Collection of Links to Dental Amalgam & Mercury critical Studies

Eine Linksammlung zu Zahnamalgam & Quecksilber kritischen Studien

Erstellt im Juli 2013 von..

INDEX

VORWORT & WARUM DIESE LISTE ?

Ich als – getrost darf man jetzt sagen EX- -- Zahnmetallgeschädigter hatte Glück , dass ich bereits in meinem damaligen Abiturlehrgang in meinen jungen Jahren einen so hervorragenden “Draht” zur Chemie & Biochemie haben durfte. Daneben meine jahrelange “Leidenschaft” diverses aus der Literatur , insbesondere in den letzten Jahren natürlich aus diesen Bereichen , in Englischer Sprache verschlingen zu wollen

Diese beiden Tatsachen halfen enorm dabei “mit zu helfen” meinen langjährigen Beschwerden , die in das Spektrum Erschöpfungszustände,Müdigkeiten ,Depression ,Ein & Durchschlafstörungen , diverse neuropathische Schmerzzustände einzukategorisieren sind , in Kooperation mit meiner Medizinerin auf den Grund zu gehen.

Beschwerden , die im Volksmund und von “unkundigen” Medizinern gerne mal in die Ecke “Hypochondrie” verschoben werden. Was allerdings gegen diese “Hypochondriehypothese” sprach , war die Tatsache , dass sich diese Beschwerden zunehmend in jene Richtung manifestierten , DASS MAN MIR DIESE PROBLEME AUCH (OPTISCH) ANSSAH ! Das galt INSBESONDERE für die Zeit am Morgen (erschöpfte ,müde Augen & verkatert) , ! Und ,.. es kam gelegentlich schon mal vor , dass ich mir von Kollegen vorhalten musste : “ Hey hast du die letzte Nacht in Schwabing durchgemacht ? Zynismus kam in solchen Fällen von meiner Seite zurück ! Natürlich schämte ich mich (insgeheim) dafür und da dieses Phänomen über die Jahre nicht verschwand , sondern die Tendenz hatte eher immer schlimmer als besser zu werden, entschloss ich mich diese Sache final anzugehen.

Hierzu fand ich auch eine wirklich sehr engagierte und aufgeschlossene Medizinerin. Diagnostiziert & ausprobiert wurde in dieser Zeit viel. Unter anderem wurde in dieser Zeit auch sehr viele Labormarker angefordert und es stellte sich an Hand der Ergebnisse vieler Labormarker heraus , dass es sich hier NICHT um Hypochondrie handeln konnte , da VIELE Labormarker ausserhalb ihrer zulässigen Referenzbereiche lagen. UND , man stellte eine HOCHGRADIGE Allergie gegen praktisch SÄMTLICHE gängigen Getreidesorten , einschließllich Gluten Huhn und Haselnuss fest. Der Allergiescan damals ergab: FÜR ALLE HÖCHSTE STUFE , STUFE III.. Damit war klar , ich WAR KEIN HYPOCHONDER !!!

Diverse Therapieversuche und Versuche mit diversen Therapiemaßnahmen ,seien es Medikamente , Nahrungsergänzungsmittel , Vitamin & Nährstofftherapien ,Therapieverfahren etc. brachten keinen DURCHSCHLAGENDEN UND/ODER DAUERHAFTEN Erfolg.

Bis schließlich im Winter 2009/10 die Quecksilberfrage das erste mal gestellt wurde: Ich hatte noch 4 alte

Amalgamplomben im Mund , die noch aus der Zeit <1987 stammten. Mein Glück hier – jetzt im nachhinein – war , dass uns (mich) unser damaliger Chemielehrer (Dr. in Chemie) in unserem Leistungskurs Chemie (2.

Bildungsweg) in der Zeit etwa von 1886 – 1989 darüber aufklärte , dass es besser ist auf Amalgamplomben zu verzichten , da in diesem Plomben sehr viel Hg verarbeitet wird und dass Hg sehr giftig ist.

Diese Aufklärungsarbeit dieses Chemielehreres ist es wohl auch zu verdanken ,dass ich heute noch lebe. Schließlich ließ mich diese Sache aufhorchen und ich verzichtete ab diesem Zeitpunkt auf Amalgamplomben. Ab der Zeit etwa >1987 wurden mir NUR noch Kunststoffplomben eingesetzt , wenn es mal was zu reparieren gab, WEIL ICH DARAUF BESTAND ! . Die 4 alten Amalgamplomben , beließ ich allerdings in meinem Mund , da ich damals dachte: “so schlimm wird's dann schon nicht werden' . GENAU DAS stellte sich später als FATALER FEHLER heraus.

In der Zeit April/Mai 2010 wurde eine Stuhlprobe von mir im Labor Bremen auf seine Hg-Belastung hin untersucht und das Ergebnis war sehr überwältigend: Es wurden 70 µg Hg/kg Stuhl gemessen , was ohne Frage für einen Kenner als monströs kategorisiert werden kann. 70 µg ?? ..und das ganz OHNE Provokation durch einen Metallchelatoren wie DMSA , DMPS oder Alpha Liponsäure ? Das Ergebnis war ohne Frage bemerkenswert und damit auch klar , ... diese Plomben müssen raus .

Im Mai 2010 wurden mir so dann diese Plomben entfernt und es folgte eine viele Monate dauernde Detoxphase ! Zu unserer aller Überraschung konnten sich im Zuge dieser Therapiemaßnahme (FAST) ALLE einstig auffälligen Labormarker nicht nur verbessern ,sie führte auch zur KOMPLETTEN Normalisierung so mancher dieser einstig auffälligen Laborparameter.

Was an dieser Sache allerdings AM MEISTEN überwältigte , war das Folgende: Ca. 1,5 Jahre nach der Entfernug der Plomben und der darauf folgenden Detox wurden die (ALLE!) Allergiescanns auf Nahrungsmittel wiederholt und ,. wie durch ein "medizinisches Wunder": **DIESE ALLERGIEN WAREN ALLE , REST & SPURLOS VERSCHWUNDEN !!**

Damit wurde ein medizinisches Dogma und Paradigma , das besagt , dass Allergien unheilbar wären verletzt und gebrochen . Der Leser wird verstehen , dass eine solche Erfahrung für das Leben eines Menschen prägenden Charakter hat und dass man sich im Zuge solcher Entwicklung selbst weiter an dieser "Amagamfront" sei es auf gesundheitspolitischer und/oder sozialer Ebene engagiert , ja , engagieren MUSS !

Wie ich eingangs schon darauf hinwies bildete mein einstiger Chemielehrgang mit einem sehr guten Verständnis für diese Sache , so wie mein gutes Verständnis für Englisch eine sehr gute Grundlage sich viel wertvolles Material hierzu selbst aus dem Netz "zu graben" . Daher war viel Zeit meiner Freizeit über die Jahre im meiner eigenen Researchtätigkeit einschlägigen Seiten gewidmet , die dieses Thema nicht populärwissenschaftlich, sondern MÖGLICHST "professionell" thematisieren.

Eine der sehr wichtigen Seiten hier bildet die Studiendatenbank von Pubmed , die mittlerweile schon rd. 25 Mio Artikel und Arbeiten aus allen Bereichen der Medizin und der Medizin affillierten Wissenschaften umfasst ! UNTER ANDEREM DORT konnte ich über die Jahre sehr viele interessante "Fälle" ausgraben , die einem in dieser Sache sehr gut weiter helfen konnten.

Das "Graben" auf einschlägigen Seiten im Netz ist oft ein sehr mühsames und zeitraubendes Geschäft und mit der Zeit vermisste ich so etwas ,. wie eine gut zusammen gestellte Linksamlung zum Thema "KRITISCHE Betrachtungen und Studien zum Thema Zahnamalgam und dem damit verbundenen Thema Toxikologie des Quecksilbers.

Das , was ich hier im Netzt über die Jahre finden konnte ist relativ dürftig ! Wenn überhaupt , enthalten sie oft nur ein paar wenige Links , oder es sind Sammlungen von Hinweisen zu Arbeiten , Autoren und/oder Titeln von Studien & Arbeiten OHNE VERLINKUNG ! Mir war es wichtig in einer Sammlung AUCH DIE JEWEILIGEN VERLINKUNGEN zu den Arbeiten vorfinden zu können (SO VIELE WIE MÖGLICH) und da es davon so gut wie nichts Brauchbares gab , entschloß ich mich diese Sache selbst in die Hand zu nehmen und erstellte nun diese Linkliste.

Diese Liste hier , die nun zweifelsohne WELTWEIT die größte Linksammlung seiner Art (geworden) sein dürfte , soll NICHT NUR dem Zweck der Kollektivierung zahnamalgam und Hg-kritischem Studienmaterials um damit dem Leser das Arbeiten in dieser Materie erleichtern zu können, dienen ,sie soll vor allem dem Bestreben im Kampf gegen das Zahnamalgam und dem weltweiten Quedcksilberverbot eine gute Schützenhilfe leisten können , indem sie SO –konzentiert – (sehr) viel Argumentationshilfe wie möglich dabei einbringen kann.

Letzteres war für mich die WICHTIGSTE Motivationsgrundlage diese Liste zu erstellen.

München im Juli 2013

ZUR VERWENDUNG DIESER LISTE.

Die Liste selbst wurde in Englischer Sprache beibehalten , da Englisch zum Einen die weltweite anerkannte Wissenschaftssprache ist und zum Anderen , weil die Titel der jeweilgen Studien , so wie die aus den Abstracts und Studies entnommenen “Coremessages” - also die Kernbotschaften die ich aus den Studien /Abstracts herauskopiert und so hier übernommen habe - so in Englischer Sprache geblieben sind . Das beibehalten dieser Arbeit in Englisch hat seine Vorteile , da sie so WELTWEIT leichter im Kampf gegen diesen Amalgam & Quecksilberwahnsinn eingesetzt werden kann .

Die Aufgliederung der Informationen und Links dieser Arbeiten erfolgt jeweils über mehre Zeilen wobei jeder Link/Arbeit entsprechend dem Kapiel dem er/sie zugehört durchnummeriert wurde. In der ersten Zeile finden wir jeweils die Nummerierung fett markiert. Daraufhin folgt in der nächsten Zeile. DER TITEL dieser Arbeit . Sodann folgt in der nächsten Zeile DER LINK zu dieser Arbeit. In der daraufhin folgenden Zeile teile ich mit , ob diese Arbeit unter diesem Link voll zugänglich ist (FULL ACCESS TO STUDY) , oder diese nur als Abstract (Abstract only) vorliegt.

Nun folgt die Angabe “Coremessage/s” . Unter dieser Angabe finden wir sodann DAS WICHTIGSTE komprimiert - als “Kernbotschaft” dieser Studie - aus dieser Studie (meist die Ergebnisse) in Beziehung zum Titel der Studie, Ich habe hierzu fast ausschließlich die entsprechenden Sätze (Botschaften) aus den unter diesen Links vorgefundenen Studies herauskopiert und hier als “Cormessage/s” eingefügt.

Diese Arbeit hier teilt sich auf auf 3 große Abschnitte. Teil 1 umfasst alle gefundenen kritischen Arbeiten die im DIREKTEN UND INDIREKTEN Zusammenhang zu Amalgam stehen. Teil 2 umfasst sämtliche kritischen Arbeiten im Bezug auf Hg (Quecksilber) OHNE direkten , oder indirekten Bezug zu Zahamalgam. Teil 3 wurde möglichen Kausalitätsketten gewidmet. So z.B. Der Sache einer möglichen – UND SOGAR SEHR WAHRSCHEINLICHEN -- genetisch bedingten Suszeptibilität gegenüber Hg , resp. Zahamalgam: GSTM1

DA ICH SELBST GSTM1 NULL -TYP bin und ich im Zusammenhang meines Engagements hier (auch im Netz) weitere Betroffene kennen lernen konnte , sind mir – einschließlich mir selbst – 4 weitere Fälle bekannt , die einer “Amalgamerkrankung/Unvertäglichkeit” zugeordnet werden können UND die allesamt EBENFALLS DEM GSTM1 NULL Typen angehören. Mir ist bis Dato noch kein Fall (eines Erkrankten) unter gekommen , der dem GSTM1 (+/+) ,(+/-),(-/+) zugeordnet werden kann . ICH FINDE DAS HOCHINTERESSANT UND VON GEWICHTIGER BEDEUTUNG !!

Die Liste steht zur FREIEN Verwendung und soll so auch fleißig ihren Einsatz , im Kampf gegen diesen weltweiten Amalgam & Hg – Wahnsinn finden können. Diese Liste kann (und soll) bei Bedarf auch auf hierzu – zu diesem Thema -- wichtigen Servern gehostet werden ! Allerding empfiehlt sich diese Arbeit nur AUF EINER Seite (Server) zu hosten , da es die Suchmaschine Google nicht gerne sieht , wenn eine Seite/Arbeit mit identischem Inhalt auf verschiedenen Webseiten gehostet wird (duplicate content & Spamverdacht !).

Die Leser sind aufgefordert diese Arbeit zu erweitern. Sie kann bei Bedarf selbst geupdated und erweitert werden mit Links , DIE NOCH NICHT Bestandteil dieser Liste sind. Dazu bedarf es allerdings meiner Zustimmung. Alternativ kann man , wenn jemand einen Link zu einer KRITISCHEN Studie kennt --- DIE NOCH NICHT Bestandteil dieser Liste ist – auch mich selbst zu diesem Zweck kontaktieren. Ich werde dann entsprechend diese Liste mit diesem Link updaten. Zu erreichen bin ich hierzu unter der Emailadresse

Wichtig: Aufgenommen werden NUR kritische Arbeiten , die noch nicht Bestandteil dieser Liste sind und sie MÜSSEN BEDINGUNGSLOS einen LINK vorweisen , auf dem WENIGSTENS der Abstract dieser Arbeit zu erreichen ist , ALLERWENIGSTENS – falls nicht mal ein Abstract vorliegt – muss der Link auf eine Seite führen , wo diese Arbeit/Studie kostenpflichtig herunter geladen werden kann. Damit nun zu den Linklisten.

TEIL 1.

PROBLEMS WITH DENTAL AMALGAM AND MERCURY TOXICITY IN DIRECT & INDIRECT RELATIONSHIPT TO DENTAL AMALGAM

1.1 DENTAL AMALGAM & MERCURY TOXICITY , GENERAL CASES .

1.1.1.
Mercury toxicity and dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/6361623
Abstract only
Coremessage:
It is generally agreed that if amalgam was introduced today as a restorative material, they would never pass F.D.A. Approval.

1.1.2.
Dental amalgam, mercury toxicity, and renal autoimmunity. =>
http://www.ncbi.nlm.nih.gov/pubmed/18540850
Abstract only
Coremessage:
We selected a group of patients (n=24) with a history of long-term exposure to mercury vapor from mercury-containing amalgam fillings and showing adverse effects that were laboratory confirmed.

1.1.3.
Dental amalgam and mercury levels in autopsy tissues: food for thought.=>
http://www.ncbi.nlm.nih.gov/pubmed/16501347
Abstract only
Coremessage:
Total mercury levels were significantly higher in subjects with a greater number of occlusal amalgam surfaces (>12) compared with those with fewer occlusal amalgams (0-3) in all types of tissue (all P < or = 0.04).

1.1.4.
Mercury exposure and risks from dental amalgam in the US population, post-2000.=>
http://www.ncbi.nlm.nih.gov/pubmed/21782213
Abstract only
Coremessage:
Based on the least conservative of the scenarios evaluated, it was estimated that some 67.2 million Americans would exceed the Hg dose associated with the reference exposure level (REL) of 0.3 µg/m(3) established by the US Environmental Protection Agency; and 122.3 million Americans would exceed the dose associated with the REL of 0.03 µg/m(3) established by the California Environmental Protection Agency.

1.1.5.
Dental amalgam fillings and the amount of organic mercury in human saliva.=>
http://www.ncbi.nlm.nih.gov/pubmed/11385194
Abstract only
The amount of organic and inorganic mercury in paraffin-stimulated saliva was significantly higher ($p<0.001$) in subjects with dental amalgam fillings (n = 88) compared to the nonamalgam study groups (n = 43 and n = 56): log(e) (organic mercury) was linearly related to log(e) (inorganic mercury, r(2) = 0.52)

1.1.6.
The Contribution of Dental Amalgam to Urinary Mercury Excretion in Children =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2022658/
FULL ACCESS TO STUDY
Coremessage:
Urinary mercury concentrations are highly correlated with both number of amalgam fillings and time since placement in children.

1.1.7.
Correlation of dental amalgam with mercury in brain tissue. =>
http://www.ncbi.nlm.nih.gov/pubmed/3480359
Abstract only
Coremessage:
Data from this project demonstrate a positive correlation between the number of occlusal surfaces of dental amalgam and mercury levels in the brain (p less than .0025 in white matter). This is indirect evidence suggesting that mercury from dental amalgam fillings may contribute to the body burden of mercury in the brain.

1.1.8.
Dental amalgam exposure and urinary mercury levels in children: the New England Children's Amalgam Trial.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2235235/
FULL ACCESS TO STUDY

1.1.9.
People with high mercury uptake from their own dental amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1128166/pdf/oenvmed00062-0052.pdf
FULL ACCESS TO STUDY
Coremessages:
Blood Hg was 12-23 micrograms/l, which is five to 10 times the average in the general population.
CONCLUSION--Although the average daily Hg uptake from dental amalgam fillings is low, there is a considerable variation between people; certain people have a high mercury uptake from their amalgam fillings.

1.1.10
Mercury (Hg) burden in children: the impact of dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/21601239
Abstract only
Coremessage:
We found that children with amalgam fillings (N=106) had significantly higher UHg-C levels than children without (N=76), with means of 3.763 µg/g creatinine versus 3.457 µg/g creatinine, respectively (P=0.019)

1.1.11.
Contribution of Dental Amalgam to Urinary Mercury Excretion in Children: Woods et al. Respond
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2265046/
FULL ACCESS TO STUDY

1.1.12.
Dental amalgam restorations: daily mercury dose and biocompatibility.=>
http://www.ncbi.nlm.nih.gov/pubmed/16393137
Abstract only
Coremessage:
The daily dose is found to be 14% of the threshold above which observable adverse neurological symptoms are expected.

1.1.13.
Dental amalgam and mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/2485649
Abstract only
Coremessage:
Mercury concentration in intraoral air and urine of seven females with dental amalgam was measured before and after intake of one hard-boiled egg. A considerable decrease in mercury concentration in intraoral air was found.

1.1.14.
Effects of bleaching on mercury ion release from dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/19329457
Abstract only
Coremessage:
There were significant increases in mercury release between control and all other hydrogen peroxide concentrations at all exposure times ($p < 0.05$).

1.1.15.
Evaluation of the mercury exposure of dental amalgam patients by the Mercury Triple Test=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1763642/pdf/v061p00535.pdf
FULL ACCESS TO STUDY

1.1.16.
Mercury in biological fluids after amalgam removal. =>
http://www.ncbi.nlm.nih.gov/pubmed/9539465
Abstract only
Coremessage:
It is concluded that the process of removing amalgam fillings can have a considerable impact on Hg levels in biological fluids. After removal, there was a considerable decline in the Hg levels of blood, plasma, and urine, which slowly approached those of subjects without any history of amalgam fillings

1.1.17.
Significant mercury deposits in internal organs following the removal of dental amalgam, & development of pre-cancer on the gingiva and the sides of the tongue and their represented organs as a result of inadvertent exposure to strong curing light (used to solidify synthetic dental filling material) & effective treatment: a clinical case report, along with organ representation areas for each tooth.=>
http://www.ncbi.nlm.nih.gov/pubmed/8914687
Abstract only

1.1.18.
Mercuric dichloride induces DNA damage in human salivary gland tissue cells and lymphocytes =>
http://www.ncbi.nlm.nih.gov/pubmed/17479252
Abstract only
Coremessages:
Amalgam is still one of the most frequently used dental filling materials. However, the possible adverse effects especially that of the mercuric component have led to continued controversy.
Increasing dose-dependent DNA migration could be demonstrated after exposure to HgCl(2) in cells of the salivary glands and lymphocytes
In both cell types a significant increase in DNA migration could be shown starting from HgCl(2)concentrations of 5 microM in comparison to the negative control.

1.1.19.
Dental amalgam mercury exposure in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/10581685
Abstract only
Coremessage:
The results showed significantly higher concentrations of mercury in the kidneys and the brains of rats in both exposed groups compared to control. Even after two months of exposure to mercury brain mercury concentration in rats with amalgam fillings was 8 times higher than in the control and 2 times higher than in rats exposed to amalgam supplemented diet.

1.1.20.
Cytotoxicity of dental composite components and mercury compounds in lung cells=>
http://www.ncbi.nlm.nih.gov/pubmed/11163377
Abstract only
Coremessages:
The toxic effect of HgCl2 and MeHgCl from the L2 cells was about 100-700-fold higher than of the dental composite components
A significant ($p<0.05$) time dependent increase of toxicity was observed with TEGDMA, HEMA and MeHgCl.

1.1.21.
Cytotoxicity of dental composite (co)monomers and the amalgam component Hg(2+) in human gingival fibroblasts.=>
http://www.ncbi.nlm.nih.gov/pubmed/16474958
Abstract only
Coremessages:
Unpolymerized resin (co)monomers or mercury (Hg) can be released from restorative dental materials (e.g. composites and amalgam). They can diffuse into the tooth pulp or the gingiva.
MeHgCl was the most toxic substance. In the BrdU assay, Hg(2+) was about fourfold less toxic than MeHgCl but Hg(2+) was about fourfold more toxic than BisGMA. In the BrdU test, a significantly ($P<0.05$) decreased toxicity was observed for Hg(2+) at 48 h, compared to the 24 h Hg(2+)-exposure
A time depending decreased toxicity was observed only for Hg(2+) which can then reach the toxic level of BisGMA.

1.1.22.
Cell death effects of resin-based dental material compounds and mercurials in human gingival fibroblasts.=>
http://www.ncbi.nlm.nih.gov/pubmed/16691427
Abstract only
Coremessage:
he results of this study indicate that resin composite components have a lower toxicity than mercury from amalgam in HGF. HEMA, BisGMA, UDMA, and HgCl2 induced mainly necrosis

1.1.23.
Cytotoxicity of dental composite components and mercury compounds in pulmonary cells. =>
http://www.ncbi.nlm.nih.gov/pubmed/11205434
Abstract only
Coremessage:
All tested substances induced a dose-dependent loss of viability in A549 and L2 cells after 24 h. The EC50 values of both mercurials were significantly ($p < 0.05$) lower compared to the values of both composite components.

1.1.24.
Effect of mercury (Hg) dental amalgam fillings on renal and oxidative stress biomarkers in children=>
http://www.ncbi.nlm.nih.gov/pubmed/22683759
Abstract only
Coremessages:
Multiple regression analyses revealed that the excretion of urinary NAG was significantly associated with the presence of dental amalgam fillings ($\beta=0.149$, $P=0.03$) and the levels of UHg-C ($\beta=0.531$, $P=0$), with an interaction between the two ($P=0$).
Urinary NAG levels were positively associated with urinary MDA levels ($\beta=0.516$, $P=0$) but not with 8-OHdG ($\beta=0.134$, $P=0.078$) after adjustment for potential confounders. Both UHg-C

1.1.25.
A study on the cytotoxicity of six filling materials in vitro=>
http://www.ncbi.nlm.nih.gov/pubmed/10680517
Abstract only
Coremessages:
The results showed that the cytotoxicity of Silver amalgam and the Gallium-Silver alloys, which were produced by mixing the conventional dental alloys powder or high copper alloys powder with Gallium, was significantly stronger than that of light curing composites and the Gallium-Silver alloys that were produced by the spherical amalgam alloys powder and Gallium. It suggested that the level of mercury and copper in the alloys can influence their cytotoxic properties.

1.1.26.
The relationship between mercury from dental amalgam and mental health.=>
http://www.ncbi.nlm.nih.gov/pubmed/2618948
Abstract only
Coremessage:
The data suggest that inorganic mercury poisoning from dental amalgam does affect the mind and emotions.

1.1.27.
Significant mercury deposits in internal organs following the removal of dental amalgam, & development of pre-cancer on the gingiva and the sides of the tongue and their represented organs as a result of inadvertent exposure to strong curing light (used to solidify synthetic dental filling material) & effective treatment: a clinical case report, along with organ representation areas for each tooth.=>
http://www.ncbi.nlm.nih.gov/pubmed/8914687
Abstract only
Coremessages:
Enhanced mercury evaporation by increased temperature and microscopic amalgam particles created by drilling may have contributed to mercury entering the lungs and G.I. system and then the blood circulation, creating abnormal deposits of mercury in the organs named above.
During the use of strong bluish curing light to create a photo-polymerization reaction to solidify the synthetic filling material, the adjacent gingiva and the side of the tongue were inadvertently exposed. This exposure to the strong bluish light was found to produce pre-cancerous conditions in the gingiva, the exposed areas of the tongue, as well as in the corresponding organs represented on those areas of the tongue, and abnormally increased enzyme levels in the liver. T

1.1.28.
Mercury release from dental amalgam restorations after magnetic resonance imaging and following mobile phone use.=>
http://www.ncbi.nlm.nih.gov/pubmed/18819554
Abstract only
Coremessage:
It appears that MRI and microwave radiation emitted from mobile phones significantly release mercury from dental amalgam restoration.

1.1.29.
Urinary porphyrin excretion in children with mercury amalgam treatment: findings from the Casa Pia Children's Dental Amalgam Trial.=>
http://www.ncbi.nlm.nih.gov/pubmed/19557617 ???
Abstract only

1.1.30.
The potential adverse health effects of dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/16042501
Abstract only
Coremessage:
Experimental evidence consistently demonstrates that Hg(0) is released from dental amalgam restorations and is absorbed by the human body. Numerous studies report positive correlations between the number of dental amalgam restorations or surfaces and urine mercury concentrations in non-occupationally exposed individuals

1.1.31.
Maternal-fetal distribution of mercury (203Hg) released from dental amalgam fillings. =>
http://www.ncbi.nlm.nih.gov/pubmed/2331037
Abstract only
Coremessage:
Results demonstrate that Hg from dental amalgam will appear in maternal and fetal blood and amniotic fluid within 2 days after placement of amalgam tooth restorations.

1.1.32.
Uptake and accumulation of mercury from dental amalgam in the common goldfish, Carassius auratus.=>
http://www.ncbi.nlm.nih.gov/pubmed/12685760
Abstract only

1.1.33
Mercury in human colostrum and early breast milk. Its dependence on dental amalgam and other factors.=>
http://www.ncbi.nlm.nih.gov/pubmed/9638609
Abstract only
Coremessage:
The Hg-M in the breast milk samples correlates positively with the number of maternal teeth with dental amalgam.

1.1.34.
Mercury in saliva and feces after removal of amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/9169079
Abstract only
Coremessage:
Before removal, the median Hg concentration in feces was more than 10 times higher than in samples from an amalgam free reference group

1.1.35.
Amalgam studies: disregarding basic principles of mercury toxicity=>
http://www.ncbi.nlm.nih.gov/pubmed/15471104
Abstract only
Coremessage:
It is widely accepted that the main source of mercury vapor is dental amalgam and it contributes substantially to mercury load in human body tissues.

1.1.36.
Serial measurements of intra-oral air mercury: estimation of daily dose from dental amalgam. =>
http://www.ncbi.nlm.nih.gov/pubmed/3860539
Abstract only
Coremessage:
Hg concentrations remained elevated during 30 min of continuous chewing and declined slowly over 90 min after cessation of chewing.

1.1.37.
Maternal amalgam dental fillings as the source of mercury exposure in developing fetus and newborn. =>
http://www.ncbi.nlm.nih.gov/pubmed/17851449
Abstract only
Coremessage:
Human placenta does not represent a real barrier to the transport of Hg(0); hence, fetal exposure occurs as a result of maternal exposure to Hg, with possible subsequent neurodevelopmental disabilities in infants
A strong positive correlation between maternal and cord blood Hg levels was found (rho=0.79; P<0.001).
Dental amalgam fillings in girls and women of reproductive age should be used with caution, to avoid increased prenatal Hg exposure.

1.1.38.
Mercury in dental practice. II. Urinary mercury excretion in dental personnel.=>
http://www.ncbi.nlm.nih.gov/pubmed/3469773
Abstract only
Coremessage:
There was a statistically significant correlation between the number of amalgam surfaces and the HgU values in the control group.

1.1.39.
Speciation of mercury excreted in feces from individuals with amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/9814717
Abstract only
Coremessage: ---

1.1.40.
[Amount of mercury from dental amalgam filling released into the atmosphere by cremation]. =>
http://www.ncbi.nlm.nih.gov/pubmed/7919469
Abstract only
Coremaessage:

The amount of mercury released from this crematorium was estimated to be approximately 9.4 kg per year, or a daily release of 26 g into the ambient air.

1.1.41.
Intra-oral air mercury released from dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/3860538
Abstract only
Coremessage:
Subjects with dental amalgams had unstimulated Hg vapor concentrations that were nine times greater than basal levels in control subjects with no amalgams.

1.1.42.
Release of mercury from dental amalgam fillings in pregnant rats and distribution of mercury in maternal and fetal tissues.=>
http://www.ncbi.nlm.nih.gov/pubmed/11516521
Abstract only
Coremessages:
The average mercury concentration in the air samples tended to decline as time elapsed but a marked amount (423.2+/-121.5 ng/day) was observed even on day 15.
The amount of mercury in the air samples increased 7--20-fold after chewing.
The placement of the single amalgam restoration (3.8--5.5 mg in weight) increased the levels of mercury approximately three to 6 times in the maternal brain, liver, lung, placenta and 20 times in the kidneys.
The highest mercury concentration among fetal organs was found in the liver, followed by the kidneys and brain.

1.1.43.
Side-effects: mercury contribution to body burden from dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/1292449
Abstract only

1.1.44.
Human exposure to mercury and silver released from dental amalgam restorations.=>
http://www.ncbi.nlm.nih.gov/pubmed/7944571
Abstract only
Coremessages:
Fecal excretions ranged from 1 to 190 micrograms Hg/24 h and from 4 to 97 micrograms Ag/24 h.
The worst-case individual showed a fecal mercury excretion amounting to 100 times the mean intake of total Hg from a normal Swedish diet.

1.1.45.
Total and inorganic mercury in breast milk in relation to fish consumption and amalgam in lactating women.=>
http://www.ncbi.nlm.nih.gov/pubmed/8687245
Abstract only
Coremessage:
The results indicated that there was an efficient transfer of inorganic mercury from blood to milk and that, in this population, mercury from amalgam fillings was the main source of mercury in milk.

1.1.46.
Amalgam risk assessment with coverage of references up to 2005]=>
http://www.ncbi.nlm.nih.gov/pubmed/15789284
Abstract only
Coremessage:
Removal of dental amalgam leads to permanent improvement of various chronic complaints in a relevant number of patients in various trials
Summing up, available data suggests that dental amalgam is an unsuitable material for medical, occupational and ecological reasons

1.1.47.
Mercury levels in plasma and urine after removal of all amalgam restorations: the effect of using rubber dams.=>
http://www.ncbi.nlm.nih.gov/pubmed/9823089=>
Abstract only
Coremessage:
The study showed that dental amalgam had a statistically significant impact on the mercury levels found in plasma and urine in the patients tested, and that the use of a rubber dam during removal of all amalgam restorations significantly reduced the peak of mercury in plasma following removal.

1.1.48.
Mercury in dental restoration: is there a risk of nephrotoxicity?=>
http://www.ncbi.nlm.nih.gov/pubmed/12018634
Abstract only
Coremessages:
Urinary excretion of NAG, gammaGT and albumin was significantly higher in persons with dental amalgam than those without
From the nephrotoxicity point of view, dental amalgam is an unsuitable filling material, as it may give rise to Hg toxicity.

1.1.49.
Mercury release from dental amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/23511257
Abstract only
Coremessage:
Groups 1 and 2 both demonstrated a significant 3-fold increase in Hg vapour levels after chewing, while levels in controls remained undetectable.

