Open Access

Recent Progress in Nutrition

LIDSEN Publishing Inc.

Original Research

Dose-Dependent Effects of Berberine Chloride Dihydrate on Proliferation of C2C12 Myoblasts and Differentiation of 3T3L1 Adipocytes

Dylan J. Stephens [1, 2], Evan B. Johnson [1], Bradley J. Johnson [1, *]

1. Department of Animal and Food Sciences, Texas Tech University, Lubbock, TX, USA; E-Mails: dylan.j.stephens@ttuhsc.edu; evan.johnson904@gmail.com; bradley.johnson@ttu.edu
2. School of Medicine, Texas Tech University Health Sciences Center, Lubbock, TX, USA

* **Correspondence:** Bradley J. Johnson; E-Mail: bradley.johnson@ttu.edu

Academic Editor: Costantino Paciolla

Recent Progress in Nutrition
2026, volume 6, issue 3
doi:10.21926/rpn.2603018

Received: February 21, 2026
Accepted: July 29, 2026
Published: July 30, 2026

Abstract

Berberine is an alkaloid chemical found in plants, including *Berberidaceae*, *Coptis rhizomes*, and *Hydrastis canadensis*. Berberine has been shown to improve insulin sensitivity, promote adipocyte loss, and is associated with higher levels of skeletal muscle protein synthesis. The purpose of this study was to examine berberine's dose-dependent effects on C2C12 myoblast proliferation and 3T3L1 adipocyte differentiation. Cells were grown out over 3 weeks, and once confluent at 90%, they were treated with berberine to examine its effect on cell survival and growth. One-way ANOVA was used to analyze differences between groups. Results showed that a 10 µM dose of berberine had an optimal effect on C2C12 myoblast proliferation at 72 hours ($p < 0.01$). In contrast, after 48 hours of inoculation, 10 µM and 100 µM doses of berberine showed suppressive effects on 3T3L1 adipocyte cell counts ($p = 0.00035$). In conclusion, berberine appeared to be advantageous for C2C12 myoblasts' survival, whereas 3T3L1 adipocytes suffered in its presence after 48 hours of inoculation.

Keywords

Berberine; C2C12; 3T3L1; cell proliferation; cell differentiation; type 2 diabetes mellitus (T2DM); AMP-activated protein kinase (AMPK); myoblasts; adipocytes

1. Introduction

Berberine is a potent alkaloid chemical found in plants such as *Berberidaceae*, *Coptis rhizomes*, and *Hydrastis Canadensis* [1]. This phytochemical has historically been used in traditional Chinese medicine to treat many conditions [2]. More recent research has examined berberine and its clinical role in weight loss and obesity [1], muscle degradation [3], insulin resistance [4], gut health [5], and anti-cancer properties [6].

Berberine has well-documented effects on altering obesity in rat models. Ilyas et al. illustrated that berberine, when supplemented in obese mouse models, decreased cholesterol absorption and plasma lipids [1]. Dyslipidemia is attenuated with supplementation of berberine, with Shen et al. noting that berberine supplementation raises ox-low-density lipoprotein (LDL)-induced macrophages [7], which could play a vital role in susceptible individuals. Similarly, weight loss has been shown to have a positive correlation with increased glucagon-like peptide (GLP) levels. Demonstrably, Wang et al. found that berberine supplementation raised both GLP-1 and GLP-2 levels, resulting in lower fasting plasma glucose [8]. These GLPs also have a vital role in nutrient absorption, and increased levels have been shown to decrease inflammation and improve intestinal integrity [9].

Many research labs have examined berberine's role in decreasing insulin resistance. Yin et al. conducted a study exploring the hypoglycemic effect of berberine on individuals with Type 2 Diabetes Mellitus (T2DM); their findings indicate that berberine decreased blood glucose levels and other T2DM indicators [10]. A systematic review by Pang et al. examined the effect of berberine on insulin sensitivity and insulin secretion, and they found that berberine enhanced insulin secretion and decreased the resting plasma glucose levels [11]. In turn, berberine could potentially decrease the possibility of individuals developing T2DM and similar etiologies.

Some research has looked at berberine's effect on increasing insulin sensitivity in tissue by way of increased glucose transporter type 4 (GLUT-4) and other beta cell receptors [12]. These receptors are crucial in the absorption of glucose into peripheral tissues, like myocytes and adipocytes, and assist in lowering blood glucose levels. Berberine has also been shown to upregulate glycolysis in part due to AMP-activated protein kinase (AMPK) activation, which has been conclusively shown to lower insulin resistance [13]. More research on GLUT-4's mechanistic relationship with berberine appears optimistic when examining the studies that have viewed them in parallel.

Relatedly, berberine has been shown to alter muscle protein synthesis (MPS). Wang et al. conducted a study where they examined MPS and degradation when berberine was introduced in insulin-resistant mice [14]. They found that berberine activated a key catabolic protein, atrogin-1, when it was applied to mice. Berberine and atrogin-1 have strong relations, which lead to increases in muscle atrophy and stunted overall mitochondrial biogenesis [14]. Their group then added peroxisome proliferator-activated receptor coactivator (PGC-1a), a well-known agonist of mitochondrial remodeling and biogenesis, and observed that PGC-1a blunted the impact of berberine and atrogin-1 [14].

