

Work-Related Respiratory Symptoms and Lung Function in New Zealand Mussel Openers

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Our objectives were to measure the prevalence of work-related and nonwork-related respiratory symptoms in a group of New Zealand mussel openers who open green-lipped mussels, and to relate these to demographic factors, work history, smoking history, and pulmonary function measurements. A cross-sectional study of respiratory symptoms and lung function was performed on 224 New Zealand mussel openers (99.6% of the study population) at nine work sites. In addition, peak expiratory flow (PEF) change across-shift was measured at one work site in 19 workers. The mean age of all mussel openers was 33.4 years and the mean duration of mussel opening was 5.0 years; 25% were male, 54.7% were current smokers, and 13.9% were ex-smokers. The reported symptom prevalences were: any wheeze, 35%; work-related wheeze, 23%; any chest tightness, 30.5%; work-related chest tightness, 20.2% (work-related symptoms were defined as symptoms improving on rest days or worse at work). Seventy-two mussel openers (32.3%) answered positively to at least 1 of 4 questions concerning work-related symptoms. The mean predicted FEV₁ (SD) for this group was 74.3% (14.5), and the mean predicted FVC (SD) was 79.2% (16.0). Nineteen workers completed serial PEF, and the mean percentage change was +1.5% at 7 hr, but 8 workers had falls ranging between 1.1–14% after either 1 or 7 hr of work. Duration of mussel opening of greater than 2 years, but less than 7 years (OR = 2.29; 95% CI, 1.07–4.91), and duration of mussel opening greater than 7 years (OR = 3.72; 95% CI, 1.52–9.11), were significant predictors of work-related respiratory symptoms. Female sex (OR = 1.73; 95% CI, 0.83–3.60) was also associated with the presence of work-related symptoms. No relationship was found with measured hygiene parameters or cleaning agents used. In conclusion, duration of work as a mussel opener was associated with the presence of work-related respiratory symptoms, after adjustment for age, sex, and smoking habit. There were marked abnormalities in mean FEV₁ and FVC, although no consistent changes across working shift were noted. Am. J. Ind. Med. 34:163–168, 1998. © 1998 Wiley-Liss, Inc.

KEY WORDS: mussel openers; occupational asthma; respiratory symptoms; pulmonary function; respiratory questionnaire

INTRODUCTION

Certain groups of workers in the seafood industry are already known to be at high risk of developing occupational asthma, and both crustaceans [Musmand et al., 1993] and noncrustaceans are known causes [Chan-Yeung and Malo, 1994; Malo and Cartier, 1993]. Indeed, many individual groups of workers worldwide are potentially exposed to the products of fish and seafood processing. We report on the

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Contract grant sponsor: Health Research Council of New Zealand; Contract grant sponsor: United Kingdom Northern Regional Health Authority

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Accepted 4 December 1997

findings of a study conducted on openers of green-lipped mussels, based in New Zealand.

Green-lipped mussels are farmed in the Nelson-Marlborough region coastal waters by suspending ropes from buoys in sheltered waters, and these ropes are subsequently transported to processing sites. On arrival in the factories, the mussels are stored for a brief period of time in a cooled state, and then time/temperature-controlled cooking takes place. Once cooked, the mussels emerge from the cooker directly onto a conveyor belt in a separate cool room and mussel openers stand adjacent to this belt, from which they manually pick mussels, open them by cutting the adductor muscle, and then drop them into a counting tube. Vapor rises from the cooked mussels in this work area and this condenses on the skin of the face, neck, and forearms or is inhaled. In addition to this main occupational exposure, it is customary to be intermittently exposed to a variety of acid-, alkali-, and chlorine-based cleaning agents.

To the best of our knowledge, occupational asthma relating to mussel exposure has not previously been described.

Initial concern about a possible link between processing green-lipped mussels (*Perna canaliculus*) and the presence of asthma symptoms related to work came from a series of cases presenting to general practitioners, chest physicians, and Occupational Safety and Health (OSH) representatives on South Island, New Zealand. At least one of these patients with recent-onset asthma had a documented improvement in pulmonary function after 6 months off work, consistent with a diagnosis of occupational asthma [Chan-Yeung and Malo, 1994], and another patient had evidence of impaired pulmonary function and a work-related fall in peak expiratory flow (PEF) on the first working day after a weekend break. In addition, there have been many anecdotal reports of respiratory problems at these work sites [Glass et al., 1996].

