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Review

eIF3f: A central regulator of the antagonism atrophy/hypertrophy in skeletal muscle[☆]



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ABSTRACT

The eukaryotic initiation factor 3 subunit f (eIF3f) is one of the 13 subunits of the translation initiation factor complex eIF3 required for several steps in the initiation of mRNA translation. In skeletal muscle, recent studies have demonstrated that eIF3f plays a central role in skeletal muscle size maintenance. Accordingly, eIF3f overexpression results in hypertrophy through modulation of protein synthesis via the mTORC1 pathway. Importantly, eIF3f was described as a target of the E3 ubiquitin ligase MAFbx/atrogen-1 for proteasome-mediated breakdown under atrophic conditions. The biological importance of the MAFbx/atrogen-1-dependent targeting of eIF3f is highlighted by the finding that expression of an eIF3f mutant insensitive to MAFbx/atrogen-1 polyubiquitination is associated with enhanced protection against starvation-induced muscle atrophy. A better understanding of the precise role of this subunit should lead to the development of new therapeutic approaches to prevent or limit muscle wasting that prevails in numerous physiological and pathological states such as immobilization, aging, denervated conditions, neuromuscular diseases, AIDS, cancer, diabetes.

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1. Introduction

Skeletal muscle mass represents approximately 40–50% of human body weight, making it the largest tissue mass and the

major protein reservoir in the body. Maintenance of muscle mass is dependent on the balance between synthesis and breakdown of myofibrillar proteins (Attaix et al., 2012). Signal transduction pathways, which promote synthesis and/or degradation of muscle proteins, mediate the regulation of muscle homeostasis as well as muscle hypertrophy or atrophy. Hypertrophy is associated with growth in muscle mass, as a result of an increase in the size of pre-existing skeletal muscle fibers accompanied by incremented protein synthesis without apparent variation in the number of myofibers. In contrast, atrophy is characterized by an alteration in protein synthesis and an increased degradation of muscle proteins

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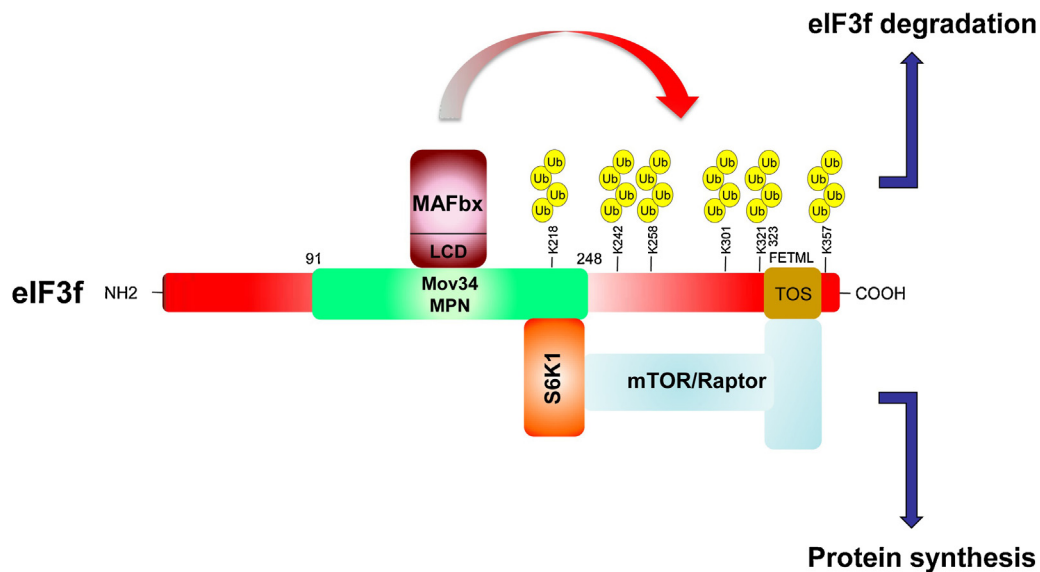


Fig. 1. Physical and functional links between eIF3f, mTOR-raptor, S6K1 and MAFbx/atrogenin-1 in skeletal muscle. The leucine charged domain (LCD) of the ubiquitin ligase MAFbx/atrogenin-1 physically interacts with the Mov34 domain of eIF3f and contributes to its ubiquitination in the C-terminal region and its subsequent degradation by the proteasome during muscle wasting. The Mov34 domain also interacts with the inactive hypophosphorylated form of S6K1. Under nutrient rich conditions, mTOR/raptor complex binds to the TOS motif of eIF3f and, thus can phosphorylate and activate S6K1. Active S6K1 is released from eIF3f complex leading to increased protein synthesis. In this model, eIF3f acts as a scaffolding protein allowing mTORC1-dependent activation of S6K1 upon insulin or growth hormone stimulation of muscle cells.

