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Tagungsband - Abstract

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Coautoren:

Titel des Abstracts:
Edelmetalle/Übergangsmetalle und Umwelt: Kfz-Katalysator verantwortlich für Erbschä

Fachgebiet: Biochemische/Klinische Analytik

Arbeitsgebiet: Med.Biochemie und Molekularbiologie

Abstract:

Edelmetalle/Übergangsmetalle und Umwelt:

Kfz-Katalysator verantwortlich für Erbschäden und Ozonloch?

Reinhold Kiehl

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Abstrakt

Hauterkrankungen, Atemwegserkrankungen und vor allem Asthma, Erbschäden sowie K
den westlichen Ballungsgebieten - ab 1990 auch überproportional in den Ostblock-Staate
(1-6).
Verantwortlich dafür sind die in unsere Atmosphäre geblasenen feinstverteilten Metalle,
- der meist signifikante atmosphärische "asthmatische" KFZ-Umwelt-Ausstoß Platin, Pal
und aromatischen Kohlenwasserstoffe sowie Rußpartikel (9-11).

Gewarnt wurde vor 15 bis 20 Jahren schon von Experten mit Ergebnissen aus entsprechen
gehört hätte. So schneit es in Grönland seit Einführung des Katalysators bis zu 120 mal h
den Platingruppen-Metallen in größtem Ausmaß kontaminiert, neben der schon bestehen
Ein PKW mit Katalysator verliert im Schnitt 1,5 Mikrogramm feinst verteiltes Platin/km
wie München werden pro Tag ein Gramm Platin freigesetzt oder ca. ein halbes Kilo/Jahr
Die direkte Einatmung durch Fußgänger sowie Schwangere und Kleinkinder erklärt
die Erkrankungsraten. Diese Tatsache ändert sich auch nicht mittels Verharmlosung dure
Defekte geben unsere Gentechniker nun vor, ein stark wachsendes neues Spielfeld mit ne
Die Hydroxid-Radikal-Konzentration in der südlichen wie nördlichen Hemisphäre
variieren signifikant seit zwei Dekaden. Das Hydroxyl-Radikal ist die dominante
oxidierende Chemikalie in der Atmosphäre und damit direkt involviert in den Ozon-Haus

Radikal-Reaktionen in dieser "Metall-Sauerstoff-Stickstoff-Kohlenstoff-Wassersuppe" si
Metalle und damit der KFZ-Verkehr sind für steigende Erkrankungsraten, Ozonloch, Tre
Kohlendioxid mit seinem Anstieg (19,20,21).

Die Konsequenz dürfte damit klar sein: schnellste Reduktion der Metalle aus unserer A

Atmosphäre erholt sich – so wie sie es schon einmal einige Millionen Jahre vorher zur Ze

Abstract

Skin diseases, diseases of the respiratory tract and above all asthma, genetic defects as well as canc converter (cat)cars – since 1990 very heavely in the east-Bloc states:fall of the wall and exchange o

Responsible for this fact are in the air finest distributed metals, like mercury/mercurials, cadmium, atmospheric asthmatic pollution platinum [5], palladium, rhodium (automobile exhaust) with or with

There came already warnings about 15 to 20 years ago by american experts, who carried out approp since the use of catalytic converters, it is snowing in Greenland over 120 times higher concentratio

The western hemisphere is assumed to be contaminated in alarming proportions in the near future v fossil fuel.

A cat car loses on average 1.5 microgram finest distributed (colloidal) platinum (plus palladium an like Munich about 300 to 400 gram per year including hydrocarbons and root particles [14-16].

The direct inhalation by pedestrians/infants and pregnant woman explains the increasing disease ra The mechanisms acting are the affinity of nickel for nitrogen and not for sulfur. Colloidal platinum additive reactions, oxidations, hydrogenations. Platinum (Cis-Platinum) inhibits/stimulates prolifer a heavely expanding new field with new market: We are changing the evolution in high speed.

There is evidence for substantial variations of the hydroxyl radical (OH) concentrations in the Sout oxidizing chemical in the atmosphere and is therefore directly involved in the ozone depletion and

Radical reactions in that "metal-oxygen-nitrogen-carbon-water soup" are responsible for the ozone rising disease rates, ozone gap, greenhouse effect, climate change and forest dieback "the" responsi

The consequence should therefore be clear: fastest reduction of the metals out of our atmosphere (b like it did some millions of years ago at the time of the saurien dead.

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Precious metals/transition metals and environment: Catalytic converter responsible IPRAC, April 1 to 4, 2001, Praque, Czech Republic, Allergie, Abstrakta, Roenik 3, Supp

Atopy/Contact Dermatitis/Asthma:Metal Ions/Cd;Hg;Ni;Pt;Pd

Key words: g-interferon, cytokines, electron transfer chain, redox potentials, metals, dise

Abbreviations: IL=interleukin, IFN=interferon, APMSF=(4-amidinophenyl)-methanesulfor azodicarboxylic acid bis (dimethylamide), DMPS = dimercaptopropan-sulfonate.

