

1-1-2004

Sister Chromatid Exchange Frequency in Lymphocytes Cultured from Cotton Gin Workers

MÜNEVVER ATMACA

HÜSEYİN BAĞCI

İBRAHİM AÇIKBAŞ

DİLİHAN GÜMÜŞ

FÜSUN DÜZCAN

Follow this and additional works at: <https://journals.tubitak.gov.tr/medical>

 Part of the [Medical Sciences Commons](#)

Recommended Citation

ATMACA, MÜNEVVER; BAĞCI, HÜSEYİN; AÇIKBAŞ, İBRAHİM; GÜMÜŞ, DİLİHAN; and DÜZCAN, FÜSUN (2004) "Sister Chromatid Exchange Frequency in Lymphocytes Cultured from Cotton Gin Workers," *Turkish Journal of Medical Sciences*: Vol. 34: No. 4, Article 5. Available at: <https://journals.tubitak.gov.tr/medical/vol34/iss4/5>

This Article is brought to you for free and open access by TÜBİTAK Academic Journals. It has been accepted for inclusion in Turkish Journal of Medical Sciences by an authorized editor of TÜBİTAK Academic Journals. For more information, please contact academic.publications@tubitak.gov.tr.

Sister Chromatid Exchange Frequency in Lymphocytes Cultured from Cotton Gin Workers

Münevver ATMACA, Hüseyin BAĞCI, İbrahim AÇIKBAŞ, Dilihan GÜMÜŞ, Füsün DÜZCAN

Department of Medical Biology, Faculty of Medicine, Pamukkale University, Denizli - Turkey

Received: January 14, 2004

Abstract: Genetic biomonitoring of human populations exposed to potential mutagens/carcinogens can be performed using different genetic markers. Sister chromatid exchange (SCE) is one of the most extensively used markers of the early biological effects of DNA damaging agents. In order to assess the genotoxicity associated with exposure to cotton dust, we determined SCE frequency in peripheral blood lymphocytes cultured from 20 cotton gin workers and 20 controls. Student's-t test indicated an increased frequency of SCE in lymphocytes of the workers (14.66 ± 4.18) compared to the controls (10.40 ± 1.37) ($P < 0.0001$). The Mann-Whitney U test indicated that the duration of exposure to cotton dust (<10 years or >10 years) and the work area were not related to the frequency of SCE ($P > 0.05$). The textile industry is rapidly expanding in Turkey, and the results of this study suggest that exposure to cotton dust may constitute a genotoxic hazard.

Key Words: genotoxicity, SCE, mutagen, cotton dust, occupational exposure

Introduction

Many people are occupationally exposed to various biohazardous agents, including radiation, dust, fibers, fumes and organic and inorganic chemicals. These hazardous agents typically are used as raw materials or in intermediate manufacturing processes. Significant increases in sister chromatid exchange (SCE) frequencies have been reported in studies of the human population occupationally exposed to wood dust, coal dust, and diesel emission particles (1).

In order to use raw cotton in the textile, oil and grain industries, the seeds and fibers are separated from each other. The separation procedure and removal of contaminating cotton fibers are performed in a section of the cotton gin. Although the effects of cotton dust on pulmonary function and the respiratory system have been described, to our knowledge, there are no reports related to the genotoxic consequences of cotton dust exposure.

Cotton gin workers are at risk of lung disease, including byssinosis and chronic bronchitis (2,3). In surveys conducted previously, relatively short-term

respiratory problems have been evaluated, and it is not clear whether exposure to cotton dust has permanent effects on pulmonary function (4).

Damage to genetic material can be cytologically observed as chromosome aberration, micronuclei or SCE (5). Therefore, the induction of these responses suggests exposure to genotoxins and possibly carcinogens. These biomarkers are used not only to assess occupational exposure but also to evaluate populations that are environmentally exposed (1,6-7). In the present study we evaluated the genotoxicity caused by exposure to cotton dust by measuring SCE in the peripheral lymphocytes of cotton gin workers.

Materials and Methods

Subjects

Twenty female cotton gin workers (29-54 years old) were included in the study. Blood was collected from the experimental group at the end of the work day. A control group (22-52 years old) included 14 females and 6 males who were not exposed to cotton dust. All volunteers

answered a detailed questionnaire in which health conditions and past and present exposure to possible genotoxic agents such as drug and alcohol usage, smoking, pesticides and herbicides were considered. Written informed consent was obtained from all volunteers.