1.1.50.
Urinary Mercury Levels in Children with Amalgam Fillings=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2453182/
FULL ACCESS TO STUDY
Coremessage:
Previous postmortem studies in humans have shown that mercury levels originating from dental amalgam surfaces and retained in tissues are higher in brain regions and thyroid than those measured in renal cortex

1.1.51.
A significant relationship between mercury exposure from dental amalgams and urinary porphyrins: a further assessment of the Casa Pia children's dental amalgam trial.=>
http://www.ncbi.nlm.nih.gov/pubmed/21053054
Abstract only
Coremessage:
Significant dose-dependent correlations between cumulative exposure to Hg from dental amalgams and urinary porphyrins associated with Hg body-burden (pentacarboxyporphyrin, precoproporphyrin, and coproporphyrin) were observed.

1.1.52.
The Contribution of Dental Amalgam to Urinary Mercury Excretion in Children=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2022658/
FULL ACCESS TO STUDY
Coremessage
Urinary mercury concentrations are highly correlated with both number of amalgam fillings and time since placement in children. Girls excrete significantly higher concentrations of mercury in the urine than boys with comparable treatment, suggesting possible sex-related differences in mercury handling and susceptibility to mercury toxicity

1.1.53.
Migration of mercury from dental amalgam through human teeth.=>
http://www.ncbi.nlm.nih.gov/pubmed/18296776
Abstract only
Coremessage:
Hg (up to approximately 10 mg g(-1)) and Zn (>100 mg g(-1)) were detected in the teeth several millimetres

from the location of the amalgams.

1.1.54.
Long-term use of nicotine chewing gum and mercury exposure from dental amalgam fillings. =>
http://www.ncbi.nlm.nih.gov/pubmed/8655765
Abstract only
Coremessage:
The impact of excessive chewing on mercury levels was considerable.

1.1.55.
Effect of occupational exposure to elemental mercury in the amalgam on thymulin hormone production among dental staff.=>
http://www.ncbi.nlm.nih.gov/pubmed/19482909
Abstract only
Coremessage:
In conclusion, our results show that dentists and dental nurses have significant exposure to mercury vapor and point to the negative impact of mercury on thymus gland functions and confirm the implication that the nitric oxide pathway is a possible mechanism for this impact.

1.1.56.
Mercury release from dental amalgam after treatment with 10% carbamide peroxide in vitro.=>
http://www.ncbi.nlm.nih.gov/pubmed/10673659
Abstract only
Coremessage:
Amalgam specimens exposed for 48 hours to 10% carbamide peroxide showed significantly higher concentrations of mercury in solution as compared with specimens treated with phosphate buffer (P <.001).

1.1.57.
The relationship between mercury from dental amalgam and oral cavity health.=>
http://www.ncbi.nlm.nih.gov/pubmed/2132561
Abstract only
Coremessage:
Comparisons between subjects with and without amalgam showed significant differences of diseases of the mouth.

1.1.58.
Lichenoid reactions of murine mucosa associated with amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/12752133
Abstract only
Coremessage:
In 97% of all patients with oral lichenoid reactions (OLR) associated with dental amalgam a removal of the fillings leads to a decline of the lesions, as a minimum

1.1.59.
Oral lichenoid reactions associated with amalgam: improvement after amalgam removal.=>
http://www.ncbi.nlm.nih.gov/pubmed/12534597
Abstract only
Coremessage:
Of all patients with OLR associated with dental amalgam fillings, 97.1% benefited from amalgam removal regardless of patch test results with amalgam or INM.
We suggest that the removal of amalgam fillings can be recommended in all patients with symptomatic OLR associated with amalgam fillings if no cutaneous LP is present.
1.1.60.
Oral lichenoid lesions (OLL) and mercury in amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/12694209
Abstract only
Coremessages:
84 patients with oral lichenoid lesions (OLL) were seen in the contact dermatitis clinic. All these patients had reticulate, lacy, plaque-like or erosive lichenoid changes adjacent to amalgam fillings
patients had replacement of their amalgam fillings, with 28 (87%) patients experiencing improvement of symptoms and signs within 3 months.

1.1.61.
The effect of bleaching agents on mercury release from spherical dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/15055616
Abstract only
Coremessage:
All groups exposed to bleach showed increased mercury release over time.

1.1.62.
Potential public health risks related to mercury/amalgam discharge from dental offices. =>
http://www.ncbi.nlm.nih.gov/pubmed/9520646
Abstract only..
Coremessage:
The use of mercury by the dental profession represents approximately 6 percent of the total annual domestic consumption and is estimated to contribute significantly to the discharge of mercury (14 percent in one study) to waste-water streams.

1.1.63.
[Amalgam and the toxicological risks of mercury. A review of the argument].
http://www.ncbi.nlm.nih.gov/pubmed/7476786
Abstract only

1.1.64.
Documented clinical side-effects to dental amalgam. =>
http://www.ncbi.nlm.nih.gov/pubmed/1292453
Abstract only
Coremessage:
The medical scientific community is now in general agreement that patients with dental amalgam fillings are chronically exposed to mercury, that the average daily absorption of mercury from dental amalgam is from 3 to 17 micrograms per day, and that the amalgam mercury absorption averages 1.25-6.5 times the average mercury absorption from dietary sources (World Health Organization, 1991). T

1.1.65.
Cracked mercury dental amalgam as a possible cause of fever of unknown origin: a case report=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2288608/
FULL ACCESS TO STUDY
Coremessage: OHNE WORTE !!!!!

1.1.66.
[Mercury and dental amalgam fillings].
http://www.ncbi.nlm.nih.gov/pubmed/9621758
Abstract only

1.1.67.
Evaluation of oral tissue response and blood levels of mercury released from dental amalgam in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/23611063
Abstract only
Coremessages:
The blood mercury levels of mothers increased significantly at six months ($P<0.01$) as compared to levels at one month.
Histopathology results from mothers showed inflammatory response at the bottom of the socket,
At six months, teeth germs showed vacuolation of the abnormal odontoblasts with globular dentine formation.
Degenerated periodontal fibres and thin trabeculae forming the bony sockets with large marrow spaces were evident.
A fibrous connective tissue capsule surrounded the amalgam mass inside the mucosa of mothers at one month and was evident also at 6 months with a huge inflammatory cell infiltrate.
Teeth germs showed elongated odontoblasts with intercellular oedema, thinner dentine and bony trabeculae with wider marrow spaces.

1.1.68.
Effects of dental amalgam and heavy metal cations on cytokine production by peripheral blood mononuclear cells in vitro.
http://www.ncbi.nlm.nih.gov/pubmed/9740009
Abstract only
Coremessages:
Freshly prepared amalgam as well as ACCM induced a decrease in the production of interferon-gamma (IFN-gamma) and interleukin-10 (IL-10), and an increase in the concentrations of tumor necrosis factor-alpha (TNF-alpha)
However, Ag+ markedly suppressed the production of IFN-gamma, IL-10, and TNF-alpha.
In summary, our results show that fresh amalgam, but not amalgam aged for 6 weeks, causes changes in the cytokine pattern of PBMC in vitro, and that these effects are due to the release of Cu2+ and Hg2+.

1.1.69.
Dental amalgam fillings and the amount of organic mercury in human saliva.=>
http://www.ncbi.nlm.nih.gov/pubmed/11385194/
Abstract only
Coremessage:
The amount of organic and inorganic mercury in paraffin-stimulated saliva was significantly higher ($p<0.001$) in subjects with dental amalgam fillings (n = 88) compared to the nonamalgam study groups (n = 43 and n = 56)

1.1.70.
Methylmercury, Amalgams, and Children's Health=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1392265/
FULL ACCESS TO STUDY
Coremessage: Heintze et al. (1983) and Lyttle et al. (1993) reported direct evidence that organic mercury in saliva is due to the transformation of bacteria.
Considering that the relevant feature of methylmercury in humans is accumulation in both adult and fetal brain, it is quite clear that, over time, the extensive exposure to methylmercury associated with dental amalgams should be taken into account.

1.1.71.
Evidence that mercury from silver dental fillings may be an etiological factor in smoking.=>
http://www.ncbi.nlm.nih.gov/pubmed/8516784
Abstract only
Coremessage:
Because mercury decreases dopamine, serotonin, norepinephrine, and acetylcholine in the brain, and nicotine has just the opposite effect on these neurotransmitters, this may help explain why persons with dental amalgams smoke more than persons without amalgams.

1.1.72.
Recurrent contact dermatitis caused by mercury in amalgam dental fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/1184258
Abstract only
Coremessage:
This case history suggests that contact dermatitis may be caused by not only the mercury in new fillings but also by that in old fillings.

1.1.73.
Long-term mercury excretion in urine after removal of amalgam fillings =>
http://www.ncbi.nlm.nih.gov/pubmed/7814102
Abstract only
Coremessage:
The exposure from amalgam fillings thus exceeds the exposure from food, air and beverages. Within 12 months after removal of all amalgam fillings the participants showed substantially lower urinary mercury levels which were comparable to those found in subjects who have never had dental amalgam fillings.

1.1.74.
Childhood urine mercury excretion: dental amalgam and fish consumption as exposure factors=>
http://www.ncbi.nlm.nih.gov/pubmed/15016596
Abstract only
Coremessage:
Using a sample of 60 children, we found that children with amalgam fillings had significantly higher UHg levels than children without amalgams

1.1.75.
The influence of amalgam fillings on urinary mercury excretion in subjects from Apulia (southern Italy).=>
http://www.ncbi.nlm.nih.gov/pubmed/9658238
Abstract only
Coremessages:
only the number of amalgam fillings ($T = 5.25$; $p = 0.025$) and the number of restored surfaces ($T = 2.33$; $p = 0.020$) were found liable to affect urinary mercury excretion in a significant manner.
In conclusion, the results of this study confirm the primary role of amalgam fillings in affecting urinary mercury excretion in those subjects who are not occupationally exposed to inorganic mercury

1.1.76.
Estimation of mercury body burden from dental amalgam: computer stimulation of a metabolic compartmental model.
http://www.ncbi.nlm.nih.gov/pubmed/3465771
Abstract only
Coremessage:
The model predicted that continuous exposure to elemental Hg vapor, at 30 micrograms/day for 10 years, would result in a total Hg body burden of 5.9 mg, of which 4.8 mg could be contained in R4. Assuming that the Hg in R4 displayed uniform distribution throughout the body, then the brain concentration was estimated to be 68 ng/g wet weight

1.1.77.
Breast-milk mercury concentrations and amalgam surface in mothers from Brasília, Brazil.=>
http://www.ncbi.nlm.nih.gov/pubmed/16116246
Abstract only
Coremessage:
The Pearson correlation coefficient was significant ($r = 0.6087$, $p = 0.0057$) between breast-milk Hg and number of amalgam surfaces

1.1.78.
Chronic neurobehavioural effects of elemental mercury in dentists =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1039326/
FULL ACCESS TO STUDY
Coremessage:
In neurobehavioural tests measuring motor speed (finger tapping), visual scanning (trail making), visuomotor coordination and concentration (digit symbol), verbal memory (digit span, logical memory delayed recall), visual memory (visual reproduction, immediate and delayed recall), and visuomotor coordination speed (bender-gestalt time), the performance of the dentists was significantly worse than that of the controls.

1.1.79.
Mercury concentrations in urine and whole blood associated with amalgam exposure in a US military population.=>
http://www.ncbi.nlm.nih.gov/pubmed/9496919
coremessage:
Significant correlations were detected between amalgam exposure and the total ($r = 0.34$, $p < 0.001$) and inorganic 0.34 ($r = 0.34$, $p < 0.001$) urinary mercury concentrations on the original scale. Stronger correlations were found for total ($r = 0.44$, $p < 0.001$) and inorganic ($r = 0.41$, $p < 0.001$) urinary Hg on the log scale, as well as for creatinine-corrected total ($r = 0.43$, $p < 0.001$) and inorganic ($r = 0.43$, $p < 0.001$) urine concentrations.

1.1.80.
Adverse immunological effects and autoimmunity induced by dental amalgam and alloy in mice.
http://www.ncbi.nlm.nih.gov/pubmed/7958626
Abstract only
Coremessages:
10 weeks of low-dose and 6 months of high-dose amalgam implantation strongly increased mitogen-induced T and B cell proliferation,
In conclusion, dental amalgam implantation in a physiological body milieu causes chronic stimulation of the immune system with induction of systemic autoimmunity in genetically sensitive mice.

1.1.81.
Dental Amalgam Exposure and Urinary Mercury Levels in Children: The New England Children's Amalgam Trial=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2235235/
Abstract only
FULL ACCESS TO STUDY
Coremessage:
In multivariate models, each additional amalgam surface present was associated with a 9% increase in current U-Hg, and each additional posterior occlusal surface-year was associated with a 3% increase in cumulative U-Hg excretion ($p < 0.001$).

1.1.82.
Symptoms and differential diagnosis of patients fearing mercury toxicity from amalgam fillings =>
http://www.ncbi.nlm.nih.gov/pubmed/9456068
Abstract only
Coremessage:
Among the patients there were 26 who had had their amalgam fillings removed and who, at the time of the follow-up, were subjectively cured.

1.1.83.
Sodium 2,3-dimercaptopropane-1-sulfonate challenge test for mercury in humans: II. Urinary mercury, porphyrins and neurobehavioral changes of dental workers in Monterrey, Mexico.=>
http://www.ncbi.nlm.nih.gov/pubmed/7815341
Abstract only
Coremessages:
The sodium salt of 2,3-dimercaptopropane-1-sulfonic acid (DMPS) challenge test (300 mg p.o. after an 11-hr fast) was given in Monterrey, Mexico to dental and nondental personnel.
The urinary coproporphyrin levels before DMPS administration, which are indicative of renal mercury content, were quantitatively associated with the urinary mercury levels among the three study groups after DMPS administration.
Regression analysis showed that the coefficient of urinary mercury was statistically and adversely associated with complex attention (switching task), the perceptual motor task (symbol-digit substitution), symptoms and mood.

1.1.84.
Exposure to mercury vapor and impact on health in the dental profession in Sweden. =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3025977/
Abstract only
Coremessages:
The objective of the present study was to carry out detailed measurements of mercury exposure in the dental profession in Sweden, and to search for adverse health effects from such exposure
The average concentration of mercury in whole blood (B-Hg) was 18 nmol/L, in plasma (P-Hg) 5.1 nmol/L, and in urine (U-Hg) 3.0 nmol/mmol creatinin
In the Q16, the number of symptoms was statistically significantly higher in the dentistry group compared with an age- and gender-matched control group ($n = 44$).

1.1.85.
Chronic neurobehavioral effects of elemental mercury in dentists.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1039326/
FULL ACCCESS TO STUDY
Coremessages:

Neurobehavioural tests were performed by 98 dentists (mean age 32, range 24-49) exposed to elemental mercury vapour and 54 controls (mean age 34, range 23-50) with no history of occupational exposure to mercury.
In neurobehavioural tests measuring motor speed (finger tapping), visual scanning (trail making), visuomotor coordination and concentration (digit symbol), verbal memory (digit span, logical memory delayed recall), visual memory (visual reproduction, immediate and delayed recall), and visuomotor coordination speed (bender-gestalt time), the performance of the dentists was significantly worse than that of the controls.
The dentists scored 3.9 to 38.9% (mean 13.9%) worse in these tests.

1.1.86.
The relationship between mercury from dental amalgam and the cardiovascular system.=>
http://www.ncbi.nlm.nih.gov/pubmed/2270468
Abstract only
Coremessage:
Comparisons between subjects with and without amalgam showed amalgam-bearing subjects had significantly higher blood pressure, lower heart rate, lower hemoglobin, and lower hematocrit. Hemoglobin, hematocrit, and red blood cells were significantly lower when correlated to increased levels of urine mercury
The amalgam subjects had a greater incidence of chest pains, tachycardia, anemia, fatigue, tiring easily, and being tired in the morning.
The data suggest that inorganic mercury poisoning from dental amalgam does affect the cardiovascular system.

1.1.87.
Whole-body imaging of the distribution of mercury released from dental fillings into monkey tissues.=>
http://www.ncbi.nlm.nih.gov/pubmed/2227216
Abstract only
Coremessage:
Whole-body images of the monkey revealed that the highest levels of Hg were located in the kidney, gastrointestinal tract, and jaw. The dental profession's advocacy of silver amalgam as a stable tooth restorative material is not supported by these findings.

1.1.88.
Mercury released from dental "silver" fillings provokes an increase in mercury- and antibiotic-resistant bacteria in oral and intestinal floras of primates.=>
http://www.ncbi.nlm.nih.gov/pubmed/8280208
Abstract only
Coremessage:
Our findings indicate that mercury released from amalgam fillings can cause an enrichment of mercury resistance plasmids in the normal bacterial floras of primates. Many of these plasmids also carry antibiotic resistance, implicating the exposure to mercury from dental amalgams in an increased incidence of multiple antibiotic resistance plasmids in the normal floras of nonmedicated subjects.

1.1.89.
Diagnosis and treatment of metal-induced side-effects.=>
http://www.ncbi.nlm.nih.gov/pubmed/17261999
Abstract only
Coremessages:
We postulate that in vivo, metal ions activate T-cells, initiating systemic inflammation, which, through cytokines, affects the brain and hypothalamus-pituitary-adrenal axis.
We postulate that in vivo metal ions will activate T-cells starting systemic inflammation which, through cytokines affect the brain and hypothalamus-pituitary-adrenal (HPA) axis.
The removal of incompatible dental material (RID) resulted in long-term health improvement in the majority of patients.

1.1.90.
A significant dose-dependent relationship between mercury exposure from dental amalgams and kidney integrity biomarkers: a further assessment of the Casa Pia children's dental amalgam trial.=>
http://www.ncbi.nlm.nih.gov/pubmed/22893351
Abstract only
Coremessage:
The results of our study suggest that dental amalgams contribute to ongoing kidney damage at the level of

the PTs in a dose-dependent fashion.

1.1.91.
Mercury vapor from dental amalgams, an in vitro study.=>
http://www.ncbi.nlm.nih.gov/pubmed/2814828
Abstract only
Coremessage:
When the surfaces of the amalgams were brushed with a tooth paste, an instant increase of mercury vapor occurred.

1.1.92.
A prospective study of prenatal mercury exposure from maternal dental amalgams and autism severity.=>
http://www.ncbi.nlm.nih.gov/pubmed/19593333
Abstract only
Coremessage:
Subjects with > or =6 amalgams were 3.2-fold significantly more likely to be diagnosed with autism (severe) in comparison to ASD (mild) than subjects with < or =5 amalgams.
Dental amalgam policies should consider Hg exposure in women before and during the child-bearing age and the possibility of subsequent fetal exposure and adverse outcomes.

1.1.93.
Neurotoxicity of dental amalgam is mediated by zinc.=>
http://www.ncbi.nlm.nih.gov/pubmed/12598557
Absract only
Coremessage:
In this study, we used cortical cell cultures to show for the first time that amalgam causes nerve cell toxicity in culture

1.1.94.
Effect of heavy metals on immune reactions in patients with infertility.=>
http://www.ncbi.nlm.nih.gov/pubmed/12920793
Abstract only
Coremessage:
In patients with metal intolerance diagnosed by the Melisa test, metal ions released from the dental materials can represent a factor, that does not cause infertility but is able to influence it negatively.

1.1.95.
[Evaluation of the dose of mercury in exposed and control subjects].=>
http://www.ncbi.nlm.nih.gov/pubmed/12197266
Abstract only
Coremessage:
The results of the present research confirmed that the U-Hg excretion in non-occupationally exposed subjects is influenced by amalgam dental fillings.

1.1.96.
Amalgam use and mercury emission in the Netherlands]. =>
http://www.ncbi.nlm.nih.gov/pubmed/11924382
Abstract only
Coremessage:
The total mercury emission from dental amalgam to the environment in the Netherlands is at the estimate 500 kg a year, with a worst case maximum of 935 kg.

1.1.97.
Corrosion of dental amalgam and mercury vapor emission in vitro.=>
http://www.ncbi.nlm.nih.gov/pubmed/3201122
Abstract 0nly
Coremessage:
The results indicate that the composition of the saliva, oral hygiene and dietary factors may be determinants of Hg0 emission from amalgams in the oral cavity.

1.1.98.
Elimination of mercury from amalgam in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/11603820
Abstract only
Coremessage:
Even two months after the amalgam had been placed in rats teeth, the amount of mercury in the urine remained 4-5 times higher than in control, and 4 times higher than in rats exposed to diet containing powdered amalgam.

1.1.99.
A comparison of mental health of multiple sclerosis patients with silver/mercury dental fillings and those with fillings removed.=>
http://www.ncbi.nlm.nih.gov/pubmed/1496084
Abstract only
Coremessages
On the Beck Depression Inventory the multiple sclerosis subjects with amalgams suffered significantly more depression while their scores on the State-Trait Anger Expression Inventory indicated the former group also exhibited significantly more anger.
On the SCL-90 Revised, subjects with amalgam fillings had significantly more symptoms of depression, hostility, psychotism, and were more obsessive-compulsive than the patients with such fillings removed.

1.1.100.
Allergic disease, immunoglobulins, exposure to mercury and dental amalgam in Swedish adolescents.=>
http://www.ncbi.nlm.nih.gov/pubmed/9192218
Abstract only
Coremessage: --

1.1.101.
[Exposure to mercury in public dental clinics in Oslo--an occupational hazard evaluation].=>
http://www.ncbi.nlm.nih.gov/pubmed/268599
Abstract only
Coremessage:
In three of the offices surveyed, mercury vapour concentration exceeded the threshold limit value of 0.05 mg/m3, implying a lack of care in handling mercury.
The manner in preparation and handling of the amalgam were considered to be the predominant comtamination factor.
1.1.102.
Allergic disease, immunoglobulins, exposure to mercury and dental amalgam in Swedish adolescents.=>
http://www.ncbi.nlm.nih.gov/pubmed/9192218
Abstract only
Coremessages:
Some experimental animals also react with autoimmunity after low doses of inorganic mercury
. A recent study of 15-year-old adolescents demonstrated an association between immunoglobulin type A (IgA) and mercury concentration in plasma (P-Hg).

1.1.103.
[Amalgam. VII. Toxic effects of mercury from amalgam fillings].=>
http://www.ncbi.nlm.nih.gov/pubmed/11822123
Abstract only
Coremessage:
In addition, based on measurements of the internal exposure of the dental team, it can be concluded that mercury vapor seems to be no significant health risk for the personnel in most dental offices.
"dental amalgam" mercury site:ncbi.nlm.nih.gov google seite 21

1.1.104.
A prospective study on the incidence of mercury levels in dental students. 2. Correlation analysis=>
http://www.ncbi.nlm.nih.gov/pubmed/1817930
Abstract only
Coremessage:
The urinary mercury levels did not correlate with sex and smoking habits, but there was a correlation with the number of amalgam fillings

1.1.105.
Mercury and arsenic levels among Lebanese dentists: a call for action. =>
http://www.ncbi.nlm.nih.gov/pubmed/12677371
No abstract available

1.1.106.
Neuropsychological effects of low mercury exposure in dental staff in Erzurum, Turkey.=>
http://www.ncbi.nlm.nih.gov/pubmed/12731695
Abstract only
Coremessage:
The dental staff group had higher whole blood (B-Hg) and urine (U-Hg) Hg levels than the control group
U-Hg had an inverse relationship with logical memory (in WMS-R test) and total retention score (in VTMP test), and a positive relationship with increased scores of Anxiety and Psychoticism (in SCL-90-R).

1.1.107.
Behavioral effects of low-level exposure to elemental Hg among dentists=>
http://www.ncbi.nlm.nih.gov/pubmed/7760775
Abstract only
Coremessages:
Exposure thresholds for health effects associated with elemental mercury (Hg degree) exposure were examined by comparing behavioral test scores of 19 exposed (mean urinary Hg = 36 micrograms/l) with those of 20 unexposed dentists.
Individual tests evaluating cognitive and motor function changed in the expected directions but were not significantly associated with urinary Hg.
However, the pooled sum of rank scores for combinations of tests within domains were significantly associated with urinary Hg, providing evidence of subtle preclinical changes in behavior associated with Hg exposure.
Coproporphyrin, one of three urinary porphyrins altered by mercury exposure, was significantly associated with deficits in digit span and simple reaction time.

1.1.108.
Mercury from maternal "silver" tooth fillings in sheep and human breast milk. A source of neonatal exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/9164660
Abstract om
Coremessage:
Results from the animal studies showed that, during pregnancy, a primary fetal site of amalgam Hg concentration is the liver, and, after delivery, the neonatal lamb kidney receives additional amalgam Hg from mother's milk.

1.1.109.
Blood mercury levels of dental students and dentists at a dental school =>
http://www.ncbi.nlm.nih.gov/pubmed/11720018
Abstract only
Coremessages:
There were statistically significant increases ($p<0.001$) in plasma mercury concentration between measurements in all groups at the end of the academic year.
Red cell mercury levels were also consistently elevated
Although the highest levels of mercury were recorded in persons working with amalgam, increased levels were also found in subjects working in the teaching classrooms

1.1.110.
Urinary porphyrin profiles as a biomarker of mercury exposure: studies on dentists with occupational exposure to mercury vapor.=>
http://www.ncbi.nlm.nih.gov/pubmed/8230299
Abstract only
Coremessages:
Porphyrins are formed as intermediates in the biosynthesis of heme. I
Mercury selectively alters porphyrin metabolism in kidney proximal tubule cells, leading to an altered urinary porphyrin excretion pattern.
Urinary porphyrin concentrations were measured in dentists participating in the Health Screening Programs conducted during the 1991 and 1992 annual meetings of the American Dental Association and compared

with urinary mercury levels measured in the same subjects.
among dentists with urinary mercury in excess of 20 micrograms/L, mean urinary concentrations of four- and five-carboxyl porphyrins as well as of precoproporphyrin were elevated three to four times those of unexposed subjects

1.1.111.
Dental amalgam affects urinary selenium excretion.=>
http://www.ncbi.nlm.nih.gov/pubmed/11899021
Abstract only
Coremessage:
Individuals with amalgam excreted less selenium (36.4 microg, median value) over 24 hours than those without amalgam (47.5 microg) (p = 0.016).

1.1.112.
The dental amalgam mercury controversy--inorganic mercury and the CNS; genetic linkage of mercury and antibiotic resistances in intestinal bacteria.=>
http://www.ncbi.nlm.nih.gov/pubmed/7716785
Abstract only
Coremessage:
Mercury (Hg) vapor exposure from dental amalgam has been demonstrated to exceed the sum of all other exposure sources
In monkeys we show that Hg, specifically from amalgam, will enrich the intestinal flora with Hg-resistant bacterial species which in turn also become resistant to antibiotics.

1.1.113.
Linkage of a novel mercury resistance operon with streptomycin resistance on a conjugative plasmid in Enterococcus faecium.=>
http://www.ncbi.nlm.nih.gov/pubmed/15907536
Abstract only
Coremessages:
It has been shown that the mercury in dental amalgam and other environmental sources can select for mercury resistant bacteria and that this can lead to an increase in resistance to antibiotics.
In this study we have cloned and sequenced the mer operon from an Enterococcus faecium strain which was resistant to mercury, tetracycline, and streptomycin=>

1.1.114.
Evidence that mercury from silver dental fillings may be an etiological factor in smoking. =>
http://www.ncbi.nlm.nih.gov/pubmed/8516784
Abstract only
Coremessage:
The amalgam group had 2.5-times more smokers per group than the non-amalgam group, which was highly significant.

1.1.115.
[Mercury exposure of the population. IV. Mercury exposure of male dentists, female dentists and dental aides].=>
http://www.ncbi.nlm.nih.gov/pubmed/1290562
Abstract only
Coremessage:
Following administration of Dimaval a significant increase of mercury excretion was observed in both groups

1.1.116.
Activation of the immune system and systemic immune-complex deposits in Brown Norway rats with dental amalgam restorations.=>
http://www.ncbi.nlm.nih.gov/pubmed/9649170
Abstract only
Coremessage:
We conclude that dental amalgam restorations release substantial amounts of their elements, which accumulate in the organs and which, in genetically susceptible rats, give rise to activation of the immune system and systemic IC deposits.

1.1.117.
Mercury in saliva and the risk of exceeding limits for sewage in relation to exposure to amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/12530606
Abstract only
Coremessage:
These results demonstrate that humans, especially in populated areas, can be a significant source of mercury pollutants. As a consequence of mercury release, bacteria may acquire mercury resistance, as well as resistance to other antimicrobial agents, thus resulting in failure of antibiotic treatment.

1.1.118.
Effect of dental amalgam on gene expression profiles in rat cerebrum, cerebellum, liver and kidney.=>
http://www.ncbi.nlm.nih.gov/pubmed/22688007
Abstract only
Coremessage:
Out of 26,962 rat genes, mercury vapor was found to increase the expression of... (several genes)

1.1.119.
[Exposure to mercury in the population. II. Mercury release from amalgam fillings].=>
http://www.ncbi.nlm.nih.gov/pubmed/2080964
Abstract only
Coremessage:
Urinary levels of mercury (HgU) were measured in 93 males and females aged 18-63 years. Subjects with amalgam fillings (n = 72) had, on average, significantly higher levels of mercury in urine (mean = 0.57 microgram Hg/l and 0.79 microgram Hg/g creatinine, respectively) than subjects without amalgam fillings

1.1.120.
Urinary mercury concentrations in Finnish dentists.=>
http://www.ncbi.nlm.nih.gov/pubmed/2594745
Abstract only
Coremessage:
The factor most closely related to high urinary mercury levels was use of amalgam by the dentist. Dentists who did not use amalgam had urinary mercury concentrations similar to those in Finns not occupationally exposed to mercury.

1.1.121.
The Chemical Forms of Mercury in Aged and Fresh Dental Amalgam Surfaces=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2866173/
FULL ACCESS TO STUDY
Coremessages:
We have explored the use of electron-yield Hg LIII X-ray absorption spectroscopy to characterize the chemical nature of dental amalgam surfaces.
We find that the method is practical, and that it shows extensive mercury depletion in the surface of the aged amalgam with significant differences between old and fresh amalgam surfaces.

1.1.122.
Psychometric evidence that mercury from silver dental fillings may be an etiological factor in depression, excessive anger, and anxiety.=>
http://www.ncbi.nlm.nih.gov/pubmed/8153237
Abstract only
Coremessages:
. Women with amalgams had significantly higher scores and reported more symptoms of fatigue and insomnia
Anger scores from the State-Trait Anger Expression Inventory showed that the women with amalgams had statistically significantly higher mean scores on expressing anger without provocation and experiencing more intense angry feelings

1.1.123.
Mercury from dental "silver" tooth fillings impairs sheep kidney function. =>
http://www.ncbi.nlm.nih.gov/pubmed/1928419
Abstract only
Coremessages:
Previous studies show that when 12 such fillings are placed in sheep teeth, the kidneys will concentrate

amalgam Hg at levels ranging from 5 to 10 micrograms Hg/g renal tissue 4-20 wk after placement.
After amalgam placement urine concentration of albumin decreased from 93.0 +/- 20.5 to 30.1 +/- 15.3 mg/l and urine Na+ concentration increased steadily from 24.8 +/- 7.7 to 82.2 +/- 20.3 mmol/l at 60 days.

1.1.124.
Dental "silver" tooth fillings: a source of mercury exposure revealed by whole-body image scan and tissue analysis.=>
http://www.ncbi.nlm.nih.gov/pubmed/2636872
Abstract only
Coremessage:
This investigation demonstrates that when radioactive 203Hg is mixed with dental Hg/silver fillings (amalgam) and placed in teeth of adult sheep, the isotope will appear in various organs and tissues within 29 days.