Berberine has been shown to have a marginal effect on the production of AMPK through the LKB1-AMPK-TORC2 signaling pathway in diabetic mice [15]. Increases in AMPK have been shown to inhibit gluconeogenesis and have effects on other mitochondrial functions [16]. This pathway leads

to lower plasma insulin and significantly lower insulin resistance and has a profound effect on the reduction of the expression of necessary gluconeogenic enzymes like phosphoenolpyruvate carboxykinase (PEPCK) and glucose-6-phosphatase (G-6-P) [16]. As a result, mitochondrial function and biogenesis are impaired.

While some in vivo studies have examined berberine's effect in vivo models, limited research is available on its impact on in vitro myocyte models. This study aimed to explore this research gap. Based on the published literature on adipocytes, we hypothesized that proliferation in C2C12 cells would decrease in the treatments supplied with berberine. Furthermore, we predicted a decrease in adipocyte cell number and associated growth with the application of berberine.

2. Materials and Methods

Berberine used in this study was manufactured by Biosynth at 95% purity and dissolved in Dimethyl Sulfoxide (DMSO), which acted as the solvent in the control (CTL) group. The C2C12 and 3T3L1 vials were stored in a Thermolyne Locator 8 FFF Cryobiological Storage System (-196°C). The cells were heated in a Thermoscientific Precision Fischer Water Bath (37°C), which took 5 to 7 minutes to fully thaw. Cells (1 mL) were added to a growth medium (GM) (25 mL) and then vortexed for 10 seconds (s) to homogeneously mix the solution. A 200 mL solution of growth media was prepared, containing 90% Dulbecco's Modified Eagle Medium (DMEM) and 10% Fetal Bovine Serum (FBS). 2 mL of Antibiotic-Antimycotic and 200 µL of Gentamicin were added to 200 mL of the GM. Finally, 10 mL of the cell suspension was added to two T-75 flasks and grown in nutrient-rich GM. Cells were incubated at 37°C and 5% carbon dioxide (CO_2) and fed every 48 hours until reaching 90% confluency. At this point, the growing phase was completed for the 3T3L1 lineage. At this point, the 3T3L1 cells were treated with berberine and allowed to differentiate over a 48-hour time frame in the incubator. Images were then taken to qualitatively visualize the effect of berberine before visualizing with GFP/DAP microscopy. At 48 h, the cells were then visualized under microscopy, and differentiation was seen via microscopy with GFP and DAP stain within their respective T-75 flasks in a dark room.

For the C2C12 cell lineages, an extensive feeding, passing, proliferation, and counting protocol was conducted from the T-75 flasks to interpret and visualize proliferation rates in each berberine dosage CTL group.

Feeding Protocol: After 24 h, the old GM was aspirated off the flask with a vacuum apparatus, then replaced with 10mL of new GM. Cells were then placed back into the Symphony Incubator by VWR.

Passing Protocol: After using the Evos XL Microscope at 1000× by Invitrogen and confirming cell confluency was at 80 to 90%, GM was aspirated, and Phosphate Buffered Saline (PBS) was applied to wash possible contaminants within the flask (10 mL). PBS was then aspirated. Cells were then trypsinized using Trypsin EDTA (3 mL per flask) and incubated for 5 min at 37°C. Cells were then broken off the flask by gently tapping against one's hand, then viewed under the microscope to ensure the cells were freely moving and completely separated from the plate. GM (7 mL) was added to each flask to neutralize the trypsin. Both flasks were pipetted into a single 50 mL conical tube. Cell solution was then vortexed for 10 s.

Counting Protocol: 100 µL of the cell solution and 100 µL of Trypan Blue Stain 0.4% by Gibco were pipetted into a 2 mL Olympus Plastics Microtube. They were then vortexed and tapped with one's

hand to guarantee a fully mixed solution. 10 µL were pipetted into both sides of an Invitrogen Countess Cell Counting Chamber Slide by Thermofischer Scientific. Averages of the cell count were taken from both sides and then converted to 25,000 cells/mL. This concentration of cells was passed into a 96-well plate at 2,500 cells/well (100 µL). 24 h after passing into the 96-well plate, 4 doses of berberine were added (0.01 µM, 0.1 µM, 1 µM, 10 µM). Two sets of three biological replicates were measured for proliferation every day from the well plates over five days (96 h). Individual wells were also considered technical replicates in this design, with a total of 6 per dose. There were also 6 negative CTL and 6 blanks measured in every assay (every 24 h).

Proliferation Assay Protocol: Promega Cell Titer 96 Aqueous One Solution Reagent was thawed to its liquid state. Starting at hour zero, 20 µL of the proliferation assay was added to the cells in a dark environment. Next, a plate vortex was used for 1 minute to ensure all components were mixed completely. The cells were then incubated at 37°C and 5% CO_2 for 1 h. Cells were taken out of the incubator and placed into aluminum foil to ensure no light exposure. Plates were then read using the BioTek Epoch Microplate Spectrophotometer set at 490 nm. Blanks and treatments were labelled, read, and analyzed into an Excel sheet. This process was repeated every 24 hours over the 96 hours. Two sets, which consisted of separate well plates, were analyzed at different time periods. Data were then extrapolated and analyzed.

