of the AhR gene battery. In contrast, incubation of the cells with isoeugenol increased CYP1B1 and AhR mRNA levels in keratinocytes. Our results indicate that keratinocytes can activate small haptens such as fragrances. This work was supported by the BMBF (IZKF BIOMAT, G11) and the European Community.

383 ELUCIDATING CHANGES IN SURFACE MARKER EXPRESSION OF DENDRITIC CELLS FOLLOWING ALLERGEN TREATMENT.

B. C. Hulette, C. A. Ryan and G. F. Gerberick. *Procter & Gamble, Cincinnati, OH.*

Dendritic cells (DC) are specialized antigen-presenting cells (APC) located in many non-lymphoid tissues, and Langerhans cells (LC), a specialized form of DC, are found in the skin. LC play a critical role in the induction of contact dermatitis and therefore have become a focal point for development of *in vitro* cell-based methods for contact sensitization testing. Because of the low abundance of skin-derived LC, cultured DC from peripheral blood (PB) are used as LC surrogates to study the effects of sensitizing chemicals on APC. It has been reported that chemical allergens induce changes in the expression of various DC surface markers. For the work presented here, DC were derived from the KG-1 cell line (KG-1 DC) or were differentiated from human PB (PBMC-DC). The phenotype of these DC was measured by flow cytometry after 48 hr treatment with the chemical allergens dinitrofluorobenzene (DNFB) and methylchloroisothiazolinone/methylisothiazolinone (MCI/MI), the irritant sodium dodecyl sulfate (SDS), lipopolysaccharide (LPS), and tumor necrosis factor-α (TNFα). It was found that only MCI/MI upregulated CD86 expression in KG-1 DC, whereas TNFα upregulated CD54 and slightly upregulated CD80 and CD86 expression. In PBMC-DC, MCI/MI and DNFB induced a slight upregulation of class II major histocompatibility expression (HLA-DR), whereas LPS and TNFα significantly upregulated CD54 and slightly upregulated CD80 and HLA-DR expression. SDS had no effect on surface marker expression in either KG-1 DC or PBMC-DC. Changes in surface marker expression in PBMC-DC treated with chemical allergens were detected in 2 of 5 donors, suggesting a limited sensitivity of PBMC-DC under these culture conditions. Futhermore, the presence of cytokines during allergen treatment masked the ability to detect changes in surface marker expression. Our data suggest that, under these culture and treatment conditions, measurement of surface marker changes using PBMC-DC or KG-1 DC does not provide a sensitive method with sufficient dynamic range for assessing the contact sensitization potential of a chemical.

384 DIVERGENT IMMUNOLOGICAL RESPONSES FOLLOWING GLUTARALDEHYDE EXPOSURE.

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Glutaraldehyde (Glut) is a potent contact sensitizer, additionally numerous cases of occupational asthma related to Glut exposure have been reported in recent years. The purpose of this study was to examine the Dose-Response relationship between Glut exposure and the development of Th1 *vs.* Th2 responses using a murine model. Initial evaluation of the sensitization potential was conducted using the standard Local Lymph Node Assay in CBA mice. Female BALB/c mice (N=5-8 per group) were used for all other studies. The IgE inducing potential was evaluated by phenotypic analysis of IgE+B220+ cells in lymph nodes draining the site of topical exposure and measurement of total serum IgE levels. The Mouse Ear Swelling Test (MEST) was used to evaluate the potential of Glut to elicit urticaria (30 min post challenge) and contact hypersensitivity (24 and 48 hrs post challenge). A dose dependent increase in lymphocyte proliferation as measured by 3H-thymidine incorporation and the %B220+ cells was observed following exposure to 0.1, 0.75, and 2.5% Glut with a positive response (SI>3 in LLNA) being observed at all concentrations tested. The MEST demonstrated an immediate response (Th2) in animals induced and challenged with 2.5% Glut, whereas animals induced with 0.1 or 0.75% and challenged with 2.5% exhibited a delayed response (Th1) at 48 hrs. post challenge. In agreement with the MEST results, only the 2.5% dose group had a significant increase ($p<0.01$) in serum IgE levels and % IgE+B220+ draining lymph nodes cells. The results of these studies indicate that the induction of a Th1 *vs.* Th2 response following dermal exposure to Glut is at least in part mediated by the exposure concentration. These studies were supported in part by NIEHS intra-agency agreement #Y1-ES-0001-01.

