

Compressive Mechanical Force's Effect on Temporomandibular Joint's Cartilage: a Systematic Review

Duarte B^{1a}, Mascarenhas P^{1b}, Costa HN^{1c}, Cavacas MA^{1d}

¹ Egas Moniz University Institute



Dentistry student (final year) EBH and CiiEM Colaborate Member PhD Orthodontic Department PhD Morphology Department and C nt and CiiEM Integrate Mer

INTRODUCTION

Mechanical loading is essential for the maintenance of chondrocyte proliferation and extracellular matrix production of temporomandibular joint's (TMJ) fibrocartilage.¹ Nonetheless, abnormal and excessive loading might disrupt this homeostasis, affecting the functional integrity, and resulting in damage to the fibrocartilage.² Changes in fibrocartilage morphology and cellular content and arrangement can compromise the normal and healthy function of TMJ, resulting in degenerative joint disease (DJD). 1,3

An example with clinical relevance of these diseases is temporomandibular joint osteoarthritis (TMJ OA), a progressive degenerative disease that affects the hard and soft tissues of TMJ. It is characterized by cartilage thinning, extracellular matrix degradation, and decreased number of chondrocytes.^{3,4} Having a high prevalence and a multifactorial etiology, it is important to study the potential causes of it, namely the excessive mechanical loading induced by compressive force. 3,4

OBJECTIVES

This systematic review aims to evaluate the morphological and cellular changes in temporomandibular joint's fibrocartilage resulting from the compressive mechanical force, in vivo.

MATERIALS AND METHODS

A specific PICO question was formulated: "What is the effect of compressive mechanical force in the temporomandibular joint's cartilage?". An electronic database search for articles published in PubMed, SCOPUS, and B-On was conducted until December 2021, using the following search strategy: "temporomandibular joint" AND "cartilage" AND "compressive".

After removing the duplicates, the remaining articles were screened by two independent calibrated reviewers.

Only in vivo experimental studies that provided knowledge about the effect of compressive force in temporomandibular joint's fibrocartilage, published in the last ten years, were included.

Studies regarding treatments for temporomandibular joint pathologies, or sex differences were excluded.

RESULTS					
		Study	Sample (Magnitude and duration of loading)		Main findings
Identification of studies via databases and registers		Magara et al. 2012 ⁵	25 rats 15 EG - 50g 10 CG	5 days	 ↓ thickness Acelular region in mature layer
Identification	Records identified from*: PubMed (n = 28) SCOPUS (n = 42) B-On (n = 45)	Du et al. 2020 ⁶	12 mice 6 EG - 40g 6 CG	7 days	 Appeared thinner ↑ IL-1β and MMP-3
	Records removed before screening: Duplicate records removed (n = 67)	Huang et al. 2021 ⁷	44 rats 22 EG 40g 22 CG	7 days	 thickness and chondrocytes ↑ TNF-a, IL-1β and IL-6
Included Screening	Records screened (n = 48) Records excluded** (n = 39)	Jiang et al. 2017 ⁸	48 rats 2 X 12 EG - 40g 2 X 12 CG	4 days 7 days	 ↓ thickness (43%= 4 days; 56%= 7 days) and chondrocytes (50%= 4 days; 61%= 7 days) ↑ chondrocytes apoptosis in 4 days EG (82%); ↓ TUNEL positive cells were observed in 7 days EG ↓ Collagen II (49%=7 days) and X (84%= 7 days) ↑ TNF-a and IL-1 in both EG
	Reports assessed for eligibility (n = 9) Reports excluded: In vitro (n = 1) Other elements of TMJ which do not include cartilage (n = 1) Studies included in review (n = 7) Fig 1. PRISMA flow diagram for the identification and selection of elegible studies.	Zhang et al. 2021 ⁹	84 rats 2 X 12 EG - 80g 2 X 12 CG	4 days 7 days	 ↓ LOXL2 (p>0.05= 4 days; p<0.05= 7 days) ↓ thickness (190.46±11.32 µm= 4 days;142.83±11.74 µm= 7 days) ↓ proteoglycans (33% =5 days; 77%= 7 days) ↓ collagen II (p>0.05= 4 days; p<0.01= 7 days) ↑ TNF-a (p<0.05= 4 days; p<0.05= 7 days)
		Li et al. 2013 ¹⁰	64 rats 4 X 8 EG - 40g 4 X 8 CG	3 days 7 days 14 days 21 days	 3 days: ↓ cell order and arrangement; ↓ thickness; ↑ chondrocytes apoptosis 7 days: ↓↓ thickness; ↓↓ chondrocytes; ↓↓ chondrocytes apoptosis 14 days: ↓↓↓ thickness (2/3); ↓↓↓ chondrocytes (50%); ↓ ECM amount; ↓ collagen II and X; ↓ chondrocytes apoptosis 21 days: ↑ thickness
		Wen et al. 2016 ¹¹	45 rats 3 X 5 EG1 - 40g 3 X 5 EG2 - 80g 3 X 5 CG	1 day 3 days 7 days	 ↓ thickness (85% =1 day EG1, 70%=3 days EG1; <60%= 7 days EG1)(75%=1 day EG2; 65%=3 days EG2; <50%=7 days EG2) ↓ chondrocytes (gradually in both EG) Apoptosis was induced at day 1 and then dropped quickly in EG1, in EG2 apoptosis was induced gradually with time reaching its highest level at 7 days

Table 1. Characteristics of the included experimental studies (EG= Experimental Group- compressive force; CG= Control Group- no mechanical loading).

DISCUSSION

The experimental in vivo studies revealed that compressive mechanical force induces the degradation of temporomandibular joint's fibrocartilage, leading to pathological changes: reduction in cartilage thickness, lower number of chondrocytes, disorder and disarranged of cell layers, and increased expression of inflammatory factors.

These changes are **co-related with the time** and **magnitude** of loading, revealing that fibrocartilage has a limited physiological tolerance. Also, the effects are compatible with those of osteoarthritis.

CONCLUSIONS

Excessive compressive force is an important etiological factor of TMJ OA, enhancing the effects with the time and magnitude of loading.

CLINICAL RELEVANCE

Understanding how TMJ's fibrocartilage responds to compressive mechanical force might play an important role not only in the treatment of TMJ disease, including osteoarthritis, but also in orthodontic treatments.

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