this varied between tools and type of exposure. Correlations between the measurement results and tool predictions also varied with tool and exposure type. Furthermore, a wide range of exposure estimates were observed when different users were asked to apply the same tools to the same scenario conditions.

Conclusion Models to estimate exposure and risk are essential elements of the toolbox of occupational hygienists and risk assessors and managers. However, there is increasing evidence that performance varies between tests, type of exposure and scenario conditions. More importantly, users appear to struggle to apply the tools consistently, leading to wide ranges in estimated exposures. There is an urgent need for the development and implementation of generic quality control procedures for use of exposure tools, to reduce the large uncertainties when applying these tools, both to prevent workers from being excessively exposed and unnecessarily implementation of stringent exposure control measures.

A QUASI EXPERIMENTAL UNCONTROLLED BEFORE-AFTER STUDY TO ASSESS IMPACT OF CASHe INTERVENTIONS IN THE YEAR 2016–2017 AT PETROCHEMICAL INDUSTRY

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Introduction To inculcate best practices in the field of OH; RIL has launched the initiative in 2003 known as CASHe in all 7 manufacturing sites. The various interventions under this project are excellent examples of team work of medical, safety, environment and technical department of respective manufacturing sites. Previously there wasn’t any scientific research approach to evaluate their outcomes at RIL-DMD-which is amongst the largest petrochemical site of RIL. So the present study was carried out to address this need.

Methods There are total 12 manufacturing plants and each of them were considered as a unit of the study. The secondary data from all these 12 plants were collected going 1 year retrospectively using semi-structured proforma regarding various CASHe interventions and compiled using MS Excel 2007. Data triangulation was done using OHC data (IH Surveys and HMIS) with plant data. After necessary editing and exclusion (i.e. projects lacking before-after data, qualitative data) student paired T test was applied to find out statistical significance.

Result There were total 187 interventions (mean-15.58/plant) addressing noise, heat, chemical exposure, ergonomics, safety and environmental hazards. Out them 142 completed and 45 in progress. Total 3316 persons (928 employee and 2388 contract workers) trained for different OH training with average 600 man hours/department. Central theme for CASHe: 2016-2017 achieved statistically significant result in term of reduction of exposure to benzene vapour by vapour recovery unit (p=0.01), noise (p=0.001) and heat etc.

Discussion CASHe projects outcomes were successful in terms of reducing hazards, workplace improvements and wellness of employee. So the present study experiments guide other industries to deal with noise, heat, highly toxic material safely and reduce their exposure along with taking care of life style diseases of their employees.
Many chemicals are classified as known human carcinogens, based at least in part on epidemiological evidence. However, occupational epidemiological studies often lack detailed and reliable individual-level exposure information, and only may be capable of qualitatively indicating increased risk among ‘exposed’ versus ‘unexposed’ groups. Although this information might be helpful for hazard identification, it is of limited use for risk assessment. Therefore, many investigators have placed greater emphasis on obtaining measurements and deriving quantitative relationships of individual exposures over time. In addition to facilitating the identification of potentially non-linear exposure response relationships, including exposure thresholds for risk, this information helps improve risk assessments. Evidence of nonlinear exposure-response sometimes aligns with knowledge about the agents’ route of exposure, mode of action, metabolism and elimination. Furthermore, the identification and application of sensitive biological markers of exposure can help define groups of workers with exposures that are biologically meaningfully different from those of other groups, allowing more precise characterisation of the risk function and possibly the shape of the underlying dose-response function. For many carcinogens, the exposure-response is becoming clearer, and for some it is not linear. Furthermore, where there is evidence of exposure thresholds, epidemiological data may provide direct evidence of the exposure level where risk is increased, i.e., a meaningful departure from background rates. This presentation will review the epidemiological evidence on several known occupational carcinogens that suggest nonlinear risk functions, drawing on examples such as hexavalent chromium, crystalline silica, ionising radiation, vinyl chloride and benzene. Possible mechanisms that give rise to the observed nonlinear relationships (e.g., production of carcinogenic metabolite, overwhelming clearance pathways or repair mechanisms, etc.) will be discussed, and recommendations on how the integration of evidence from different lines of inquiry holds promise for identifying nonlinear exposure-response relationships for occupational carcinogens.

