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Occupational risk assessment of oxidative stress and DNA damage in tannery workers exposed to Chromium in Pakistan

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Abstract
Trivalent chromium is extensively used in tanning process which appears to be associated with genetic damages in workers. Therefore, the present study was undertaken to investigate the oxidative stress parameters and DNA damage in chromium exposed tanners. DNA damage in lymphocytes was measured by the comet assay. The results showed that blood chromium, DNA damage, superoxide dismutase (SOD) and malondialdehyde (MDA) levels were significantly higher ($p<0.001$), while glutathione (GSH) level was significantly lower ($p<0.001$) in exposed groups as compared to control group. In Pearson correlation analysis, blood chromium level showed significant correlation with oxidative stress parameters and DNA damage. The mean tail length of two exposure groups was significantly higher as compared to control. These findings showed that during long-term chromium exposure, chromium is absorbed in the body, which may be distributed in the various tissues and organs of exposed workers. The present study revealed that occupational exposure to trivalent chromium can lead to oxidative stress and DNA damage in tannery workers. DNA damage and blood chromium level may serve as an efficient biomarker in tannery workers exposed to trivalent chromium.

Keywords: Trivalent chromium, oxidative stress, DNA damage, superoxide dismutase, malondialdehyde

1. Introduction
Chromium is one of the toxic heavy metals, which is extensively discharged from the tanning industry to the environment. Chromium has the binding potential of biomolecules in living systems and cause toxicity to biological life. Chromium is found in two valence states, the most stable and common forms are hexavalent chromium [Cr (VI)] and trivalent chromium [Cr(III)] [1]. Hexavalent chromium is highly toxic, whereas trivalent chromium is an essential micronutrient for living organisms [2]. Chromium has been widely used in industries such as tannery, dyes, glass, inks and ceramics [3]. Cr (III) is used as a basic tanning agent in the leather tanning industry [4]. Tannery workers are mainly exposed to chromium salts, e.g., potassium dichromate in the leather tanning department. The tannery workers are under constant threat of chromium in the tanning department [5]. Exposure of tannery workers to chromium can occur through oral, dermal, or by inhalation. The cell membrane is impermeable to Cr (III) whereas, Cr (VI) can pass through the cell membrane and undergoes reduction [6]. During reduction inside the cell, spontaneous reactions occur with intercellular reductants as ascorbate and glutathione, generating various reactive species of chromium like Cr (V), Cr (IV), Cr (II) and Cr (III).The reductive metabolism of Cr (VI) to Cr(III) can cause various forms of DNA damage, which results from either oxidative damage by reactive intermediates or from DNA adduction by Cr(III) [7, 8].
In the cytoplasm, the oxidation of Cr (IV) to Cr (VI) produces reactive oxygen species (ROS) that react with DNA-protein complex [9]. Other reactive species including hydroxyl radical or singlet oxygen are also produced leading to oxidative damage of the cell [10]. In addition, Cr (IV) can easily bind to genetic materials, altering their physiological functions. The intermediates that are generated from Cr (VI) reduction are toxic for protein and nucleic acids [11]. Inside the cell, Cr (III) forms complexes with various intracellular macromolecules leading to Cr (III)-DNA adducts as well as DNA damage [12]. In addition, the formation of Cr-DNA adduct and L-cysteine-Cr (III)-DNA likely increase both mutagenicity and genotoxicity in

