

3 September 2009

This supplement has been prepared to present scientific and technical news items that may be of more interest to technical personnel at RDT&E activities and the labs, or the medics rather than the broader readership of the basic CB Daily. Due to the nature of the material, the articles, if available online, are usually only available through subscription services thus making specific links generally unavailable. Thus, usually only the bibliographic citation is available for use by an activity's technical library.

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Chem-Bio News – S&T Edition

- 1. ETS2 REGULATING NEURODEGENERATIVE SIGNALING PATHWAY OF HUMAN NEURONAL (SH-SY5Y) CELLS EXPOSED TO SINGLE AND REPEATED LOW-DOSE SARIN (GB):** *"The overall data delineate an in vitro experimental model suitable for studying the neuropathology of cells and may provide novel insights into therapeutic interventions.."*
- 2. EXPRESSION OF EBOLAVIRUS GLYCOPROTEIN ON THE TARGET CELLS ENHANCES VIRAL ENTRY:** *"These findings have important implications in our current understanding of virus entry and superinfection interference.."*
- 3. INHIBITION OF NK CELL ACTIVITY BY IL-17 ALLOWS VACCINIA VIRUS TO INDUCE SEVERE SKIN LESIONS IN A MOUSE MODEL OF ECZEMA VACCINATUM:** *"Given low NK cell activities and increased IL-17 expression in atopic dermatitis patients, these results can explain the increased susceptibility of atopic dermatitis patients to eczema vaccinatum.."*
- 4. ANTIGEN-SPECIFIC MEMORY B-CELL RESPONSES TO VIBRIO CHOLERAЕ O1 INFECTION IN BANGLADESH:** *"Such memory B cells could mediate anamnestic responses on reexposure to V. cholerae."*
- 5. BICARBONATE INDUCES VIBRIO CHOLERAЕ VIRULENCE GENE EXPRESSION BY ENHANCING TOXIN ACTIVITY ANTIGEN-SPECIFIC MEMORY B-CELL RESPONSES TO VIBRIO CHOLERAЕ O1 INFECTION IN BANGLADESH:** *"Given that bicarbonate is present at high concentration in the upper small intestine where V. cholerae colonizes, bicarbonate is likely an important chemical stimulus that V. cholerae senses and that induces virulence during the natural course of infection."*
- 6. THE INHIBITION OF THE INTERACTION BETWEEN THE ANTHRAX TOXIN AND ITS CELLULAR RECEPTOR BY AN ANTI-RECEPTOR MONOCLONAL ANTIBODY:** *"To our knowledge, this is the first report to illustrate that an anti-CMG2 antibody could inhibit the PA-CMG2 interaction and therefore interfere with the intoxication of anthrax toxin."*
- 7. FEMTOMOLAR DETECTION OF THE ANTHRAX EDEMA FACTOR IN HUMAN AND ANIMAL PLASMA:** *"This simple and robust combination of enzymatic reaction and enzyme immunoassay for the diagnosis of anthrax toxemia could prove useful in biological threat detection as well in research and clinical practice.."*
- 8. CHOLERA TOXIN INHIBITS IL-12 PRODUCTION AND CD8 ALPHA(+) DENDRITIC CELL DIFFERENTIATION BY cAMP MEDIATED INHIBITION OF IRF8 FUNCTION:** *"Therefore, because IRF8 is essential for IL-12 production and the differentiation of CD8 alpha(+) cDCs and pDCs, these data suggest that CT and other Gs-protein agonists can affect IL-12 production and DC differentiation via a common mechanism involving IRF8.."*
- 9. UCSB SCIENTISTS DISCOVER POTENTIAL DRUG DELIVERY SYSTEM:** *"The scientists developed a peptide, a small piece of protein that can carry "cargo" for delivery into the cell. The cargo could be a nanoparticle, or even a cell. Riding on the peptide, the cargo gets out of the blood vessel and penetrates the tissue."*
- 10. VIBRIO CHOLERAЕ PROTEOME-WIDE SCREEN FOR IMMUNOSTIMULATORY PROTEINS IDENTIFIES PHOSPHATIDYLSERINE DECARBOXYLASE AS A NOVEL TOLL-LIKE RECEPTOR 4 AGONIST:** *"Taken together, these results provide evidence for the identification of V. cholerae PSD as a novel TLR4 agonist and further demonstrate the potential application of PSD as a vaccine adjuvant."*

11. CHIP DETECTS MICROORGANISMS IN AMBIENT AIR: "Researchers from six Fraunhofer Institutes have now developed a test system capable of carrying out such tests on the spot and in less than half an hour."

