

Chromosome Damage in Lymphocytes of Stainless Steel Welders

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ABSTRACT Genotoxic effect of welding fumes generated by Manual Metal Arc (MMA), Metal Inert Gas (MIG) and Oxy-acetylene welding on the lymphocytes of 75 welders, 25 from each group and equal number of healthy matched controls, was investigated by studying Mitotic Index (MI), Chromosome Aberrations (CAs), Sister Chromatid Exchanges (SCEs), and Satellite Associations (SAs) The MI showed significant increase in all the three groups (4.25 - 7.66, 4.35 - 7.63, 4.63 - 7.54). The background frequency for CAs was normal (0.88, 0.84, 0.92). It significantly increased in all the three groups (4.68, 3.84, 2.96). Among controls only chromatid gaps and breaks were observed, while chromosome type aberrations could not be seen. Among exposed samples both chromatid type viz. gaps, breaks and isochromatid exchanges and chromosome type aberrations viz. dicentrics, rings, acentric fragments, translocations, chromosome gaps, chromosome breaks and diplochromosomes were observed. The frequency of SCEs also increased (3.98 - 7.84, 4.16 - 4.72, 4.57 - 6.43). However, the increase was significant in group-1 and group-3 welders only. Synergistic effect of smoking and alcohol was noticed in both CAs and SCEs, the frequencies being the highest in smoker-alcoholics and lowest in non smoker - non alcoholics. The CAs and SCEs also revealed correlation with the duration of exposure. The frequencies of SAs also depicted significant increase (4.8 - 13.8, 5.64 - 11.48, 5.56 - 9.28). The D-G type of associations outnumbered all other types. It is concluded that the welding fumes, containing chromium (Cr VI) and nickel, cause considerable chromosomal damage. The welders, in their occupational settings are prone to high genetic risk.

INTRODUCTION

Pollutants are constantly poured into the environment by various occupational settings. Welding is amongst one of the most common occupations which constitutes 0.5% to 2% of the industrial workers (Stern and Hansson 1986). A generic term *welding* deals with various techniques for joining metals, mostly steel and aluminium viz. Manual Metal Arc welding (MMA), Metal Inert Gas welding (MIG) and Oxyacetylene welding (Autogen).

Welders are exposed to a large range of chemical and physical agents. Physical factors include heat, UV radiations, infrared radiations, noise and electro - magnetic field. The composition and quantity of fumes and gases produced vary with the kind of technique involved and the type of

metal welded. These comprise fumes of oxides of various metals, fumes of crystalline nature, viz. NaF, CaF, K₂CaF₃, MgO, K₂CO₃, Na₂CO₃, MnFe₂O₄, toxic gases like carbon dioxide, carbon monoxide, nitrogen oxides and fluorine. The fumes from MMA/SS and MIG/SS welding contain Cr and Ni in a wide range of oxidation states and solubilities (Stern and Hansson 1986).

Epidemiological studies on welders have confirmed a vast variety of diseases viz. cancer mortality (IARC 1990), siderosis (Buck Up and Schweisseriunge 1960), chronic bronchitis (Peters et al. 1973), frequent attacks of sore throat, hoarseness, colds and fever (Mari et al. 1977), pulmonary tumours (Sjogren 1980), asthma (Keskenen et al. 1980), and dermatitis (Brown 1969).

There have been contradicting reports on the genetic effects of welding fumes on human beings. Welding fumes generated in the manual metal arc (MMA) method on stainless steel have shown higher toxicity and mutagenicity in bacteria and mammalian cells compared to fumes generated by other welding methods (Hedenstedt et al. 1977; Koshi 1979; Stern et al. 1988). Bacterial and eukaryotic test systems revealed association between the water soluble hexavalent chromium (Cr VI) present in the welding fumes and measured mutagenic activity (Hedenstedt et al. 1977). Elevated levels of CAs (Bigliev et al. 1977) and SCEs (Sarto et al. 1982) have been reported in lymphocytes from welders. Others found no increase in CAs (Husgafvel - Pursianinen et al. 1982; Elias et al. 1989) or SCEs (Nagaya 1989; Popp et al. 1991). Keeping in mind the above cytogenetic damage and vast variety of diseases in welders, this comparative analysis was carried out in the different groups of welders.

