Bulletin Board

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CHEMWATCH

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ASIA PACIFIC

Changes to the public section of the Australian Inventory of Chemical Substances (AICS) and to related AICS features

2012-09-21

The National Industrial Chemicals Notification & Assessment Scheme (NICNAS) recently published a notice informing users of the Australian Inventory of Chemical Substances (AICS) of a number of updates made to the AICS search function, AICS forms and public AICS records. The AICS is central to NICNAS' role of regulating industrial chemicals by providing a legal mechanism for distinguishing between existing chemicals and new chemicals. Maintaining the accuracy of the AICS requires that NICNAS regularly monitors the AICS and corrects errors and inaccuracies when NICNAS becomes aware of them. The effectiveness of the role the AICS plays in regulating industrial chemicals is not only affected by its accuracy, but also depends upon an efficient search facility. Maintaining both these aspects – the accuracy of the AICS and the usability of the search facility – also results in fewer calls to the AICS Manager. Recent concurrent developments have impacted on the utility of the AICS:

- Following on from the recent Chemical Abstract Service (CAS) audit of the public AICS, the CAS numbers and names of several chemicals have been updated. NICNAS would like to encourage Industry to adopt these updated chemical details, however we are also aware that Industry will need time to adapt to these changes. To allow for this, NICNAS has incorporated a number of new features in the AICS and the AICS search function.
- Further improvements to the AICS search function have been made following responses to the 2010 NICNAS survey of stakeholders. The survey results indicated that AICS users required improvements to be made to the usability of the AICS search tool, particularly for the less frequent users of the AICS, whom the survey found are generally less confident in their ability to use and interpret AICS search outcomes.
- In addition to these improvements, changes to other AICS functions and AICS forms have been made following the Cost Recovery Impact Statement (CRIS). As advised in the "Changes to AICS fees: 2012-13" the non-confidential (public) AICS search service provided by NICNAS for an administrative fee, is no longer available as of 1 July 2012 due

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to a lack of demand for the service following the advent of the online search tool.

- As advised in the March and August 2012 Chemical Gazette Notices, NICNAS has been removing some chemicals ineligible for listing on the AICS, so as to improve its accuracy.

As a result of the above drivers, NICNAS has made specific improvements to the public AICS records, search function and associated guidance material. These changes, which are designed to assist users of the AICS adjust to such things as updated particulars for some chemicals, and to provide a more efficient and effective search experience, are:

1. Improvements following the CAS audit:
    a. Updated CAS numbers and names on the AICS.
    b. Creation of a new field entitled "Superseded CAS No", to enable searching using superseded CAS numbers for chemicals amended as a result of the CAS audit changes.
    c. A text warning informing searchers that they have searched using a superseded CAS number will appear in red text at the top of search page results.
    d. Superseded chemical names have been moved to the "Associated Names" field, enabling searching using superseded names.
2. AICS search improvements following CRIS and the stakeholder surveys:
    a. As NICNAS no longer offers public AICS searches as a fee-for-service. Form AICS 4C (AICS Search Request Form – Non-Confidential Section) has been removed from the NICNAS web site.
    b. A pop-up warning has been added to warn searchers when an invalid CAS number has been entered, so searchers can recheck the CAS number before continuing with the search.
    c. The AICS search "No results returned" page, now includes possible reasons for a nil result to assist users in determining what to do next.
    d. Form AICS 5C (AICS Search Request Form – Confidential Section) has been modified to ensure users have correctly undertaken a search of the public (nonconfidential) AICS
3. Removal of ineligible chemicals from the AICS to improve its currency:
    a. Non-stoichiometric alloys are considered to be mixtures and have been removed from the AICS. Note that the component elements remain on the AICS.

b. Ions have been removed from the AICS as they are not considered to be eligible chemicals.

To assist users of the AICS to adjust to these changes, NICNAS has also included pointers on the AICS search tool and extra advice in the AICS search guidance.

NICNAS, 14 September 2012

http://www.nicnas.gov.au

Transition arrangements - Australian regulatory framework for biologicals

2012-09-21

On 31 May 2012, the Australian regulatory framework for biologicals came into effect. A three year transition period is specified in the Therapeutic Goods Act 1989 for manufacturers and sponsors of currently supplied biologicals to meet the new regulatory arrangements, including inclusion of biologicals in the Australian Register of Therapeutic Goods (ARTG). The transition arrangements are described in Appendix 12 of the Australian Regulatory Guidelines for Biologicals (ARGB) and the transition period ceases on 31 May 2014. A key date for sponsors of currently supplied biologicals is 30 November 2012. Sponsors will be able to continue to supply a product until the TGA has reached a decision, even in the unlikely event that this decision is reached after 31 May 2014 if:

A key date for sponsors of currently supplied biologicals is 30 November 2012.

- the biological was supplied prior to 31 May 2011 and
- an acceptable application is received by the Therapeutic Goods Administration (TGA) for inclusion of the biological into the ARTG on or before 30 November 2012.

The Australian Government has made special funding arrangements for funding of the direct regulatory costs for Australian publicly funded facilities and not-for-profit hospital supply units, and this continues until 31 May 2014. The 'direct regulatory costs' for biologicals include, but are not limited to:

- application fees
- dossier evaluation fees
- variation fees
- manufacturing inspection fees.

Facilities that have been given 'non-profit' status by the TGA in the past are required to re-apply to the TGA under the new arrangements. The

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transition arrangements for the Australian regulatory framework for biologicals apply to products that were supplied before 31 May 2011. New biologicals need to be included in the Australian Register of Therapeutic Goods (ARTG) before they can be legally supplied in Australia.

There are three types of currently supplied product:

1. Biologicals that were previously exempt from ARTG entry but licensed for GMP by the TGA are exempt from inclusion in the ARTG until 31 May 2014. Sponsors need to apply for inclusion of a biological in the ARTG for the product before the transition period ends. During this period existing requirements in relation to the GMP licence will continue to apply.
2. Biologicals that were previously exempt from both ARTG entry and GMP licensing requirements are exempt from these requirements until 31 May 2014. Sponsors need to: ensure that the manufacturer has applied to the TGA for a GMP licence after the GMP licence application has been made, the sponsor must lodge an application for inclusion of the biological in the ARTG during the transition period. It is recommended that sponsors of products previously exempt or excluded from ARTG inclusion submit an application to include their product in the ARTG as a biological before 30 November 2012. If an application is acceptable and is made before 30 November 2012, then a product will be able to be continually supplied until the TGA makes a decision on the application, even if this decision is made after 31 May 2014.
3. Biologicals currently being supplied as therapeutic goods and included in the ARTG will continue to be on the ARTG. Sponsors do not need to take any action. Current entries on the ARTG will continue to be regulated under the old requirements until the TGA creates a new entry on the biologicals part of the ARTG and cancels the current entry. This will occur as soon as practicable, and the TGA will notify the sponsor once this has been completed. Supply of the product can continue without interruption throughout this process. There will be no fees payable for transferring products to the biologicals part of the ARTG. Only one set of annual charges for ARTG inclusion will be imposed for that product during the year of transfer.

TGA, 18 September 2012

http://www.tga.gov.au/

Legislation

Taiwan to amend testing methods for synthetic food contact materials

2012-09-21

Taiwan's Food and Drug Administration (FDA) is consulting on amendments to the testing methods for formaldehyde-melamine food contact materials, according to the Executive Yuan Gazette. Interested parties should submit comments by 8 October 2012. Further information is available (in Chinese) at: http://gazette.nat.gov.tw/EG_FileManager/eguploadpub/eg018178/ch08/type3/gov70/num29/Eg.htm

Chemical Watch, 19 September 2012

http://chemicalwatch.com

Taiwan's Food and Drug Administration (FDA) is consulting on amendments to the testing methods for formaldehyde-melamine food contact materials, according to the Executive Yuan Gazette.

AMERICA

Chemical Security Efforts Criticised

2012-09-21

After six years and having spent nearly $500 million to establish a program to safeguard the nation's chemical facilities against terrorist threats, the Department of Homeland Security (DHS) has made little progress, Republican lawmakers and a government watchdog group have charged. "For all the support Congress has given over the years, [DHS's Chemical Facility Anti-Terrorism Standards (CFATS) program] should have more to show" than wasteful spending and delayed implementation, Rep. John M. Shimkus (R-Ill.) remarked at a hearing held on 11 September by a subcommittee of the House of Representatives' Energy & Commerce Committee. The DHS antiterrorism effort has been plagued with problems since its inception. An internal DHS memo late last year exposed flaws in the program, including poor hiring decisions and mismanagement of resources. CFATS requires high-risk chemical facilities to conduct vulnerability assessments and then design and implement site security plans that meet risk-based performance standards set by DHS. DHS Undersecretary Rand Beers told the subcommittee that DHS is "working as quickly as possible" to implement a 95-item action plan to correct deficiencies in CFATS. Beers said the security program covers 4,433 high-risk chemical facilities, of which 3,660 have developed security plans for review. Although DHS has given final approval to only two site security plans and conditional approval to 73 others, the department hopes to conduct 300 more approval inspections over the next year, noted

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David Wulf, head of DHS's Infrastructure Security Compliance Division. Expressing frustration with the program's delay, Rep. Tim F. Murphy (R-Pa.) said it would take "a few centuries" for DHS to inspect all of the covered facilities. Rep. Bill Cassidy (R-La.) asked why no DHS employees have been fired over the troubled program. "I see something that smacks of cronyism. Frankly, I'm wondering why we're giving you any money," he told the two DHS officials. Cathleen A. Berrick, managing director of the homeland security team at the Government Accountability Office, testified that DHS is "still in the early phase" of correcting course and making CFATS an effective regulatory program.