12. MECHANISED NANOCAPSULES TARGET DRUG DELIVERY: "US researchers have developed a nano-sized drug delivery system that only releases its payload in specific pH conditions,....."

CB Daily Report

Chem-Bio News

ETS2 REGULATING NEURODEGENERATIVE SIGNALING PATHWAY OF HUMAN NEURONAL (SH-SY5Y) CELLS EXPOSED TO SINGLE AND REPEATED LOW-DOSE SARIN (GB)

Ecology, Environment & Conservation Business
August 22, 2009

"The mechanistic understanding of low-level sarin-induced neurotoxicity after single or repeated doses has yet to be explored at a cellular level. Using the microarray (Affymetrix-GeneChips) transcription profiling approach, the present study examined gene expression in human SH-SY5Y cells exposed to, single Q and 24 h) or repeated (2 x 24 h) doses of sarin (5 μ g/mL) to delineate the possible mechanism."

"Two hundred twenty-four genes whose expression was significantly ($P < 0.01$) altered by at least 3-fold were selected by GeneSpringGX analysis. The comparative gene expression data confirmed the transcriptional changes to be related to dose and exposure time of sarin. The effect of a single noncytotoxic sarin dose on gene transcription was variable, whereas repeated doses over 48 h persistently down-regulated genes linked to neurodegenerative mechanisms. Thirty persistently altered genes were validated using real-time quantitative reverse transcription-polymerase chain reaction (qRT-PCR). Similar qRT-PCR profiles obtained in sarin-treated SH-SY5Y and HCN-1A cells confirmed the cell-independent alterations in expression levels. Genes (ETS2, APOE, PSEN1, DDC, and CD9) implicated mainly in the regulation of sarin-induced neuropathogenesis were further confirmed by Western blot and double-immunofluorescence assays. The regulome pathway suggests a new feasible mechanism by which sarin increases ETS2 expression and takes control over other genes involved in the neurodegenerative pathway."

"The overall data delineate an in vitro experimental model suitable for studying the neuropathology of cells and may provide novel insights into therapeutic interventions.."

The full article can be found at: (A. Pachappan, et. al., "ETS2 Regulating Neurodegenerative Signaling Pathway of Human Neuronal (SH-SY5Y) Cells Exposed to Single and Repeated Low-Dose Sarin (GB)". Chemical Research in Toxicology, 2009;22(6):990-996). Link not available.

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EXPRESSION OF EBOLAVIRUS GLYCOPROTEIN ON THE TARGET CELLS ENHANCES VIRAL ENTRY

Drug Week
September 4, 2009

"Entry of Ebolavirus to the target cells is mediated by the viral glycoprotein GP. The native GP exists as a homotrimer on the virions and contains two subunits, a surface subunit (GP1) that is involved in receptor binding and a transmembrane subunit (GP2) that mediates the virus-host membrane fusion."

"Previously we showed that over-expression of GP on the target cells blocks GP-mediated viral entry,

which is mostly likely due to receptor interference by GP1. In this study, using a tetracycline inducible system, we report that low levels of GP expression on the target cells, instead of interfering, specifically enhance GP mediated viral entry. Detailed mapping analysis strongly suggests that the fusion subunit GP2 is primarily responsible for this novel phenomenon, here referred to as trans enhancement. Our data suggests that GP2 mediated trans enhancement of virus fusion occurs via a mechanism analogous to eukaryotic membrane fusion processes involving specific trans oligomerization and cooperative interaction of fusion mediators."

"These findings have important implications in our current understanding of virus entry and superinfection interference.."

The full article can be found at: (B. Manicassamy, et. al., "Expression of Ebolavirus glycoprotein on the target cells enhances viral entry". *Virology Journal*, 2009;6():75). Link not available.

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INHIBITION OF NK CELL ACTIVITY BY IL-17 ALLOWS VACCINIA VIRUS TO INDUCE SEVERE SKIN LESIONS IN A MOUSE MODEL OF ECZEMA VACCINATUM

Medical Letter on the CDC & FDA

August 30, 2009

"Individuals with atopic dermatitis are excluded from smallpox vaccination because of their propensity to develop eczema vaccinatum, a disseminated vaccinia virus (VACV) infection."