human cells [13, 14]. Interaction of trivalent chromium with DNA leads to DNA strand breaks and oxidative DNA base modification such as production of 8-hydroxydeoxyguanosine (8-OH-dG) [15]. Cr (VI) can cause oxidative stress in the cell leading to DNA damage [16]. Chromium (VI) is considered carcinogen and human mutagen. Chromium has potential to inhibit macromolecular synthesis and induce various types of DNA lesions and gene mutation. Chromium induced mutations; DNA–DNA interest and crosslinks and mutations in the tumor suppressor gene *p53* are the important factors that play a key role in determining genotoxicity [17]. Oxidative DNA damage can be caused by a reduction in intracellular antioxidants. Moreover, glutathione (GSH) and the cystein also play a key role in this process [18]. The reduction of trivalent chromium in divalent chromium leads to oxidative stress by intracellular reducing agents. The reaction between Cr (II) and hydrogen peroxide leads to lipid peroxidation [19]. Lipid peroxidation is also caused by the massive production of free radicals [20]. As a result, defense mechanisms of the cell are activated, which detoxify the effects of lipid peroxidation [21, 22]. Superoxide dismutase (SOD) helps in the conversion of two superoxide molecules to generate oxygen and hydrogen peroxide, while GSH is capable to decompose peroxides safely. GSH containing sulfhydryl group is the most abundant non proteinous tripeptide in all cells and it has a protective role against oxidative injury. It has been reported that cultured cells exposed to chromium, lead to the production of Cr (III)–DNA adducts, which induce carcinogenesis and mutagenesis [23-25]. Under the standard condition of comet assay, as Cr–DNA adducts may lead to strand breakage [26, 27]. Sheikhupura is well known in the leather industry in Pakistan [28]. The effluents from tanneries are released into the open land, water resources and agricultural land, thus population living in Sheikhupura is at high risk of deleterious health effects of chromium [29].

The current study describes the results of total chromium in blood samples used as an internal biomarker in tannery workers and the relationship between oxidative stress and DNA damage. In addition, the study demonstrates that occupational exposure to chromium (III) causes oxidative stress and DNA damage in tannery workers.

2. Materials and Methods

Subjects 120 male tannery workers were selected from tanneries in Sheikhupura city, Punjab province of Pakistan. Control population belonging to a similar age group and socioeconomic strata, who never had any occupational exposure in the leather tanneries were selected. Tannery workers worked for at least 8–10 hr. a day for six days per week. These individuals were divided into three groups: (i) exposed group I (Short term exposure) included 60 tannery workers (30 smokers and 30 nonsmokers); (ii) exposure group II (Long term exposure) included 60 tannery workers (36 smokers and 24 nonsmokers); (iii) 120 controls (61 smokers and 59 nonsmokers) (Table 1). The average age of exposure group I was 20-35 years, exposure group II was 36-50 years and controls were 20-50 years (Table 1). The exposure time of worker groups I and II was from5-10 years (mean 6.12± 2.403 years) and 11-20 years (15.86± 3.673 years). Tannery workers were actively involved in the chrome tanning process. The consent was obtained from all individuals about the study. Ethical clearance for the present research investigation was obtained from the ethical committee, University of Peshawar, Pakistan. Only rubber gloves and aprons were used by all tannery workers as protective measures. Blood samples (5ml) were collected from all subjects. A questionnaire was filled by tannery workers and different demographic characteristics were analyzed.

2.1 Workplace Environment

The exposed workers were selected from the tanning department. All the workers were working in the ill-ventilated environment. Chromium may enter the body by dust breathing, food consumption as well as through direct cutaneous contact. Since chromium in the workplace environment is mostly in the Cr (III) form, it is this particular chemical species which needs investigation. In addition, chromium contamination may also occur through handling of tanning agents like chromium sulphate and chromate.

2.2 Blood Chromium analysis

Blood samples were collected from individuals at the end of the working day and stored at 4 °C in an ice box and transported to the laboratory. Chromium levels in erythrocytes and lymphocytes were measured by graphite furnace atomic absorption spectrophotometer Perkin Elmer model 700 (Perkin Elmer, CA, USA) with Zeeman background correction based on the method described by Gao *et al*., (1993) [30]. Samples were then left for 30–40 min at room temperature to let the blood to separate into two fractions: fraction 1, the supernatant, contained the plasma and white blood cells, and fraction 2 the residual, contained predominantly red blood cells. Three milliliters of supernatant (fraction 1) were layered on 3ml of histopaque 1077 (Sigma, St. Louis, Mo., USA) and centrifuged for 30min at 1800 rpm. The interphase containing the lymphocytes was removed and washed 3 times with phosphate buffered saline. The lymphocyte pellet was transferred to an Eppendorf tube, brought up to a final volume of 1 ml, and centrifuged for 5min at 4000 rpm. The supernatant was discarded and the lymphocytes were resuspended in 0.1ml deionized distilled water.