Human Lymphocyte Culture and SCE Assay

Five milliliters blood was collected from each donor with heparinized syringes. Eight drops of the blood were added to 5 ml of RPMI 1640 medium containing L-glutamine (Biochrom) plus 0.2 ml of phytohemagglutinin (Biochrom). The cultures were incubated at 37 °C for 72 h. After 24 h, 5-bromo-2-deoxyuridine (Sigma) was added to the culture medium at a final concentration of 10 mg/ml, and the incubation was continued in the dark. Colchicines (Sigma) was added 2 h prior to the harvest at

a final concentration of 10 mg/ml. Metaphase cells were harvested by centrifugation, treated with 0.075 M KCl and fixed in methanol-acetic acid 3:1. Slides were prepared and stained by the fluorescence-plus-Giemsa technique (8). Twenty-five well spread metaphases were scored blindly per donor.

Results

Twenty-five metaphases from each donor were evaluated for SCE. SCE frequencies were 14.66 in the subject group compared with 10.44 in the control group, a significant difference ($P < 0.0001$). Data for the individual subjects, along with certain features of the subject and control groups, are summarized in Table.

The effect of age on SCE frequency was evaluated in the subjects and controls using the Mann–Whitney U Test,

Table. Comparison of SCE frequencies in the subject and control groups.

Controls			Subjects				
Control No.	Age (years)	Mean SC SD:±1.37	Subject No.	Age (years)	Duration of Employment (years)	Work Environment	Mean SCE SD:±4.18
C-1	22	11.72	S-1	49	21	C	16.40
C-2	22	8.92	*S-2	52	18	C	11.32
C-3	23	13.28	S-3	47	17	C	23.56
C-4	25	10.40	S-4	38	16	C	10.04
C-5	26	9.16	S-5	52	15	O	10.88
C-6	27	12.84	*S-6	48	14	C	10.60
C-7	28	10.24	S-7	48	14	O	18.36
C-8	31	8.76	S-8	51	14	O	10.48
C-9	33	9.16	S-9	38	13	C	16.96
C-10	35	10.76	S-10	48	12	O	10.32
C-11	37	10.20	S-11	46	10	C	20.04
C-12	38	10.32	S-12	40	10	C	18.20
C-13	39	7.84	S-13	37	7	C	18.20
C-14	39	11.60	*S-14	54	6	O	9.88
C-15	41	10.24	S-15	48	5.5	O	16.76
C-16	41	10.60	S-16	29	5	C	20.32
C-17	41	11.68	S-17	42	4	C	12.64
C-18	45	9.20	S-18	32	4	O	13.64
C-19	46	10.04	S-19	35	4	C	11.32
C-20	52	11.20	S-20	45	2	C	13.28

*Hypertension, O: Open area, C: close proximity to mechanical equipment, SD: Standard Deviation

and no significant differences were found within the groups. Additionally, because of our subject group consisted of all women volunteers, we cannot make any statistical analysis on the sex effect on SCE in our study.

The cotton gin workers were grouped according to their work place environment, either as working in an enclosed area in close proximity to mechanical equipment or in an open area.

The enclosed area consists of 13 workers, 11 seasonal (6 month each year) and 2 continuous whose mean duration of employment is 10.84 years. The open area consists of 5 seasonal and 2 continuous workers whose mean duration of employment is 10.07 years. Differences in SCE stratified by work environment were not significant according to the Mann-Whitney U Test, although workers in the confined areas had a slightly higher frequency of SCE ($P > 0.05$).

Discussion

Cytogenetic assays have been used since the early 1960s to evaluate the exposure of workers to potentially mutagenic and carcinogenic agents (9). SCE analysis has been used in many of these studies. In order to determine agent-related effects in such studies, it is very important to collect samples from a control group that is matched as closely as possible to the experimental group in all regards except for the exposure being evaluated (10). In our study, we have chosen individuals for both the subject and control groups who are non-smokers, do not use alcohol, and have no exposure to pesticides, chemicals or known mutagens. The only possible exception was for 3 individuals in the subject group who had been using antihypertensive drugs. Telez et al. (11), however, found that antihypertensive drugs have no effects on SCE frequency; therefore, we included these 3 individuals in the experimental group. Because cotton gin workers in our region are almost all women, our donor group was exclusively made up of females. Several studies indicate no relationship between SCE frequency and age in individuals less than 60 years old (12-14), and the SCE frequencies of the volunteers in our study did not show any age-related changes. Although, closed area workers' SCE value was slightly higher, we can not find statistically

important differences between workers in open and closed areas, because the closed area environment is well ventilated and working conditions do not have any dose effect differences between the two.

