Journal of Nutrition & Intermediary Metabolism 17 (2019) 100099

Contents lists available at ScienceDirect

Journal of Nutrition & Intermediary Metabolism

journal homepage: www.elsevier.com/locate/jnim

Effects of day-time feeding on murine skeletal muscle growth and synthesis

Shinya Aoyama[a,b,1,2], Shuichi Kojima[a,2], Keisuke Sasaki[a], Takeru Shimoda[a], Kengo Takahashi[a], Rina Hirooka[a], Yu Tahara[a], Shigenobu Shibata[a,*]

[a] Graduate School of Advanced Science and Engineering, Waseda University, Tokyo, 1628480, Japan
[b] Organization for University Research Initiatives, Waseda University, Tokyo, 1628480, Japan

HIGHLIGHTS

- Day-time feeding attenuated overloading-induced muscle hypertrophy.
- It also attenuated overloading-induced protein synthesis.
- Feeding time did not affect the muscle weight under normal and atrophic conditions.

ARTICLE INFO

Keywords:
Feeding time
Muscle hypertrophy
Protein synthesis
Time-restricted feeding

ABSTRACT

Muscle mass is controlled by the balance between muscle synthesis and degradation. Although nutrition is important for the maintenance of muscle mass and growth, the effects of feeding time have remained unclear. In the present study, we aimed to evaluate the effects of day- or night-time-restricted feeding on the muscle volume using muscle atrophy and hypertrophy mouse models. The day- and night-time-restricted feeding was conducted from zeitgeber time 2 (ZT2) to ZT10 and ZT14 to ZT22, respectively. In the unilateral immobilization-induced atrophy model, the decrease in immobilized muscle weight did not significantly change with the feeding time. However, the contralateral non-immobilized muscle weight was lower in the mice fed at day time (inactive phase) than in those fed at night time (active phase). In the overloading-induced hypertrophy model, muscle hypertrophy and protein synthesis were attenuated by day-time feeding. These results suggest that day-time feeding attenuated muscle growth via the inhibition of muscle synthesis. Feeding at an irregular time such as a late-night meal could be detrimental for muscle growth.

1. Introduction

The skeletal muscle mass is controlled by the balance between muscle synthesis and degradation. It is considered that the activation of muscle synthesis or degradation is induced by muscle hypertrophy and atrophy, respectively [1]. Dietary amino acids activate the synthesis of muscle protein via the mammalian target of rapamycin (mTOR) pathway [2]. In addition, co-ingestion of carbohydrates and proteins also activates the synthesis of muscle protein [3]. Further, the optimal nutritional intake has a key role in the maintenance and increase of muscle mass.

The late meal time and/or breakfast skipping lead to some health problems such as obesity and diabetes [4,5]. In contrast, time-restricted eating without calorie restriction reduces the risk of metabolic dysfunctions, indicating that nutritional intake at the optimal time is important to maintain healthy metabolic function [6–8]. Studies using mouse models have showed that time-restricted feeding in the resting phase attenuates the repair of DNA in the skin and the function of immune system [9,10]. However, there are only a few studies focused on the effects of feeding time on the skeletal muscle functions.

Several muscle functions such as muscle force and myogenesis exhibit day–night oscillation [11–13]. In our recent study, we demonstrated that *Atrogin1*, which is one of the key muscle catabolic factors, shows day–night oscillation under atrophic condition [14]. In addition,

Abbreviations: BW, body weight; DRF, day-time restricted feeding; GAS, gastrocnemius muscle; Immo, immobilization; mTOR, mammalian target of rapamycin; NRF, night-time restricted feeding; OL, overloading; PLA, plantaris muscle; SE, standard error; SUnSET, surface sensing of translation

* Corresponding author.
E-mail address: shibatas@waseda.jp (S. Shibata).
[1] Present address: Graduate school of Biomedical Science, Nagasaki University, Nagasaki.
[2] These authors contributed equally to this study.

https://doi.org/10.1016/j.jnim.2019.100099
Received 26 March 2019; Received in revised form 27 July 2019; Accepted 18 August 2019
Available online 19 August 2019

another study has reported that the muscle protein synthesis, assessed by the activation of mTOR signaling, exhibits day–night oscillation [15]. Considering that nutrition affects the muscle growth and/or degradation system(s) as described above, the occurrence of time-dependent response to nutrition is expected, based on the results of studies on day–night oscillation of muscle-volume-related factors. In this study, we aimed to evaluate the effects of the night- or day-time feeding on the skeletal muscle mass and muscle protein synthesis.