The second fraction was diluted with 3ml of sodium chloride (0.9%) up to the initial volume of blood collected, allowed to stand at room temperature for 10min, and centrifuged for 10min at 3000 rpm. Haematocrit 2 measurements (Ht 2) and RBC counts were made just before centrifuging for the last washing. The erythrocyte pellet after the last wash was diluted to the final volume of the blood sample with 0.1% Triton X-100.

2.3 Comet assay

Whole blood was analyzed using comet assay by the method of Singh *et al*, with minor modifications [31]. The whole blood sample (20µl) was used to carry out comet assay. Duplicate slides per sample were prepared. For preparation of base layer, 50 ml of normal melting agarose (1% DW) was coated on slides and were air dried. For the preparation of second layer, 20 µl of blood sample was mixed with 80 µl low melting point agarose (0.5% in PBS) and it was kept at 4°Cfor 10 minutes. After removing the cover slip, third layer of 100 µl low melting point agarose (0.5% in PBS) was pipetted out on the second layer. Slides were subjected to tank filled with lysing solution (2.5 M NaCl, 200 mm Na_2 EDTA, 10 Mm Tris–HCl, 10% DMSO and 1% Triton X 100) for 1 h at 4 °C and the slides were then subjected to electrophoresis box and covered with buffer (1 mM Na_2 EDTA, 0.3 M NaOH, pH 13) for 20 min at 4 °C to allow unwinding of DNA. The slides were then placed in electrophoresis 18V (0.7–1.0 V/cm) 300mA for 20 min at 4 °C, after that the slides were

neutralized with Tris–HCl buffer (400 mM, pH 7.4) for 5min. The slides were stained with 75 µL EtBr (ethidium bromide) (20µg/ml).

A total of 50 cells/sample were screened using fluorescent microscope (Nikon Eclips 80i) equipped with an excitation filter of 450-490nm and the tail length of the comet was expressed as a micrometer.

2.4 Superoxide dismutase (SOD)

SOD level was measured by the method of McCord and Fridovich with slight modifications[32].Accurately measured blood sample (200µl)was washed with ice cold normal saline and then centrifuged three times at10000 r/min. RBCs were hemolyzed by adding 1.5 volume of water and kept at 0–4 °C. Hemoglobin (Hb) was precipitated by adding chloroform (180 ml) and cold ethanol (300 ml) followed by vigorous mixing and centrifuged supernatant was used for determination of SOD. Two parallel reaction setups were performed. The first setup tube 1 of 0.3 ml (186 µM) phenazine methosulphate was mixed with 0.2 ml (0.052 M) of sodium pyrophosphate, 0.3 ml (300 µM) of nitrobluetetrazoliumand0.2 ml of enzyme source. All the above reagents were present in the second setup reaction except the enzyme source. After adding l, ml and 0.8 ml distilled water in both reaction setups, 0.2 ml (780 mM) of reduced Nicotinamide adenine dinucleotide (NADH) disodium salt was added and the reaction was started. After 90 s, 1 ml of glacial acetic acid was added to both the tubes of reaction setups. The absorbance was measured at 560 nm on the spectrophotometer and expressed as a unit per gram per hemoglobin.

2.5 Reduced glutathione (GSH)

Blood GSH levels were measured according to the method of Beutler *et al* [33]. Blood sample (200 ml) was added to cold distilled water (1.8 ml) and incubated at 37 °C for 10 min. Sulphosalicylic acid (600 ml) was mixed with a reaction mixture followed by centrifugation for 15 min at 2000 rpm.400 ml of phosphate buffer was mixed with 200 ml of supernatant and 80 ml of 3, 5-dithiobis-2-nitrobenzoic acid, as a result the yellow color was developed. The absorbance was measured at 412 nm and GSH level was expressed as microgram per milliliter.