Accordingly, we conducted a cross-sectional study of nine mussel-processing sites. This study was designed in response to the concerns raised by the constellation of initial case reports. We report here the findings on the prevalence of respiratory symptoms, abnormalities of lung function, and across-shift changes in pulmonary function.

MATERIALS AND METHODS

Questionnaire

Two hundred and twenty-four mussel openers were approached to take part in this study; these workers comprised the total population of mussel openers working at the nine processing sites. Each consenting worker completed a questionnaire inquiring about demographic data, smoking habit, work history, and respiratory symptoms. In addition, a set of specific questions was included to inquire about work-related respiratory symptoms (in this study defined as

worse on the working shift or improving on rest days). These comprised three questions on wheeze and two on chest tightness. For example, the questions on wheeze were as follows: 1) Do you wheeze? 2) Does it come on at work? and 3) Does it improve away from work? For purposes of analysis, all those workers reporting at least one work-related symptom were classified as having work-related symptoms. These workers comprised the cases for further statistical analysis in comparison to noncases.

Pulmonary Function

Pulmonary function was assessed in all workers. This consisted of an FEV₁ and FVC using the best of three forced expiratory maneuvers in the standing position after instruction. This was measured using a calibrated portable spirometer (Microspiro HI-501). The same operator (P.P.) and the same spirometer were used for all worker assessments in this study. For purposes of analysis, all FEV₁ and FVC values were then converted into a percentage predicted value for each individual worker [Quanjer, 1983]. Ethnic differences in predicted values were allowed for by appropriate reduction in predicted values for those with Polynesian ethnicity, as described. This led to a 15% reduction in predicted values of FEV₁ and FVC for Polynesian workers in comparison to Caucasian workers.

Work Environment

Standard features of the working environment were documented by measuring indoor and outdoor air temperature in degrees Celsius, the relative humidity in the work area by using a Casella whirling hygrometer, and air movement using a Kata thermometer. These were measured in order to investigate any relationship between work-related symptoms and differences in temperature and humidity experienced by different workers. In addition, all current cleaning agents were recorded for each site, and the volume of the opening room was calculated.

Across-Shift PEF Change

Three weeks after the initial cross-sectional study, one of the factory sites (site 8) was revisited in order to measure changes in pulmonary function associated with working shift in the mussel openers who had participated in the questionnaire phase. This site was chosen due to its close proximity to the OSH laboratory, and as these data were not regarded as the main theme of the study, only one site was studied further in this way. PEF, using the best of three recordings and one PEF meter, was measured prior to the working shift, one hour into the shift, and again at 7 hr into the working shift. All data relating to this are expressed as a percentage change from the baseline value after 1 and 7 hr of work.

TABLE I. Demographic Data of 224 Mussel Openers Studied at Various Work Sites in New Zealand, 1996

Factory no.	n	Male (%)	Mean age (years)	Mean duration	Current	Ex-smokers (%)	Nonsmokers (%)	Polynesian (%)	Other (%)
				as opener (years)	smokers (%)				
1	78	29.5	34.7	5.4	44.9	19.2	35.9	24.4	75.6
2	19	15.8	32.8	5.6	63.2	5.3	31.6	36.8	68.4
3	13	23.0	23.6	2.2	84.6	7.7	7.7	0.0	100.0
4	10	40.0	33.3	6.1	70.0	10.0	20.0	30.0	70.0
5	14	0.0	38.0	6.8	57.1	28.6	14.3	35.7	62.3
6	13	38.5	35.8	3.5	46.2	15.4	38.5	0.0	100.0
7	39	23.0	36.5	5.0	48.7	12.8	38.5	38.5	66.7
8	23	20.1	27.9	4.2	65.2	4.4	30.4	12.5	87.5
9	14	28.6	31.0	5.0	64.3	7.1	28.6	78.6	42.9
Totals	223	25.0	33.4	5.0	54.7	13.9	31.4	28.1	71.9

