MEDI CHEM

NEWSLETTER

July 2008

Medichem 2008 Board election

With the last Newsletter you received the call for candidates for this year's Medichem Board election.

The following Medichem members were nominated to the secretary and consented to stand as Board candidates:

Stephen Borron and Kevin Trangle (both USA), Peter Boogaard (The Netherlands), Adrian Combrinck (South Africa), Elpida Emmanouil-Nikoloussi (Greece), Jiin Ger (Taiwan), Jorge Morales (Mexico), Abed bin Onn (Malaysia), and Edwin Whiteside (New Zealand).

Les Yee and Frank Rose, after having served on the Board for three and two terms, respectively, have decided not to run for another term. The Medichem Board gratefully acknowledges the outstanding service both have provided to Medichem in the past and conveys our sincere thanks and best wishes for the future.

Since this year up to eight seats on the Medichem Board are to be distributed, there are candidates from as many countries as there are vacancies on the Board, and thus a part of this year's election goes with a "silent vote".

According to Article 5 Sect. 4.1 "to ensure maximum international representation, each Board Member shall be from a different country." Thus, Stephen Borron and Kevin Trangle, both being from the same country, have to be elected in a formal procedure.

Dear Members, please therefore **vote for one of the two** nominated candidates. Please show your personal interest in Medichem by casting your vote, either by fax or mail. The necessary ballot form for the election has been provided with this Newsletter.

To be valid, the ballot form must reach the Secretary **no later than August 24th, 2008**. Only Medichem members in good standing may vote.

Forms coming from anonymous voters have to be considered void. It goes without saying that the names of those voting will only be used to identify voters as members in good standing and will be known to no-one other than the Secretary. I would like to thank you for your kind cooperation.

In conclusion: please vote for one of the two candidates from the USA.

Dr. Georg Wultsch
(Graz, Austria)

MEDICHEM - Occupational and Environmental Health in the Production and Use of Chemicals

Founded 1972 in Ludwigshafen, Germany

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Nearly 20% of the working population in Europe and North America is engaged in shiftwork, and - owing to the nature of the production processes involved - the chemical industry is particularly dependent on this type of work organization. In December 2007, an expert Working Group convened by the IARC Monographs programme has concluded on the basis of "limited evidence in humans for the carcinogenicity of shift-work that involves nightwork", and "sufficient evidence in experimental animals for the carcinogenicity of light during the daily dark period (biological night)" that shiftwork that involves circadian disruption is probably carcinogenic to humans (Group 2A)."

The IARC further explained: "Epidemiological studies have found that long-term nightworkers have a higher risk of breast cancer risk than women who do not work at night. These studies have involved mainly nurses and flight attendants. The studies are consistent with animal studies that demonstrate that constant light, dim light at night, or simulated chronic jet lag can substantially increase tumour development. Other experimental studies show that reducing melatonin levels at night increases the incidence or growth of tumours. These results may be explained by the disruption of the circadian system that is caused by exposure to light at night. This can alter sleep-activity patterns, suppress melatonin production, and disregulate genes involved in tumour development. Among the many different patterns of shiftwork, those that include nightwork are most disruptive to the circadian system."

Up to now, only a short "Policy Watch" notice on this decision has been published in Lancet oncology, and a look at the reference section of this notice leaves open the question whether the "limited evidence" in humans should rather be regarded as "insufficient evidence".

Cohort study of cancer risk among male and female shift workers

(Judith Schwartzbaum, Anders Ahlbom, Maria Feychting; Scand J Work Environ Health 2007; 33:336-343.)

Melatonin, a Hormone that inhibits experimentally induced cancers, is suppressed by nighttime exposure to light so that nighttime shift workers may be at an increased risk of cancer. Previous studies of shift workers found an increased risk of breast cancer among women and suggested a possible increased risk of colon cancer among women and prostate cancer. The present study was conducted to see whether these previous findings could be confirmed and whether shift workers are at elevated risk for cancer at additional sites. Methods Altogether 2,102,126 male and l,148,661 female workers were identified who worked in both 1960 and 1970. Their Jobs were classified according to the percentage of shift workers, and they were followed from 1971 through 1989 or until they were diagnosed with cancer or died. Standardized incidence ratios (SIR) were used to compare the adjusted cancer incidence rates for shift workers with those for nonshift workers.