Mercury, metalloproteases, IgE-level, inflammation and allergic manifestations

In search for an assay system more closely related to the in vivo conditions of atopic eczema patients, we decided to directly investigate the blood samples of these patients. During our first attempt we titrated the blood samples with activators and inhibitors of proteases, since some of these compounds were thought to be involved in triggering atopic eczema. Particularly the metalloprotease activator mercury should have been able, in our opinion, to influence gIFN-levels by activation of metalloproteases for degradation of this important regulatory factor. Mercury has been suspected for decades now of triggering allergic manifestations via the immune system.

The effect of mercury on IgE-levels was seen at concentration ranges of 0,5 to 1 mM; concentrations which are about 1 mio times higher than the normal range in blood of control or atopic eczema persons. Mobilization of mercury by DMPS results in 10^2 to 10^3 times higher values in these persons, which is still about 10^4 to 10^5 times lower than our measured effective concentrations of Hg on IgE-levels. Nevertheless, someone describes immune changes (in the lymphocyte-subpopulations) induced in their opinion by mercury mobilization. However, these changes, especially in patients with allergic diseases, were not verified. In anothe injected into rats, which corresponds to about 5 mg/l blood (a concentration near our de toxic mercury concentrations seem not to be responsible for the changes in IgE-levels in

Matrix metalloproteinases (collagenase, gelatinase, stromelysin) are highly glycosylated activity, and are therefore inhibited by chelating agents (like EDTA) and have the ability tissue cells such as fibroblasts and from neutrophils as inactive proenzymes, and can different mercurial compounds, or reactive oxygen species (ROS). They are also inhibite their secretion is suppressed by immunosuppressive drugs, like glucocorticoids .

Activation of isolated metalloproteases requires μM concentrations of mercurials; 10 approximately 4 hrs. These conditions were obtained in our patients after mercury mob However, under normal conditions the circulating protease and lactoferrin concentratior been done by ELISA. ELISA measures only protein concentrations. In blood samples of and the anticoagulant heparin from reaction with substrate or binding to antibodies (fo EDTA plasma. α^2- macroglobulin is inactive and residual heparin and/or TIMP prot concentrations (collagenase ca. 90ng/ml gelatinase ca. 600 ng/ml, and lactoferrin ca. 300 (coagulated blood), α^2- macroglobulin is inactive, heparin missing and therefore almos concentrations (and activities) of the proteases (and of lactoferrin) were then obtained in

The few measurements with capillary blood samples (collected under heparin protection activation processes exist. The few heparin molecules, possibly in here available, metalloproteases for binding to antibodies during ELISA (competition). On this ground,

We could show (1) that circulating immune complexes and IgE in the patients blood acti with further enhancement of aggregation (thrombosis). This process could be related to process starts with rising IgE concentrations in the circulating blood or affected skin aggregation results presumably in a changed energy metabolism in these particles with with elevation of histamine, inactivation of α_2-macroglobulin and activation of metallo cells to UV-light and responsible for development of skin carcinomas.. Nitric acid (abnormalities of the L-arginine: NO pathway could contribute to the pathophysiology of

gIFN-molecules were significantly degraded by metalloproteases (at least by activated l such behavior. However, one should keep in mind that the concentrations of the circula Degradation of two plasma components, namely C1-inhibitor and α_1-proteinase metalloprotease regulation is evident, and the impact of the changing active gIFN-conc point, we compared total IgE measurements using samples of circulating blood with sk either (2).

The standardized titration of blood samples from different patients (IgE ca. 1000 U/ml)
their IgE-values, suggesting involvement of a redox reaction in the Hg-IgE-interaction; I
EDTA elevates IgE-levels (at least in the experiments where Hg^{2+} induces positive v
metalloprotease on IgE concentrations, however the results of the Hg^{2+} - titrations are in

The serine protease inhibitor APMSF itself has no effect on IgE-level, which means tha
proteases are not involved in our measured metalloprotease activities.

APMSF probably interacts with serine residues after they are liberated by EDTA treatn
involvement of external Mg^{2+} - or Ca^{2+}- sensitive serine residues in the signal tranductio

The detergent Triton X- 100 (and probably other detergents too) drastically lowers IgE-l
The IgE-level was also reduced by cycloheximide, a protein synthesis inhibitor, which sl
measured. A similar result, although during days of growing, has been obtained in cell cu

Patients' IgE-level in the circulating blood system is regulated by degradation and (re)
regulated by various factors, including interleukins and gIFN. We were able to demonstr
the O_2^- -build- up in neutrophils) in our simple assay system, although the background le
well as synthesis rates of the patients' steady state IgE-level, by doing a few assays.