1.1.125.
Cerebrospinal fluid protein changes in multiple sclerosis after dental amalgam removal. =>
http://www.ncbi.nlm.nih.gov/pubmed/9727079
Abstract only
Coremessages:
This study documents objective biochemical changes following the removal of these fillings along with other dental materials
The dramatic changes in photolabeling of cerebrospinal fluid (CSF) proteins following these dental interventions suggest CSF photolabeling may serve as an objective biomarker for monitoring MS.

1.1.126.
Minor changes in serum levels of cytokines after removal of amalgam restorations=>
http://www.ncbi.nlm.nih.gov/pubmed/22475563
Abstract only
Coremessage:
After amalgam removal a decrease towards the median value of the reference group was found for GM-CSF, IL-8, and IL-7. In conclusion, removal of all dental amalgam restorations and replacement with other dental restorative materials was associated with decreased concentrations of Th1-type proinflammatory markers in serum.

1.1.127.
Changes in health complaints after removal of amalgam fillings=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3229679/
FULL ACCESS TO STUDY
Coremessage:
Comparisons between the groups showed that reductions in intra-oral and general health complaints in the treatment group were significantly different from the changes in the reference group. The mechanisms behind this remain to be identified.

1.1.128.
Traces of mercury in organs from primates with amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/2115006
Abstract only
Coremessages:
It was found that amalgam fillings (total, 0.7-1.2 g) caused deposition of mercury in the following tissues: spinal ganglia, anterior pituitary, adrenal, medulla, liver, kidneys, lungs, and intestinal lymph glands.
In monkeys with maxillary silver amalgam implants (total, 0.1-0.3 g), mercury was found in the same organs except for liver, lungs, and intestinal lymph glands.
These results strongly support what has been suggested previously that dental fillings in primates cause absorption of mercury released from amalgam fillings through lungs and intestinal tract, and that depending on exposure mercury is distributed to most organs and will eventually be found in the central nervous system.

1.1.129.
Mercury deposits in neurons of the trigeminal ganglia after insertion of dental amalgam in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/8043992
Abstract only
Coremessage:

Within the trigeminal ganglia, nerve cells with mercury deposits were observed in seven out of 12 rats

1.1.130.
Impact of nocturnal bruxism on mercury uptake from dental amalgams.=>
http://www.ncbi.nlm.nih.gov/pubmed/9249192
Abstract only
Coremessage:
In a regression model with bruxism as the only explanatory variable, no significant effect of bruxism was found, but when the number of amalgam fillings, chewing gum use, and other background variables were taken into account, there was a limited impact of bruxism on Hg in plasma.

1.1.131.
Effect of teeth amalgam on mercury levels in the colostrums human milk in Lenjan.=>
http://www.ncbi.nlm.nih.gov/pubmed/21494835
Abstract only
Coremessage:
Obtained results showed that only dental amalgam significantly increased the mercury level in human milk ($p < 0.001$)

1.1.132.
Removal of dental amalgam decreases anti-TPO and anti-Tg autoantibodies in patients with autoimmune thyroiditis.=>
http://www.ncbi.nlm.nih.gov/pubmed/16804512
Abstract only
Coremessage:
Removal of mercury-containing dental amalgam in patients with mercury hypersensitivity may contribute to successful treatment of autoimmune thyroiditis

1.1.133.
A model for recording mercury release from an amalgam surface.=>
http://www.ncbi.nlm.nih.gov/pubmed/4052550
Abstract only
Coremessage:
After brushing the surface of the amalgam during two subsequent periods, in a manner similar to toothbrushing, an increase in corrosion current was measured indicating the removal of loosely bound corrosion products.

1.1.134.
Treatment of health complaints attributed to amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/18362317
Abstract only
Coremessage:
Both removal groups showed a significant decrease in steady-state levels of inorganic mercury compared with the no-removal group. Thus, all 3 interventions were associated with clinically relevant improvements.

1.1.135.
Dentinal and pulpal uptake of mercury from lined and unlined amalgam restorations in minipigs.=>
http://www.ncbi.nlm.nih.gov/pubmed/9298366 =>
Abstract only
Coremessages:
Silver-enhanced mercury was found in all teeth with amalgam, whereas teeth with composites were devoid of mercury
Mercury could be traced in the odontoblast processes, in the body of odontoblasts, and on rare occasions in the nerve tissue of the pulp from lined and from unlined amalgam restorations.
The present study thus demonstrates transport of mercury through dentinal tubules to the pulp

1.1.136.
Mercury content in amalgam tattoos of human oral mucosa and its relation to local tissue reactions.=>
http://www.ncbi.nlm.nih.gov/pubmed/9527359
Abstract only

1.1.137.
Inorganic mercury and methylmercury in placentas of Swedish women.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1240842/
FULL ACCESS TO STUDY
Coremessage:
I-Hg levels in placenta increased with an increasing number of maternal dental amalgam fillings ($p < 0.001$).

1.1.138.
Mercury in biological fluids after amalgam removal.=>
http://www.ncbi.nlm.nih.gov/pubmed/9539465
Abstract only
Coremessage:
It is concluded that the process of removing amalgam fillings can have a considerable impact on Hg levels in biological fluids

1.1.139.
Heavy-metal mitogenesis: Zn++ and Hg++ induce cellular cytotoxicity and interferon production in murine T lymphocytes.=>
http://www.ncbi.nlm.nih.gov/pubmed/2448223
Abstract only
Coremessage:
Both Zn++ and Hg++ activated splenic lymphocytes to display lectin-dependent cytotoxicity and to produce acid-labile interferon.

1.1.140.
Maternal amalgam dental fillings as the source of mercury exposure in developing fetus and newborn.=>
http://www.ncbi.nlm.nih.gov/pubmed/17851449/
Abstract only
Coremessage:
Levels of Hg in the cord blood were significantly associated with the number of maternal amalgam fillings (rho=0.46, P<0.001) and with the number of years since the last filling (rho=-0.37, P<0.001); these associations remained significant after adjustment for maternal age and education.
Dental amalgam fillings in girls and women of reproductive age should be used with caution, to avoid increased prenatal Hg exposure.

1.1.141.
Dissolution of mercury from amalgam into saline solution.=>
http://www.ncbi.nlm.nih.gov/pubmed/3476540
Abstract only
Coremessage:
The study revealed that as high as 55% of the mercury ions liberated from the amalgams and the amalgam phases was adsorbed onto the walls of the vials in which the specimens were aged.

1.1.142.
Orofacial granulomatosis associated with hypersensitivity to dental amalgam.=>
http://www.ncbi.nlm.nih.gov/pubmed/21684771
Abstract only
Coremessage:
After the removal of the suspected allergens, all patients experienced recovery within 1 month

1.1.143.
Does mercury from amalgam restorations constitute a health hazard?=>
http://www.ncbi.nlm.nih.gov/pubmed/2270464
Abstract only
Coremessages:
Amalgam restorations continuously emit mercury vapour, which is absorbed in considerable quantities via the lungs
Animal studies suggest the possibility of immune system reactions to mercury, i.e. development of autoimmunity, that are not primarily dose-dependent, but rather depend on genetic susceptibility.
From a toxicological point of view, amalgam is an unsuitable material for dental restorations.

1.1.144.
Urinary mercury excretion in dental personal =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1008416/pdf/brjindmed00075-0068.pdf
FULL ACCESS TO STUDY
Coremessages:
The potential health hazard in the extensive use of mercury in dental surgeries is a current cause for concern (Journal ofthe American Dental Association,1971;
Table 1 gives a summary of the results. In both groups of workers the DSAs had a higher mean and greater range of urinary mercury concentrations than the dentists; the NHS workers had similarly,
in both dentists and DSAs, a higher mean and greater range of concentrations. Legende: DSA = dental surgery assistants NHS = National Health Service (Dentists)

1.1.145.
Mercury burden of human fetal and infant tissues.=>
http://www.ncbi.nlm.nih.gov/pubmed/7957411
Abstract only
Coremessage:
The Hg-K (n = 38) and Hg-L (n = 40) of fetuses and Hg-K (n = 35) and Hg-C (n = 35) of older infants (11-50 weeks of life) correlated significantly with the number of dental amalgam fillings of the mother.

1.1.146.
The effect of occupational exposure to the mercury vapour on the fertility of female dental assistants.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3025977/
FULL ACCESS TO STUDY
Coremessages:
Women with high occupational exposure to mercury were less fertile than unexposed controls.
The fecundability (probability of conception each menstrual cycle) of women who prepared 30 or more amalgams per week and who had five or more poor mercury hygiene factors was only 63% of that for unexposed women (95% CI 42%-96%) after controlling for covariates

1.1.147.
30-year follow-up of residual effects on New Zealand School Dental Nurses, from occupational mercury exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/17615119
Abstract only
Coremessages:
This paper reports possible residual adverse effects from occupational mercury exposure in dentistry
Thirty years ago, the all-women exposed group worked with both silver and copper amalgam filling material without protective gloves or a ventilation system, resulting in chronic mercury exposure.
In general, the study suggests that acute symptoms from mercury exposure may be reversible, while some residual health effects may be becoming more of a concern with the women's increasing age.

1.1.148.
The association between a genetic polymorphism of coproporphyrinogen oxidase, dental mercury =>
http://www.ncbi.nlm.nih.gov/pubmed/16343843
Abstract only
Coremessages:
We previously described a polymorphism in exon 4 of the gene encoding the heme biosynthetic pathway enzyme, coproporphyrinogen oxidase (CPOX4), which significantly modifies the effect of mercury exposure on urinary porphyrin excretion in humans.
Here, we examined potential consequences of this polymorphism
These exploratory findings suggest that the CPOX4 polymorphism may affect susceptibility for specific neurobehavioral functions associated with mercury exposure in human subjects.

1.1.149.
A case of high mercury exposure from dental amalgam=>
http://www.ncbi.nlm.nih.gov/pubmed/8831068
Abstract only
Coremessages:
Analysis of mercury in plasma and urine showed unexpectedly high concentrations, 63 and 223 nmol/l, respectively.
Following removal of the amalgam fillings, the urinary excretion of mercury became gradually normalized,

and her symptoms declined.

1.1.150.
Methylmercury, Amalgams, and Children's Health: Björnberg et al. Respond=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1392266/
FULL ACCESS TO STUDY
Coremessage:
In the study by Leistevuo et al. (2001), 15–18% of total mercury in saliva (5–12.5 nmol/L) was organic in a group of subjects with amalgam fillings.

1.1.151.
High mercury emissions from dental clinics despite amalgam separators =>
http://www.ncbi.nlm.nih.gov/pubmed/16054673
Abstract only
Coremessages:
All waste water was collected for four consecutive working days, initially at ordinary operating conditions and a second time after a thorough revision and cleaning of the discharge system.
The study also indicates that banning Hg in dentistry is the one long-term way to stop Hg emissions from dental amalgam.

1.1.152.
Mercury poisoning from dental amalgam through a direct nose-brain transport.=>
http://www.ncbi.nlm.nih.gov/pubmed/2566770
NO Abstract available.

1.1.153.
Skeletal muscle abnormalities associated with occupational exposure to mercury vapours. =>
http://www.ncbi.nlm.nih.gov/pubmed/10963110
Abstract only
Coremessages:
Dental personnel are frequently exposed to inhalation of metallic mercury vapours.
All of them presented symptoms of chronic mercury poisoning.
Selective atrophy of type IIB muscle fibres was found in patients, and in one of them there was fibre grouping.
Most of the muscles showed increased fibre area per capillary.
. Some capillaries were altered, showing endothelial infoldings into the lumen, thickened basement membrane and partial or total occlusion.

1.1.154.
Long-term dissolution of mercury from a non-mercury-releasing amalgam.
http://www.ncbi.nlm.nih.gov/pubmed/1860296
Abstract only
Coremessages:
This study examined the mercury release from a "non-mercury-releasing" dental amalgam, Composil, over a 104-week period.
This study showed that Composil releases mercury in quantities that far exceed those detected in other amalgam systems.

1.1.155.
Dental amalgam as one of the risk factors in autoimmune diseases.=>
http://www.ncbi.nlm.nih.gov/pubmed/12743535
Abstract only
Coremessage:
Results imply that, in some patients with thyroiditis, mercury from dental amalgam can stimulate the production of antinuclear antibodies. Dental amalgam may be a risk factor in some patients with autoimmune disease.

1.1.156.
Health and neuropsychological functioning of dentists=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1740287/
FULL ACCESS TO STUDY
Coremessages:

Dentists had, on average, urinary mercury concentrations over four times that of control subjects, but all but one dentist had urinary mercury below the Health and Safety Executive health guidance value.
Dentists were significantly more likely than control subjects to have had disorders of the kidney and memory disturbance.
Differences were found between the psychomotor performance of dentists and controls after adjusting for age and sex, but there was no significant association between changes in psychomotor response and mercury concentrations in urine, hair, or nails.

1.1.157.
Recovery from mercury-induced burning mouth syndrome due to mercury allergy.=>
http://www.ncbi.nlm.nih.gov/pubmed/15473333
Abstract only
Coremessage:
Full recovery from BMS and complete remission of systemic dermatitis were achieved after the mercury tooth filling was removed. Mercury is thought to be an allergen implicated in BMS as well as in the systemic reactivation of allergic contact dermatitis.

1.1.158.
The release, tissue distribution and excretion of mercury from experimental amalgam tattoos.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2013129/
FULL ACCESS TO STUDY
Coremessage:
Raised mercury levels were detected in the blood, bile, kidneys, liver, spleen and lungs of implanted animals; by far the highest concentrations were found in the renal cortex. Mercury was excreted with the urine and, to a lesser extent, the faeces. The pattern of mercury redistribution resembled that seen following chronic exposure to mercuric compounds.

1.1.159.
Chronic low-level mercury exposure and neuropsychological functioning.=>
http://www.ncbi.nlm.nih.gov/pubmed/3805254
Abstract only
Coremessages:
Workers with elevated mercury levels scored significantly less well on the Recurrent Figures
Chronic subtoxic levels of inorganic mercury appear to produce mild changes in short-term nonverbal recall and heightened distress generally, and particularly in categories of obsessive compulsion, anxiety and psychoticism,

1.1.160.
Urinary mercury after administration of 2,3-dimercaptopropane-1-sulfonic acid: correlation with dental amalgam score.=>
http://www.ncbi.nlm.nih.gov/pubmed/1563599
Absrct only
Coremessage:
Linear regression analysis indicated a highly significant positive correlation between the mercury excreted in the urine 2 h after DMPS administration and the dental amalgam scores.

1.1.161.
Amalgam dental fillings and hearing loss.=>
http://www.ncbi.nlm.nih.gov/pubmed/19085401
Abstract only
Coremessage:
he results suggest an association between more amalgam fillings and poorer thresholds at higher frequencies, which could contribute to presbyacusis in developed countries. This provides further argument for the use of amalgams to be phased out where suitable alternatives exist.

1.1.162.
Changed clinical chemistry pattern in blood after removal of dental amalgam and other metal alloys supported by antioxidant therapy.=>
http://www.ncbi.nlm.nih.gov/pubmed/17916968
Abstract only
Coremessage:

The variables serum lactate dehydrogenase (serum LDH) and serum sodium differed significantly both when comparing Control with Before ($p < 0.01$) and Before with After ($p < 0.01$). The variables white blood cell count (WBC), blood neutrophil count, blood eosinophil count, blood basophil count, blood lymphocyte count, blood monocyte count, serum potassium, and serum creatinine differed in the Before/After test ($p < 0.05$).

1.1.163.
Removal of dental amalgam and other metal alloys supported by antioxidant therapy alleviates symptoms and improves quality of life in patients with amalgam-associated ill health.=>
http://www.ncbi.nlm.nih.gov/pubmed/12500173
Abstract only
Coremessage:
The hypothesis that metal exposure from dental amalgam can cause ill health in a susceptible part of the exposed population was supported =>

1.2. DENTAL AMALGAM & DEPRESSION AND ANXIETY

1.2.1.
Psychometric evidence that mercury from silver dental fillings may be an etiological factor in depression, excessive anger, and anxiety.=>
http://www.ncbi.nlm.nih.gov/pubmed/8153237
Abstract only
Coremessages:
. Women with amalgams had significantly higher scores and reported more symptoms of fatigue and insomnia
Anger scores from the State-Trait Anger Expression Inventory showed that the women with amalgams had statistically significantly higher mean scores on expressing anger without provocation and experiencing more intense angry feelings

1.2.2.
Physical and mental problems attributed to dental amalgam fillings: a descriptive study of 99 self-referred patients compared with 272 controls.=>
http://www.ncbi.nlm.nih.gov/pubmed/9021864
Abstract only
Coremesaage:
The dental amalgam sample reported significantly more physical symptoms from all body regions.

1.2.3.
Results of dental amalgam removal and mercury detoxification using DMPS and neural therapy.=>
http://www.ncbi.nlm.nih.gov/pubmed/10895513
Abstract only
Coremessages:
The most distressing symptoms were headache and backache, fatigue, and memory and concentration problems
Headache and backache responded best to treatment, but all symptoms showed considerable improvement on average. Of the respondents, 78% reported that they were either satisfied or very satisfied with the results of treatment, and 9.5% reported that they were disappointed.

1.2.4.
Changes in health complaints after removal of amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3229679/
FULL ACCESS TO STUDY
Coremessage:
In the treatment group, there were significant reductions in intra-oral and general health complaints from

inclusion into study to the 3-year follow-up. In the reference group, changes in the same period were not significant.

1.2.5.
Personality Traits in (mercury) Miners with Past Occupational Elemental Mercury Exposure =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1367847/
FULL ACCESS TO STUDY
1.2.6.
Defensive characteristics in individuals with amalgam illness as measured by the percept-genetic method Defense Mechanism Test.=>
http://www.ncbi.nlm.nih.gov/pubmed/8811140
Abstract only
Coremessage:
The objective was to try to distinguish the group with amalgam illness from the non-patient group by means of the DMT. The results showed that it was possible to distinguish the two groups significantly from each other.
The most characteristic traits of the patient group were a general lateness in perception and few emotional responses compared with the non-patient group and, especially, an inability to perceive the aggressive component in the stimulus picture.
The DMT seems to be a powerful method in the effort to understand the mechanisms underlying the problems of amalgam illness.

1.3. DENTAL AMALGAM & MCS (MULTIPLE CHEMICAL SENSITIVITY)

1.3.1.
The Search for Reliable Biomarkers of Disease in Multiple Chemical Sensitivity and Other Environmental Intolerances=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3155329/
FULL ACCESS TO STUDY
Coremessages:
(Mercury) Indeed, tissue contamination through dental root and jaw bone transmission has also been proven
Inorganic Hg contained in dental amalgam is transformed into organic methyl-mercury by residential microorganisms in the mouth and in the gastrointestinal tract
Chronic Hg intoxication has been connected with a wide variety of symptoms, ranging from cutaneous and oral mucosa signs [58] with increased infection susceptibility, to persistent fatigue with joint and muscle pain, neurological symptoms [59] and vegetative disorders, headache, migraine, lack of concentration, low memory capacity, depression, sleeping disorders.
Clinical reports have linked dental amalgam implants with prevailing skin and mucosa manifestations of autoimmune diseases
Few rigorous evidences have also linked dental amalgam to increased risk of multiple sclerosis,

1.4. DENTAL AMALGAM & MERCURY IN BRAIN AND NERVE TISSUE

1.4.1.
Correlation of dental amalgam with mercury in brain tissue.=>
http://www.ncbi.nlm.nih.gov/pubmed/3480359
Abstract only
Coremessage:
Data from this project demonstrate a positive correlation between the number of occlusal surfaces of dental amalgam and mercury levels in the brain (p less than .0025 in white matter).

1.4.2.
Dental amalgam and mercury levels in autopsy tissues: food for thought.=>
http://www.ncbi.nlm.nih.gov/pubmed/16501347
Abstract only
Coremessage:
Total mercury levels were significantly higher in subjects with a greater number of occlusal amalgam surfaces (>12) compared with those with fewer occlusal amalgams (0-3) in all types of tissue (all P < or = 0.04).
Mercury levels were significantly higher in brain tissues compared with thyroid and kidney tissues in subjects with more than 12 occlusal amalgam fillings (all P < or = 0.01) but not in subjects with 3 or less occlusal amalgams (all P > or = 0.07).

1.4.3.
Mercury in human brain, blood, muscle and toenails in relation to exposure: an autopsy study =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2098763/
FULL ACCESS TO STUDY
Coremessage:
I-Hg in both blood and occipital cortex, as well as total-Hg in pituitary and thyroid were strongly associated with the number of dental amalgam surfaces at the time of death.

1.4.4.
Silver concentrations in human tissues. their dependence on dental amalgam and other factors =>
http://www.ncbi.nlm.nih.gov/pubmed/8825980
Abstract only
Coremessage:
In this sub-group statistically significant correlations were found between the number of teeth with dental amalgam and the Ag concentrations in the cerebral cortex and the liver

1.4.5.
Relation between mercury and selenium in pituitary glands of dental staff. =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009861/pdf/brjindmed00138-0071.pdf
FULL ACCES TO STUDY
Coremessage:
Analyses of mercury concentration have shown high concentrations of mercury in the pituitary glands of dental staff.'

1.4.6.
Mercury accumulation in tissues from dental staff and controls in relation to exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/2603127
Abstract only
Coremessage:
The results revealed high mercury concentrations (median 815, range 135-4,040 micrograms Hg/kg wet weight) in pituitaries from the dental staff cases compared to controls (N = 23, median 23 range 6-1, 170 micrograms Hg/kg).

1.4.7.
Dentists, dental nurses, and brain tumours =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1339649/pdf/bmjcred00224-0024.pdf
FULL ACCESS TO STUDY
Coremessage:
The table shows that among dentists and dental nurses glioblastoma was about twice as common as expected.

1.4.8.
MERCURY IN PITUITARY GLANDS OF DENTISTS (Nylander)=>
http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(86)92395-0/fulltext
NO ACCESS TO STUDY , NO ABSTRACT

1.4.9.
Mercury concentrations in the human brain and kidneys in relation to exposure from dental amalgam fillings.=>
http://www.ncbi.nlm.nih.gov/pubmed/3481133

Abstract only
Coremessages:
. Results from 34 individuals showed a statistically significant regression between the number of tooth surfaces containing amalgam and concentration of mercury in the occipital lobe cortex
It is concluded that the cause of the association between amalgam load and accumulation of mercury in tissues is the release of mercury vapour from amalgam fillings

1.4.10.
Uptake of inorganic mercury in the olfactory bulbs via olfactory pathways in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/9600806
Abstract only
Coremessages:
We propose that in these rats the mercury is taken up from the blood into the olfactory neurons and then moves along the axons to their terminations in the olfactory bulbs.
In humans a continuous exposure of the nasal cavity to mercury vapor (Hg0), released from amalgam fillings and oxidized to Hg2+ in the olfactory mucosa, as well as a potential uptake of Hg2+ in the olfactory neurons from the blood, may lead to considerable concentrations of the metal in the olfactory bulbs.

1.5. DENTAL AMALGAM , IMMUNITY & AUTOIMMUNITY

1.5.1.
Dental amalgam as one of the risk factors in autoimmune diseases.=>
http://www.ncbi.nlm.nih.gov/pubmed/12743535
Abstract only
Coremesssage:
Increased production of SSB/La autoantibodies in the media following stimulation of peripheral blood lymphocytes with HgCl2 was found in all cases.
Results imply that, in some patients with thyroiditis, mercury from dental amalgam can stimulate the production of antinuclear antibodies

1.5.2.
Removal of dental amalgam decreases anti-TPO and anti-Tg autoantibodies in patients with autoimmune thyroiditis.=>
http://www.ncbi.nlm.nih.gov/pubmed/16804512
Abstract only
Coremessage:
patients with mercury hypersensitivity who underwent amalgam replacement (Group IIA) showed a significant decrease in the levels of both anti-Tg (p=0.001) and anti-TPO (p=0.0007) autoantibodies.

1.5.3.
The beneficial effect of amalgam replacement on health in patients with autoimmunity. =>
http://www.ncbi.nlm.nih.gov/pubmed/15349088
Abstract only
Coremessage:
Results of lymphocyte reactivity measured with MELISA indicate that in vitro reactivity after the replacement of dental amalgam decreased significantly to inorganic mercury, silver, organic mercury and lead. Out of 35 patients, 25 patients (71%) showed improvement of health.

1.5.4.
Acute glomerulonephritis, Henoch-Schönlein purpura and dental amalgam in Swedish children: a case-control study.=>
http://www.ncbi.nlm.nih.gov/pubmed/8931347
Abstract only
Coremessage:

1.5.5.
Adverse immunological effects and autoimmunity induced by dental amalgam and alloy in mice.
http://www.ncbi.nlm.nih.gov/pubmed/7958626

Abstract only
Coremessages:
10 weeks of low-dose and 6 months of high-dose amalgam implantation strongly increased mitogen-induced T and B cell proliferation,
In conclusion, dental amalgam implantation in a physiological body milieu causes chronic stimulation of the immune system with induction of systemic autoimmunity in genetically sensitive mice.

1.5.6.
Mercury and nickel allergy: risk factors in fatigue and autoimmunity=>
http://www.ncbi.nlm.nih.gov/pubmed/11462117
Abstract only
Coremessage:
To evaluate clinical relevance of positive in vitro findings, the replacement of amalgam with metal-free restorations was performed in some of the patients. At a six-month follow-up, patients reported considerably alleviated fatigue and disappearance of many symptoms previously encountered; in parallel, lymphocyte responses to metals decreased as well.

1.5.7.
Linking mercury amalgam to autoimmunity.=>
http://www.ncbi.nlm.nih.gov/pubmed/20080446
No abstract available

1.6. DENTAL AMALGAM & MS (MULTIPLE SCLEROSIS)

1.6.1
Evidence that mercury from silver dental fillings may be an etiological factor in multiple sclerosis.=>
http://www.ncbi.nlm.nih.gov/pubmed/8191275
Abstract only
Coremessages:
MS subjects with amalgams were found to have significantly lower levels of red blood cells, hemoglobin and hematocrit compared to MS subjects with amalgam removal.
Thyroxine levels were also significantly lower in the MS amalgam group and they had significantly lower levels of total T Lymphocytes and T-8 (CD8) suppressor cells.
The MS amalgam group had significantly higher blood urea nitrogen and lower serum IgG.
Hair mercury was significantly higher in the MS subjects compared to the non-MS control group

1.6.2.
Cerebrospinal fluid protein changes in multiple sclerosis after dental amalgam removal. =>
http://www.ncbi.nlm.nih.gov/pubmed/9727079
Abstract only
Coremessages:
This study documents objective biochemical changes following the removal of these fillings along with other dental materials
The dramatic changes in photolabeling of cerebrospinal fluid (CSF) proteins following these dental interventions suggest CSF photolabeling may serve as an objective biomarker for monitoring MS.

1.6.3.
A comparison of mental health of multiple sclerosis patients with silver/mercury dental fillings and those with fillings removed.=>
http://www.ncbi.nlm.nih.gov/pubmed/1496084
Abstract only
Coremessages:
In the SCL-90 Revised, subjects with amalgam fillings had significantly more symptoms of depression, hostility, psychotism, and were more obsessive-compulsive than the patients with such fillings removed.
On a questionnaire containing 18 mental health symptoms multiple sclerosis subjects with amalgam fillings reported a history of 43% more symptoms than those without amalgam fillings over the past 12 months.
These data suggested that the poorer mental health status exhibited by multiple sclerosis subjects with dental amalgam fillings may be associated with mercury toxicity from the amalgam.

1.6.4.
Epidemiology, etiology, and prevention of multiple sclerosis. Hypothesis and fact=>
http://www.ncbi.nlm.nih.gov/pubmed/6837537
Abstract only
Coremessages:
Slow, retrograde seepage of ionic mercury from root canal or Class V amalgam fillings inserted many years previously, recurrent caries and corrosion around filling edges, and the oxidizing effect of the purulent response may lead to multiple sclerosis in middle age.
Clinical and epidemiologic data also suggest that a second heavy metal, lead, may operate almost interchangeably with mercury.

1.6.5.
Health effects of dental amalgam exposure: a retrospective cohort study.=>
http://www.ncbi.nlm.nih.gov/pubmed/15155698
Abstract only
Coremessages:
Of conditions allegedly associated with amalgam, multiple sclerosis had an adjusted hazard ratio (HR) of 1.24 (95% CI: 0.99, 1.53, P = 0.06),

1.6.6.
Comperative epidemiology of multiple sclerosis and dental caries.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1060938/
FULL ACCESS TO STUDY
Coremessages:
The geographical distribution and other epidemiological characteristics of multiple sclerosis (MS) are compared with those of dental caries.
The rates of death due to MS in Australian states are linearly related to the numbers of decayed, missing, and filled (DMF) teeth found in individuals from those states (r=0.97, P less than 0.002)
In the United States of America, a strong positive correlation (r=0.55, P less than 0.001) also exists between MS death rates and dental caries indices

1.6.7.
Dental amalgam and multiple sclerosis: a systematic review and meta-analysis=>
http://www.ncbi.nlm.nih.gov/pubmed/17436982
Abstract only
Coremessage:
The pooled OR for the risk of MS among amalgam users was consistent, with a slight, nonstatistically significant increase between amalgam use and risk of MS. (da waren die "Researcher" aber sehr sparsam im Vergleich zu den Mercuryergebnissen (siehe unten unter Mercury & MS))

1.7. DENTAL AMALGAM , THYROID & ENDOCRIN SYSTEM

1.7.1.
The relation between human exposure to mercury and thyroid hormone status.=>
http://www.ncbi.nlm.nih.gov/pubmed/22426797
Abstract only
Coremessages:
There was a positive correlation between maternal THg and children's TSH
Mothers with dental amalgam fillings had significantly lower T4 and fT4 levels.
Our results suggest that low-level exposure to Hg can affect thyroid hormone status during prenatal and early postnatal exposure depending on the form of Hg, gender, ethnicity, lifestyle, or socioeconomic status (dental amalgam fillings).

1.8. DENTAL AMALGAM & SCHIZOPHRENIA

1.8.1.
Personality variables in patients with self-reported reactions to dental amalgam. =>
http://www.ncbi.nlm.nih.gov/pubmed/14763784
Abstract only
Coremessage:
On MMPI-2, the amalgam patients presented a 'conversion V' pattern, and elevated psychasthenia and schizophrenia scales, reflecting an increased prevalence of psychological and somatic complaints compared with the controls
This indicates that amalgam patients experience ill health, as their personality profiles bear several similarities with other groups with long-lasting symptoms

1.9. DENTAL AMALGAM & PARKINSON

NOTHING FOUND **YET** ! (siehe hierzu auch die umfangreiche Sammlung unten im Abschnitt "Mercury & Parkinson)

1.10. DENTAL AMALGAM & ALS (Amyotropic lateralsclerosis)

1.10.1.
Recovery from amyotrophic lateral sclerosis and from allergy after removal of dental amalgam fillings=>
http://www.ncbi.nlm.nih.gov/pubmed/23511261
No Abstract available

1.11. DENTAL AMALGAM & CFS (CHRONIK FATIGUE SYNDROME)

1.11.1.
Physical and mental problems attributed to dental amalgam fillings: a descriptive study of 99 self-referred patients compared with 272 controls. =>
http://www.ncbi.nlm.nih.gov/pubmed/9021864
Abstract only
Coremessage:
One third of the dental amalgam patients reported symptoms of chronic fatigue syndrome compared with none in the dental control sample and only 2 and 6%, respectively, in the two clinical comparison samples.

1.12. DENTAL AMALGAM & ASTHMA

1.12.1.
Mercury--is it a respiratory tract allergen?=>
http://www.ncbi.nlm.nih.gov/pubmed/2187473
Abstract only
Coremessage
This observation suggests that mercury in the form of dental amalgam may also be an allergen of the respiratory tract, which should not be surprising, bearing in mind the work that shows the existence of mercury vapours from dental amalgam.