2.6 Malondialdehyde (MDA)

MDA level in blood plasma was determined by the method of Ohkawa *et al* [34]. 1 ml of 20% acetic acid and 200 µL of 8% SDS (pH 4) were added to 200 µL of blood plasma.1.5 ml of 0.8% thiobarbituric acid (TBA) and 1.1 ml of distilled water were added to the reaction mixture and incubated for 1 hour. After this 3 ml of n-butanol was added to the reaction mixture. A clear butanol fraction was obtained. The absorbance was measured at 532 nm. MDA level was expressed as nano mole per milliliter of blood plasma.

2.7 Statistical analysis

The results were shown in terms of mean, percentages and standard deviations. For the comparison of two means, independent student's t-test was used while ANOVA was applied to identify the variation in the data. The association pattern of chromium concentration with DNA damage and oxidative stress was analyzed using Pearson's correlation test. P-value < 0.05 is considered as statistically significant. The data was analyzed using "IBM SPSS software" 20.0 version.

3. Results

Chromium concentration in blood Hexavalent chromium levels in erythrocyte of the exposed and control groups are shown in Table 2.The exposed groups showed significantly (p<0.001) higher erythrocyte Cr concentration than that of the control group. Cr concentration in erythrocytes were found to be 0.79µg/l and 1.16µg/l in smoking control groups I and II respectively, which was increased significantly to 4.20µg/l and 5.06µg/l smoking exposed groups I and II respectively, while in nonsmoking control groups I and II were found to be 0.54µg/l and 0.68µg/l respectively, which was increased significantly to 3.78µg/l and 4.71µg/linnon smoking exposed groups I and II respectively (Fig 1).Cr concentration in lymphocytes were found to be 29.23µg/10^{12} cells and 33.14µg/10^{12} cells in smoking control groups I and II respectively, which were increased significantly to 42.35µg/10^{12} cells and 61.56µg/10^{12} cells in smoking exposed groups I and II respectively. Similarly in nonsmoking control groups I and II concentration of Cr in lymphocytes was found to be 27.15µg/10^{12} cells and 30.98µg/10^{12} cells respectively, which were increased significantly to 38.12µg/10^{12} cells and 57.38µg/10^{12} cells in nonsmoking exposed groups I and II respectively.