Data Analysis: Two T-75 flasks were imaged with 3 images per flask at different areas for analysis of 3T3L1 differentiation for a total of 6 biological replicates per group. For C2C12 myoblasts, 2 separate sets of well plates were analyzed with 3 biological replicates per plate, with a total of 6 biological replicates per group. Outliers were tested within each combination of set, treatment, and time, and removed if the deviation from the quartile was greater than 3 × IQR. Data was analyzed by one-way ANOVA within each time point, with set included in the model as a random effect. Pairwise comparison was conducted with Tukey adjustment protected by ANOVA. The denominator degrees of freedom were calculated with the Kenward-Roger adjustment. Significance compared to $\alpha = 0.05$.

2.1 Ethics Statement

This study utilized animal-origin immortalized cell lines obtained from the Department of Animal and Food Sciences at Texas Tech University. As no live animal tissues were studied, no ethical approval was required to perform this study.

3. Results

C2C12 Proliferation: Our group was interested in determining the dose-dependent effects of berberine on the rate of cellular proliferation of C2C12 myoblasts. Table 1 illustrates the time and dose-dependent response of proliferation in response to berberine supplementation. The C2C12 cells followed a normal increase in cellular proliferation over the 96-h period. A trend was noted advancing toward a statistically significant p-value as the time course went on (linear and quadratic). Absorbance is a direct and predictive measure of the myoblast's proliferation. At 0 h, minimal cell proliferation occurred across all dosages. For CTL and 0.01 µM, nominal proliferation was observed. The 0.1 µM dose had lower proliferation at 0 h. This trend continued for the 1 µM at 0 h. As the concentration of berberine increased, it was anticipated that proliferation would follow. This relationship was noted; 10 µM had the highest proliferation at 0 h. Standard Error of the Mean (SEM)

at 0 h was 0.0195. The linear p-value was statistically insignificant (p = 0.34). At 24 h, proliferation increased approximately twofold across all treatments. The linear p-value was insignificant (p = 0.30). SEM at 24 h was 0.0336. Approximately a threefold increase was observed between 24 and 48 h. SEM at 48 h was 0.1430. Although this period was insignificant (p = 0.69), proliferation was advancing toward a statistically significant outcome across all treatments. The rate of increase was to a lesser extent, but 72 h provided the optimal proliferation across all the doses, with 10 µM showing statistical significance from the rest of the groups, apart from 1 µM. According to Figure 1, proliferation proved to be significant quadratically (p < 0.01). SEM at 72 h was 0.0975. At 96 h, the higher doses (1 µM, 10 µM) decreased in proliferation. In contrast, the lower concentrations (CTL, 0.01 µM, 0.1 µM) continued to increase steadily. Despite this observation, the linear p-value was insignificant (p = 0.71). SEM at 96 h was 0.1600.

Table 1 Proliferation assay absorbance results from 6 biological replicates of C2C12 cells treated with varying doses of berberine and CTL.

Absorbance of C2C12 0-96 hours with berberine								
Time	0 µM	0.01 µM	0.1 µM	1 µM	10 µM	SEM	Linear P Value	Quadratic P Value
0 h	0.070	0.070	0.069	0.060	0.088	0.019	0.34	0.63
24 h	0.157	0.157	0.157	0.161	0.197	0.033	0.30	0.55
48 h	0.510	0.510	0.510	0.514	0.550	0.143	0.69	0.30
72 h	0.625^{b}	0.628^{b}	0.654^{b}	0.891^{ab}	0.929^{a}	0.097	0.02	<0.01
96 h	0.841	0.841	0.845	0.874	0.889	0.1600	00.71	0.82

$^{a, b}$: Groups that don't share a letter are statistically significant (p < 0.05).

Absorbance: unitless outcome.