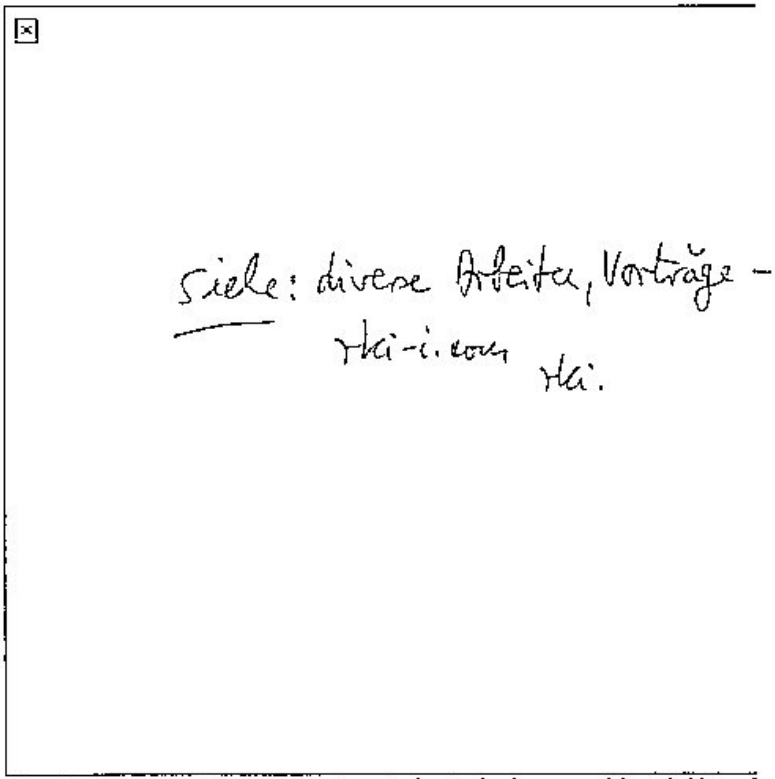
Furthermore, and even more importantly, the results obtained with Hg^{2+} indicate the invc

Involvement of a redox/thiol-disulfide interchange mechanism in the regulation of I

This dithiol/disulfide redox state is sensitive to Hg^{2+}, Diamide, gIFN and IL- 4, ht
of the involved protein, etf (2) in such a way that two associated thiols become vi
the effective concentration of gIFN by a factor of 10^2 to 10^3 or more . IL-4 reac
directions and at different time scales (minutes vs days) . The interaction of conformatior
varying IgE values in the blood samples of these patients on addition of Hg^{2+}. The descri

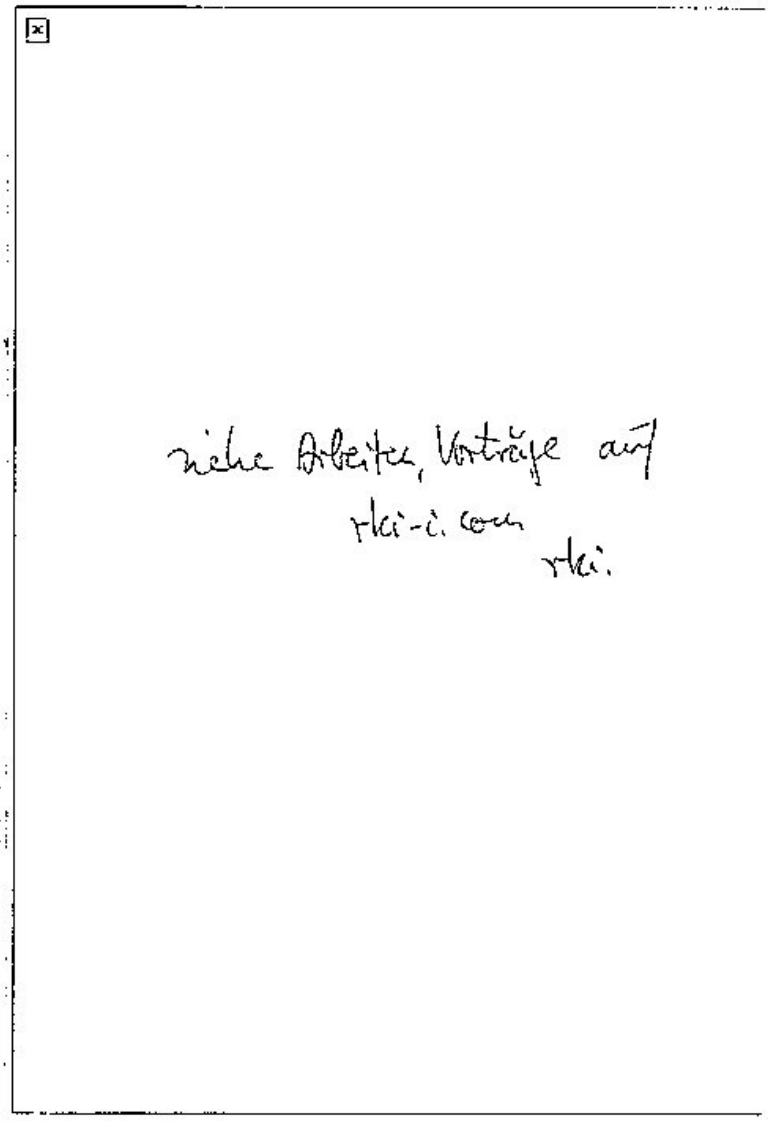
Our ineffective titrations of the redox state, indicated by the various IgE levels, with
clearly that the thiol groups involved were located inside the involved B cells: glutathior
then, of course, not able to replace Hg^{2+} or Diamide in the described dithiol/disulfide in
the cells , most likely with a dithiol-containing protein localized, at least for some time, o
the described mechanism require the involvement of a membrane-bound protein. NEI
cellular glutathione concentrations were too high (up to 10 mM) to be involved in the
protein. The different results obtained when using blood samples or cell culrures may be
experiments, in contrast to cell cultures which were grown in artificial systems using mit

Elektron transfer chain, NADPH to DNA



Siele: diverse Arbeiten, Vorträge –
Hai-i. com Hai.