Cancer rates were not elevated for the male shift workers or for the female shift workers (all sites combined)

No evidence was found for an association between shift work and breast or prostate cancer, or all cancer sites combined among shift workers.

Recently, H.A. Kolstad from the Department of Occupational Medicine of the Aarhus University Hospital in Denmark published a systematic review that concerns the role of nightshift work in the risk of breast cancer or other cancers:

Nightshift work and risk of breast cancer and other cancers - a critical review of the epidemiologic evidence

(Kolstad HA; Scand J Work Environ Health; 2008; 34:5-22)

METHODS: Studies that specifically included information on nightshift or shift work and reported cancer occurrence were focused upon. A systematic search of

Medline and the Science Citation Index was conducted until May 2007. The quality of each paper was discussed with respect to design, exposure and outcome information, bias, confounding, and exposure-response assessment.

RESULTS: Thirteen relevant reports were found, and eight reported the relative risk for breast cancer, three for prostate cancer, three for colon cancer, and four for all cancers. Most of the studies had crude information about nightshift work, four register-linked studies had no individual exposure information but relied on exposure probabilities assessed on a group level, and no studies analyzed cancer risk according to the cumulative number of night shifts (however, most of the studies did so according to the number of years of nightshift work). Confounding did not seem to be of major concern. The presentation of the results was not always complete, and it would have been appreciated if the reasons for leaving some findings out had been reported. There were indications of a long-term effect of nightshift work (more than 20-30 years), but the number of positive studies was small. In addition, they were all conducted among nurses, and the risk estimates were only moderately increased. This situation makes the results sensitive to bias, chance, and confounding.

CONCLUSIONS: There is limited evidence for a causal association between nightshift work and breast cancer, while there is insufficient evidence for prostate cancer, colon cancer, and overall cancer.

I'm afraid that "further studies are needed …"

Dr. Georg Wultsch
(Graz, Austria)
Dr. Michael Nasterlack
(Ludwigshafen, Germany)

Parental occupational exposure to pesticides and the risk of childhood leukemia in Costa Rica

(Patricia Monge, Catharina Wesseling, Jorge Guardado, LicComp, Ingvar Lundberg, Anders Ahlbom, Kenneth P Cantor, Elisabete Weiderpass, Timo Partanen; Scand J Work Environ Health 2007; 33:293-303)

Parental exposure to pesticides and the risk of leukemia in offspring were examined in a population-based case - control study in Costa Rica.

All cases of childhood leukemia in 1995-2000, were identified at the Cancer Registry and the Children's Hospital. Population controls were drawn from the National Birth Registry. Interviews of parents were conducted using conventional and icon-based calendar forms. An exposure model was constructed for 25 pesticides in five time periods

Mothers' exposures to any pesticides during the year before conception and during the first and second trimesters were associated with the risk [odds ratio (OR) 2.4, 95% confidence interval (95% CI) 1.0-5.9; OR 22, 95% CI 2.8-171.5; OR 4.5, 95% CI 1.4-14.7, respectively] and during any time (OR 2.2, 95% CI 1.0-4.8).

An association was found for fathers' exposures to any pesticides during the second trimester (OR 1.5, 95% CI 1.0-2.3).

An increased risk with respect to organophosphates was found for mothers during the first trimester (OR 3.5, 95% CI l.0-12.2) and for fathers during the year before conception and the first trimester (OR l.5, 95% CI l.0-2.2 and OR 1.6, 95% CI 1.0-2.6, respectively), and benzimidazoles during the first, second, and third trimesters of pregnancy (OR 2.2, 95% CI l.0-4.4; OR 2.2, 95% CI l.0-5.0; OR 2.2, 95% CI l.0-5.2, respectively). There was a suggestion of an exposure-response gradient for fathers as regards picloram, benomyl, and paraquat. Age at diagnosis was positively associated with fathers' exposures and inversely associated with mothers' exposures.

The results suggest that parental exposure to certain pesticides may increase the risk of leukemia in offspring.

