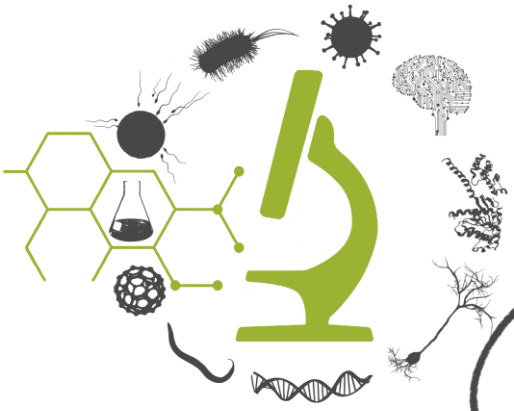
III CONGRESO NACIONAL DE JÓVENES INVESTIGADORES EN BIOMEDICINA

III National Congress of Young Researchers in Biomedicine



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BOOK OF ABSTRACTS

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P04-3 How to strengthen the recommendations for the informed consent process in health-related studies: The i-CONSENT project methodology

Jaime Fons-Martínez; Júlia García-Bayarri; Javier Díez-Domingo.

FISABIO

i-CONSENT is a H2020 project (GA 741856) that aims to develop a series of guidelines to improve the informed consent process (ICP) in health-related studies, being scientific evidence an important input for this outcome. Systematic and narrative reviews of the literature, hard law and soft law were performed in order to provide an in-depth analysis of the following issues and factors associated with the ICP: • Strategies to improve the ICP • Gender and age-related issues associated with acquisition of informed consent (IC) • Ethical and legal issues concerning the ICP • Socio-cultural and psychological perspectives toward IC. The results of this research where used to elaborate the first version of the guidelines. Many gaps related to the information on informed consent were identified during this process and some of the evidences that supported the recommendations were considered weak. To strengthen and reinforce these recommendations, a workshop with representatives of the main stakeholders involved in the ICP (Regulators, Pharmaceutical Industry, Investigators, Patient Organizations and Ethical Advisory Boards) was organised. Key recommendations were identified, put into flashcards and specific questions on them and on how to put them in practice were prepared to guide the discussion. The flashcards were distributed to the stakeholders considered more suitable to address the issue (2 stakeholders by each flashcard) and each of them discussed independently the topics assigned. After the workgroup the conclusions were presented by each stakeholder to the rest of attendants and a final discussion to complete the perspective was conducted. The results and conclusions by stakeholder were analysed and the perspectives were compared. Some final recommendations were elaborated combining the information from the literature with the expertise and point of view of the stakeholders involved, increasing the robustness of these recommendations and filling some of the gaps identified. This communication includes examples.

P04-4 Metformin and salicylate reduce polyglutamine aggregation through synergistic activation of AMPK in *C. elegans*

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AMPK is an enzyme which functions as a master regulator of the homeostasis of energy levels in the cell. In recent years, it has been shown that activation of AMPK, in in vitro and in vivo models, reduces the toxicity induced by polyglutamines (polyQ). AMPK is an obligate heterotrimer (AMPKα, AMPKβ and AMPKγ) that can be activated allosterically by AMP, which binds to the regulatory subunit AMPKβ. This enzyme is also activated by direct phosphorylation of AMPKα. When active, AMPK restores reduced ATP levels, by inhibiting anabolic pathways, and activating catabolic reactions and other processes that may produce energy, such as autophagy. Moreover, it has been described elsewhere that AMPK can be synergistically activated by other substances, such as metformin and salicylate. Previous results from our group show that metformin, which is an indirect activator of AMPK, improves the aggregation pattern in worm and mouse models of polyQ toxicity. In this work, we demonstrate that metformin and salicylate activate synergistically AMPK, and through this they reduce polyQ aggregation in body wall muscles. Activation of AMPK by these substances is also able to induce neuroprotection of mechanosensory neurons in C. elegans. Moreover, our findings show that the neuroprotection effect induced by both drugs requires the function of AMPKα (catalytic subunit) and AMPKβ2 (regulatory subunit 2), while AMPKβ1 (regulatory subunit 1) is just partially required. Finally, our results indicate that autophagy could play a role in the modulation of polyQ aggregation after activation by metformin and salicylate.

P04-5 Inhibition of pcsk9 as a promising therapeutic target to prevent cardiovascular events in patients with familial hypercholesterolemia.

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Introduction and Objectives: Proprotein convertase subtilisin/kexin type 9 (PCSK9) is an enzyme ubiquitously expressed in many tissues and cell types. It binds to LDL-C receptor and promotes its degradation. Gain-of-function mutations in PCSK9 gene are found in Familial Hypercholesterolemia (FH)