Chemical & Engineering News, 17 September 2012

http://pubs.acs.org/cen/news

AWWA Releases Response to New Lead Reduction Act

2012-09-21

After a shareholder meeting with the EPA, AWWA releases 10 suggestions on how to best implement a new lead reduction act.

During a shareholder meeting on 16 August, the United States Environment Protections Agency called for members to submit opinions and suggestions that they may have regarding the implementation of the "Reduction of Lead in Drinking Water Act" (Public Law 111-380). This law outlines two basic objectives: lowering the allowable amount of lead in pipes, fittings, and plumbing fixtures coming into contact with water and creating a small number of exemptions to this lead-free definition. In response to this, the American Water Works Association released a detailed statement of ideas regarding the law and how best to integrate it into the water industry. In this statement, released 31 August, AWWA outlined ten recommendations about moving forward toward clean water. Calling for a simple and direct execution of this act, AWWA's suggestions ranged from focusing on those two objectives to shying away from elaborate implementations of this act. "It is essential that the Agency facilitate timely implementation of Public Law 111-380 and not introduce additional uncertainty or complicate work that is already underway to achieve compliance with the law's intent," said Thomas Curtis, AWWA's deputy executive director, in a letter accompanying the suggestions. The full list of recommendations go to: http://www.awwa.org/publications/breakingnewsdetail.cfm?itemnumber=59443

Environmental Protection News, 6 September 2012

http://www.eponline.com

Legislation

Canada to stop opposing listing asbestos as hazardous

2012-09-21

Recently, Canada dropped its long time opposition to the international listing of asbestos as a hazardous material, a designation intended to curb the use abroad of the fire-resistant substance, which can cause cancer and other illnesses. Canada had been the main opponent of such a listing, which would require exporters to warn importing countries of the hazards of asbestos, and would allow countries to ban its import. The listing would not of itself ban its sale. Industry Minister Christian Paradis said he made the decision as a logical consequence of plans by Quebec's provincial premier-designate, Pauline Marois, effectively to end the production of the substance. Quebec is the only place in Canada where it's produced. Canada had long worked against the listing under the United Nations' Rotterdam Convention, fearful that it would put Quebec asbestos mining out of business. "It would be illogical for Canada to oppose the inclusion of chrysotile (asbestos) in...the Rotterdam Convention when Quebec, the only province that produces chrysotile, will prohibit its exploitation," Paradis said. He made the announcement in Thetford Mines, his birthplace in the heart of his electoral district, and once a huge player in asbestos production. Canada has been the only Western developed country to export asbestos, which is estimated to kill more than 100,000 people around the world every year. It had continued to export it even though it strictly regulated its use domestically. From 1900 through 2003, it accounted for one-third of all worldwide production of all types of asbestos, according to the U.S. Geological Survey. Only Kazakhstan and Russia collectively produced more. But asbestos production had been dwindling in Canada. According to the U.S. Geological Survey, it dropped to fifth on the list of asbestos producers in 2011, with production less than a third of what it had been five years previously. Only six countries were producing quantifiable amounts of asbestos last year: Brazil, Canada, China, India, Kazakhstan and Russia. It is still used to strengthen cement products and roof shingles, and the industry says those uses are safe. Successive Canadian governments had refused to step in and ban asbestos production, arguing that if used appropriately it was not harmful, but they were embarrassed at international meetings when they defended its use. The outgoing Liberal government in Quebec had announced a C$58 million ($60 million) loan to restart the Jeffrey Mine, which would have been the only active asbestos mine in the province, but the incoming

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Parti Quebecois, elected on 4 September, has pledged to cancel that loan, with the resulting end of asbestos output in Canada.

Reuters Health, 14 September 2012

http://www.reuters.com/news/health

EUROPE

EU to get better safety monitoring of dangerous drugs

2012-09-21

The potentially harmful effects of medicinal products will be better monitored in the future as changes to existing EU legislation, adopted on 11 September, tighten up the European system. Problems with medical products in any member state will also be evaluated. The new rules - agreed between the European Parliament and EU ministers - will introduce an automatic emergency procedure, including an EU safety evaluation and possible EU-wide withdrawal if, for example, a member state wants to withdraw a medicinal product from the market. The procedure would also be triggered if a company decided not to renew a marketing authorisation for safety reasons. This new system is designed to prevent any more cases like that of the French diabetes drug Mediator, which allegedly caused between 500 and 2000 deaths. The Mediator case showed that there are loopholes in the EU system that needs to be closed. Mediator (benfluorex) was authorised to treat diabetes, but was also prescribed as an appetite suppressant. It was on the market for over 30 years in France, Portugal, Luxembourg, Greece, Italy and Spain. Although concerns about it surfaced in 1999, it was not withdrawn on the biggest market, France, until 2009. "It's a shame that it often takes a scandal to bring about higher standards in legislation," said parliamentary rapporteur Linda McAvan (Socialist and Democrats, Britain)."But the changes agreed today will hopefully close the loopholes that the Mediator case has highlighted - by strengthening the hand of regulators, and by putting companies under a lot more scrutiny" she added. French MEP Gilles Pargneaux (S&D) stated that it is in Europe that adverse effects of drugs must be identified, assessed and prevented. "After the affair with Mediator, the European Commission conducted a stress test that "identified weaknesses" in the current legislation. This work has led to these revisions," Pargneaux said. The changes to the EU legislation will also force companies to be more transparent. If for example a company withdraws a medicinal product from the market, it will have to state explicitly whether

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it has done so for safety reasons. The aim is to determine whether the commercial reasons sometimes given by companies for withdrawing a product in fact was due to safety concerns. In addition, the European Medicines Agency will have to set up a system to make sure that all new medicines and any medicines for which regulators have ongoing safety concerns are labelled with a black symbol. This should enable patients and healthcare professionals to identify them. The new rules will enter into force in 2013.

Euractiv, 12 September 2012

http://www.euractiv.com/

JRC reviews measurement requirements for implementation of EU Commission nano definition

2012-09-21

The European Commission's Joint Research Centre (JRC) has published a report reviewing the measurement methods available that can be used to classify materials under the definition for nanomaterial adopted by the European Commission in October 2011. The report details the requirements for particle size measurement of nanomaterials based on the definition and reviews the capabilities and limitations of currently-available measurement methods. Related issues, such as the implementation of the definition through methods other than measurement or methods for the detection of specific nanomaterials are expected to be addressed in a follow up report. To read the full report go to: JRC report

The European Commission's Joint Research Centre (JRC) has published a report reviewing the measurement methods available that can be used to classify materials under the definition for nanomaterial adopted by the European Commission in October 2011.

Chemical Watch, 18 September 2012

http://chemicalwatch.com

http://chemicalwatch.com

Businesses seek to block EU's ban on harmful greenhouse gases

2012-09-21

Businesses trying to block a European Union ban on hydrofluorocarbons (HFC) appear to have started "a lobbying frenzy" to ensure they can continue to use the powerful greenhouse gases in refrigerators, foams and air conditioning systems. The European Commission is currently reviewing its legislation on restricted fluorinated (F) gases, which includes

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HFCs. The gases can often be thousands times more harmful than carbon dioxide and are estimated to account for about two per cent of EU greenhouse gases (GHGs). But transparency organisation Corporate Europe Observatory (CEO) is concerned that industries using F-gases have launched a multi-million Euro campaign to prevent ambitious restrictions from coming into force. In a report published 13 September, CEO found a spike in the number of companies and trade associations with an interest in F-gases registering their lobbying interests with the EU at the end of 2011. More than 50 F-gas organisations and firms joined the EU Transparency Register, which lists organisations seeking to influence EU policy. CEO cannot prove the companies' ambitions, because it is optional for lobbyists to state their cause on the register and many choose not to do so. However, it is concerned they could be seeking to repeat the success of the 2006 lobbying battle in which the HFC industry successfully avoided a ban on HFCs. Green groups and industry lobbyists promoting alternative refrigeration systems are also campaigning for an outright ban on F-gases in the new legislative proposals. However, CEO found that green groups' voices and spending power were outnumbered by at least 10 to one by the mainstream HFC industry. Registered HFC industry organisations declared a total lobbying budget of €23.9m (£19.2m). In contrast, eight green groups lobbying on HFCs declared a budget of €2.2m and another eight companies supporting natural refrigerants declared €900,000. "This is a classic case of vested interests in industry mobilising massive financial resources to block effective climate regulation," report author David Leloup said. "Industry lobbyists are targeting the EU in a bid to be able to continue with business as usual, regardless of the fact that safe, cost-effective alternatives are readily available on the market, and regardless of the devastating impact on the climate."

Business Green, 14 September 2012

http://www.businessgreen.com

REACH Update

New version of the QSAR Toolbox to be introduced in a workshop in November

2012-09-21

The European Chemicals Agency (ECHA), in collaboration with the OECD, has invited industry users to a workshop introducing version 3.0 of the QSAR Toolbox on 20 November in Helsinki. Companies have been requested to express their interest in participating to the event by 17 October. The workshop aims to present the latest version of the OECD QSAR Toolbox. Emphasis will be put on how registrants can use the OECD QSAR Toolbox version 3.0 for filling gaps in (eco)toxicity data to fulfil their information requirements. In addition, ECHA and the OECD wish to collect feedback from industry on the use of the Toolbox and identify needs for implementing additional features in the tool and for providing other kind of support. Version 3.0 will contain many new features, hence a live demo will be an integral part of the event. A maximum of 30 participants is foreseen for this workshop and every effort will be made to have a representative audience from different industry sectors. ECHA has asked that expressions of interest for the workshop be lodged by 17 October 2012 by filling in the webform. New features of the QSAR Toolbox 3.0 include:

- Inclusion of additional data sources
- Advanced search engine
- 22 new mechanistically and endpoint specific profiling schemes
- Quantitative mixtures toxicity prediction
- Tautomeric set prediction
- Prediction accounting for metabolism
- Implementation of Adverse Outcome Pathways (AOPs) related to skin sensitisation supplemented by three new databases containing AOPs data for skin sensitisation.
- Four new simulators (autoxidation and hydrolysis)
- Enhanced reporting engine to handle mixtures, tautomers and metabolites

The QSAR Toolbox is an OECD software tool developed for filling data gaps in (eco)toxicity data needed for assessing the hazards of chemicals and for grouping chemicals into categories. It can also be used for the development of integrated testing strategies. Importantly, REACH

The European Chemicals Agency (ECHA), in collaboration with the OECD, has invited industry users to a workshop introducing version 3.0 of the QSAR Toolbox on 20 November in Helsinki.

registrants can use the QSAR Toolbox as a tool to generate an alternative method to animal testing to fulfil their information requirements.

