Lung function in sisal ropemakers

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ABSTRACT Lung function was measured by spirometry in 66 workers in a sisal ropemaking factory, and in their matched controls. The major atmospheric contaminant was the lubricant (or a component part thereof) used to soften the fibre. The concentration of airborne matter was generally less than $1 \mu\text{g m}^{-3}$. There was no difference in lung function between the two groups before the start of the working shift, that is, the mixture of softening lubricant and sisal caused no long-term effects. Although there was no change in lung function over the working shift in the group making sisal rope, the control group did show a significant increase in lung function over the same period. This suggests that an effect attributable to the lubricant and sisal dust did exist. In previous studies little mention has been made of the softeners used in the processing of sisal fibre. These additives may exert a significant effect on ventilatory capacity and may act in conjunction with sisal dust.

At present there is little evidence that inhalation of sisal dust causes permanent lung damage. Stott (1958) found that lung function measurements in sisal cardroom workers exposed to high dust levels differed significantly from those of a less-exposed control group. However, Žuškin *et al.* (1972) found no significant difference in any measurements of lung function in sisal workers or their controls before starting the work shift. They suggested that, as the average duration of exposure was only five years, this period may have been too short to lead to any chronic respiratory changes.

There are conflicting views concerning exposure to sisal dust and acute changes in lung function. Whereas Gilson *et al.* (1962) gave no clear evidence of an acute effect of sisal dust on ventilatory capacity, several investigators did find a fall in respiratory function during the work shift (McKerrow *et al.*, 1965; Munt *et al.*, 1965; Valić and Žuškin, 1972; Žuškin *et al.*, 1972). McKerrow *et al.* (1965) showed that this decrease was related to the dust concentration.

Nicholls *et al.* (1973) showed that sisal dust and the oil used to soften the fibres both caused histamine release. They postulated that this was the cause

of the acute changes in ventilatory capacity over the working shift.

The objectives of this study were to determine whether chronic exposure to sisal dust and the lubricant used to soften the fibre caused permanent changes in lung function, and whether an acute reduction in lung function did occur over the course of a working shift.

Population and methods

The total work force of 66 black males employed in a small ropemaking factory was studied. The raw materials to which this group was exposed were sisal fibre (species *Agave Sisalana Perrine*) and a lubricating/softening mixture of bitumen, dark green petroleum jelly, paraffin, woolgrease and Mobisol Ty 30 used to process the fibre. The control group comprised 66 black males not exposed to sisal dust or its softener. These controls were matched to the sisal-exposed workers for age, height and smoking habits and were chosen from 680 randomly selected men working at a sister factory manufacturing steel rope on the same site.

The forced vital capacity (FVC), forced expiratory volume in one second (FEV_{1.0}) and the forced expiratory flow in the middle half of expiration (FEF_{25-75%}) were measured in all subjects using a

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Vitalograph wedge bellows spirometer (waterless). Tests were performed on weekdays before the start of the morning shift (which began at 6 00 am) and subsequently two hours and six hours later (8 00 am and 12 00 noon) on the same day. On each occasion, three recordings were made, and the spirometric trace having the highest values was used. Values were corrected to BTPS. The participants of both groups at each session were selected randomly to minimise bias.

Strategic sampling was performed with a Casella Hexhlet loaded with pre-weighed glass-fibre filters. Particulate concentrations were measured at the sisal-loading conveyor and at three additional sites in the factory.

Results

In the vicinity of the sisal-loading conveyor, where only three people worked, total dust levels of the order of 10 mg m^{-3} were found, with approximately 1 mg m^{-3} being respirable. Away from the loading area, total concentrations of all airborne contaminants were less than $1 \mu\text{g m}^{-3}$. Fibres were detected in general atmospheric samples collected by settlement on glass slides, but the major contaminant, as shown by the appearance of the Hexhlet filters, was the fibre-softener (or one of its components) sprayed initially at the end of the loading conveyor and consequently present at all the rope-manufacturing operations.

Comparison of the sisal-exposed and control groups showed no significant differences ($p > 0.05$) between the means or the standard deviations of the matching variables of age, height and smoking habit (Table 1).

Table 1 Comparison of the sisal-exposed and control groups with respect to the matching variables age, height, and smoking habit

Variable	Control workers (N = 66)	Sisal workers (N = 66)	t
Age (years)			
Mean	32.6	33.3	0.54
Standard deviation	8.3	8.2	(NS)
Height (cm)			
Mean	167.0	167.2	0.21
Standard deviation	5.8	6.0	(NS)
Smoking habit			
Non-smokers			
Number	24	25	
Smokers			
Number	42	41	
Mean (cigarettes/day)	10.2	10.1	0.13
Standard deviation	5.4	4.8	(NS)

NS = Not significant at the 5% level.

t = Student's t test.