(Goldspink et al., 1986). Muscle atrophy that results in a state of weakness and emaciation of the body is the terminal phase of certain diseases or chronic infections such as cancer, AIDS, diabetes, bacterial infections and nerve degeneration. Muscle atrophy is also observed during aging, immobilization and trauma to the muscle.

The ubiquitin-proteasome system has been particularly involved in muscle atrophy after the discovery of the E3 ubiquitin ligases MAFbx/atrogenin-1 (muscle atrophy F-box protein) and MuRF-1 (muscle RING finger-1), which are both overexpressed in various models of atrophy (fasting, cancer, diabetes, immobilization) (Bodine et al., 2001; Gomes et al., 2001). The knockout of genes coding for these factors confers resistance to certain types of atrophy, suggesting a major role of these E3 ligases in mediating muscle atrophy (Bodine et al., 2001). The function of E3 ubiquitin ligases is to ubiquitinate specific proteins to target them for recognition and degradation by the proteasome. MAFbx/atrogenin-1 is responsible for the degradation of the transcription factor MyoD and the eukaryotic initiation factor of translation eIF3f. Therefore, MAFbx/atrogenin-1 promotes muscle atrophy by inhibiting the transcription and translation of muscle genes, and prevents the replacement of degraded proteins (Lagrand-Cantaloube et al., 2009, 2008; Tintignac et al., 2005). In this review, we summarize the current state of knowledge concerning the central role of eIF3f in the control of skeletal muscle mass, particularly during muscle wasting.

2. Structure of eIF3f and its function in non-muscle cells

Mammalian eukaryotic initiation factor 3f (eIF3f, p-47) is one of the 13 subunits (designated eIF3a to eIF3m) of the eukaryotic initiation factor eIF3, a multi-protein complex required for the initiation of protein synthesis. eIF3 (800 kDa) is the largest and one of the most complex initiation factors known (Asano et al., 1997; Benne and Hershey, 1976). It is a multifunctional initiation factor that plays a major role in translation by operating at different levels of the initiation pathway; including the assembly of the ternary complex eIF2-GTP-Met-tRNA, binding of this complex and other

components of the 43S PIC (preinitiation complex) to the 40S ribosomal subunit, mRNA recruitment to the 43S PIC, and scanning mRNA for AUG recognition. The eIF3f subunit is a member of the Mov34 family and contains a conserved (≈ 140 -aa) domain named MPN (Mpr1/Pad1/N-terminal) (Fig. 1). This motif is found in two macromolecular complexes, homologous to eIF3, the COP9 signalosome, and the lid of 19S proteasome (Hofmann and Bucher, 1998). Moreover, both eIF3f and eIF3h interact directly with each other, supporting a possible scaffolding role for these subunits (Zhou et al., 2008).

The function of eIF3f within the eIF3 complex is not totally characterized. However, eIF3f is essential for *Schizosaccharomyces pombe* (*S. pombe*) viability, and its depletion markedly decreases the global protein synthesis in fission yeast (Zhou et al., 2005). The same effect on translation was described in cells infected with the severe acute respiratory syndrome coronaviruses and coronavirus infectious bronchitis virus (Xiao et al., 2008). Interestingly, eIF3f overexpression has been also associated with inhibition of HIV-1 replication (Valente et al., 2009). Furthermore, studies by Doldan et al. (2008a,b) showed that eIF3f is downregulated in several human tumors. The same group found that eIF3f overexpression in cancer cells negatively regulates cell growth by affecting the translation efficiency and the activation of apoptosis (Shi et al., 2006). Moreover, caspase-processed isoform of the cell division kinase 11 (Cdk11^{P46}) binds and phosphorylates eIF3f at Ser-46 and Thr-119 during apoptosis in A375 melanoma cells, leading to an inhibition of translation (Shi et al., 2003). In a recent study, Wen et al. (2012) reported that eIF3f exhibited a tumor suppressive function in pancreatic cancer. Mechanistically, the authors found that eIF3f inhibits both cap-dependent and cap-independent translation and that eIF3f promotes rRNA degradation through direct interaction with heterogeneous nuclear ribonucleoprotein K (Wen et al., 2012). From these data, it appears that eIF3f can act in different ways according to cellular contexts. Thus, this protein is essential for *S. pombe* viability but in cancer cells, it is a negative regulator of cell growth and protein translation, and it plays a central role in apoptotic signaling (Marchione et al., 2013).