Dr. Georg Wultsch
(Graz, Austria)

The topic of pesticides and childhood cancer is indeed a never-ending one. I have dealt with it repeatedly in the past, and I confess that it appears almost hopeless to get a secure grip on it. The possible link between pesticides and different childhood cancers has been examined in many studies. Exposure assessment included maternal or paternal or both; occupational and home use; active application or passive exposure; farming, gardening and other environments. Exposures were estimated prior to, during or after pregnancy, with and without allowing for latency periods or time lag prior to date of first diagnosis. Some studies examined interactions between host susceptibility factors, notably enzyme polymorphisms, and the resulting cancer risks. The majority of studies have been carried out in leukemia, followed by brain cancer, often without differentiation among histological subgroups and thus in many cases probably "comparing apples with pears". Reviews have been published in 1997, 1998 and 2006 where the evidence was found suggestive but not conclusive. The review quoted in the following was an extended update of the latter one.

Pesticides and childhood cancer: an update

(Nasterlack M; Int J Hyg Environ Health 2007; 210:645-657)

METHODS: The PubMed database was searched to identify published studies on this topic issued between 1998 and 2006.

RESULTS: Thirty-six new studies have been identified for this review. Some cohort studies and the majority of the case-control studies suggest an increased risk for the cancer types studied, associated with exposure to pesticides in at least one of a large variety of exposure categories. However, the evidence is conflicting with regard to cancer types as well as to causative factors across studies. The major shortcomings concern exposure assessment, where, e. g., ''farming'' is treated equal to ''exposure to pesticides'', disregarding other possible exposures, e.g., to biological or infectious agents, and hitherto unidentified lifestyle factors. Also, many exposure categories used, mainly in case–control studies, lack chemical or toxicological plausibility. In most studies exposures were categorized as ''ever vs. never'', with little regard of exposure intensity or duration.

CONCLUSIONS: The available literature does not allow firm conclusions with regard to pesticides and any type of childhood cancer. But even if the reported associations were true, exposure to pesticides could not explain the vast majority of childhood cancer cases. Investing in the acquisition and critical review of exposure information appears to be the crucial step for causal assessment in future research. However, focusing on the presence of pesticides, and not asking the question why they were used, might mask relevant associations to other causative agents.

Dr. Michael Nasterlack
(Ludwigshafen, Germany)

Population-based study on occupational risk factors for preeclampsia and gestational hypertension

(Edwige Haelterman, Sylvie Marcoux, Agathe Croteau, Michele Dramaix ; Scand J Work Environ Health 2007; 33:304-317)

Preeclampsia is a leading cause of maternal and perinatal morbidity. Work-related factors may influence the occurrence of this disorder. This case-control study estimated the associations between work-related physical and psychosocial factors and the risk of preeclampsia and gestational hypertension.

The eligible women consisted of a random sample of the women who delivered a singleton live birth in 1997-1999 in six regions of Quebec

and worked during pregnancy. Cases of preeclampsia (N=102) and gestational hypertension (N=99) were compared with normotensive controls (N=4381). Information on occupational exposures at the onset of pregnancy was collected during phone Interviews a few weeks after delivery. Detailed information was obtained on work schedule, postures, physical exertion, work organization, noise, Vibration, and extreme temperature. Adjusted odds ratios (aOR) were estimated through polytomous logistic regression.

Women standing daily at least l hour consecutively without walking experienced a higher risk of preeclampsia [aOR 2.5, 95% confidence interval (95% CI) 1.4-4.6], as well as women climbing stairs frequently (aOR 2.3, 95% CI l.2-4.l) and women working more than 5 consecutive days without a day-off (aOR 3.0, 95% CI l .0-9.5). Squatting or kneeling, pushing or pulling objects, whole-body vibration, forced pace, job strain, and no control on breaks were positively, but nonsignificantly, associated with preeclampsia. The associations were weaker for gestational hypertension

These findings suggest that being exposed to physically demanding and stressful occupational conditions at the onset of pregnancy increases the risk of preeclampsia.