ECHA, 13 September 2012

http://echa.europa.eu

ECHA urges 574 registrants of intermediates to improve the quality of their dossiers

2012-09-21

The European Chemicals Agency (ECHA) has conducted a new IT-based screening of all REACH intermediate registration dossiers, which has raised serious quality and potential compliance concerns. The concerned registrants have been directly informed via REACH-IT and are requested to review and update their dossiers with correct information over the next three months. After this period, ECHA plans to screen these dossiers again and identify those that will be subject to further regulatory actions. REACH allows intermediates manufactured and used under strictly controlled conditions to be registered with reduced information on their properties and without a chemical safety report. ECHA has earlier reported on the outcome of previous screenings of intermediate dossiers undertaken in 2010 and 2011. Those screenings raised serious concerns in terms of compliance. The most problematic dossiers that seemed to be inconsistent with the definition of intermediates and/or respecting strictly controlled conditions have been pursued through clarification requests and where needed compliance checks. Therefore, there are substances currently registered as intermediates for which potentially important information on their hazards and risks has not been gathered. ECHA has now undertaken a more systematic IT-screening of the approximately 5 500 registrations for intermediates. The analysis of the reported uses in these dossiers revealed that 2388 dossiers included uses that do not, or are very unlikely to, fulfil the definition of intermediates and/or are used under strictly controlled conditions. These dossiers with deficiencies and a potential for incompliance represent 760 substances. The Agency has sent letters to 574 registrants with potentially non-compliant intermediate registrations, asking them to carefully review the reported uses and update their registration dossiers within three months. In addition, ECHA has added to this letter practical advice for registrants on how to better report intermediates in IUCLID 5.4 or how to update the registration to a full Article 10 Registration. Following the end of the three month period, ECHA will undertake a new screening of dossiers and identify those that require regulatory action. More information about the IT-screening and examples

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of the observed deficiencies can be found in a detailed background document: http://echa.europa.eu/documents/10162/13583/intermediate_status_scc_background_note_en.pdf

ECHA, 14 September 2012

http://echa.europa.eu

ECHA strengthens its compliance check strategy

2012-9-21

The European Chemicals Agency (ECHA) is introducing a new systematic targeting of compliance checks based on specified concerns. This will increase the chances for dossiers of insufficient compliance to be scrutinised and ensure higher compliance of REACH registrations. To ensure increased compliance of REACH registration dossiers, ECHA carries out both full compliance checks of the dossiers and now more effectively also targets its evaluation to specific parts of them. In a full compliance check, ECHA addresses the full dossier content in a single evaluation exercise, especially for randomly selected registration dossiers. This means that ECHA performs a systematic evaluation of all information requirements in the technical dossier (e.g. physico-chemical, environmental and human health endpoints), including the corresponding elements and conclusions provided in the chemical safety report (i.e. PBT/vPvB assessment, classification and labelling, exposure assessment and risk characterisation). Where a dossier is non-compliant with an information requirement, ECHA will request the information in a single decision. The decision is taken in cooperation with the Member States. In a targeted compliance check, ECHA evaluates only a specific part of the registration dossier based on specified concerns. Selected (groups of) endpoints or criteria, called areas of concern, have been identified that are in particular relevant for the safe use of substances. The ultimate goal is to focus on those endpoints that matter for human health and the environment. Emphasis will be given to Persistent, Bioaccumulative and Toxic (PBT); Carcinogenic, Mutagenic or Toxic to reproduction (CMR); or sensitising (S) properties of a substance. IT-assisted targeting combined with expert judgement will help to achieve the necessary increased compliance of the registration dossiers. The chances of non-compliant dossiers being picked up for compliance check are now higher. Dossiers submitted individually outside an existing joint submission and dossiers with obviously incomplete essential elements will be automatically selected for compliance check. The consequence of the targeted compliance check is that if the dossier contains several non-compliances

The European Chemicals Agency (ECHA) is introducing a new systematic targeting of compliance checks based on specified concerns.

REACH Update

registrants may in the near future receive more than one draft decision per registration dossier at different moments in time. ECHA invites registrants who receive a first draft decision as a result of these targeted compliance checks, to re-assess the overall quality of their registration dossier and prepare updates where necessary. In this context ECHA reminds the registrants that for the most common non-compliance issues that have been identified in the past, a list of recommendations for improving dossier quality can be found in the Annual evaluation report. Furthermore, ECHA starts a series of webinars in which registrants will be advised on how to bring their registration dossiers in compliance with REACH. Further information is available on the ECHA website.

ECHA, 17 September 2012

http://echa.europa.eu

Janet's Corner

Top 20 Amazing Science Facts

2012-09-21

1. There are 62,000 miles of blood vessels in the human body – laid end to end they would circle the earth 2.5 times

2. At over 2000 kilometres long, The Great Barrier Reef is the largest living structure on Earth

3. The risk of being struck by a falling meteorite for a human is one occurrence every 9,300 years

4. A thimbleful of a neutron star would weigh over 100 million tons

5. A typical hurricane produces the energy equivalent of 8,000 one megaton bombs

6. Blood sucking hookworms inhabit 700 million people worldwide

7. The highest speed ever achieved on a bicycle is 166.94 mph, by Fred Rompelberg

8. We can produce laser light a million times brighter than sunshine

9. 65% of those with autism are left handed

10. The combined length of the roots of a Finnish pine tree is over 30 miles

11. The oceans contain enough salt to cover all the continents to a depth of nearly 500 feet

12. The interstellar gas cloud Sagittarius B contains a billion, billion, billion litres of alcohol

13. Polar Bears can run at 25 miles an hour and jump over 6 feet in the air

14. 60-65 million years ago dolphins and humans shared a common ancestor

15. Polar Bears are nearly undetectable by infrared cameras, due to their transparent fur

16. The average person accidentally eats 430 bugs each year of their life

17. A single rye plant can spread up to 400 miles of roots underground

18. The temperature on the surface of Mercury exceeds 430 degrees C during the day, and, at night, plummets to minus 180 degrees centigrade

Janet's Corner

19. The evaporation from a large oak or beech tree is from ten to twenty-five gallons in twenty-four hours

20. Butterflies taste with their hind feet, and their taste sensation works on touch – this allows them to determine whether a leaf is edible

List Verse

http://listverse.com

Hazard Alert

Aniline

2012-09-04

Aniline, phenylamine or aminobenzene is an organic compound with the formula $C_6H_5NH_2$. Consisting of a phenyl group attached to an amino group, aniline is the prototypical aromatic amine. Like most volatile amines, it possesses the somewhat unpleasant odour of rotten fish. It ignites readily, burning with a smoky flame characteristic of aromatic compounds. Aniline is colourless to slightly yellow, but it slowly oxidises and resinifies in air, giving a red-brown tint to aged samples. [1] Aniline does not readily evaporate at room temperature and is slightly soluble in water and mixes readily with most organic solvents. [2]

USES [3,4]

Aniline is predominantly used as a chemical intermediate. It is used in rubber accelerators and anti-oxidants, dyes and intermediates, photographic chemicals, as isocyanates for urethane foams, in pharmaceuticals, explosives, petroleum refining; and in production of diphenylamine, phenolics, herbicides and fungicides. Aniline is also used in the manufacture of polyurethanes, rubber processing chemicals, pesticides, fibres, dyes and pigments, photographic chemicals, and pharmaceuticals.

Aniline, phenylamine or aminobenzene is an organic compound with the formula C6H5NH2.

SOURCES & ROUTES OF EXPOSURE [3,4]

Sources of Exposure

Aniline can be formed from the breakdown of certain pollutants found in outdoor air, from the burning of plastics, or from burning tobacco. Airborne exposure to aniline may occur from breathing contaminated air, from smoking tobacco or proximity to someone who is smoking, or from being near industrial sources that use large quantities of aniline. Occupational exposure to aniline could occur in industries that use aniline to make other chemicals. In addition, small amounts of aniline may be found in some foods, such as corn, grains, rhubarb, apples, beans, and rapeseed cake (animal feed). Aniline has also been found as a volatile component of black tea. Aniline has been detected in drinking water and has also been found in surface water.

Routes of Exposure

The major routes of exposure for aniline include absorption into the body via inhalation of the vapour, through the skin and by ingestion.

HEALTH EFFECTS [3]

Acute Effects

Acute inhalation exposure to high levels of aniline in humans has resulted in effects on the lung, such as upper respiratory tract irritation and congestion. Aniline has been classified as very toxic in humans, with a probable oral lethal dose in humans at 50 to 500 milligrams per kilogram body weight (mg/kg). It is considered to have high acute toxicity, based on short-term animal tests in rats.

Chronic Effects

The major effect from chronic inhalation exposure to aniline in humans is the formation of methemoglobin, which can cause cyanosis (interference with the oxygen-carrying capacity of the blood). Aniline is severely irritating to mucous membranes and affects the eyes, skin, and upper respiratory tract in humans. Significant amounts of aniline can be absorbed through the skin. Animal studies have reported a dose-related decrease in red blood cell count, haemoglobin levels, and hematocrit. The Reference Concentration (RfC) for aniline is 0.001 milligrams per cubic meter (mg/m3) based on spleen toxicity in rats.