1.13. DENTAL AMALGAM & FIBROMYALGIA

1.13.1.
Mercury exposure from dental amalgam fillings in the etiology of primary fibromyalgia: a pilot study=>
http://www.ncbi.nlm.nih.gov/pubmed/8596179
No abstract available

1.14.DENTAL AMALGAM & AUTISM

1.14.1.
A prospective study of prenatal mercury exposure from maternal dental amalgams and autism severity.=>
http://www.ncbi.nlm.nih.gov/pubmed/19593333
Abstract only
Coremessage:
Subjects with > or =6 amalgams were 3.2-fold significantly more likely to be diagnosed with autism (severe) in comparison to ASD (mild) than subjects with < or =5 amalgams.

1.15. DENTAL AMALGAM & SOCIAL PHOBIA

NOTHING FOUND **YET** !

1.16. DENTAL AMALGAM & ADHD / ADHS

NOTHING FOUND **YET** !

1.17.DENTAL AMALGAM & OXIDATIVE STRESS

1.17.1.
Effect of mercury (Hg) dental amalgam fillings on renal and oxidative stress biomarkers in children=>
http://www.ncbi.nlm.nih.gov/pubmed/22683759
Abstract only
Coremessages:
Multiple regression analyses revealed that the excretion of urinary NAG was significantly associated with the presence of dental amalgam fillings (β=0.149, P=0.03) and the levels of UHg-C (β=0.531, P=0), with an interaction between the two (P=0).
Urinary NAG levels were positively associated with urinary MDA levels (β=0.516, P=0) but not with 8-OHdG (β=0.134, P=0.078) after adjustment for potential confounders. Both UHg-C

1.17.2.
Cytotoxicity of root perforation repair materials.=>
http://www.ncbi.nlm.nih.gov/pubmed/9693574
Abstract only
Coremessage:
Results demonstrated that both material and time affected cell viability ($p < 0.0001$), with amalgam eluate significantly inhibitory on cell viability at 24 h, compared with control and the two other tested materials.

1.17.3.
Release of mercury from dental amalgam and its influence on salivary antioxidant activity.=>
http://www.ncbi.nlm.nih.gov/pubmed/11799733
Abstract only
Coremessage:
The present study provides, for the first time, evidence of a pro-oxidant role of the amalgam Hg chronically released in saliva.

1.17.4.
Influence of amalgam fillings on Hg levels and total antioxidant activity in plasma of healthy donors.=>
http://www.ncbi.nlm.nih.gov/pubmed/11799720
Abstract only
Coremessage:
TAA negatively correlated with Hg plasma revealing a pro-oxidant role of Hg released from amalgam fillings.

TEIL 2.

PROBLEMS WITH MERCURY & MERCURY TOXICOLOGY IN GENERAL , WITHOUT DIRECT & INDIRECT RELATIONSHIP TO DENTAL AMALGAM

2.1. MERCURY TOXICOLOGY & ENVIRONMENTAL PROBLEMS , GENERAL CASES.

2.1.1
[Neurotoxic effect of exposure to low doses of mercury]. =>
http://www.ncbi.nlm.nih.gov/pubmed/12197270
Abstract only
Coremessage:
In conclusion, this study supports the finding of early alterations of motor function and neuroendocrine secretion at very low exposure levels of inorganic Hg, below the current ACGIH BEI and below the most recent exposure levels reported in the literature.

2.1.2
Calculation of Mercury's Effects on Neurodevelopment=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3548290/
FULL ACCESS TO STUDY
Coremessages:
Prospective data justify a lower threshold Hg level of 0.58 µg/g hair corresponding to 50% of the reference dose (Grandjean and Budtz-Jørgensen 2007). In addition, a 1-µg/g increase in hair Hg concentration is more likely associated with an average adverse impact of 0.465 IQ points, as discussed by Pichery et al. (2012).

2.1.3.
Case of Mercury Poisoning =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1958561/pdf/brmedj02869-0054.pdf
FULL ACCESS TO STUDY

2.1.4.
Subjective symptoms and neurobehavioral performances of ex-mercury miners at an average of 18 years after the cessation of chronic exposure to mercury vapor. Mercury Workers Study Group.=>
http://www.ncbi.nlm.nih.gov/pubmed/8344236
Abstract only
Coremessage:
Although the extent of the workers' symptoms caused by mercury poisoning, termed erethismus merculialis, markedly decreased after the cessation of exposure, the prevalence of neurological symptoms (such as hand tremors, headaches, and slurred speech) and symptoms of senility (such as low-back pain, loss of sexual desire) in the ex-miners was significantly higher than those in the controls.

2.1.5.
Sensitivity of dopaminergic neuron differentiation from stem cells to chronic low-dose methylmercury exposure =>
http://toxsci.oxfordjournals.org/content/early/2011/03/07/toxsci.kfr054.full.pdf (Achtung umfangreich)
FULL ACCESS TO STUDY !

2.1.6.
Differing Effects of Toxicants (Methylmercury, Inorganic Mercury, Lead, Amyloid β, and Rotenone) on Cultured Rat Cerebrocortical Neurons: Differential Expression of Rho Proteins Associated With Neurotoxicity =>
http://toxsci.oxfordjournals.org/content/126/2/506.full

FULL ACCESS TO STUDY !

2.1.7.
Psychological performance and long-term exposure to mercury vapors.=>
http://www.ncbi.nlm.nih.gov/pubmed/6740275
Abstract only
Coremessage:
The more heavily exposed workers performed more poorly on the verbal intelligence test (Similarities) than the referents did. Impairments in the memory tests showed a statistically significant correlation with the actual exposure level, especially with the actual concentration of mercury in blood

2.1.8.
Prognosis of mercury poisoning in mercury refinery workers.=>
http://www.ncbi.nlm.nih.gov/pubmed/6497343
Abstract only
Coremessages:
They were reexamined after 2 months of hospital admission and chelation treatment with unithiol or sodium dimercaptosuccinate (Na-DMS). Based on clinical evaluation, the condition of the patients in both groups had all improved, even in the severe cases.

2.1.9.
Effect of occupational exposure to elemental mercury on short term memory.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009214/pdf/brjindmed00056-0053.pdf
FULL ACCESS TO STUDY
Coremessages:
Previous studies have indicated that exposure to elemental mercury is associated with increased short term memory scanning time.
Despite lower urinary mercury concentrations in this second group (0.11 mg/l average), a statistical association was again observed relating urine mercury to reduced short term memory capacity.

2.1.10.
Effects of low exposure to inorganic mercury on psychological performance.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1035109/
FULL ACCESS TO STUDY
Coremessage:
The personality of the occupationally exposed workers was found to be considerably changed compared with that of the control group

2.1.11.
Residual neurologic deficits 30 years after occupational exposure to elemental mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/11022856
Abstract only
Coremessages:
Quantitative assessment of resting tremor was nearly significantly associated with cumulative mercury exposure (p=0.07).
The statistically significant associations with mercury exposure were observed in spite of greater mortality among the exposed group than the unexposed group
These results suggest that substantial occupational mercury exposure can have long-term adverse effects on the peripheral nervous system detectable decades after cessation of exposure

2.1.12.
Mercury accumulation in the squirrel monkey eye after mercury vapour exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/8604479
Abstract only
Coremessage:
Mapping of the mercury distribution in the eye revealed that the non-myelin-containing portion of the optic disc was densely loaded with mercury deposits, which are mostly confined to the capillary walls and the glial columns.
This finding indicates that mercury is trapped within the melanocytes, which keeps potentially dangerous material from reaching the neural retina. In addition, the retinal capillary walls were densely loaded with mercury deposits, even 3 years after exposure.

2.1.13.
Glutathione level after long-term occupational elemental mercury exposure =>
http://www.ncbi.nlm.nih.gov/pubmed/17706633
Abstrsct only
Coremessages:
The mean CAT activity in miners and retired miners was significantly higher ($p<0.05$) than in the controls
The mean concentrations of GSH (mmol/g Hb) in miners (13.03+/-3.71) were significantly higher ($p<0.05$) than in the control group (11.68+/-2.66)
A positive correlation between GSSG and present U-Hg excretion ($r=0.41$, $p=0.001$) in the whole group of ex-mercury miners was observed.

2.1.14.
Occupational mercury exposure and its consequences for behaviour.=>
http://www.ncbi.nlm.nih.gov/pubmed/7129652
Abstract only
Coremessages:
Mercury is a known neurotoxin. Evidence from animal studies show behavioural impairment which can be long-lasting, after low-level exposure to mercury
The mercury-exposed group showed poorer psychomotor co-ordination and premature fatigue, although simple motor responses were not affected

2.1.15.
[Clinico-neuro-psychological evaluation of workers exposed to metallic mercury in the electric lamp industry =>
http://www.ncbi.nlm.nih.gov/pubmed/1342526
Abstract only
Coremessage:
(80.30%) of chronically poisoned workers showed poor psychomotor co-ordination, 56 (78.88%) showed neurological impairments, 51 (71.83%) decreases in memory capacity, 47 (66.20%) pathological findings in the clinical exam, 45 (63.38%) psychiatric disturbances and 37 (52.10%) poor performance in the concentration test.

2.1.16.
Evaluation of mercury in urine as an indicator of exposure to low levels of mercury vapor.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241455/
Abstract only
Coremessage: --

2.1.17.
Heavy metals and fertility.=>
http://www.ncbi.nlm.nih.gov/pubmed/9726782
Abstract only
Coremessage:
. Significant correlations were found between different heavy metals and clinical parameters (age, body mass index, nationality) as well as gynecological conditions (uterine fibroids, miscarriages, hormonal disorders).

2.1.18.
Effects of prenatal exposure to mercury on cognitive and psychomotor function in one-year-old infants: epidemiologic cohort study in Poland.=>
http://www.ncbi.nlm.nih.gov/pubmed/16275013
Abstract only
Coremessage:
The geometric mean (GM) for maternal blood mercury level for the group of infants with normal neurocognitive performance was lower (GM = 0.52 mug/L; 95% confidence interval [CI], 0.46-0.58) than that observed in the group with delayed performance (GM = 0.75 mug/L; 95% CI, 0.59-0.94), and this difference was significant ($p = 0.010$).

2.1.19.
Impact of heavy metals on hormonal and immunological factors in women with repeated miscarriages.=>
http://www.ncbi.nlm.nih.gov/pubmed/9741713
Abstract only
Coremessage:

We conclude that heavy metals seem to have a negative impact on ovarian as well as on pituitary function.

2.1.20.
Renal disposition of mercury in rats after intravenous injection of inorganic mercury and cysteine.=>
http://www.ncbi.nlm.nih.gov/pubmed/7723073
Abstract only
Coremessage:
These data indicate that coadministration of a nontoxic dose of inorganic mercury with a twofold higher amount (in moles) of cysteine increases significantly the clearance of mercury from the blood and increases the accumulation of inorganic mercury in the renal cortex and outer stripe of the outer medulla during the initial 1 h after injection

2.1.21.
DEPOSITION OF MERCURY IN THE EYE =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC510665/pdf/brjopthal00677-0052.pdf (VON 1937! !!)
FULL ACCESS TO STUDY
Coremessage:
A case of deposition of mercury in the eyelids, the bulbar conjunctiva, the region of Descemet's membrane, and anterior lens capsule after long-continued local application of mercury ointment is described.

2.1.22.
MERCURY IN THE LENS (HYDRARGYROSIS ENTIS) =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1315201/pdf/taos00055-0084.pdf
FULL ACCESS TO STUDY
Coremessage: ---

2.1.23.
Basolateral uptake of inorganic mercury in the kidney.=>
http://www.ncbi.nlm.nih.gov/pubmed/9705903
Abstract only
Coremessages:
The disposition of mercury was studied in the kidneys, liver, and blood 1 h after treatment. In rats given only mercuric chloride, the renal burden of mercury was approximately 20-25% of the administered dose of mercury, which is approximately 50% of the renal burden of mercury detected on average in normal rats. Overall, the findings from the present study provide additional evidence that there is basolateral uptake of inorganic mercury in the kidneys and that the primary mechanism involved in this basolateral uptake is dependent on the activity of the organic anion transporter.
More importantly, the present findings also show that GSH and cysteine enhance the basolateral uptake of mercuric ions in the kidney when they are coadministered with inorganic mercury (presumably in the form of mercuric conjugates).

2.1.24.
Effects of exercise training on the distribution of metallic mercury in mice. =>
http://www.ncbi.nlm.nih.gov/pubmed/7946505
Abstract only
Coremessage:
The purpose of this study was to correlate exercise induced changes of antioxidant enzymes with the distribution of mercury after mercury vapour exposure in mice.
Mercury concentrations in the Ex group were significantly higher than the N.Ex group in the heart, whole blood, red blood cells and the brain at 24 and 48 h; and in the plasma and kidneys at 24 h. 4. It was concluded that exercise training is a factor in distribution changes of mercury after exposure to mercury vapour

2.1.25.
Inorganic lead (Pb)- and mercury (Hg)-induced neuronal cell death involves cytoskeletal reorganization =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3188729/ =>
FULL ACCESS TO STUDY !

2.1.26.
A quantitative evaluation of brain dysfunction and body-burden of toxic metals =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3560777/
FULL ACCESSS TO STUDY
Coremessages:
The present study associated brain dysfunction with Hg body-burden in a cohort of patients diagnosed with NDs, but the contributions of other heavy metals or genetic factors cannot be ruled-out.

2.1.27.
Body burden of mercury is associated with acute atopic eczema and total IgE in children from southern Germany.=>
http://www.ncbi.nlm.nih.gov/pubmed/15341030
No abstract available

2.1.28.
Low dose mercury toxicity and human health.=>
http://www.ncbi.nlm.nih.gov/pubmed/21783611
Abstract only
Coremessage:
Decreased performance in areas of motor function and memory has been reported among children exposed to presumably safe mercury levels
Similarly, disruption of attention, fine motor function and verbal memory was also found in adults on exposure to low mercury levels

2.1.29.
Mercury Toxicity=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3440017/
FULL ACCESS TO STUDY
Coremessage:
OHNE WORTE !

2.1.30.
Fish Consumption, Low-Level Mercury, Lipids, and Inflammatory Markers in Children =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3267839/
FULL ACCESS TO STUDY
Coremessages:
This study of a pediatric population is the first to document an association between blood Hg, systemic inflammation, and endocrine disruption in humans, in a pediatric sample.
Therefore, the data indicates that while fish consumption in children leads to a more atheroprotective lipid profile, there is also a significant increase in nonessential toxic heavy metal exposure.

2.1.31.
Uptake and distribution of mercury in rats after repeated administration of mercuric chloride.=>
http://www.ncbi.nlm.nih.gov/pubmed/11759913
Abstract only
Coremessages:
Both male and female rats accumulated significantly more mercury in the kidneys than the other organs.
Mercury content in the kidneys of females was 39.9 and 40.9 microg/g at 2.0 and 4.0 mg/kg/day, respectively and of males was 34.9 and 41.0 microg/g at 2.0 and 4.0 mg/kg/day, respectively.
Mercury content in the kidneys of both of sexes was significantly higher than the other organs.

2.1.32.
Immunomodulation by mercuric chloride in vitro: application of different cell activation pathways=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1868882/
FULL ACCESS TO STUDY
Coremessages:
Results show that Hg doses above 15 ng/ml significantly reduced cell vitality ($P < 0{\cdot}05$)
Lower doses elicited distinct effects on T helper 1 (Th1) and Th2 cytokine expression depending on cellular activation pathways.
Taken together, we conclude that low-level exposure to Hg, in the absence of inflammation, polarizes the immune response toward Th2

2.1.33
Glutamate: a potential mediator of inorganic mercury neurotoxicity=>
http://www.ncbi.nlm.nih.gov/pubmed/8776719
Abstract only
Coremessage: (MC = Mercurychloride)
We have shown that: 1) 1 microM MC lowers the threshold of GLU neurotoxicity, 2) the combined neurotoxic effect of GLU plus MC is attenuated by DTT but not by GSH, which is consistent with the involvement of impaired astrocytic GLU transport, and 3) neuronal damage induced by GLU plus MC becomes less accentuated in a medium with dizocilpine (MK-801), a noncompetitive NMDA receptor antagonist.

2.1.34.
Increased inorganic mercury in spinal motor neurons follwoing chelating agents. =>
http://www.ncbi.nlm.nih.gov/pubmed/8856730
Abstract only
Coremessage:
Mercury deposits occupied significantly more volume in motor neurons after both DMPS (7.4%, SD +/- 0.7%) and DMSA (8.0% +/- SD 0.7%) treatment than in controls (4.3%, SD +/- 1.7%).

2.1.35.
Ecological effects, transport, and fate of mercury: a general review.=>
http://www.ncbi.nlm.nih.gov/pubmed/10789973
Abstract only
Coremessages:
A wide variety of physiological, reproductive and biochemical abnormalities have been reported in fish exposed to sublethal concentrations of mercury
Birds fed inorganic mercury show a reduction in food intake and consequent poor growth.
With few exceptions, terrestrial plants (woody plants in particular) are generally insensitive to the harmful effects of mercury compounds.

2.1.36.
Mercury in food items from the Idrija Mercury Mine area.=>
http://www.ncbi.nlm.nih.gov/pubmed/23683522
Abstract only
Coremessages:
As a consequence of over 500 years of mining and smelting activities (1490-1995), and of its natural geological occurrence, the soil in the Idrija region is highly contaminated with Hg
Based on data from previous studies, we can conclude that the levels of Hg in food have not diminished significantly during the past 15 years after closure of the Hg mi

2.1.37.
Sensitization to inorganic mercury could be a risk factor for infertility.=>
http://www.ncbi.nlm.nih.gov/pubmed/16136024
Abstract only
Coremessage:
In patients with metal intolerance diagnosed by the MELISA test the release of metal ions from dental materials can be one of the stimulating factors which may adversely affect fertility.

2.1.38.
Relationship of Blood Mercury Levels to Health Parameters in the Loggerhead Sea Turtle (Caretta caretta)=>
http://www.ncbi.nlm.nih.gov/pubmed/16136024
FULL ACCESS TO STUDY
Coremessage
Blood mercury concentrations were positively correlated with hematocrit and creatine phosphokinase activity, and negatively correlated with lymphocyte cell counts and aspartate amino-transferase.
In vitro exposure of peripheral blood leukocytes to methylmercury resulted in suppression of proliferative responses for B cells (0.1 µg/g and 0.35 µg/g) and T cells (0.7 µg/g).

2.1.39.
Human Exposure and Health Effects of Inorganic and Elemental Mercury=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3514464/
FULL ACCESS TO STUDY
Coremessage: Ohne Worte !

2.1.40.
Tissue levels of mercury in autopsy specimens of liver and kidney=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2366391/pdf/bullwho00462-0088.pdf
FULL ACCESS TO STUDY
Coremessage: ---

2.1.41.
Nephrotoxic actions of low-dose mercury in mice: protection by zinc **.**=>
http://www.ncbi.nlm.nih.gov/pubmed/12194165
Abstract only
Coremessage:
. Both metals reduced significantly ($p < .05$) the absolute and relative kidney weights of the animals. Zinc-treated animals showed normal kidney histology that was comparable with that of the control. Mercury treatment produced necrosis and widening of the glomeruli, whereas a combination of both metals resulted in protection from the toxic effects, with most nephrons resembling the control.

2.1.42.
Mercury toxicity: Genetic susceptibility and synergistic effects =>
http://www.medicalveritas.com/images/00070.pdf
Abstract only

2.1.43.
Dietary Mercury Exposure Resulted in Behavioral Differences in Mice Contaminated with Fish-Associated Methylmercury Compared to Methylmercury Chloride Added to Diet=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3412318/
FULL ACCESS TO STUDY
Coremessages:
Methylmercury (MeHg) is a potent neurotoxin, and humans are mainly exposed to this pollutant through fish consumption. However, in classical toxicological studies, pure methylmercury chloride (MeHgCl) is injected, given to drink or incorporated within feed assuming that its effects are identical to those of MeHg naturally associated to fish. In the present study, we wanted to address the question whether a diet containing MeHg associated to fish could result in observable adverse effects in mice as compared to a diet containing the same concentration of MeHg.

2.1.44.
Prenatal coexposure to metallic mercury vapour and methylmercury produce interactive behavioural changes in adult rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/8709923
Abstract only
Coremessages:
In the swim maze test, the MeHg + Hg degrees and Hg degrees groups evidenced longer latencies to reach a submerged platform, which they had learned to mount the day before, compared to either the control or MeHg groups. I
During the learning trial, the same groups (i.e., MeHg + Hg degrees and Hg degrees) showed longer latencies and made more errors in acquiring all eight pellets.
Generally, the results indicate that prenatal exposure to Hg degrees causes alterations to both spontaneous and learned behaviours, suggesting some deficit in adaptive functions.

2.1.45.
Human Exposure and Health Effects of Inorganic and Elemental Mercury=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3514464/
FULL ACCESS TO STUDY
Coremesssages:
Elemental mercury may also enter the brain from the nasal cavity through the olfactory pathway.

2.1.46.
A Biomonitoring Study of Lead, Cadmium, and Mercury in the Blood of New York City Adults =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2022653/
FULL ACCESS TO STUDY
Coremessage: New Yorker Städter drei mal so viel Hg im Blut als ihre Kollegen auf dem Lande

2.1.47.
Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1543702/
FULL ACCESS TO STUDY
Coremessages:
A cohort of 1022 consecutive singleton births was generated during 1987–1988 in the Faroe Islands, where increased methylmercury exposure occurs from traditional seafood diets that include pilot whale meat.
Blood and hair samples obtained from the participants were analyzed for mercury.
Indicators of prenatal methylmercury exposure were significantly associated with deficits in finger tapping speed, reaction time on a continued performance task, and cued naming.
The effects on brain function associated with prenatal methylmercury exposure therefore appear to be multi-focal and permanent.

2.1.48.
Mercury in hair as an indicator of total body burden=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2366395/
FULL ACCESS TO STUDY
Coremessage:
In the patients studied, the peak body burden ranged from 0.8 to 4.4 mg/kg in cases showing mild symptoms, from 1.5 to 6 mg/kg in cases with moderate symptoms, and from 3 to 12 mg/kg in cases with severe symptoms.

2.1.49.
Role of Bacteria in Bioaccumulation of Mercury in the Oyster Crassostrea virginica=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC187121/
FULL ACESS TO STUDY
Coremessage:
Mercury concentrations were 200 times greater in tissue fractions of oysters dosed with mercury-metabolizing bacteria compared with the oysters held under control conditions without mercury-metabolizing bacteria.

2.1.50.
Higher faecal excretion and lower tissue accumulation of mercury in Wistar rats from contaminated fish than from methylmercury chloride added to fish=>
http://www.ncbi.nlm.nih.gov/pubmed/15207387
Abstract only
Coremessage: ---

2.1.51.
A small dose of ethanol increases the exhalation of mercury in low-level-exposed humans.
http://www.ncbi.nlm.nih.gov/pubmed/10872631
Abstract only
Coremessage:
A marked increase, in general about fivefold, in mercury concentrations in end-exhaled air was seen in all subjects 30 min after intake of alcohol, regardless of the level of mercury exposure.
Higher ethanol doses resulted in higher mercury levels in end-exhaled air and longer time periods before a return to background levels
low levels of mercury can be detected in end-exhaled air also in individuals without amalgam fillings.
About a fivefold increase was seen 30 min after alcohol intake, and the relative increase seemed to be independent of the body burden of mercury.

2.1.52.
Ethanol-increased exhalation of mercury in mice. =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1008413/pdf/brjindmed00075-0047.pdf
FULL ACCESS TO STUDY
Coremessage:
Ethanol treatment led to an eight-fold increase of counts accumulated on a filter over a four-hour period, compared with water-treated mice.

2.1.53.
Elemental mercury exposure: peripheral neurotoxicity =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1008958/

FULL ACCESS TO STUDY
Coremessage:
Significant correlations between increasing urine mercury concentrations and prolonged motor and sensory distal latencies were established.

2.1.54.
Accumulation of inorganic mercury in hair of rats exposed to methylmercury or mercuric chloride.=>
http://www.ncbi.nlm.nih.gov/pubmed/17146195
Abstract only
Coremessage:
These findings suggest that the inorganic mercury is also taken up by rat hair from the blood circulation, as is the MeHg, irrespective of the consequences of the biotransformation of MeHg or exposure to inorganic mercury itself.
Accordingly, a selective quantification of inorganic mercury in human hair may be useful in detecting inorganic mercury exposure.

2.1.55.
Relationship between mercury in blood and 24-h ambulatory blood pressure in Greenlanders and Danes.=>
http://www.ncbi.nlm.nih.gov/pubmed/15882543
Abstract only
Coremessage:
Pulse pressure increased with higher mercury content in the blood

2.1.56.
Pneumonitis after inhalation of mercury vapours=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2539016/
FULL ACCESS TO STIDY
Coremessage:
A 43-year-old man presented to hospital with pneumonia but only after discharge from hospital did he admit to deliberate prior inhalation of mercury. His pulmonary involvement appeared to resolve almost completely with antibiotics and supportive care. Nevertheless, persisting elevated urinary excretion of mercury required two courses of chelation therapy. No serious systemic sequelae were observed.

2.1.57.
Exposure to low-dose mercury (from thimerosal) & premature puberty - a new avenue for research with the vaccine safety datalink.=>
http://www.ncbi.nlm.nih.gov/pubmed/20424297
No abstract available

2.1.58.
Toxic Effects of Mercury on the Cardiovascular and Central Nervous Systems =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3395437/
FULL ACCESS TO STUDY
Coremessages:
Many studies show that high exposure to mercury induces changes in the central nervous system, potentially resulting in irritability, fatigue, behavioral changes, tremors, headaches, hearing and cognitive loss, dysarthria, incoordination, hallucinations, and death.
In the cardiovascular system, mercury induces hypertension in humans and animals that has wide-ranging consequences, including alterations in endothelial function.

2.1.59.
Methylmercury poisoning in the Iraqi suckling infant: a longitudinal study over five years. =>
http://www.ncbi.nlm.nih.gov/pubmed/6892222
Abstract only
Coremessage:
Abnormal neurological signs in these infants became more obvious with time: hyperreflexia was observed in 8 of 22 infants at first examination, and in 17 of 22 at second examination.
The frequency of pathological reflexes and delayed motor developmental milestones was so high as to be considered significant even in the absence of a controlled study.

2.1.60.
Chronic low-level mercury exposure, BDNF polymorphism and associations with cognitive and motor function.=>
http://www.ncbi.nlm.nih.gov/pubmed/16301096
Abstract only
Coremessages:
Statistically significant adverse associations with HgU ($p<.05$) were found for nine measures among DDs (Digit Span (Forward), Digit and Spatial Span(Backward), Visual Reproduction, Finger Tapping(Dominant, Alternate, and Alternate Partialed), Hand Steadiness, and Tracking), and eight measures among DAs (Digit Span(Forward), Visual Reproduction, Pattern Discrimination(Rate), Symbol Digit(Rate), Trailmaking B, Finger Tapping(Dominant and Alternate Partialed), and Hand Steadiness).
Performance on verbal intelligence and reaction time were not associated with either HgU or BDNF status
Our findings are applicable to exposure levels of the general population and identify a potentially vulnerable group with a BDNF polymorphism.

2.1.61.
The absorption, blood levels, and excretion of mercury after a single dose of mercury vapor in humans=>
http://www.ncbi.nlm.nih.gov/pubmed/9630463
Abatract only
Coremessages:
The median retention of Hg0 was 69% of the inhaled dose
About 1.0% of the absorbed Hg was excreted via urine during the first 3 days after exposure, whereas the estimated amount excreted during 30 days ranged from 8 to 40%.
The daily Hg dose was estimated to 5-9 micrograms/day in subjects with an ordinary number of amalgam fillings.

2.1.62.
Case of Chronic Mercurial Poisoning =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2103529/pdf/procrsmed01361-0086a.pdf
FULL ACCESS TO STUDY
Coremessage:
Case of a **PATIENT, a male, A. S., aged 51, thermometer maker.**

2.1.63.
The Distribution and Excretion of Inhaled Mercury Vapour=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1039182/pdf/brjindmed00204-0047.pdf
FULL ACCESS TO STUDY
Coremessage:---

2.1.64.
Diet -related mercury poisoning resulting in visual loss (Case report) =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1857490/
FULL ACCESS TO STUDY
Coremessage:
A 36-year-old man with progressive peripheral neuropathies was referred to the eye clinic with failing visual acuity
No cause could be found for his ongoing weight loss, poor appetite, disturbed sleep and worsening painful neuropathies after extensive investigations.
Blood tests identified a markedly raised mercury concentration, and further inquiries identified his diet to be rich in fish caught in the Caribbean
Tests carried out on the fish he provided showed a high concentration of mercury in the tissue.
INTERESSANTER FALL !!!

2.1.65.
Tissue content of mercury in rats given methylmercuric chloride orally: influence of intestinal flora.=>
http://www.ncbi.nlm.nih.gov/pubmed/7387196
Abstract only
Coremessage:
Antibiotic-treated rats given [203Hg]-labeled methylmercuric chloride orally had significantly more mercury in their tissues, especially in kidney, brain, lung, blood, and skeletal muscle, and also excreted less mercury in the feces than conventional rats.

2.1.66.
Cytogenotoxicity in uroepithelial cells of women exposed to mercury in a mining area.=>
http://www.ncbi.nlm.nih.gov/pubmed/20798028
Abstract only
Coremessage:
Living in a mining area with exposure to inorganic mercury and having higher mercury levels in urine increased the risk of developing uroepithelial cytogenotoxicity.

2.1.67.
Mercury as a health hazard - Archives of Disease in Childhood (Case Report) =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1778295/pdf/archdisch00700-0087.pdf
FULL ACCESS TO STUDY
Coremessage:
Pink disease has virtually disappeared since teething powders were withdrawn.' We describe a case in a boy who was exposed to metallic
mercury vapour. We discuss the potential health hazard of spilled elemental mercury in the house and the difficulties of removing it from the environment.

2.1.68.
Mercury intoxication presenting with hypertension and tachycardia =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1717944/pdf/v080p00556.pdf
FULL ACCESS TO STUDY
Coremessage:
An 11 year old girl presented with hypertension and tachycardia**.**
symptoms included insomnia and weight loss, and she was found to have a raised concentration of mercury in blood and urine**.**
Mercury intoxication should be considered in the differential diagnosis of hypertension with tachycardia even in patients presenting without the skin lesions typical of mercury intoxication and **without a history of** exposure

2.1.69.
Abiotic methylation of mercury in the aquatic environment.=>
http://www.ncbi.nlm.nih.gov/pubmed/16226793
Abstract only
Coremessages:
Results of our laboratory-based investigations of aqueous mercury reactions with some potential methyl donors, including MeCo(dmg)(2)(H2O), a simple model for methylcobalamin, various methyltin compounds and methyl iodide, are presented
In each reaction, the yield of methylmercury and the rate of methylation depend strongly on environmental factors such as pH, temperature, and the presence of complexing agents, especially chloride.

2.1.70.
Intravenous mercury: a three year follow-up =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2449044/pdf/ulstermedj00072-0069.pdf
Coremessage:
Serum mercury (black line) showed a rapid initial fall, urinary mercury (grey line) a transient rise on treatment. Both remain
consideratby elevated above normal after three years.(Even after 3 Years of DMPS - treatment)

2.1.71.
Six cases of poisoning after a parenteral organic mercurial compound (Merthiolate)=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2495252/pdf/postmedj00331-0029.pdf
FULL ACCESS TO STUDY
Coremessage:
The case histories of four children and two adults who were accidentally given toxic amounts of Merthiolate are recorded. The possible modes of action of Merthiolate in causing symptoms are discussed.
Five out of the six patients died, and necropsy showed extensive renal tubular necrosis in each case, and in two, evidence of diffuse intravascular coagulation.

2.1.72.
Predicted mercury concentrations in hair from infant immunizations: cause for concern.=>
http://www.ncbi.nlm.nih.gov/pubmed/11770890
Abstract only
Coremessages:
Modeled hair Hg concentrations in infants exposed to vaccinal TMS are in excess of the Environmental Protection Agency (EPA) safety guidelines of 1 ppm for up to 365 days, with several peak concentrations within this period.
More sensitive individuals and those with additional sources of exposure would have higher Hg concentrations.