**Results**

There is evidence for a strong association between high cumulative exposure to benzene and AML, but little support for an etiologic relation with peak exposure; in contrast, peak benzene exposure has been associated with risk of myelodysplastic syndrome. Peak, but not cumulative formaldehyde exposure has been associated with various LHM. For styrene, no relationship was seen between number of peaks and several cancers of interest. These patterns may be due to variable definitions of peak exposures or may reflect differences in toxicokinetic and carcinogenic mechanisms of these chemicals.

**Discussion**

A peak exposure should be defined quantitatively in terms of exposure intensity, duration, and frequency of occurrence. Future epidemiologic research should apply standardized definitions that can be applied to existing datasets and in newly initiated epidemiologic studies that are consistent with or shed light on the underlying disease processes.

**Abstracts**

### 1715a

**EPIDEMIOLOGICAL EVIDENCE FOR NON-LINEAR EXPOSURE-RESPONSES FOR OCCUPATIONAL CARCINOGENS**

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Effects of crystalline silica on the respiratory tract have been demonstrated in a large number of epidemiological studies. Crystalline silica is a known occupational carcinogen with the lung as main target organ and can cause silicosis as well as chronic obstructive pulmonary disease (COPD). While these hazards are well characterised, there is an ongoing debate on the quantitative exposure-response relationships for crystalline silica and these respiratory endpoints.

Both for regulatory and preventive purposes, the demonstration of an exposure threshold which almost excludes any human health risk would be highly desirable. Another option would be the derivation of an exposure-risk relationship associating a given exposure level with a specific lifetime risk, e.g. for lung cancer. However, chronic inflammation – believed to be a threshold effect – is currently considered as the most likely mechanism relevant for both the development of silicosis and lung cancer, while it is unclear whether silica-induced lung cancer requires the presence of silicosis.

This presentation will review the current epidemiological evidence for the derivation of a threshold with respect to the development of lung cancer and silicosis focusing on high-intensity exposures, often characterised by short-term high intensities, are well established as major etiological contributors to acute adverse health outcomes. Associations between peak chemical exposures and risk of occupational cancers have been contrasted with observed effects related to more conventional metrics, cumulative exposure and exposure duration. However, the definitions of peak exposure have been highly idiosyncratic, which complicates data interpretation, risk assessment and ultimately setting occupational exposure standards. Thus, there is a need to develop a standardised epidemiologic framework for defining and assessing peak exposures in occupational epidemiology studies of chemical carcinogens, with consideration of underlying toxicological mechanisms, exposure assessment requirements, and policy implications.

**Methods**

We reviewed and contrasted cancer risk findings for peak and cumulative exposures from influential occupational epidemiology studies of benzene and formaldehyde, both classified by IARC as causes of lymphohematopoietic malignancies (LHM) in humans, and for some other possible chemical carcinogens.

**Results**

There is evidence for a strong association between high cumulative exposure to benzene and AML, but little support for an etiologic relation with peak exposure; in contrast, peak benzene exposure has been associated with risk of myelodysplastic syndrome. Peak, but not cumulative formaldehyde exposure has been associated with various LHM. For styrene, no relationship was seen between number of peaks and several cancers of interest. These patterns may be due to variable definitions of peak exposures or may reflect differences in toxicokinetic and carcinogenic mechanisms of these chemicals.

**Discussion**

A peak exposure should be defined quantitatively in terms of exposure intensity, duration, and frequency of occurrence. Future epidemiologic research should apply standardized definitions that can be applied to existing datasets and in newly initiated epidemiologic studies that are consistent with or shed light on the underlying disease processes.