2. Materials and methods

2.1. Animals

Six- and seven-week-old male Kwl:ICR mice were purchased from Tokyo Laboratory Animals Sciences (Tokyo, Japan). The mice were placed in a room maintained at 22 ± 2 °C and 60 ± 5% humidity, with a 12-h light (08:00–20:00 h)–dark cycle. Zeitgeber time 0 (ZT0) was designated as the "lights on" time and ZT12 as the "lights off" time. The mice were provided a standard diet (EF; Oriental Yeast, Tokyo, Japan) and water *ad libitum* during the acclimation period. After randomized grouping, the experiments were conducted in a nonblinded manner. Body weight (BW) were measured every week. This study was approved by the Committee for Animal Experimentation at the Waseda University (2017-A065), and the mice were treated in accordance with the committee's guidelines.

2.2. Time-restricted feeding

The mice were maintained under day-time- (DRF: ZT2 to ZT10) or night-time (NRF: ZT14 to ZT22)-restricted feeding schedule. Time-restricted feeding was performed using an automated time-restricted feeding device (Natsume Seisakusho, Tokyo, Japan) as reported previously [14].

2.3. Hindlimb immobilization

Unilateral hindlimb immobilization was performed as described previously [16]. Briefly, the mice were anesthetized with isoflurane, and then the leg on one side was immobilized. The contralateral side leg was not treated and used as the control. The immobilized leg was fixed with the knee in an extended position and the ankle in a plantar-flexed position. The hindlimb immobilization was conducted at approximately ZT6 on day 7 after time-restricted feeding.

2.4. Synergist ablation

The mice were anesthetized with isoflurane. Thereafter, functional overload of the plantaris muscle was induced by unilateral surgical ablation of the distal tendons of the gastrocnemius and soleus muscles as described previously [17,18]. The incision in the contralateral leg was closed without surgical ablation of the distal tendons, and its plantaris muscle served as the sham muscle. This surgery was conducted at approximately ZT6 on day 7 after time-restricted feeding.

2.5. Recording of locomotor activity

Locomotor activity of mice was monitored with an area sensor (F5B, Omron, Tokyo, Japan) and analyzed with ClockLab software (Actimetrics, Wilmette, IL, USA), as previously described [14]. Locomotor activity was continuously monitored during the experimental period.

2.6. Measurement of protein synthesis and western blotting

For the assessment of protein synthesis using the surface sensing of translation (SUnSET) method, puromycin (40 μmol/kg BW) was intraperitoneally injected exactly 30 min before sampling of the hindlimb muscle. Immediately after sampling, the muscle samples were frozen in liquid nitrogen and stored at −80 °C until analysis [19,20]. The frozen muscle samples were powdered using a frozen cell crusher (Cryo-Press; MICROTEC, Tokyo, Japan) and homogenized using TissueLyser II (Qiagen, Frederick, MD, USA) with homogenizing buffer (40 mM Tris, pH 7.5; 1 mM EDTA; 5 mM EGTA; 0.5% Triton X-100) in the presence of Protease Inhibitor Cocktail (Roche Diagnostics, Indianapolis, IN, USA) and Phosphatase Inhibitor Cocktail (Nacalai Tesque, Kyoto, Japan). After homogenization, the samples were rotated for 1 h at 4 °C, and then centrifuged at 14,000 g for 30 min at 4 °C. The concentration of proteins was measured using the BCA Protein Assay Kit (Thermo Fisher Scientific, Waltham, MA). Western blotting analysis was conducted as described previously [21]. Sodium dodecyl sulfate polyacrylamide gel electrophoresis was performed with 10 or 50 μg of protein sample, and the proteins in the gel were then transferred onto polyvinylidene difluoride membranes (GE Healthcare, Buckinghamshire, UK). The membranes were then incubated overnight at 4 °C with anti-Puromycin (1:25 000 dilution; clone 12D10; Merck Millipore, Burlington, MA), anti-p-S6 (1:1000 dilution; P–S6 Ribosomal Protein (S240/244) Rabbit Ab #2215; Cell Signaling Technology, Danvers, MA) or anti-S6 (1:1000 dilution; S6 Ribosomal Protein (5G10) Rabbit mAb #2217; Cell Signaling Technology, Danvers, MA). After wash and incubation with HRP-conjugated anti-rabbit or mouse IgG (Cell Signaling Technology, Danvers, MA), the bands of immunoreactive proteins were detected using an enhanced chemiluminescence kit (GE Healthcare, Buckinghamshire, UK) and quantified using the LAS-3000 system (GE healthcare, Buckinghamshire, UK) and ImageJ. After the detection of bands, membranes were stained by Coomassie Brilliant Blue (CBB).