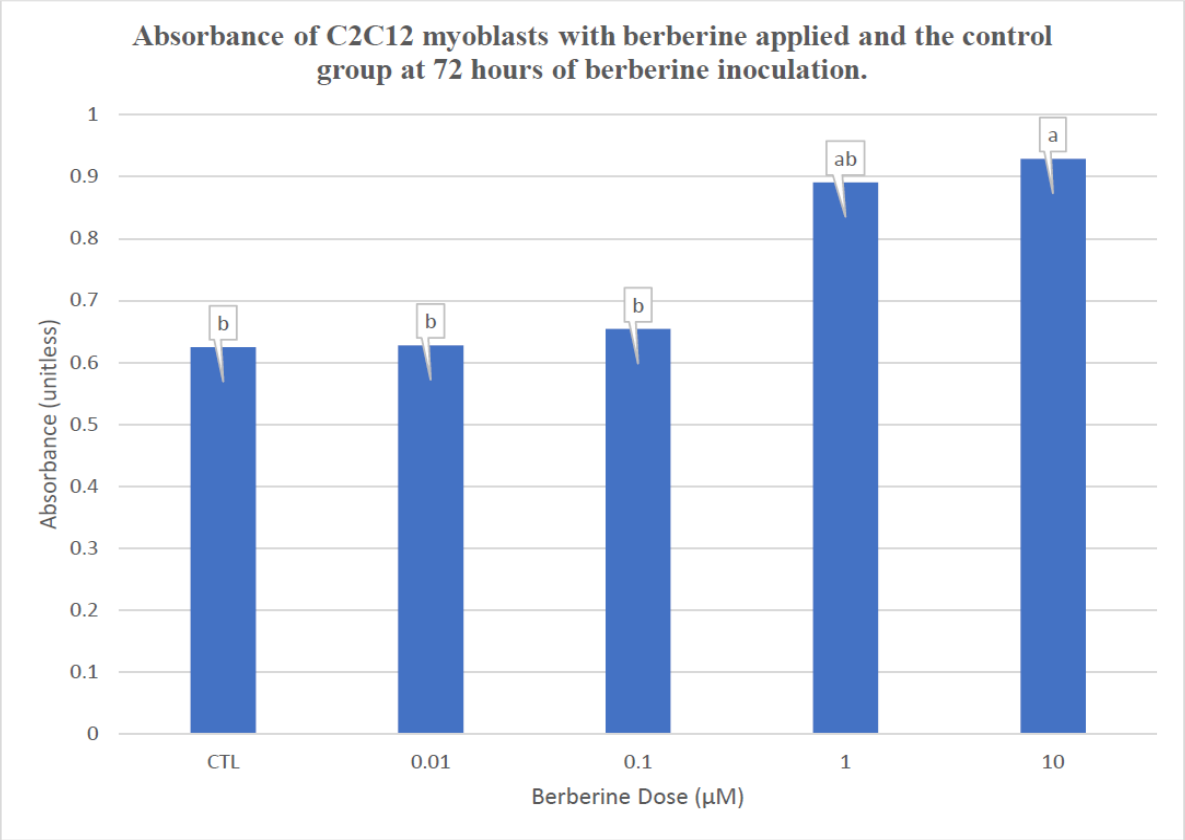

Figure 1 Absorbance of C2C12 myoblasts treated with berberine at 72 hours of berberine inoculation.

Qualitative Visualization of C2C12 Myoblasts Fusion: Figure 2 visualizes the C2C12 myoblasts differentiation over a 96-h period. The time course visualizes the fusion process from 0 h to 96 h of exposure to 1.0 µM of berberine. The addition of berberine appeared to stimulate both the extent of myoblast fusion to myotubes and the morphological size of the myoblasts. Furthermore, Figure 3 displays the differing levels of myotube fusion between the CTL, 0.1 µM, and 1 µM groups, with the 1 µM group showing the most extensive network of myotube fusion and growth. As expected, the 0.1 µM showed the second-highest level of fusion, followed by the CTL group. The 10 µM group wasn't visualized, as the berberine was performed on the 3T3L1 adipocytes first, which showed strong suppression of cell growth and number.

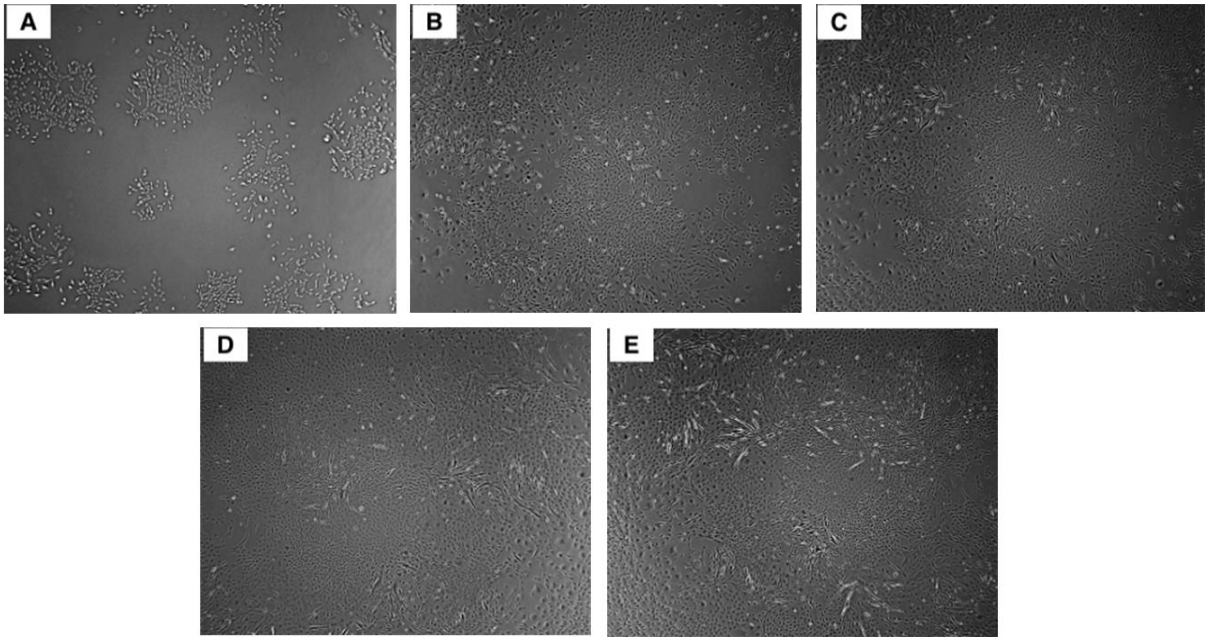

Figure 2 1000× magnification of 1.0 µM supplemented C2C12 myoblasts growth and fusion over a 96-hour time course. (A) 0 hours of berberine inoculation. (B) 24 hours of berberine inoculation. (C) 48 hours of berberine inoculation. (D) 72 hours of berberine inoculation. (E) 96 hours of berberine inoculation.