Reproductive/Developmental Effects

No information is available on the reproductive or developmental effects of aniline in humans. Birth defects were observed in animals given aniline by gavage (placing the chemical experimentally in the stomachs of the animals). The total number of offspring in mice given aniline by gavage was lower than in the control group even though the average number of offspring per litter was not affected. However, some of the pregnant mice treated with aniline died during pregnancy. Survival of offspring in the aniline-treated group was decreased.

Carcinogenicity

A study of British workers in the chemical dye industry exposed to aniline and other chemicals concluded that there was insufficient evidence to suggest that aniline itself is a cause of bladder tumours. Animal studies have demonstrated an increase in tumours of the spleen in rats exposed to aniline hydrochloride. EPA considers aniline to be a probable human carcinogen (cancer-causing agent) and has ranked it in EPA's Group B2.

SAFETY [5]

First Aid Measures

- Eye Contact: Check for and remove any contact lenses. Immediately flush eyes with running water for at least 15 minutes, keeping eyelids open. Cold water may be used. Get medical attention. Finish by rinsing thoroughly with running water to avoid a possible infection.
- Skin Contact: In case of contact, immediately flush skin with plenty of water. Cover the irritated skin with an emollient. Remove contaminated clothing and shoes. Cold water may be used. Wash clothing before reuse. Thoroughly clean shoes before reuse. Get medical attention.
- Serious Skin Contact: Wash with a disinfectant soap and cover the contaminated skin with an anti-bacterial cream. Seek immediate medical attention.
- Inhalation: If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention immediately.
- Serious Inhalation: Evacuate the victim to a safe area as soon as possible. Loosen tight clothing such as a collar, tie, belt or waistband. If breathing is difficult, administer oxygen. If the victim is not breathing, perform mouth-to-mouth resuscitation. WARNING: It may be hazardous to the person providing aid to give mouth-to-mouth resuscitation when the inhaled material is toxic, infectious or corrosive. Seek immediate medical attention.
- Ingestion: Do NOT induce vomiting unless directed to do so by medical personnel. Never give anything by mouth to an unconscious person. If large quantities of this material are swallowed, call a physician immediately. Loosen tight clothing such as a collar, tie, belt or waistband.

Handling & Storage

Precautions

- Keep locked up
- Keep away from heat.
- Keep away from sources of ignition.
- Ground all equipment containing material.
- Do not ingest.
- Do not breathe gas/fumes/ vapour/spray.

- Wear suitable protective clothing. In case of insufficient ventilation, wear suitable respiratory equipment.
- If ingested, seek medical advice immediately and show the container or the label.
- Avoid contact with skin and eyes.
- Keep away from incompatibles such as oxidising agents, metals, acids, alkalis.

Storage

- Aniline is air and light sensitive. Store in light-resistance container.
- Keep container in a cool, well-ventilated area.
- Keep container tightly closed and sealed until ready for use.
- Avoid all possible sources of ignition (spark or flame).

Exposure Controls/Personal Protection

Engineering Controls

- Provide exhaust ventilation or other engineering controls to keep the airborne concentrations of vapours below their respective threshold limit value.
- Ensure that eyewash stations and safety showers are proximal to the workstation location.

Personal Protection

The following personal protective equipment is recommended when handling aniline:

- Splash goggles
- Lab coat
- Vapour respirator (be sure to use an approved/certified respirator or equivalent)
- Gloves

Personal Protective Equipment for Large Spills:

- Splash goggles
- Full suit
- Vapour respirator
- Boots
- Gloves

- A self-contained breathing apparatus should be used to avoid inhalation of the product.
- NOTE: Suggested protective clothing might not be sufficient; consult a specialist BEFORE handling this product.

REGULATION [4,5]

Exposure Limits

United States

ACGIH:

- TWA (TVL): 7.6 (mg/m3)
- SKIN TWA: 2 (ppm)

OSHA:

- TWA (PEL): 5 (ppm)
- TWA (PEL): 19 (mg/m3)

Australia:

Worksafe Australia:

- TWA: 2 ppm 7.6 mg/m<3
- STEL: not given in Worksafe Australia.

Canada

- SKIN TWA: 2 ppm 7.6 (mg/m3)

United Kingdom

- TWA: 1 (ppm)
- TWA: 4 (mg/m3)

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Gossip

While pregnant, traffic pollution near home raises risk for preeclampsia

2012-09-06

A new study by Australian researchers has found that exposure to traffic-related air pollution can greatly raise a pregnant woman's risk for preeclampsia. Preeclampsia can cause serious health problems for mother and baby. The strongest effects were observed among women exposed during the third trimester and for those who had gestational diabetes. One of a handful of studies to examine the link between air pollution and preeclampsia, it is unique because the scientists measured the women's exposures to local air pollutants generated by road traffic, including cars and diesel trucks. Preeclampsia occurs when a pregnant woman has significantly elevated blood pressure along with protein in the urine. Symptoms can include swelling, rapid weight gain and headaches. Scientists don't fully understand its origins but suspect it is related to how the placenta's blood vessels grow, develop and attach to the uterus during pregnancy. The condition usually begins after the 20th week of pregnancy. Most cases are diagnosed late in the third trimester after the 34th week. Some cases occur as late as one month after birth. This pregnancy complication can lead to serious health problems – and even death – for the mother and developing baby. Preeclampsia can reduce foetal growth and increase the risk of early birth. The condition is a leading cause of maternal death and medically related pre-term birth. Delivery is the only way to alleviate preeclampsia. However, regular prenatal medical care can identify the condition in its early stages so it can be managed to help protect the mother and baby. Preeclampsia affects 3 to 6 percent of pregnancies. Women who are older, obese or already have hypertension, diabetes or kidney disease are at increased risk of developing preeclampsia. Pregnancies conceived during the summer often have higher rates of preeclampsia than pregnancies conceived during other seasons. Air pollution may play a role in how and why preeclampsia develops. Like preeclampsia, air pollution varies by season. In addition, exposure to air pollution is strongly linked to cardiovascular disease, such as heart attack and high blood pressure. Preeclampsia shares some characteristics with cardiovascular disease, too. Some research suggests that exposure to air pollution is a risk factor for birth problems, including small, underweight babies and pre-term birth. Cars and other vehicles are a primary contributor to urban air pollution, yet the relationship between traffic pollution and preeclampsia is not well studied.

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During the new study, the researchers investigated the association between preeclampsia in pregnant women and exposure to traffic-related air pollution, as measured by the estimated levels of nitrogen dioxide near the women's homes. They examined medical records of 23,452 pregnancies in Perth, Western Australia, between January 2000 and December 2006. The mothers' residential addresses and preeclampsia diagnoses were available from government databases. The study included women who were at least 30 weeks pregnant with a singleton pregnancy. The researchers collected air pollution, weather and traffic volume data from routine local monitoring stations throughout the region. They added the data to a statistical modelling program to estimate average nitrogen dioxide levels at each woman's home for every week of pregnancy. Nitrogen dioxide is one of the main components of traffic-related air pollution. Its levels indicate the severity of the air pollution. The researchers examined average nitrogen dioxide levels during the entire course of pregnancy as well as the average levels during each trimester. They adjusted for several factors, including diabetes, previous pregnancies, socioeconomic status, age, smoking and ethnicity. The results showed that higher levels of traffic-related air pollution near the women's homes over the entire pregnancy resulted in a 12 percent greater risk of the women developing preeclampsia. They looked at air pollution levels by trimester. During the third trimester, exposure to higher air pollution levels was associated with a 30 percent greater risk of developing preeclampsia. The largest association was observed for women who already had or developed diabetes during pregnancy, although diabetes itself is a risk factor for preeclampsia. In this subset of 1,049 women with existing or gestational diabetes during the third trimester exposure to higher levels of air pollution was associated with a 326 percent increase in the risk of preeclampsia. Over the entire pregnancy, there was a 53 percent increase in risk. Of these diabetic women, 67 developed preeclampsia. These findings suggested that pregnant women with the highest exposures to traffic related air pollution during their whole pregnancy had an increased risk for preeclampsia. This is the first published study that used field measurements of traffic related air pollution to examine the association between air pollution and preeclampsia. While scientists don't fully understand how preeclampsia develops, the basis for the condition could begin early in pregnancy. One of the most interesting aspects of this study is that the link between air pollution and preeclampsia appears to be greatest during the third trimester – although the third trimester is also when most preeclampsia cases develop and are diagnosed. One explanation for this increased importance of third trimester exposure may be that among pregnant women who are predisposed to develop

preeclampsia – such as women with diabetes – the exposure to the elevated levels of traffic-related air pollution may trigger overt disease. Air pollution can be a challenging environmental exposure to study. While data on ambient air pollution are readily available because many governmental agencies monitor air quality, it is difficult to know exactly how much of the pollution any one person breathes. For instance, this study uses an estimate of the level of nitrogen dioxide due to traffic present at the woman's address as listed on her medical record. If the woman moved during her pregnancy, if she spends the majority of her time elsewhere or if she is rarely outside, then the exposure level assigned to her based on the stated address may be incorrect. Even with this limitation, however, this study suggests that air pollution may contribute to another adverse health outcome.