2.7. Statistical analyses

The data are presented as mean ± standard error (SE). GraphPad Prism version 7 (GraphPad Software, San Diego, CA) was used for the statistical analyses. Differences with $P < 0.05$ were considered statistically significant. To test whether the data showed equal or biased variation, we used an F-test or Brown–Forsythe's test. Statistical significance was determined by the two-way analysis of variance with Sidak test (if an interaction and/or the main effect were significant) for *post-hoc* analysis.

3. Results

Under normal condition, which is no induction of muscle atrophy or hypertrophy in both legs, the BW, gastrocnemius muscle weight, and its relative weight to BW did not differ between the NRF and DRF groups (Supplementary Fig. 1).

The BW of mice was not altered in the NRF and DRF groups (Fig. 1A). On day 7 after unilateral immobilization, there was a decrease in the gastrocnemius muscle weight in the NRF and DRF groups (Fig. 1B and C). Although the immobilized muscle did not significantly differ between the NRF and DRF groups, the contralateral non-immobilized muscle weight was higher in the NRF group than in the DRF group. The daily locomotor activity level during experimental period was not changed in the NRF and DRF groups, although the circadian pattern of locomotor activity was different in these groups (Supplementary Fig. 2). Further, we evaluated the muscle protein synthesis at three time points on day 3 and 4 after unilateral immobilization. In the sham muscle (the contralateral non-immobilized muscle), the muscle protein synthesis in the NRF group tended to be higher than that in the DRF group at ZT6 but not at ZT18. In the immobilized muscles, muscle protein synthesis did not differ between the NRF and DRF groups (Fig. 1D and E). The phosphorylated S6 protein level of the sham and immobilized muscles in NRF group tended to be higher or significantly higher than that in the DRF group at ZT18 (Fig. 1G). The total S6 protein level was not changed in the sham muscle. In contrast, it tended