385 RANKING OF ALLERGENIC POTENCY OF LATEX CHEMICALS IN A MODIFIED LOCAL LYMPH NODE ASSAY.

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A modified local lymph node assay (LLNA) with *ex vivo* tritium thymidine (3H-TdR) labelling of the proliferating lymph node cells was used for determination of the allergenic potency of chemicals used in the production of latex medical gloves. Fifteen chemicals, known to induce allergic contact dermatitis in man, including various thiuram, carbamate, and benzothiazole compounds and one amine were tested. The EC3 (effective concentration inducing a threefold increase in proliferation of lymph node cells (Stimulation Index, SI=3)) was calculated with non-linear regression analysis, including a bootstrap method for determination of the 5%-95% confidence interval of the EC3 value. The compounds tested were thiurams: TMTM (tetramethylthiurammonosulfide), TMTD (tetramethylthiuramdisulfide), TETD (tetraethylthiuramdisulfide), TBTD (tetrabutylthiuramdisulfide), PTD (dipentamethylenethiuramdisulfide), and PTT (dipentamethylenethiuramtetrasulfide); carbamates: ZDMC (zinc dimethyldithiocarbamate), ZDEC (zinc diethyldithiocarbamate), ZDBC (zinc dibutyldithiocarbamate), ZPC (zinc pentamethylenedithiocarbamate); benzothiazoles: MBT (2-mercaptobenzothiazole), MBTS (2, 2-dibenzothiazyldisulfide), ZMBT (zinc mercaptobenzothiazole), and MBI (mercaptobenzimidazole); and DEA (diethylamine). This procedure identified 14 out of the 15 chemicals tested as sensitiser, while for one chemical, ZDBC, no EC3 could be calculated due to low responses and a lack of a Dose-Response relationship in the data obtained. The ranking order of the chemicals with increasing EC3 values (and thus decreasing allergenic potency) was found to be: ZDEC, TMTD, TETD, ZPC, ZDMC, MBTS, PTD, TMTM, MBT, MBI, PTT, ZMBT, TBTD, DEA, and ZDBC. Our results indicate that the chemicals of choice for use in latex production would be for the thiuram compounds TBTD, for the carbamates ZDBC and for the benzothiazoles ZMBT. However, one has to be aware that besides potency also the total amount of residual chemical present in the final product is important for allergy induction.

386 STUDY TO DETERMINE THE PRESENCE OF ANTIPOLYMER ANTIBODIES IN A GROUP OF DUTCH WOMEN WITH A SILICONE BREAST IMPLANT (SBI).

W. H. De Jong, M. Kallewaard, C. A. Goldhoorn, J. W. Bijlsma, J. S. Schouten and H. Van Loveren. *Immunobiology and Haematology, RIVM, Bilthoven, Netherlands.*