2.1.73.
Recent reports have described neurobehavioral impairments in human subjects carrying a V66M polymorphism in the gene encoding brain-derived neurotrophic factor (BDNF)=>
http://www.ncbi.nlm.nih.gov/pubmed/15254338
Abstract only
Coremessages:
we examined the potential effect of this BDNF polymorphism on symptoms and mood in an established cohort of dental practitioners with chronic low-level Hg degrees exposure.
These results indicate that among DAs very low levels of occupational Hg degrees exposure are associated with increased symptoms

2.1.74.
A cascade analysis of the interaction of mercury and coproporphyrinogen oxidase (CPOX) polymorphism on the heme biosynthetic pathway and porphyrin production.=>
http://www.ncbi.nlm.nih.gov/pubmed/16214298
Abstract only
Coremessages:
Mercury (Hg) exposure in various forms remains a persistent public health concern in many parts of the world. In previous studies, we have described a biomarker of mercury exposure characterized by increased urinary concentrations of specific porphyrins.
an atypical porphyrinogenic response (APR) has been observed in approximately 15% of Hg-exposed persons, in which the three porphyrins that are affected by Hg, i.e., 5-CP, 4-CP and, KICP, are excreted in substantial excess of that predicted on the basis of Hg exposure alone
This APR has been attributed to a specific polymorphism in exon 4 of the CPOX gene (CPOX4)
In the present study, we sought to further confirm the hypothesis that the observed changes in porphyrin excretion patterns might serve as a biomarker of Hg exposure and potential toxicity by statistically modeling the cascading effects on porphyrin concentrations within the heme biosynthetic pathway of Hg exposure and CPOX4 polymorphism in a human population with long-term occupational exposure to elemental mercury
Our results are highly consistent with this hypothesis.
These findings lend further support to the proposed utility of urinary porphyrin changes as a biomarker of exposure and potential toxicity in subjects with mercury exposure.

2.1.75.
Chronic mercury poisoning: Report of two siblings=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2923418/
FULL ACCESS TO STUDY
Coremessage:
In this study, two siblings (one a 7-year-old boy and the other a 13 years old girl) are reported who developed chronic mercury poisoning as a result of long-term contact with batteries.

2.1.76.
Metal toxicity and the respiratory tract.
http://www.ncbi.nlm.nih.gov/pubmed/2178966
Abstract only
Coremessage:
The fumes or gaseous forms of several metals, e.g. cadmium (Cd), manganese (Mn), mercury (Hg), nickel carbonyl (Nl(CO)4, zinc chloride (ZnCl2), vanadium pentoxide (V2O5), may lead to acute chemical pneumonitis and pulmonary oedema or to acute tracheobronchitis

2.1.77.
Cytotoxicity and accumulation of Hg, Ag, Cd, Cu, Pb and Zn in human peripheral T and B lymphocytes and monocytes in vitro.=>

http://www.ncbi.nlm.nih.gov/pubmed/7721038
Abstract only
Coremessage:
Examination by scanning electron microscopy showed that the heavy metals caused serious destruction of the cell membranes.

2.1.78.
Evaluation of cytotoxicity attributed to thimerosal on murine and human kidney cells.=>
.http://www.ncbi.nlm.nih.gov/pubmed/18049999
Abstract only
Coremessage:
These data demonstrate that the higher cytotoxicity produced by thimerosal on renal cells with respect to similar compounds without Hg may be related to this metal content.

2.1.79.
Shaking up the Salpêtrière Jean-Martin Charcot and mercury-induced tremor=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462583/
FULL ACCESS TO STUDY
Coremessage: --

2.1.80.
Thiol-modulated mechanisms of the cytotoxicity of thimerosal and inhibition of DNA topoisomerase II alpha=>
http://www.ncbi.nlm.nih.gov/pubmed/18197631
Abstract only
Coremessage:
Thimerosal-induced single and double strand breaks in K562 cells were consistent with a rapid induction of apoptosis
In conclusion, these studies have elucidated some of the chemistry and biological activities of the interaction of thimerosal with topoisomerase II alpha and protein and nonprotein thiols and with DNA.

2.1.81.
High temperature enhances cytotoxicity of mercury (HgCl2) on HeLa S3 cells.=>
http://www.ncbi.nlm.nih.gov/pubmed/1707405
Abstract only
Coremessage:
These results suggest that the cytotoxicity of mercury to cell growth was enhanced at the higher temperature and that this enhancement is related to the increased inhibitory effect of mercury on DNA and RNA synthesis and on the cell cycle at high temperatures.

2.1.82.
Mercury compounds disrupt neuronal glutamate transport in cultured mouse cerebellar granule cells=>
http://www.ncbi.nlm.nih.gov/pubmed/15635608
Abstract only
Coremessage:
We suggest that a direct inhibition of glutamate uptake triggers an imbalance in cell homeostasis, leading to neuronal failure and Cl(-)-regulated cellular glutamate efflux. Our results demonstrate that neuronal glutamate transport is a novel target to be taken into account when assessing mercury-induced neurotoxicity.

2.1.83.
Pneumonitis after inhalation of mercury vapours=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2539016/
FULL ACCESS TO STUDY
Coremessage:
A 43-year-old man presented to hospital with pneumonia but only after discharge from hospital did he admit to deliberate prior inhalation of mercury. His pulmonary involvement appeared to resolve almost completely with antibiotics and supportive care. Nevertheless, persisting elevated urinary excretion of mercury required two courses of chelation therapy.

2.1.84.
In vitro and whole animal evidence that methylmercury disrupts GABAergic systems in discrete brain regions in captive mink.=>
http://www.ncbi.nlm.nih.gov/pubmed/20060493
Abstract only
Coremessage:
These results show that chronic exposure to environmentally relevant levels of MeHg disrupts GABAergic signaling. Given that GABA is the main inhibitory neurotransmitter in the mammalian nervous system, prolonged disruptions of its function may underlie the sub-clinical impacts of MeHg at relevant levels to animal health.

2.1.85.
Low doses of heavy metals disrupt normal structure and function of rat platelets.=>
http://www.ncbi.nlm.nih.gov/pubmed/11215709
Abstract only
Coremessage:--

2.1.86.
Cytotoxicity and genotoxicity of low doses of mercury chloride and methylmercury chloride on human lymphocytes in vitro=>
http://www.ncbi.nlm.nih.gov/pubmed/15933784
Abstract only
Coremessage:
A significant increase ($P < 0.05$) in the relative frequency of chromosome aberrations was observed for all concentrations of CH_3HgCl when compared to control, whether alone or in an evident sinergistic combination with $HgCl_2$

2.1.87.
Hair Mercury Negatively Correlates with Calcium Pump Activity in Human Term Newborns and Their Mothers at Delivery=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2235233/
FULL ACCESS TO STUDY
Coremessage:
Maternal hair Hg negatively correlates with Ca pump activity in maternal and cord blood erythrocytes.

2.1.88.
Thimerosal neurotoxicity is associated with glutathione depletion: protection with glutathione precursors.=>
http://www.ncbi.nlm.nih.gov/pubmed/15527868
Abstract only
Coremessages:
Cultured neuroblastoma cells were found to have lower levels of GSH and increased sensitivity to thimerosol toxicity compared to glioblastoma cells that have higher basal levels of intracellular GSH.
levels of intracellular GSH. Thimerosal-induced cytotoxicity was associated with depletion of intracellular GSH in both cell lines.

2.1.89.
In vivo genotoxicity of mercury chloride and lead acetate: Micronucleus test on acridine orange stained fish cells=>
http://www.ncbi.nlm.nih.gov/pubmed/17889980
Abstract only
Coremessage:
The results of this study indicate that LA and MC have genotoxic and cytotoxic damage in fish and confirmed that AO staining is a suitable technique for in vivo MN test in fish.

2.1.90.
In Vivo Effects of Mercury (II) on Deoxyuridine Triphosphate Nucleotidohydrolase, DNA Polymerase (α, β), and Uracil-DNA Glycosylase Activities in Cultured Human Cells: Relationship to DNA Damage, DNA Repair, and Cytotoxicity=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2901161/
FULL ACCESS TO STUDY
Coremessage:
In fact, there was a time- and dose-dependent activation of uracil-DNA glycosylase activity with maximum

activation occurring in cells exposed to 50 µM mercuric acetate.
The inhibition of dUTPase and DNA polymerase a activities and the activation of uracil-DNA glycosylase activity correlated with the induction of single-strand breaks in DNA by mercuric acetate and with the decrease in cell viability.

2.1.91.
Mercury in the hair of pregnant and lactating Chilean mothers=>
http://www.ncbi.nlm.nih.gov/pubmed/8540996
Abstract only
Coremessage:
The total mercury concentration in hair was significantly higher in women who indicated that they ate fish seven or more times per week; in those who said they ate fish, shellfish, or algae five or more times per week; and in those who had lived 20 or more years in their village.

2.1.92.
Blood Mercury Reporting in NHANES: Identifying Asian, Pacific Islander, Native American, and Multiracial Group=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1367827/
FULL ACCESS TO STUDY
Coremessage:
Study subjects in NHANES who self-identified as Asian, Pacific Islander, Native American, or multiracial had a higher prevalence of elevated blood mercury than all other racial/ethnic participants in the survey.

2.1.93.
Death Following Inhalation of Mercury Vapor at Home =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1272162/pdf/westjmed00238-0061.pdf
FULL ACCESS TO STUDY
Coremessage:
THE INHALATION of toxic fumes and metallic vapors is a fairly common occurrence in industry.
There are ample accounts describing the effects In the following report we discuss what appears to be the first death of an adult exposed to mercury vapor in the home **.**

2.1.94.
Elevation of mercury in human blood from controlled chronic ingestion of methylmercury in fish.=>
http://www.ncbi.nlm.nih.gov/pubmed/6724592
Abstract only
Coremessage:
The results indicate that a daily intake of 1 microgram methylmercury would, at equilibrium, produce a blood mercury concentration of 0.8 micrograms/kg
2.1.95.
Exposure and toxic effects of elemental mercury in gold-mining activities in Ecuador.=>
http://www.ncbi.nlm.nih.gov/pubmed/21925580
Abstract only
Coremessages:
Biomarker concentrations among merchants were statistically significantly higher than among miners and referents; also the miners differed from the referents
Thus, the gold merchants have a much higher exposure and risk than the miners, in whom the exposure varies over time.

2.1.96.
Effect of milk on mercury absorption and gut retention in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/497464
Abstract only
Coremessage: --

2.1.97.
Mechanisms of methylmercury-induced neurotoxicity.=>
http://www.ncbi.nlm.nih.gov/pubmed/7516300
Abstract only
Coremessage:
Thus, the early cellular effects of exposure to MeHg are diverse and cell damage likely occurs by more than one mechanism, the effects of which may be additive or synergistic.

2.1.98.
Maternal-to-fetus transfer of mercury in metallothionein-null pregnant mice after exposure to mercury vapor.=>
http://www.ncbi.nlm.nih.gov/pubmed/12049849
Abstract only
Coremessages:
This study examined the role of placenta metallothionein (MT) in maternal-to-fetal mercury transfer in MT-null and wild-type mice after exposure to elemental mercury (Hg(0)) vapor.
In contrast to mercury levels in maternal organs, fetal mercury levels were significantly higher in MT-null mice than in wild-type mice.
These results suggest that MT in the placenta has a defensive role in preventing maternal-to-fetal mercury transfer.

2.1.99.
Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers. Part I: History, neuropsychological findings and chelator effects.=>
http://www.ncbi.nlm.nih.gov/pubmed/1352115
Abstract only
Coremessages:
There were significant correlations between neuropsychological tests and indices of mercury exposure.
Serial mercury in the blood and urine verified the long half-life and large volume of distribution of mercury.
Chelation therapy with both drugs resulted in the mobilization of a small fraction of the total estimated body mercury.
However, DMSA was able to increase the excretion of mercury to a greater extent than NAP.
These observations demonstrate that acute exposure to elemental mercury and its vapour induces acute, inorganic mercury toxicity and causes long-term, probably irreversible, neurological sequelae.

2.1.100.
Residual neurologic deficits 30 years after occupational exposure to elemental mercury
http://www.ncbi.nlm.nih.gov/pubmed/11022856
Abstract only
Coremessage:
These results suggest that substantial occupational mercury exposure can have long-term adverse effects on the peripheral nervous system detectable decades after cessation of exposure.

2.1.101
A case of chronic inorganic mercury poisoning with progressive intentional tremor and remarkably prolonged latency of P300].=>
http://www.ncbi.nlm.nih.gov/pubmed/10349350
Abstract only
Coremessage:
Chronic inorganic mercury poisoning has been reported to produce organic changes in the brain and P300 is considered to be useful to detect these changes

2.1.102.
Auto-aggressive metallic mercury injection around the knee joint: a case report=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3226429/
FULL ACCESS TO STUDY
Coremessage:
Herein we present the case of a 29-year-old male patient who developed an obsessive-compulsive disorder causing auto-aggressive behaviour with injection of elemental mercury and several other foreign bodies into the soft tissues around the left knee about 15 years before initial presentation.

2.1.103
Phospholipase A2 stimulation by methyl mercury in neuron culture=>
http://www.ncbi.nlm.nih.gov/pubmed/8294933
Abstract only
Coremessage: --

2.1.104.
Accidental ethyl mercury poisoning with nervous system, skeletal muscle, and myocardium injury.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC490489/

FULL ACCESS TO STUDY
Coremessage:
The clinical, electrophysiological, and toxicological, and in two of the patients the pathological data, showed that this organic mercury compound has a very high toxicity not only for the brain, but also for the spinal motoneurones, peripheral nerves, skeletal muscles, and myocardium.

2.1.105.
Chronic elemental mercury intoxication: clinical and field studies in lampsocket manufacturers =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1127959/pdf/oenvmed00052-0051.pdf
FULL ACCESS TO STUDY
Coremessages:
Four workers chronically exposed to elemental mercury in a lampsocket manufacturing factory were studied. The clinical manifestations were severe in one, mild in another, and suspicious in the remaining two. Correlation between severity of clinical features and increased urinary mercury concentrations was found.

2.1.106.
Changes in the nervous system due to occupational metallic mercury poisoning=>
http://www.ncbi.nlm.nih.gov/pubmed/9513954
Abstract only
Coremessage:
The authors stressed the irreversibility of central nervous disorders despite cessession of the exposure to Hg.

2.1.107.
Ultrastructural studies of the nervous system after mercury intoxication. II. Pathological changes in the nerve fibers.=>
http://www.ncbi.nlm.nih.gov/pubmed/5047107
No abstract available

2.1.108.
Effect of kidney damage on the mobilisation of mercury by thiol-complexing agents =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1008679/pdf/brjindmed00066-0020.pdf
Abstract only
Coremessage:
In the present work the ability of three thiol-complexing agents, D-penicillamine, N-acetyl-D, L-penicillamine, and 2,3-dimercaptosuccinic acid (DMSA) to remove mercury from a damaged
kidney and to increase the urinary excretion of mercury were studied.
It was shown that both renal uptake and urinary excretion of mercury were decreased in animals
with damaged kidneys.

2.1.109.
Urinary mercury in twelve cases of cutaneous mercurous chloride (calomel) exposure: effect of sodium 2,3-dimercaptopropane-1-sulfonate (DMPS) therapy.=>
http://www.ncbi.nlm.nih.gov/pubmed/9365436
Abstract only
Chronic topical application of 5.9% HgCl cream was associated with clinical mercurialism in two subjects and with high urinary mercury level in all the cases.

2.1.110.
Tremor in workers with low exposure to metallic mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/3766401
Abstract only
Coremessage:
The study indicates that exposure to metallic mercury below the current TLV (50 micrograms/m3) may increase the tremor of the finger.

2.1.111.
Early biochemical effects of an organic mercury fungicide on infants: "dose makes the poison".=>
http://www.ncbi.nlm.nih.gov/pubmed/2857500
Abstract only
Coremessage:
The results support the threshold concept of the systemic toxicity of metals. gamma-Glutamyl transpeptidase

is a useful and sensitive marker for preclinical effects of toxic metals.

2.1.112.
Quantitative evaluation of urinary porphyrins as a measure of kidney mercury content and mercury body burden during prolonged methylmercury exposure in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/11353132
Abstract only
Coremessage:
These findings demonstrate that urinary porphyrin concentrations relate quantitatively to DMPS-mobilizable mercury in the kidney and, therefore, serve as a biochemical measure of renal mercury content.

2.1.113.
[Deposits of mercury in the lungs and abdomen of a 20-year-old psychopath =>
http://www.ncbi.nlm.nih.gov/pubmed/432193
No Abstract available

2.1.114.
Low-dose exposure to inorganic mercury accelerates disease and mortality in acquired murine lupus.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241605/
FULL ACCESS TO STUDY
Coremessage:
Inorganic mercury (iHg) is known to induce autoimmune disease in susceptible rodent strains. Additionally, in inbred strains of mice prone to autoimmune disease, iHg can accelerate and exacerbate disease manifestations.
Our results indicate that a 2-week exposure to low-dose iHg (20 or 200 micro g/kg every other day) to donor and host mice ending 1 week before GVHD induction can significantly worsen parameters of disease severity, resulting in premature mortality.

2.1.115.
Mercury exposure in French Guiana: levels and determinants. =>
http://www.ncbi.nlm.nih.gov/pubmed/9709995Abstract only
Coremessages:
Overall, 12% of the samples contained mercury levels in excess of 10 microg/g, but in some Amerindian communities up to 79% of the children had hair mercury levels that exceeded 10 microg/g.
The results of this study indicated that (a) diet played a predominant role in total mercury burden, and (b) in some communities, mercury contamination exceeded safe levels.

2.1.116.
Chelation: Harnessing and Enhancing Heavy Metal Detoxification—A Review=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3654245/
FULL ACCESS TO STUDY

2.1.117.
[A case of embolism caused by metallic mercury in a drug addict].=>
http://www.ncbi.nlm.nih.gov/pubmed/2483639
Abstract only
Coremessage:
We describe the case of pulmonary embolism from metallic mercury after an deliberate intravenous injection in a drug addict. Metallic mercury embolisation is extremely rare and it is very important to remark the role of elementary mercury in chronic poisoning.

2.1.118.
Intravenous injection of elemental mercury: A report of two cases =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2740532/
FULL ACCESS TO STUDY
Coremessage:
Two cases of intravenous injection of elemental mercury are described in this report. One patient succumbed and the other remains asymptomatic two years after the surgical removal of all the injected mercury. Management of intravenous injection of elemental mercury (intended to be an aphrodisiac in these two cases) is discussed here and the need for surgical removal of all accessible mercury has been emphasized

2.1.119.
Measurement of hand tremor induced by industrial exposure to metallic mercury =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009173/pdf/brjindmed00054-0084.pdf
FULL ACCESS TO STUDY !

2.1.120.
Differences in frequency of finger tremor in otherwise asymptomatic mercury workers=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1035292/pdf/brjindmed00048-0046.pdf
FULL ACCESS TO STUDY
Coremessage:
Tremor was measured from the index finger during low force, position holding in 18 control subjects and 18 battery workers with low level exposure to mercury.
The findings also confirm other reports that currently permitted exposures to mercury are associated with subtle but distinctive differences in tremor accompanying voluntary movement.

2.1.121.
SKN-1/Nrf2 Inhibits Dopamine Neuron Degeneration in a Caenorhabditis elegans Model of Methylmercury Toxicity =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3003544/
FULL ACCESS TO STUDY !

2.1.122.
Involvement of environmental mercury and lead in the etiology of neurodegenerative diseases. =>
http://www.ncbi.nlm.nih.gov/pubmed/16898674
Abstract only !

2.1.123.
Mercury-induced toxicity of rat cortical neurons is mediated through N-methyl-D-Aspartate receptors=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462706/
FULL ACCESS TO STUDY
Coremessages:
We found that inorganic mercuric chloride ($HgCl_2$ –at 0.025 to 25µM) not only caused neuronal degeneration but also perturbed neuronal excitability.
Collectively, our data show that $HgCl_2$-induced toxic effects on central neurons are triggered by an over-activation of NMDA receptors, leading to cytoskeleton instability.

2.1.124.
Organic and Inorganic Mercury in Neonatal Rat Brain after Prenatal Exposure to Methylmercury and Mercury Vapor =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2831924/
FULL ACCESS TO STUDY
Coremessage:
Statistical analysis using linear mixed effects models showed that MeHg dose was the primary determinant of both organic and inorganic brain Hg levels.
For both outcomes, we also found significant interactions between MeHg and Hg vapor exposure.
This interaction, heretofore not reported, suggests that coexposure to MeHg and Hg vapor at levels relevant to human exposure might elevate neurotoxic risks

2.2. MERCURY & MULTIPLE SCLEROSIS

2.2.1.
The mercury-multiple sclerosis connection.=>
http://www.ncbi.nlm.nih.gov/pubmed/3855877
No abstract available

2.2.2.
Theoretical considerations on the etiology of multiple sclerosis. Is multiple sclerosis a mercury allergy=>
http://www.ncbi.nlm.nih.gov/pubmed/5985125
No abstract available.

2.2.3.
Atrophy of large myelinated motor axons and declining muscle grip strength following mercury vapour inhalation in mice=>
http://www.ncbi.nlm.nih.gov/pubmed/16326402
Abstract only
Coremessage:
The group exposed to Hg(0) showed a significant reduction in the mean axon caliber, $p < .0001$. Gaussian spectral analysis of axon diameter distribution showed atrophy principally to large myelinated fibers, a subpopulation of axons that is also affected in MND.

2.2.4.
Serum mercury level and multiple sclerosis=>
http://www.ncbi.nlm.nih.gov/pubmed/22068727
Abstract only
Coremessage
Blood samples were collected and serum mercury content was determined. Serum mercury level in MS patients was significantly higher than controls (9.6±10.17 vs. 5.7±8.6, P=0.037). Concerning all MS patients, serum mercury value was significantly higher than the mercury concentration founded in control subjects {odd ratio: 2.39 (CI, 1.96-2.94), P=0.00}

2.2.5.
A case of multiple sclerosis improvement following removal of heavy metal intoxication: lessons learnt from Matteo's case.=>
http://www.ncbi.nlm.nih.gov/pubmed/22438029
Abstract only
Coremessage:
We studied the case of a patient affected by MS, who has been unsuccessfully treated for some years with current therapies. We examined his levels of toxic heavy metals in the urine, following intravenous "challenge" with the chelating agent calcium disodium ethylene diamine tetraacetic acid (EDTA).The patient displayed elevated levels of aluminium, lead and mercury in the urine. Indeed, he was subjected to treatment with EDTA twice a month. Under treatment, the patient revealed in time improved symptoms suggestive of MS remission. The clinical data correlated with the reduction of heavy metal levels in the urine to normal range values. Our case report suggests that levels of toxic metals can be tested in patients affected by neurodegenerative diseases as MS.

2.2.6.
Effect of mercury on rabbit myelin CNP-ase in vitro.=>
http://www.ncbi.nlm.nih.gov/pubmed/3031563
Abstract only
Coremessage:
2',3'-cyclic nucleotide 3'-phosphodiesterase (CNPase) catalyzes hydrolysis of 2',3'-cyclic nucleotides to form the corresponding 2'-monophosphates. Rabbit myelin fraction with CNPase specific activity between 30-40 mumoles/min/mg protein was incubated in the presence of various inorganic and organic heavy metal compounds: HgCl2; (CH3Hg)OH; Pb(NO3)2; Pb(C2H302)2.3H20; (C2H5)2Pb; (C2H5)3SnCl.
The enzyme has been shown to be almost exclusively sensitive to mercurials in microM concentration range.

2.2.7.
Peripheral neuropathy following intraneural injection of mercury compounds=>
http://www.ncbi.nlm.nih.gov/pubmed/6263220
Absract only
Coremessage:
The predominant effect of mercuric chloride was on Schwann cells which showed cytoplasmic swelling and necrosis, associated with extensive segmental demyelination

2.2.8.
Lethal effects of inorganic mercury on cells and tissues of Trichomycterus brasiliensis (Pisces; Siluroidei).=>
http://www.ncbi.nlm.nih.gov/pubmed/9091098
Abstract only
Coremessages:
For Trichomycterus brasiliensis, inorganic mercury is lethal above 0.1 mg.l(-1) in 24 hours. The gills were severely affected: an increased cell proliferation in the interlamellar regions leads to a thickening of the

secondary lamellae.
The liver is increasingly damaged:
After 24 hours, necrosis is almost complete and blood comes out of all capillaries
Nerves such as the optic, show disorganized disposition of axons and mainly disruption and dissociation of myelin sheaths,

2.3. MERCURY & ALZHEIMER

2.3.1.
Alzheimer disease: mercury as pathogenetic factor and apolipoprotein E as a moderator.=>
http://www.ncbi.nlm.nih.gov/pubmed/15580166
Abstract only
Coremessage:
Some studies have shown higher mercury concentrations in brains of deceased and in blood of living patients with Alzheimer's disease.
perimental studies have found that even smallest amounts of mercury but no other metals in low concentrations were able to cause all nerve cell changes, which are typical for Alzheimer's disease

2.3.2.
Mercury(II) promotes the in vitro aggregation of tau fragment corresponding to the second repeat of microtubule-binding domain: Coordination and conformational transition.=>
http://www.ncbi.nlm.nih.gov/pubmed/20665688
Abstract only
Coremessages:
For the first time, we investigated the impacts of mercury(II) ions on the folding and aggregation of Alzheimer's tau fragment R2 (residues 275-305: VQIIN KKLDL SNVQS KCGSK DNIKH VPGGGS), corresponding to the second repeat unit of the microtubule-binding domain, which was believed to be pivotal to the biochemical properties of full tau protein.
By ThS fluorescence assay and electron microscopy, we found that mercury(II) dramatically promoted heparin-induced aggregation of R2 at an optimum molar ratio of 1: 2 (metal: protein), and the resulting R2 filaments became smaller. Isothermal titration calorimetry (ITC) experiment revealed that the strong coordination of mercury(II) with R2 was an enthalpy-controlled, entropy-decreased thermodynamic process.
This study was undertaken to better understand the mechanism of tau aggregation, and evaluate the possible role of mercury(II) in the pathogenesis of AD

2.3.3.
Trace element imbalances in isolated subcellular fractions of Alzheimer's disease brains=>
http://www.ncbi.nlm.nih.gov/pubmed/2085723
Abstract only
Coremessage:
Comparison of element ratios revealed increased Hg/Se, Hg/Zn and Zn/Se mass ratios in AD

2.3.4.
Does inorganic mercury play a role in Alzheimer's disease? A systematic review and an integrated molecular mechanism.=>
http://www.ncbi.nlm.nih.gov/pubmed/20847438
Abstract only
Coremessages:
Some autopsy studies found increased mercury levels in brain tissues of AD patients.
In vitro models showed that inorganic mercury reproduces all pathological changes seen in AD, and in animal models inorganic mercury produced changes that are similar to those seen in AD.
Its high affinity for selenium and selenoproteins suggests that inorganic mercury may promote neurodegenerative disorders via disruption of redox regulation.

2.3.5.
Mercury vapor inhalation inhibits binding of GTP to tubulin in rat brain: similarity to a molecular lesion in Alzheimer diseased brain.=>
http://www.ncbi.nlm.nih.gov/pubmed/9291481
Abstract only

Coremessages:
Hg2+ interacts with brain tubulin and disassembles microtubules that maintain neurite structure
The identical neurochemical lesion of similar or greater magnitude is evident in Alzheimer brain homogenates from approximately 80% of patients, when compared to human age-matched neurological controls.
By 14 d Hg0 exposure, photoaffinity labelling on the beta-subunit of the tubulin dimer with [alpha 32P] 8N3 GTP in brain homogenates was decreased 41-74%, upon analysis of SDS-PAGE autoradiograms.

2.3.6.
Mercury and Alzheimer's disease=>
http://www.ncbi.nlm.nih.gov/pubmed/17628833
Abstract only
Coremessage:
Higher mercury concentrations were found in brain regions and blood of some patients with Alzheimer's disease (AD).
Low levels of inorganic mercury were able to cause AD- typical nerve cell deteriorations in vitro and in animal experiments

2.3.7.
Levels of organic and inorganic mercury in human blood predicted from measurements of total mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/20981860
Abstract only
Coremessages:
In conclusion, the relevant concentrations of inorganic Hg in plasma and organic Hg in cells can reliably be calculated from measurements of total Hg and from assumed e/p ratios.
This means a sizeable reduction of analytical work, and also provides specific information in cases of low-level co-exposure to both Hg species.

2.3.8.
Apolipoprotein E genotyping as a potential biomarker for mercury neurotoxicity.=>
http://www.ncbi.nlm.nih.gov/pubmed/12897404
Abstract only
Coremessage:
Apolipoprotein-E (apo-E) genotyping has been investigated as an indicator of susceptibility to heavy metal (i.e., lead) neurotoxicity
Moreover, the apo-E epsilon (epsilon)4 allele is a major risk factor for neurodegenerative conditions, including Alzheimer's disease (AD)

2.3.9.
The Relationship of Toxic Effects of Mercury to Exacerbation of the Medical Condition classified as Alzheimer’s Disease
http://www.fda.gov/ohrms/dockets/dailys/02/Sep02/091602/80027dd5.pdf
FULL ACCESS TO STUDY
Coremessage:
Body Haley on Alzheimer & Mercury

2.3.10.
Factors Affecting the Amount of Mercury in Human Scalp Hair=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1775849/pdf/amjph00792-0038.pdf
FULL ACCESS TO STUDY

2.3.11.
Regional brain trace-element studies in Alzheimer's disease.=>
http://www.ncbi.nlm.nih.gov/pubmed/3393299
Abstract only
Coremessage:
The elevation of mercury in AD nbM, as compared to age-matched controls, is the largest trace-element imbalance observed to date in AD brain. I

2.3.12.
Increased blood mercury levels in patients with Alzheimer's disease. =>
http://www.ncbi.nlm.nih.gov/pubmed/9588761

Abstract only
Coremessage:
These results demonstrate elevated blood levels of mercury in AD, and they suggest that this increase of mercury levels is associated with high CSF levels of A beta, whereas tau levels were unrelated.
Possible explanations of increased blood mercury levels in AD include yet unidentified environmental sources or release from brain tissue with the advance in neuronal death.

2.3.13.
Mercury induces cell cytotoxicity and oxidative stress and increases beta-amyloid secretion and tau phosphorylation in SHSY5Y neuroblastoma cells.=>
http://www.ncbi.nlm.nih.gov/pubmed/10617124
Abstract only
Coremessage:
Tau phosphorylation was significantly increased in the presence of mercury ($n = 9$, $p<0.001$), whereas melatonin preincubation reduced the phosphorylation to control values. These results indicate that mercury may play a role in pathophysiological mechanisms of AD.

2.3.14.
Challenges Associated with Metal Chelation Therapy in Alzheimer's Disease=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2931820/
Abstract only
Coremessage:--

2.4. MERCURY IN BRAIN AND NERVE TISSUE:

2.4.1
Metal toxicity in the central nervous system.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1474439/
FULL ACCESS TO STUDY
Coremessage:
The developing nervous system is especially susceptible to damage by methylmercury. It has been discovered that microtubules are destroyed by this form of mercury and this effect may explain the inhibition of cell division and cell migration, processes that occur only in the developmental stages.

2.4.2.
The Chemical Nature of Mercury in Human Brain Following Poisoning or Environmental Exposure=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3400271/
FULL ACCESS TO STUDY
Coremessage:---

2.4.3.
Uptake and localization of mercury in the brain of rats after prolonged oral feeding with mercuric chloride. =>
http://www.ncbi.nlm.nih.gov/pubmed/939707
Abstract only !

