MUSCLE OS

P 2

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The first chapter of the first domain — Mechanical Tension, Metabolic Stress & Muscle Damage, including the three mechanisms of hypertrophy.

Domain 1: Training Foundations

1. Mechanical Tension, Metabolic Stress & Muscle Damage

1.1 The Three Mechanisms of Hypertrophy

Skeletal muscle hypertrophy is driven by three primary mechanisms that operate synergistically: mechanical tension, metabolic stress, and muscle damage. While each contributes to the hypertrophic response, mechanical tension is universally accepted as the primary driver. Understanding how these mechanisms interact allows the coach to design training programmes that maximise adaptive signalling while managing recovery.

Key Distinction

Mechanical tension is for hypertrophy. Metabolic stress and muscle damage are mechanisms that can augment the response but cannot independently produce substantial hypertrophy in the absence of mechanical tension.

1.2 Mechanical Tension

Mechanical tension refers to the force produced by a muscle during contraction, combined with the passive tension generated through muscle stretch. The total tension stimulus is the product of force production and muscle length at peak tension. This is why exercises that load a muscle in its lengthened position (deep stretch under load) produce superior hypertrophic responses — they maximise both active force and passive tension simultaneously. The mechanotransduction pathway converts this mechanical signal into a biochemical response: integrins, focal adhesion kinase (FAK), and the YAP/TAZ pathway transduce sarcolemmal strain into mTOR activation and subsequent protein synthesis.

1.3 Metabolic Stress

Metabolic stress refers to the accumulation of metabolites — lactate, inorganic phosphate (Pi), hydrogen ions (H+), and reactive oxygen species — during high-repetition, moderate-load, short-rest training. These metabolites contribute to hypertrophy through several pathways: cell swelling (volume expansion of the muscle fibre activates anabolic signalling via the osmosensitive transcription factor TonEBP/NFAT5), hypoxia-induced transcription factors (HIF-1a upregulates VEGF, supporting capillary density and nutrient delivery), and

metabolite-mediated growth factor release (lactate has been shown to stimulate muscle stem cell activity independent of pH changes).

1.4 Muscle Damage

Exercise-induced muscle damage involves Z-disc disruption, sarcomere disorganisation, and the resulting inflammatory response. Satellite cell activation is the primary repair mechanism: quiescent satellite cells are activated by damage signals (HGF release from the extracellular matrix) and proliferate to donate nuclei to existing myofibres, increasing the myonuclear domain and supporting further hypertrophy. The inflammatory response includes neutrophil infiltration, macrophage polarisation (M1 to M2 transition), and release of cytokines (IL-6, IL-10) that coordinate repair and remodelling. While some damage is necessary for adaptation, excessive damage impairs recovery and interferes with subsequent training quality.

1.5 Synergistic Operation

Table 1.1 The Three Hypertrophy Mechanisms

MECHANISM	STIMULUS	PRIMARY VARIABLE	KEY EVIDENCE
Mechanical tension	Force production x muscle stretch	Load, full ROM, maximally stretched position under load	Schoenfeld (2010); Hornberger & Chien (2006)
Metabolic stress	Metabolite accumulation (lactate, H+, Pi)	Rep range (8–15+), rest intervals (30–90 s), continuous tension	Schoenfeld (2013); Burd et al. (2012)
Muscle damage	Z-disc disruption, eccentric loading	Novel exercises, eccentric emphasis, high volume	Clarkson & Hubal (2002); Toigo & Boutellier (2006)

CHAPTER SUMMARY

Mechanical tension is the primary hypertrophy driver, with metabolic stress and muscle damage playing adjuvant roles. Exercises that load the muscle in its lengthened position maximise the tension stimulus. Cell swelling from metabolite accumulation and satellite cell activation from muscle damage contribute to but cannot substitute for mechanical tension. A well-designed programme exploits all three mechanisms through appropriate exercise selection, loading parameters, and rest intervals.