely due to the detoxification effect of TP, as accounted for by The world of plants, 2008.[19]

SM was significantly (P-value <0.01) reduced in animals treated with Pb only. This observation is in support of the findings of Ping-Chi et al., 1998,[22] that lead exposure might reduce SM. However, the SM of animals treated with TP along with Pb is not significantly different from that of the control. This further establishes the fact that TP has a protective ability against Pb toxicity. In a similar way, SMP was significantly (P-value <0.01) lowered in animals treated with Pb only. This observation is in support of the findings of Spomenka et al., 2000,[23] and that of Pinon-Lataillade et al., 1995,[24] that Pb causes an increase in abnormal sperm head morphology. However, the SMP of animals treated with TP along with Pb is not significantly different from that of the control. This also supports the fact that TP has a protective ability against Pb toxicity.

On the contrary, the STL of animals exposed to lead only was found not to be significantly (P-value >0.05) different from STL of the control. This is in support of the findings of Pinon-Lataillade et al., 1995,[24] that Pb does not affect the levels of follicle stimulating hormone and LH in blood and that of testosterone in both blood and testes. This, therefore, suggests that the hypothalamic–pituitary–testicular axis is not adversely affected by exposure to lead. In a similar way, the STL of animals treated with TP along with Pb was not significantly (P-value >0.05) different from that of the control, which means that the effects of Pb and/or TP noticed on the male reproductive system were not as a result of changes/alteration in the plasma or testicular testosterone levels.

There was no significant (P-value >0.05) difference in the SOD activity of both the plasma and the testes of the control and that of the animals treated with tomato along with Pb. But, there was a significant (P-value <0.01) decrease in the plasma and testicular SOD activity in animals treated with Pb only compared with the control. This finding is in agreement with that of Ping-Chi and Yueliang (2002)[25] and is at the same time in support of Lycopersicon esculentum (tomato) as an antioxidant.

There was a significant (P-value <0.05) decrease in both plasma and testicular CAT activity of animals treated with Pb only relative to the control. There was, however no significant (P-value >0.05) difference between the control and the animals treated with tomato along with Pb in this respect. This further establishes that TP must have reduced the oxidative stress that Pb could cause.

Finally, there was no significant (P-value >0.05) difference in both the plasma and the testicular MDA concentration of the control and those of the animals treated with tomato along with Pb, whereas animals treated with Pb only showed a significant (P-value <0.05) increase in both plasma and testicular MDA concentration. This confirms that it was TP, the source of antioxidants,[7,8] that reduced the oxidative stress that Pb exposure could have caused in the tomato-treated animals.

It can, therefore, be concluded that exposure to lead significantly reduces testicular weight, SC, LDR, SM, and SMP while TP prevents these adverse effects of Pb on the male reproductive parameters. However, neither Pb alone nor Pb with TP could have any significant effect on the

**Table 13: Testicular MDA concentration across the three groups**

|                      | Group A             | Group B             | Group C             |
|----------------------|---------------------|---------------------|---------------------|
| Testicular MDA conc. | 1393.47 ± 2.818     | 2235.47 ± 3.732**   | 1485.53 ± 2.655     |
| P-value (when compared with control) | 0.0039              | 0.2103              |

**Note:** *P*-value <0.01
serum and testicular testosterone level. These adverse effects of Pb on the male reproductive parameters is most likely due to the oxidative stress that Pb causes by interfering with the activities of SOD and that of CAT and 35 thereby, given freedom to free radicals (e.g., reactive oxygen species) to cause oxidation, which manifests as an increase in the concentration of MDA (in the case of lipid peroxidation). TP, therefore, prevents the adverse effects of Pb on the male reproductive functions. This is due to the antioxidant property of TP.

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