The redox signal of gIFN for O_2 or IgE production (activation process) is probably med
subunit via G-protein (Rac-2). This system, thus, very much resembles the receptor-li
NADPH to DNA (IgE) (2). The defect in NADPH to DNA (IgE) at atopic eczema patie
redox regulation of IgE synthesis is difficult to answer. The mechanism of signal tr
gIFNmRNA and gIFN production in mitogen activated T-culture cells by IL-4 takes
antiproliferative effects (2). During short time intervals, IL-4 transduces opposite to gIFN
of etf/ref (via G protein?) may be responsible for this behavior . The down regulation
prostaglandin E2 and superoxide production by IL-4 implies the possibility that IL-4 may



siehe Arbeiten, Vorträge auf
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Extremely high serum IgE levels exist in patients with the so called hyper IgE syndrom chain should be in the full reduced form. The defect in NADPH to IgE for electron-tra eczema patients. All the factors regulating NADPH oxidase also, of course, influence th phosphorylation and dephosphorylation of the involved proteins by kinases (e.g. PkC oxidative stress conditions .

The redox potential is responsible for stress protein IgE or 0_2-synthesis and prolifer

The adaption of cells to oxidative stress , to heat shock, to environmental stress, etc. is no

The general scheme of activation of this defense mechanism seems to be the use of stim (TNF) and IL-1 control NADPH oxidase (nonphagocytes), TNF and IL-1 control collag oxidase (O_2 production) occurs simultaneously to the expression of former enzymes.

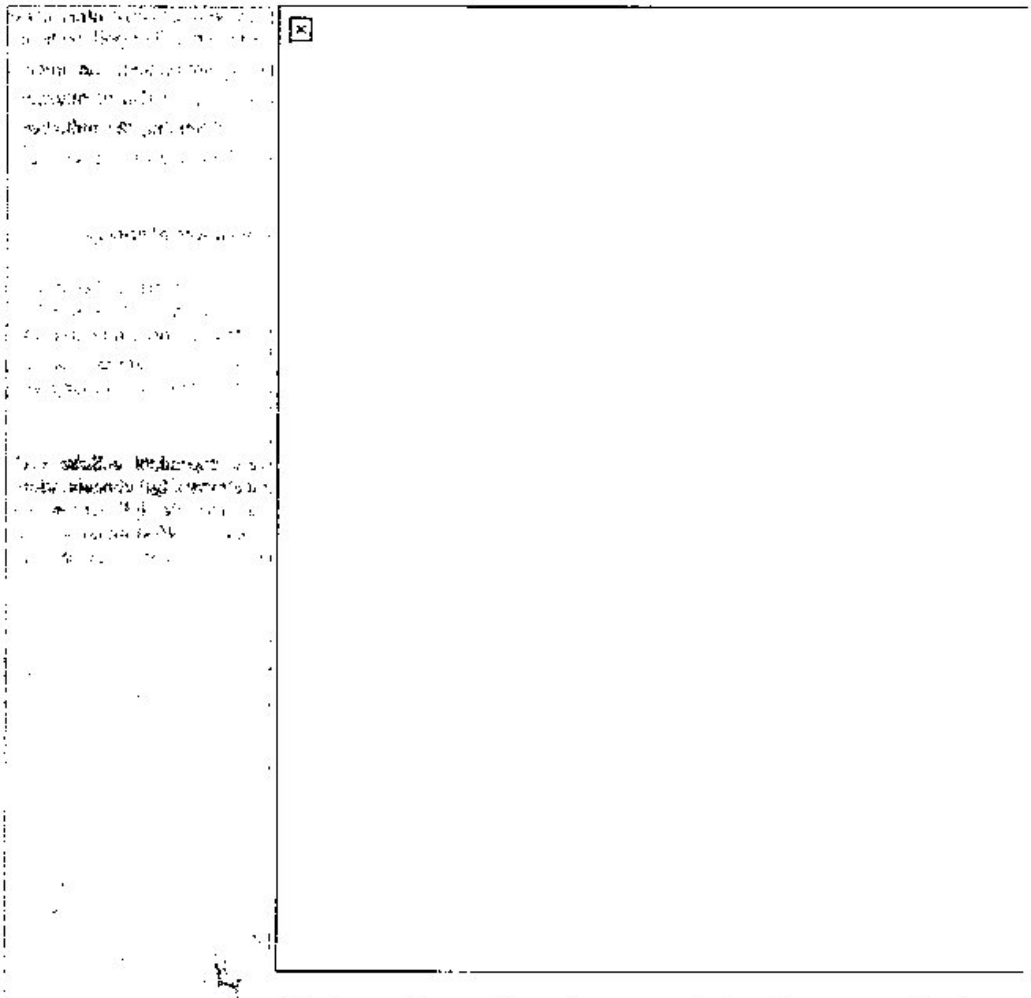
In the case of IgE synthesis (and probably also in the expression of some other comp irreversibly with the involved essential dithiol / disulfide redox state. The pollutants incl the electron transfer chain in the reduced form (low or no O_2 production) and, under s oxidized form is not able to synthesize IgE but instead O_2, and the risk of mitogen s compound, CO (and NO), binds to the NADPH oxidase , preventing the reduction c accompanied by the enhanced probability of IgE synthesis. Depending on concentratio human studies .