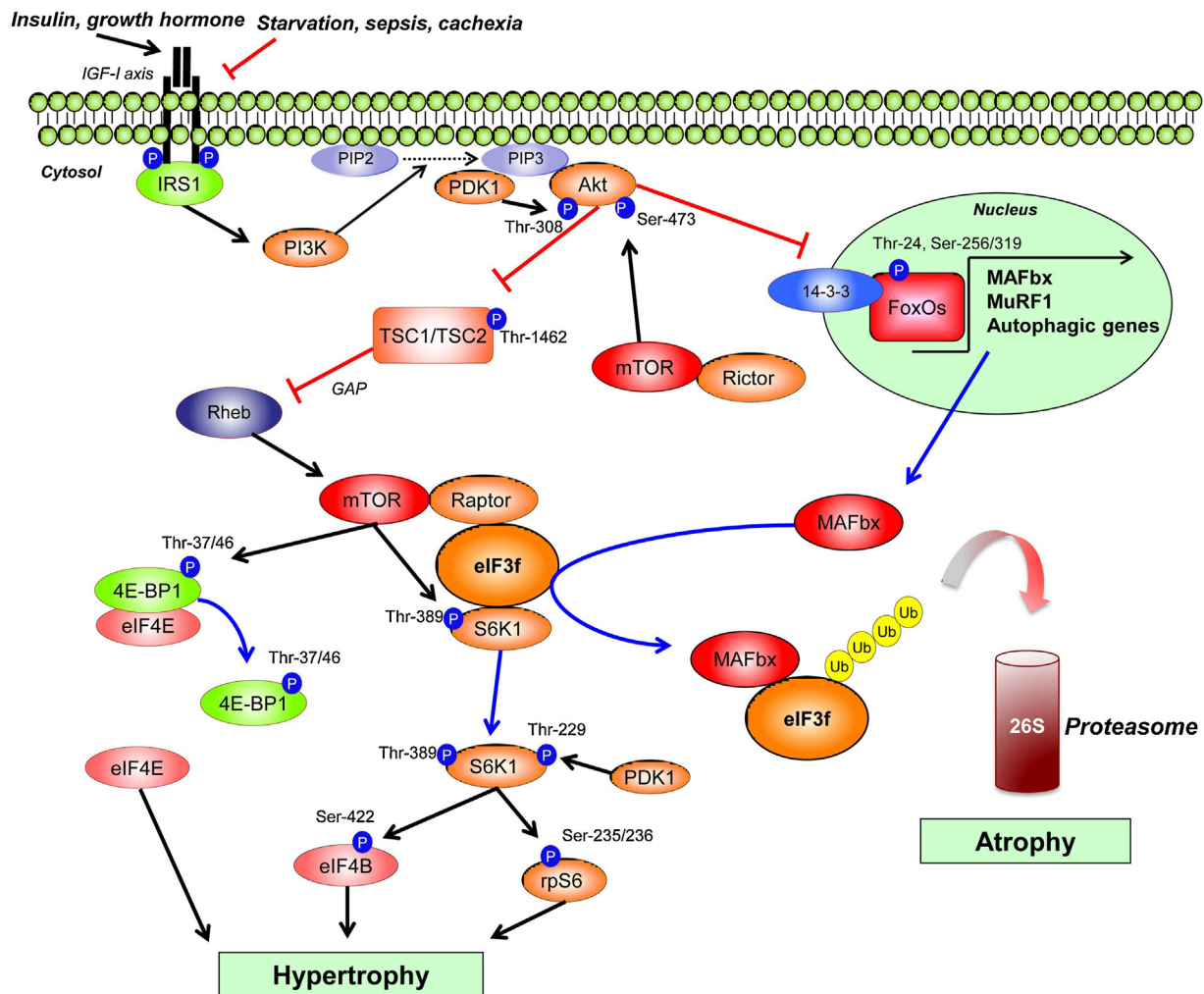


Fig. 2. eIF3f is at the crossroads between the protein synthesis and protein degradation signaling pathways in skeletal muscle. Upon IGF-1 axis activation, the IGF-1 receptor becomes phosphorylated and recruits IRS1, leading to the activation of the lipid kinase PI3K. PI3K catalyzes the transfer of a phosphate group to the membrane-bound PIP2. Phosphorylation of PIP2 generates PIP3, which recruits two additional kinases, Akt and PDK1. Akt is bound and activated by phosphorylation at Thr-308 by PDK1. Akt inactivates TSC1/TSC2 complex, a Rheb inhibitor, by phosphorylation. Finally, mTOR activated by Rheb, binds to the scaffold protein eIF3f and exerts its activity on protein synthesis by phosphorylation of the S6K1 protein. mTOR in association with rictor phosphorylates and fully activates Akt on Ser-473. While FoxOs transcription factors are directly phosphorylated and inhibited by Akt via 14-3-3 binding in response to insulin/growth factor stimulation, under condition of energy stress, dephosphorylation of FoxO factors leads to its nuclear entry and E3 ubiquitin ligase MAFbx/atrogen-1 expression. Proteasomal degradation of eIF3f mediated by the overexpressed MAFbx/atrogen-1 under starvation and diseases is leading to decreased protein synthesis and muscle wasting. Abbreviations not defined in the text: IGF-1: insulin like growth factor 1; IRS1: insulin receptor substrate 1; PDK1: phosphoinositide-dependent protein kinase-1; PI3K: phosphatidylinositol 3-kinase; PIP2: phosphatidylinositol 4,5-bisphosphate; PIP3: phosphatidylinositol 3,4,5-triphosphate; Rheb: ras homologous enriched in brain; TSC1/2: tuberous sclerosis complex1/2.

3. eIF3f regulates protein synthesis and skeletal muscle mass

3.1. Anabolic process and eIF3f

Increased protein synthesis, mainly associated with the activation of the canonical IGF-1/Akt/mTORC1 pathway leads to an increase of the diameter of muscle fibers (Fig. 2). The mammalian target of rapamycin complex 1 (mTORC1) modulates protein synthesis through regulation of its downstream effectors, the eukaryotic translation initiation factor 4E-binding protein 1 (4E-BP1) and the ribosomal protein S6 kinase 1 (S6K1) (Fig. 2). Mechanistically, phosphorylation of 4E-BP1 at Thr-37/46 by the mammalian target of rapamycin (mTOR) induces its dissociation from the eukaryotic translation initiation factor 4E (eIF4E) providing the assembly of the preinitiation complex (Youtani et al., 2000). In addition, after an initial phosphorylation by mTOR at Thr-389, S6K1 phosphorylates substrates involved in the regulation of protein translation, such as the ribosomal protein S6 and