MATERIALS AND METHODS

Workers exposed to weldings fumes in a Bicycle Industry were grouped according to the type of welding into three groups: group 1 - Metal Inert Gas (MIG), welders; group 2 - Manual Metal

Arc (MMA) welders, group 3 - Oxyacetylene Gas (Autogen) welders. The different types of welding were carried out, in different areas of the industry, for welding different parts of the bicycle. All identified subjects agreed to participate in the study. Welders from each group comprising 25 individuals and an equal number of healthy controls matched with respect to age, sex, smoking habits and alcohol consumption, constituted the materials for the present investigation. Persons who neither had previous history of pollutant exposure, nor had any ailment, were taken as the control subjects. They included the persons mostly at the managerial and clerical posts as also the security personnel of the industry.

The age range was the same for both controls and exposed individuals ranging from 21-52 years in all the three groups. Criteria for classifying smokers and alcoholics were the same in the exposed and control workers i.e. the number of cigarettes and amount of alcohol consumed were approximately the same. Heparinized venous blood samples (2 ml) were obtained from each subject and transported to the laboratory in insulated ice box. Short-term lymphocyte cultures were set up within 3 h. of sampling according to the method of Moorhead et al. (1960) with minor modifications (Yadav et al. 1999). For mitotic index (MI) a minimum of 5000 cells per individual were counted from Giemsa-stained slides. Mitotic index was calculated by using the formula:

$$MI = \frac{\text{No. of dividing cells}}{\text{Total no. of cells scored}} \times 100$$

Lymphocytes were harvested after 48 h. for chromosomal aberrations (CAs). Routine chro-

mosome preparations were made and slides were stained with 4% Giemsa. For each person, 100 well spread metaphases were analysed.

For sister chromatid exchanges (SCEs), 5-bromodeoxyuridine (10 ug/ml culture) was added 24 h after setting up of the cultures. Harvesting was done after 72 h. Metaphase chromosomes were prepared and stained with Hoechst 33258 and 4% Giemsa following the method of Perry and Wolff (1974). For calculating the frequency of SCEs per cell, 25 metaphases were analysed as per standard practice.

The criteria described by Hansson (1970) were followed for evaluating the satellite associations (SAs).

Exposure assessment was carried out and characteristics of welding fumes were given in table 1. Fumes were collected by Casella Air sampler and analyzed gravimetrically. For analysing the gases the Bregger polymer/Bregger long term detector was used, and for measuring UV radiations UV digital meter was used. Epidemiological survey showed that all Autogen welders were healthy, while most of the MIG and MMA welders complained of irritation and watering in eyes with frequent and recurrent cold, sore throat and fever. As many as 10 out of 25, MIG welders and 19 out of 25 MMA welders were asthmatic. A single electric (MMA) welder had a cancerous growth in the nose. Various parameters viz. chromosome aberrations (CAs), sister chromatid exchanges (SCEs), mitotic index (MI) and satellite associations (SAs) were investigated and analyzed.

For statistical analysis of results, Harvey's least square analysis, Regression analysis, Correlation matrix, ANOVA (Analysis of variance),

Table 1 : Characteristics of welding fumes

	ELEMENTAL ANALYSIS Mean concentration mg/m				GASES Mean concentration			UV mean irradiance level
	Cr	Ni	Mn	F	Oxides of Nitrogen (PPM)	Carbon monoxide	Gaseous Fluorides mg/m ³	UV/Cm ²
*TLV	0.050	1.000	1.000	2.50	5.000	50.000	2.500	0.100
MIG	0.097	0.500	0.200	1.020	2.200	2.500	1.110	0.283
Group-1 MMA	0.081		0.190	0.140	2.700	2.890		0.154
Group-2 Autogen Gas	0.060		0.120		3.050	3.020		0.152
Group-3								

*TLV = Threshold Limit Value.

students 't' test and Chi square test were applied.

RESULTS AND DISCUSSION

All the three exposed groups had higher mitotic index (MI) as compared to controls (Table 2). Group-1 had the highest (7.66) and group-3 the lowest MI (7.54). All the values were significant at p<0.05. Similar results have been reported by Rupa et al. (1989), Yadav and Kaushhik (1996), Yadav and Seth (1998 a, b), Yadav et al. (1999), Yadav and Chhillar (2001).