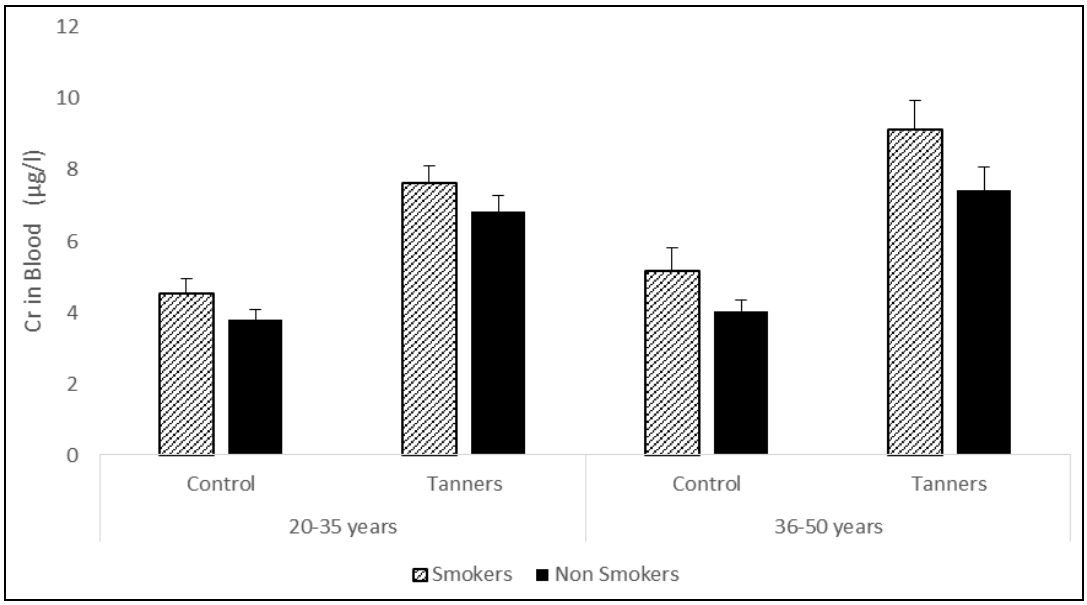


Fig 1: The levels of chromium in blood of smoking and nonsmoking tanners and control groups in error bar graph (p = < 0.001, n=60)

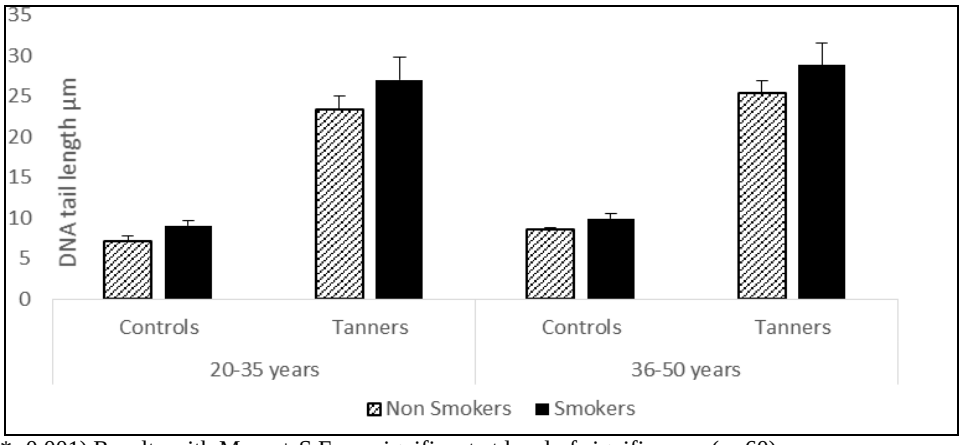
Table 1: Summary of statistical analysis of DNA damage (µm), MDA (nmol-ml^{-1}), GSH (µg-ml^{-1}) and SOD (unit-g^{-1}-Hb^{-1}) levels in tannery workers and controls.

Age	Health	20-35 years			36-50 years		
Groups	**parameters**	**Controls**	**tanners**	**t-statistic**	**Controls**	**Tanners**	**t-statistic**
Non Smokers	DNA	7.18± 0.57	23.32± 6.021	-34.411*	8.51± 0.59	25.41± 7.19	-30.113*
Smokers	tail length	9.03± 0.27	26.99± 2.651	-57.541*	9.90± 0.52	28.79± 2.15	-64.878*
Non Smokers		8.47 ± 0.61	11.11 ± 1.43	7.1043*	8.83 ± 0.61	12.13 ± 0.61	12.164*
Smokers	MDA	5.24 ± 1.42	6.21 ± 1.62	2.521*	6.21 ± 0.65	8.36 ± 1.42	4.95*
Non Smokers		65.22 ± 1.15	55.39 ± 1.10	22.6715*	71.10 ± 0.27	58.82 ± 1.624	43.0012*
Smokers	GSH	62.65 ± 0.63	48.81 ± 1.08	31.6073*	68.43 ± 1.12	54.51 ± 1.20	26.7828*
Non Smokers		55.47 ± 0.73	66.75± 2.21	21.1015*	63.55 ± 0.58	74.23 ± 2.21	21.7904*
Smokers	SOD	55.29 ± 0.66	71.10 ± 1.21	32.1104*	66.23 ± 1.25	78.42 ± 1.52	25.3646*

Table 2: Concentration of hexavalent chromium in lymphocytes (µg/l) of tannery Workers and control population