Dr. Georg Wultsch
(Graz, Austria)

Occupational exposure to endocrine-disrupting compounds and biliary tract cancer among men

(Wolfgang Ahrens, Chinara Mambetova, Nicole Bourdon-Raverdy, Agustin Llopis-Gonzalez, Pascal Guenel, LennartHardell, Franco Merletti, Maria Morales-Sua-rez-Varela, Jörn Olsen, Häkan Olsson, Mogens Vyberg, Paola Zambon; Scand J Work Environ Health 2007; 33:387-96)

This study investigated the association between cancer of the extrahepatic biliary tract and exposure to endocrine-disrupting compounds.

Altogether 183 men with histologically confirmed carcinoma of the extrahepatic biliary tract and 1938 matched controls were interviewed between 1995 and 1997 in the frame of an international multicenter case-control study in six European countries (Denmark, France, Germany, Italy, Spain, and Sweden). Self-reported Job descriptions were converted to semiquantitative variables (intensity, probability, and duration of exposure) for 14 endocrine-disrupting compounds. The cases were compared with 1421 population controls and 517 colon adenocarcinoma patients. Odds ratios (OR) and 95% confidence intervals (95% CI) were obtained with unconditional logistic regression and adjusted for age, country, and gallstones.

Occupational exposure to endocrine-disrupting compounds resulted in an OR of l.4 (95% CI l.0-2.l) with no dose-effect relationship for cumuJative exposure (low: OR l.3, 95% CI 0.6-3.0; medium: OR l.5, 95% CI 0.8-2.7; high: OR 1.4, 95% CI 0.9-2.4) (only index participants). The elevated risk was restricted to extrahepatic bile ducts and ampulla Vateri (OR 1.7, 95% CI 1.0-2.6). The adjusted OR for cancer of the extrahepatic biliary tract after exposure to pofychlorinated biphenyls was 2.8 (95% CI 1.3-5.9, only index participants).

The data show some associations between exposure to endocrine-disrupting compounds in the workplace and the risk for cancer of the extrahepatic biliary tract among men, particularly for the extrahepatic bile duct and ampulla of Vater. Polychlorinated biphenyls could possibly be a strong risk factor.

Dr. Georg Wultsch
(Graz, Austria)

A long-standing controversy in environmental and occupational medicine regards the alleged carcinogenicity of formaldehyde in humans. In June 2004, IARC recommended that formaldehyde should be regarded as an IARC Group 1 carcinogen. At European level, the IARC recommendation triggered a review of formaldehyde's existing category 3

classification, the lowest available EU category for suspected carcinogens. In consideration of the mechanistic data the German MAK Commission assigned formaldehyde to Category 4 of carcinogenic substances, which means that under the conditions of the MAK value (currently at 0.3 ppm) "no significant contribution to human cancer risk is expected" at the workplace.
An overview over the current debate and the underlying scientific evidence (from an industry point of view!) is available at http://www.formaldehyde-europe.org/
However, this perspective appears not to be significantly different from the conclusions drawn by "independent researchers."

Carcinogenic potential of formaldehyde in occupational settings: a critical assessment and possible impact on occupational exposure levels.

(Duhayon S, Hoet P, Van Maele-Fabry G, Lison D; Int Arch Occup Environ Health 2008; 81:695-710)

OBJECTIVES: To review epidemiological studies which led to a change in the classification of formaldehyde by the International Agency for Research on Cancer (IARC) in 2004 as well as studies published thereafter, with the objective to examine whether occupational exposure levels for formaldehyde should be adapted.

METHOD: Cohort and case-control studies investigating the association between occupational exposure to formaldehyde and nasopharyngeal cancer (NPC) and reporting estimates of formaldehyde exposure as well as the most recent meta-analyses, published after 1994, were reviewed.