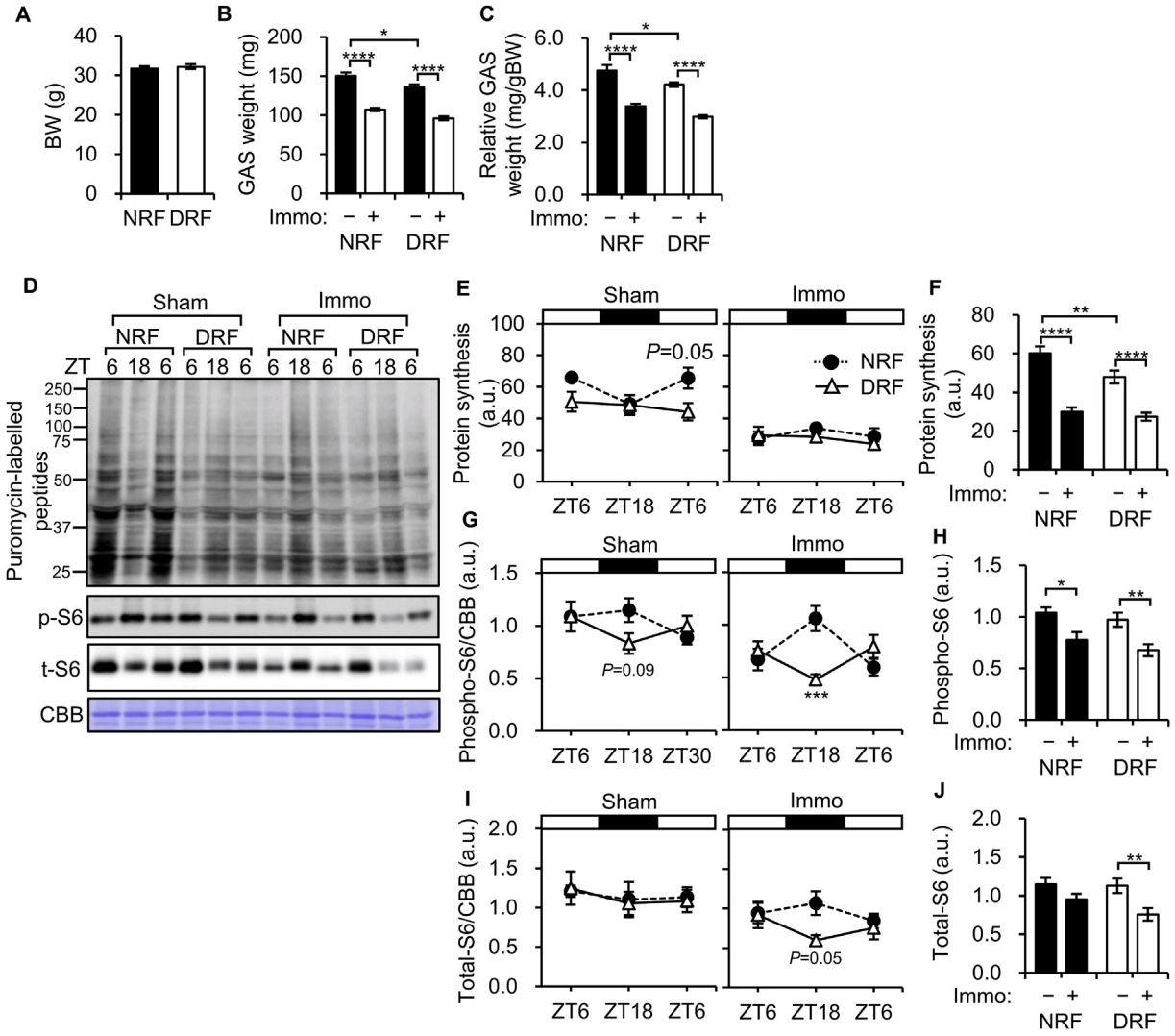

Fig. 1. Effects of day-time feeding on the skeletal muscle mass and protein synthesis in unilateral hindlimb immobilized mice. (A) Body weight (BW), (B) gastrocnemius muscle (GAS) weight, and (C) relative GAS weight to BW in mice maintained under the night- or day-time restricted feeding (NRF or DRF) on day 7 after unilateral hindlimb immobilization (Immo) (A–C: n = 8; D: n = 5). (D) representative blot of puromycin-labeled peptides, phosphorylated (p-)S6, total (t-)S6 and Coomassie Brilliant Blue (CBB) stain and (E, G, I) the day–night variation in protein synthesis, p-S6 and t-S6 in the GAS muscle of NRF or DRF mice on day 3 after unilateral hindlimb immobilization (n = 4–5/time point). (F, H, J) The average value of protein synthesis, p-S6 and t-S6 at three time points (n = 14–15). Mean ± SE. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$, ****$P < 0.0001$, Sidak. . (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

to be lower in the immobilized muscle of DRF group at ZT18 compared with that of NRF group at the same time point (Fig. 1I). The average level of muscle protein synthesis was significantly higher in the sham muscle of the NRF group than that of the DRF group although the effect of feeding time on the muscle protein synthesis was not observed in the immobilized muscles (Fig. 1F). The average level of phosphorylated and total S6 protein was not altered between the NRF and DRF groups, although it was decreased by immobilization (Fig. 1H, J).

We evaluated the effects of feeding time on skeletal muscle hypertrophy using the unilateral hindlimb overloaded model. The BW did not differ between the NRF and DRF groups. Unilateral hindlimb overloading increased the plantaris muscle weight and its relative weight in both the groups (Fig. 2B and C). The increase in muscle weight was significantly attenuated by DRF (Fig. 2C). The result of daily locomotor activity level was not changed in the NRF and DRF groups (Supplementary Fig. 3). This result was similar to the second experiment using immobilized model.