Frequencies of all types of chromosome aberrations (CAs) was significantly higher in the exposed groups as compared to their controls (Table 3). Frequency of total CAs was highest in group-1 (4.680) and lowest in group-3 (2.960). Similar results were shown by Elias et al. (1989). The total chromatid type aberrations were more than the total chromosome type aberrations in all the exposed workers and controls. The background frequency in controls 0.88, 0.84 and 0.92 (Table 3) tallied with the earlier reports (Galloway

et al. 1986, Bender et al. 1988; Yadav and Seth 2000).

In all the three groups the least square means of CAs and SCEs in smoker alcoholics was higher than the non-smoker non-alcoholics (Table 4). All these values were higher in the exposed smokers and alcoholics as compared to the controls. The values for the least square means were highest in group-1 for both CAs (4.77) and SCEs (7.47) after the longest exposure of 16-20 years (Table 5). With respect to age the least square means for CAs was 3.66, 3.47 and 2.58 in group-1, 2 and 3 respectively (Table 6). Least square analysis showed the highest frequency of SCEs (7.26) in group 1 and the lowest (4.50) in group-2 (Table 6). Thus MIG welders and Autogen welders showed significant increase in SCEs. This is in agreement with earlier results (Etienne et al. 1982; Koshi et al. 1984).

The values for analysis of variance for SCEs were highly significant in smokers alcoholics for group-2 and 3 (Table 7.1). With regard to CAs the

Table 2 : Mitotic index (MI) in workers exposed to welding fumes

		No. of Samples	No. of cells screened	No. of metaphases	MI	SD ±
Group-1	Control	25	123277	5241	4.25	0.77
	Exposed	25	125760	9639	7.66*	0.80
Group-2	Control	25	122643	5743	4.35	1.05
	Exposed	25	126696	9639	7.63*	0.80
Group-3	Control	25	123845	5743	4.63	1.12
	Exposed	25	124344	9387	7.54*	0.92

Group-1: MIG welders; Group-2: MMA welders; Group-3: Oxyacetylene Gas welders
*P<0.05

Table 3: Chromosome aberration frequency in welders and controls

	Chromatid type aberrations				Chromosome type aberrations							Total CAs	Level of Significance	
	Gap	Break	Iso Exch.	Total	Dicentric	Ring	Ac. Frag.	Trans	Cg	Cb	Dipl			Total
Group 1														
C	44 (1.76)	22 (0.880)	—	22 (0.880)	—	—	—	—	—	—	—	—	22 (0.880)	P<0.01
E	66 (2.640)	61 (2.440)	6 (.240)	67 (2.680)	10 (0.400)	7 (0.280)	16 (0.640)	6 (0.320)	8 (0.320)	2 (0.080)	1 (0.40)	50 (2.000)	117 (4.680)	
Group 2														
C	37 (1.48)	21 (0.840)	—	21 (0.840)	—	—	—	—	—	—	—	—	21 (0.840)	P<0.01
E	71 (2.840)	58 (2.320)	3 (0.120)	61 (2.440)	6 (0.240)	6 (0.240)	11 (0.440)	6 (0.240)	3 (0.120)	3 (0.120)	—	35 (1.400)	96 (3.840)	
Group 3														
C	33 (1.320)	23 (0.920)	—	23 (0.920)	—	—	—	—	—	—	—	—	23 (0.920)	P<0.01
E	61 (2.440)	53 (2.120)	2 (0.080)	55 (2.200)	4 (0.160)	3 (0.120)	7 (0.280)	2 (0.80)	2 (0.80)	1 (0.040)	—	19 (0.760)	74 (2.960)	

E = Exposed, C = Control, AC Frag = Acentric Fragment, Trans = Translocation, Cg = Chromosome gap, Cb= Chromosome break, Dipl. = Diplochromosome, Dic = Dicentric, Iso Exch = Isochromatid Exchange; Figures in parenthesis indicate the value per 100 metaphases.