RESULTS: Evidence of an association between occupational formaldehyde exposure and NPC appears debatable. Results of the cohort studied by Hauptmann et al. (Am J Epidemiol 159 (12):1117-1130, 2004) were key findings in the IARC evaluation. In this study, mortality from NPC was elevated compared with that of the US general population. However, internal comparison analysis using alternative categorization revealed that none of the relative risk for NPC was statistically significantly increased in any category of exposure (Marsh and Youk in Regul Toxicol Pharmacol 42(3):275-283, 2005) and re-analyses of the data highlighted the inappropriateness of the exposure assessment used by Hauptmann et al. (Am J Epidemiol 159(12):1117-1130, 2004) and Marsh et al. (Regul Toxicol Pharmacol 47(1):59-67, 2007). Two other cohorts (Coggon et al. in J Natl Cancer Inst 95(21):1608-1615, 2003; Pinkerton et al. in Occup Environ Med 61(3)193-200, 2004) reported no increase in NPC. Two case-control studies brought some evidence of an increased risk of NPC but the assessment of exposure levels was uncertain. DISCUSSION: Human studies fail to raise a convincing conclusion concerning the carcinogenicity of formaldehyde and are not helpful to delineate a possible dose-response relationship. Experimental data indicate that in rats, the carcinogenic activity of formaldehyde is associated with cytotoxic/ proliferative mechanisms. Therefore protecting from these effects associated with formaldehyde exposure should be sufficient to protect from its potential carcinogenic effects, if any in humans.

CONCLUSION: Current occupational exposure levels to formaldehyde, set to protect against local irritation, should not be adapted.

Dr. Michael Nasterlack
(Ludwigshafen, Germany)

Even if it may not be a relevant human carcinogen, formaldehyde in sufficiently high concentrations is a classical example for a strong airway irritant - which leads us to another unresolved controversy: pro or contra inhalatory cortisone as a first treatment after inhalation trauma. Double-blind randomized trials have (to my knowledge) not been carried out in real-world conditions, and may never be available for a variety of reasons. We are thus depending on analogy,

personal experience, and sometimes even on belief in the absence of convincing evidence. The following study may add a little something to the "analogy" section (although I am fully aware that cortisone was administered before ozone exposure, and ozone is not necessarily representative for "any irritant").

Fluticasone propionate protects against ozone-induced airway inflammation and modified immune cell aktivation markers in healthy volunteers

(Alexis NE, Lay JC, Haczku A, Gong H, Linn W, Hazucha MJ, Harris B, Tal-Singer R, Peden DB; Environ Health Perspect 2008; 116:799-805)

Ozone exposure induces airway neutrophilia and modifies innate immune monocytic cell-surface phenotypes in healthy individuals. High-dose inhaled corticosteroids can reduce O_3-induced airway inflammation, but their effect on innate immune activation is unknown.

The authors used a human O_3 inhalation challenge model to examine the effectiveness of clinically relevant doses of inhaled corticosteroids on airway inflammation and markers of innate immune activation in healthy volunteers.

Seventeen O_3-responsive subjects [> 10% increase in the percentage of polymorphonuclear leukocytes (PMNs) in sputum, PMNs per milligram vs. baseline sputum] received placebo, or either a single therapeutic dose (0.5 mg) or a high dose (2 mg) of inhaled fluticasone proprionate (FP) 1 hr before a 3-hr O_3 challenge (0.25 ppm) on three separate occasions at least 2 weeks apart. Lung function, exhaled nitric oxide, sputum, and systemic biomarkers were assessed 1-5 hr after the O_3 challenge. To determine the effect of FP on cellular function, we assessed sputum cells from seven subjects by flow cytometry for cell-surface marker activation.

FP had no effect on O_3-induced lung function decline. Compared with placebo, 0.5 mg and 2 mg FP reduced O_3-induced sputum neutrophilia by 18% and 35%, respectively. A similar effect was observed on the airway-specific serum biomarker Clara cell protein 16 (CCP16). Furthermore, FP pretreatment significantly reduced O_3-induced modification of CD11b, mCD14, CD64, CD16, HLA-DR, and CD86 on sputum monocytes in a dose-dependent manner.

This study confirmed and extended data demonstrating the protective effect of FP against O_3-induced airway inflammation and immune cell activation.

Dr. Michael Nasterlack
(Ludwigshafen, Germany)

Media Rips Carbon Nanotubes

There have been a number of articles published since May 20 regarding a possible link between carbon nanotubes and the development of precursors of mesothelioma resulting from a recent letter published in Nature Nanotechnology.