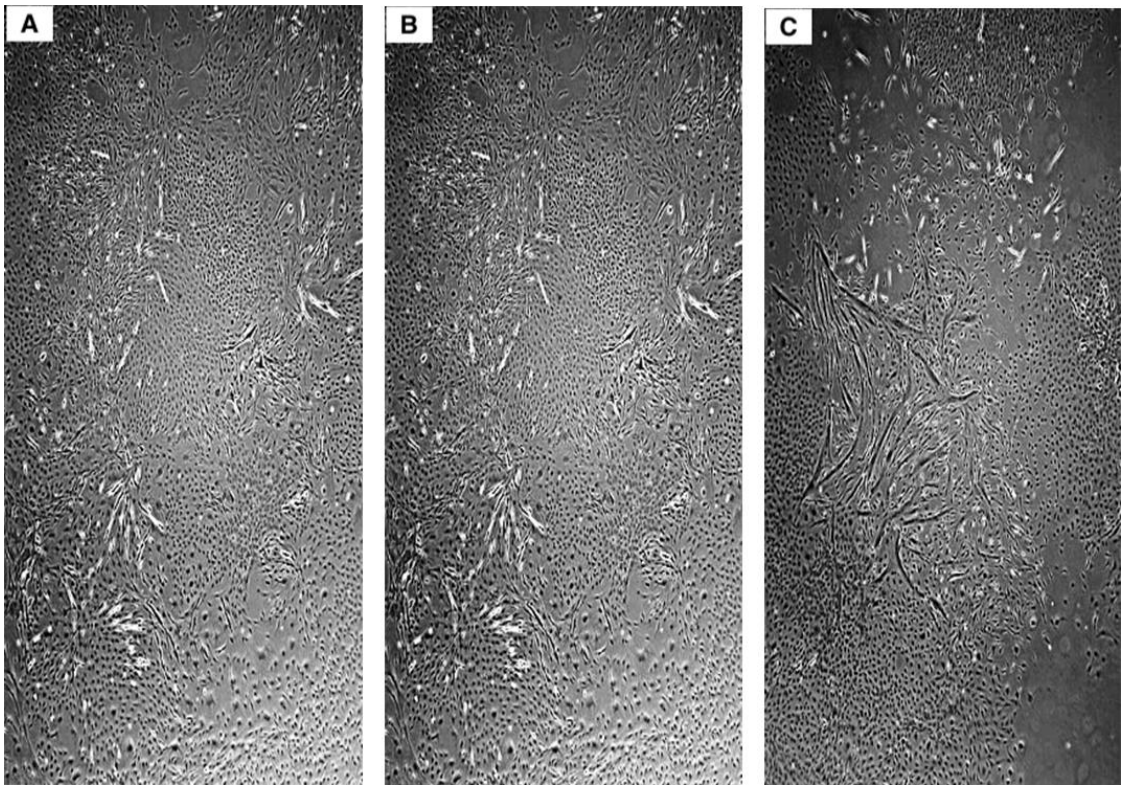

Figure 3 1000× magnification displaying differentiated C2C12 myoblasts at 96 hours of berberine inoculation. (A) Control group. (B) 0.1 µM group. (C) 1.0 µM group.

Quantitative Cell Count of 3T3L1 Adipocytes: Berberine displayed a strong inhibitory effect on the 3T3L1 cell lineage, seen both qualitatively in Figure 4 and quantitatively in Table 2. Specifically,

after 48 h of berberine inoculation, berberine markedly reduced the cell number when applied to the 10 µM and 100 µM 3T3L1 groups, which is consistent with possible apoptosis. Average cell counts of the 10 µM and 100 µM groups were 6.97 and 2.57, respectively. This showed strong statistical significance of berberine's anti-differentiation effects when compared to the CTL, 0.1 µM, and 1.0 µM groups with cell counts of 194.71, 143.5, and 127.72, respectively. There was also a statistical significance between the 1 µM and CTL groups. For the total data analysis, SEM was 9, and the p-value among all groups was 0.00035, which displays berberine's potent effects on inhibiting growth of the 3T3L1 cell lineage.