Previous studies indicate that exposure to wood dust and diesel emission results in increased frequencies of SCE (1). There is little data as to whether or not exposure to cotton dust causes a long-term loss of pulmonary function. Christiani et al. (4) have an ongoing study examining the chronic effects of cotton dust exposure on the respiratory system which had continued for 15 years. They have observed a positive relationship between exposure to cotton dust and loss of pulmonary function. Although, it is known that cotton dust produces respiratory illness, there have been no reports on the potential genotoxic, mutagenic, or carcinogenic effects of cotton dust in the literature.

Our results found a significant increase in the SCE frequencies of lymphocytes cultured from exposed workers as compared with controls, suggesting that exposure to cotton dust may have genotoxic consequences. It will be necessary to confirm these preliminary findings using larger study populations, perhaps using prospective studies with factory workers, and using other biomarkers of genotoxicity more mechanistically linked with human disease, such as chromosome aberrations and micronuclei.

Acknowledgments

The authors thank Mehmet Zencir for the statistical analysis. This work was supported by the Pamukkale University Research Foundation.

Correspondece author:

Hüseyin BAĞCI

Pamukkale University,

Medical Faculty,

Department of Medical Biology,

Morphology Building, Kinikli Kampusu,

Denizli - TURKEY

E-mail: hbagci@pamukkale.edu.tr

References

1. Keshava N, Ong T. Occupational exposure to genotoxic agents. *Mutat Res* 437: 175-94, 1999.
2. Bakırcı N, Tümerdem N. Pamuk İplik Üretimi ve İşçi Sağlığına Etkileri. *Turkish Journal of Occupational Health and Safety* 9: 12-23, 2002.
3. Sigsgaard T, Pederson OF, Juul S et al. Respiratory disorders and atopy in cotton, wool and other textile mill workers in Denmark. *Am J Ind Med* 22: 163-84, 1992.
4. Christiani DC, Wang XR, Pan L et al. Longitudinal changes in pulmonary function and respiratory symptoms in cotton textile workers. *Am J Respir Crit Care Med* 163: 847-53, 2001.
5. Zhang Z, Yang L, Zhang Q et al. Studies on the utilization of a plant SCE test in detecting potential mutagenic agents. *Mutat Res* 261: 69-73, 1991.
6. Lazutka JR, Lekevicius R, Dedonyte V et al. Chromosomal aberrations and sister-chromatid exchanges in Lithuanian populations: effects of occupational and environmental exposures. *Mutat Res* 445: 225-39, 1999.
7. Kassie F, Parzefall W, Knasmüller S. Single cell gel electrophoresis assay: a new technique for human biomonitoring studies. *Mutat Res* 463: 13-31, 2000.
8. Perry P, Wolff S. New Giemsa method for the differential staining of sister chromatids. *Nature* 251: 156-58, 1974.
9. Hagmar L, Strömberg U, Tinnerberg H et al. The usefulness of cytogenetic biomarkers as intermediate endpoints in carcinogenesis. *Int J Hyg Environ Health* 204: 43-7, 2001.
10. Albertini RJ, Anderson D, Douglas GR et al. IPCS guidelines for the monitoring of genotoxic effects of carcinogens in humans. International Programme on Chemical Safety. *Mutat Res* 463: 111-72, 2000.
11. Telez M, Martinez B, Criado B et al. Evaluation of the cytogenetic damage induced by the antihypertensive drug nimodipine in human lymphocytes. *Mutagenesis* 16: 345-51, 2001.
12. Albertini RJ, Atland K, Carrano AV et al. Environmental Health Criteria. International Programme on Chemical Safety Guidelines for the Study of Genetic Effects in Human Populations. World Health Organization, Geneva. 1985. pp: 46-1984.
13. Barale R, Chelotti L, Davini T et al. Sister chromatid exchange and micronucleus frequency in human lymphocytes of 1,650 subjects in an Italian population: II. Contribution of sex, age, and lifestyle. *Environ Mol Mutagen* 31: 228-42, 1998.
14. Bolognesi C, Abbondandolo A, Barale R et al. Age-related increase of baseline frequencies of sister chromatid exchanges, chromosome aberrations, and micronuclei in human lymphocytes. *Cancer Epidemiol Biomarkers Prev* 6: 249-56, 1997.