Table 4: Least square means for alcohol and smoking

	Group 1		Group 2		Group 3	
	E	C	E	C	E	C
1. ++	6.74 + 0.41 (8.05 + 0.40)	1.43 + 0.34 (4.09 + 0.31)	6.05 + 0.24 (5.65 + 0.09)	1.04 + 0.23 (4.20 + 0.21)	4.38 + 0.52 (7.13 + 0.08)	1.07 + 0.18 (5.07 + 0.18)
2. +-	4.30 + 0.42 (7.08 + 0.41)	0.78 + 0.34 (3.83 + 0.31)	3.57 + 0.27 (4.92 + 0.10)	0.78 + 0.26 (4.04 + 0.24)	2.55 + 0.47 (6.59 + 0.07)	0.80 + 0.24 (4.24 + 0.25)
3. -+	2.42 + 0.38 (6.94 + 0.38)	0.65 + 0.41 (3.53 + 0.37)	2.94 + 0.29 (4.34 + 0.11)	0.50 + 0.32 (4.14 + 0.29)	2.01 + 0.67 (6.17 + 0.10)	0.68 + 0.33 (3.43 + 0.34)
4. --	1.16 + 0.52 (6.98 + 0.51)	0.12 + 0.32 (3.57 + 0.30)	1.33 + 0.29 (3.11 + 0.14)	0.61 + 0.31 (3.03 + 0.28)	1.38 + 0.68 (5.70 + 0.10)	0.68 + 0.34 (3.73 + 0.35)

Upper values in each row represent CAs and value in brackets represent the SCEs.
 ++ = Smokers & Alcoholics, +- = Smokers, -+ = Alcoholics, -- = Non smokers Non Alcoholics, E = Exposed, C = Control.

Table 5: Least square means for duration of exposure

Exposure (in years)	Group 1		Group 2		Group 3	
	CA	SCEs	CA	SCEs	CA	SCEs
0 - 5	2.76 + 0.70	7.35 + 0.68	3.28 + 0.43	4.31 + 0.16	2.19 + 0.71	5.99 + 0.11
6 - 10	3.27 + 0.46	7.35 + 0.45	3.29 + 0.27	4.47 + 0.10	2.14 + 0.48	6.35 + 0.07
11 - 15	3.81 + 0.43	6.89 + 0.42	3.45 + 0.36	4.37 + 0.13	2.64 + 0.51	6.49 + 0.08
16 - 20	4.77 + 0.36	7.47 + 0.36	3.87 + 0.31	4.87 + 0.12	3.65 + 0.58	6.77 + 0.09

Table 6: Least square means of CAs and SCEs with respect to age

	Group 1		Group 2		Group 3	
	Exposed	Control	Exposed	Control	Exposed	Control
Total CAs	3.66 + 0.26	0.75 + 0.25	3.47 + 0.16	0.73 + 0.14	2.58 + 0.29	0.81 + 0.14
Total SCEs	7.26 + 0.25	3.75 + 0.23	4.50 + 0.06	3.94 + 0.13	6.40 + 0.04	4.12 + 0.15
1. 21 - 30 years	3.64 + 0.47 (7.50 + 0.31)	0.62 + 0.72 (4.12 + 0.25)	3.11 + 0.30 (4.59 + 0.11)	0.90 + 0.18 (4.45 + 0.17)	3.10 + 0.63 (6.56 + 0.15)	0.88 + 0.46 (4.47 + 0.16)
2. 31 - 40 years	3.84 + 0.32 (7.50 + 0.31)	0.75 + 0.27 (4.12 + 0.25)	3.74 + 0.24 (4.43 + 0.09)	0.59 + 0.24 (3.71 + 0.22)	2.03 + 0.43 (6.34 + 0.06)	0.74 + 0.24 (3.76 + 0.25)
3. 41 years and above	3.49 + 0.74 (6.80 + 0.73)	0.87 + 0.16 (3.88 + 0.14)	3.56 + 0.42 (4.49 + 0.16)	0.71 + 0.34 (3.67 + 0.31)	2.61 + 0.85 (6.30 + 0.13)	0.94 + 0.14 (3.74 + 0.31)

In rows 1, 2 & 3 upper values represent CAs and values in brackets SCEs.

Table 7.1: Anova table for SCEs

Group	Source	Mean sum of Square MSS	F. Value	Level of Significance
1.	Effect No.1	0.4191	0.5435	Non-significant
	2	0.3959	0.5134	Non-significant
	3	1.8217	2.3628	Non-significant
2.	Effect No.1	0.0241	0.36800	Non-significant
	2	0.2089	3.1832	Non-significant
	3	5.5290	84.2202	Highly Significant at P < .05
3.	Effect No.1	0.0430	1.2180	
	2	0.2607	7.3714	Significant at P < .05
	3	0.9437	26.6840 at P < .05	Highly Significant

Effect 1 : Effect of Age
 Effect 2 : Duration of Exposure
 Effect 3 : Smoking and Alcoholics

Group 1 : MIG Welders
 Group 2 : MMA Welders
 Group 3 : Autogen Welders

values were found highly significant in smokers and alcoholics in all the three groups (Table 7.2).