"To study the underlying mechanism of the vulnerability of atopic dermatitis patients to VACV infection, we developed a mouse model of eczema vaccinatum. Virus infection of eczematous skin induced severe primary erosive skin lesions, but not in the skin of healthy mice. Eczematous mice exhibited lower natural killer (NK) cell activity but similar cytotoxic T lymphocyte activity and humoral immune responses. The role of NK cells in controlling VACV-induced skin lesions was demonstrated by experiments depleting or transferring NK cells. The proinflammatory cytokine interleukin (IL)-17 reduced NK cell activity in mice with preexisting dermatitis."

"Given low NK cell activities and increased IL-17 expression in atopic dermatitis patients, these results can explain the increased susceptibility of atopic dermatitis patients to eczema vaccinatum.."

The full article can be found at: (Y. Kawakami, et. al., "Inhibition of NK cell activity by IL-17 allows vaccinia virus to induce severe skin lesions in a mouse model of eczema vaccinatum". *Journal of Experimental Medicine*, 2009;206(6):1219-1225). Link not available.

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ANTIGEN-SPECIFIC MEMORY B-CELL RESPONSES TO VIBRIO CHOLERAE O1 INFECTION IN BANGLADESH

Immunotherapy Weekly

September 2, 2009

Infection with *V. cholerae* elicits long-term protection against subsequent disease in countries where the disease is endemic. Although the mechanism of this protective immunity is unknown, it has been hypothesized that a protective mucosal response to *V. cholerae* infection may be mediated by anamnestic responses of memory B cells in the gut-associated lymphoid tissue."

"To characterize memory B-cell responses to cholera, we enrolled a cohort of 39 hospitalized patients with culture-confirmed cholera and evaluated their immunologic responses at frequent intervals over the subsequent 1 year. Memory B cells to cholera antigens, including lipopolysaccharide (LPS), and the protein antigens cholera toxin B subunit (CTB) and toxin-coregulated pilus major subunit A (TcpA) were enumerated using a method of polyclonal stimulation of peripheral blood mononuclear cells followed by a standard enzyme-linked immunospot procedure. All patients demonstrated CTB, TcpA, and LPS-

specific immunoglobulin G (IgG) and IgA memory responses by day 90. In addition, these memory B-cell responses persisted up to 1 year, substantially longer than other traditional immunologic markers of infection with *V. cholerae*. While the magnitude of the LPS-specific IgG memory B-cell response waned at 1 year, CTB- and TcpA-specific IgG memory B cells remained significantly elevated at 1 year after infection, suggesting that T-cell help may result in a more durable memory B-cell response to *V. cholerae* protein antigens."

"Such memory B cells could mediate anamnestic responses on reexposure to *V. cholerae*."

The full article can be found at: (A.M. Harris, et. al., "Antigen-specific memory B-cell responses to *Vibrio cholerae* O1 infection in Bangladesh". *Infection and Immunity*, 2009;77(9):3850-6). Link not available.

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BICARBONATE INDUCES VIBRIO CHOLERAEE VIRULENCE GENE EXPRESSION BY ENHANCING TOXT ACTIVITY

Drug Week

September 4, 2009

"Growth under the inducing conditions or infection of a host initiates a complex regulatory cascade that results in production of ToxT, a regulatory protein that directly activates transcription of the genes encoding cholera toxin (CT), toxin-coregulated pilus (TCP), and other virulence genes. Previous studies have shown that sodium bicarbonate induces CT expression in the *V. cholerae* El Tor biotype. However, the mechanism for bicarbonate-mediated CT induction has not been defined. In this study, we demonstrate that bicarbonate stimulates virulence gene expression by enhancing ToxT activity. Both the classical and El Tor biotypes produce inactive ToxT protein when they are cultured statically in the absence of bicarbonate. Addition of bicarbonate to the culture medium does not affect ToxT production but causes a significant increase in CT and TCP expression in both biotypes. Ethoxzolamide, a potent carbonic anhydrase inhibitor, inhibits bicarbonate-mediated virulence induction, suggesting that conversion of CO₂ into bicarbonate by carbonic anhydrase plays a role in virulence induction. Thus, bicarbonate is the first positive effector for ToxT activity to be identified."