Table 2 Comparison of the sisal-exposed and control groups at 6 00 am

Ventilatory function	Control workers (N ₁ = 66)	Sisal workers (N ₂ = 66)	t
FVC (litre)			
Mean	4.34	4.24	0.71
Standard deviation	0.65	0.94	(NS)
FEV _{1.0} (litre)			
Mean	3.59	3.43	1.31
Standard deviation	0.60	0.84	(NS)
FEF _{25-75%} (litre s ⁻¹)			
Mean	3.92	3.78	0.54
Standard deviation	1.54	1.59	(NS)

NS = Not significant at the 5% level.

t = Student's t test for independent samples (degrees of freedom = N₁ + N₂ - 2 = 130).

Table 3 Comparison of the sisal-exposed and control groups for the time interval 6 00 am to 12 00 noon. (The earlier readings are subtracted from the later)

Ventilatory function	Control workers (N = 66)	Sisal workers (N = 66)	Control-Sisal (N = 66)
FVC (litre). Mean differences	0.09	0.02	0.07
SD of differences	0.32	0.35	0.48
t	2.24*	0.55(NS)	1.12(NS)
FEV _{1.0} (litre). Mean differences	0.09	0.03	0.06
SD of differences	0.31	0.35	0.46
t	2.35*	0.79(NS)	1.01(NS)
FEF _{25-75%} (litre s ⁻¹). Mean differences	0.28	-0.05	0.33
SD of differences	0.89	0.73	1.29
t	2.59**	0.54	2.08*

t = Student's paired t test; SD = Standard deviation; NS = Not significant; * = Significant at the 5% level; ** = Significant at the 1% level.

Before the work shift there were no significant differences ($p > 0.05$) between the ventilatory measurements of the sisal rope workers and controls (Table 2).

Neither group showed any change in ventilatory function over the first two hours of the shift but statistically significant improvements in lung function were observed in the control group over the period 6 00 am to noon ($p < 0.05$; Table 3). Mean differences of 90 ml, 90 ml and 280 ml s⁻¹ were observed for FVC, FEV_{1.0} and FEF_{25-75%} respectively. The sisal-exposed workers showed no significant lung function changes over this same time interval.

The differences in FVC and FEV_{1.0} between the control and sisal-exposed groups were not significant but the net difference of 330 ml s⁻¹ in FEF_{25-75%}

Table 4 Comparison of the sisal-exposed and control groups at 6 00 am: dependence on years of sisal exposure. (Differences are controls minus sisal-exposed)

Ventilatory function	Exposure of different groups Comparison	Ex < 4 years		4 years ≤ Ex < 10 years		Ex ≥ 10 years	
		(N = 26) Controls	Sisal	(N = 19) Controls	Sisal	(N = 21) Controls	Sisal
FVC (litre)	Mean Difference T	4.30	-0.25	4.39 0.14 6.83 (df = 2)*	4.25	4.35 0.50	3.85
FEV _{1.0} (litre)	Mean Difference T	3.69	-0.03	3.64 0.26 1.90 (df = 2):NS	3.38	3.43 0.32	3.11
FEF ₂₅₋₇₅ % (litre s ⁻¹)	Mean Difference T	4.24	-0.05	3.98 0.52 1.29 (df = 2):NS	3.46	3.47 0.05	3.42

Ex = Exposure period; T = Kruskal-Wallis T-Value; NS = Not significant; * = Significant at the 5% level; df = Degrees of freedom.

Table 5 Comparison of the sisal-exposed and control groups for the period 6 00 am to 12 noon: dependence on smoking habit. (Differences are controls minus sisal-exposed)

Ventilatory function	Comparison	Smoking habit Smokers (N = 41)		Diff	t	Non-smokers (N = 25)		Diff	t
		Controls	Sisal			Controls	Sisal		
FVC (litre)	Mean changes	0.06	0.08	-0.02	0.29 (NS)	0.13	-0.07	0.20	1.82 (NS)
	SD t	0.26	0.32	0.41	1.87(NS)	0.42	0.37	0.56	
FEV _{1.0} (litre)	Mean changes	0.06	0.09	-0.03	0.39 (NS)	0.13	-0.06	0.19	2.11*
	SD t	0.29	0.37	0.44	1.94(NS)	0.35	0.30	0.46	
FEF ₂₅₋₇₅ % (litre s ⁻¹)	Mean changes	0.12	0.04	0.08	0.49 (NS)	0.54	-0.19	0.73	2.48*
	SD t	0.85	0.69	1.11	2.03*	0.91	0.79	1.48	

Diff = Difference; SD = Standard deviation; t = Student's t test (unpaired for smoker/non-smoker comparison); NS = Not significant; * = Significant at the 5% level.

between these groups was significant at the 5% level (Table 3).