TABLE II. Percentage of Respiratory Symptoms Among Mussel Openers by Work Sites

	Factory no.									Totals
	1	2	3	4	5	6	7	8	9	
Number of workers	78.0	19.0	13.0	10.0	14.0	13.0	39.0	23.0	14.0	223.0
Any wheeze	25.6	36.8	53.9	40.0	50.0	7.7	38.5	39.1	57.1	35.0
Wheeze worse at work	18.0	26.3	7.7	30.0	42.9	7.7	23.1	26.1	42.9	23.0
Wheeze better on rest days	12.8	26.3	7.7	20.0	42.9	7.7	10.3	21.7	14.3	16.1
Any chest tightness	25.6	36.8	53.9	20.0	35.7	15.4	33.3	26.1	42.9	30.5
Chest tightness worse at work	16.7	31.6	23.1	20.0	28.6	15.4	15.4	21.7	28.6	20.2
Chest tightness better on rest days	12.8	26.3	15.4	10.0	28.6	7.7	10.3	17.4	0.0	13.9
Any shortness of breath	24.4	21	30.8	30.0	28.6	7.7	46.1	21.7	64.3	30.0
Woken at night short of breath	15.4	10.5	7.7	20.0	14.3	0.0	12.8	0.0	21.4	12.1
Worker reported asthma	10.3	15.8	23.1	20.0	28.6	7.7	25.6	8.3	28.6	16.5
Any work-related symptom	26.9	42.1	30.8	30.0	42.9	23.1	28.2	39.1	50.0	32.3

Data Analysis

PC by the SAS Institute [1988] was used for all analyses. Continuous variables were compared between groups by estimating the mean and standard deviation. Prevalence odds ratios were calculated using the Mantel-Haenszel method [Mantel and Haenszel, 1989]. Logistic regression was performed to calculate adjusted odds ratios (adjusted for age, sex, current, ex-, or never smoker of cigarettes, and the other risk factors in the model) for each risk factor for the presence of work-related symptoms.

All workers in this study gave written informed consent to take part in the study.

RESULTS

Two hundred and twenty-three workers from the potential study population of 224 workers participated (response

rate, 99.6%). Table I illustrates the demographic and smoking data for all workers and between individual sites. These factors were generally uniform across different sites, although site 3 had a younger workforce with a higher proportion of current smokers in comparison to other sites.

There was moderate variation observed in the prevalence of respiratory symptoms between processing sites as shown in Table II, although the prevalence of any work-related symptom was generally high and relatively uniform across working sites.

Table III summarizes the univariate and adjusted analysis (for all variables in the model) for risk factors associated with the presence of work-related respiratory symptoms. The total duration in years as a mussel opener has been taken as a measure of previous exposure to mussels. The most striking finding relates to the association between the presence of work-related respiratory symptoms and work as

TABLE III. Risk Factors for Work-Related Respiratory Symptoms in a Study of Mussel Openers in New Zealand

		Symptoms (n = 72) (32.3%)	No. of symptoms (n = 151) (67.7%)	Odds ratio (95% CI)	Adjusted odds ratio (95% CI) ^a
Sex	Male	13	43	1.00	1.00
	Female	59	108	1.81 (0.90–3.63)	1.73 (0.83–3.60)
Smoking	Never	20	50	1.00	1.00
	Ex	12	19	1.58 (0.65–3.85)	1.31 (0.52–3.32)
	Current	40	82	1.22 (0.64–2.32)	1.08 (0.54–2.14)
Ethnicity	Other	49	107	1.00	1.00
	Polynesian	23	44	1.14 (0.62–2.10)	1.07 (0.56–2.04)
Age	≤24	21	51	1.00	1.00
	25–34	17	35	1.18 (0.55–2.55)	0.87 (0.39–1.97)
	35–44	21	30	1.70 (0.80–3.62)	1.25 (0.56–2.77)
	45+	13	35	0.90 (0.40–2.04)	0.58 (0.23–1.44)
Total duration of mussel opening (years)	0–1.9	12	53	1.00	1.00
	2.0–6.9	38	70	2.39 (1.14–5.03)	2.29 (1.07–4.91)
	7.0+	22	28	3.47 (1.50–8.04)	3.72 (1.52–9.11)

