

## บทความที่น่าสนใจประจำเดือน สิงหาคม 2556

<b>Title :</b>	<a href="#">Calcineurin expression and localisation during porcine oocyte growth and meiotic maturation</a>
<b>Author :</b>	Lenka Tůmová, Jaroslav Petr, Tereza Žalmanová, Eva Chmelíková, Tomáš Kott, Hana Tichovská, Veronika Kučerová-Chrpová, Kristýna Hošková, František Jílek
<b>Journal :</b>	Animal Reproduction Science, Available online 6 August 2013 : In Press, Accepted Manuscript, <a href="http://dx.doi.org/10.1016/j.anireprosci.2013.07.011">http://dx.doi.org/10.1016/j.anireprosci.2013.07.011</a>
<b>Abstract :</b>	<p>The processes of oocyte growth, acquisition of meiotic competence and meiotic maturation are regulated by a large number of molecules. One of them could be calcineurin consisting of catalytic subunit A (A<math>\alpha</math>, A<math>\beta</math>, A<math>\gamma</math> isoforms) and regulatory subunit B (B1, B2 isoforms). Calcineurin is involved in the meiotic maturation of oocytes in invertebrates or in lower vertebrates. In the mammalian oocytes, the possible role of calcineurin in the regulation of oocyte meiosis has not been clarified to date. In this study, to investigate the role of calcineurin during porcine oocyte growth, acquisition of meiotic competence and meiotic maturation, we analysed the expression and localisation of calcineurin subunits and the mRNA expression of calcineurin isoforms. Calcineurin was expressed in growing porcine oocytes, in fully-grown oocytes and during their in vitro meiotic maturation. We found both subunits of calcineurin. Calcineurin A and calcineurin B were localised mainly in cortex in all porcine oocytes. The changes in the intracellular localisation of separate calcineurin subunits during meiotic maturation were determined. We detected mRNA for calcineurin isoforms A<math>\beta</math>, A<math>\gamma</math>, B2 in oocytes and mRNA for calcineurin isoforms A<math>\beta</math>, A<math>\gamma</math>, B1, and B2 in cumular cells. To our knowledge, this is the first confirmation of calcineurin presence in porcine oocytes.</p>
<b>Database :</b>	ScienceDirect

<b>Title :</b>	<a href="#">Combined Hepatocellular and Cholangiocarcinoma (Biphenotypic) Tumors: Imaging Features and Diagnostic Accuracy of Contrast-Enhanced CT and MRI</a>
<b>Author :</b>	Kathryn J. Fowler, Arman Sheybani, Rex A. Parker III, Sean Doherty, Elizabeth M. Brunt, William C. Chapman, Christine O. Menias
<b>Journal :</b>	American Journal of Roentgenology: August 2013, Volume 201, Number 2, p 332-339
<b>Abstract :</b>	<p>The purpose of this study was to evaluate the diagnostic accuracy of preoperative imaging for diagnosis of combined hepatocellular cholangiocarcinoma tumors and to evaluate the clinical and imaging features and demographics of patients presenting to our institution with such tumors.</p>
<b>Database :</b>	Free Medical Journals

<b>Title :</b>	<a href="#">A Radiologist's Guide to Treatment Response Criteria in Oncologic Imaging: Functional, Molecular, and Disease-Specific Imaging Biomarkers</a>
<b>Author :</b>	Pedram Rezai, Mark J. Pisaneschi, Chun Feng and Vahid Yaghamai
<b>Journal :</b>	American Journal of Roentgenology: August 2013, Vol. 201, Number 2, p 246-256
<b>Abstract :</b>	<p>This article reviews the functional, molecular, and disease-specific imaging biomarkers of treatment response.</p>
<b>Database :</b>	Free Medical Journals

<b>Title :</b>	<a href="#">Lipid mediator serum profiles in asthmatics significantly shift following dietary supplementation with omega-3 fatty acids (pages 1378–1389)</a>
<b>Author :</b>	Susanna L. Lundström, Jun Yang, John D. Brannan, Jesper Z. Haeggström, Bruce D. Hammock, Parameswaran Nair, Paul O'Byrne, Sven-Erik Dahlén, Craig E. Wheelock
<b>Journal :</b>	Molecular Nutrition & Food Research (Special Issue: Lipidomics: Approaches and Applications in Nutrition Research), August 2013, Volume 57, Issue 8, pages 1378–1389
<b>Abstract :</b>	In contrast to well-characterized PUFA levels in serum, little is known regarding their downstream metabolic products. However, many of these compounds are lipid mediators with prominent roles during pro- and antiinflammatory processes.
<b>Database :</b>	Wiley Online Library