Environmental Health News, 4 September 2012

http://www.environmentalhealthnews.org/

Possible Association Between Cardiovascular Disease, Chemical Exposure, Study Suggests

2012-09-06

In a new study of 1,216 individuals, published recently online in the journal Archives of Internal Medicine, exposure to perfluorooctanoic acid (PFOA), an artificial chemical used in the manufacture of some common household products, appears to be associated with cardiovascular disease and peripheral arterial disease. Surveys have suggested that PFOA (widely used in the manufacture of products such as lubricants, polishes, paper and textile coatings, and food packaging) is detectable in the blood of more than 98 percent of the U.S population. Evidence from previous research has suggested that an association may be biologically plausible between PFOA exposure and cardiovascular disease (CVD), according to the study background. "Cardiovascular disease (CVD) is a major public health problem. Identifying novel risk factors for CVD, including widely prevalent environmental exposures, is therefore important," according to the study background. Anoop Shankar, M.D., Ph.D., and colleagues from the West Virginia University School of Public Health, Morgantown, examined the association between serum (blood) levels of PFOA and the presence of CVD and PAD, a marker of atherosclerosis, in a nationally representative group of adults. The study used merged data from the 1999-2000 and 2003-2004 National Health and Nutrition Examination Survey (NHANES). The study suggests that increasing serum PFOA levels were positively associated with the presence of CVD and PAD, and the

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association appeared to be independent of confounders such as age, sex, race/ethnicity, smoking status, body mass index, diabetes mellitus, hypertension and serum cholesterol level, the authors comment.
"Our results contribute to the emerging data on health effects of PFCs [perfluoroalkyl chemicals], suggesting for the first time that PFOA exposure is potentially related to CVD and PAD. However, owing to the cross-sectional nature of the present study, we cannot conclude that the association is causal," the authors comment. Compared with the reference level of PFOA in quartile 1, the multivariable odds ratio among participants in quartile 4 was 2.01 for CVD and 1.78 for PAD, according to the results. "In summary, in a representative cross-sectional sample of the U.S population, we found that higher PFOA levels are positively associated with self-reported CVD and objectively measured PAD. Our findings, however, should be interpreted with caution because of the possibility of residual confounding and reverse causality. Future prospective studies are needed to confirm or refute our findings," the authors conclude. In a commentary, Debabrata Mukherjee, M.D., M.S., of Texas Tech University Health Sciences Centre, El Paso, writes: "These results contribute to the evolving data on the adverse health effects of PFOA, suggesting that PFOA exposure may be potentially related to CVD." "However, a major limitation is the cross-sectional nature of the study. Given this significant limitation, causality or the temporal nature of the association between PFOA and CVD cannot be concluded from the current analysis," Mukherjee continues. "Although it seems clear that additional prospective research is needed to tease out the true adverse cardiovascular effects of PFOA, given the concerns raised by this and prior studies, clinicians will need to act now. From a societal point of view, it would make sense to limit or to eliminate the use of PFOA and its congeners in industry through legislation and regulation while improving water purification and treatment techniques to try and remove this potentially toxic chemical from our water supply," Mukherjee concludes.

Science Daily, 3 September 2012

http://www.sciencedaily.com

"Sunshine" vitamin D found to speed tuberculosis recovery

2012-09-06

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In a recent study, researchers have demonstrated how and why the "sunshine" vitamin D can speed recovery in tuberculosis (TB) patients, helping explain why the so-called heliotherapy of a bygone, pre-antibiotic era may have done some good.

era may have done some good. From the late 1800s - well before the development of antibiotics in 1930s - TB patients were often sent to retreats where they were encouraged to soak up the sun's rays in what was known as heliotherapy or phototherapy. The new study, led by British researchers, has discovered that high doses of vitamin D - which is made in the body when exposed to sunlight - given alongside antibiotic treatment, appears to help patients recover more quickly from the infectious lung disease. The findings suggest high doses of the vitamin dampen down the body's inflammatory response to infection, reducing damage to the lungs, said Adrian Martineau, a senior lecturer in respiratory infection and immunity at Queen Mary University of London, who led the study. "Sometimes these inflammatory responses can cause tissue damage leading to ... cavities in the lung," he said. "If we can help these cavities to heal more quickly, then patients should be infectious for a shorter period of time, and they may also suffer less lung damage." In addition, the researchers said they think vitamin D's ability to dampen inflammatory responses without interfering with the action of antibiotics suggests supplements might be useful for patients taking antibiotics for diseases like pneumonia, sepsis and other lung infections. TB, which people in wealthier parts of the world often mistakenly believe to be a thing of the past, is proving a tough disease to beat. In 2010, it infected 8.8 million people worldwide and killed 1.4 million. The infection destroys lung tissue, causing patients to cough up the bacteria which then spreads through the air and can be inhaled by others. In recent years, rates of drug-resistant TB have been spreading fast across the world, causing alarm among public health officials and prompting calls for more research into new and more effective treatments. The researchers, whose study was published by the Proceedings of the National Academy of Sciences, split 95 TB patients who were on standard antibiotic treatment into two groups. For the first eight weeks of their treatment, 44 of them were also given high dose vitamin D, while the remaining 51 got placebos. Anna Coussens from Britain's National Institute for Medical Research measured signs of inflammation in blood samples to see what effect the vitamin D had on immune responses. "We found that a large number of these inflammatory markers fell further and faster in patients receiving vitamin D," she said. The researchers also found that Mycobacterium tuberculosis, the bacteria that cause TB, cleared from the phlegm coughed up from deep in the lungs faster in patients on vitamin D, taking an average of 23 days to become undetectable under the microscope compared to 36 days in those on placebo. Martineau said it was too early to recommend all TB patients take high-dose vitamin D

alongside antibiotics, as more research with a larger group of patients was needed first.

Reuters Health, 4 September 2012

http://www.reuters.com/news/health

Asbestos takes toll years later

2012-09-06

Researchers have discovered that females who spent their teenage years in a mining town in Western Australia between roughly 1943 and 1966 have had a 20-47% greater risk of dying from any cause than the general population of Western Australia, while males have had a 50-83% increased risk. "Wittenoom kids" who spent their childhoods exposed to asbestos in the north-west of Western Australia are now developing a range of cancers or dying at a rate well above the average population, according to a new study by researchers from The University of Western Australia for the UWA-affiliated Western Australian Institute for Medical Research (WAIMR). Mining of the deadly blue asbestos at Wittenoom, 1106km north of Perth, ceased in 1966 and the town was later closed after airborne fibres in dust from mining operations were found to cause malignant mesothelioma, lung cancer, asbestosis and other serious diseases. While data has been collected previously looking at asbestos-related diseases caused by occupational asbestos exposure among men (either working in asbestos mining towns or using asbestos products), this new study is the first to look at the long-term health of children who were exposed to asbestos at Wittenoom. The study, which has been published in the American Journal of Industrial Medicine, shows that girls up to the age of 15 who lived in Wittenoom have been more likely to develop mesothelioma, ovarian and brain cancers and have had increased death rates. Boys who spent their childhood and early teenage years in Wittenoom during the years that asbestos was mined (1943-1966) now have elevated rates of mesothelioma, leukaemia, prostate, brain and colorectal cancer, diseases of the circulatory and nervous system, and excessive death rates. "The original township was only 1.6km from the mine," said leading researcher on the paper, WAIMR's Associate Professor Alison Reid. "Later in 1947, when the population grew, the township was moved 12km away from the mine site but tailings from the mine were used throughout the town. "These tailings, rich in crocidolite fibres, were used to pave roads, footpaths, parking areas, the local racecourse and school playgrounds. They were even used in people's backyards, where, of course, children often played," she said. "These "Wittenoom kids" are now reaching the age

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where chronic adult diseases are becoming more prevalent and many have died." Associate Professor Reid said the study by WAIMR researchers from UWA's School of Population Health found that a total of 2460 former Wittenoom children were documented to have been exposed to blue asbestos before the age of 15. The median age of their first exposure was at three years of age. Of the people studied, 63 per cent were either born in Wittenoom or had moved to the mining town by the time they were three years old. The vast majority (93.5 per cent) left Wittenoom by the time they were 16, so were exposed to asbestos only during their childhoods. By the end of 2007, 228 former residents had died from a range of causes. By the end of 2009, there were 215 cases of cancer in 207 individuals. This means that compared with the general population in Western Australia, Wittenoom girls have had a 20-47 per cent greater risk of dying from any cause, while boys have had a 50-83 per cent increased chance of dying from any cause. "We will continue to follow this group to provide important information on the long-term implications of exposure to asbestos during childhood," Associate Professor Alison Reid said.

Science Alert, 5 September 2012

http://www.sciencealert.com.au

Early puberty? Girls exposed to household chemical menstruate earlier, CDC study finds.

2012-09-06

In a new study, researchers have discovered that girls exposed to high levels of a common household chemical had their first period seven months earlier than girls with lower exposures. "This study adds to the growing body of scientific research that exposure to environmental chemicals may be associated with early puberty," said Danielle Buttke, a reproductive physiologist at the U.S. Centres for Disease Control and Prevention who was the study's lead author. Age of menarche – when a girl has her first period – has fallen over the past century, from an average of 16-17 years to 12-13 years. Experts are not sure why, but they suspect that better nutrition and rising obesity rates play a major role. There is also some evidence that points to chemicals in consumer products that can mimic oestrogen, a hormone that is critical to the timing of puberty. The CDC study is the first to link the chemical dichlorobenzene and the age of girls' first period. Dichlorobenzene, a solvent, is used in some mothballs and solid blocks of toilet bowl deodorizers and air fresheners. Nearly all people tested by the CDC in the 1990s had its residue in their bodies, and breathing indoor air is the primary exposure. Buttke said the role that

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environmental factors may play in the timing of puberty is not easy to tease out but it's a serious public health issue that warrants further study. Early menarche raises the risk of developing breast cancer and other diseases in adulthood. Whether there are health effects from exposure to dichlorobenzene is largely unknown. It is classified as a possible human carcinogen and previous studies have linked prenatal exposure to low birth weight in boys. Because it appears to alter hormones in lab animals, the U.S. Environmental Protection Agency has named it a priority for screening for hormonal effects. During the new study, the researchers examined 440 girls aged 12 to 16 who participated in the CDC's National Health and Nutrition Examination Survey. For those with the highest levels of dichlorobenzene metabolites in their urine, the average age of first menstruation was 11.8 years, while for girls with the lowest levels it was 12.4 years, or more than seven months later, according to the study published online in Environmental Health Perspectives. The CDC scientists found no significant association between age of first period and levels of other potential endocrine-disrupting chemicals found in consumer products, including bisphenol A (BPA), phthalates and parabens. Blacks and Hispanics had higher levels of the dichlorobenzene residue than white girls. But the scientists adjusted their data on menstruation to include racial factors, which can play a role in the timing of puberty. Previous studies found that heavier girls as well as blacks and Hispanics tend to have their first period at an earlier age.