	20-35 years age group			36-50 years age group		
	Controls	**Tanners (exposed group I)**	**t-statistic**	**Controls**	**Tanners (exposed group II)**	**t-statistic**
Smokers	29.23 ± 0.05	42.35 ± 0.28	32.428*	33.14 ± 0.52	61.568 ± 0.72	21.215*
Non Smokers	27.15± 0.23	38.12 ± 0.22	26.037*	30.98 ± 0.32	57.388 ± 0.54	25.254*

*Valuesare significant at p<0.001



(*=0.001) Results with Mean ± S.E are significant at level of significance. (n=60)

Fig 2: The DNA tail length of smoking and non-smoking tanners and control groups inerror bar graph (p = < 0.001 and n=60)

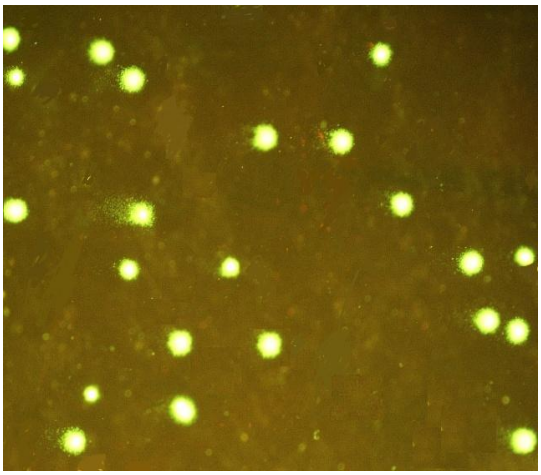


Fig 3(A): DNA tail length of control population

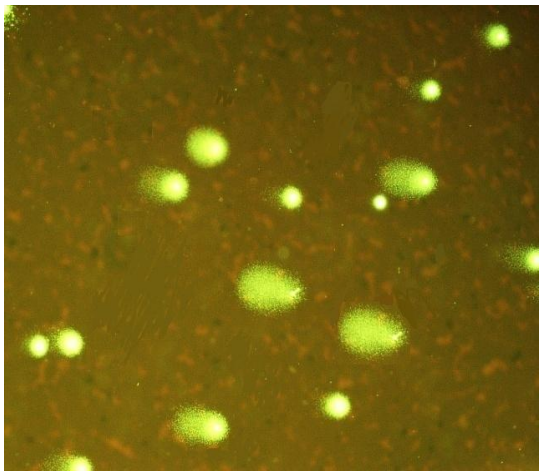


Fig 3 (B): DNA tail length of exposed tannery workers

Table 3: The Pearson correlation coefficient (R) of chromium with DNA damage, MDA, GSH and SOD levels

				DNA	MDA	GSH	SOD
Chromium in blood	20-35 years	Controls	Smokers	0.704037	0.764323*	-0.08753	0.670723*
			Non smokers	-0.179981	0.303978	0.001343	0.278318
		Tanners	Smokers	0.90739**	0.837364*	-0.87018*	0.754174*
			Non smokers	0.700059*	0.801548*	-0.79264*	0.731335*
	36-50 years	Controls	Smokers	0.726981*	0.795646*	-0.71961*	0.743368*
			Non smokers	0.480398	0.321396	-0.52303	0.732138*
		Tanners	Smokers	0.97630**	0.93933**	-0.9614**	0.97938**
			Non smokers	0.877128*	0.822787*	-0.75321*	0.74321**

The level of significant * p=0.01 ** p=0.001 for correlation (r) n=60

3.1 DNA damage

Comet tail length of nonsmoking tannery workers was found to be 7.189µm and 8.510µm in nonsmoking control group I and II respectively, which was increased significantly to 23.326µm and 25.416µm in nonsmoking exposed groups I and II respectively. Comet tail length of exposed groups was significantly (p<0.001) higher than unexposed groups. While comet tail lengths were found to be 9.032µm and 9.907µm in smoking control groups respectively, which were increased significantly to 26.992µmand 28.791µm in smoking exposed groups I and Irrespectively (Table 3; Figure 3 A, B). Tannery workers in group II with the long-term Cr exposure were most affected and showed larger comet tail length as compared to group 1 and control. Table 3 shows that comet tail length of smoking workers was significantly higher than those of nonsmoking workers in exposed groups I and II (P>0.05).Table 3 shows the correlation between comet tail length and blood chromium level. In Pearson correlation analysis, a significant positive correlation was observed for DNA damage with Cr concentration (r 0.907397, p<0.001) and (r 0.700059, p<0.01) in smoking and nonsmoking exposed group I respectively. Similarly, DNA damage also showed significant positive correlation with Cr level (r 0.976309, p<0.001) and (r 0.877128, p<0.01) in smoking and nonsmoking exposed group II respectively.