the eukaryotic translation initiation factor 4B (Holz and Blenis, 2005; Saitoh et al., 2002). Moreover, S6K1 can interact with the phosphoinositide-dependent kinase-1 (PDK1) that induces a second round of phosphorylation at Thr-229 for full activation of S6K1 (Pullen et al., 1998) (Fig. 2).

The molecular basis of the connection between mTORC1 and eIF3f has been investigated during terminal differentiation (Csibi et al., 2010). Thus, several studies demonstrated that eIF3f plays a fundamental role in skeletal muscle mass regulation through the control of mTORC1 signaling pathway (Csibi et al., 2010; Goodman et al., 2011). A conserved TOS (TOR signaling) motif in eIF3f (FETML, amino acids 323–327) is highly conserved among species (Schalm and Blenis, 2002). This motif links mTOR/raptor complex to eIF3f, and allows mTOR to regulate downstream effectors and cap-dependent translation initiation (Fig. 1). In this model, eIF3f acts as a scaffold that interconnects mTOR/raptor with S6K1 as it was previously found in HEK293 cells (Holz et al., 2005). Our group proposed that S6K1 and mTOR physically interact with two different domains of eIF3f in muscle cells. The Mov34 motif, involved in

MAFbx/atrogin-1-mediated polyubiquitination of eIF3f is responsible for the interaction with the inactive hypophosphorylated form of S6K1 (Fig. 1). Inactive S6K1 is bound to eIF3f, and upon amino acid/growth factor/mitogen stimulation, mTOR binds to eIF3f, leading to the phosphorylation and activation of S6K1 (Csibi et al., 2010; Holz and Blenis, 2005). Active phosphorylated S6K1 is then released from eIF3f complex and is able to phosphorylate its substrates. It is important to note that eIF3f induces a specific and robust recruitment of endogenous eIF4G, raptor and rpS6 to the m7-GTP cap complex, suggesting that this protein is not only an activator of the cap-dependent translation but also a major actor of the proper assembly of the translation initiation complex in myotubes (Csibi et al., 2010).