^aAdjusted for all factors listed.**TABLE IV.** Lung Function Results Between Work-Related Symptom Groups in a Study of Mussel Openers in New Zealand

Work-related symptom group	PPFEV ₁ Mean (SD), 95% CI	PPFVC Mean (SD), 95% CI
Symptoms (n = 72)	73.19 (14.40), 69.86–76.50	77.59 (15.43), 74.03–81.15
No symptoms (n = 151)	74.95 (14.64) 72.56–77.20	80.08 (16.32) 77.40–82.58

a mussel opener for 7 years or more. Work as a mussel opener for between 2–7 years was also a significant risk factor for the presence of symptoms, although the odds ratio was lower, suggesting a dose-response relationship between duration of work and symptom reporting. Female sex was also associated with the presence of symptoms.

Table IV compares the mean percentage predicted lung function values between those workers with and without work-related respiratory symptoms. Although the FEV₁ and FVC values were lower in those with symptoms, the most obvious feature of the lung function results relates to the global reduction seen in both groups in comparison to expected values.

Furthermore, evidence of mild airways obstruction was found in a significant number of workers. The mean ratio of FEV₁/FVC (subsequently referred to as FEV%) for all workers was 0.81, and 26 workers (11.7%) had an FEV% of less than 0.70. A further 17 workers (7.6%) had a FEV% of less than 0.75 but greater than 0.70.

Eight individual workers (42.1%) out of the total of 19 that underwent PEF testing across the working shift sustained falls in PEF related to work, although the magnitude of these falls was small (range, 1.1–14.0% fall at either 1 or 7 hr of work). There was no relationship between the presence of a fall in PEF and the presence of work-related symptoms. For example, 1 of the 7 workers with work-related symptoms demonstrated a fall in PEF in comparison to 3 of the remaining 12 workers with no respiratory symptoms (OR = 0.44; 95% CI, 0.04–5.59). The same absence of correlation was found for the relationship between the reporting of ever being diagnosed with asthma and a work-related fall in PEF. These data must be treated and interpreted with caution, in view of the natural (nonwork-related) diurnal variation in PEF that exists in the airways of many asthmatics.

There was no observed relationship between measured air temperature inside the factory site, air humidity or air movement, and the presence of respiratory symptoms.

Although the main risk factor identified for the presence of work-related respiratory symptoms related to duration of exposure to the products of mussel opening, a further analysis was undertaken to investigate the relationship between current use of different cleaning chemicals and the presence of symptoms. The current use of a total of 18 chemicals was individually compared to the prevalence of work-related respiratory symptoms, and the use of none of these was associated significantly with respiratory symptoms. When chemicals were grouped together into either acid-based or chlorine-based agents, there were no differ-

ences in the prevalence of respiratory symptoms between those exposed and nonexposed to these groups. For example, 32.5% of workers exposed to acid-based chemicals reported work-related respiratory symptoms in comparison to 31.8% of nonexposed workers (OR = 1.03; 95% CI, 0.56–1.91).

DISCUSSION

This study has documented, for the first time, symptoms suggestive of occupational asthma and abnormalities in lung function associated with work as an opener in a green-lipped mussel-processing plant. The study found a high prevalence of workers with any work-related respiratory symptom (32.3% of all mussel openers), and the presence of these symptoms related independently to the total lifetime duration of mussel opening for each worker (with a particularly strong risk after 7 years as a mussel opener) and did not appear to relate to any measured hygiene parameter or chemical usage at each factory.

In addition, pulmonary function levels were globally reduced in both workers with and without work-related symptoms, implying a significant reduction from expected values in an adult working population. Furthermore, 26 workers (12%) had an FEV₁/FVC ratio of less than 70%, suggesting the presence of airways obstruction. When across-shift PEF measures were considered, 6 workers of the 19 studied developed falls in pulmonary function after 7 hr of work, although predictably, not all of these workers reported work-related respiratory symptoms. These PEF data need to be interpreted with some caution, as only one site was revisited, and certain workers sustained relatively small falls in PEF.

