



Giorgia Cavioli

● ESPERIENZA LAVORATIVA

01/11/2021 – ATTUALE Roma, Italia

DOTTORATO IN MORFOGENESI E INGEGNERIA TISSUTALE UNIVERSITÀ DI ROMA, LA SAPIENZA

19/01/2021 – 31/10/2021 Roma, Italia

RICERCATRICE TIROCINANTE UNIVERSITÀ LA SAPIENZA

Ricerca sperimentale con tecniche di Biologia Molecolare e Cellulare, *in vivo* ed *in vitro*
Analisi dati tramite Excell.

● ISTRUZIONE E FORMAZIONE

10/10/2018 – 19/01/2021 Roma (RM), Italia

LAUREA MAGISTRALE (LM-6) IN GENETICA E BIOLOGIA MOLECOLARE Università La Sapienza

Indirizzo Roma (RM), Italia

04/10/2015 – 24/10/2018 Roma (RM), Italia

LAUREA TRIENNALE (L13) IN SCIENZE BIOLOGICHE Università La Sapienza

Indirizzo Roma (RM), Italia

10/09/2010 – 09/07/2015 Zagarolo (RM), Italia

DIPLOMA LICEO SCIENTIFICO Liceo Scientifico "Giovanni Falcone e Paolo Borsellino"

Indirizzo Via Colle dei Frati, Zagarolo, Zagarolo (RM), Italia

● COMPETENZE LINGUISTICHE

Lingua madre: **ITALIANO**

Altre lingue:

	COMPRESIONE		ESPRESSIONE ORALE		SCRITTURA
	Ascolto	Lettura	Produzione orale	Interazione orale	
INGLESE	B1	B1	A2	B1	B1

Livelli: A1 e A2: Livello elementare B1 e B2: Livello intermedio C1 e C2: Livello avanzato

● COMPETENZE DIGITALI

Padronanza del Pacchetto Office (Word Excel PowerPoint ecc) | Gestione autonoma della posta e-mail | R/R-Studio

● ULTERIORI INFORMAZIONI

PUBBLICAZIONI

[Thyroid Hormone Protects from Fasting-Induced Skeletal Muscle Atrophy by Promoting Metabolic Adaptation](#)

– 2019

Thyroid hormones regulate a wide range of cellular responses, via non-genomic and genomic actions, depending on cell-specific thyroid hormone transporters, co-repressors, or co-activators. Skeletal muscle has been identified as a direct target of thyroid hormone T3, where it regulates stem cell proliferation and differentiation, as well as myofiber metabolism. However, the effects of T3 in muscle-wasting conditions have not been yet addressed. Being T3 primarily responsible for the regulation of metabolism, we challenged mice with fasting and found that T3 counteracted starvation-induced muscle atrophy. Interestingly, T3 did not prevent the activation of the main catabolic pathways, i.e., the ubiquitin-proteasome or the autophagy-lysosomal systems, nor did it stimulate de novo muscle synthesis in starved muscles. Transcriptome analyses revealed that T3 mainly affected the metabolic processes in starved muscle. Further analyses of myofiber metabolism revealed that T3 prevented the starvation-mediated metabolic shift, thus preserving skeletal muscle mass. Our study elucidated new T3 functions in regulating skeletal muscle homeostasis and metabolism in pathological conditions, opening to new potential therapeutic approaches for the treatment of skeletal muscle atrophy.

[Neurohypophyseal hormones and skeletal muscle: a tale of two faces.](#) – 2020

The neurohypophyseal hormones vasopressin and oxytocin were invested, in recent years, with novel functions upon striated muscle, regulating its differentiation, trophism, and homeostasis. Recent studies highlight that these hormones not only target skeletal muscle but represent novel myokines. We discuss the possibility of exploiting the muscle hypertrophy activity of oxytocin to revert muscle atrophy, including cancer cachexia muscle wasting. Furthermore, the role of oxytocin in cardiac homeostasis and the possible role of cardiac atrophy as a consequence of death in cachectic patients is discussed.

[Cytoplasmic HDAC4 regulates the membrane repair mechanism in Duchenne Muscular Dystrophy.](#) –

2022

Histone deacetylase 4 performs crucial functions in the cytoplasm of dystrophic muscles, by mediating the muscle repair response to damage, an important role in ensuring muscle homeostasis, probably by stabilizing Trim72 mRNA. Consequently, the cytoplasmic functions of HDAC4 should be stimulated rather than inhibited in muscular dystrophy treatments, a fact to be considered in future therapeutic approaches.

[Functional Nutrients to Ameliorate Neurogenic Muscle Atrophy](#) – 2022

Neurogenic muscle atrophy is a debilitating condition that occurs from nerve trauma in association with diseases or during aging, leading to reduced interaction between motoneurons and skeletal fibers. Current therapeutic approaches aiming at preserving muscle mass in a scenario of decreased nervous input include physical activity and employment of drugs that slow down the progression of the condition yet provide no concrete resolution. Nutritional support appears as a precious tool, adding to the success of personalized medicine, and could thus play a relevant part in mitigating neurogenic muscle atrophy. We herein summarize the molecular pathways triggered by denervation of the skeletal muscle that could be affected by functional nutrients. In this narrative review, we examine and discuss studies pertaining to the use of functional ingredients to counteract neurogenic muscle atrophy, focusing on their preventive or curative means of action within the skeletal muscle. We reviewed experimental models of denervation in rodents and in amyotrophic lateral sclerosis, as well as that caused by aging, considering the knowledge generated with use of animal experimental models and, also, from human studies.

CONFERENZE E SEMINARI

24/04/2022 – 29/04/2022 – Online

EMBO Workshop “Muscle formation, maintenance, regeneration and pathology”.

20/10/2022 – 23/10/2022 – Assisi

19th IIM meeting

Autorizzo il trattamento dei miei dati personali presenti nel CV ai sensi dell'art. 13 d. lgs. 30 giugno 2003 n. 196 - "Codice in materia di protezione dei dati personali" e dell'art. 13 GDPR 679/16 - "Regolamento europeo sulla protezione dei dati personali".

Roma , 08/12/2022