3.2 MDA concentration

The MDA concentration of non-smoking control I and II were found to be 5.270nmol/ml and 6.118nmol/ml in control groups, respectively, which was increased significantly to 6.622nmol/ml and 8.778nmol/ml in nonsmoking exposed groups I and II respectively. MDA concentration of exposed groups was significantly (p<0.001) higher as compared to unexposed groups. While the concentration of MDA was found to be 8.353nmol/ml and 8.737nmol/ml in smoking control groups I and II respectively, which was increased significantly to 10.124nmol/ml and 11nmol/ml in smoking exposed groups I and II respectively. MDA concentration of exposed groups was significantly (p<0.001) higher than control groups. Tannery workers in group II with the long-term Cr exposure were most affected and showed high MDA concentration as compared to group 1 and control which was exposed for a short time period and control group (4). A significant positive correlation was observed for MDA concentration with Cr level (r 0.837364, p<0.01) and (r 0.801548, p<0.01) in smoking and nonsmoking exposed group I. Similarly MDA concentration showed significant positive correlation with Cr level (r 0.939335, p<0.001) and (r 0.822787, p<0.01) in smoking and nonsmoking exposed group II Table 3 respectively.

3.3 GSH concentration

GSH levels were found to be 64.329±1.053µg/ml and 72.168±0.379µg/ml in smoking control groups, which was decreased significantly to 54.499±2.006µg/ml and 59.926±1.594µg/ml in smoking exposes groups I and II respectively. MDA concentration of exposed groups was significantly (p<0.001) lower than control groups. GSH concentration of nonsmoking control groups I and II were found to be 61.756±0.537µg/ml and 69.569±1.009µg/ml in respectively, which was decreased significantly to 49.711±2.094µg/ml and 55.702±2.303µg/ml in nonsmoking exposed groups I and II respectively. GSH concentration of exposed groups was significantly (p<0.001) lower than those of unexposed groups. The tannery workers in group II have significantly lowered GSH level due to long-term Cr exposure as compared to group I and control groups (Table 2,3).A significant negative correlation was observed for GSH with Cr concentration (r -0.87018, p<0.01) (r -0.79264, p<0.01) in smoking and nonsmoking exposed group I. GSH also showed significant negative correlation with Cr level (r -0.96141, p<0.001) and (r-0.75321, p<0.01) in smoking and nonsmoking exposed group II Table 2,3 respectively.

3.4 SOD concentration

SOD concentration in nonsmoking control groups I and II was found to be 59.699U/gHb and 64.758U/gHb in respectively, which was significantly increased to 70.048U/gHb and 79.528U/gHb in nonsmoking exposed groups I and II respectively. SOD concentration of exposed groups was significantly (p<0.001) higher than those of unexposed groups. While the concentration of SOD was found to be 56.675U/gHb and 64.758U/gHb in smoking control groups I and II respectively, which was significantly increased to 65.999U/gHb and 75.178U/gHb in smoking exposed groups I and II respectively. SOD concentration of exposed groups was significantly (p<0.001) higher as compared to unexposed groups. Long-term Cr exposed tannery workers in the group II exhibit significantly higher SOD level as compared to group I with short term Cr exposure Table 2.

SOD level showed significant positive correlation with Cr concentration (r 0.754174, p<0.01) and (r 0.731335, p<0.01) in smoking and nonsmoking exposed group I. A Significant positive correlation was also observed in SOD level and Cr concentration (r 0.979388, p<0.001) (r 0.743214, p<0.001) in smoking and nonsmoking exposure group II Table 3.