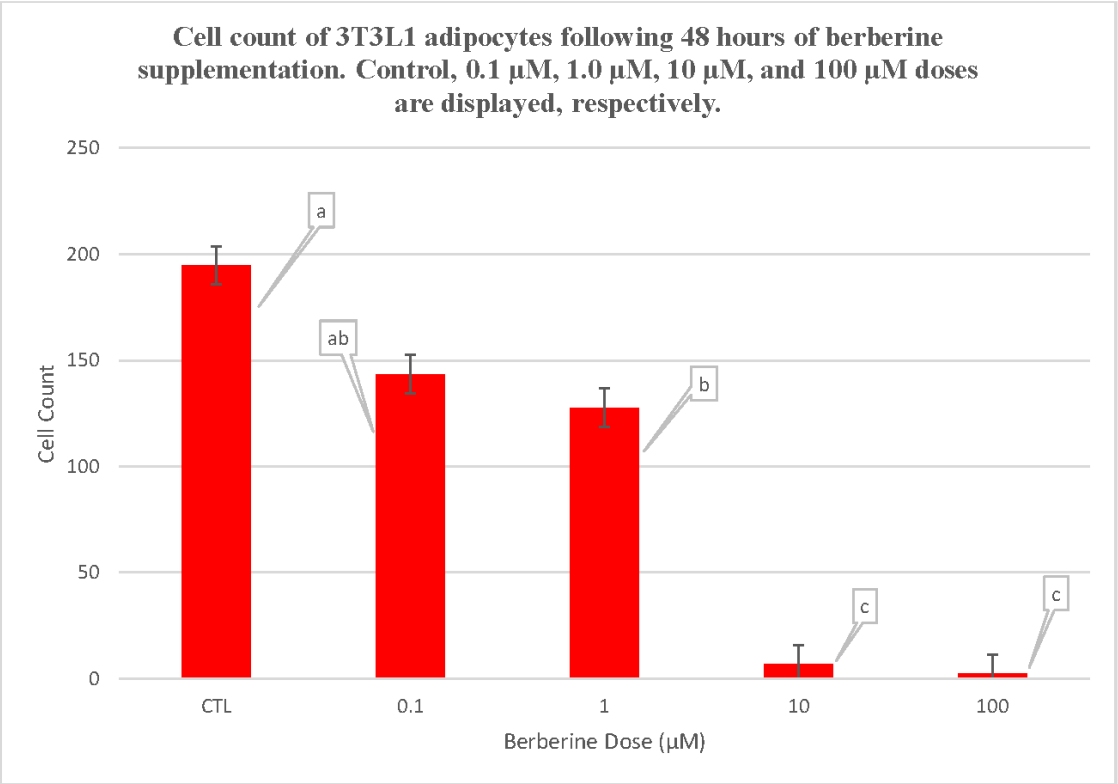

Figure 4 Cell count of 3T3L1 adipocytes following 48 hours of berberine supplementation. Control, 0.1 µM, 1.0 µM, 10 µM, and 100 µM doses are displayed, respectively.

Table 2 Cell Count of 2 sets of T-75 flasks holding 3T3L1 Adipocytes 48 hours following the application of berberine.

Cell count of 3T3L1 48 hours after inoculation of berberine						
CTL	0.1 µM	1 µM	10 µM	100 µM	SEM	P-value
194.71^{a}	143.5^{ab}	127.72^{b}	6.97^{c}	2.57^{c}	9	0.00035

$^{a, b, c}$: Groups that don't share a letter are statistically significant ($p < 0.05$).

Qualitative Effects of Berberine on 3T3L1 Cell Count and Differentiation: As highlighted by Figure 5, there was an inverse qualitative effect on the differentiation and survival of 3T3L1 cells 48 h following application of berberine. Progressively, as the dose was increased, cell count diminished drastically, showing that berberine may have inhibited growth and differentiation of 3T3L1 cells at

higher doses. Figure 6 shows the 3T3L1 cells 48 h after supplementation of berberine, before the staining process took place.

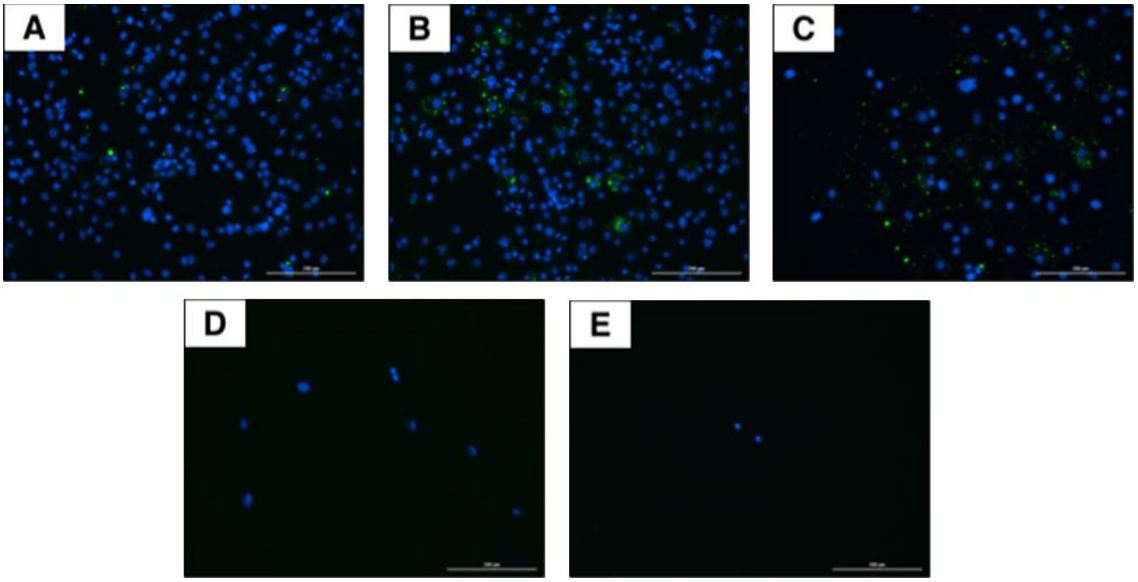

Figure 5 Visualization of stained, differentiated 3T3L1 adipocytes at 48 hours of berberine supplementation. (A) Control group. (B) 0.1 µM group. (C) 1.0 µM group. (D) 10 µM group. (E) 100 µM group. CTL; control.