Buttke and other scientists said it's impossible to say from this study alone whether exposure to the chemical actually causes girls to menstruate earlier. The CDC study is the first to link the chemical dichlorobenzene and the age of girls' first period. One major weakness is that the researchers measured the chemical in the girls' urine when they were 12 to 16 years old, after most had already started menstruating. As a result, they do not know what the girls were exposed to before their periods started, which most likely would be the critical time. "Future studies should look at exposure in the years leading up to menarche as well as exposures in utero," Buttke said. Dichlorobenzene is short-lived inside the body. It breaks down quickly and is removed from the body through urine within hours of exposure. "While it's entirely possible that chemicals with endocrine-disrupting properties can influence timing of puberty, it's unclear whether chemical exposures during certain periods of child development can have a bigger impact than at other times," said Mary Wolff, director of the Centre for Children's Environmental Health and Disease Prevention Research at Mount Sinai Hospital in New York. She was not involved in the current study. In addition, it is possible that women who experience

early puberty have some difference in the way their body metabolises the dichlorobenzene, leading to higher levels in urine. "Perhaps the chemical has nothing to do with when they started menstruating and is simply a marker of some physiological difference," said Dr. Richard Stahlhut, an environmental health researcher and preventive medicine specialist at the University of Rochester Medical Centre. Nationwide tests found dichlorobenzene residue in 98 percent of people tested in the early 1990s. It can pass to a foetus in the womb, and it also has been widely found in breast milk. Because of its cancer risk and its widespread discovery in sewage, California in 2006 banned dichlorobenzene, also known as PDCB, in toilet and urinal deodorisers and solid room fresheners. That regulation aimed to reduce indoor air levels in California by 60 percent, according to a report by the California Air Resources Board. "It is clear from the data presented that PDCB in toilet/urinal blocks, and solid air fresheners, along with mothballs are a major source of human exposure," the California report said. Representatives of the Chlorobenzene Producers Association, which represents manufacturers of dichlorobenzene, were not available for comment on the new CDC study. Earlier research has linked premature puberty to other contaminants. Adolescent girls with high levels of brominated flame-retardants had their first periods earlier than other girls in a 2011 study. Furthermore, girls prenatally exposed to other, now-banned flame-retardants called PBBs began to menstruate at a younger age, according to one study of Michigan women who in 1973, while pregnant, ate food contaminated with the chemicals. In-the-womb exposure to the banned insecticide DDT was associated with early menarche in a study of mothers and daughters in the Great Lakes region in the 1970s and 1980s. While the new study found no link between BPA or phthalates and early menarche, some previous studies suggest that these chemicals, used in many consumer products, may play a role in lowering the age of puberty in humans and animals.

Environmental Health News, 31 August 2012

http://www.environmentalhealthnews.org/

Non-infectious diseases hit the globe

2012-09-06

Non-communicable diseases (NCDs) such as heart disease, cancer and diabetes are no longer just a problem in wealthy nations – the rate of NCDs in low-to-middle income countries are increasing faster than in developed countries. This major public health issue was the focus of the Director's Seminar presented by Professor Rob Moodie from the University

Non-communicable diseases (NCDs) such as heart disease, cancer and diabetes are no longer just a problem in wealthy nations – the rate of NCDs in low-to-middle income countries are increasing faster than in developed countries.

of Melbourne's School of Population Health. "Globally 14.2 million people between the ages of 30 and 69 die each year prematurely from diseases which are preventable. Risk factors for these diseases include tobacco use, unhealthy diets and physical inactivity," Professor Moodie told a packed audience at Burnet. "There is a common view that only people in wealthy nations die from NCDs but it is a new epidemic in low-to-middle income countries that needs to be addressed." Professor Moodie said it was particularly concerning to be told recently by a surgeon at a hospital in Fiji that he was amputating one leg a day from patients suffering sepsis related to diabetes. "Seven trillion dollars of lost output in developed countries is attributable to NCDs," he said. "We need to start looking at these new epidemics as they are major global problems that should have our attention." Professor Moodie founded Burnet's Centre for International Health in 1995. Current head of the Centre, Professor Mike Toole said it was Professor Moodie's unique vision that laid the foundation for the continued success and impact of the Centre's work.

Science Alert, 31 August 2012

http://www.sciencealert.com.au

Waste water harnessed to make electricity and plastics

2012-09-06

Treating wastewater is energy intensive. In the US, it sucks up the equivalent output of four of the country's biggest power plants every year. But it needn't be such a drain on resources - soon it might be able to earn its keep. In a new study, a team of researchers from Oregon State University, led by Hong Liu, have plans for microbial fuel cells that will reclaim energy from wastewater and produce around 2.87 watts per litre of wastewater. That is almost double the amount of electrical power usual for such a cell. Furthermore, its by-products could be harnessed to create cheap, biodegradable plastics. Wastewater holds huge amounts of energy, bound up in organic molecules, but it can be difficult to access. The Oregon fuel cells run on microbes that would normally digest organic matter to produce water. In a fuel cell, though, isolated from oxygen, that conversion stalls and electrons, which are bundled with protons and oxygen to form water, are pulled away from the microbes by the potential between a cathode and an anode, creating an electrical current. As well as tweaking the mixture of microbes on the electrodes, the Oregon design has also managed to squash far more electrodes into the fuel cell than on previous versions. Liu says her lab aims to scale up the device within the next five years and make it cheaper (Energy & Environmental

In a new study, a team of researchers from Oregon State University, led by Hong Liu, have plans for microbial fuel cells that will reclaim energy from waste water and produce around 2.87 watts per litre of waste water.

Gossip

Science, doi.org/h66). The by-products of wastewater treatment can be harnessed too. Engineers are working on a way to convert methane into biodegradable plastics. The dream plastic would be biodegradable, made from organic materials, and break down easily. At the moment, polyhydroxyalkanoate (PHA) seems like the best bet. But PHA plastics are manufactured by genetically modified bacteria fed on sugars in a process that is both expensive and complex, making it hard for them to compete with conventional plastics. In the past, researchers have used the by-products of wastewater treatment to generate fuel and sometimes even to create plastics, but nearly all these attempts have focused on the "sludge" of sediment, solid waste and chemicals. Because the sludge is made of many diverse components, it produces a less stable plastic. So Molly Morse of Mango Materials in California and colleagues are now using methane, another major by-product of treating wastewater. Methanotrophs, simple organisms that feed on methane, are much better at converting it into polymers than typical bacteria are at converting sugar into plastics. Methane is pumped into a vat of methanotrophs - harvested from the wastewater treatment plant itself - along with a bubbling stream of oxygen and a few other nutrients. The end result is a polymer powder that can be separated from the mass of bacteria and turned into pellets for shaping into commercial plastic products. Morse envisions that their wastewater plastic could be used for all kinds of temporary or disposable applications, ranging from packaging materials to beauty products. Craig Criddle at Stanford University in California, who is on the firm's advisory board, says when methane itself is sold as fuel it first needs to be cleaned up. Then it will bank about 60 to 80 cents for 3 to 4 kilograms, whereas the same amount of methane could yield a kilogram of plastic, bringing in 4 to 5 dollars. "There's huge value added in going from biogas to plastic," he says. Clean water itself is one of the products that can be gleaned from waste. In Singapore, clean water reclaimed from human sewage is sold as NEWater. The country, which has tended to rely on Malaysia for clean water, plans to produce half of the water it consumes through waste reclamation by 2060. After conventional treatment, the wastewater passes through a membrane with a very fine mesh to remove large particles before reverse osmosis draws out bacteria and other contaminants. It is then zapped with ultraviolet radiation to kill off any remaining bugs. It is mainly used for industrial applications in cooling but is also blended with reservoir water to be used as drinking water.

