

SELEZIONE N. 2021S31, PER ESAMI, PER LA STIPULA DI N. 1 CONTRATTO DI LAVORO A TERMINE, CATEGORIA D, POSIZIONE ECONOMICA D1, AREA TECNICA, TECNICO-SCIENTIFICA ED ELABORAZIONE DATI, TEMPO PIENO, PER 12 MESI, AI SENSI DEL D.LGS. 30.03.2001, N. 165 E S.M.I., DEL D.LGS. 15.06.2015, N. 81 IN QUANTO COMPATIBILE E DEL C.C.N.L. DEL 19.04.2018, PRESSO IL DIPARTIMENTO DI SCIENZE BIOMEDICHE - DSB - TECNICO A SUPPORTO DELLA RICERCA SU MECCANISMO D'AZIONE DEI CORRETTORI DEL CFTR E GENERAZIONE DI MODELLI ARTIFICIALI DI MUSCOLO, PRESSO L'UNIVERSITA' DEGLI STUDI DI PADOVA. (avviso pubblicato all'Albo ufficiale il 22 giugno 2021)

QUESITI COLLOQUIO

Busta 1 — Testo 1

- 1) Endoplasmic Reticulum associated degradation (ERAD): meccanismo d'azione
- 2) Metodi per la down regolazione dell'espressione di un gene
- 3) Metodi per la valutazione del differenziamento del tessuto muscolare (in vitro)

Informatica: Definizione di memoria ram e rom

Mitofusin 2: from functions to disease Mitochondria are highly dynamic organelles whose functions are essential for cell viability. Within the cell, the mitochondrial network is continuously remodeled through the balance between fusion and fission events. Moreover, it dynamically contacts other organelles, particularly the endoplasmic reticulum, with which it enterprises an important functional relationship able to modulate several cellular pathways. Being mitochondria key bioenergetics organelles, they have to be transported to all the specific high—energy demanding sites within the cell and, when damaged, they have to be efficiently removed. Among other proteins, Mitofusin 2 represents a key player in all these mitochondrial activities (fusion, trafficking, turnover, contacts with other organelles), the balance of which results in the appropriate mitochondrial shape, function, and distribution within the cell. Here we review the structural and functional properties of Mitofusin 2, highlighting its crucial role in several cell pathways, as well as in the pathogenesis of neurodegenerative diseases, metabolic disorders. cardiomyopathies, and cancer.

Busta 2 — Testo 2

- 1) Sistema ubiquitina proteasoma
- 2) Produzione di matrici extracellulari deeciularizzate e ricellularizzazione
- 3) qRT-PCR: descrizione della procedura Informatica: Cosa si intende per dispositivi di input e output in informatica?

Calsequestrins in skeletal and cardiac muscle from adult Danio rerio Calsequestrin (Casq) is a high capacity, low affinity Ca^{2+} —binding protein, critical for Ca^{2+} -buffering in cardiac and skeletal muscle sarcoplasmic reticulum. All vertebrates have multiple genes encoding for different Casq isoforms. Increasing interest has been focused on mammalian and human Casq genes since mutations of both cardiac (Casq2) and skeletal muscle (Casq1) isoforms cause different, and sometime severe, human pathologies. Danio rerio (zebrafish) is a powerful model for studying function and mutations of human proteins. In this work, expression, biochemical properties cellular and sub—cellular localization of D. rerio native Casq isoforms are investigated. By quantitative PCR, three mRNAs were detected in skeletal muscle and heart with different abundances. Three zebrafish Casqs: Casq1a, Casq1b and Casq2 were identified by mass spectrometry (Data

are available via ProteomeXchange with identifier PXD002455). Skeletal and cardiac zebrafish calsequestrins share properties with mammalian Casq1 and Casq2. Skeletal Casqs were found primarily, but not exclusively, at the sarcomere Z—line level where terminal cisternae of sarcoplasmic reticulum are located.

Busta 3 — Testo 3

1) Unfolded protein response

2) Immunoprecipitazione delle proteine

3) Editing genomico mediante CRISPR/CasQ Informatica: Quali programmi conosce per l'analisi di immagini? Bacterial toxins with intracellular protease activity The recent determination of their primary sequence has led to the discovery of the metallo—proteolytic activity of the bacterial toxins responsible for tetanus, botulism and anthrax. The protease domain of these toxins enters into the cytosol where it displays a zinc-dependent endopeptidase activity of remarkable specificity. Tetanus neurotoxin and botulinum neurotoxins type B, D, F and G cleave VAMP, an integral protein of the neurotransmitter containing synaptic vesicles. Botulinum neurotoxins type A and E cleave SNAP—25, while the type C neurotoxin cleaves both SNAP—25 and syntaxin, two proteins located on the cytosolic face of the presynaptic membrane. Such specific proteolysis leads to an impaired function of the neuroexocytosis machinery with blockade of neurotransmitter release and consequent paralysis. The lethal factor of *Bacillus anthracis* is specific for the MAPkinase—kinases which are cleaved within their amino terminus. In this case, however, such specific biochemical lesion could not be correlated with the pathogenesis of anthrax. The recently determined sequence of the vacuolating cytotoxin of *Helicobacter pylori* contains within its amino terminal domain elements related to serine—proteases, but such an activity as well as its cytosolic target remains to be detected.

Busta 4 — Testo 4

1) Modifiche post-traduzionali delle proteine nel reticolo endoplasmatico

2) Analisi della ubiquitinazione delle proteine

3) Analisi della localizzazione cellulare delle proteine

Informatica: Quali programmi conosce per l'analisi statistica di dati sperimentali?

Mechanisms regulating skeletal muscle growth and atrophy

Skeletal muscle mass increases during postnatal development through a process of hypertrophy, i.e. enlargement of individual muscle fibers, and a similar process may be induced in adult skeletal muscle in response to contractile activity, such as strength exercise. and specific hormones, such as androgens and β -adrenergic agonists. Muscle hypertrophy occurs when the overall rates of protein synthesis exceed the rates of protein degradation. Two major signaling pathways control protein synthesis, the IGF1-Akt—mTOR pathway, acting as a positive regulator, and the myostatin—Smad2/3 pathway, acting as a negative regulator, and additional pathways have recently been identified. Proliferation and fusion of satellite cells, leading to an increase in the number of myonuclei, may also contribute to muscle growth during early but not late stages of postnatal development and in some forms of muscle hypertrophy in the adult. Muscle atrophy occurs when protein degradation rates exceed protein synthesis, and may be induced in adult skeletal muscle in a variety of conditions, including starvation, denervation, cancer cachexia. heart failure and aging. Two major protein degradation pathways, the proteasomal and the autophagolysosomal pathways, are activated during muscle atrophy and variably contribute to the loss of muscle mass. These pathways involve a variety

of atrophy—related genes or atrogenes, which are controlled by specific transcription factors, such as FoxO3, which is negatively regulated by Akt, and NF—KB, which is activated by inflammatory cytokines.