Mitochondrial oxidative phosphorylation serves as sole producer of energy

B-cells have a considerable need for energy. Their proliferation, synthesis and excretion c fuel is glutamine instead of glucose . Thus, it is not surprising that the process of NADPI energy formation . All the compounds influencing mitochondrial energy formation (2) th Dermal and intestinal dysbiosis, food, as well as psychogenic stress (2) are the main trig C. albicans, play a definite role in inducing allergic reactions in patients . Carbohydrate response by changing the energy metabolism of lymphocytes .

Psychogenic stress elevates norepinephrine levels, lowers dependent cellular cAM prostaglandin, leukotriene, cytokine concentration, etc.) and elevates IgE concentration.' allergens. It should be stressed that the total (unspecific plus specific) IgE concentratio gIFN independent IgE production by cultured cells on IL-4 and CD 40 stimulation is reli and resembles autoimmune diseases. The described pathogenesis of atopic eczema and le

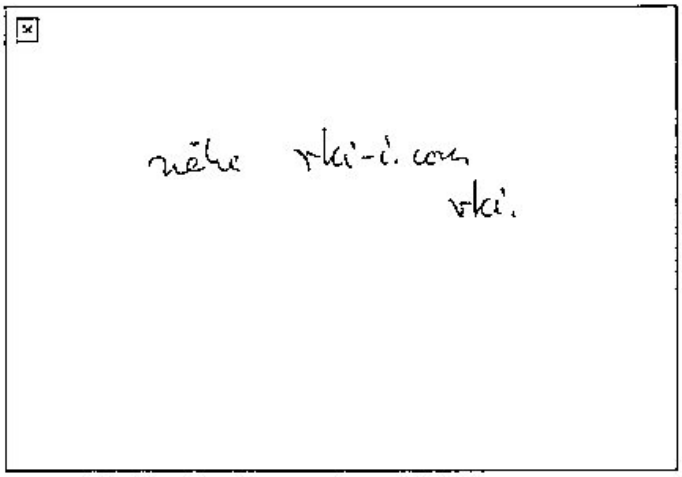
Ni, Pt; Pd: Contact dermatitis/ Asthma



Skin diseases, diseases of the respiratory tract and above all asthma, genetic defects as since 1990 very heavely in the east-Bloc states/ fall of the wall and exchange of old cars

The most commen occupational as well as public contact allergen Ni (8) and the mo responsible for this fact (6,7,9): In the air finest distributed Pt at a metropolis like Munich

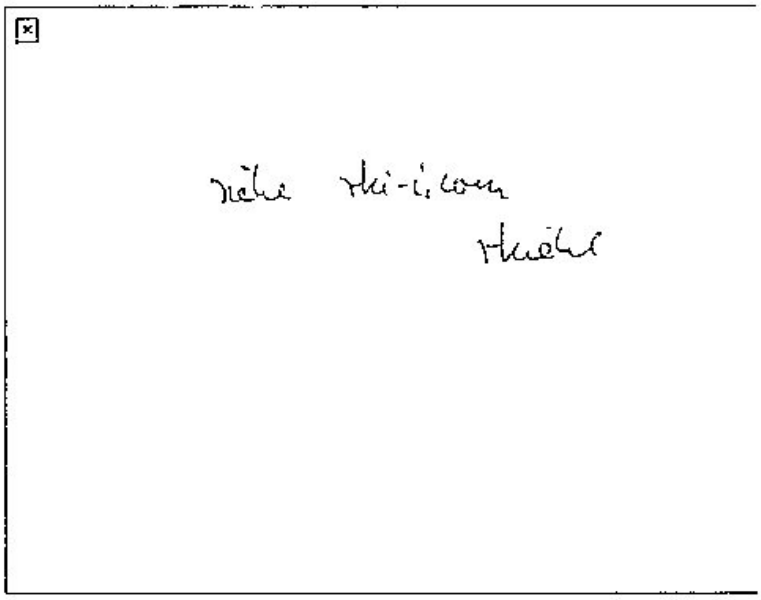
plus haevy pollutions of aliphatic and aromatic hydrocarbons (10), root particles (10-12) acting: Ni does not bind to sulfur but to N. Colloidal Pt(Pd) and Ni for instance hav hydrogenations. Pt (Cis-Pt) inhibits/ stimulates proliferations/ IgE-synthesis.