"Given that bicarbonate is present at high concentration in the upper small intestine where *V. cholerae* colonizes, bicarbonate is likely an important chemical stimulus that *V. cholerae* senses and that induces virulence during the natural course of infection."

The full article can be found at: (B.H. Abuaita, et. al., "Bicarbonate Induces *Vibrio cholerae* virulence gene expression by enhancing ToxT activity". *Infection and Immunity*, 2009;77(9):4111-20). Link not available.

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THE INHIBITION OF THE INTERACTION BETWEEN THE ANTHRAX TOXIN AND ITS CELLULAR RECEPTOR BY AN ANTI-RECEPTOR MONOCLONAL ANTIBODY

Medical Letter on the CDC & FDA

September 6, 2009

"The high affinity binding of the anthrax protective antigen (PA) to one of its receptors, capillary morphogenesis protein 2 (CMG2), is essential for the intoxication process of anthrax toxin. To acquire novel research tools to study the PA-CMG2 interaction, we generated several anti-CMG2 monoclonal antibodies (MAbs)."

"We demonstrated that one of the MAbs, 4B5, could inhibit PA-CMG2 binding and could also protect the sensitive cells against an anthrax lethal toxin challenge. We identified the epitope recognized by 4B5 and confirmed that the key residues of the epitope were the residues (YI)-Y-119-LK125 of CMG2. Based on our results, we propose that 4B5 binds to the E122 pocket of CMG2 and interrupts the interaction

between the pocket and the PA 2 beta 3-2 beta 4 loop."

"To our knowledge, this is the first report to illustrate that an anti-CMG2 antibody could inhibit the PA-CMG2 interaction and therefore interfere with the intoxication of anthrax toxin."

The full article can be found at: (G.L. Li, et. al., "The inhibition of the interaction between the anthrax toxin and its cellular receptor by an anti-receptor monoclonal antibody". Biochemical and Biophysical Research Communications, 2009;385(4):591-595). Link not available.

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FEMTOMOLAR DETECTION OF THE ANTHRAX EDEMA FACTOR IN HUMAN AND ANIMAL PLASMA

Medical Letter on the CDC & FDA
August 30, 2009

"Edema factor (EF), a calmodulin-activated adenylyl cyclase, is a toxin which contributes to cutaneous and systemic anthrax. As a novel strategy to detect anthrax toxins in humans or animals infected by *Bacillus anthracis*, we have developed a sensitive enzymatic assay to be able to monitor functional EF in human and animal plasma."

"Samples containing EF are incubated in the presence of calmodulin and ATP, which is converted to cAMP. After oxidation and derivatization, cAMP is monitored by competitive enzyme immunoassay. Because of the high turnover of EF and the sensitivity of cAMP detection, EF can be detected at concentrations of 1 pg/mL 10 fM in 4 h in plasma from humans or at 10 pg/mL in the plasma of various animal species using only a blood volume of 5 μ L. The assay has good reproducibility with intra- and interday coefficients of variation in the range of 20% and is not subject to significant interindividual matrix effects. In an experimental study performed in mice infected with the Berne strain, we were able to detect EF in serum and ear tissues."

"This simple and robust combination of enzymatic reaction and enzyme immunoassay for the diagnosis of anthrax toxemia could prove useful in biological threat detection as well in research and clinical practice.."

The full article can be found at: (E. Duriez, et. al. "Femtomolar Detection of the Anthrax Edema Factor in Human and Animal Plasma". Analytical Chemistry, 2009;81(14):5935-5941). Link not available.

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CHOLERA TOXIN INHIBITS IL-12 PRODUCTION AND CD8 ALPHA(+) DENDRITIC CELL DIFFERENTIATION BY cAMP-MEDIATED INHIBITION OF IRF8 FUNCTION

Biotech Week
August 26, 2009

"Prior studies have demonstrated that cholera toxin (CT) and other cAMP-inducing factors inhibit interleukin (IL)-12 production from monocytes and dendritic cells (DCs). We show that CT inhibits Th1 responses in vivo in mice infected with *Toxoplasma gondii*."

