which he refers, it is nevertheless possible that simpler and less difficult approaches than dust control in spinning mill processes could contribute to improvements in at least some developing countries.

This opinion is based on findings on cotton growing and primary processing in Uganda between 1955 and 1971. The peasant grower would take his produce by bicycle to the buying shed or cotton gin where it was sold by weight. The ginneries (roller gins for processing high grade long staple cottons) sold their finished bales to the Lint Marketing Board to formulae that, among other things, assumed a certain ratio of baled, ginned, cotton weight to raw cotton weight purchased. There was, in consequence, an incentive to eliminate weight loss between picking and baling in at least two stages in the passage from grower to the user (the cotton spinner).

Provided that the cotton buyer could be satisfied that the picked cotton was not unduly contaminated there was little incentive to the grower to be overwhelmingly concerned to eliminate bract fragments—at considerable extra trouble—in the picking, although it is understood that such carryover is less than in machine harvested cottons. More to the point is the situation in the gin. Here the first process of “opening” was intended mechanically to tease out the fibres to prepare them for the roller gins in which the lint was separated from the cotton seed. This provided the only, but potentially very effective, method of removing a proportion of dust contaminants—including, of course, bract fragments—at any point between harvest and delivery to the spinning mill bale opening area. Exhaust ventilation on the cotton opener was an essential ingredient in its successful execution, and cotton openers were built as standard with the necessary fans and ductwork incorporated. These were fairly effective, but at the period described, it was considered that the optimum practicable performance was far from being achieved. Although, byssinosis in Uganda ginneries workers never became a problem—owing, it is generally believed, to the short term and seasonal exposures to cotton dust—good maintenance and proper operation of openers were, none the less, major ingredients in achieving a reasonably low dust atmosphere throughout the rest of the ginning

processes. Whether or not they constituted a long term hazard to health, the dusty conditions that otherwise occurred in ginneries undoubtedly had adverse effects on the wellbeing of workers. Consequently opener function was kept under surveillance both by Lint Marketing Board inspectors concerned with national export product quality and by factories inspectors concerned with working conditions. Nevertheless, there was a commercial disincentive for the ginner to install openers with the most efficient possible dust removal arrangements, as shown by the necessity for the factories inspectorate to take court action against ginneries in which surreptitious means had been adopted to render the openers still less effective by making the exhaust system completely inoperative: visible contaminants in the lint could still be eliminated but the microscopic fragments of the dust content were not, so that the net outturn of baled cotton was somewhat higher than should have been the case. Profit margins could thereby be increased, but in consequence unsatisfactory dust concentrations resulted elsewhere in the gin.

In answer to Parikh's comments, therefore, I suggest that whereas it may be difficult to devise a system to give incentive to growers to reduce bract content in the picked cotton, simple improvements can be made in ginneries openers. Adequate regulatory surveillance of their function, while further improving dust conditions and wellbeing of ginneries workers, might also result in the baled cotton that proceeds to spinning mills having a content of byssinosis generating material sufficiently low that any further dust control measures are unnecessary. At the least, this would permit the use of less sophisticated devices of lower efficiency in achievement of adequate working conditions.

It is recognised that this approach may only be effective in cotton growing areas where cotton is hand picked and roller ginning is practised, and that it may only be workable where the marketing procedures have been adjusted to take account of effects on economic returns throughout the progression from field to finished yarn.

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Renal and immunological effects of occupational exposure to inorganic mercury

Sir,—We read with interest the study by Langworth *et al* on the renal and immunological effects of occupational exposure to inorganic mercury (1992;49:394–401) in which seven indices of renal dysfunction (excretion of albumin, orosomucoid, β_2 -microglobulin, N-acetylglucosaminidase (NAG), and copper; serum creatinine concentration and relative clearance of β_2 -microglobulin) were determined in 89 mercury exposed workers and 75 controls. Urinary mercury concentration (U-Hg) was moderate (25.4 $\mu\text{g/g}$ creatinine in the exposed *v* 1.9 $\mu\text{g/g}$ creatinine in the controls) and no glomerular or tubular effects were found except for a slight increase in NAG.

Recently, we conducted a comparable cross sectional study in mercury exposed chloroalkali workers to validate a new segment specific renal marker, intestinal type human alkaline phosphatase (IAP).¹ In the normal human kidney IAP is exclusively located at the brush border of the tubule epithelial cells present in the S3-segment of the proximal tubule and IAP can thus be considered a specific marker for effects at the S3-segment of the human proximal tubule, a part of the nephron particularly susceptible to some toxins.² An accurate and easy to perform immunoassay using the high affinity specific monoclonal IAP250-antibody has been developed.³

In our study, 83 exposed workers were compared with 84 controls. Their exposure was lower than in the Swedish study (13.4 $\mu\text{g/g}$ creatinine in exposed *v* 1.3 $\mu\text{g/g}$ creatinine in controls) but IAP ($p < 0.001$), NAG ($p < 0.02$), and tissue non-specific alkaline phosphatase (TNAP) ($p < 0.05$) were significantly increased (Mann-Whitney U test). Exposed workers were older than the controls (39.1 years *v* 32.7 years) and multiple regression analyses with each of the enzymes as the dependent variable showed that NAG and TNAP were related only to age. The increased NAG and TNAP excretion in the exposed workers can thus be partly explained by the age difference. For IAP, on the other hand, 12% of the variance in IAP could be explained by urinary mercury whereas age accounted only for a further 2%.