2.4.4.
Interactions of mercury in rat brain.=>
http://www.ncbi.nlm.nih.gov/pubmed/7682830
Abstract only
Coremessage:
Experiments showed that HgO vapor exposure can induce the stimulation of rat brain MT(Metallothioneine) synthesis.

2.4.5.
[Comparison of the body burden of the population of Leipzig and Munich with the heavy metals cadmium, lead and mercury--a study of human organ samples].=>
http://www.ncbi.nlm.nih.gov/pubmed/8043965
Abstract only
Coremessage:
significantly higher mercury concentrations were found in the brain samples from Leipzig than from Munich.

2.4.6.
Sexual differences in the distribution and retention of organic and inorganic mercury in methyl mercury-treated rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/3757970
Abstract only
Coremessages:
At 56 days of age, male and female Long-Evans rats received 1 mumole of 203Hg-labeled methyl mercuric chloride per kilogram sc and total, organic, and inorganic mercury contents and concentrations in tissues were determined for up to 98 days postdosing. Whole body clearance of mercury was faster in females than in males, and females attained higher peak percentages of the methyl mercury dose in kidney and brain than did males.

2.4.7.
Brain, kidney and liver 203Hg-methyl mercury uptake in the rat: relationship to the neutral amino acid carrier.=>
http://www.ncbi.nlm.nih.gov/pubmed/2780503
Abstract only
Coremessage:
Brain 203Hg concentrations L-cysteine treated animals were significantly higher compared with saline treated animals (P less than 0.05) at 3 min., 7 hr and 96 hr.

2.4.8.
Antioxidant system breakdown in brain of feral golden grey mullet (Liza aurata) as an effect of mercury exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/20309630
Abstract only
Coremessage:
An overall antioxidant depletion was verified in brain of fish collected at the mercury-contaminated stations, since total glutathione content and the studied antioxidant enzymes (catalase-CAT, glutathione peroxidase-GPx, glutathione-S-transferase-GST and glutathione reductase-GR) significantly decreased. In addition, this breakdown of the redox-defense system was significantly correlated with the accumulated T-Hg levels.

2.4.9.
Uptake of elemental mercury by brain in relation to concentration of glutathione and activity of glutathione peroxidase.=>
http://www.ncbi.nlm.nih.gov/pubmed/6623515
Abstract only
Coremessage:
. After an intraventricular injection of iodoacetate, activity of glutathione peroxidase in brain was inhibited 19% and the content of reduced glutathione was decreased 20%. In these animals mercury uptake by brain increased 66% relative to controls.

2.4.10.
Hyperintense lesions in brain MRI after exposure to a mercuric chloride-containing skin whitening cream.=>
http://www.ncbi.nlm.nih.gov/pubmed/21052738
Abstract only
Coremessage:
Under treatment with dimercapto-1-propansulficacid, Hg level in the urine raised to 1,175.5 µg/l, neurological deterioration occurred, and brain magnetic resonance imaging (MRI) showed on fluid attenuated inversion recovery sequences new hyperintense lesions in the subcortical white matter
Although urinary excretion could be enhanced during chelation therapy, signs and symptoms of intoxication could be worsened.

2.4.11.
Brain and tissue levels of mercury after chronic methylmercury exposure in the monkey.=>
http://www.ncbi.nlm.nih.gov/pubmed/2499694
Abstract only
Coremessages:
One monkey that died while still being dosed had brain mercury levels three times higher than levels in blood.s
Brain mercury levels were at least three orders of magnitude higher than those predicted by assuming the half-life in brain to be the same as that in blood.
These data clearly indicate that brain half-life is considerably longer than blood half-life in the monkey under

conditions of chronic dosing.

2.4.12.
Uptake of Mercury by the Brain =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1008814/pdf/brjindmed00116-0075.pdf
FULL ACCESS TO STUDY
Coremessage:
A technique has been developed for injecting metallic mercury intravenously in aqueous solution. Thirty seconds after intravenous injection of rats with 0·1 µg. metallic mercury labelled with 203Hg nearly 20% of the dose had been exhaled and the concentration in the brain was nearly as high as in the blood.

2.4.13.
Speciation of mercury in the primate blood and brain following long-term exposure to methyl mercury.
http://www.ncbi.nlm.nih.gov/pubmed/8122267
Abstract onbly
Coremessage:
The Hg concentrations in brain (7-22 micrograms Hg/g) were considerably higher than those in normal weight monkeys, due to the high blood Hg levels in combination with a high brain-to-blood distribution ratio.

2.4.14.
Demonstration of mercury in the human brain and other organs 17 years after metallic mercury exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/8793247
Abstract only
Coremessage:
Histological examination of the tissue by the Danscher and Schroder method, which is specific for mercury, showed a highly positive staining in the majority of nerve cells and cells of other organs

2.4.15.
Mercury in human spinal motor neurons. =>
http://www.ncbi.nlm.nih.gov/pubmed/9829816
Abstract only
Coremessage:
Inorganic mercury has been proposed as a neurotoxin that could cause sporadic motor neuron disease (SMND). We were therefore interested to see if mercury could be detected in the upper and lower motor neurons of SMND patients, and if mercury accumulated within motor neurons during life.
Mercury was found in the spinal motor neurons of 36% of adult control cases and 45% of adult SMND cases,

2.4.16.
Localization of mercury in CNS of the rat. II. Intraperitoneal injection of methylmercuric chloride (CH3HgCl) and mercuric chloride (HgCl2).=>
http://www.ncbi.nlm.nih.gov/pubmed/2330591
Abstract only
Coremessage:
mercury was detected in the gray matter of the spinal cord mercury. Particularly large deposits were present in the anterior horn motoneurons
At the cellular level, the heaviest staining intensity was seen in neurons, although the cytoplasm of glia and ependymal cells also showed significant deposits in sections from rats exposed to CH3HgCl.

2.4.17.
Localization of mercury in CNS of the rat. V. Inhalation exposure to metallic mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/1605734
Abstract only
Coremessage:
The autometallographical technique has been used to determine the distribution and cellular localization of mercury deposits in the Wistar rat CNS after exposure to elemental mercury vapor (50-550 micrograms Hg/m3 of air for 4-24 h).
Animal exposure to 550 micrograms Hg/m3 for 24 h resulted in visible mercury deposits in the cerebellar and cerebral cortices
Neurons in the thalamus contained heavy accumulations of mercury.
glia cells also contained scattered mercury deposits.

2.4.18.
Distribution of dietary mercury in a dog. Quantitation and localization of total mercury in organs and central nervous system=>
http://www.ncbi.nlm.nih.gov/pubmed/2717923
Abstract only
Coremessages:
An Alsatian dog which had been fed fish contaminated with methyl mercury for 7 years
In the exposed dog, mercury was found in all of the organs examined; the highest concentrations were found in the kidneys.
In the central nervous system (CNS) the mercury was fairly uniformly distributed, with 93% in the inorganic state, whereas the skeletal muscles contained approximately 30% inorganic mercury
High amounts of histochemically demonstrable mercury were observed in the liver, thyroid gland and kidney.
In the control dogs, all the organs examined were practically devoid of deposits.

2.4.19.
Uptake of inorganic mercury in the olfactory bulbs via olfactory pathways in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/9600806
Abstract only
Coremessages:
Uptake and transport in the olfactory neurons may be an important means by which some heavy metals gain access to the brain.
These data indicate that our results can be ascribed to a movement of the mercury along the olfactory axons to their terminal parts in the glomeruli and not to circulatory uptake from the mucosal vasculature.
An uptake of 203Hg2+ in the glomerular layer of the olfactory bulbs was also seen in rats given the metal intraperitoneally.
The intraperitoneal injections in addition resulted in an uptake of the 203Hg2+ in the olfactory epithelium
We propose that in these rats the mercury is taken up from the blood into the olfactory neurons and then moves along the axons to their terminations in the olfactory bulbs.
In humans a continuous exposure of the nasal cavity to mercury vapor (Hg0), released from amalgam fillings and oxidized to Hg2+ in the olfactory mucosa, as well as a potential uptake of Hg2+ in the olfactory neurons from the blood, may lead to considerable concentrations of the metal in the olfactory bulbs.

2.4.20.
Accumulation of methylmercury and inorganic mercury in the brain.=>
http://www.ncbi.nlm.nih.gov/pubmed/2484587
Abstract only
Coremessage:
After exposure to metallic mercury vapor, inorganic mercury, probably bound to selenium, accumulates in the brain

2.4.21.
The Relationship of Toxic Effects of Mercury to Exacerbation of the Medical Condition classified as Alzheimer's Disease
http://www.fda.gov/ohrms/dockets/dailys/02/Sep02/091602/80027dd5.pdf
FULL ACCESS TO STUDY
Coremessage:
Umfangreiche Arbeit von Body Haley

2.4.22.
Entry of low doses of mercury vapor into the nervous system.=>
http://www.ncbi.nlm.nih.gov/pubmed/9498219
Abstract only
Inorganic mercury remains within neurons indefinitely and has been implicated in some human neurodegenerative diseases.
We were interested in finding the lowest dose of mercury vapor that resulted in mercury deposition in neurons
low doses of mercury vapor, well within WHO guidelines for safe human occupational exposure, enter and remain within motor neurons of mice.

2.4.23.
Effects of long-term treatment with methyl mercury on the developing rat brain=>
http://www.ncbi.nlm.nih.gov/pubmed/1769362
Abstract only
Coremesssage:

2.4.24.
Abnormal auditory brainstem responses for mice treated with mercurial compounds: involvement of excessive nitric oxide.=>
http://www.ncbi.nlm.nih.gov/pubmed/11311454
Abstract only
Coremessage:
n this paper, we attempted to construct an animal (mouse) model for monitoring the oto-neurotoxicity of mercuric sulfide, comparing its toxicity with the well-known (organic) mercury compound methyl-mercury. Mice were treated with either mercuric sulfide (HgS, 0.1 and 1.0 g/kg per day) or methyl-mercury (MeHg, 0.2, 2.0 and 10 mg/kg per day) by gastric gavage for 7 consecutive days. Analysis of auditory brainstem response (ABR) indicated that significant elevation of the physiological hearing threshold as well as significant prolongation of interwave latency I-V was observed for MeHg -- (2.0 and 0.2 mg/kg per day) or HgS -- (1.0 g/kg per day, but not 0.1 g/kg per day) treated mice
These results suggest a correlation between the Hg-elicited hearing dysfunction and the availability of mercury in brain tissue.

2.4.25.
Administration of Thimerosal to Infant Rats Increases Overflow of Glutamate and Aspartate in the Prefrontal Cortex: Protective Role of Dehydroepiandrosterone Sulfate=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3264864/
FULL ACCESS TO STUDY
Coremessages:
Thimerosal, a mercury-containing vaccine preservative, is a suspected factor in the etiology of neurodevelopmental disorders. We previously showed that its administration to infant rats causes behavioral, neurochemical and neuropathological abnormalities similar to those present in autism.
Application of thimerosal to the PFC in perfusion fluid evoked a rapid increase of glutamate overflow.
Since excessive accumulation of extracellular glutamate is linked with excitotoxicity, our data imply that neonatal exposure to thimerosal-containing vaccines might induce excitotoxic brain injuries, leading to neurodevelopmental disorders.

2.4.26.
Psychiatric aspects of methylmercury poisoning=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC494811/
FULL ACCESS TO STUDY
Coremessage:
Forty-three patients with methylmercury poisoning were studied; 74·4% showed some degree of depression. Their blood levels of mercury were higher than the average values for the whole group, and considerably higher than the blood levels of the non-depressed patients. Irritability was observed in 44·2% of the patients, all except one of the 19 being under 30 years of age.
Mercury binding compounds did not seem to have a significant effect in enhancing recovery from the depressive state. The possibility of there being two distinct syndromes, due to organic and inorganic mercury poisoning, is discussed.

2.4.27.
Motor neuron uptake of low dose inorganic mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/8926498
Abstract only
Coremessages:
In animals, inorganic mercury can bypass the blood brain barrier and enter motor neurons.
We sought to determine the lowest injected dose of mercury that could be detected in mouse motor neurons.
Mice were injected intraperitoneally with mercuric chloride
Five days after injection, mercury granules were detected at doses from 0.2 microgram/g upwards in the cell bodies of spinal and brain stem motor neurons, more granules being seen at the higher doses

2.4.28.
Persistent mercury in nerve cells 16 years after metallic mercury poisoning.=>
http://www.ncbi.nlm.nih.gov/pubmed/3226504
Abstract only
Coremessage:
Danscher & Schroeder's method for mercury showed many positively staining lysosomal dense bodies in a large proportion of nerve cells, and the presence of mercury was confirmed by elemental X-ray analysis. The mercury content of the brain was increased, much of it being present in colloidal form.

2.4.29.
Follow-up of methylmercury concentration in brain areas of developing rats exposed during prenatal life using cold-vapor absorption spectrometry=>
http://www.ncbi.nlm.nih.gov/pubmed/2095813
Abstract only
Coremessage:
The distribution of mercury in brain samples showed that the metal distributes to all brain areas, but with different levels. The amount of mercury in the brain areas was about 10-100 times higher, depending on the tested area, in MMC exposed rats than in control,

2.4.30.
Long term persistence of mercury in the brain.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009672/pdf/brjindmed00150-0001.pdf
FULL ACCESS TO STUDY
Coremessage:
Interessante Reportage im "British Journal ofIndustrial Medicine"

2.4.31.
Chronic mercury poisoning from a single brief exposure=>
http://www.ncbi.nlm.nih.gov/pubmed/690736
Abstract only
Coremessage:
Although thess patients had symptoms which are not pathognomonic of chronic mercury poisoning, we feel the events described strongly suggest their relationship to a single brief exposure and represent a form of chronic mercurialism.

2.4.32.
A young man with a heavy heart (Case Report) =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1729226/
FULL ACCESS TO STUDY
Coremessage:
chest radiography and cardiac ultrasound were performed. Both revealed metal dense deposits in the heart. On questioning, the patient revealed that he had self injected with mercury 15 years before

2.4.33.
Acute mercury poisoning: a case report=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2848228/
FULL ACCESS TO STUDY
Coremessage:
This article presents a 36-year-old case admitted to emergency department (ED) due to exposure to metallic mercury

2.4.34.
Mercury chronic toxicity might be associated to some cases of hydrocephalus in adult humans?=>
http://www.ncbi.nlm.nih.gov/pubmed/22521429
Abstract only
Coremessage:
In our best knowledge, hydrocephalus and stenosis of aqueduct of Sylvius have been described only in animals exposed to methylmercury during their gestation. We think that this case of hydrocephalus might be associated with the chronic mercury exposure and therefore this etiology must be taken in account in a patient with hydrocephalus of unknown etiology

2.4.35.
Mercury vapor inhalation and poisoning of a family.=>
http://www.ncbi.nlm.nih.gov/pubmed/22906171
Abstract only
Coremessage:--

2.4.36.
quantitative and qualitative distribution of mercury in organs from arctic sledgedogs: an atomic absorption spectrophotometric and histochemical study of tissue samples from natural long-termed high dietary organic mercury-exposed dogs from Thule, Greenland.=>
http://www.ncbi.nlm.nih.gov/pubmed/8884882
Abstract only
Coremessage:
The highest concentration of total mercury was found in mesenterial lymph nodes followed by liver and kidneys, which indicates that the lymphatic system might play an important role in the regulating transport of mercury to target organs.

2.4.37.
Mercury distribution and speciation in different brain regions of beluga whales (Delphinapterus leucas).=>
http://www.ncbi.nlm.nih.gov/pubmed/23624002
Abstract only
Coremessage:
There was a positive association between SeT and HgT in all brain regions ($p<0.05$) suggesting that Se may play a role in the detoxification of Hg in the brain. The concentration of HgT in the cerebellum was significantly associated with HgT in other organs. Therefore, HgT concentrations in organs that are frequently sampled in bio-monitoring studies could be used to estimate HgT concentrations in the cerebellum, which is the target organ of MeHg toxicity

2.4.38.
Mercury accumulations in brains from populations exposed to high and low dietary levels of methyl mercury=>
http://www.ncbi.nlm.nih.gov/pubmed/10429339
Abstract only
Coremessages:
This suggests an age dependent accumulation of total mercury and a slow transformation of methyl mercury to inorganic mercury in the brain.
The autometallographically demonstrable mercury was primarily located in glia cells.

2.4.39.
Distribution of mercury in metallothionein-null mice after exposure to mercury vapor: amount of metallothionein isoform does not affect accumulation of mercury in the brain.=>
http://www.ncbi.nlm.nih.gov/pubmed/22863856
Abstract only
Coremessages:
After exposure to mercury vapor, significant Hg accumulation was observed in the brains of wild-type and MT-I/II null and MT-III null mice, as well as in the liver and kidneys.
Gel chromatograms of cerebrum soluble fractions revealed that a significant amount of Hg existed as an MT-bound form in all the mouse strains.
The present study demonstrated that brain uptake of Hg(0) and its accumulation as Hg(2+) did not depend on the amount of MT isoform in the tissue, at least in the early phase.

2.4.40.
Accumulation of mercury in neurosecretory neurons of mice after long-term exposure to oral mercuric chloride. =>
http://www.ncbi.nlm.nih.gov/pubmed/10477110
Abstract only
Coremessages:
Electron microscopy demonstrated that mercury deposits in neurosecretory neurons were detected exclusively within lysosomes. Mercury was also present in small vesicles, 40-70 nm in diameter, and in endocytic vacuoles within the axon terminals of the neurohypophysis.

2.4.41.
Retrograde axonal transport of mercury in rat sciatic nerve.=>
http://www.ncbi.nlm.nih.gov/pubmed/7687797
Abstract only
Coremessage:
The findings indicate that mercury is transported retrogradely in axons of ventral horn motoneurons and dorsal root ganglion cells.

2.4.42.
Thimerosal and Animal Brains: New Data for Assessing Human Ethylmercury Risk=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280369/
FULL ACCESS TO STUDY
Coremessage:
However, the proportion of inorganic mercury in the brain was much higher in the thimerosal group (21–86% of total mercury) compared to the methylmercury group (6–10%). Brain concentrations of inorganic mercury were approximately twice as high in the thimerosal group compared to the methylmercury group. Inorganic mercury remains in the brain much longer than organic mercury, with an estimated half-life of more than a year.

2.4.43.
Demethylation of methyl mercury in different brain sites of Macaca fascicularis monkeys during long-term subclinical methyl mercury exposure.=>
http://www.ncbi.nlm.nih.gov/pubmed/7570604
Abstract only
Coremessages:
The concentration of I-Hg in the thalamus did not decrease during the clearance period (6 months), while I-Hg in the pituitary continued to increase in spite of no additional exposure. The MeHg exposed monkeys had several times higher I-Hg concentrations in the brain than monkeys exposed to HgCl2, indicating that I-Hg was formed by demethylation of MeHg in the brain, and not by brain uptake of I-Hg formed by demethylation elsewhere in the body.

2.4.44.
Distribution and toxicity of mercury in rats after oral administration of mercury-contaminated whale red meat marketed for human consumption.=>
http://www.ncbi.nlm.nih.gov/pubmed/16263377
Abstract only
Coremessage:
The administration of red meat significantly elevated T-Hg concentrations in the liver, kidney, erythrocytes, cerebral cortex and medulla oblongata from the control levels but did not elevate the T-Hg concentration in serum, showing the typical distribution pattern of M-Hg, not of inorganic Hg

2.4.45.
Detection of mercury in rat spinal cord and dorsal root ganglia after exposure to mercury vapor. =>
http://www.ncbi.nlm.nih.gov/pubmed/8519348
Abstract only
Coremessage:
The quantitative mercury measurements demonstrated that spinal cords from rats exposed to mercury vapor for 6 or 8 weeks contained a significantly higher concentration of mercury than those from control animals.

2.5. MERCURY & ALS (Amyotrophic Lateral Sclerosis)

2.5.1.
ALS and mercury intoxication: a relationship? =>
http://www.ncbi.nlm.nih.gov/pubmed/17719172
Abstract only
Coremessages:

We report the case of an 81-year-old woman in whom clinical signs and features of electromyographic activity patterns were consistent with amyotrophic lateral sclerosis (ALS).
Increased blood level and massive urinary excretion of mercury proved mercury intoxication.
Despite a chelation treatment with Meso 2-3 dimercaptosucininc acid (DMSA), she died after 17 months.

2.5.2.
Mercury intoxication simulating amyotrophic lateral sclerosis=>
http://www.ncbi.nlm.nih.gov/pubmed/6864963
Abstract only
Coremessage:
A 54-year-old man had a syndrome resembling amyotrophic lateral sclerosis after a brief but intense exposure to elemental mercury.

2.5.3.
ALS, mercury exposure, and chelation therapy.?>
http://www.ncbi.nlm.nih.gov/pubmed/18054425
No abstract available

2.5.4.
Inorganic mercury intoxication reminiscent of amyotrophic lateral sclerosis=>
http://www.ncbi.nlm.nih.gov/pubmed/722351
Abstract only
Coremessage:
Two employees in a mercuric oxide manufacturing plant developed neurologic changes not previously reported from the exposure to inorganic mercury or elemental mercury vapor. The symptoms, physical findings and laboratory studies resembled those found in amyotrophic lateral sclerosis (ALS) and organic mercury intoxication.

2.5.5.
The association of exposure to lead, mercury, and selenium and the development of amyotrophic lateral sclerosis and the epigenetic implications.=>
http://www.ncbi.nlm.nih.gov/pubmed/20689252
Abstract only
Coremessage: --

2.5.6.
A syndrome clinically resembling amyotrophic lateral sclerosis following chronic mercurialism.=>
http://www.ncbi.nlm.nih.gov/pubmed/13751207
No abstract available

2.5.7.
Amyotrophic lateral sclerosis and metallic toxins.=>
http://www.ncbi.nlm.nih.gov/pubmed/4879837
No abstract available

2.5.8.
Exposure to an Environmental Neurotoxicant Hastens the Onset of Amyotrophic Lateral Sclerosis-Like Phenotype in Human Cu2+/Zn2+ Superoxide Dismutase 1 G93A Mice: Glutamate-Mediated Excitotoxicity=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3141904/
FULL ACCESS TO STUDY
Coremessage:
Thus in G93A mice Zn2+ apparently contributed measurably to the MeHg-induced effect. This is the initial demonstration of accelerated onset of ALS-like phenotype in a genetically susceptible organism by exposure to low concentrations of an environmental neurotoxicant.

2.5.9.
Mercury intoxication simulating amyotrophic lateral sclerosis.=>
http://www.ncbi.nlm.nih.gov/pubmed/6864963
Abstract only
Coremessage:
-A 54-year-old man had a syndrome resembling amyotrophic lateral sclerosis after a brief but intense

exposure to elemental mercury

2.5.10.
Amyotrophic lateral sclerosis after accidental injection of mercury. =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1073965/pdf/jnnpsyc00018-0100.pdf
FULL ACCES TO STUDY
Coremessage:
View Case: "Amyotrophic lateral sclerosis after accidental injection of mercury"

2.5.11.
Inorganic mercury is transported from muscular nerve terminals to spinal and brainstem motoneurons=>
http://www.ncbi.nlm.nih.gov/pubmed/1383815
Abstract only
Coremessage:
The distribution of mercury within the brainstem and spinal cord of mice was investigated with the autometallographic technique after intramuscular administration of a single dose of mercuric mercury (HgCl2). Deposits of mercury were localized to motor neurons of the spinal cord and to brainstem motor nuclei; i.e., neurons with their peripheral projections outside the blood-brain barrier.
The possible link between this process and the development of motor neuron degeneration in ALS is discussed.

2.5.12.
Mercury in the spinal cord after inhalation of mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/22364490
Abstract only
Coremessage:
Autoradiograms showed that Hg was deposited inside the spinal cord.

2.5.13.
Mercury in hair of patients with ALS=>
http://www.ncbi.nlm.nih.gov/pubmed/2805505
Abstract only
Coremessage:
Statistically it is significant (p less than 0.05) between that in ALS in Nara and Mie and that in normal controls. 6 cases (40%) with ALS in Nara and Mie have the value above the mean +2 standard deviation of controls

2.5.14.
[Amyotrophic lateral sclerosis and mercury--preliminary report].=>
http://www.ncbi.nlm.nih.gov/pubmed/2085936
Abstract only
Coremessage:
From these data mercury with low content of selenium might be one of the environmental factors which are thought to be involved in producing of ALS.

2.5.15.
Symptomatology of the amyotrophic lateral sclerosis in chronic mercury poisoning or polyneuritis in chronic alcoholism, mercurial intoxication and Pick's atrophy=>
http://www.ncbi.nlm.nih.gov/pubmed/14360364
No Abstract available

2.5.16.
Chronic mercurialism; a cause of the clinical syndrome of amyotrophic lateral sclerosis.=>
http://www.ncbi.nlm.nih.gov/pubmed/13206485
No abstract available

2.6. MERCURY & AUTISM

2.6.1.
Mercury and autism: accelerating evidence? =>
http://www.ncbi.nlm.nih.gov/pubmed/16264412
Abstract only
Coremessage:
In vitro, mercury and thimerosal in levels found several days after vaccination inhibit methionine synthetase (MS) by 50%. Normal function of MS is crucial in biochemical steps necessary for brain development, attention and production of glutathione, an important antioxidative and detoxifying agent.

2.6.2.
Safety and efficacy of oral DMSA therapy for children with autism spectrum disorders: Part B - Behavioral results=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2770991/
FULL ACCESS TO STUDY
The groups receiving one round and seven rounds of DMSA had significant improvements on all the assessment measures.

2.6.3.
Porphyrinuria in childhood autistic disorder: implications for environmental toxicity.=>
http://www.ncbi.nlm.nih.gov/pubmed/16782144
Abstract only
Coremessages:
Coproporphyrin levels were elevated in children with autistic disorder relative to control groups.
The atypical molecule precoproporphyrin, a specific indicator of heavy metal toxicity, was also elevated in autistic disorder ($P < 0.001$) but not significantly in Asperger's.

2.6.4.
Correlations Between Gene Expression and Mercury Levels in Blood of Boys With and Without Autism=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3006666/
FULL ACCESS TO STUDY
Coremessage:
Gene expression in blood was correlated with mercury levels in blood of 2- to 5-year-old boys with autism (AU) compared to age-matched typically developing (TD) control boys.

2.6.5.
Chronic inorganic mercury exposure induces sex-specific changes in central TNFα expression: Importance in autism?=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3443965/
FULL ACCESS TO STUDY
Coremessage:
Therefore, we utilized an in vivo model to assess the effects of mercury exposure on neuroimmune signaling. In prairie voles, 10 week mercury exposure (60 ppm $HgCl_2$ in drinking water) resulted in a male-specific increase in TNFα protein expression in the cerebellum and hippocampus. These findings are consistent with our previously reported male-specific mercury-induced deficits in social behavior and further support a role for heavy metals exposure in neuropathologies such as autism.
For example, levels of proinflammatory cytokines, including tumor necrosis factor alpha (TNFα), consistently are elevated in the central nervous system (CNS) of autism patients [9, 35, 53

2.6.6.
A prospective assessment of porphyrins in autistic disorders: a potential marker for heavy metal exposure. =>
http://www.ncbi.nlm.nih.gov/pubmed/17000470
Abstract only
Coremessages:
An apparent dose-response effect was observed between autism severity and increased urinary coproporphyrins
Patients with non-chelated autism had significantly increased median coproporphyrin levels versus controls.

2.6.7.
Blood levels of mercury are related to diagnosis of autism: a reanalysis of an important data set. =>
http://www.ncbi.nlm.nih.gov/pubmed/18006963
Abstract only
Coremessage:
the hair sample analysis results offer some support for the idea that persons with autism may be less efficient and more variable at eliminating mercury from the blood.

2.6.8.
Mercury, lead, and zinc in baby teeth of children with autism versus controls.=>
http://www.ncbi.nlm.nih.gov/pubmed/17497416
Abstract only
Coremessage:
Children with autism had significantly (2.1-fold) higher levels of mercury but similar levels of lead and similar levels of zinc.
higher use of oral antibiotics in the children with autism may have reduced their ability to excrete mercury, and hence may partially explain the higher level in baby teeth

2.6.9.
. A case series of children with apparent mercury toxic encephalopathies manifesting with clinical symptoms of regressive autistic disorders. =>
http://www.ncbi.nlm.nih.gov/pubmed/17454560
Abstract only
Coremessage:
Based upon differential diagnoses, 8 of 9 patients examined were exposed to significant mercury from Thimerosal-containing biologic/vaccine preparations during their fetal/infant developmental periods, and subsequently, between 12 and 24 mo of age, these previously normally developing children suffered mercury toxic encephalopathies that manifested with clinical symptoms consistent with regressive ASDs.

2.6.10.
Environmental mercury release, special education rates and autism disorder: an ecological study of Texas=>
http://www.ncbi.nlm.nih.gov/pubmed/16338635
Abstract only
Coremessage:
There was a significant increase in the rates of special education students and autism rates associated with increases in environmentally released mercury

2.6.11.
Age-dependent lower or higher levels of hair mercury in autistic children than in healthy controls.=>
http://www.ncbi.nlm.nih.gov/pubmed/20628443
Abstract only
Coremessage:
Autistic children significantly differed from healthy peers in the concentrations of mercury in hair: younger autistics had lower levels, while older - higher levels than their respective controls. The results suggest that autistic children differ from healthy children in metabolism of mercury, which seems to change with age.

2.6.12.
The plausibility of a role for mercury in the etiology of autism: a cellular perspective=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3173748/
FULL ACCESS TO STUDY
Coremessage:
Sehr umfangreich

2.6.13.
Efficacy of DMSA Therapy in a Sample of Arab Children with Autistic Spectrum Disorder=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3566884/
FULL ACCESS TO STUDY
Coremessage:
Our data supports evidence that detoxification treatment with oral DMSA has beneficial effect on ASD patients.

2.6.14.
Toxic Metals and Essential Elements in Hair and Severity of Symptoms among Children with Autism =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3484795/
FULL ACCESS TO STUDY

2.7. MERCURY & EPILEPSY

2.7.1.
Methylmercury: A Potential Environmental Risk Factor Contributing to Epileptogenesis=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3285480/
FULL ACCESS TO STUDY
Coremessage:
Patients or animals with acute or chronic MeHg poisoning often display epileptic seizures or show increased susceptibility to seizures, suggesting that MeHg exposure may be associated with epileptogenesis.

2.7.2.
Chronic low-dose maternal exposure to methylmercury enhances epileptogenicity in developing rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/10568690
Abstract only
Coremessage:
Effects of continuous low-dose maternal methylmercury intoxication on the induction and propagation of ictal epileptiform activity induced by 3-aminopyridine, were investigated on the neocortex of 4-weeks-old offspring rats
Epileptogenicity was significantly increased in offspring of mercury-treated animals compared to those of controls,

2.8. MERCURY, IMMUNITY & AUTOIMMUNITY

2.8.1.
Thiol compounds inhibit mercury-induced immunological and immunopathological alterations in susceptible mice =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1904555/pdf/cei0107-0068.pdf
FULL ACCESS TO STUDY
Coremessage:
In vitro mercury induces a high proliferative response in splenic lymphocytes and in vivo it induces a systemic autoimmune disease in susceptible mouse strains.

2.8.2.
Anti-fibrillarin autoantibodies in mercury-treated mice.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1534831/
FULL ACCESS TO STUDY
Coremessages:
Using indirect immunofluorescence (IF) with HEp-2 cells as a substrate serially bled SJL mice were found to gradually develop a high titre of anti-nucleolar antibodies (ANuA) after 3-5 weeks of s.c. injections of 1.6 mg $HgCl_2$/kg body weight every third day
ome mercury-treated SJL mice also developed a significantly increased titre of anti-histone antibodies of the IgM class.
In addition, a fraction of the mercury-treated SJL mice developed serum antibodies reacting with 10-15 and 60-70 kD nucleolar proteins in immunoblotting.