3.2. Catabolic process and eIF3f

Conversely to hypertrophy, atrophy is linked to a decrease in muscle fiber size as a result of reduced protein synthesis and increased protein breakdown. The forkhead box O (FoxO) transcription factors related pathway plays a major role in the control of protein breakdown during muscle atrophy through the regulation of the ubiquitin proteasome and the autophagic systems (Mammucari et al., 2007; Sandri et al., 2004). MAFbx/atrogin-1 interacts with the Mov34 domain of eIF3f and promotes its ubiquitination resulting in proteasome-mediated degradation of eIF3f during muscle wasting (Lagirand-Cantaloube et al., 2008) (Fig. 1). The degradation of eIF3f suppresses S6K1 activation by mTOR in a MAFbx/atrogin-1-induced atrophy model (Csibi et al., 2010). Furthermore, berberine, an inhibitor of mitochondrial respiration, induces atrophy through MAFbx/atrogin-1-mediated degradation of eIF3f in a mice model of type 2 diabetes (Wang et al., 2010). The authors found a decrease in eIF3f expression, mTORC1 signaling activity and protein synthesis, as well as an increase in protein degradation after berberine treatment. Importantly, the knockdown of MAFbx/atrogin-1 attenuated these berberine-induced effects on skeletal muscle, particularly on alterations in eIF3f expression and mTORC1 activity (Wang et al., 2010). In accordance with these observations, it was described that eIF3f expression decreases during various atrophic conditions (i.e. glucocorticoid treatment, starvation and oxidative stress-induced atrophy) and knockdown of MAFbx/atrogin-1 or inhibition of the proteasome system prevents this decrease (Lagirand-Cantaloube et al., 2008). Genetic activation of eIF3f induces hypertrophy associated with an increase in phosphorylation of mTORC1 substrates S6K1 and 4E-BP1, and limits atrophy by increasing expression of muscle structural proteins during starvation (Csibi et al., 2010). In contrast, genetic blockade of eIF3f induces myotubes atrophy associated with a decrease in the phosphorylation of mTORC1 substrates (Csibi et al., 2010). Interestingly, six highly conserved lysine residues on the C-terminal of eIF3f have been found to be necessary for MAFbx/atrogin-1 mediated polyubiquitination (Fig. 1). The mutation of these residues (eIF3f K5-10R) confers increased stability to eIF3f (Csibi et al., 2009) and this mutant is able to attenuate starvation-mediated atrophy by maintaining persistent phosphorylation of S6K1 and rpS6 (Csibi et al., 2010, 2009). Moreover, overexpression of eIF3f K5-10R mutant induces a significant increase in protein synthesis and hypertrophy compared to the wild type protein under normal condition (Csibi et al., 2010, 2009).

4. Conclusion

The eukaryotic initiation factor eIF3f has become a key player in the regulation of skeletal muscle mass. Indeed, eIF3f acts as a scaffold that interconnects mTORC1 with its downstream effectors in order to initiate mRNA translation and increase protein

synthesis. Under basal conditions, overexpression of eIF3f in differentiated muscle cells is sufficient to induce hypertrophy via increased activity of mTORC1. In addition, overexpression of eIF3f K5-10R mutant resistant to MAFbx/atrogin-1-dependent degradation is not only associated with protection against muscle atrophy but also with induction of hypertrophy. In sum, the role of eIF3f in muscle as a central element of both atrophy and hypertrophy pathways makes it an attractive new target for therapeutic intervention. A better knowledge of the function and regulation of eIF3f associated to structural information about MAFbx/eIF3f/mTORC1/S6K1 interactions are now required to develop specific and efficient compounds to prevent and/or slow down skeletal muscle atrophy. In this regard, future research using eIF3f transgenic mice will likely uncover new physiological roles of this initiation factor in the maintenance of skeletal muscle homeostasis.

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