<b>Title :</b>	<a href="#">Prevention of diet-induced obesity by apple polyphenols in Wistar rats through regulation of adipocyte gene expression and DNA methylation patterns (pages 1473–1478)</a>
<b>Author :</b>	Noemi Boqué, Rocío de la Iglesia, Ana L. de la Garza, Fermín I. Milagro, Mónica Olivares, Óscar Bañuelos, Ana Cristina Soria, Sonia Rodríguez-Sánchez, José Alfredo Martínez, Javier Campián
<b>Journal :</b>	Molecular Nutrition & Food Research (Special Issue: Lipidomics: Approaches and Applications in Nutrition Research), August 2013, Volume 57, Issue 8, pages 1473–1478
<b>Abstract :</b>	This study was conducted to determine the mechanisms implicated in the beneficial effects of apple polyphenols (APs) against diet-induced obesity in Wistar rats, described in a previous study from our group. Supplementation of high-fat sucrose diet with AP prevented adiposity increase by inhibition of adipocyte hypertrophy. Rats supplemented with AP exhibited improved glucose tolerance while adipocytes isolated from these rats showed an enhanced lipolytic response to isoproterenol. AP intake led to reduced Lep, Plin, and sterol regulatory element binding transcription factor 1 (Srebf1) mRNA levels and increased aquaporin 7 (Aqp7), adipocyte enhancer binding protein 1 (Aebp1), and peroxisome proliferator-activated receptor gamma co-activator 1 alpha (Ppargc1a) mRNA levels in epididymal adipocytes. In addition, we found different methylation patterns of Aqp7, Lep, Ppargc1a, and Srebf1 promoters in adipocytes from apple-supplemented rats compared to high-fat sucrose fed rats. The administration of AP protects against body weight gain and fat deposition and improves glucose tolerance in rats. We propose that AP exerts the antiobesity effects through the regulation of genes involved in adipogenesis, lipolysis, and fatty acid oxidation, in a process that could be mediated in part by epigenetic mechanisms.
<b>Database :</b>	Wiley Online Library

<b>Title :</b>	<a href="#">Visual size perception and haptic calibration during development</a>
<b>Author :</b>	Monica Gori, Luana Giuliana, Giulio Sandini, David Burr
<b>Journal :</b>	Developmental Science: Early View (Online Version of Record published before inclusion in an issue), Article first published online: 12 SEP 2012 DOI: 10.1111/j.1467-7687.2012.01183.x

<b>Abstract :</b>	It is still unclear how the visual system perceives accurately the size of objects at different distances. One suggestion, dating back to Berkeley's famous essay, is that vision is calibrated by touch. If so, we may expect different mechanisms involved for near, reachable distances and far, unreachable distances. To study how the haptic system calibrates vision we measured size constancy in children (from 6 to 16 years of age) and adults, at various distances. At all ages, accuracy of the visual size perception changes with distance, and is almost veridical inside the haptic workspace, in agreement with the idea that the haptic system acts to calibrate visual size perception. Outside this space, systematic errors occurred, which varied with age. Adults tended to overestimate visual size of distant objects (over-compensation for distance), while children younger than 14 underestimated their size (under-compensation). At 16 years of age there seemed to be a transition point, with veridical perception of distant objects. When young subjects were allowed to touch the object inside the haptic workspace, the visual biases disappeared, while older subjects showed multisensory integration. All results are consistent with the idea that the haptic system can be used to calibrate visual size perception during development, more effectively within than outside the haptic workspace, and that the calibration mechanisms are different in children than in adults.
<b>Database :</b>	Wiley Online Library