The pattern of these findings, and in particular the relationship between duration of work and the presence of symptoms, suggest that the workers are possibly becoming sensitized to a particular agent encountered at work, and this may well be a high molecular weight protein derived from the mussel. It is perhaps surprising that duration of work as a mussel opener was a risk factor for the presence of respiratory symptoms in the setting of a cross-sectional study, as one might expect those with work-related symptoms to leave employment early to find a more acceptable work environment. Our findings may relate to the fact that work as a mussel opener is highly skilled, and well-paid in the current economic climate; alternative similarly paid work is probably not easy to find.

Evidence for the associations between work with other types of seafood and the diagnosis of occupational asthma comes largely from studies based within each type of seafood processing industry [Malo and Cartier, 1993], although, in addition, a recent population survey of adult New Zealanders also identified food processors (other than

bakers) at an independently increased risk of various definitions of asthma [Fishwick et al., 1997].

Most reported seafood asthma relates to sensitization to large molecular weight proteins or fish-derived proteins [Malo and Cartier, 1993], and these proteins then elicit a classical IgE-mediated allergic reaction after a period of sensitization. This mechanism is common to other classical examples of occupational asthma such as laboratory animal asthma (due to animal-derived proteins) and baker's asthma (due to flour derived from various cereals [Chan-Yeung and Malo, 1994]).

Previous work in oyster workers, prawn processors, cuttlefish handlers, and trout workers [Malo and Cartier, 1993; Gaddie et al., 1980; Tomaszunas et al., 1988; Sherson et al., 1989] has shown high levels of work-related respiratory symptoms suggestive of asthma, in addition to symptoms of rhinoconjunctivitis and urticarial reactions, a cutaneous manifestation of allergy. In each of these studies, specific IgE to the seafood allergen was elevated in workers with likely occupational asthma in comparison to workers with no symptoms. Furthermore, a recent study of shrimp and clam workers [Desjardins et al., 1995] noted similar levels of occupational asthma and sensitization, in addition to measuring airborne levels of these agents.

Perhaps the most studied group of workers with seafood asthma are those working with snow crabs. Cartier et al. [1984] and Hudson et al. [1985] identified a group of snow crab workers in the early 1980s who underwent full questionnaire assessment, lung function, and skin prick testing to extract of crab in addition to the water used to boil the crabs as part of the cooking process. Interestingly, approximately 16% of workers were confirmed to have occupational asthma. This number was approximately half the number that had previously been thought to have occupational asthma solely on the basis of reported symptoms during an earlier respiratory questionnaire. The main risk factors associated with the presence of occupational asthma in this study were positive skin prick reactions and current smoking. The authors concluded that it is important to carefully evaluate further all those with suggestive symptoms of occupational asthma, so that the true cases can be identified more accurately.

More recently again, a study of salmon processors in the United Kingdom [Douglas et al., 1995] documented 8.2% of workers to have occupational asthma related to exposure to aerosols generated from the process. Furthermore, specific IgE levels were associated with the presence and severity of respiratory symptoms in addition to a marker of exposure (the distance away from the source of the aerosol where the worker was standing). This study also concluded that current cigarette smoking increased the risk of specific IgE production to the salmon and stressed the importance of intervention with standard hygiene measures, as no newly starting workers developed occupational asthma if they began work

after the modification of the local ventilation system had been carried out.

Relatively limited information [Hudson et al., 1985] is available on the follow-up of workers with occupational asthma related to seafood and, in particular, what happens to respiratory symptoms and lung function when the exposure ceases (although two of the index cases fueling this study improved after a short period away from work [Glass et al., 1996]). Follow-up studies of other seafood workers show that recovery after exposure ceases is slow and many workers remain symptomatic, using asthma treatment and exhibiting bronchial hyperresponsiveness. In the case of the snow crab workers, a plateau of improvement was seen 2 years after exposure ceased [Malo and Cartier, 1993; Malo et al., 1988].

This study has noted a high prevalence of work-related respiratory symptoms in this group of workers. Furthermore, these work-related symptoms appear to relate to the duration of employment as a mussel openers, after adjustment for age, sex, and smoking habit, suggesting that increasing sensitization to the etiological agent may occur with increasing duration of exposure.