C. Poland, et al., "Carbon nanotubes introduced into the abdominal cavity of mice show asbestos-like pathology in a pilot study," NATURE NANOTECHNOLOGY, May 20, 2008.

The letter's authors related the results of an in vivo study in which they injected various types of carbon nanotubes into the mesothelial abdominal lining of mice. The study was driven, in part, because of prior speculation regarding an outward resemblance between certain carbon nanotubes and asbestos fibers, as well as prior studies showing possible adverse EHS effects from exposure to certain types of carbon nanoparticles under laboratory conditions. While not actually causing mesothelioma, the scientists "observed that long MWCNTs produced inflammation FBGCs and granulomas similar to the foreign body inflammatory response caused by long asbestos fibres." Of course, the mice did not actually inhale carbon nanotubes (of any size) in the experiment, nor did the nanotubes end up in the chest cavity. The researchers further

concluded that the "study does not address whether CNTs would be able to reach the mesothelium in sufficient numbers to cause meso-thelioma following inhalation exposure." To those judging whether media coverage of the issue has been "fair and balanced," below are some of the more notable articles we have come across since the Poland study was published: "Are Nanotubes the Next Asbestos?;" "CANCER; Carbon Nanotubes That Look Like Asbestos, Behave Like Asbestos;" "Cancer concerns over carbon nanotubes;" "Cancer risk seen in nanotechnology;" "Tiny cylinders used in some products act like asbestos, a study finds;" "Carbon nanotube has similar effects to asbestos;" "Carbon nanotubes as bad as asbestos, says study;" "Carbon nanotubes behave like asbestos, study shows;" "Carbon Nanotubes Could Pose Health Risks Akin to Asbestos;" "Carbon nanotubes, key ingredient in nanotechnology work, mimic asbestos in mouse tests;" "Carbon nanotubes may be as hazardous to health as asbestos;" "Carbon nanotubes mimic asbestos in early study;" "Carbon nanotubes that look like asbestos just as cancerous;" "Comparison of Nanotubes to Asbestos Spurs Call for EPA, Hill Action;" "Effects of Nanotubes May Lead to Cancer, Study Says;" "Fears over wonder nanotubes;" "Health threat of nanotubes may be similar to asbestos, study warns;" "Hi-Tech Fibres Scare;" "How safe are nanoparticles?;" "In Study, Researchers Find Nanotubes May Pose Health Risks;" "Nano-fibres lead to pre-cancer symptoms in mice;" "Nanofibres linked to cancer;" "Nanotech could cause mesothelioma;" "Nanotubes could cause lung disease like asbestos;" "Nanotubes, Like Asbestos, Could Threaten Health;" "Nanotubes may cause cancer hazard;" "Nanotubes may pose risk that asbestos does, study reports;" "New cancer alert;" "New technology may be as bad as asbestos;" "Some nanotubes as dangerous as asbestos;" "Some nanotubes could cause cancer threat – study;" "Study Comparing Nanotubes, Asbestos Prompts Call for EPA Action;" "Study Finds Certain Nanotubes Could Be as Dangerous as Asbestos;" "Study links nanotubes to possible lung illness;" "Study: 'Nanotubes' Pose Same Danger as Asbestos;" "Study Seen Impacting Expected Cal/EPA Nanotechnology Bill;" "Study Waves Cautionary Flag About Nanotubes;" and "The microparticles that could pose the same risk as asbestos."

(Taken from
John C. Monica,
Michael E. Heintz,
Nanotechnology Law Report,
June 2008)

Welcome to new members

Lin, Chun-Chi, M.D., Division of clinical toxicology, Taipei Veterans General Hospital (Taiwan)

Dr. **Stephanie Reutemann**, Beiersdorf AG, Hamburg (Germany)

Forthcoming Events

The 36th Medichem Congress **Innovation in Occupational Health** will take place Sept. 10th to 11th in Amsterdam.

Preceding the congress, on 8th and 9th September 2008 an Advanced Hazmat Life Support (AHLS) Provider course will be held. This is a 16 hour, two-day course that gives health professionals a timely and effective response strategy in the medical management of hazmat incidents. Participants will receive a four-year verification status upon successful completion of the course.

Find more information and the registration form at http://www.medichem.org/medichem2008.