<b>Title :</b>	<a href="#">Episodic autobiographical memory in amnesic mild cognitive impairment: What are the neural correlates?</a>
<b>Author :</b>	Christine Bastin, Dorothee Feyers, Haroun Jedidi, Mohamed Ali Bahri, Christian Degueldre, Christian Lemaire, Fabienne Collette, Eric Salmon
<b>Journal :</b>	Human Brain Mapping: August 2013, Volume 34, Issue 8, pages 1811–1825
<b>Abstract :</b>	Autobiographical memory in amnesic Mild Cognitive Impairment (aMCI) is characterized by impaired retrieval of episodic memories, but relatively preserved personal semantic knowledge. This study aimed to identify (via FDG-PET) the neural substrates of impaired episodic specificity of autobiographical memories in 35 aMCI patients compared with 24 healthy elderly controls. Significant correlations between regional cerebral activity and the proportion of episodic details in autobiographical memories from two life periods were found in specific regions of an autobiographical brain network. In aMCI patients, more than in controls, specifically episodic memories from early adulthood were associated with metabolic activity in the cuneus and in parietal regions. We hypothesized that variable retrieval of episodic autobiographical memories in our aMCI patients would be related to their variable capacity to reactivate specific sensory-perceptual and contextual details of early adulthood events linked to reduced (occipito-parietal) visual imagery and less efficient (parietal) attentional processes. For recent memories (last year), a correlation emerged between the proportion of episodic details and activity in lateral temporal regions and the temporo-parietal junction. Accordingly, variable episodic memory for recent events may be related to the efficiency of controlled search through general events likely to provide cues for the retrieval of episodic details and to the ability to establish a self perspective favouring recollection. Hum Brain Mapp, 2013. © 2012 Wiley Periodicals, Inc.
<b>Database :</b>	Wiley Online Library

<b>Title :</b>	<a href="#">Suppression of interactions between prostate tumor cell-surface integrin and endothelial ICAM-1 by simvastatin inhibits micrometastasis</a>
<b>Author :</b>	Belal AL-Husein, Anna Goc, Payaningal R. Somanath
<b>Journal :</b>	Journal of Cellular Physiology: November 2013, Volume 228, Issue 11, pages 2139–2148
<b>Abstract :</b>	Cancer micrometastasis relies on the ability of cancer cells to secrete angiogenic modulators, to interact with the vascular endothelium, and to overcome the resistance offered by the endothelial-barrier. Being an essential step prior to metastasis, blockage of micrometastasis can have potential applications in cancer therapy and metastasis prevention. Due to poorly known molecular mechanisms leading to micrometastasis, developing therapeutic strategies to target prostate cancer utilizing drugs that block micrometastasis is far from reality. Here, we demonstrate the potential benefits of simvastatin in the inhibition of prostate cancer micrometastasis and reveal the novel molecular mechanisms underlying this process. First, we showed that simvastatin inhibited the ability of human PC3 prostate cancer cells for transendothelial migration in vitro. Second, our data indicated that simvastatin modulates the expression of tumor-derived factors such as angiopoietins and VEGF-A at the mRNA and protein levels by the PC3 cells, thus preventing endothelial-barrier disruption. Third, simvastatin directly activated endothelial cells and enhances endothelial-barrier resistance. Apart from this, our study revealed that simvastatin-mediated effect on PC3 micrometastasis was mediated through inhibition of integrin $\alpha\beta 3$ activity and suppression of interaction between prostate cancer cell integrin $\alpha\beta 3$ with endothelial ICAM-1. J. Cell. Physiol. 228: 2139–2148, 2013. © 2013 Wiley Periodicals, Inc.
<b>Database :</b>	Wiley Online Library