New Scientist, 3 September 2012

http://www.newscientist.com/

Gossip

Sleep Apnoea Linked With Increased Risk of Cancer Death

2012-09-06

In a new study, researchers have found an association between sleep apnoea severity and increased cancer mortality. The research, which was presented 4 September 2012 at the European Respiratory Society's (ERS) Annual Congress in Vienna, adds to evidence presented earlier this year highlighting a link between severe sleep apnoea and cancer. Two further studies presented at the ERS Congress, also show evidence suggesting an increase in cancer incidence among sleep apnoea patients and an association between the spread of cancer and sleep apnoea. In the first study, over 5,600 patients from 7 different sleep clinics in Spain were analysed to investigate the link between sleep apnoea and cancer mortality. The severity of sleep apnoea, was then measured, using an hypoxaemia index. This index measures the amount of time during the night that a person suffers from low levels of oxygen in the blood (less than 90% oxygen saturation). The results showed that people with sleep apnoea who spent more than 14% of their sleep with levels of oxygen saturation below 90% (usually severe sleep apnoea patients) had approximately double the relative risk of death due to cancer (odds ratio 1.94), than people without sleep apnoea. The results showed that this association was even higher in men and younger people. People with sleep apnoea can be treated using continuous positive airway pressure (CPAP) therapy, which generates a stream of air to keep the upper airways open during sleep. In the first study, patients who were not using this device consistently had an increased relative risk (odds ratio 2.56) of death from cancer. Lead author, Dr Miguel Angel Martinez-Garcia from La Fe University Hospital in Valencia, Spain, said: "We found a significant increase in the relative risk of dying from cancer in people with sleep apnoea. This adds to evidence presented earlier this year that found for the first time a link between cancer and sleep apnoea mortality. Our research has only found an association between these disorders but this does not mean that sleep apnoea causes cancer. Similar results were also found in the second study, which showed an increase in all-type cancer incidence in people with severe sleep apnoea. The link was present even when factors such as age, sex, weight and other comorbidities of participants, were controlled for. Lead author, Dr Francisco Campos-Rodriguez from Valme University Hospital in Seville, Spain, said: "Further studies are necessary to corroborate our results and analyse the role of CPAP treatment on this association. We hope the findings of our studies will encourage people to get their sleep apnoea diagnosed and treated early to help maintain a

In a new study, researchers have found an association between sleep apnoea severity and increased cancer mortality.

good quality of life." In a third study, researchers used a mouse model of skin cancer (melanoma) to investigate tumour spread (metastasis) and whether this was associated with sleep apnoea. The results showed that the spread of cancer was more abundant in mice that had been subjected to intermittent hypoxic air, with low levels of oxygen as in sleep apnoea, than those who breathed normal air during the experiment. Lead author, Professor Ramon Farre from University of Barcelona in Spain, said: "The data from this study in animals strongly suggests a link between the spread of cancer and sleep apnoea. This provides strong evidence to encourage further study in this area to understand in more detail the links between sleep apnoea and cancer."

Science Daily, 4 September 2012

http://www.sciencedaily.com

Food for thought: Eat your way to dementia

2012-09-06

In a new study, researchers have discovered that by changing the way the rats' brains respond to insulin, can result in dementia.

Suzanne De La Monte's rats were disoriented and confused. Navigating their way around a circular water maze - a common memory test for rodents - they quickly forgot where they were, and couldn't figure out how to locate the hidden, submerged safety platform. Instead, they splashed around aimlessly. "They were demented. They couldn't learn or remember," says de la Monte, a neuropathologist at Brown University in Providence, Rhode Island. A closer look at her rats' brains uncovered devastating damage. Areas associated with memory were studded with bright pink plaques, like rocks in a climbing wall, while many neurons, full to bursting point with a toxic protein, were collapsing and crumbling. As they disintegrated, they lost their shape and their connections with other neurons, teetering on the brink of death. Such changes are the hallmarks of Alzheimer's disease, and yet they arose in surprising circumstances. De la Monte had interfered with the way the rats' brains respond to insulin. The hormone is most famous for controlling blood sugar levels, but it also plays a key role in brain signalling. When de la Monte disrupted its path to the rats' neurons, the result was dementia. Poor sensitivity to insulin is typically associated with type 2 diabetes, in which liver, fat and muscle cells fail to respond to the hormone. But results such as de la Monte's have led some researchers to wonder whether Alzheimer's may sometimes be another version of diabetes - one that hits the brain. Some have even renamed it "type 3 diabetes". If they are right - and a growing body of evidence suggests they might be - the implications are deeply troubling. Since calorific foods are known to impair our body's response

to insulin, we may be unwittingly poisoning our brains every time we chow down on burgers and fries. People with type 2 diabetes, who have already developed insulin resistance, may be particularly at risk. "The epidemic of type 2 diabetes, if it continues on its current trajectory, is likely to be followed by an epidemic of dementia," says Ewan McNay of the University at Albany in New York. "That's going to be a huge challenge to the medical and care systems." All of which highlights the importance of eating healthier foods and taking exercise to reduce your risk of dementia. It may even be possible to reverse - or at least decelerate - some of the cognitive decline in people who already have Alzheimer's, by targeting the underlying insulin resistance. If so, that would suggest new treatments for the disease, which has so far evaded any attempt to treat it. A new understanding of Alzheimer's can't come soon enough; it plagues an estimated 5.4 million adults in the US, whose care cost $130 billion in 2011 alone. Worldwide, 36 million people have the disease, a figure that will rise as the population continues to grow. "We are desperate for an effective therapy," says John Morris, a neurologist specialising in Alzheimer's disease at the Washington University School of Medicine in St Louis. For a long time, the finger of blame has pointed squarely at the beta amyloid plaques that amass in the brains of people with the disease. Alois Alzheimer, the German psychiatrist and neuropathologist for whom the disease is named, first described these strange protein deposits over a century ago, when he noticed apparently normal brain cells filled with strange fibrils. In the areas where the disease had progressed, the fibrils had merged and moved to the surface inside the cell, where they folded together in thick bundles. "Eventually, the nucleus and the cell disintegrate, and only a tangled bundle of fibrils indicates the place which had formerly been occupied by a ganglion cell," he wrote. The origin of these plaques is only partially understood; we know that beta amyloid is a fragment of a larger protein that helps form cell membranes in the brain and other parts of the body. It is also thought to carry out important functions of its own, such as fighting microbes, transporting cholesterol and regulating the activity of certain genes. What prompts the protein to clump into the deadly plaques is something of a mystery, but if the new research is right, a diabetes-like illness might be a trigger.

This new focus follows a growing recognition of insulin's role in the brain. Until recently, the hormone was typecast as a regulator of blood sugar, giving the cue for muscles, liver and fat cells to extract sugar from the blood and either use it for energy or store it as fat. We now know that it is a master multitasker: it helps neurons, particularly in the hippocampus and frontal lobe, take up glucose for energy, and it also regulates

neurotransmitters, like acetylcholine, which are crucial for memory and learning. What's more, it encourages plasticity - the process through which neurons change shape, make new connections and strengthen others. And it is important for the function and growth of blood vessels, which supply the brain with oxygen and glucose. As a result, reducing the level of insulin in the brain can immediately impair cognition. Spatial memory, in particular, seems to suffer when you block insulin uptake in the hippocampus; the effect is almost the same as that of morphine, says McNay. Conversely, a boost of insulin seems to improve it's functioning. McNay points out that this role in the brain "makes evolutionary sense", since it would help us to remember the location of a food source. As our ancestors gorged on berries in the savannah, for instance, the spike in glucose and the subsequent rush of insulin would signal "remember this, it's important", causing the brain to crystallise the memory. But as we know from type 2 diabetes, processes that evolved to help us meet the challenges of prehistory can easily backfire in the modern world. When people frequently gorge on fatty, sugary food their insulin spikes repeatedly until it sticks at a higher level. Muscle, liver and fat cells then stop responding to the hormone, meaning they don't mop up glucose and fat in the blood. As a result, the pancreas desperately works overtime to make more insulin to control the glucose - and levels of the two molecules skyrocket. "It's like you are knocking on the door and the person inside is ignoring your call. So you knock louder and louder," says de la Monte. The pancreas can't keep up with the demand indefinitely, however, and as time passes people with type 2 diabetes often end up with abnormally low levels of insulin. Weight gain seems to amplify the problem - 80 per cent of people with type 2 diabetes are also overweight or obese. Though the mechanism is still unclear, obesity seems to trigger the release of inflammatory and metabolic stress molecules inside liver and fat cells that disrupt insulin action, leading to high blood glucose levels and, eventually, insulin resistance. If McNay and de la Monte are correct, a similar process may lead to Alzheimer's. They think that constantly high levels of insulin, triggered by the fat and sugar content of the western diet, might begin to overwhelm the brain, which can't constantly be on high alert. Either alongside the other changes associated with type 2 diabetes, or separately, the brain may then begin to turn down its insulin signalling, impairing your ability to think and form memories before leading to permanent neural damage. "I believe it starts with insulin resistance," says de la Monte. "If you can avoid brain diabetes you'll be fine. But once it gets going you are going to need to attack on multiple fronts." Her study on the demented rats was one of the first experiments to make this link. At the time she was interested in the impact of alcohol on the brain, which is known to

decrease its number of insulin receptors. To probe the consequences she used a chemical to wipe out all the brain cells carrying the insulin receptor; the result looked surprisingly similar to Alzheimer's, including the build-up of the deadly beta amyloid plaques. De la Monte's finding is now just one of many discoveries to confirm that a disrupted insulin system can lead to the symptoms of Alzheimer's. William Klein at Northwestern University in Evanston, Illinois, for instance, has found that triggering diabetes created Alzheimer's-like brain changes in rabbits, including a sharp rise in the number of beta amyloid proteins (The Journal of Alzheimer's Disease, DOI: 10.3233/JAD-2012-120571). "It's the first time that any culprit has been singled out as an instigator of sporadic Alzheimer's disease pathology, one of the big mysteries in the field," he says. McNay and Suzanne Craft at the University of Washington in Seattle, meanwhile, fed rats a high fat diet for 12 months, which destroyed their ability to regulate insulin and led to diabetes. Once again, high beta amyloid levels in the brain accompanied the change. In addition, they had trouble navigating a maze and looked "much like an Alzheimer's patient", says McNay. Of course, animal studies can only tell you so much about a human disease, but an almost Frankensteinian demonstration confirms that the brains of people with Alzheimer's are insulin-resistant. Using brains from cadavers, Steven Arnold at the University of Pennsylvania bathed various tissue samples in insulin to see how they would react. Tissue from people who had not had Alzheimer's seemed to spring back to life, triggering a cascade of chemical reactions suggestive of synaptic activity. In contrast, the neurons of those who had had Alzheimer's barely reacted at all (Journal of Clinical Investigations, vol 122, p 1316). "The insulin signalling is paralysed," says Arnold.