To determine whether or not lung function deteriorated with increasing duration of sisal exposure, the sisal-exposed group was classified in such a way that approximately one-third of the sample of 66 fell into one of three groups, defined as follows:

- exposed for less than four years;
- exposed for between four years and ten years;
- exposed for ten years or more.

A significant effect was not apparent for any measurement of lung function, with the exception of FVC before the work shift (Table 4). In this instance differences of -250 ml, 140 ml and 500 ml were observed between the control and sisal-exposed workers in the groups having less than four years', between four and ten years', and more than ten years' exposure respectively.

To determine whether smoking exerted an effect in conjunction with sisal and the lubricant, the changes in lung function (from 6 00 am to noon) of the sisal-exposed versus control groups were analysed separately, for smokers and non-smokers. The only significant difference was that for change in FEF₂₅₋₇₅ % ($p < 0.05$; Table 5). The mean difference in improvement of lung function, from 6 00 am to noon, between the control and sisal-exposed workers, was 80 ml s⁻¹ for the smokers and 730 ml s⁻¹ for the non-smokers.

Discussion

In the literature (Stott, 1958; Gilson *et al.*, 1962; McKerrow *et al.*, 1965; Munt *et al.*, 1965; Valić and Žuškin, 1972; Žuškin *et al.*, 1972) no mention is made of the mixtures used to process and soften sisal

fibre. Nicholls *et al.* (1973) did suggest, however, that, over a working shift, histamine released by sisal dust and fibre-softener might cause acute changes in ventilatory capacity.

In our factory, the major atmospheric contaminant was the fibre-softener (or one of its components). Only three workers at the start of the loading conveyor were exposed to significant levels of raw sisal fibre.

It has been shown that work in the rope factory did not cause significant acute reductions of ventilatory capacity over a work shift. This contrasts with previous surveys (McKerrow *et al.*, 1965; Valić and Žuškin, 1972; Žuškin *et al.*, 1972) in which atmospheric concentrations of sisal dust were of greater magnitude. The controls did, however, show a significant increase ($P < 0.05$) in ventilatory capacity over a work period. This contrast with the sisal-exposed group suggests an effect caused by acute exposure to sisal and fibre-softener.

An increase in ventilatory capacity for a control group over a working shift was shown by Gilson *et al.* (1962), and McKerrow *et al.* (1965). As a possible explanation, McDermott (1966) suggested the normal diurnal change in airways resistance, that is, a decrease during the course of the day with a subsequent rise in the evening. Guberan *et al.* (1969) also showed a circadian rhythm in shift workers as FEV_{1.0} and FVC increased significantly during the morning shift then subsequently declined in the afternoon and evening shifts.

In this survey there was no significant difference in ventilatory capacity between the two groups at 6 00 am, in other words, exposure to sisal and fibre-softener did not cause any long-term problems. This corroborates the findings of Gilson *et al.* (1962), Munt *et al.* (1965) and Žuškin *et al.* (1972).

To establish whether an effect existed which could be related to length of exposure, the workers were classified in this respect, but no significant differences were obtained with the exception of the FVC at 6 00 am (Table 4). This isolated result, which is significant only at the 5% level, has no biological explanation.

The effect on lung function of sisal dust and fibre-softener over the work shift was most noticeable in non-smokers. This seems to be because, compared with smokers, non-smoking controls showed a greater improvement and non-smoking sisal ropemakers a more marked deterioration over the work shift. Although this effect has been demonstrated with exposure to other types of atmospheric contaminants (Lawther and Waller, 1973; Hankinson *et al.*, 1977) the converse has been found with hemp

and jute (El Ghawabi, 1978).

It has been shown in this survey that exposure to the fibre-softener and low concentrations of sisal dust caused no long-term problems. The acute effects demonstrated over a work shift in the presence of very low concentrations of sisal dust are probably dependent on airborne contaminants arising from the softener. To clarify this point, the effects of the various additives used in processing raw sisal and other vegetable fibres should be investigated more fully.

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