Muscle protein synthesis was assessed at ZT6 because muscle protein synthesis in the NRF mice in the preliminary experiment was relatively higher at ZT6. The muscle protein synthesis was significantly increased on day 3 after overloading in the NRF group but not in the DRF group (Fig. 2D and E). However, on day 7 after overloading, the muscle protein synthesis was increased in the overloaded muscles of both groups, and there was no significant difference between the groups. Similar response was observed in the level of phosphorylated S6 protein, but not total S6 protein (Fig. 2F and G). The phosphorylation of S6 was increased by overloading, with the increase being less in the DRF group than in the NRF group.

4. Discussion

In this study, we examined the effects of feeding time on the muscle mass using the muscle atrophy and hypertrophy models. The intact muscle mass did not change with feeding time (Supplementary Fig. 1), suggesting that feeding time does not affect the muscle mass under normal conditions. In addition, the immobilized muscle weight did not change unexpectedly with the feeding time. On the contrary, the non-immobilized muscle weight was decreased by DRF. Considering that the

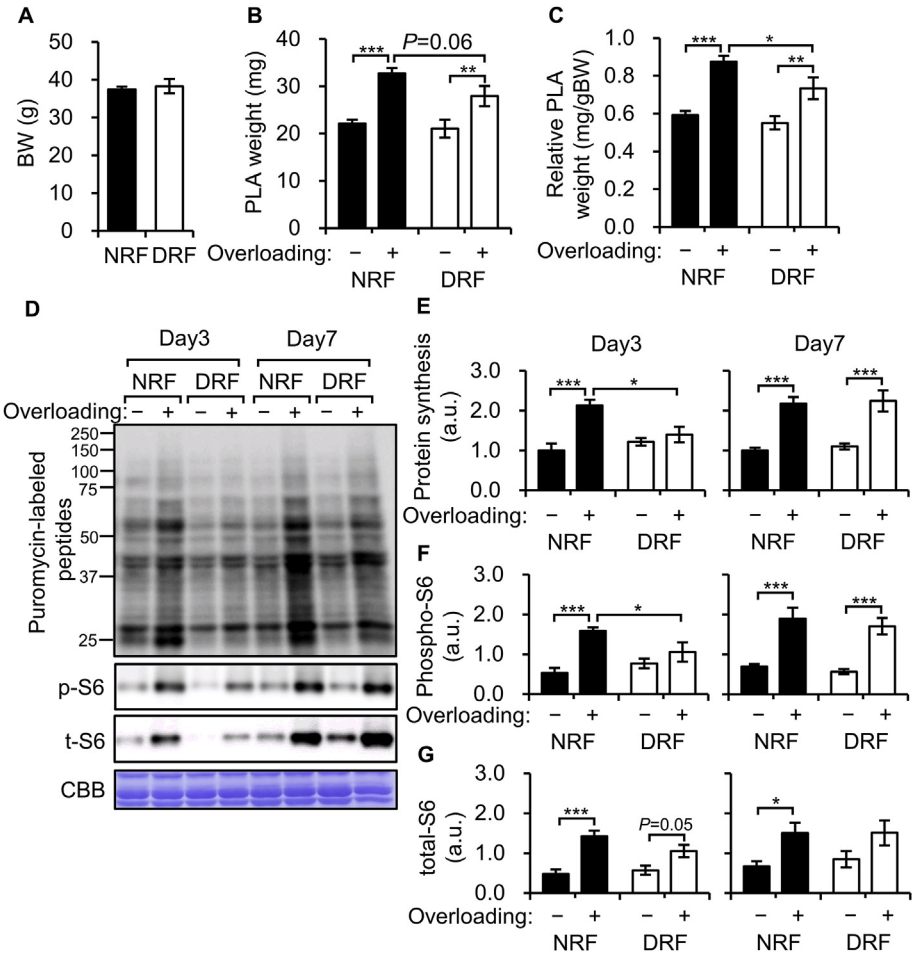

Fig. 2. Effects of day-time feeding on skeletal muscle hypertrophy and protein synthesis in unilateral hindlimb overloaded mice. (A) Body weight (BW), (B) plantaris muscle (PLA) weight, and (C) relative PLA muscle weight to BW in mice maintained under the night- or day-time restricted feeding (NRF or DRF) on day 7 after unilateral overloading (n = 5). (D) Representative blot of puromycin-labeled peptides, phosphorylated (p-)S6, total (t-)S6 and Coomassie Brilliant Blue (CBB) stain and (E, F, G) the protein synthesis, p-S6 and t-S6 at ZT6 in the plantaris muscle of NRF or DRF mice on days 3 and 7 after unilateral hindlimb overloading (OL) (n = 4–5). Mean ± SE. $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$, Sidak. . (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