It's not yet fully understood exactly why disrupted insulin signalling would lead to the other kinds of brain damage associated with Alzheimer's, such as the build up of plaques, though the emerging research suggests many, possibly interlinked, mechanisms. One line of evidence, for instance, has shown the same protein-chomping enzyme breaks down both that insulin and beta amyloid. Under normal circumstances that enzyme can successfully deal with both, but if too much insulin is washing around, the hormone overwhelms the enzyme, and the beta amyloid gets neglected. Instead of being broken down, it accumulates, perhaps building into the toxic plaques that kill brain cells (Proceedings of the National Academy of Sciences, vol 100, p 4162). Exacerbating the problem, beta amyloid can then stop neurons from responding to insulin, leading to further damage. By studying dishes of rat neurons, Klein has found that toxic clusters of the protein attack and destroy regions of synapses that are covered in

insulin receptors; they also stop new receptors appearing, making the neuron insulin-resistant (FASEB Journal, vol 22, p 246). The result would be an immediate impairment in cognition. Worse still, this insulin resistance tells the cells to make even more beta amyloid, which then goes on to harm more brain cells. "It triggers a vicious cycle," says Klein. Things only get worse if the pancreas becomes exhausted by the high demand for insulin, lowering levels of the hormone in the brain. Klein has found that a moderate level of insulin is protective, offsetting beta amyloid damage by blocking its landing sites on brain cells. "But when people age or have diabetes, the insulin signalling in the brain becomes weaker, possibly opening a window for amyloid beta toxin to start destroying the neurons," he says. It is still early days for this work - and the researchers are keen to point out that they haven't solved every aspect of the puzzle. Klein, for example, thinks that lack of insulin in the brain may be just one of many triggers for beta amyloid toxins, so he's searching for other culprits. Suzanne Craft, who has been a pioneer in insulin and Alzheimer's research, agrees that it is probably one of many paths to the disease. After all, most people with Alzheimer's don't have full-blown type 2 diabetes - though many do have some problems with the insulin signalling in their bodies, even if they don't match every criterion for the disease. Even so, the research should ring warning bells for the future. Thanks to our addiction to fast food, type 2 diabetes is constantly on the rise. In the US alone, 19 million people have now been diagnosed with the condition, while a further 79 million are considered "prediabetic", showing some of the early signs of insulin resistance. If Alzheimer's and type 2 diabetes do share a similar mechanism, levels of dementia may follow a similar trajectory as these people age. Even if someone doesn't develop diabetes, a bad diet might be enough to set the wheels in motion for brain degeneration, according to an ongoing study led by Craft. For one month a group of volunteers - none of whom had diabetes - ate foods that were high in saturated fat and sugar while a control group ate a diet low in sugar and saturated fat. In just four weeks, those gorging on the high-sugar diet had higher levels of insulin and significantly higher beta amyloid levels in their spinal fluid. The control group showed decreases in both. "An unhealthy diet disrupts normal insulin function in the brain, increases inflammation and oxidative stress, and impairs amyloid regulation," says Craft. When these three converge they can lead to Alzheimer's, she says. When you consider that obesity is a big risk factor for both diabetes and dementia, all the signs suggest that our addiction to junk foods could spell trouble for our mental health in the future. On the plus side, a new understanding of the disease might lead to new treatments for those who already have Alzheimer's.

Gossip

Craft, for instance, is investigating whether a boost of insulin might improve symptoms. So far, she has tested out a device that delivers insulin deep into the nose, where it then travels to the brain. The study was short, lasting just four months and involving only 104 people, but the results were promising. In memory tests those who received the treatment could recall more details of stories, had longer attention spans, regained more interest in their hobbies and were better able to care for themselves. The glucose metabolism in their brains also improved (Archives of Neurology, vol 69, p 29). Given insulin's many roles in the brain, the nasal spray may work for a number of reasons. A blast of the hormone might help struggling cells to return to normal activity. Alternatively, Craft points out that it might decrease inflammation and oxidative stress caused by reactive oxygen-containing compounds - both of which are problems for people with Alzheimer's. Klein, meanwhile, thinks that Craft's approach may work because insulin helps prevent the beta amyloid toxins from docking with brain cells. "It is a struggle between insulin and the toxins for synaptic survival," he says. He suspects it might also curb the build-up of these toxins in the first place. A better understanding of this process might come from Craft's next project; she has just been awarded $7.9 million by the US National Institutes of Health in Bethesda, Maryland, to test the nasal insulin spray on 240 volunteers showing signs of dementia. Teams across the US will monitor learning, memory, daily function and any brain changes using PET scans. There are several other possible lines of attack: clinical trials are investigating the use of approved diabetes drugs such as metformin, exenatide, liraglutide and pioglitazone, which try to restore the balance of insulin and glucose in the blood or improve the insulin sensitivity of an organ. Arnold, for instance, plans to study the effect of metformin by measuring amyloid levels in the spinal fluid and testing the blood flow in the brain before and after treatment. "We want to see if these medicines work to decrease levels of these abnormal proteins in Alzheimer's disease and ultimately improve the patients' cognitive abilities, or at least prevent them from getting worse," he says. "We'll also see whether the drugs restore other insulin functions like promoting synapse formation and regrowing neural connections." Other groups plan to use advanced brain imaging to see if these diabetes medications can shrink the beta amyloid plaques, which might reverse some of the brain damage. For the time being, there are measures that everyone can take to help stave off cognitive decline. Since insulin resistance emerges from a bad diet, laying off fatty and sweet foods might help to reduce the risk of developing Alzheimer's. Conversely, diets rich in certain kinds of fatty acids might help the brain to maintain good insulin signalling. Exercise, too, can encourage the body to conquer insulin resistance - which may

explain why regular physical activity reduces your risk of Alzheimer's by 40 per cent (*Annals of Internal Medicine*, vol 144, p 73). "Even if you are 400 pounds and you haven't seen the back of the couch for six months, it's not too late. It's likely that any exercise will help, even in patients who've been diabetic for a long time. Get some of the insulin sensitivity back and stop accumulating so much amyloid," says McNay. "Potentially, even some of the amyloid that's built up might get broken down. As for the rest of us, extra trips to the gym are always a good idea, and this work shows that they help your brain as well as your body."

New Scientist, 3 September 2012

http://www.newscientist.com/

DNA data unlocks map to genetic disease

2012-09-06

A massive DNA database has generated a map of the genetic switches, which impact everything from hair loss to cancer and opened the door to revolutionary treatments for a host of deadly diseases, researchers say. "This is a major step toward understanding the wiring diagram of a human being," said lead researcher Michael Snyder of Stanford University. The Encyclopedia of DNA Elements - or ENCODE - has enabled scientists to assign specific biological functions for 80 per cent of the human genome and has helped explain how genetic variants affect a person's susceptibility to disease. It also exposed previously hidden connections between seemingly unrelated diseases such as asthma, lupus and multiple sclerosis, which were found to be linked to specific genetic regulatory codes for proteins that regulate the immune system. A key insight revealed in a host of papers published in the journals Nature, Science and Cell is that many diseases result from changes in when, where and how a gene switches on or off rather than a change to the gene itself. ``Genes occupy only a tiny fraction of the genome, and most efforts to map the genetic causes of disease were frustrated by signals that pointed away from genes," said co-author John Stamatoyannoupoulos, a researcher at the University of Washington. ``Now we know that these efforts were not in vain, and that the signals were in fact pointing to the genome's operating system." Another significant finding is that this blueprint of genetic switches can be used to pinpoint cell types that play a role in specific diseases without needing to understand how the disease actually works. For instance, it took researchers decades to link a set of immune cells with the inflammatory bowel disease Crohn's. The ENCODE data was able to swiftly identify that the genetic variants associated with Crohn's

A massive DNA database has generated a map of the genetic switches, which impact everything from hair loss to cancer and opened the door to revolutionary treatments for a host of deadly diseases, researchers say.

were concentrated in that subset of cells. This in-depth map of the human genetic code has also altered scientific understanding of how DNA works. The first sketch of the human genome described DNA as a string, which contained genes in isolated sections that make up just two per cent of its length. The space in between was dubbed ``junk DNA'' and many researchers did not believe it served a function. Attention was focused on the 'coding' genes, which carried instructions for making the proteins that carried out basic biological functions. ENCODE confirmed more recent theories that the bulk of this 'junk' is actually littered with switches that determine how the genes work and act as a massive control panel. ``Our genome is simply alive with switches: millions of places that determine whether a gene is switched on or off,'' said lead analysis coordinator Ewan Birney of the EMBL-European Bioinformatics Institute. ``We found a much bigger part of the genome - a surprising amount, in fact - is involved in controlling when and where proteins are produced, than in simply manufacturing the building blocks.'' Perhaps most importantly, the database has been made available to the scientific community - and the general public - as an open resource in order to facilitate research. The project combined the efforts of 442 scientists in 32 labs in the United States, Britain, Spain, Switzerland, Singapore and Japan. The researchers used about 300 years worth of computer time to study 147 tissue types and identified over four million different regulatory regions where proteins interact with the DNA.

The Australian, 6 September 2012

http://www.theaustralian.com.au

Technical Notes

(Note: Open your Web Browser and click on Heading to link to section)

ENVIRONMENTAL

MEDICAL

OCCUPATIONAL

Technical Notes

PUBLIC HEALTH

Dietary exposure estimates of 16 polycyclic aromatic hydrocarbons (PAHs) in Xuanwei and Fuyuan, counties in a high lung cancer incidence area in China

Exposure assessment of tetrafluoroethylene and ammonium perfluorooctanoate 1951-2002

Lead Poisoning Among Arab American and African American Children in the Detroit Metropolitan Area, Michigan

Human maternal and umbilical cord blood concentrations of polybrominated diphenyl ethers

Blood lead levels and cumulative blood lead index (CBLI) as predictors of late neurodevelopment in lead poisoned children

SAFETY

Micro-discharge energy-saving high-pressure engine master part apparatus of heat-accumulated exhaust gas heat energy recovery

Face part disinfection and drying device for anti-gas mask