Thus exposed smokes and alcoholics showed

more damage than their respective controls. Obe and Herha (1975) also observed that chronic alcohol consumers exhibit a significantly higher fre-

Table 7.2: Anova table for CAs

Group	Source	Mean sum of Square MSS	F. Value	Level of Significance
1.	Effect No.1	0.1637	0.2021	Non-significant Non-significant Highly significant at P < 0.05
	2	0.1974	2.437	
	3	36.2391	44.737	
2.	Effect No.1	0.4100	0.9294	Highly Significant at P < .05
	2	0.2654	0.6016	
	3	20.9472	47.2202	
3.	Effect No.1	1.5420	1.1034	Highly Significant at P < .05
	2	1.0416	0.7453	
	3	4.8033	3.4370	

Effect 1 : Effect of Age
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quency of aberrations in peripheral lymphocytes compared to the control group. Many other studies also reported high values of CAs in smokers of both exposed, and control groups (Vijaylaxmi and Evans 1982; Littlefield and Joiner 1986; Yadav and Thakur 2000 a,b). Rudiger et al. (1976) have reported that IM concentrations of benzo (a) pyrene, the suspected precarcinogen of cigarette smoke, increases the frequency of SCEs almost two fold in human lymphocytes *in vitro*. Similar to the results obtained in the present study, Kussin and Kaley (1974) and Obe and Herha (1975) have also found that smoking coupled with alcohol consumption causes higher frequency of CAs. Thus exposed welders who are both smokers and alcoholics are at a greater risk of having genetic damage from welding fumes.

Association between the human satellite chromosomes has received much attention because

of its possible relation to non-disjunction and translocation (Hansson 1975). During the present investigation the chromosomes of the exposed workers showed a marked increase in various type of SAs per cell in comparison to controls (Table 8). The MIG welders showed highest incidence of SAs per cell (13.80) and number of translocations (6). MMA welders had 11.48 SAs and 6 translocations, whereas the Autogen welders had 9.28 SAs and only 2 translocations. D-G type associations of chromosomes were found to be the highest and 3D group were the lowest. It can be inferred that SAs are related to the incidence of translocations to some extent.

A strong correlation between the frequency of CAs and duration of exposure was observed in group-2 (Table 9.1). A highly significant correlation was evident between the frequency of SCEs and duration of exposure in group 2 and 3 while a

Table 8: Frequency of satellite association in workers exposed to welding fumes

Sample	n	Total no. of metaphases	Satellite association between the D and G acrocentric chromosome							Total	Ass. per cell	SEM
			DD	DG	GG	2DG	2GD	2D2G	3D			
Group 1												
Control	25	2500	18 (0.72)	64 (2.56)	20 (0.80)	4 (0.16)	4 (0.16)	8 (0.32)	2 (0.08)	120	4.8	0.27
Exposed	25	2500	72 (2.88)	138 (5.52)	50 (2.00)	33 (1.32)	27 (1.08)	14 (0.56)	11 (0.44)	345	13.8	0.09
Group 2												
Control	25	2500	33 (1.32)	71 (2.84)	19 (0.76)	7 (0.28)	6 (0.24)	1 (0.04)	4 (0.16)	141	5.64	0.50
Exposed	25	2500	53 (2.12)	116 (4.64)	47 (1.88)	36 (1.44)	17 (0.68)	10 (0.41)	8 (0.32)	287	11.48	0.35
Group 3												
Control	25	2500	27 (1.08)	70 (2.80)	19 (0.76)	10 (0.40)	5 (0.20)	5 (0.20)	3 (0.12)	139	5.56	0.52
Exposed	25	2500	49 (1.96)	101 (4.04)	36 (1.44)	23 (0.92)	12 (0.48)	6 (0.24)	5 (0.20)	232	9.28	0.38

Figures in parenthesis indicate the values per 100 metaphases

negative correlation was observed in group-1 (-0.058). Similarly a very strong correlation was found between age and duration of exposure in the three groups (Table 9.1). A highly significant correlation was also observed between CAs and SCEs in the three groups (Table 9.2). The highest correlation was observed in group-2 (0.8777) and the lowest in group-1 (0.4965). A positive correlation was observed between age and CAs in all the three groups (Table 9.3). the

Table 9.1: Correlation between CAs, SCEs and age with duration of exposure in three different groups

	Group 1	Group 2	Group 3
Age	0.7692**	00.8447**	0.7489**
CAs	0.2815	00.4536**	0.4003
SCEs	- 0.0580	0.4143**	0.6666**

** Highly Significant at P<0.05 Group 1: MIG Welders, Group 2 : MMA Welders, Group 3 : Autogen welders

Table 9.2: Correlation between SCEs and CAs in different groups.