"This correlated with low serum IL-12 levels and a selective reduction in the numbers of CD8 alpha(+) conventional DCs (cDCs) in lymphoid organs. CT inhibited the function of interferon (IFN) regulatory factor (IRF) 8, a transcription factor known to positively regulate IL-12p35 and p40 gene expression, and the differentiation of CD8 alpha(+) and plasmacytoid DCs (pDCs). Fluorescence recovery after photobleaching analysis showed that exposure to CT, forskolin, or dibutyryl (db) cAMP blocked LPS and IFN-gamma-induced IRF8 binding to chromatin. Moreover, CT and dbcAMP inhibited the binding of IRF8 to the IFN-stimulated response element (ISRE)-like element in the mouse IL-12p40 promoter, likely by blocking the formation of ISRE-binding IRF1-IRF8 heterocomplexes. Furthermore, CT inhibited the differentiation of pDCs from fms-like tyrosine kinase 3 ligand-treated bone marrow cells in vitro."

"Therefore, because IRF8 is essential for IL-12 production and the differentiation of CD8 alpha(+) cDCs and pDCs, these data suggest that CT and other Gs-protein agonists can affect IL-12 production and DC differentiation via a common mechanism involving IRF8.."

The full article can be found at: (A. Lasala, et. al., "Cholera toxin inhibits IL-12 production and CD8 alpha(+) dendritic cell differentiation by cAMP-mediated inhibition of IRF8 function". Journal of Experimental Medicine, 2009;206(6):1227-1235). Link not available.

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UCSB SCIENTISTS DISCOVER POTENTIAL DRUG DELIVERY SYSTEM

Nanotechwire.com

August 25, 2009

"Scientists at UC Santa Barbara have discovered a potential new drug delivery system. The finding is a biological mechanism for delivery of nanoparticles into tissue. The results are published in this week's Proceedings of the National Academy of Sciences."

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"The scientists developed a peptide, a small piece of protein that can carry "cargo" for delivery into the cell. The cargo could be a nanoparticle, or even a cell. Riding on the peptide, the cargo gets out of the blood vessel and penetrates the tissue.

The drug is located at one end of the peptide. At the other is the "C terminal," which has the "motif" -- an amino acid sequence including arginine or lysine, that causes the tissue penetration. This terminal has to be open, the researchers found. The strict requirement for the C terminal led the group to coin a new name, the "C-end rule," or CendR, pronounced "sender."

The full article can be found at: <http://nanotechwire.com/news.asp?nid=8446>

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VIBRIO CHOLERAE PROTEOME-WIDE SCREEN FOR IMMUNOSTIMULATORY PROTEINS IDENTIFIES PHOSPHATIDYLSERINE DECARBOXYLASE AS A NOVEL TOLL-LIKE RECEPTOR 4 AGONIST

By Ann Thanawastien, Wagner R. Montor, Joshua LaBaer, John J. Mekalanos, Sang Sun Yoon

PLoS Pathogens

August 21, 2009

"Abstract

Recognition of conserved bacterial components provides immediate and efficient immune responses and plays a critical role in triggering antigen-specific adaptive immunity. To date, most microbial components that are detected by host innate immune system are non-proteinaceous structural components. In order to identify novel bacterial immunostimulatory proteins, we developed a new high-throughput approach called "EPSIA", Expressed Protein Screen for Immune Activators. Out of 3,882 *Vibrio cholerae* proteins, we identified phosphatidylserine decarboxylase (PSD) as a conserved bacterial protein capable of activating host innate immunity. PSD in concentrations as low as 100 ng/ml stimulated RAW264.7 murine macrophage cells and primary peritoneal macrophage cells to secrete TNF α and IL-6, respectively. PSD-induced proinflammatory response was dependent on the presence of MyD88, a known adaptor molecule for innate immune response. An enzymatically inactive PSD mutant and heat-inactivated PSD induced ~40% and ~15% of IL-6 production compared to that by native PSD, respectively. This suggests that PSD induces the production of IL-6, in part, via its enzymatic activity. Subsequent receptor screening determined TLR4 as a receptor mediating the PSD-induced

proinflammatory response. Moreover, no detectable IL-6 was produced in TLR4-deficient mouse macrophages by PSD. PSD also exhibited a strong adjuvant activity against a co-administered antigen, BSA. Anti-BSA response was decreased in TLR4-deficient mice immunized with BSA in combination with PSD, further proving the role of TLR4 in PSD signaling in vivo. Taken together, these results provide evidence for the identification of *V. cholerae* PSD as a novel TLR4 agonist and further demonstrate the potential application of PSD as a vaccine adjuvant.