2.8.3.
Effects of mercury on the immune system.
http://www.ncbi.nlm.nih.gov/pubmed/9046578
No abstract available

2.8.4.
Mercuric chloride induces a strong immune activation, but does not accelerate the development of dermal fibrosis in tight skin 1 mice=>
http://www.ncbi.nlm.nih.gov/pubmed/15140057
Abstract only
Coremessage:
In susceptible mice, mercuric chloride induces a systemic autoimmune disease characterized by increased serum levels of immunoglobulin (Ig) G1 and IgE, production of anti-nucleolar autoantibodies (ANolA) and formation of renal IgG deposits.only
As a support for our hypothesis, we observed that in Tsk1/+ mice, B cells were spontaneously hyperactive and that treatment with mercury induced a strong immune/autoimmune response in these mice, but not in their non-Tsk (+/+) littermates. This response was characterized by the formation of high numbers of splenic IgG1, IgG2b and IgG3 antibody-secreting cells, increased serum levels of IgE, production of IgG1 antibodies against single-stranded DNA (ssDNA), trinitrophenol (TNP) as well as thyroglobulin and the development of renal IgG1 deposits.

2.8.5.
Immunology of mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/19076354
Abstract only
Coremessage:
. In genetically susceptible mice or rats, subtoxic doses of mercury induce the production of highly specific autoantibodies as well as a generalized activation of the immune system.

2.8.6.
Mercury compounds and the immune system: a review.=>
http://www.ncbi.nlm.nih.gov/pubmed/9524402
Abstract only
Coremessages:
Repeated administration of mercuric chloride to rats, mice and rabbits can induce autoimmune response and a membranous nephropathy

2.8.7.
Toxicology of Autoimmune Diseases=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3076021/
FULL ACCESS TO STUDY
Coremessage: Mercury großer Abschnitt gewidmet.

2.8.8.
Mercury exposure, serum antinuclear/antinucleolar antibodies, and serum cytokine levels in mining populations in Amazonian Brazil: A cross-sectional study=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2873228/
FULL ACCESS TO STUDY
Coremessages:
We have reported elevated levels of ANA and ANoA in human populations exposed to mercury in artisanal gold mining
We measured ANA, ANoA, and cytokine concentrations in serum and compared results from mercury-exposed artisanal gold miners to those from diamond and emerald miners working under similar conditions and with similar socioeconomic status and risks of infectious disease. Mercury-exposed gold miners had higher prevalence of detectable ANA and ANoA and higher titers of ANA and ANoA as compared to diamond and emerald miners with no occupational mercury exposure.

2.8.9.
Methyl mercury-induced autoimmunity in mice.=>
http://www.ncbi.nlm.nih.gov/pubmed/9931279
Abstract only
Coremessage:
Therefore, demethylation of MeHg probably increased the concentration of inorganic mercury in the body sufficiently to reactivate the immune system. This reactivation indicated that genetically susceptible mice are not resistant to challenge with mercury, making them distinctly different from rat

2.8.10.
Autoreactive T cells in mercury-induced autoimmunity. Demonstration by limiting dilution analysis.=>
http://www.ncbi.nlm.nih.gov/pubmed/2974423
Abstract only
Coremessage:
Mercuric chloride is responsible in Brown-Norway rats for an autoimmune disease that is autoregulated. Previous studies have shown that this agent induces T cell-dependent polyclonal B cell activation in these rats.

2.8.11.
Mercury-induced autoimmunity in mice.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241265/
FULL ACCESS TO STUDY
Coremessage:--

2.8.12.
Immunosuppressive and autoimmune effects of thimerosal in mice.=>
http://www.ncbi.nlm.nih.gov/pubmed/15808517
Abstract only
Coremessage:
The lymph node expression of IL-2 and IL-15 mRNA was increased after 2 days, and of IL-4 and IFN-gamma mRNA after 6 and 14 days. During the first 14 days treatment, the number of splenocytes, including T and B cells as well as Ig-secreting cells decreased.
In conclusion, the organic mercury compound thimerosal (EtHg) has initial immunosuppressive effects similar to those of MeHg. However, in contrast to MeHg, thimerosal treatment leads in genetically susceptible mice to a second phase with strong immunostimulation and autoimmunity, which is T-cell dependent, H-2 linked and may at least partly be due to the inorganic mercury derived from the metabolism of ethyl mercury.

2.8.13.
Mercury and autoimmunity: implications for occupational and environmental health.=>
http://www.ncbi.nlm.nih.gov/pubmed/16023690
Abstract only
Coremessage:
Mercury (Hg) has long been recognized as a neurotoxicant; however, recent work in animal models has implicated Hg as an immunotoxicant
In particular, Hg has been shown to induce autoimmune disease in susceptible animals with effects including overproduction of specific autoantibodies and pathophysiologic signs of lupus-like disease

2.8.14.
Xenobiotic metal-induced autoimmunity: mercury and silver differentially induce antinucleolar autoantibody production in susceptible H-2s, H-2q and H-2f mice=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1808646/
FULL ACCESS TO STUDY
Coremessage:
Xenobiotic-metals such as mercury (Hg) and silver (Ag) induce an H-2 linked antinucleolar autoantibody (ANolA) production in susceptible mice

2.8.15.
Organic mercury compounds and autoimmunity.=>
http://www.ncbi.nlm.nih.gov/pubmed/15990073
Abstract only
Coremessage:
Recent studies have confirmed that organic mercurials such as methyl mercury (MeHg) and ethyl mercury (EtHg) are much more potent immunosuppressors than inorganic mercury (Hg). However,
Recent studies in mice with a susceptible genotype has revealed that the immunosuppressive effect of MeHg and EtHg will within 1-3 weeks be superseded by immunostimulation causing an HgIA-like syndrome.

2.8.16.
Mercury-induced autoimmunity in the absence of IL-4=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1905075/
FULL ACCESS TO STUDY
Coremessage:

In susceptible H-2s mice, mercuric chloride (HgCl2) induces an autoimmune syndrome characterized by production of anti-nucleolar antibodies (ANoA) and increased serum levels of IgG1 and IgE antibodies.

2.8.17.
Mechanism of mercury-induced autoimmunity: both T helper 1- and T helper 2-type responses are involved.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2326774/
FULL ACCESS TO STUDY
Coremessage:
To further explore the role of Th1/Th2 cytokines in the mercury model, we performed anti-interferon-γ antibody treatment in IL-4-deficient mice together with mercury treatment and found that the production of IgG2a and IgG3, but not IgG2b, antibodies was downregulated. This indicated that besides Th2-type cytokines, Th1-type and other cytokines were involved as well in mercury-induced autoimmune response.

2.8.18.
Interleukin-4 gene expression in mercury-induced autoimmunity.=>
http://www.ncbi.nlm.nih.gov/pubmed/7871386
Abstract only
Coremessage:
Mercuric chloride (HgCl2) induces autoimmunity in Brown Norway (BN) rats, with necrotizing vasculitis in the gut

2.8.19.
Mercury-induced renal autoimmunity in BN-->LEW.1N chimeric rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/8168152
Abstract only
Coremessage:
Repeated exposure to relatively low doses of mercuric chloride causes a variety of autoimmune responses in rats of the Brown Norway (BN) strain.

2.8.20.
Mechanisms of heavy metal-induced autoimmunity.=>
http://www.ncbi.nlm.nih.gov/pubmed/15829271
Abstract only
We review in this article some of the mechanisms by which heavy metal exposure can lead to autoimmunity.

2.8.21.
Mercury-induced renal autoimmunity: changes in RT6+ T-lymphocytes of susceptible and resistant rats.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1519727/
FULL ACCESS TO STUDY
Coremessage: --

2.8.22.
Autoimmunity-inducing metals (Hg, Au and Ag) modulate mast cell signaling, function and survival.=>
http://www.ncbi.nlm.nih.gov/pubmed/22103852
Abstract only
Coremessage:
The three heavy metals, mercury, gold and silver commonly and specifically induce aberrant immunological responses leading to autoimmune disorders in genetically susceptible animals and humans
Furthermore, the metals have considerable impacts on mast cell survival, which also species seems to be involved in the development of metal-induced autoimmune disorders.

2.8.23.
Mercury and silver induce B cell activation and anti-nucleolar autoantibody production in outbred mouse stocks: are environmental factors more important than the susceptibility genes in connection with autoimmunity?=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2665687/
FULL ACCESS TO STUDY
Coremessages:
Four weeks of treatment with both mercury and silver induced a strong B cell activation characterized by increased numbers of splenic antibody-secreting cells of at least one or more immunoglobulin (Ig) isotype(s) in all treated stocks.

The three stocks also exhibited a marked increase in the serum IgE levels in response to mercury, but not silver.
Thus, the findings of this study suggest that long-term exposure to certain environmental factors can activate the immune system to produce autoimmunity per se, without requiring specific susceptible gene

2.8.24.
Autoimmunity and heavy metals.=>
http://www.ncbi.nlm.nih.gov/pubmed/7704000
Abstract only
Coremessage:
Similarly, there is solid evidence that mercury can induce autoimmune disease both in humans and experimental animals.

2.8.25.
Mercury Induces an Unopposed Inflammatory Response in Human Peripheral Blood Mononuclear Cells in Vitro=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2799469/
FULL ACCESS TO STUDY
Coremessage:
Dysregulation of cytokine signaling appears to play an important role in the etiology of Hg-induced autoimmunity in animal models

2.9. MERCURY , THYROID & ENDOCRINOLOGY

2.9.1.
Mercury and thyroid autoantibodies in U.S. women, NHANES 2007-2008.=>
http://www.ncbi.nlm.nih.gov/pubmed/22280926
Abstract only
Coremessage:
Results suggest an association between mercury and thyroglobulin autoantibody positivity

2.9.2.
Effects of mercury on the endocrine system.=>
http://www.ncbi.nlm.nih.gov/pubmed/19545200
No abstract available

2.9.3
The endocrine effects of mercury in humans and wildlife.=>
http://www.ncbi.nlm.nih.gov/pubmed/19280433
Abstract only
Coremessage:
Mercury (Hg) is well studied and research continues as our knowledge of its health risks increases. One expanding area of research not well emphasized to date is the endocrine effects of Hg. This review summarizes the existing literature on the effects of Hg on the endocrine system and identifies gaps in the knowledge.

2.9.4.
Metals in blood and urine, and thyroid function among adults in the United States 2007-2008.=>
http://www.ncbi.nlm.nih.gov/pubmed/23044211
Abstract only
Coremessage:
When including all blood metals, mercury was associated with decreases in T(3) and T(4)

2.9.5.
To breed or not to breed: endocrine response to mercury contamination by an Arctic seabird.=>
http://www.ncbi.nlm.nih.gov/pubmed/23720523
Abstract only

Coremessages:
These results suggest that mercury contamination may disrupt GnRH input to the pituitary
Thus, high mercury concentration could affect the ability of long-lived birds to modulate their reproductive effort (skipping or breeding) according to ongoing environmental changes in the Arctic, thereby impacting population dynamics.

2.9.6.
The Endocrine Disruptive Effects of Mercury =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2723593/pdf/12199_2008_Article_BF02931255.pdf
FULL ACCESS TO STUDY
Coremessages:
2.9.7.
The endocrine disruptive effects of mercury have recently become one of the major public concerns.
In this report, the adverse effects of mercury on the hypothalamus, pituitary, thyroid, adrenal gland, and gonads (testis and ovary) in laboratory animals as well as in humans are reviewed.

2.9.8.
Endocrine disruptor & nutritional effects of heavy metals in ovarian hyperstimulation=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3241832/
FULLACCESS TO STUDY
Coremessage:
Hair mercury concentration showed a negative correlation with oocyte yield ($p<0.05$,βcoefficient 0.38) and follicle number ($p=0.03$,β coefficient0.19) after ovarian stimulation.

2.9.9.
The effects of metals as endocrine disruptors.=>
http://www.ncbi.nlm.nih.gov/pubmed/19466673
Abstract only
Coremessage: --

2.9.10.
Suppressed adrenocortical responses and thyroid hormone levels in birds near a mercury-contaminated river.=>
http://www.ncbi.nlm.nih.gov/pubmed/19731714
Abstract only
Coremessage:
Nestlings from the contaminated sites had blood Hg concentrations that exceeded those from the reference sites by more than an order of magnitude (354 +/- 22 vs 17 +/- 1 ppb wet weight
adrenocortical responses, plasma triiodothyronine, and thyroxin concentrations were suppressed, relative to reference levels, by the end of the nestling period.
These results suggest that (1) Hg may disrupt endocrine systems of terrestrial avian young and (2) adverse effects of Hg on endocrine systems may be most evident once endocrine axes are fully developed.

2.9.11.
Thyroid hormones in pregnancy in relation to environmental exposure to organochlorine compounds and mercury.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280346/
FULL ACCESS TO STUDY
Coremessage:
and mercury are global envirThe aim of this study was to examine thyroid hormone levels during pregnancy and in cord blood in relation to blood concentrations of organochlorine compounds (OCs) and Hg in healthy women recruited during pregnancy. onmental contaminants that can disrupt the endocrine system in animals and humans.
Cord serum free thyroxin was negatively correlated with inorganic Hg. These results suggest that at even low levels of exposure, persistent environmental contaminants can interfere with thyroid status during pregnancy

2.9.12.
Thyrotoxicity of the chlorides of cadmium and mercury in rabbit.=>
http://www.ncbi.nlm.nih.gov/pubmed/1449659
Abstract only
Coremessages:
The present study was aimed at establishing a direct relationship between heavy metal poisoning and thyroid

dysfunction
Cadmium and mercury treatment at LD50 levels resulted in severe thyrotoxicosis in the rabbit
Within 24 h of intramuscular administration of cadmium chloride 15 mg.kg-1 body weight (bw) and mercury chloride 20 mg.kg-1 bw, thyroid peroxidase activity increased significantly over the control with a concomitant rise in the triiodothyronine (T3) titre.
On the other hand, there was a remarkable fall in the thyroxine (T4) level, and the T3/T4 ratio was high as compared with the control.
Evidence indicates that acute heavy metal lethality will induce immediate hyperthyroidism.

2.9.13.
Postnatal endocrine dysfunction induced by prenatal methylmercury or cadmium exposure in mice=>
http://www.ncbi.nlm.nih.gov/pubmed/722189
Abstract only
Coremessage:
The subtle and delayed effects of two heavy metals, cadmium and mercury, on the pituitary-adrenal axis of mice were examined
Exposure to methylmercury resulted in diminished hepatic metabolism of corticosterone in vitro due to a loss of liver mass.

2.9.14.
In vitro inhibition of thymulin production in mercury-exposed thymus of young mice=>
http://www.ncbi.nlm.nih.gov/pubmed/11327382
Abstract only
Coremessage:
Thymulin production decreased by 70, 74, 82 and 86% and by 55, 66, 73 and 81% for mercury concentrations of 10(-6) M and 10(-8) M, respectively, after 2, 4, 5 and 6 h.
Mercury toxic effect on thymulin kinetics may be directly exerted to thymulin synthesis in epithelial cells, although it is less dramatic than that of cycloheximide (CHX), known as a potent inhibitor of protein synthesis in such cells

2.9.15.
Thyroid Hormones and Methylmercury Toxicity=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3637991/
FULL ACCESS TO STUDY
Coremessage:
This review addresses the possibility that high exposures to the organometal, methylmercury (MeHg), may perturb neurodevelopmental processes by selectively affecting thyroid hormone homeostasis and function.

2.9.16.
In vitro and in vivo effects of mercuric chloride on thymic endocrine activity, NK and NKT cell cytotoxicity, cytokine profiles (IL-2, IFN-gamma, IL-6): role of the nitric oxide-L-arginine pathway.=>
http://www.ncbi.nlm.nih.gov/pubmed/16428073
Abstract only
Coremessages:
Mercury (Hg2+) affects cell-mediated immunity, including thymulin production.
Since L-arginine is the substrate for NO production, it may compensate for the cell-mediated immune defect induced by HgCl2, via the arginine-NO-pathway. L-arginine is also able to reduce glomerular kidney IgG antibodies deposits induced by higher dose of HgCl2 administration.

2.9.17.
Estradiol reduces cumulative mercury and associated disturbances in the hypothalamus-pituitary axis of ovariectomized rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/16406600
Abstract only
Coremessage:
A decrease in plasma levels of luteinizing hormone (LH) was also detected after administration of 7.5 mg/kg MeHgCl. These disturbances in LHRH and LH secretion induced by mercury were abolished or superimposed (respectively) by estrogenic replacement therapy (0.025 mg/kg 17beta estradiol cypionate, intramuscular).

2.9.18.
Thyroid Hormones in Relation to Lead, Mercury, and Cadmium Exposure in the National Health and Nutrition Examination Survey, 2007–2008=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3569681/
FULL ACCESS TO STUDY
Coremessages:
Background: Heavy metals, such as lead (Pb), mercury (Hg), and cadmium (Cd), are known toxicants, but their associations with the thyroid axis have not been well quantified at U.S. background levels.
Objectives: We investigated the relationships between thyroid hormones (total and free thyroxine [TT4 and FT4], total and free triiodothyronine [TT3 and FT3], thyroid-stimulating hormone [TSH], and thyroglobulin [Tg]) and levels of Pb, Hg, and Cd in blood and Cd in urine.
In adults, blood Hg was inversely related to TT4, TT3, and FT3 and urinary Cd was positively associated with TT4, TT3, FT3, and Tg, but there were no associations with Pb
Our analysis suggests an inverse association between Hg exposure and thyroid hormones,

2.9.19.
Endocrine function in mercury exposed chloralkali workers=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1128033/
FULL ACCESS TO STUDY
Coremessages:
OBJECTIVE--The aim was to study whether functional impairment of the pituitary, thyroid, testes, and adrenal glands of humans occupationally exposed to mercury (Hg) vapour can be shown as a result of accumulation of Hg in these glands.
The serum free T4 concentration and the ratio free T4/free T3 were slightly, but significantly, higher in the subgroups with the highest exposure, and the serum free T3 was inversely associated with cumulative Hg exposure. This indicates a possible inhibitory effect of mercury on 5'-deiodinases, which are responsible for the conversion of T4 to the active hormone T3.

2.9.20.
Adverse effects of mercuric chloride on thyroid of mice, Musculus albinus and pattern of recovery of the damaged activity.=>
http://www.ncbi.nlm.nih.gov/pubmed/15303713
Abstract only
Coremessage:
Daily treatment of HgCl2 for 7, 14 and 21 days decreased serum cholesterol, TPO and T4 activity
Simultaneous administration of EPL (25 mg/mice) restored thyroid function in mice by maintaining serum thyroid hormone concentration almost normal.

2.9.21.
Effect of long-term uptake of mercuric sulphide on thyroid hormones and glutathione in mice=>
http://www.ncbi.nlm.nih.gov/pubmed/1450564
No abstract available

2.9.22.
Effect of mercury on glutathione and thyroid hormones.=>
http://www.ncbi.nlm.nih.gov/pubmed/2322688
No abstract available

2.9.23.
Thyroid function in chronic mercury poisoning =>
http://www.ncbi.nlm.nih.gov/pubmed/14428756
No abstract available

2.9.24.
[Changes of thyroid function in chronic mercury poisoning=>
http://www.ncbi.nlm.nih.gov/pubmed/19280433
No abstract available

2.9.25.
The relation between human exposure to mercury and thyroid hormone status.=>
http://www.ncbi.nlm.nih.gov/pubmed/22426797
Abstract only

Coremessage:
The aim of this study was to investigate total mercury (THg) and methylmercury (MeHg) exposure of 75 mother-child pairs in relation to their thyroid hormone status (thyroid-stimulating hormone (TSH), triiodothyronine (T3), free triiodothyronine (fT3), thyroxine (T4), and free thyroxine (fT4)).

2.9.26.
Mercury in molar excess of selenium interferes with thyroid hormone function in free-ranging freshwater fish.=>
http://www.ncbi.nlm.nih.gov/pubmed/22794667
Abstract only
Coremessages:
In organisms, binding of Hg in a Se-Hg complex results in a detoxification of Hg.
However, formation of Se-Hg complexes also affects Se bioavailability, disrupting functions of Se-dependent enzymes, such as TH deiodinases, which convert thyroxine (T4) to the physiologically active TH, triiodothyronine (T3)
This suggests that Se availability is impaired by Hg and results in altered selenoenzyme activities and loss of optimal control of TH balance in free-ranging freshwater fish.

2.9.27.
Effects of low mercury vapour exposure on the thyroid function in chloralkali workers.=>
http://www.ncbi.nlm.nih.gov/pubmed/11180271
Abstract only
Coremessages:
The median serum concentration of reverse triiodothyronine (rT3) was statistically significantly higher in the exposed subjects compared with the referents (268 pmol l (-1) and range 161-422 vs 240 pmol l(-1) and range 129-352; P = 0.009)
The difference between the exposed subjects and the referents was most pronounced in the highest exposed sub-groups
The free thyroxine (T4)/free T3 ratio was also higher in the highest exposed subgroups compared with the referents
The study could indicate a slight effect of low mercury vapour exposure on the function of the enzyme type I iodothyronine deiodinase, possibly modified by comparatively low urinary iodine concentrations.

2.9.28.
[Toxicologic hazards at the endocrine level of heavy metals=>
http://www.ncbi.nlm.nih.gov/pubmed/3842814
Abstract only
The effects of heavy metals (in particular Pb, Cd, Cr, Mn and Hg) on pituitary, thyroid, adrenal gland, pancreas and gonads are reviewed. The effect of these metals on the CNS centers regulating endocrine function is emphasized. On this time, the reviewed data stress the importance of studying the mechanisms of preclinical damage and the biological markers of it.

2.10. MERCURY & SOCIAL PHOBIA

NOTHING FOUND YET

2.11. MERCURY & SCHIZOPHRENIA

2.11.1
[Schizophrenia & mercury poisoning. Contribution to the study of the schizophreniforme psychoses due to mercury].=>
http://www.ncbi.nlm.nih.gov/pubmed/14950105
No abstract available

2.12. MERCURY & NEUROPATHIC PAIN

2.12.1
Neuropathic pain in children after exposure to mercury=>
http://www.ncbi.nlm.nih.gov/pubmed/19076591
No abstract available

2.12.2
Pain relief by carbamazepine in mercury poisoning.=>
http://www.ncbi.nlm.nih.gov/pubmed/9537499
No abstract available

2.12.3.
Mercury intoxication and neuropathic pain.=>
http://www.ncbi.nlm.nih.gov/pubmed/18312528
No abstract available

2.13. MERCURY & CFS (CHRONIC FATIGUE SYNDROME)

2.13.1.
The frequency of mercury intolerance in patients with chronic fatigue syndrome and healthy controls.=>
http://www.ncbi.nlm.nih.gov/pubmed/10416724
No abstract available

2.13.2.
Thimerosal, micromercurialism and chronic fatigue syndrome.=>
http://www.ncbi.nlm.nih.gov/pubmed/15780514
No abstract available

2.13.3.
[Reactions to metals in patients with chronic fatigue and autoimmune endocrinopathy=>
http://www.ncbi.nlm.nih.gov/pubmed/10951876
Abstract only
Coremessages:
Fatigue regardless of the underlying disease is primarily associated with hypersensitivity to inorganic and organic mercury, nickel, and gold.
Statistical analysis of data obtained from professionals and controls revealed a higher incidence of positivity to organic and inorganic mercury and nickel in professionals.

2.13.4.
Chronic Fatigue Syndrome (CFS) and related illnesses: A case history supporting subacute mercury poisoning or “micromercurialism” =>
http://www.medicalveritas.com/images/00071.pdf
Abstract only

2.13.5.
Improved Chronic Fatigue Symptoms after Removal of Mercury in Patient with Increased Mercury Concentration in Hair Toxic Mineral Assay: A Case Report =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3481032/
FULL ACCESS TO STUDY
Coremessage:
In the current case, we learned the patient had consumed many slices of raw tuna and was initially

diagnosed with chronic fatigue syndrome.
Our patient's toxic chronic fatigue symptoms improved after he was given mercury removal therapy, indicating that he was correctly diagnosed with chronic exposure to organic mercury.

2.13.6.
Mercury toxicity presenting as chronic fatigue, memory impairment and depression: diagnosis, treatment, susceptibility, and outcomes in a New Zealand general practice setting (1994-2006).=>
http://www.ncbi.nlm.nih.gov/pubmed/16891999
Abstract only
Coremessage:
In a group of 465 patients diagnosed as having chronic mercury toxicity (CMT), 32.3% had severe fatigue, 88.8% had memory loss, and 27.5% had depression.

2.14.MERCURY , OXIDATIVE STRESS , MITOCHONDRIA & APOPTOSIS

2.14.1.
Oxidative damage to nuclic acids in motor neurons containing mercury =>
http://www.ncbi.nlm.nih.gov/pubmed/9741394
Abstract only
Coremessages:
In each control-mercury pair (four pairs per group) significantly more perikaryal fluorescence was seen in mercury-containing than in control motor neurons (Mann-Whitney testing).
Mercury within the motor neuron perikaryon therefore leads to increased avidin binding, an indicator of oxidative damage to DNA. The findings support the hypothesis that an environmental toxin such as mercury can enter and damage motor neurons.

2.14.2.
Lead and mercury mutagenesis: role of H2O2, superoxide dismutase, and xanthine oxidase.=>
http://www.ncbi.nlm.nih.gov/pubmed/9654245
Abstract only
Coremessage:
hese results demonstrate that Pb2+ and Hg2+ disrupt the redox status of AS52 cells by enhancing the activities of CuZn-SOD and XO. Furthermore, the results of these studies also demonstrate that there is a causal relationship between the induction of H2O2 by these metals and mutagenesis.

2.14.3.
Oxidative metabolism of neutrophils in vitro and human mercury intolerance.=>
http://www.ncbi.nlm.nih.gov/pubmed/20654420
Abstract only
Coremessages:
Neutrophils from 22 patients and 15 healthy controls were exposed in vitro to mercuric chloride and phenyl mercuric acetate in increasing doses and the superoxide anion production of the isolated cells was measured using the NBT (nitroblue tetrazolium) test
A significant difference in the NBT reduction of unstimulated neutrophils and one concentration of mercuric chloride was found between the tolerant and intolerant patients.
Neutrophils from tolerant patients showed a peak NBT value at lower concentrations of mercuric chloride than did cells from the healthy controls and the intolerant patients

2.14.4.
Mercury induced time-dependent alterations in lipid profiles and lipid peroxidation in different body organs of cat-fish Heteropneustes fossilis.=>
http://www.ncbi.nlm.nih.gov/pubmed/2708789
Abstract only
Coremessages:
The effects of mercuric chloride (HgCl2) on lipid profiles and lipid peroxidation in different body organs of fresh water cat-fish Heteropneustes fossilis were studied.

The daily exposure of HgCl2 0.2 mg/L for 10, 20 and 30 days depleted the total lipids in brain. But the content of phospholipids enhanced significantly at 30 days.
Significant increment in lipid peroxidation was discernible in brain, liver and muscle. In kidney the rate of lipid peroxidation was significantly reduced. The results suggest that exposure of HgCl2 enhances the peroxidation of endogenous lipids in brain, liver and muscle.

2.14.5.
Chelation in Metal Intoxication=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2922724/
FULL ACCESS TO STUDY
Coremessage: In dieser sehr umfangreichen Arbeit findet sich der Abschnitt: "Oxidative Stress in Metal Toxicity and the Role of Antioxidants"

2.14.6.
Induction of oxidative stress by non-lethal dose of mercury in rat liver: possible relationships between apoptosis and necrosis=>
http://www.ncbi.nlm.nih.gov/pubmed/21186712
Abstract only
Coremessage:
The elevations of aspartate transaminase (AST) and alanine transaminase (ALT) levels measured exhibited increase of 287.5 and 214.5% after 48 hr of exposure respectively which were found to be highly significant compared with control.

2.14.7.
Cytotoxicity of inorganic mercury in murine T and B lymphoma cell lines: involvement of reactive oxygen species, Ca(2+) homeostasis, and cytokine gene expression.=>
http://www.ncbi.nlm.nih.gov/pubmed/12849721
Abstract only
Coremessages:
Mercury concentration-dependently decreased cell viability, membrane integrity, and proliferation in both EL4 and A20 cells
Mercury increased the reactive oxygen species (ROS) production in both EL4 and A20 cells, and pretreatment with antioxidants reversed mercury-induced ROS generation
Mercury increased gene expression of IL-4 and TNFalpha in EL4 cells;

2.14.8.
Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnish men=>
http://www.ncbi.nlm.nih.gov/pubmed/7828289
Abstract only
Coremessages:
Even though previous studies have suggested an association between high fish intake and reduced coronary heart disease (CHD) mortality, men in Eastern Finland, who have a high fish intake, have an exceptionally high CHD mortality. We hypothesized that this paradox could be in part explained by high mercury content in fish.
These data suggest that a high intake of mercury from nonfatty freshwater fish and the consequent accumulation of mercury in the body are associated with an excess risk of AMI as well as death from CHD, CVD, and any cause in Eastern Finnish men and this increased risk may be due to the promotion of lipid peroxidation by mercury.

2.14.9.
Inorganic mercury causes pancreatic beta-cell death via the oxidative stress-induced apoptotic and necrotic pathways.=>
http://www.ncbi.nlm.nih.gov/pubmed/20006636
Abstract only
Coremessages:
Mercury is a well-known highly toxic metal. In this study, we characterize and investigate the cytotoxicity and its possible mechanisms of inorganic mercury in pancreatic beta-cells
The intracellular mercury levels were markedly elevated in HgCl2-treated HIT-T15 cells. Taken together, these results suggest that HgCl2-induced oxidative stress causes pancreatic beta-cell dysfunction and cytotoxicity involved the co-existence of apoptotic and necrotic cell death.

2.14.10.
Stress proteins and oxidative damage in a renal derived cell line exposed to inorganic mercury and lead=>
http://www.ncbi.nlm.nih.gov/pubmed/19720107
Abstract only
Coremessage:
Our results clearly demonstrated that mercury increases ROS and RNS levels and the expressions of Hsp25 and inducible Hsp72. These findings are corroborated by evident mitochondrial damage, apoptosis or necrosis.

2.14.11.
Renal oxidant injury and oxidant response induced by mercury =>
http://www.ncbi.nlm.nih.gov/pubmed/8872981
Abstracht only
Coremessages:
We demonstrate that I.L.C-PK1 cells, exposed to HgCl2, generate massive amounts of hydrogen peroxide, the latter completely quenched by the hydrogen peroxide scavenger, pyruvate.
The redox sensitive enzyme, heme oxygenase, was markedly up-regulated in the kidney in response to HgCl2
HgCl2 also induced members of the bcl family, bcl2 and bclx, genes that protect against apoptosis and oxidant injury
In summary, we demonstrate that HgCl2 potently stimulates renal generation of hydrogen peroxide in vitro and in vivo and such generation of peroxide contributes to renal dysfunction in vitro and in vivo.

2.14.12.
Accumulation of mercury and its effect on antioxidant enzymes in brain, liver, and kidneys of mice=>
http://www.ncbi.nlm.nih.gov/pubmed/10390852
Abstract only
Coremessage:
The results showed that the highest oral dose of mercury significantly increased antioxidant enzymes in kidneys and liver. The increased antioxidant enzymes enhance the antioxidant potential of the organs to reduce oxidative stress.

2.14.13.
Thimerosal-Derived Ethylmercury Is a Mitochondrial Toxin in Human Astrocytes: Possible Role of Fenton Chemistry in the Oxidation and Breakage of mtDNA =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3395253/
FULL ACCESS TO STUDY !

2.14.14.
Uncoupling effect of mercuric chloride on mitochondria isolated from an hepatic cell line.
http://www.ncbi.nlm.nih.gov/pubmed/11481667
Abstract only !

2.14.15.
Mercuric chloride: toxicity and apoptosis in a human oligodendroglial cell line MO3.13.=>
http://www.ncbi.nlm.nih.gov/pubmed/12504520
Abstract only
Coremessage:
These results indicate that HgCl(2) is toxic at low concentrations for oligodendroglial cells and that the MO3.13 cell line dies in an apoptotic manner when exposed to low concentrations of HgCl(2).

