**Open Access** 

# Protein Intake and Exercise-Induced Muscle Damage: A Bibliometric and Visual Analysis

#### Wei Li\*

Department of Physical Therapy and Motion Analysis, University of South Alabama, 307 N University Blvd, Mobile, AL 36688 ,USA

#### Introduction

Numerous research have addressed exercise-induced muscle damage (EIMD) issues, from dietary recommendations to recovery techniques, but few have successfully explored and assessed substantial amounts of scientific output. Exercise-induced muscle damage (EIMD) is a phenomena that happens after new or unfamiliar exercise, especially if the exercise contains a lot of eccentric contractions [1]. Normal side effects of the damage process include a temporary reduction in muscle function (including muscle force and range of motion) [2,3] an increase in the swelling of the involved muscle group, an increase in the circulation of muscle-specific proteins, and delayed-onset muscle soreness (DOMS). The majority of EIMD symptoms and signs appear right after the first exercise session and last for up to 14 days. Despite the fact that the underlying causes of its development are still unknown, these variables are frequently employed to measure the severity of muscle injury, with DOMS being the most regularly utilised marker [4,5].

The Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines were used to compile this systematic literature review. The effectiveness of nitrate intake in recovering from fatigue brought on by exercise was studied in experimental and quasi-experimental research, which the authors selected as inclusion criteria. Clear standards for the replication and updating of systematic reviews served as the foundation for the choice to conduct a systematic review related to the goal of the current investigation [6].

#### Discussion

Similar to the lung, salivary gland cells showed evidence of TRPV4 and AQP5 channel cooperation. The apical area of the acinar cells in the mouse submandibular gland is where both channels are located. The analysis of salivary gland cells from AQP5-/- and AQP5+/+ mice showed that the HTS-stimulated Ca2+ entry (for which the TRPV4 channel is presumably involved) was dramatically reduced in cells separated from AQP5-/- animals when they were put in HTS (hypotonic external solution). N and C terminus-truncated AQP5 channels were created in order to more thoroughly examine the connection between AQP5 and TRPV4.

### Conclusion

Patients who have recovered from moderate to severe ARDS-related COVID-19 damage have irreversible functional deficits. In the post-discharge pulmonary rehabilitation, exercise is essential. Despite being secure and

\*Address for Correspondence: Wei Li, Department of Physical Therapy and Motion Analysis, University of South Alabama, 307 N University Blvd, Mobile, AL 36688, USA, E-mail:weil@gmail.com

**Copyright:** © 2022 Li W. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Date of Submission: 14 June 2022, Manuscript No. jsmds-22-77382; Editor Assigned: 16 June 2022, PreQC No. P-77382; Reviewed: 28 June 2022, QC No. Q-77382; Revised: 04 July 2022, Manuscript No. R-77382; Published: 11 July, 2022, DOI: 10.37421/2161-0673.2022.12.263

the standard form of training, CONC exercises provide exercise-limiting cardiovascular stress, dyspnea, and fatigue. Therefore, lowered tolerance and training compliance can significantly reduce prospective advantages. ECC, on the other hand, is a cutting-edge form of training that is often employed by athletes but much less frequently in therapeutic settings. Recent studies show that COPD patients who exercise with ECC as opposed to CONC experience significantly greater gains in functional capacity and muscle mass as well as fewer complaints of fatigue and dyspnea. However, there are few outpatient data following COVID-19.

## **Conflict of Interest**

None.

#### References

- Clarkson, Priscilla M., and Monica J. Hubal. "Exercise-induced muscle damage in humans." Am J Phys Med 81, (2002): S52-S69.
- Clarkson, Priscilla M., Kazunori Nosaka, and Barry Braun. "Muscle function after exercise-induced muscle damage and rapid adaptation." *Med Sci Sports Exerc* 24(1992): 512-520.
- Brown, S., S. Day, and A. Donnelly. "Indirect evidence of human skeletal muscle damage and collagen breakdown after eccentric muscle actions." J Sports Sci 17(1999): 397-402.
- Yu, Ji-Guo, Jing-Xia Liu, Lena Carlsson, Lars-Eric Thornell, and Per S. Stål. "Reevaluation of sarcolemma injury and muscle swelling in human skeletal muscles after eccentric exercise." *PloS one* 8 (2013): e62056.
- McHugh, Malachy P. "Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise." Scand J Med Sci Sports 13 (2003): 88-97.
- Howatson, Glyn, and Ken A. Van Someren. "The prevention and treatment of exercise-induced muscle damage." J Sports Med 38(2008): 483-503.

How to cite this article: Li, Wei. "Protein Intake and Exercise-Induced Muscle Damage: A Bibliometric and Visual Analysis." J Sports Med Doping Stud 12 (2022): 263.