lymphomas (NHL) were evaluated in a case-referent study encompassing 54 cases of HD, 106 cases of NHL, and 275 referents, all alive. Exposure information was obtained by questionnaires posted to the subjects. Crude rate ratios were increased for various occupational exposures, including solvents, welding, wood preservatives, phenoxy acids, and fresh wood (sawmill workers, lumberjacks, paper pulp workers). After further analyses based on logistic regression occupational exposures to welding and creosote remained as significant risk factors for HD. For NHL, occupational exposures to solvents, phenoxy acids, and creosote but also work as carpenter or cabinet maker and contacts with pets (other than dogs, cats, and birds) were associated with significantly increased risks.
---Authors' abstract

783/89 **THE LEAD-EXPOSED WORKER**
D. Rempel, JAMA, 262(4):532-534, July 28, 1989. 19 refs.

The lead standard established by the federal Occupational Safety and Health Administration in 1978 requires physicians and employers to follow very specific guidelines when treating lead-exposed workers. For example, if a worker's blood lead level is 2.90 μmol/L of whole blood or greater, the worker must be removed from work, with full pay and retention of seniority, until the blood level falls below 1.95 μmol/L. Physicians play a key role in the implementation of the lead standard; the standard specifies frequency of blood lead measurements, frequency and extent of medical monitoring, and medical removal from work. This article reviews the lead standard as it applies to physicians and makes recommendations about managing the worker with lead poisoning.
---Author's abstract

784/89 **MUTUAL METABOLIC SUPPRESSION BETWEEN BENZENE AND TOLUENE IN MAN**
O. Inoue, et al, International Arch. Occup. Environ. Health 60:15-20, 1988

The exposure intensity during a shift and the metabolite levels in the shift-end urine were examined in male workers exposed to either benzene (65 subjects; the benzene group), toluene (35 subjects; the toluene group), or a mixture of both (55 subjects; the mixture group). In addition, 35 non-exposed male workers (the control group) were similarly examined for urinary metabolites to define background levels. A linear relationship was established between the intensity of solvent exposure and the corresponding urinary metabolite levels (i.e., phenol, catechol and quinol from benzene, and hippuric acid and o-cresol from toluene) in each case when one of the three exposed groups was combined with the control group for calculation. Comparison of regression lines in combination with regression analysis disclosed that urinary levels of phenol and quinol (but not catechol) were lower in the mixture group than in the benzene group when the intensities of exposure to benzene were comparable, indicating that the biotransformation of benzene to phenolic compounds (excluding catechol) in man is suppressed by co-exposure to toluene. Conversely, metabolism of toluene to hippuric acid was suppressed by benzene co-exposure. Conversion of toluene to o-cresol was also reduced by benzene, but to a lesser extent. The significance of the present findings on the mutual suppression of metabolism between benzene and toluene is discussed in relation to solvent toxicology and biological monitoring of exposure to the solvents.
---Authors' abstract

785/89 **STUDIES OF ACUTE NEPHROTOXIC POTENTIAL OF TRICHLOROETHYLENE IN FISCHER 344 RATS**
S. K. Chakrabarti and B. Tuchweber, J. Toxicol. Environ. Health 23:147-158, 1988

Group of male Fischer 344 rats, after pretreatment with phenobarbital (80 mg/kg, ip 3 d), were treated ip in corn oil with 0, 5.5, 11.0, and 22.0 mmol trichloroethylene (TRI) per kg body weight. Urines were collected 24 h after the treatment and the animals were then sacrificed. The nephrotoxicity of TRI was then studied by measuring certain biochemical parameters characteristic of renal injury and its in vivo metabolism by quantitating the TRI principal urinary metabolites. Treatment of rats with TRI up to 11 mmol/kg did not influence any of the measured biochemical parameters of nephrotoxicity. On the other hand, significant increases in the urinary level of N-acetyl-β-glucosaminidase (NAG) and glucose as well as serum urea nitrogen were observed at 24 h only at the highest dose level (22 mmol/kg) or TRI. Urinary excretions of both trichloroethanol and trichloroacetic acid reached an apparent saturation at the highest dose level of TRI. In inhalation studies, urinary levels of γ-glutamyltranspeptidase, NAG, glucose, proteins, and serum urea nitrogen were significantly increased at 24 h when rats were exposed to either 1000 or 2000 ppm TRI for 6 h. The capacity of renal cortical slices to accumulate p-aminohippurate was significantly reduced 24 h after the exposure to 22 mmol TRI/kg (ip), or to 1000 or 2000 ppm TRI. These results have demonstrated that TRI exerts its acute nephrotoxic potential at a very high dose level and produces nephrotoxic insult at the proximal tubules and possibly glomerular regions of the rat kidney, whether exposed by inhalation or by ip route. These data further indicate the involvement of a capacity-limited metabolism in the expression of acute nephrotoxicity due to TRI in Fischer 344 rats.
---Authors' abstract