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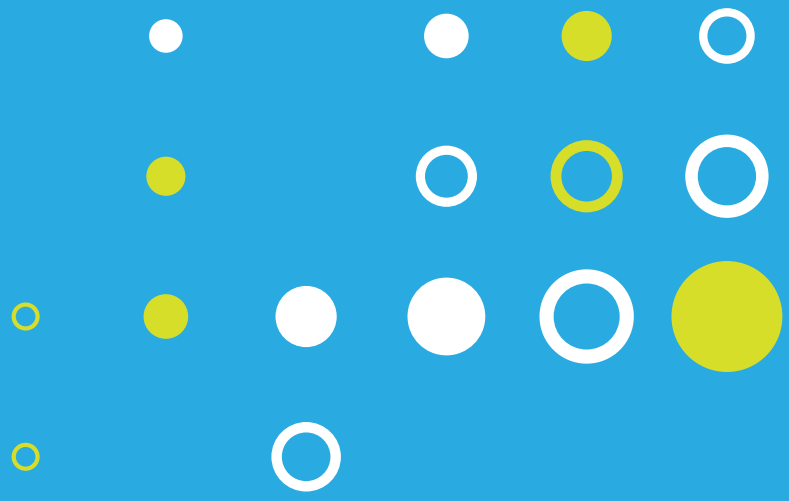

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CB-P49.

DIPHENYL DISELENIDE MODULATES MACROPHAGE ACTIVATION BY DECREASING PRO- AND ANTI-INFLAMMATORY MARKERS

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The biological importance of selenium led to the development of pharmacologically active organoselenium compounds. Diphenyl diselenide (PhSe)2 is an organoselenium compound whose biological activities have been poorly described. Previously, we evaluated its antioxidant and anti-inflammatory properties in an *in vitro* model of inflammation. We found that (PhSe)2 was able to prevent the production of reactive oxygen species, nitric oxide, the expression of iNOS, the peroxynitrite modification of proteins (nitrotyrosine immunostaining) and the antigen presentation capacity of LPS stimulated macrophages (Mph). Next, we focused on studying the ability of (PhSe)2 to modulate the alternative activation of Mph, which encompasses an upregulation of anti-inflammatory mediators. We isolated peritoneal Mph and stimulated them with dexamethasone to induce the alternative activation phenotype. We observed that activated Mph presented higher expression of IL-10, which was downregulated by (PhSe)2. Then, we examined the surface expression of mannose receptor (CD206), and found that dexamethasone treatment augmented the expression of CD206, while (PhSe)2 was able to downregulate this molecule. Finally, preliminary results indicate that (PhSe)2 decreased the ratio of IL-10/IL-12 mRNA in dexamethasone treated Mph. These findings suggest that (PhSe)2 could be used to modulate the activation of Mph.

CB-P50.

NICOTINIC ACETYLCHOLINE RECEPTOR CLUSTERS LOCALIZATION ON LIPID DOMAINS OF THE PLASMALEMMA

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In the present work we have attempted to establish whether there is a correlation between antibody-induced nicotinic acetylcholine receptor (AChR)-rich clusters and the physical state of the underlying cell membrane in living cells. For this purpose di-4-ANEPPDHQ, a fluorescent probe that differentiates liquid-ordered from liquid-disordered phases in model membranes was used in combination with labeling of the AChR in CHO-K1/A5, a clonal cell line expressing adult muscle-type AChR. The so-called generalized polarization ("GP") of di-4-ANEPPDHQ was measured in regions of the cell-surface membrane associated with the AChR platforms clearly identified using conventional wide-field fluorescence microscopy. Under control conditions AChR clusters are roughly equally distributed among liquid-ordered and liquid-disordered domains. This distribution changes upon cyclodextrin-mediated cholesterol depletion or by Latrunculin disruption of the actin cytoskeleton. Association of AChR clusters with lipid domains with different biophysical properties may have consequences on AChR trafficking processes.

CB-P51.

YACON LEAVES AMELLIORATE TGF-ß1/SMAD SIGNALING IN DIABETIC KIDNEY

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Nephropathy is a common cause of morbidity and mortality in diabetic patients. Prevention of this complication is clearly required. *Smallanthus sonchifolius* (yacon) leaves containing mainly polyphenolic acids and the lactone enhydrin have been shown to ameliorate hyperglycemia in streptozotocin-induced diabetic rats. In the present study, we examined the beneficial effects of yacon leaves decoction in diabetic kidney and explored the possible underlying mechanism.
Diabetic rats were orally administered with 10% yacon leaves water decoction (140mg dry extract/kg b.w.) for 30 days. Biochemical parameters in blood and urine, immunohistochemistry, western immunoblotting and qRT-PCR analysis were developed.
Yacon decoction significantly decreased high blood glucose level in diabetic rats and improved insulin production. Diabetic-dependent alterations in urinary albumin excretion, creatinine clearance, kidney hypertrophy and basement membrane thickening were attenuated by yacon decoction. These findings were associated with a marked decrease in TGFß1, TGFRII and p-Smad2/3. The expression of extracelullar matrix proteins as collagen IV, laminin-1, fibronectin and collagen III were also diminished in the yacon-treated group. These results suggest that yacon leaves is a protective agent against renal damage in diabetic nephropathy, whose action can be mediated by TGFß/Smads signals.

CB-P52.

IMPLICATION OF PKC ISOFORMS IN REVERSION OF MAMMARY TUMORS MALIGNANCY IN RESPONSE TO RETINOIC ACID

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In this work we have overeexpressed in LM3 murine mammary cells the and isoforms of PKC in order to study whether these genetically modified sublines are more sensitive to retinoid treatment (ATRA).
Through a reporter gene assay, using the retinoic acid responsive elements upstream luciferase gene (RARE-Luciferase), we could determine that only PKC overexpression induced an increase in the activity of these sites. This result correlates with previous assays showing that PKC traslocates to the nucleus coupled to retinoid receptors. ATRA effect was also studied *in vivo* and *in vitro*, evaluating parameters related to tumor growth and dissemination. While PKC overexpression induced an important increase in the *in vitro* proliferative capacity, only these overexpressors become sensitive to ATRA treatment showing a proliferative delay. Moreover, LM3-PKC cells also showed a higher migratory capacity, also reversed by retinoid treatment. *In vivo* assays showed that only PKC overexpression induced an increase in tumor growth and metastatic potential, and ATRA treatment was able to limit the malignant progression of these tumors. Our results suggest that, PKC overexpression confers a more aggressive phenotype but make the cells sensitive to ATRA effects, while PKC is necessary for retinoid receptors traslocation but is insufficient to alter cellular response to retinoid treatment.