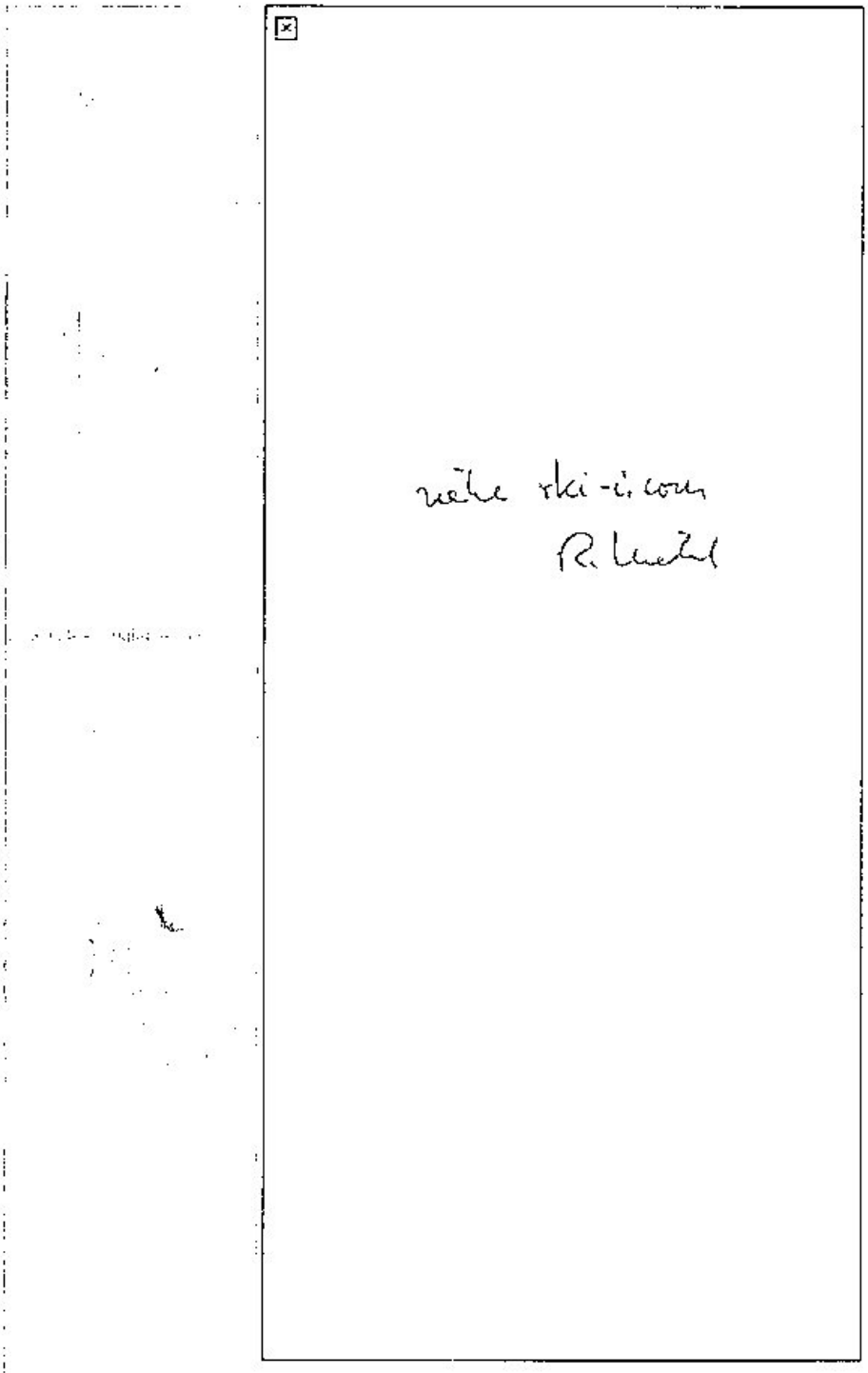


Direct labeling without enzymes

Using Nucleic Acid Labeling Kits:

A derivative of cis-platin reacts with the N-7 position of guanine residues, providing a stable linka
Nucleic Acid Labeling Kits).