daily locomotor activity level was not changed in the NRF and DRF groups, the involvement of daily locomotor activity could be small in the lower muscle mass of the DRF-fed mice. From these results we speculate one possibility. Exercise-like-stimulated muscles such as overloaded muscles are sensitive to the effects of feeding time. In addition to the effect of feeding time on non-immobilized muscle, the protein synthesis and muscle mass were higher in the overloaded muscles of the NRF-fed mice than that of DRF-fed mice. It is considered that the intake of nutrition, such as protein and amino acids, activates exercise-induced protein synthesis, while the effect of nutrition declines under sedentary condition [3]. It suggests that mealtime affects the skeletal muscle weight under exercise-like-stimulated condition. In the second experiment of the present study, in which the effects of feeding time on immobilized muscles were investigated, although we did not treat the non-immobilized muscles, the muscle weight and protein synthesis of non-immobilized muscle in NRF-fed mice were higher than that in DRF-fed mice. Because mice must use only non-immobilized leg without immobilized leg for locomotor activity, the non-immobilized muscles may receive an overloading-like stimulation. Therefore, it is suggested that the effects of feeding time on the muscle mass and protein synthesis are sensitive to a muscle condition such as overloading.

On day 3 after overloading, the overloading-induced protein synthesis and phosphorylation of S6 ribosomal protein were attenuated by DRF, while these attenuations were not observed on day 7 after overloading. It has been reported that the activation of mTOR in the early phase after overloading affects overloading-induced skeletal muscle hypertrophy [22]. It is suggested that DRF inhibits the protein synthesis by less activation of mTOR pathway in the early phase after overloading, and its inhibition relates to the attenuation of overloading-induced muscle hypertrophy.

It is well-known that the activation of the mTOR and insulin pathways via amino acids and carbohydrates is involved in the nutrition-induced muscle synthesis [23,24]. The activation of the mTOR and insulin pathways showed day–night variation in several tissues, and their activation was relatively higher in the active phase [15,25–29]. Considering that these pathways are activated by feeding, it is possible that the feeding cycle drives their day–night variations. In fact, it has been reported that fasting reduces the day–night activation of these pathways [15]. On the contrary, the day–night activation of protein translation was not observed in muscle-specific *Bmal1* knock-out mice, which showed normal feeding cycle and locomotor activity [29]. Therefore, it is thought that the day–night activation of the mTOR and insulin pathways is driven by multi-factors including the feeding cycle and muscle clock genes. In the present study, western blotting analysis at three time points with 12-h intervals revealed that NRF induces daily changes in the protein synthesis, while such variations were not observed in DRF mice. Wang et al. reported that day-time feeding attenuated UV-induced DNA replication in the murine skin and showed the possibility that the mismatch of timing between feeding-cycle-induced oxidative capacity and circadian-clock-induced DNA replication was the reason for the attenuation of DNA replication according to day-time feeding [10]. In our study, the decrease in muscle protein synthesis according to DRF could be due to the mismatch of timing between circadian-clock-driven protein synthesis related factors and feeding time. Their day-night variations could not be assessed with sufficient accuracy because we studied only three time points with 12-h intervals. It would be necessary to assess their day-night variations at more time points with short intervals.

5. Conclusion

Our study showed that skeletal muscle growth was attenuated by DRF under overloading condition. Although metabolic dysfunctions have been observed in subjects who skipped breakfast and those who had night-eating syndrome [4,5], the muscle functions in these subjects remain unclear. In the future, studies should focus on the molecular mechanism of feeding-time-dependent effects on the skeletal muscle functions.

Funding

This work was partially supported by the Council for Science, Technology, and Innovation, SIP, "Technologies for creating next-generation agriculture, forestry, and fisheries" (funding agency: Bio-oriented Technology Research Advancement Institution, NARO) (Shibata S.) and the Japan Society for the Promotion of Science (JSPS) KAKENHI [grant number 17K18176] (Aoyama S.).

Author statement

S.A., S.K. and S.S. planned experiments; S.A., S.K., K.S., T.S., K.T., R.H. and Y.T. performed experiments; S.A. and S.K. analyzed data; S.A. and S.S wrote the manuscript.