As mercury primarily targets the S3-segment of the human proximal tubule,⁴ our study validated the segment specificity of IAP. We believe that IAP is a more sensitive indicator of renal effects in workers exposed to mercury than NAG and TNAP because even at exposures well under the proposed threshold values (50 µg/g creatinine) IAP excretion was significantly increased and related to the exposure (U-Hg). This sensitivity could be the result of the segment specificity of this marker, thus expressing specific changes that may go undetected by other less specific markers such as NAG, present in the proximal tubules as well as in deeper parts of the nephron.⁵ In a test battery applied in workers exposed to mercury, IAP can be a useful addition. As for all other early renal markers, the clinical relevance and predictive value remain unknown and a multicentre European study, Science and Technology for Environment Protection (STEP) has been set up. In STEP I (1989-90) a battery of more than 25 tests of renal function were studied in more than 1000 workers exposed to different potential nephrotoxins including mercury, solvents, cadmium, and lead. STEP II (1991-93) is a follow up study of workers in which the long term effects of a selected battery of relevant tests will be determined. This study will give some clues as to the clinical predictability of selected renal tests in the context of environmental pollution.

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- 2 Verpooten GF, Nouwen EJ, Hoylaerts MF, Hendrix PG, De Broe ME. Segment-specific localisation of intestinal-type alkaline phosphatase in human kidney. *Kidney Int* 1989; 36:617-25.
- 3 Verpooten GF, Nuyts GD, Hoylaerts MF, et al. Immunoassay in urine of a specific marker for the human proximal tubular S3-segment. *Clin Chem* 1992;38:642-7.
- 4 Bulger RE. Renal damage caused by heavy metals. *Toxicol Pathol* 1986; 14:58-65.
- 5 Guder WG, Ross BD. Enzyme distribution along the nephron. *Kidney Int* 1984;24:101-11.

NOTICES

Minesafe International 1993—The second international conference on occupational health and safety in the minerals industry, Burswood Convention Centre, Perth, Western Australia, 21-26 March 1993.

This event provides the mining industry with a major international forum for the exchange of ideas and developments in occupational health and safety.

The theme of the conference will be "from principals to practice", with the object of facilitating the subsequent implementation of practices throughout the industry that are discussed and examined at the conference. Among the major safety issues to be explored in the plenary sessions of the conference will be: health and safety management at the workplace; man and machinery; accident causation and solutions; lifestyle and health promotion; workplace hazards and their control; future regulation of occupational health and safety in the mining industry.

Workshop sessions will follow some plenary sessions to allow individual topics to be dealt with in greater detail. An exhibition will be staged in association with the conference. Full details can be obtained from: Mr John W Brown, Public Relations Officer, Government of Western Australia, 115 Strand, London WC2R 0AJ. Telephone (071) 240 2881; Fax (071) 240 6637.

The Royal Society of Chemistry environment and toxicology subject groups in association with the health and safety group of the Society of Chemical Industry—the laboratory environment: working with dangerous substances, Tuesday 30 March 1993 at the Scientific Societies Lecture Theatre, Burlington Place (Off Savile Row), London.

The handling and disposal of noxious substances (chemical, radiochemical, and biological) can present a variety of hazards and risks both for laboratory personnel and the environment, and are subject to regulatory control through the implementation of the COSHH Regulations 1988, the

Environmental Protection Act 1990, and the Health and Safety at Work Etc Act 1974, among other legal requirements.

In this symposium techniques for the safe handling and disposal of potentially dangerous and reactive substances will be reviewed, together with the health and insurance implications of working with these materials in the laboratory. Improvements in control and personal protection measures through advances in laboratory design will also be discussed.

This symposium will be of value to laboratory managers, and safety and environment officers in industry, research institutes, and government departments.

Further information may be obtained from Pauline A Sim, Gascoigne Secretarial Services, 24 Southfield Drive, Hazlemere, High Wycombe, Bucks HP15 7HB. Tel: 0494 713664; fax 0494 714516.

University of Cincinnati, NIOSH Educational Resource Center: Environmental and Occupational Health & Safety continuing education courses. January-July 1993 513/558-1730

Pulmonary function testing: NIOSH approved spirometry (NIOSH 010) 5-7 April, 1993; 12-14 July, 1993 (Summer Institute).

All aspects of screening spirometry in the occupational health and outpatient setting are presented through lectures and laboratories, with hands on experience. Successful completion of this course satisfies government training requirements for certain industries. 2.1 CEUs. Call 513/558-1730 for information. Fee: \$395 (\$495 when taken with interpretation of PFT), \$445—Summer Institute.

Interpretation of pulmonary functions tests, 15 July, 1993

This course provides additional pulmonary function training for practising professionals. The focus is on interpretation of test results and reviewing the latest changes and updates in pulmonary function testing. 0.7 CEUs. Call 513/558-1730 for information. Fee: \$125 (\$495 when taken with PFT).