Author Summary

Innate immune responses are the first line of defense and involve the early recognition of pathogenic microorganisms. Furthermore, these early innate responses can help shape and influence the development of more specific adaptive immune responses. One way that innate immunity is triggered is by activation of TLRs, or Toll-like Receptors. TLRs recognize a wide spectrum of microbes by binding to pathogen-associated molecular patterns (PAMPs), which are conserved microbial products. Here, we have used a high-throughput method to understand more about how a pathogen can trigger early innate immune responses and also how these early responses to infection can influence the adaptive, more specific, immune response. This technique can also be utilized for adjuvant discovery which is important in vaccine development since different adjuvants can induce or enhance different kinds of immune responses to a particular antigen. Using this method, we identified a novel bacterial protein that activates a TLR and further characterized its role as an adjuvant. Identifying the TLRs, their ligands, and the signal transduction events that they initiate has provided insight into our understanding of how the immune response to infection begins, and how these factors also collectively influence the adaptive immune response.”

The full article can be found at: <http://www.plospathogens.org/article/info%3Adoi%2F10.1371%2Fjournal.ppat.1000556;jsessionid=269618E943725A56C6A4C1B2BFB795B4>

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CHIP DETECTS MICROORGANISMS IN AMBIENT AIR

Nanotechwire.com

September 01, 2009

“Researchers from six Fraunhofer Institutes have now developed a test system capable of carrying out such tests on the spot and in less than half an hour. “We take a plastic chip and apply a gel to it. We embed special fluorescent-marked antibodies in this gel. These detect very specific microorganisms which can then be viewed under a fluorescence microscope,” explains Gerd Sulz, project manager at the Fraunhofer Institute for Physical Measurement Techniques IPM in Freiburg. The test system monitors the ambient air by accumulating different microorganisms and particles – including dust – on the gel, and segregating only particles between one and ten micrometers in size. The antibodies in the gel bind to specific microorganisms, which fit into them like a key in a lock. They do not bind to specks of dust or other germs. The task of the Freiburg scientists is to detect the identified microorganisms using an optical technique. The first step is a wash cycle that removes all the antibodies not bound to microorganisms. This is done by applying an electrical voltage – the unbound antibodies are so small that they can be moved through the gel by the electric field, whilst the antibodies that have latched onto a microorganism remain trapped. A glance at the chip reveals whether any antibodies, and how many, have bound to microorganisms – the trapped antibodies glow red due to the fluorescent marking. The result provides information about the type and number of potentially dangerous microorganisms in the air.

A prototype of the test system has already been built. The researchers have used it successfully to carry out measurement cycles with relevant test particles and are now working on software to fully automate the process. The researchers will be presenting the test system at the Anuga food trade fair in Cologne from October 10 to 14 (Hall 5, Stand B020).”

The full article can be found at: <http://www.nanotechwire.com/news.asp?nid=8498>

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MECHANISED NANOCAPSULES TARGET DRUG DELIVERY

By Matt Wilkinson
Chemistry World
September 02, 2009

“US researchers have developed a nano-sized drug delivery system that only releases its payload in specific pH conditions, a feature that could prove particularly useful for targeted delivery of cancer treatments.”

.....

“But now, a collaboration between US researchers in Fraser Stoddart's team at Northwestern University, Illinois and Jeffrey Zink's team at the University of California, Los Angeles (UCLA), has led to the development of 'mechanised nanoparticles' that release their payload in response to a change in pH.

At the heart of the system is a mesoporous silica nanoparticle of around 200nm in size which would carry the drug and is easily absorbed by cells. But the payload would leak through the particle's pores without what Stoddart describes as all-important 'nanovalves'.

These nanovalves are 'stalks' that carry a ring, like a cyclodextrin or cucurbituril, explains Stoddart, which controls the release of the cargo from the core of the mesoporous particle.

The stalks are made of a chain consisting of three nitrogen atoms, separated by a series of carbon atoms positioned so that they hydrogen bond with the oxygen atoms around the rims of the cucurbituril ring.

Two ammonium groups close to the nanoparticle are separated by four carbon atoms while the 'terminal' anilinium nitrogen is separated from the central nitrogen atom by six carbon atoms.”

The full article can be found at:

<http://www.rsc.org/chemistryworld/News/2009/September/02090903.asp>

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