Conflicts of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Acknowledgements

We would like to thank Editage [http://www.editage.com] for editing and reviewing this manuscript for English language.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jnim.2019.100099.

References

[1] S. Schiaffino, K.A. Dyar, S. Ciciliot, B. Blaauw, M. Sandri, Mechanisms regulating skeletal muscle growth and atrophy, FEBS J. 280 (2013) 4294–4314.
[2] T. Moro, S.M. Ebert, C.M. Adams, B.B. Rasmussen, Amino acid sensing in skeletal muscle, Trends Endocrinol. Metab. 27 (2016) 796–806.
[3] P.T. Reidy, B.B. Rasmussen, Role of ingested amino acids and protein in the promotion of resistance exercise-induced muscle protein anabolism, J. Nutr. 146 (2016) 155–183.
[4] H. Bi, Y. Gan, C. Yang, Y. Chen, X. Tong, Z. Lu, Breakfast skipping and the risk of type 2 diabetes: a meta-analysis of observational studies, Public Health Nutr. 18 (2015) 3013–3019.
[5] C. McCuen-Wurst, M. Ruggieri, K.C. Allison, Disordered eating and obesity: associations between binge-eating disorder, night-eating syndrome, and weight-related comorbidities, Ann. N. Y. Acad. Sci. 1411 (2018) 96–105.
[6] V.D. Longo, S. Panda, Fasting, circadian rhythms, and time-restricted feeding in healthy lifespan, Cell Metabol. 23 (2016) 1048–1059.
[7] J. Rothschild, K.K. Hoddy, P. Jambazian, K.A. Varady, Time-restricted feeding and risk of metabolic disease: a review of human and animal studies, Nutr. Rev. 72 (2014) 308–318.
[8] A. Zarrinpar, A. Chaix, S. Panda, Daily eating patterns and their impact on health and disease, Trends Endocrinol. Metab. 27 (2016) 69–83.
[9] Y.M. Cisse, J.C. Borniger, E. Lemanski, W.H. Walker 2nd, R.J. Nelson, Time-restricted feeding alters the innate immune response to bacterial endotoxin, J. Immunol. 200 (2018) 681–687.
[10] H. Wang, E. van Spyk, Q. Liu, M. Geyfman, M.L. Salmans, V. Kumar, A. Ihler, N. Li, J.S. Takahashi, B. Andersen, Time-restricted feeding shifts the skin circadian clock and alters UVB-induced DNA damage, Cell Rep. 20 (2017) 1061–1072.
[11] B.D. Harfmann, E.A. Schroder, K.A. Esser, Circadian rhythms, the molecular clock, and skeletal muscle, J. Biol. Rhythm. 30 (2015) 84–94.
[12] S. Schiaffino, B. Blaauw, K.A. Dyar, The functional significance of the skeletal muscle clock: lessons from Bmal1 knockout models, Skelet. Muscle 6 (2016) 33.
[13] S. Aoyama, S. Shibata, The role of circadian rhythms in muscular and osseous physiology and their regulation by nutrition and exercise, Front. Neurosci. 11 (2017) 63.
[14] S. Aoyama, S. Kojima, K. Sasaki, R. Ishikawa, M. Tanaka, T. Shimoda, Y. Hattori, N. Aoki, K. Takahashi, R. Hirooka, M. Takizawa, A. Haraguchi, S. Shibata, Day-Night Oscillation of Atrogin1 and Timing-dependent Preventive Effect of Weight-Bearing on Muscle Atrophy, EBioMedicine, 2018.
[15] T. Shavlakadze, T. Anwari, Z. Soffe, G. Cozens, P.J. Mark, C. Gondro, M.D. Grounds, Impact of fasting on the rhythmic expression of myogenic and metabolic factors in skeletal muscle of adult mice, Am. J. Physiol. Cell Physiol. 305 (2013) C26–35.
[16] J.S. You, G.B. Anderson, M.S. Dooley, T.A. Hornberger, The role of mTOR signaling in the regulation of protein synthesis and muscle mass during immobilization in mice, Dis. Model. Mech. 8 (2015) 1059–1069.