Group	Correlation Coefficient
1.	0.4964 **
2.	0.8777 **
3.	0.6284 **

** Highly Significant at P<0.05

Table 9.3: Correlation between CAs and SCEs with age

	Group 1	Group 2	Group 3
CAs	0.3142	0.4252 **	0.5517 **
SCEs	0.0483	0.3537	0.7129

AGEs and SCEs showed positive correlation in group-2 and 3, while this correlation was negative in group-1 (Table 9.3). The values were highly significant for CAs in group-2 and for both CAs and SCEs in group-3.

In the present study, it has been found that with increase in duration of exposure and age there is increase of CAs in all the three groups of weld-

ers. However, with regard to SCEs there is no general trend. As a person's age increases his resistance decreases. Moreover, with age, duration of exposure is also increasing. This may lead to more damage as the individual inhales more fumes for a longer period of time and with less resistance. Bochkov (1972) and Galloway et al. (1986) reported a significant increase in the frequency of CAs with increasing age, while Goh (1981) and Waksvik et al. (1981) have reported similar relationship for SCE frequency.

Variation in CAs ($r^2 = 10.26, 21.19$ and 30.47) and SCEs ($r^2 = 0.34, 17.17$ and 57.16) could be explained by duration of exposure and age in group-1, 2 and 3 respectively (Table 10). The rate of determination with regard to CAs and SCEs to be the highest in group-3 and these values were lowest in group-1 (Table 10).

From air analysis (Table 1) it is evident that only Cr (VI) and UV radiations exceeded the threshold limit values (TLV) given in 1992 by the ACGIH (American Conference of Governmental Industrial Hygiene) and hence are responsible for the above cytogenetic damage. It is also evident from the literature that welders are exposed to UV rays (Okuno 1987; Elias et al. 1989). UV radiation induces pyrimidine dimers which are mutagenic photoproducts in mammalian cells. 8-Methoxy prosalen alone was not clastogenic but concomitant exposure to non-damaging doses of UV light caused large increase in the incidence of CAs of all types. This was due to the formation of photo products during exposure to UV light (Dean et al. 1991). Replication of UV damaged templates occurs *in vitro* as it does *in vivo* and that this replication results in mutation fixation. UV is one of the primary reasons for increase, in the incidence of skin cancer (Schauberger et al.1992). The International Agency for Research on Cancer has classified fumes and gases from welding as possible carcinogenics to humans (classification group 2B, IARC 1990). The immuno-suppressant azathio-

Table 10: Regression analysis and rate of determination in three groups. Both variables i.e. both CAs and SCEs are related to age and duration of exposure

S.No.		a	x_1	x_2	r^2 Value
Group 1	CAs	0.811	0.911	0.049	10.26
	SCEs	7.717	0.0008	0.012	0.34
Group 2	CAs	1.295	0.038	0.116	21.19
	SCEs	3.920	0.0146	0.0694	17.17
Group 3	CAs	- 1.296	- 0.1143	- 0.0081	30.47
	SCEs	4.672	0.0431	0.0274	57.16

a = Constant incept, x_1 = Regression coefficient in relation to age, x_2 = Regression coefficient with duration of exposure, r^2 = Coefficient of determination.

prine and long wave ultraviolet light have been postulated to have a synergistic effect on DNA resulting in carcinogenic change. Increase in SCEs has been observed in renal transplant patients in response to UV exposure (Dalton et al. 1990). It is evident from the above that UV damages DNA which results in cytogenetic damage. Hexavalent chromium, which constitutes 50-100% of the total released chromium in welding, crosses biological membrane and gets converted to trivalent chromium which binds to protein and nucleic acids, thereby damaging DNA and causing mutations in bacteria, cultured mammalian cells and experimental animals *in vivo* (Blomquist et al. 1977; Stern and Hansson 1986).

It is evident from above that welding fumes containing hexavalent chromium and ultraviolet rays are highly dangerous as they are genotoxic as well as carcinogenic and lead to increased chromosome damage. Duration of exposure and hence age, alcohol consumption and smoking habits add to the damage.

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