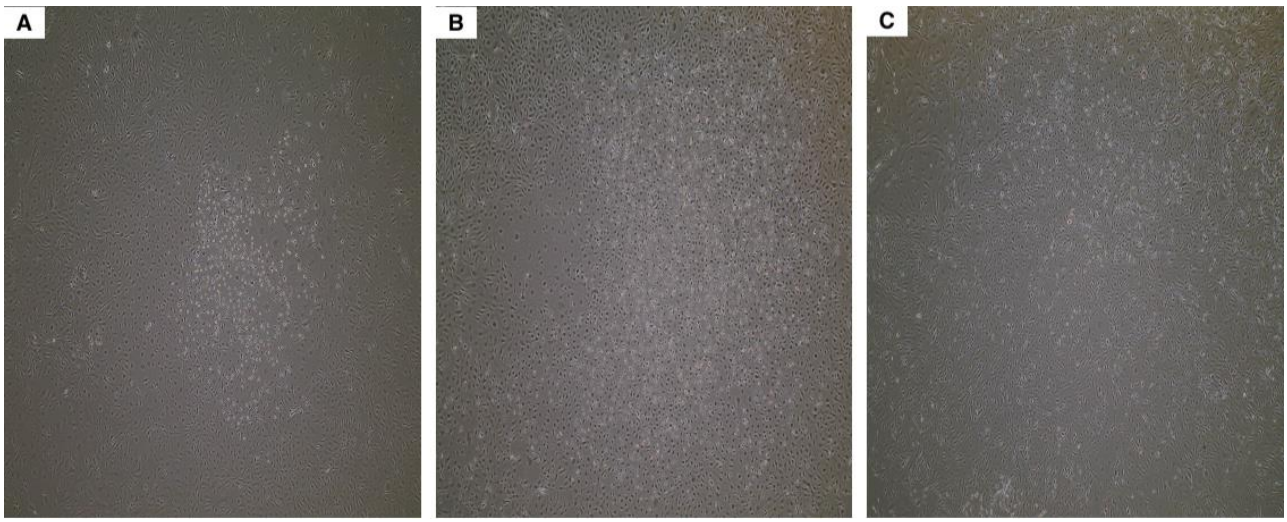

Figure 6 1000× magnification showing unstained 3T3L1 adipocytes after 48 hours of differentiation before application of berberine. (A) Control group. (B) 0.1 µM group. (C) 1.0 µM group. 10 µM and 100 µM groups weren't included due to a lack of visualization and low cell count.

4. Discussion

Berberine has become a popular supplement for the suppression of glucose modulation. As previously mentioned, berberine has been demonstrated to decrease plasma glucose in diabetic patients [10] and inhibit gluconeogenesis in diabetic rats [15]. Furthermore, berberine leads to

lower levels of vascular smooth muscle cell proliferation and apoptosis [17], while also attenuating the expression of certain genetic components associated with adipogenesis [18].

Our results suggest that berberine may play a role in promoting proliferation and differentiation in C2C12 myoblasts and suppressing differentiation of 3T3L1 adipocytes. 72 h appears to be the optimal point for proliferation in skeletal muscle. After this point, the cells likely differentiated into multinucleated myotube blasts. This shift in differentiation may explain the decrease in proliferation observed at 96 h. Possible reasons for this reduction are depletion of nearby nutrients or increased confluency of the myoblasts. Qualitatively visualized, myotube size was larger with higher doses of berberine (Figure 3). On the contrary, berberine appeared to coordinate a decrease in cell count in the 3T3L1 adipocytes, seen both quantitatively and qualitatively. Our results suggest berberine may play a role in various pathways, with some described in this study. Further studies to examine key metabolic pathways, proteins, and enzymes to explain and confirm these preliminary findings.

Our results conflict with previous in vivo studies on berberine's impact on muscle atrophy [14]. This study aimed to fill the current research gap of berberine in in vitro muscle lineages, and our results propose a major discrepancy between in vivo and in vitro models. This pilot study shows that both time and dose may play a role in berberine's effect on muscle growth.

4.1 Limitations

Our study didn't examine polymerase chain reaction (PCR), Western blot, and related tests to further examine the key underlying pathways of AMPK, mTOR, apoptosis, and differentiation markers described in the text. Similarly, toxicity assays weren't assessed during berberine inoculation, which could provide valuable insights into the results of the essay. Furthermore, quantitative analysis of images and figures was not performed, as this was a pilot study on the novelty of berberine. One-way ANOVA statistical analysis was performed, which is a notable limitation. The impact of berberine was examined on the 3T3L1 cells before the C2C12 cells, and the decreased number of cells was a crucial reason for not having a 100 µM dose for the C2C12 cells. Future studies may benefit from exploring increased doses of berberine on muscle lineages, as ours suggest it may be beneficial in muscle lineage growth in vitro models. Lastly, no controls for solvent effects were employed during the study.

5. Conclusions

Our study found that berberine attenuated 3T3L1 adipocyte cell numbers with increasing dose, with a reduction in cell count consistent with possible apoptosis seen in cells at the 10 µM and 100 µM dosages. In contrast, it appears that berberine supplementation significantly enhanced C2C12 myoblast proliferation, notably at the 72-h mark with the 10 µM dose, both linearly and quadratically. Future research should continue to examine the use of berberine on adipocyte and myoblast cell lines, with a focus on deepening the knowledge it has on molecular assays and pathways in vitro and video models. Our study suggests these effects, but further experimental studies are needed to confirm our conjecture. Our results show that berberine may have a positive interaction with in vitro myoblasts at various cell cycle checkpoints, facilitating opportunistic growth.