[17] S.M. Terena, K.P. Fernandes, S.K. Bussadori, A.M. Deana, R.A. Mesquita-Ferrari, Systematic review of the synergist muscle ablation model for compensatory hypertrophy, Revista da Associacao Medica Brasileira 63 (2017) (1992) 164–172.
[18] C.F. Bentzinger, S. Lin, K. Romanino, P. Castets, M. Guridi, S. Summermatter, C. Handschin, L.A. Tintignac, M.N. Hall, M.A. Ruegg, Differential response of skeletal muscles to mTORC1 signaling during atrophy and hypertrophy, Skelet. Muscle 3 (2013) 6.
[19] C.A. Goodman, D.M. Mabrey, J.W. Frey, M.H. Miu, E.K. Schmidt, P. Pierre, T.A. Hornberger, Novel insights into the regulation of skeletal muscle protein synthesis as revealed by a new nonradioactive in vivo technique, FASEB J. 25 (2011) 1028–1039.
[20] E.K. Schmidt, G. Clavarino, M. Ceppi, P. Pierre, SUnSET, a nonradioactive method to monitor protein synthesis, Nat. Methods 6 (2009) 275–277.
[21] S. Aoyama, H. Jia, K. Nakazawa, J. Yamamura, K. Saito, H. Kato, Dietary genistein prevents denervation-induced muscle atrophy in male rodents via effects on estrogen receptor-alpha, J. Nutr. 146 (2016) 1147–1154.
[22] N. Ito, U.T. Ruegg, A. Kudo, Y. Miyagoe-Suzuki, S. Takeda, Activation of calcium signaling through Trpv1 by nNOS and peroxynitrite as a key trigger of skeletal muscle hypertrophy, Nat. Med. 19 (2013) 101–106.
[23] R.P. Rhoads, L.H. Baumgard, S.W. El-Kadi, L.D. Zhao, Physiology and Endocrinology Symposium: roles for insulin-supported skeletal muscle growth, J. Anim. Sci. 94 (2016) 1791–1802.
[24] J.P. De Bandt, Leucine and mammalian target of rapamycin-dependent activation of muscle protein synthesis in aging, J. Nutr. 146 (2016) 2616s–2624s.
[25] J.O. Lipton, E.D. Yuan, L.M. Boyle, D. Ebrahimi-Fakhari, E. Kwiatkowski, A. Nathan, T. Guttler, F. Davis, J.M. Asara, M. Sahin, The circadian protein BMAL1 regulates translation in response to S6K1-mediated phosphorylation, Cell 161 (2015) 1138–1151.
[26] F. Sinturel, A. Gerber, D. Mauvoisin, J. Wang, D. Gatfield, J.J. Stubblefield, C.B. Green, F. Gachon, U. Schibler, Diurnal oscillations in liver mass and cell size accompany ribosome assembly cycles, Cell 169 (2017) 651–663 e614.
[27] K.A. Dyar, S. Ciciliot, L.E. Wright, R.S. Bienso, G.M. Tagliazucchi, V.R. Patel, M. Forcato, M.I. Paz, A. Gudiksen, F. Solagna, M. Albiero, I. Moretti, K.L. Eckel-Mahan, P. Baldi, P. Sassone-Corsi, R. Rizzuto, S. Bicciato, H. Pilegaard, B. Blaauw, S. Schiaffino, Muscle insulin sensitivity and glucose metabolism are controlled by the intrinsic muscle clock, Mol Metab. 3 (2014) 29–41.
[28] Y. Wang, L. Song, M. Liu, R. Ge, Q. Zhou, W. Liu, R. Li, J. Qie, B. Zhen, Y. Wang, F. He, J. Qin, C. Ding, A proteomics landscape of circadian clock in mouse liver, Nat. Commun. 9 (2018) 1553.
[29] K.A. Dyar, M.J. Hubert, A.A. Mir, S. Ciciliot, D. Lutter, F. Greulich, F. Quagliarini, M. Kleinert, K. Fischer, T.O. Eichmann, L.E. Wright, M.I. Pena Paz, A. Casarin, V. Pertegato, V. Romanello, M. Albiero, S. Mazzucco, R. Rizzuto, L. Salviati, G. Biolo, B. Blaauw, S. Schiaffino, N.H. Uhlenhaut, Transcriptional programming of lipid and amino acid metabolism by the skeletal muscle circadian clock, PLoS Biol. 16 (2018) e2005886.