2.14.16.
Methylmercury Induces Acute Oxidative Stress, Altering Nrf2 Protein Level in Primary Microglial Cells =>
http://toxsci.oxfordjournals.org/content/116/2/590.full
FULL ACCESS TO STUDY !

2.14.17.
The impact of long-term past exposure to elemental mercury on antioxidative capacity and lipid peroxidation in mercury miners =>
http://www.ncbi.nlm.nih.gov/pubmed/15139389
Abstract only
Coremessage:

Among the observed lipid peroxidative products, the mean concentration of U-MDA was statistically higher ($p < 0.01$) in miners (0.21 micromol/mmol creatinine) than in the controls (0.17 micromol/mmol creatinine).

2.14.18.
Mercury Abolishes Neurotrophic Factor–Stimulated Jak-STAT Signaling in Nerve Cells by Oxidative Stress =>
http://toxsci.oxfordjournals.org/content/94/1/129.full.pdf
FULL ACCESS TO STUDY !

2.14.19.
Mercury-induced H2O2 production and lipid peroxidation in vitro in rat kidney mitochondria. =>
http://www.ncbi.nlm.nih.gov/pubmed/1768276
Abstract only !

2.14.20.
Mercury exposure, nutritional deficiencies and metabolic disruptions may affect learning in children=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2773803/
FULL ACCESS TO STUDY
Coremessage:
Nutritional deficiencies and mercury exposure have been shown to alter neuronal function and increase oxidative stress among children with autism.
Mercury, either individually or in concert with other factors, may be harmful if ingested in above average amounts or by sensitive individuals.

2.14.21.
Comparative cytotoxicity of cadmium and mercury in a human bronchial epithelial cell line (BEAS-2B) and its role in oxidative stress and induction of heat shock protein 70.=>
http://www.ncbi.nlm.nih.gov/pubmed/17454561
Abstract only
Coremessages:
Electron spin resonance (ESR) tests showed that hydroxyl radical generation was greater in the reaction of cells with Hg compared to Cd.
Depletion of sulfhydryl groups of cellular proteins and generation of ROS may be involved in metal-induced lung cell damage.

2.14.22.
Cytotoxicity of metal ions to human oligodendroglial cells and human gingival fibroblasts assessed by mitochondrial dehydrogenase activity=>
http://www.ncbi.nlm.nih.gov/pubmed/18023858
Abstract only
Coremessage:
whereas mercury showed the highest cytotoxic effects on HGF (TC50 74 microM) comparing with other tested metals.

2.14.23.
Rapid cell death induced by methyl mercury in suspension of cerebellar granule neurons.=>
http://www.ncbi.nlm.nih.gov/pubmed/2642297
Abstract only
Coremessage:
. The results suggest that additional cytotoxic mechanisms beyond perturbations of the main metabolic pathways are involved in the neurotoxic mechanism of action of MeHg in cerebellar granule neurons.

2.14.24.
Paradoxical effect of methyl mercury on mitochondrial protein synthesis in mouse brain tissue. =>
http://www.ncbi.nlm.nih.gov/pubmed/3627362
Abstract only !

2.14.25.
Methylmercury inhibits electron transport chain activity and induces cytochrome c release in cerebellum mitochondria. =>
http://www.ncbi.nlm.nih.gov/pubmed/21628953

Abstract only !

2.14.26.
Methylmercury has a selective effect on mitochondria in cultured astrocytes in the presence of [U-(13)C]glutamate. =>
http://www.ncbi.nlm.nih.gov/pubmed/11454325
Abstract only !

2.14.27.
Inhibition of mitochondrial ATPase by Hg++ ions. =>
http://www.ncbi.nlm.nih.gov/pubmed/150609
Abstract only !

2.14.28.
Mercuric chloride induces apoptosis via a mitochondrial-dependent pathway in human leukemia cells. =>
http://www.ncbi.nlm.nih.gov/pubmed/12505371
Abstract only !

2.14.29.
Mitochondria as an important target in heavy metal toxicity in rat hepatoma AS-30D cells. =>
http://www.ncbi.nlm.nih.gov/pubmed/18501399
Abstract only !

2.14.30.
Methylmercury (MeHg) elicits mitochondrial-dependent apoptosis in developing hippocampus and acts at low exposures =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3256128/
FULL ACCESS TO STUDY !

2.15. MERCURY & ADHS/ADHD

2.15.1.
Attention-deficit hyperactivity disorder and blood mercury level: a case control study in Chinese children=>
http://www.ncbi.nlm.nih.gov/pubmed/17177150
Abstract only
Coremessage:
High blood mercury level was associated with ADHD. Whether the relationship is causal requires further studies.

2.15.2.
Prenatal Methylmercury, Postnatal Lead Exposure, and Evidence of Attention Deficit/Hyperactivity Disorder among Inuit Children in Arctic Québec=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3491943/
FULL ACCESS TO STUDY
Coremesssage:
Cord blood mercury concentrations were associated with higher TRF symptom scores for attention problems and DBD scores consistent with ADHD

2.15.3.
Low blood levels of lead and mercury and symptoms of attention deficit hyperactivity in children: a report of the children's health and environment research (CHEER).=>
http://www.ncbi.nlm.nih.gov/pubmed/19100765
Abstract only
Coremessage:
The observed association between blood lead concentration and the appearance of ADHD symptoms in Korean children suggests that lead, even at low concentrations, is a risk factor for ADHD.

2.16. MERCURY & DIVERENT SYNDROMS

2.16.1.
Apallic syndrome in chronic mercury poisoning.=>
http://www.ncbi.nlm.nih.gov/pubmed/199444
Abstract only
Coremessage: --

2.16.2.
Minimal-change nephrotic syndrome due to occupational mercury vapor inhalation=>
http://www.ncbi.nlm.nih.gov/pubmed/19761728
Abstract only
Coremessage:
A 25-year-old man developed nephrotic syndrome and severe hypertension following occupational exposure to mercury vapor whilst working at a fluorescent light factory

2.16.3.
Stevens-Johnson syndrome in a child with chronic mercury exposure and 2,3-dimercaptopropane-1-sulfonate (DMPS) therapy.=>
http://www.ncbi.nlm.nih.gov/pubmed/18568806
Abstract only
Coremessage:
The reported association suggests that SJS may be a potential complication of DMPS therapy, and this should be considered in the risk-benefit analysis of chelation.

2.16.4.
Baboon syndrome induced by mercury - first case report in China.=>
http://www.ncbi.nlm.nih.gov/pubmed/17577379
Abstract only
Coremessage:
A case of mercury-induced baboon syndrome was reported. A 31-year-old woman presented with itching papules and vesicles in the right axilla, which extended to the left axilla, arms, fossa poplitea, buttocks, and groin. A mercury thermometer was broken 2 days before exanthema.

2.16.5.
Kawasaki's disease, acrodynia, and mercury.=>
http://www.ncbi.nlm.nih.gov/pubmed/19075648
Abstract only
Coremessage:
Coinciding with the largest increase (1985-1990) of thimerosal (49.6% ethyl mercury) in vaccines, routinely given to infants in the U.S. by 6 months of age (from 75microg to 187.5microg), the rates of Kawasaki's Disease increased ten times, and, later (1985-1997), by 20 times.

2.16.6.
Mucocutaneous lymph node syndrome: is there a relationship to mercury exposure=>
http://www.ncbi.nlm.nih.gov/pubmed/2801647
No abstract available

2.16.7.
Reference levels of blood mercury and association with metabolic syndrome in Korean adults.=>
https://www.ncbi.nlm.nih.gov/m/pubmed/23824410/?i=2&from=/17784549/related
Abstract only
Coremessage:
Furthermore, blood Hg is associated with metabolic syndrome, in which Hg exposure may play a role as a possible risk factor for cardiovascular diseases.

2.16.8.
Case report: heavy metal burden presenting as Bartter syndrome.=>
http://www.ncbi.nlm.nih.gov/pubmed/21194246

Abstract only
Coremessage: --

2.16.9.
BRONCHITIS DUE TO ACUTE MERCURY INHALATION. REPORT OF TWO CASES.=>
http://www.ncbi.nlm.nih.gov/pubmed/14068440
No abstract available

2.16.10.
Morvan's fibrillary chorea and acrodynic syndrome following mercury treatment =>
http://www.ncbi.nlm.nih.gov/pubmed/6522913
Abstract only
Coremessages:
Blood and particularly urine mercury levels were elevated
Administration of dimercaprol (BAL) considerably increased urinary excretion of mercury and there was progressive improvement and finally recovery after two months of BAL treatment.This case exemplifies the possible co-existence of fibrillary chorea and acrodynia

2.16.11.
A whitened face woman with nephrotic syndrome.=>
http://www.ncbi.nlm.nih.gov/pubmed/12500245
Abstract only
Coremessage:
The authors report on a 34-year-old Indonesian domestic helper who presented with nephrotic syndrome secondary to membranous nephropathy. It was subsequently found that she used a skin whitening cream regularly that was found to contain a mercury level of almost 2,000 times above the allowable limit.

2.16.12.
Nephrotic syndrome after contact with mercury. A report of five cases, three after the use of ammoniated mercury ointment.=>
http://www.ncbi.nlm.nih.gov/pubmed/13866316
No abstract available

2.16.13.
Was Young's syndrome caused by exposure to mercury in childhood?=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1697782/
FULL ACCESS TO STUDY
Coremessage:
The decline in incidence of Young's syndrome in those born after 1955 is similar to that observed with pink disease, suggesting that both conditions may have had a similar aetiology--mercury intoxication

2.16.14.
Mercury inhalation poisoning and acute lung injury.=>
http://www.ncbi.nlm.nih.gov/pubmed/9735669
Abstract only
Coremessage:
We experienced a patient with acute respiratory distress syndrome (ARDS) after illicit use of mercury vapor for hemorrhoid treatment; he developed acute chemical pneumonitis following exposure to mercury vapor.

2.16.15.
Minimal change disease caused by exposure to mercury-containing skin lightening cream: a report of 4 cases.=>
http://www.ncbi.nlm.nih.gov/pubmed/23537684
Abstract only
Coremessage:
With regard to renal pathology, apart from membranous nephropathy, minimal change disease should be included as another pathological entity caused by mercury exposure or intoxication.

2.16.16.
Fanconi syndrome caused by mercury chloride poisoning=>
http://www.ncbi.nlm.nih.gov/pubmed/2756202
No abstract available

2.16.17.
Genitourinary diseases mortality in mercury miners=>
http://www.ncbi.nlm.nih.gov/pubmed/17175931
Abstract only
Coremessage:
his paper shows an excess in the mortality due to genitourinary diseases, specially in nephritis, nephrotic syndrome and nephrosis, whose estimation and significance increases when compared to the population of Castilla-La Mancha.

2.16.18.
Toxicological evaluation of two children diagnosed as Munchausen syndrome by proxy.=>
http://www.ncbi.nlm.nih.gov/pubmed/23094539
Abstract only
Coremessage:
Toxicology detected 3.5 ng/ml mercury (Hg) in the fluid and 9.4 microg Hg/g creatinine in the urine

2.16.19.
Systemic allergic dermatitis syndrome caused by mercury: a reply=>
http://www.ncbi.nlm.nih.gov/pubmed/18844708
No abstract avilable

2.16.20.
[Transient tubular syndrome due to intoxication with an organic mercury compound (Glyceromerfen) in a one and one-half-year old girl=>
http://www.ncbi.nlm.nih.gov/pubmed/4702989
No abstract avaliable

2.17. MERCURY & PARKINSON

2.17.1.
Parkinson's disease, macular degeneration and cutaneous signs of mercury toxicity.=>
http://www.ncbi.nlm.nih.gov/pubmed/16832218
Abstract only !
Coremessage:
Mercury may play a role in the etiology of Parkinson disease and Grover's disease.

2.17.2.
Epidemiologic study on the association between body burden mercury level and idiopathic Parkinson's disease. =>
http://www.ncbi.nlm.nih.gov/pubmed/2725805
Abstract only !

2.17.3.
[Parkinsonism in chronic occupational metallic mercury intoxication]. =>
http://www.ncbi.nlm.nih.gov/pubmed/15098329
Abstract only !
2.17.4.
[Parkinson disease, mercury and other heavy metals]. =>
http://www.ncbi.nlm.nih.gov/pubmed/7900145
No abstract available !!

2.17.5.
Parkinson's disease mortality and the industrial use of heavy metals in Michigan. =>
http://www.ncbi.nlm.nih.gov/pubmed/8419812
Abstract only

2.17.6.
The enigma of parkinsonism in chronic borderline mercury intoxication, resolved by challenge with

penicillamine. =>
http://www.ncbi.nlm.nih.gov/pubmed/8784840
Abstract only (Fall eines Exzahnarztes) !

2.17.7.
Occupational metal exposures and the risk of Parkinson's disease.=>
http://www.ncbi.nlm.nih.gov/pubmed/10545782
Abstract only !

2.17.8.
Industrial toxicants and Parkinson’s disease =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3299826/
Abstract only !

2.17.9.
Parkinson's Disease and Mercury =>
http://www.researchgate.net/publication/224954026_Parkinson's_Disease_and_Mercury/file/9fcfd50b9da1f89514.pdf
FULL ACCESS TO STUDY !

2.17.10.
Toxic Exposures and Parkinsons: the Mercury Connection Bernard Windham(Ed.)- Chemical Engineer/Biostatistician =>
http://www.flcv.com/parkins.html
FULL ACCESS TO STUDY !

2.17.11.
Parkinson's disease and occupational exposure to organic solvents, agricultural chemicals and mercury--a case-referent study.=>
http://www.ncbi.nlm.nih.gov/pubmed/7347910
Abstract only
Coremessages
The outcome of the study does not support the hypothesis that occupational exposure to organic solvents in general increases the risk of Parkinson's disease, but the confidence intervals of the odds ratios do not rule out such possibilities.

2.17.12.
Parkinson's disease, macular degeneration and cutaneous signs of mercury toxicity.=>
http://www.ncbi.nlm.nih.gov/pubmed/16832218
Abstract only !
Coremessage:
Mercury may play a role in the etiology of Parkinson disease and Grover's disease.

TEIL 3.

SPECULTATIONS & POSSIBILITIES , POSSIBLE CHAINS OF CAUSALITIES

3.1.POSSIBLE CHAIN OF CAUSALITY Nr. 1 : (Mobilefunc ,Mercury , Dental Amalgam & Thyroidhormones)

According to Hall Huggins , TSH -levesls go up in case of heavy metall intoxication ! http://curezone.com/forums/fm.asp?i=2024767 So they do too, if a person is a frequent mobilphone user and this phaenomenon has already been investigated by a Study (Study Nr. 2) , that confirmed that there is a distinct influence by frequent mobilephone use on thyroid hormone levels , esp. TSH . Frequent mobile phone use leads to increasing TSH-levels and to a derangement of thyroid hormones .

As we can see in Study Nr. 1 it could be found ,that mobile phone us can also lead to increasing release of mercury from dental amlagam fillings. ! So , the question is: Couldn't it be possible , that the REAL reason for increasing TSH Levels ist not mobilefunc by it's self , but through it's mercury releasing effect ? To answer this question it would be usefull to contact both authors of both studies to bring them together for the purpose of answering this question by conducting a FURTHER study in which this possibility could be investigated.

For example in this way: Arrange to groups of heavy mobile phone users ! One group bears dental amalgam fillings , the other one does not ! Investigate the TSH (& thyroid hormone leves) of both groups.

3.1.1.
Study Nr. 1 finds that mobile phone use leads to increasing Hg release from dental amalgam restorations:

Mercury release from dental amalgam restorations after magnetic resonance imaging and following mobile phone use.=>
http://www.ncbi.nlm.nih.gov/pubmed/18819554
Abstract only
Coremessage:
It appears that MRI and microwave radiation emitted from mobile phones significantly release mercury from dental amalgam restoration.

3.1.2.
Study Nr.2 finds that frequent mobile phone use leads to increasing TSH – Levels :

Alterations in TSH and Thyroid Hormones following Mobile Phone Use=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3243874/
FULL ACCESS TO STUDY
Coremessage:
The levels in the students who severely used mobile phones were 1.18±0.30, 7.75±1.14 and 3.75±2.05 respectively. In non-users, the levels were 1.15±0.27, 8.42±2.72 and 2.70±1.75, respectively. The difference among the levels of TSH in these 3 groups was statistically significant ($P<0.05$).

3.2. POSSIBLE CHAIN OF CAUSALITY Nr. 2 : (GSTM1 null in (nearly) all known neurodegenerative diseases)

The enzyme Glutathion -S -Transferase Type M1 , GSTM1 plays a major role in detoxification of esp. aromatic toxins (mainly out of the family of benzoles , phenoles & polyphenole componds) and heavy metals. It is the „instrument" that is responsible for the coupling of these compunds (&heavy metals) to its associated carrier „Glutathion through which these toxins can be excreted by the body through its natural pathways . I.e. Mainly by bile & feces. If this pathway of excretion is blocked through the lack of this enzyme , mercury (and other toxins) must be stored within the body , or , they are excreted through other ways , as we can find in study Nr. 3 . The Gene GSTM1 codes for the expression of this enzyme , Glutathion S – Transferase. If there is a deletion of this Gen (GSTM1 NULL) , the body of the carrier of this genetic deficiency can not handle and excrete (these) toxins. This increases the susceptibility of the subject to esp. Mercury and (very probably) to every other toxin as well.

Mercury is one of the most powerfull (neuro)-toxins we know and i think and i believe that Mercury plays the most dominant role in all possible (chronic!) inroxications ,men can acquire ! Not only because mercury ist the most potent neurotoxin we know , but also because of its enormous ubiquity ! Finaly ,. nearly every one carries this toxin within his mouth ! Out of these reason it is obvoius , that mercury should be responsible for the MOST FREQUENT (CHRONIC) intoxications in men (& wildlife too.).

Becaus of this fact , and becaus of the fact that GSTM1 plays such an important role in the natural mercury detoxification of our body , i began to investigate & research the frequeny of GSTM1 NULL cases in relation to all known neurodegenerative diseases we know on pubmed and to my surprise i found studies , that showed , THAT IN (NEARLY) ALL KNOWN AND FREQUENT NEURODEGENARTIVE DISEASES GSTM1 NULL . TYPS ARE DOMINANT !!

Out of this reason and out of the fact that GSTM1 ist the most important natural „detoxifier" of our bodies of Mercury - as shown in study Nr. 3 - it is quite obvious for me , that mercury **should** play the most dominant role in the generation of all these neurodegenerative diseases..

3.2.1.
Study Nr. 3 finds that GSTM1 plays a central role in the ability of our body to detoxify our bodies of mercury:

Glutathione-S-transferase polymorphism, metallothionein expression, and mercury levels among students in Austria.=
http://www.ncbi.nlm.nih.gov/pubmed/17716707
Abstract only
Coremessage:
We focused on the relationship between polymorphisms in glutathione-S-transferase (GST) genes and mercury concentrations in blood, urine, and hair. The correlation between blood mercury levels, GSTT1 and GSTM1 polymorphism, and gene expression of certain metallothionein subgroups (MT1, MT3) was evaluated in a further group of students (N=30).
hair mercury concentrations are significantly increased in persons with the double deleted genotype (GSTT1-/- and GSTM1-/-) as compared to persons with the intact genotype
We conclude that the epistatic effect of the GSTT1 and the GSTM1 deletion polymorphism is a risk factor for increased susceptibility to mercury exposure

In all of the following neurological disorders and neurodegenerative diseases , the GSTM1 NULL Type is dominant:

3.2.2.
GSTM1 0/0 Genotype dominant in Parkinson ! =>
http://www.ncbi.nlm.nih.gov/pubmed/18591034
Abstract only
Coremessage:
We have examined the occurrence of GSTM1 null, one of the glutathione S-transferase mu genes, in a control and a Parkinson's disease group. By using the polymerase chain reaction (PCR) we found 67% of non-expressors compared with 51% in a control group (chi(1)(2) = 5.535; p < 0.025). These results suggest that a deletion of the GSTM1 gene may be associated with a susceptibility to Parkinson's diseas

3.2.3.
GSTM1 0/0 Genotype dominant in Epilepsy ! =>
http://www.ncbi.nlm.nih.gov/pubmed/12499585
Abstract only
Coremessage:
The frequency of GSTM1(-) of the epileptics with intractable seizure was significantly higher than that of the group with good seizure control and that of the normal subjects.

3.2.4.
GSTM1 0/0 Genotype dominant in Schizophrenia ! =>
http://www.ncbi.nlm.nih.gov/pubmed/11181039
Abstract only
Coremessage:
Our findings suggest that the GSTM1*0 is associated with an increased susceptibility to schizophrenia, particularly disorganized type of the disease. It is therefore likely that the GSTM1 gene deletion constitutes to vulnerability for disease states of this kind, rather than being the direct cause of schizophrenic conditions.

3.2.5.
GSTM1 0/0 Genotype dominant in Multiple Sclerosis (here at least in women) ! =>
http://www.ncbi.nlm.nih.gov/pubmed/17437619
Abstract only
Coremessage:
A significantly increased frequency of GSTM1 null genotype was found amongst female patients (65.5%) as compared with males (33.3%, P =0.04)

3.2.6.
GSTM1 0/0 Genotyp dominant bei Chronik Fatigue Syndrom ! =>
http://www.umweltmedizin.de/content/articles/511/539/348/index.html?catid=348&artid=5146&nosum=1

3.2.7.
GSTM1 0/0 Genotyp dominant in Autism ! =>
http://www.faqs.org/patents/app/20080293058#ixzz1VCYYLKPC
Coremessage: sehr umfangreiche Analyse.

3.2.8.
GSTM1 NULL dominant in anxiety ,depression & emotional disorders =>
http://www.ncbi.nlm.nih.gov/pubmed/22661588
Coremessages:
Patients with homozygous GSTM1 gene deletion reported higher anxiety (mean null genotype = 47.3 ± 9.2, non-null = 43.9 ± 7.8; P = .04), more depression (null = 51.0 ± 9.8, non-null = 47.0 ± 9.4; P = .03), and more global distress (null = 50.2 ± 9.7, non-null = 45.2 ± 9.9; P = .01).
GSTM1 gene deletion was consistently associated with greater psychological distress in medulloblastoma survivors across multiple domains, suggesting that this genotype may predispose patients for increased emotional late effects.

3.2.9.
GSTM1 NULL dominant in Alzheimer =>
http://www.ncbi.nlm.nih.gov/pubmed/22381228
Coremessages:
Differences in genotype distributions between AD patients and controls were found only for the GSTM1 null genotype

Our outcome suggests that the GSTM1 null genotype is a risk factor for AD in Italian patients.

3.3. POSSIBLE CHAIN OF CAUSALTIY Nr.3 : (Mercury & oxidative Stress , MDA elevated in all neurodegenerative Diseases)

MDA (Malone dialdehyde) is the most important marker for oxidative stress and it is a marker for lipid peroxidaton. The higher this marker is meassured , the higher the degree of lipid peroxidation , induced -- for example – through toxins like mercury. When lipid peroxidation is high , the level of this marker (MDA ,Malone dialdehyde) rises. In study Nr. 4 it is shown ,that havy metals lead to lipid peroxidation. If lipid peroxidation occurs , the marker MDA (Malone dialdehyde) rises. So we can find in Study Nr. 5. that mercury miners are chronicly exposed to mercury an that their „corresponding“ MDA – levels are (chronicly) elevated to.

And – as with GSTM1 – out of this reason and because of the fact , that mercury ist one of the most potent neurotoxins that exist AND because of its ubiquity i guessed the possibility , that in all known neurodegenerative disorders MDA levels SHOULD be elevated in case toxins , esp. Mercury. are/is the primary candidate as a cause for these dieseases & disorders. Out of this possible perspective and possibility , i did a research on pubmed and i was NOT dissapointed ! My findings showed , THAT IN ALL CASES OF NEUREDEGENERATIVE DISEASES AND KNOWN NEUROLOGICAL DISORDERS, MDA LEVELS ARE ELEVATED !!

Could this be a mere coincidence ? No ! With respect to these search results , i dont believe so (any more) ! This result – in association to my findings in relationship to GSTM1- shows (should show) AT LEAST ONE certainty: That (environmental) toxins – is it mercury or not -- are THE MAIN CAUSE OF ALL THIS DISORDERS ! And , as mercury ist the most potent neurotoxin on earth and because at the same time mercury ist the MOST UBIQUITOUS toxin , mercury should be considered as the main culprit as a cause for all these diseases..

3.3.1.
Study 4 finds & shows that exposer to heavy metals (mercury) leads to lipid peroxidation & elevated MDA -levels:

Experimentally induced lipid peroxidation after exposure to chromium, mercury or silver: interactions with carbon tetrachloride.=<
http://www.ncbi.nlm.nih.gov/pubmed/1579547
Abstract only
Coremessage:
Chromium, along with other heavy metals, induces an increased production of malondialdehyde (MDA), an indicator of lipid peroxidation, in liver and kidney tissue of NMRI male mice.

3.3.2.
Study 5 finds that MDA-levels in mercury miners are (prob.chronicly) elevated:

The impact of long-term past exposure to elemental mercury on antioxidative capacity and lipid peroxidation in mercury miners.=>
http://www.ncbi.nlm.nih.gov/pubmed/15139389
Coremessage:
Among the observed lipid peroxidative products, the mean concentration of U-MDA was statistically higher ($p < 0.01$) in miners (0.21 micromol/mmol creatinine) than in the controls (0.17 micromol/mmol creatinine). I

3.3.3.
MDA elevated in Epilepsy:

Increased plasma malondialdehyde associated with cerebellar structural defects =>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1717310/
FULL ACCESS TO STUDY
Coremessages:
Measurements of MDA concentrations in plasma were compared among healthy children (n=31), patients with neurological disorders or epileptic syndromes (n=15)
Compared with healthy controls and the neurological/epileptic group, the 31 children with pontocerebellar structural defects had significantly increased MDA values

3.3.4.
MDA elevated in Restles leg syndrome:

Assessment of nitric oxide, advanced oxidation protein products, malondialdehyde, and thiol levels in patients with restless legs syndrome.=>
http://www.ncbi.nlm.nih.gov/pubmed/22469072
Abstract only
Coremessage:
Increased advanced oxidation protein products, malondialdehyde levels, and decreased thiol and nitric oxide levels, may suggest that patients with RLS are under oxidative stress.

3.3.5.
MDA elevated in Autismus:

Evaluation of oxidative stress in autism: defective antioxidant enzymes and increased lipid peroxidation. =>
http://www.ncbi.nlm.nih.gov/pubmed/20845086
Abstract only
Coremessages:
. We compared levels of SOD, GSH-Px, and MDA in children with autism and controls.
In children less than 6 years of age, levels of SOD, and GSH-Px were significantly lower in autistic children compared with their controls, while MDA was significantly higher among patients than controls.

3.3.6.
MDA elevated in Schizophrenia & Bipolar Disorder:

Lipid peroxidation and antioxidant enzyme levels in patients with schizophrenia an bipolar disorder=>
http://perweb.firat.edu.tr/personel/yayinlar/fua_1019/1019_24209.pdf
FULL ACCESS TO STUDY
Coremessage:
There was a significant increase in MDA levels of patients with schizophrenia and bipolar disirder compared with controls.

3.3.7.
MDA elevated in (adult) ADHD :

Malondialdehyde levels in adult attention-deficit hyperactivity disorder.=>
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2077350/
FULL ACCESS TO STUDY
Coremessage:
To evaluate the biochemical basis of adult attention-deficit hyperactivity disorder (A-ADHD), we compared lipid peroxidation status in the plasma of A-ADHD patients, and that of control subjects without A-ADHD by quantifying the levels of malondialdehyde (MDA), an end product of fatty acid oxidation
The mean (standard deviation [SD]) MDA levels in patients (2.44 [0.84] nmol/mL) were significantly higher than those of control subjects (0.36 [0.20] nmol/mL) ($t = 11.013$, $df = 39$, $p < 0.01$)

3.3.8.
MDA (dramaticaly) elevated in Obsessive-Compulsive Disorder:

Serum Antioxidant Vitamins and Malondialdehyde Levels in Patients with Obsessive-Compulsive Disorder =>
http://www.gjpsy.uni-goettingen.de/gjp-article-shohag.pdf
FULL ACCESS TO STUDY
Coremessage:
MDA levels were found significantly higher in OCD subjects

3.3.9.
MDA elevated in Fibromyalgia:

Free radicals and antioxidants in primary fibromyalgia: an oxidative stress disorder? =>
http://www.ncbi.nlm.nih.gov/pubmed/14689230
Abstract only !
Coremessagse:
The role of free radicals in fibromyalgia is controversial. In this study, 85 female patients with primary fibromyalgia and 80 age-, height-, and weight-matched healthy women were evaluated for oxidant/antioxidant balance
Malondialdehyde levels were significantly higher and superoxide dismutase levels significantly lower in fibromyalgic patients than controls.

3.3.10.
MDA (dramaticaly) elevated in Social Phobia Patients:

Antioxidant enzyme and malondialdehyde levels in patients with social phobia.=>
http://www.ncbi.nlm.nih.gov/pubmed/18339429
Abstract only
Coremessage:
The mean MDA, SOD, GSH-Px and CAT levels in the patient group were significantly higher than those in the control group.

3.3.11.
MDA elevated in Neuropathic pain:

Role of oxidative stress in pathophysiology of peripheral neuropathy and modulation by N-acetyl-L-cysteine in rats.=>
http://www.ncbi.nlm.nih.gov/pubmed/16214382
Abstract only
Coremessage:
The malondialdehyde levels of ligated sciatic nerves were significantly increased compared to non-ligated sciatic nerves (sham operated).

3.3.12.
MDA elevated in chronic fatigue (CFS) Syndrome:

Blood parameters indicative of oxidative stress are associated with symptom expression in chronic fatigue syndrome.=>
http://www.ncbi.nlm.nih.gov/pubmed/10905542
Abstract only
Coremessages:
Full blood counts, ESR, CRP, haematinics and markers for oxidative stress were measured for 33 patients diagnosed with chronic fatigue syndrome
CFS patients had increases in malondialdehyde ($P < 0.006$), methaemoglobin ($P < 0.02$), mean erythrocyte volume ($P < 0.02$) and 2,3-diphosphoglycerate ($P < 0.04$) compared with controls.

3.3.13.
MDA (dramaticaly) elevated in Alzheimer:

Determination of malonaldehyde in Alzheimer's disease: a comparative study of high-performance liquid chromatography and thiobarbituric acid test..=>
http://www.ncbi.nlm.nih.gov/pubmed/9222750
Abstract only
Coremessage:
Significant increases in the concentration of MDA of SDAT subjects were found in comparison with the two other groups, indicating that the measurement of MDA in erythrocytes could be used as a marker of oxidative damage in Alzheimer's disease.

3.3.14.
MDA elevated in Parkinson:

Oxidative stress indicators are elevated in de novo Parkinson's disease patients.=>
http://www.ncbi.nlm.nih.gov/pubmed/10568214
Abstract only
Coremessage:
The increased levels of reactive oxidative species (malondialdehyde content and superoxide radical production) in peripheral blood, and excessive activity of protective enzymatic systems (glutathione reductase Cu, and Zn-superoxide-dismutase) could indicate an additional systemic reaction related to a chronic oxidative stress state in the brain

3.3.15.
MDA elevated in MS (Mulitple Sclerosis) - Patients:

[Parameters of antioxidant protection in multiple sclerosis].
http://www.ncbi.nlm.nih.gov/pubmed/19891349
Abstract only
Coremessage:
A study of parameters of oxidant and antioxidant systems has been carried out in 79 patients with multiple sclerosis (MS) and in 75 mentally and physically healthy subjects.
The analysis of peripheral blood parameters in patients with MS has showed the substantial increase of pro-oxidant processes (malondialdehyde level).

-ENDE-

LETZTE AKTUALISIERUNG DER LISTE: JULI 2013