Abbreviations

MPS	Muscle protein synthesis
AMPK	AMP-activated protein kinase
T2DM	Type 2 Diabetes Mellitus
mTOR	mechanistic Target of Rapamycin
LDL	low-density lipoprotein
GLP	glucagon-like peptide
GLUT-4	glucose transporter type 4
PGC-1a	proliferator-activated receptor coactivator
PEPCK	phosphoenolpyruvate carboxykinase
G-6-P	glucose-6-phosphatase
GM	growth media
DMEM	Dulbecco's Modified Eagle Medium
FBS	Fetal Bovine Serum
µM	micromolar
mL	milliliter
CTL	control
PBS	Phosphate Buffered Saline
SEM	Standard Error of the Mean
PCR	polymerase chain reaction

Author Contributions

Dylan J. Stephens: Conceptualization, methodology, investigation, data curation, visualization; writing – original draft; review and editing. Evan J. Johnson: Conceptualization, methodology, investigation, data curation, visualization; writing – original draft; review and editing. Bradley J. Johnson: Conceptualization, methodology, investigation, data curation, visualization; writing – original draft; review and editing; project administration. All authors have read and approved the published version of the manuscript.

Funding

This study was supported by the Gordon W. Davis Reagents Chair in Meat Science and Muscle Biology Endowment at Texas Tech University.

Competing Interests

The authors have declared that no competing interests exist.

References

1. Ilyas Z, Perna S, Al-Thawadi S, Alalwan TA, Riva A, Petrangolini G, et al. The effect of Berberine on weight loss in order to prevent obesity: A systematic review. Biomed Pharmacother. 2020; 127: 110137.

2. Shen CY, Jiang JG, Yang L, Wang DW, Zhu W. Anti-ageing active ingredients from herbs and nutraceuticals used in traditional Chinese medicine: Pharmacological mechanisms and implications for drug discovery. Br J Pharmacol. 2017; 174: 1395-1425.
3. Choi YH. Berberine hydrochloride protects C2C12 myoblast cells against oxidative stress-induced damage via induction of Nrf-2-mediated HO-1 expression. Drug Dev Res. 2016; 77: 310-318.
4. Cheng J, Ma X, Yan G, Yu Q, Huang Z, Lin G, et al. High fructose-induced skeletal muscle insulin resistance could be alleviated by berberine via AMPD1 and ADSL. Food Chem Toxicol. 2023; 175: 113731.
5. Zhang L, Wu X, Yang R, Chen F, Liao Y, Zhu Z, et al. Effects of berberine on the gastrointestinal microbiota. Front Cell Infect Microbiol. 2021; 10: 588517.
6. Xiong RG, Huang SY, Wu SX, Zhou DD, Yang ZJ, Saimaiti A, et al. Anticancer effects and mechanisms of berberine from medicinal herbs: An update review. Molecules. 2022; 27: 4523.
7. Shen X, Chen Y, Yu L, Zheng J, Shang S, Wang J. Berberine improves Ox-LDL-induced macrophage efferocytosis dysfunction and alleviates inflammation via the PPARγ/LXRα pathway. Tohoku J Exp Med. 2025; 267: 185-201.
8. Wang Y, Liu H, Zheng M, Yang Y, Ren H, Kong Y, et al. Berberine slows the progression of prediabetes to diabetes in Zucker diabetic fatty rats by enhancing intestinal secretion of glucagon-like peptide-2 and improving the gut microbiota. Front Endocrinol. 2021; 12: 609134.
9. Abdalqadir N, Adeli K. GLP-1 and GLP-2 orchestrate intestine integrity, gut microbiota, and immune system crosstalk. Microorganisms. 2022; 10: 2061.
10. Yin J, Xing H, Ye J. Efficacy of berberine in patients with type 2 diabetes mellitus. Metabolism. 2008; 57: 712-717.
11. Pang B, Zhao LH, Zhou Q, Zhao TY, Wang H, Gu CJ, et al. Application of berberine on treating type 2 diabetes mellitus. Int J Endocrinol. 2015; 2015: 905749.
12. Baska A, Leis K, Gałązka P. Berberine in the treatment of diabetes mellitus: A review. Endocr Metab Immune Disord Drug Targets. 2021; 21: 1379-1386.
13. Cicero AF, Tartagni E. Antidiabetic properties of berberine: From cellular pharmacology to clinical effects. Hosp Pract. 2012; 40: 56-63.
14. Wang H, Liu D, Cao P, Lecker S, Hu Z. Atrogin-1 affects muscle protein synthesis and degradation when energy metabolism is impaired by the antidiabetes drug berberine. Diabetes. 2010; 59: 1879-1889.
15. Jiang SJ, Dong H, Li JB, Xu LJ, Zou X, Wang KF, et al. Berberine inhibits hepatic gluconeogenesis via the LKB1-AMPK-TORC2 signaling pathway in streptozotocin-induced diabetic rats. World J Gastroenterol. 2015; 21: 7777-7785.
16. Wu S, Zou MH. AMPK, mitochondrial function, and cardiovascular disease. Int J Mol Sci. 2020; 21: 4987.
17. Wang L, Deng L, Lin N, Shi Y, Chen J, Zhou Y, et al. Berberine inhibits proliferation and apoptosis of vascular smooth muscle cells induced by mechanical stretch via the PDI/ERS and MAPK pathways. Life Sci. 2020; 259: 118253.
18. Yang Y, Liu F, Lu R, Jia J. Berberine inhibits adipogenesis in porcine adipocytes via AMP-activated protein kinase-dependent and-independent mechanisms. Lipids. 2019; 54: 667-678.