

externalization, and did not affect ROS generation. Conversely, addition of antioxidants fully prevented Ca^{2+} influx and eryptosis. Either staurosporin or Z-DEVD-FMK blunted PS externalization. Oxysterols inhibited APLT and inward transport of PS. Only 7-ketocholesterol and cholestan-3 β ,5 α ,6 β -triol were individually active. Significant eryptosis was observed *ex vivo*.

Conclusions: Oxidative stress triggers oxysterol-induced eryptosis, with coordinated and complementary processes not strictly dependent on calcium activity. Potential relevance for eryptotic activity of oxysterols *in vivo* is suggested.

Study and testing of medical devices for remote monitoring of patients with chronic conditions.

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Remote healthcare systems have received increasing attention in the last decade, explaining why intelligent systems for e-health care are an emerging area of development(1). Telemonitoring is a tool based on the use of communication technologies to monitor simple clinical variables, in order to enable early detection of any alteration, providing an opportunity to prevent hospitalization(2). We used a physiological status monitor (PSM) embedded into a compression shirt to enable patients to measure and transmit vital metrics. We monitored 12 individuals and each individual was monitored for 8 hours. The components of the PSM were: 1) two sensors for the detection of ECG and the heart rate (HR), 2) an electric piezo-resistive sensor for respiratory rate (RR), 3) a triaxial accelerometer for posture of the individuals, 4) a control unit, integrated with the shirt, that recorded all signals in an internal memory and transferred the data to an external unit by Bluetooth. The subjects were enabled to introduce markers during the recording, whenever they felt discomfort. All the data were then transferred to a medical records repository for an easy consultation. The physician could access the repository, in a secure way, to analyze the record and evaluate any significant alteration of the ECG, HR and RR. It would be interesting to make a cloud network that allow patients to share their vital parameters in the net and medical personnel to be able to see the information of each individual, through the electronic health dossier, in real time.

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Codon 12 and codon 13 mutations in K-RAS differentially affect therapies response of colorectal carcinoma cells.

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The colorectal cancer (CRC) is a heterogeneous disease, which develops as a result of numerous genetic and epigenetic alterations. A gene family frequently mutated in tumors is that of ras which consists of three main oncogenes (H-, K- and N-Ras) on different chromosomes and encode 21 KDa G-proteins. The RAS proteins are involved in a highly conserved signal transduction pathways that regulates proliferation, differentiation and apoptosis and their activating mutations have oncogenic effects. K-RAS mutations, usually point mutations in codons 12 or 13, are common in CRC. Several studies show that mutations in different codons or different mutations in the same codon of K-Ras may have different biological effects and lead to a different response

to therapies based on Cetuximab, an anti-EGFR monoclonal antibody. K-RAS mutations are in fact considered to be a resistance factor to this drug, but it has been reported that tumors with codon 13 K-RAS mutations might be less resistant to Cetuximab than tumors with codon 12 mutations. To gain more information on this point, we analyzed HT-29 clones stably transfected with cDNAs codifying K-RASG12V (clone K12) and K-RASG13D (clone K13) under the control of an inducible promoter. We observed that Cetuximab affects the cell cycle and proliferation of the K13 clone expressing K-RASG13D but not the K12 cells induced to express K-RASG12V. These results support the hypothesis that tumors with different K-RAS mutations could respond differently to therapies. Moreover preliminary data show that Cetuximab has no effect on the rate of cell proliferation of the parental HT29 cell line, in which the Ras genes are wild type but there is a mutation in BRAF, nor on its cell cycle. However, western blot analysis show a Cetuximab effect on the level of expression and phosphorylation (i.e., activation) of the tumor suppressor protein P53, but not on that of one of the P53 targets, the cell cycle regulatory protein P21.

Bacteria consortia and deterioration of archaeological waterlogged wood: identification by molecular and microscopy techniques

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Keywords: **Waterlogged wood, Biodeterioration, Molecular investigation**

In this study molecular tools are applied to reveal and identify bacterial colonization in waterlogged wood to assessing the changes induced in anatomical structure, previously observed by Optical and Scanning Electron Microscopy (1). The results obtained by observation of wooden thin sections (OM), shown the presence of black and dark-brown areas and mineral concretions. The SEM analysis revealed a specific cell walls alteration, attributable to bacterial activity, other than abundant pyrite framboids (FeS₂). The presence of sulfur compounds in archaeological waterlogged wood can indicate both long-term burial in anoxic environment and colonization by sulfate-reducing bacteria. Molecular methods allow us extract microbial genomic DNA from wood samples and *in vitro* amplify (PCR) bacteria DNA target sequences (16S, ITS-rRNA) (2). Through sequences analysis of PCR products cellulosolytic and ligninolytic bacteria, such as *Pseudomonas*, *Cellulomonas*, *Xanthomonas* and *Bacillus* spp, have been revealed. Moreover the presence of *Marinobacter* sp. and *Desulforudis audaxviator*, respectively iron-oxidizing and sulfate-reducing bacteria, are identify. We hypothesize that this investigation approach, can be applied to a variety of wooden artifacts of archaeological findings for both characterization of microbial colonization in order to understanding the main degradation phenomena, indispensable for a correct conservation strategies.

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