A summation of the causes for multifactorial diseases, e.g. allergies, respiratory tract dise

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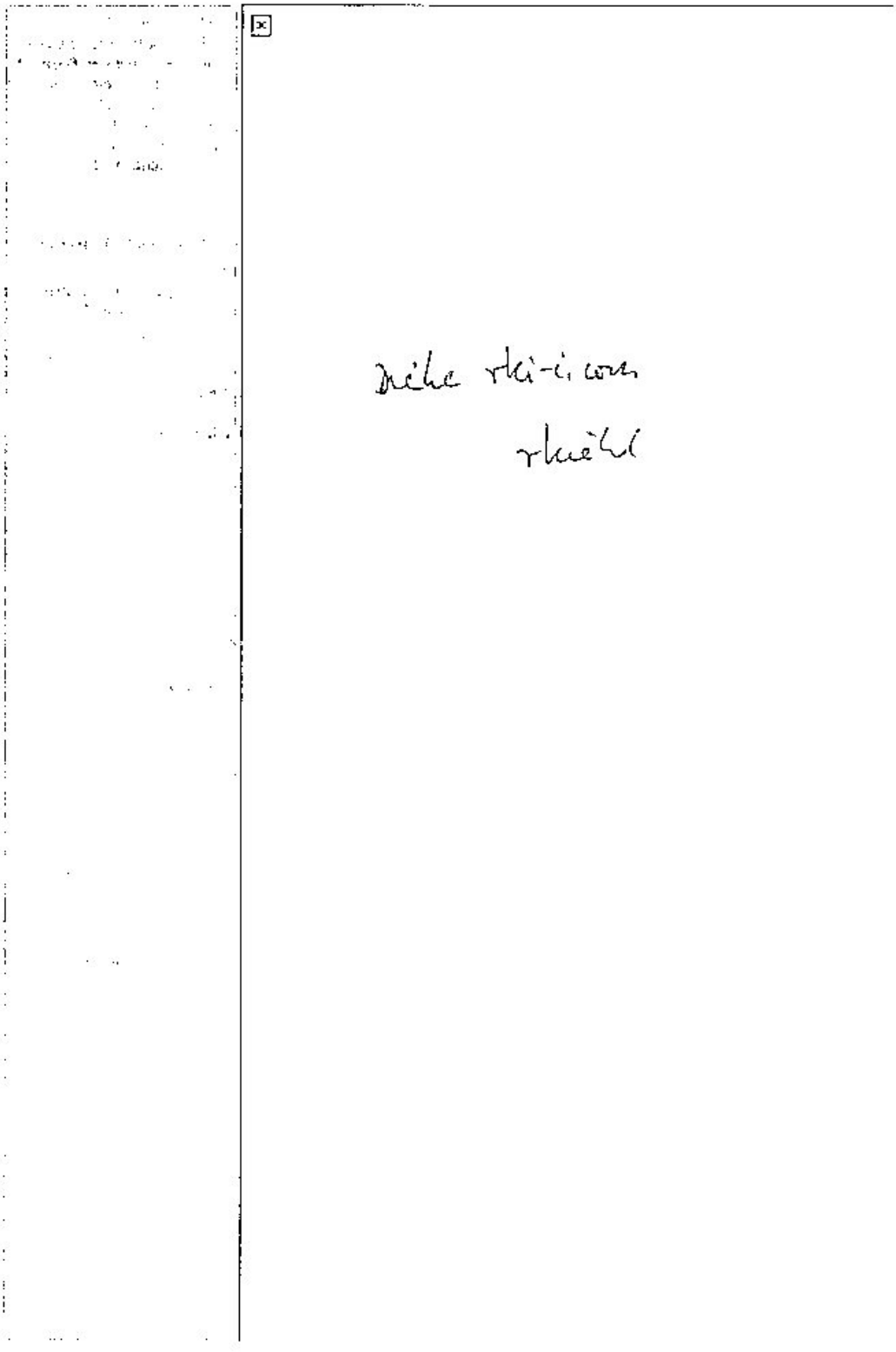

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R.Kichl, Buch, Literatur und Dateien unter http://www.rki-i.com/

H.Marquardt, S.G.Schäfer, Lehrbuch der Toxikologie

Ändern?

EAACI – München 2005

Catalytic converter responsible for genetic defects and ozone hole?

Kiehl,R., RKI-Institute, Saliterweg 1, D-93437 Furth i.W., ,

Skin diseases, diseases of the respiratory tract and above all asthma, genetic defects as well as cancer are rapidly increasing in the western conurbations with the use of catalytic converter (cat)cars – since 1990 very heavely in the east-Bloc states:fall of the wall and exchange of old cars without cat against cat cars.

Responsible for this fact are in the air finest distributed metals, like mercury/mercurials, cadmium, the most commen occupational as well as public contact allergen nickel and the most significant atmospheric asthmatic pollution platinum, palladium, rhodium (automobile exhaust) with or without heavy pollutions of aliphatic and aromatic hydrocarbons , as well as root particles. Since the use of catalytic converters, it is snowing in Greenland over 120 times higher concentrations of platinum and rhodium.

The western hemisphere is assumed to be contaminated in alarming proportions in the near future with the platinum group metals, beside the already existing contamination with mercury out of fossil fuel.

A cat car loses on average 1.5 microgram finest distributed (colloidal) platinum (plus palladium and rhodium) per kilometer drive. In a metropolis like Munich about 300 to 400 gram per year including hydrocarbons and root particles.

The direct inhalation by pedestrians/infants and pregnant woman explains the increasing disease rates. The mechanisms acting are the affinity of nickel for nitrogen and not for sulfur. Colloidal platinum/palladium and nickel for instance have a preference for carbon, alkene, alkine; alkylate, catalyse additive reactions, oxidations, hydrogenations. Platinum (Cis-Platinum) inhibits/stimulates proliferations/IgE-synthesis. Against the emerging genetic defects are our genetic engineers now fighting, a heavely expanding new field with new market: We are changing the evolution in high speed.