Recently the presence of antipolymer antibodies (APA) has been reported to be correlated with the disease status in silicone breast implant (SBI) recipients (and fibromyalgia patients. A cross sectional study was conducted in a population of Dutch women with SBI and self reported severe complaints. SBI recipients (n=42) were clinically examined and blood samples were obtained. In the APA assay the binding of immunoglobulins to acrylamide polymer-coated strip (2) was demonstated after incubation with secondary antibodies and visualization by a colorimetric method. In 12 of 42 SBI recipients an increase in the level of polymer binding immunoglobulins was detected compared to a negative reference sample, 3 of these 12 showing a positive and 9 a weakly positive response. In 3 out of 12 non-SBI recipients, included for control on the performance of the APA assay, an increased level of polymer binding immunoglobulins was demonstrated, 2 of these 3 showing a positive and 1 a weakly positive response. In the general population in 5 out of 80 women a positive APA was found, and one of 80 showed a weakly positive response. The study population of SBI recipients was categorised in severity subgroups (limited, mild, moderate, advanced) based on the functional capacity and the physicians general assessment of pain and disease activity. Most (34 of 42) SBI recipients belonged to the limited severity subgroup. In conclusion, we did not find a high prevalence of polymer binding immunoglobulins (APA) in a group of Dutch SBI recipients with self reported severe complaints. A discrepancy was present between the self-reported severe complaints and the observed mild clinical symptoms. SBI exposure (mean 17 years) did not result in induction of polymer binding immunoglobulins in this minimal symptomatic study group. Antigen strips for the APA assay were kindly provided by Dr R.B. Wilson, Autoimmune Technologies, L.C.C., New Orleans, LA, USA.

387 EXPOSURE TO SINGLE OR TERTIARY MIXTURES OF N, N-DIETHYL-M-TOLUAMIDE (DEET), PYRIDOSTIGMINE BROMIDE (PYR), JP-8 JET FUEL AND STRESS IN AUTOIMMUNE PRONE MICE.

G. S. Gilkeson, M. M. Peden-Adams, J. G. EuDaly, S. Dabra, A. EuDaly, R. Hayes, L. Heesemann, J. Miller, J. Smyth and D. E. Keil. *Medical University of SC, Charleston, SC.*

Exposures to chemicals such as DEET, PYR, and JP-8 jet fuel (JP-8) are common in military environments, as well as in some occupations. During the Gulf War, military service personnel were exposed to PYR, DEET, JP-8, and stress. Previous studies determined the lowest adverse effect level of immunological parameters to be 15.5 mg/kg DEET, 2 mg/kg PYR, 500 mg/kg JP-8, and 20 minutes exercise stress for 14 days. To investigate the contribution of these agents in accelerating or exasperating autoimmune disease, a strain of autoimmune prone mice, MRL lpr/lpr, were exposed for 14 days at age 12 weeks. At 18 weeks of age, immunological parameters in mice were assessed. No remarkable changes occurred in complete

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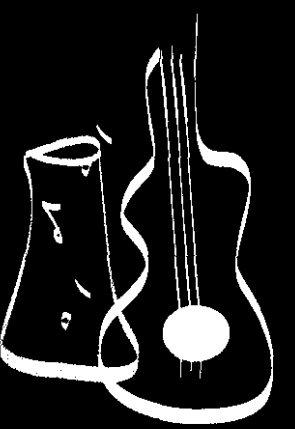

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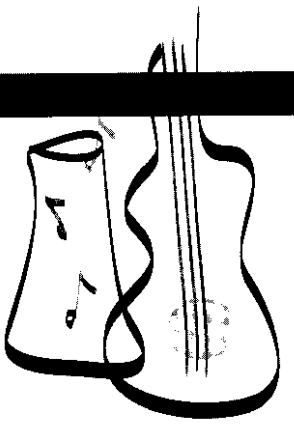

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Preface

This issue of *The Toxicologist* is devoted to the abstracts of the presentations for the symposium, platform, poster discussion, workshop, roundtable, and poster sessions of the 41st Annual Meeting of the Society of Toxicology, held at the Opryland Hotel and Convention Center, Nashville, Tennessee, March 17–21, 2002.

An alphabetical Author Index, cross referencing the corresponding abstract number(s), begins on page 385.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 411.

The abstracts are reproduced as accepted by the Program Committee of the Society of Toxicology and appear in numerical sequence.

Additional Late-Breaking Abstracts are issued in a supplement to this publication and are available at the 41st Annual Meeting and through the Society of Toxicology Headquarters office.

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