We are not able to comment further on the potential etiological agent, as we did not assess any measure of allergic sensitization to mussels in this study; neither can we comment on the possible mechanisms of action, as no measures nonspecific bronchial provocation to assess bronchial responsiveness (BHR) or allergic sensitization were made. This is the focus of a continuing theme of research in this group of mussel openers. Again, although we performed routine hygiene assessments at each site, air sampling was not performed, although consideration is now being given to sampling certain sites in order to identify airborne proteins that may be potentially the cause of the problem.

ACKNOWLEDGMENTS

The Wellington Asthma Research Group is funded by a Programme Grant from the Health Research Council of New Zealand. D.F. was funded in part by the Northern Regional Health Authority of the United Kingdom. There are no conflicts of interest in the publication of this paper. We thank all the workers who participated in, or assisted with, the study, and the managers of the mussel-opening sites for their continued cooperation.

REFERENCES

- Cartier A, Malo J, Forest F, Lafrance M, Pineau L, St-Aubin JJ, Dubois JY (1984): Occupational asthma in snow crab processing workers. *J Allergy Clin Immunol* 74:261–269.
- Chan-Yeung M (1995): Assessment of asthma in the workplace. *Chest* 108:1084–1117.
- Chan-Yeung M, Malo J-L (1994): Aetiological agents in occupational asthma. *Eur Respir J* 7:346–371.
- Desjardins A, Malo J, L'Archevêque J, Cartier A, McCants M, SB L (1995): Occupational IgE-mediated sensitisation and asthma caused by clam and shrimp. *J Allergy Clin Immunol* 96:608–617.
- Douglas J, McSharry C, Blaikie L, Morrow T, Miles S, Franklin D (1995): Occupational asthma caused by automated salmon processing. *Lancet* 346:737–740.
- Fishwick D, Pearce N, D'Souza W, Lewis S, Town I, Armstrong R, Kogevinas M, Crane J (1997): Occupational asthma in New Zealanders: A population based study. *Occup Environ Med* 54:301–306.
- Gaddie J, Legge J, Friend J (1980): Pulmonary hypersensitivity in prawn workers. *Lancet* No 8208 vol 2. 1350–1353.
- Glass W, Powers P, Burt R, Fishwick D, Bradshaw L, Pearce N (1996): "Respiratory Symptoms and Lung Function Measurements in Mussel Openers—A Preliminary Study." Division of Occupational Safety and Health, Department of Labour, Wellington, New Zealand.
- Hudson P, Cartier A, Pineau L, Lafrance M, St.-Aubin JJ, Dubois JY, Malo JL (1985): Follow-up of occupational asthma caused by crab and various agents. *J Allergy Clin Immunol* 76:682–688.
- Malo JL, Cartier A (1993): Occupational reactions in the seafood industry. *Clin Rev Allergy* 11:223–240.
- Malo J, Cartier A, Ghezze H, Lafrance M, McCants M, Lehrer Study Leave (1988): Patterns of improvement in spirometry, bronchial hyperresponsiveness, and specific IgE antibody levels after cessation of exposure in occupational asthma caused by snow-crab processing. *Am Rev Respir Dis* 138:807–812.
- Mantel N, Haenszel WH (1959): Statistical aspects of the analysis of data from retrospective studies of disease. *JNCI* 22:719–748.
- Musmand J, Daul C, Lehrer Study Leave (1993): Crustacean allergy. *Clin Exp Allergy* 23:722–732.
- Quanjer PH (1983): Standardised lung function testing. *Bull Eur Physio-pathol Respir [Suppl]* 5.
- SAS Institute (1988): "Language Guide for Personal Computers." Carey, NC: SAS Institute.
- Sherson D, Hansen I, Sigsgaard T (1989): Occupationally related respiratory symptoms in trout processing workers. *Allergy* 44:336–341.
- Tomaszun S, Weclawik Z, Lewinski M (1988): Allergic reactions to cuttlefish in deep-sea fishermen. *Lancet* No 8594 vol 1. 1116–1117.