There is evidence for substantial variations of the hydroxyl radical (OH) concentrations in the Southern and Northern Hemisphere during the last two decades. The hydroxyl radical is the dominant oxidizing chemical in the atmosphere and is therefore directly involved in the ozone depletion and the greenhouse effect.

Radical reactions in that "metal-oxygen-nitrogen-carbon-water soup" are responsible for the ozone gap and the greenhouse effect. The catalyst metals, and therefore the automobile traffic, are for rising disease rates, ozone gap, greenhouse effect, climate change and forest dieback "the" responsible causes and not carbon dioxide with its dramatic rise.

The consequence should therefore be clear: fastest reduction of the metals (beside of carbon dioxide) out of our atmosphere – maybe we are in luck and the atmosphere is regenerating – like it did some millions of years ago at the time of the saurien dead.

Kiehl, R. rki-i.com, materialien , (pdf- and html-files).

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9. März 2005

Edelmetalle/Übergangsmetalle verantwortlich für Erbschäden und Ozonloch?

Kiehl, R., RKI-Institute, lab and res for mol med/biology, Salterweg 1, D-93437 Furth
E-mail: , Internet: .

Hauterkrankungen, Atemwegserkrankungen und vor allem Asthma, Erbschäden sowie Krebserkrankungen, wie Leukämie, steigen rapide seit der Einführung des Katalysators in den westlichen Ballungsgebieten - ab 1990 auch überproportional in den Ostblock-Staaten mit Wegfall der Mauer und Austausch alter Kraftfahrzeuge gegen Katalysator-Autos.

Verantwortlich dafür sind die in unsere Atmosphäre geblasenen feinstverteilten Metalle, wie Hg, Cd, das am häufigsten verbreitete Kontaktallergen Ni sowie - der meist signifikante atmosphärische "asthmatische" KFZ-Umwelt-Ausstoß Pt, Pd, Rh, mit oder ohne die ebenfalls zur Emission gelangenden aliphatischen und aromatischen Kohlenwasserstoffe sowie Rußpartikel.

Gewarnt wurde vor 15 bis 20 Jahren schon von Experten mit Ergebnissen aus entsprechenden Versuchen mit dem Katalysator in den USA, ohne daß irgend jemand darauf gehört hätte. So schneit es in Grönland seit Einführung des Katalysators bis zu 120 mal höhere Konzentrationen an Pt und Rh. Die westliche Hemisphäre wird mit den Platingruppen-Metallen in größtem Ausmaß kontaminiert, neben der schon bestehenden Kontamination durch Hg aus fossilen Brennstoffen.

Ein PKW mit Katalysator verliert im Schnitt 1.5 Mikrogramm feinst verteiltes Pt/km Fahrt. In einem Stau mit langsamer Fahrt sind dies etwa 1.1 Gramm. In einer Großstadt wie München werden pro Tag ein Gramm Pt freigesetzt oder ca. ein halbes Kilo/Jahr plus Kohlenwasserstoffe und Rußpartikel.

Die direkte Einatmung durch Fußgänger sowie Schwangere und Kleinkinder erklärt die Erkrankungsraten. Diese Tatsache ändert sich auch nicht mittels Verharmlosung durch die Kraftfahrzeug-Industrie oder andere. Gegen die entstehenden genetischen Defekte gehen unsere Gentechniker nun vor, ein stark wachsendes neues Spielfeld mit neuem Markt: Wir ändern die Evolution im Schnelldurchgang...

Die Hydroxid-Radikal-Konzentration in der südlichen wie nördlichen Hemisphäre variieren signifikant seit zwei Dekaden. Das Hydroxyl-Radikal ist die dominante oxidierende Chemikalie in der Atmosphäre und damit direkt involviert in den Ozon-Haushalt und den "Treibhaus-Effekt".

Radikal-Reaktionen in dieser "Metall-Sauerstoff-Stickstoff-Kohlenstoff-Wassersuppe" sind verantwortlich für das Ozon-Loch und den Treibhaus-Effekt. Die Katalysator-Metalle und damit der KFZ-Verkehr sind für steigende Erkrankungsraten, Ozonloch, Treibhaus-Effekt, Klima-Änderung und Waldsterben "der" Verursacher und nicht Kohlendioxid mit seinem Anstieg.

Die Konsequenz dürfte damit klar sein: schnellste Reduktion der Metalle aus unserer Atmosphäre (neben der Reduktion von CO_2) – vielleicht haben wir Glück und die Atmosphäre erholt sich – so wie sie es schon einmal einige Millionen Jahre vorher zur Zeit des Aussterbens der Dinosaurier getan hat.

Kiehl, R. Internet, rki-i.com , materialien , (frei herunterladbare pdf- und html-files unter Publikationen, Materialien – Buchkapitel und Powerpoint-Präsentationen).