4. Discussion

Basic chromium (III) sulphate [$Cr(H_2O)_5(OH)\ SO_4$] is extensively used in tanning industries as a primary tanning agent [35]. Tannery workers are continuously exposed to Cr (III) and are thus affected their health. On the other hand, hexavalent chromium has the ability to cross cell membrane in the presence of phosphate and sulfate anion-exchange carrier pathway. Once inside the cell, Cr (VI) reduces to reactive intermediates and is eventually converted into kinetically stable Cr(III), which is more reactive than hexavalent chromium toward DNA and protein [36]. In the blood stream, trivalent chromium is largely bound to plasma proteins, amino acids and other organic acids. The complexes of Cr(III) bound to low-molecular-weight ligands in plasma can easily cross the cell membrane [37-39]. It has been concluded from the previously reported studies that trivalent chromium may cause different genotoxic effects *in vitro* studies [40]. In the present investigation, health risk factors associated with trivalent chromium were elucidated in terms of DNA damage and oxidative stress. The findings of previous studies have shown significantly higher Cr (III) levels in blood of exposed population [41]. Similar results were observed in our investigation in tannery workers exposed to chromium. It was observed that blood chromium level in exposed groups I and II were significantly higher (P<0.01) than control groups. Similarly, high concentration of chromium was reported in chromium exposed population in some previous conducted studies [41-43]. The research findings revealed that in longer duration of exposure, higher amount of chromium is absorbed in the body. Further, the comet tail length of groups I and II are significantly longer as compared to controls, showing that there was more DNA damage in chromium exposed tannery workers. Sellappa *et al.* and

Medeiros *et al*. also reported similar reported that there was an increased rate in DNA damage in tannery workers [7, 44]. We found that there was a significant positive correlation between DNA damage and chromium level in the blood.

Danadevi *et al* have reported that DNA damage was positively correlated with blood chromium concentration [42]. The mechanism of Cr toxicity has been related to oxidative stress. Excessive production of reactive oxygen species causes lipid peroxidation, which adversely affects cell structures, nucleic acids and lipids [45, 46].

In our study, MDA concentrations of exposed groups were found to be higher than those of unexposed groups. Higher level of MDA induced oxidative stress in tannery workers. Similar results were reported by Harris and Shi [47]. Goulart *et al*, has also documented that tannery workers showed significantly higher urinary MDA level compared with unexposed populations [12]. In the present study, smoking habits have shown significant effects on the MDA level in chromium exposed tannery workers. Bridges *et al*. has also reported that MDA concentration increased with age due to smoking habit [48].

In the present study, there was a significant positive correlation between MDA concentration and chromium level. This higher MDA level in tannery workers may be due to occupational exposure to high level of chromium in the work place. While GSH level was significantly lower in the exposed population than that of control groups. Similar results were documented by Lenton *et al*. [16]. Low level of GSH in tannery workers is due to its involvement in detoxification of lipid peroxidation. The marked decrease in GSH concentration, toxic effect of reactive oxygen species and can cause cell injury and death [49]. There is a negative correlation between GSH and the blood Cr level in the exposed groups. These findings showed that low GSH level may be associated with oxidative stress and chromium exposure. SOD activity was significantly higher in tannery workers compared with control.

5. Conclusion

Our data demonstrated the fact that trivalent chromium causes oxidative stress and DNA damage in exposed tannery workers. Moreover, oxidative stress and DNA damage were significantly elevated in tannery workers exposed to Cr (III). In addition, chromium concentration in erythrocytes is a sensitive indicator of recent Cr exposure. Lymphocytes are also capable of great uptake, intracellular reduction and accumulation of chromium (VI). As their life span ranges from a few months to years, their chromium content represents the amount accumulated over a long period of time and may express the blood cell body burden more accurately.

However, additional studies are required to unfold the adverse effects of trivalent chromium exposure on human health.

6. Acknowledgement

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