and duration of exposure, the exposures have been recent and of limited duration. The full manifestation of the effects of shipyard employment would not yet be expected to be present in this group" (Ex. 162-C, p. 1).

In comparison with the mortality observed in white males in Connecticut, the overall mortality for the cohort with 11.5 years of exposure was significantly (p less than 0.05) elevated (356 observed, 316 deaths expected). Mortality from cancer at all sites was also in excess (90 observed, 80 expected). The major sites of cancer increase were the lung (35 observed, 26 expected) and the gastrointestinal tract (19 observed, 15 expected), two cancer sites known to be related to asbestos exposure. These excesses were seen in both production and support workers, whereas office employees from the same shipyards experienced mortality similar to that of the general male population of Connecticut. Finding such excesses in a cohort that had relatively short employment and that had been followed for a relatively short period of time was, in the authors' words, "unexpected" and leads to augmented concern for the next two or three decades" (Ex. 162-C, pp. 3, 4).

The study (Ex. 162-C) provides additional qualitative evidence of the excess risk of lung cancer mortality and GI cancer mortality experienced by asbestos-exposed workers. Although these investigators were surprised to find such excesses following relatively recent asbestos exposure, other authors (Ex. 306-B, Ex. 320) have noted that significant increases in the lung cancer death rate begin to appear 10 to 14 years after the first exposure and peaks between 30 and 35 years after (Ex. 306-B, p. 57).

Zoloth and Michaels (Ex. 163-E) performed a proportionate mortality analysis of 381 deaths that occurred among white males who had been members of a local New York chapter of the Sheet Metalworkers International Association for at least 10 years. Specific estimates of asbestos exposure levels were not given; however, exposure was described as being intermittent and incidental. Half of the local union members were employed in installation of metal ducts. The expected distribution of deaths was based on U.S. white male mortality rates, with adjustments for age and date of death.

There was significant (p less than 0.05) excess mortality from all cancers (PMR-152), lung cancer (PMR-160), colorectal cancer (PMR-232), and non-Hodgkins lymphoma (PMR-236). In addition, three deaths from mesotheloma were observed. The authors calculated standardized mortality odds ratios (SMOR) using arteriosclerotic heart disease as a referent to offset some of the potential biases in PMRs. The calculated SMORs were reported to be virtually identical to the PMRs, indicating the absence of any significant biases in the PMR's for cancer. The authors concluded that this study, with an overall pattern of observed mortality consistent with that found in other populations exposed to asbestos, "strongly suggests the presence of significant asbestos-related illness is [sic] a population with 'secondary' asbestos exposure" (Ex. 163-E, p. 11).

The interpretation of these results is limited by the design of proportionate mortality studies. Although the investigators reported that half of the local union members were employed in installations of metal ducts, and thus were must likely to be exposed intermittently to asbestos, it is not known what proportion of the deceased members were so employed. Moreover, although the observed deaths occurred in a predominantly metropolitan population, the expected distribution of deaths was based on general U.S. mortality rates; the resultant comparison is not ideal because of the generally recognized differences in mortality patterns of urban populations in comparison to those of the overall U.S. population. These investigators did strengthen their study results by calculating SMORs.

2. Evidence of an Excess Risk of Lung Cancer and Mesothelioma at Low Cumulative Exposures of Asbestos

In establishing whether an existing permissible exposure limit is inadequate for protecting workers against the risk of occupational disease, the Agency relies principally on the findings of quantitative risk assessments and an evaluation of the significance of the risk presented by exposure at the existing PEL. After conducting the quantitative risk assessment for asbestos, OSHA concludes that the 2-f/cc PEL is inadequate for worker protection and that reduction of the PEL is warranted (see Section V, Quantitative Risk Assessment, and Section VI, Significance of Risk). OSHA's finding that the 2 f/cc PEL is inadequate is supported by the observations of excess cancer mortality among workers who have been exposed to cumulative levels of asbestos lower than would be permitted by lifetime exposure to 2 f/cc. These observations, first referred to in the November proposal and discussed above, were derived from the studies by Dement et al. (Exs. 84-36; 84-37), Henderson and Enterline (Ex. 84-48), Finkelstein (Ex. 84-240), and Seidman et al. (Exs. 84-87, 261-A). In addition, a number of studies have recorded cases of mesothelioma among members of the families of asbestos workers (Anderson et al., Exs. 84-16, 84-17; Vianna and Polon, Ex. 84-186); Li et al., Ex. 84-149). Mesothelioma has also been observed among community members living near asbestos mines and factors (Wagner et al., Ex. 2-21; Newhouse and Thompson, Ex-84-70). For example, in 1976, Anderson et al. (Ex. 84-16) reported that 4 cases of pleural mesothelioma had been diagnosed among 620 family contacts of amosite factory workers. Presumably, family contacts received their exposure to asbestos from dust carried home on the worker's clothing, and especially during the laundering of dusty clothes. Although exposure measurements were not taken for family contacts, OSHA considers it very likely that their cumulative exposure was less than the cumulative exposure that would result from lifetime exposure to the 2 f/cc standard. OSHA believes that these findings, as well as the observation in epidemiological studies of excess mortality resulting from low cumulative exposures to asbestos, further support the Agency's finding from the risk assessment that the 2 f/cc PEL is inadequate for protecting workers against the risk from lung cancer and mesothelioma.

3. Experimental Evidence

Several animal studies are contained in the record that show that experimental animals, when administered asbestos fiber by inhalation, injection, or implantation, develop malignant tumors at a rate higher than unexposed animals (Exs. 84-338; 84-320; 84-205; 94-96; 84-197; 84-120; 84-128; 84-240; 84-193; 84-195). No rulemaking participant questioned the causal relationship between asbestos exposure and the development of malignancies in experimental animals. OSHA believes that, while these studies in general support the findings of epidemiology studies, they are more germaine to the issues brought up during the rulemaking regarding the relationship between fiber type and dimension and the carcinogenicity of asbestos. OSHA discusses these experimental studies in a later section that deals with the issues of fiber type and size.

4. Summary of the Evidence of Lung Cancer and Mesothelioma

After reviewing the studies discussed above, OSHA finds that the evidence for