<b>Title :</b>	<a href="#">Acetaminophen attenuates doxorubicin-induced cardiac fibrosis via osteopontin and GATA4 regulation: Reduction of oxidant levels</a>
<b>Author :</b>	Kathryn J. Schunke, Luke Coyle, Gary F. Merrill, David T. Denhardt
<b>Journal :</b>	Journal of Cellular Physiology: October 2013, Volume 228, Issue 10, pages 2006–2014
<b>Abstract :</b>	It is well documented in animal and human studies that therapy with the anti-cancer drug doxorubicin (DOX) induces fibrosis, cardiac dysfunction, and cell death. The most widely accepted mechanism of cardiac injury is through production of reactive oxygen species (ROS), which cause mitochondrial damage, sarcomere structural alterations, and altered gene expression in myocytes and fibroblasts. Here we investigated the effects of acetaminophen (APAP, N-acetyl-para-aminophenol) on DOX-induced cardiac injury and fibrosis in the presence or absence of osteopontin (OPN). H9c2 rat heart-derived embryonic myoblasts were exposed to increasing concentrations of DOX $\pm$ APAP; cell viability, oxidative stress, and OPN transcript levels were analyzed. We found a dose-dependent decrease in cell viability and a corresponding increase in intracellular oxidants at the tested concentrations of DOX. These effects were attenuated in the presence of APAP. RT-PCR analysis revealed a small increase in OPN transcript levels in response to DOX, which was suppressed by APAP. When male 10–12-week-old mice (OPN $^{+/+}$ or OPN $^{-/-}$ ) were given weekly injections of DOX $\pm$ APAP for 4 weeks there was substantial cardiac fibrosis in OPN $^{+/+}$ and, to a lesser extent, in OPN $^{-/-}$ mice. In both groups, APAP decreased fibrosis to near baseline levels. Activity of the pro-survival GATA4 transcription factor was diminished by DOX in both mouse genotypes, but retained baseline activity in the presence of APAP.

	These effects were mediated, in part, by the ability of APAP, acting as an anti-inflammatory agent, to decrease intracellular ROS levels, consequently diminishing the injury-induced increase in OPN levels. J. Cell. Physiol. 228: 2006–2014, 2013. © 2013 Wiley Periodicals, Inc.
<b>Database :</b>	Wiley Online Library

<b>Title :</b>	<a href="#">Pharmacokinetic Study of Adenosine Diphosphate-Encapsulated Liposomes Coated with Fibrinogen <math>\gamma</math>-Chain Dodecapeptide as a Synthetic Platelet Substitute in an Anticancer Drug-Induced Thrombocytopenia Rat Model</a>
<b>Author :</b>	Kazuaki Taguchi, Hayato Ujihira <sup>1</sup> , Hiroshi Watanabe, Atsushi Fujiyama, Mami Doi <sup>3</sup> , Shinji Takeoka, Yasuo Ikeda, Makoto Handa, Masaki Otagiri, Toru Maruyama
<b>Journal :</b>	Journal of Pharmaceutical Sciences: Early View (Online Version of Record published before inclusion in an issue), Article first published online: 5 AUG 2013 DOI: 10.1002/jps.23692
<b>Abstract :</b>	A fibrinogen $\gamma$ -chain (dodecapeptide HHLGGAKQAGDV, H12)-coated, adenosine diphosphate (ADP)-encapsulated liposome [H12-(ADP)-liposome] was designed to achieve optimal performance as a homeostatic agent and expected as a synthetic platelet alternative. For the purpose of efficient function as platelet substitute, H12-(ADP)-liposomes should potentially have both acceptable pharmacokinetic and biodegradable properties under conditions of an adaptation disease including thrombocytopenia induced by anticancer drugs. The aim of this study was to characterize the pharmacokinetics of H12-(ADP)-liposomes in busulphan-induced thrombocytopenic rats using <sup>14</sup> C, <sup>3</sup> H double radiolabeled H12-(ADP)-liposomes, in which the encapsulated ADP and liposomal membrane (cholesterol) were labeled with <sup>14</sup> C and <sup>3</sup> H, respectively. After the administration of H12-(ADP)-liposomes, they were determined to be mainly distributed to the liver and spleen and disappeared from organs within 7 days after injection. The encapsulated ADP was mainly eliminated in the urine, whereas the outer membrane (cholesterol) was mainly eliminated in feces. The successive dispositions of the H12-(ADP)-liposomes were similar in both normal and thrombocytopenic rats. However, the kinetics of H12-(ADP)-liposomes in thrombocytopenic rats was more rapid, compared with the corresponding values for normal rats. These findings, which well reflect the clinical features of patients with anticancer drug-induced thrombocytopenia, provide useful information for the development of the H12-(ADP)-liposomes for future clinical use. © 2013 Wiley Periodicals, Inc. and the American Pharmacists Association J Pharm Sci
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