Data in Brief 40 (2022) 107773

Contents lists available at ScienceDirect

Data in Brief

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

Data Article

Dataset for liver metabolomic profile of highland barley *Monascus purpureus* went extract-treated golden hamsters with nonalcoholic fatty liver disease

Mei-Ning Zhu[a,1], Cui-Zhu Zhao[a,1], Chong-Zhi Wang[b,c], Jian-Bo Rao[a], Yong-Wei Qiu[a], Yan-Ping Gao[a], Xiao-Yun Wang[a], Ya-Mei Zhang[a], Guang Wu[a], Jie Chen[a], Qin-Ge Ma[a,b,c,*], Guo-Yue Zhong[a,*], Rong-Rui Wei[a,**]

[a] *Research Center of Natural Resources of Chinese Medicinal Materials and Ethnic Medicine and Key Laboratory of Modern Preparation of Chinese Medicine of Ministry of Education, Jiangxi University of Traditional Chinese Medicine, Nanchang 330004, China*
[b] *Tang Center for Herbal Medicine Research, The University of Chicago, Chicago 60637, United States*
[c] *Department of Anesthesia and Critical Care, The University of Chicago, Chicago 60637, United States*

ARTICLE INFO

Article history:
Received 19 December 2021
Revised 26 December 2021
Accepted 28 December 2021
Available online 30 December 2021

Keywords:
Highland barley *Monascus purpureus* went
Nonalcoholic fatty liver disease
Metabolites
High-fat
High-fructose
High-cholesterol diet

ABSTRACT

Nonalcoholic Fatty Liver Disease (NAFLD) is a serious problem endangering human health in the world. The pathogenesis of this disease is often accompanied by lipid metabolism disorder and can cause liver lipid accumulation. Highland barley *Monascus purpureus* Went extract (HBMPWE) can inhibit the liver lipid accumulation caused by a high-fat, high-fructose, high-cholesterol diet. However, it is not clear what changes have taken place in the process of liver lipid metabolism after HBMPWE administration. To fill this knowledge gap and to support the findings published in the companion research article entitled "Highland Barley *Monascus purpureus* Went Extract Ameliorates High-Fat, High-Fructose,

* Corresponding author at: Key Laboratory of Modern Preparation of Chinese Medicine of Ministry of Education, Jiangxi University of Traditional Chinese Medicine, Nanchang 330004, China.
** Corresponding author at: Research Center of Natural Resources of Chinese Medicinal Materials and Ethnic Medicine, Jiangxi University of Traditional Chinese Medicine, Nanchang 330004, China.
E-mail addresses: 20181020@jxutcm.edu.cn (Q.-G. Ma), 20111025@jxutcm.edu.cn (G.-Y. Zhong), 20171029@jxutcm.edu.cn (R.-R. Wei).
[1] Co-first authors: Mei-Ning Zhu and Cui-Zhu Zhao.

https://doi.org/10.1016/j.dib.2021.107773

High-Cholesterol Diet Induced Nonalcoholic Fatty Liver Disease by Regulating Lipid Metabolism in Golden Hamsters" [1], we provided important information related to the liver differential metabolites and identified twenty-one differential metabolites of liver metabolism. In the model group, the levels of lactate, linoleic acid, and malic acid increased significantly. After HBMPWE treatment, the expressions of these metabolites reduced significantly. Therefore, these liver differential metabolites could be used as biological signatures reflecting the severity of NAFLD and HBMPWE treatment outcomes.

Specifications Table

Subject	Health and Medical Sciences
Specific subject area	Liver, metabolic diseases, NAFLD,biological signature
Type of data	Figures and Tables
How data was acquired	Q Exactive UPLC-MS/MS
Data format	Raw, Analysed
Description of data collection	Metabolomic liver profiles of golden hamsters, NAFLD golden hamsters and HBMPWE-treated NAFLD golden hamsters were investigated. Metabolomic liver profiling from NAFLD model group and HBMPWE-treated group.
Data source location	Data were collected at Jiangxi University of Traditional Chinese Medicine, Nanchang, China
Data accessibility	Data is provided within this article
Related research article	C.Z. Zhao, W. Jiang, Y.Y. Zhu, C.Z. Wang, W.H. Zhong, G. Wu, J. Chen, M.N. Zhu, Q.L. Wu, X.L. Du, Y.Y. Luo, M. Li, H.L. Wang, H. Zhao, Q.G. Ma, G.Y. Zhong, R.R. Wei, Highland barley *Monascus purpureus* Went extract ameliorates high-fat, high-fructose, high-cholesterol diet induced nonalcoholic fatty liver disease by regulating lipid metabolism in golden hamsters, J. Ethnopharmacol. 10.1016/j.jep.2021.114922.

Value of the Data

- These data of differential metabolites are closely correlated with lipid metabolism index, body weight, and liver index. The changes of differential metabolites play important role in predicting and studying the mechanism of NAFLD.
- Researchers working on pathogenesis, mechanism, biological function of metabolites of NAFLD can benefit from these data. It can be judged the pathophysiological state of the liver, speculated the mechanism, and verified the biological function according to differential metabolites of NAFLD.
- These data might be used for studying targeted metabonomics and looking for genes related to metabolic pathways according to differential metabolites, so as to clarify the mechanism of NAFLD.

1. Data Description

1.1. Screening of liver differential metabolites after HBMPWE treatment

The orthogonal partial least squares discriminant analysis (OPLS-DA)-supervised pattern recognition method was applied to identify the overall metabolic differences between two

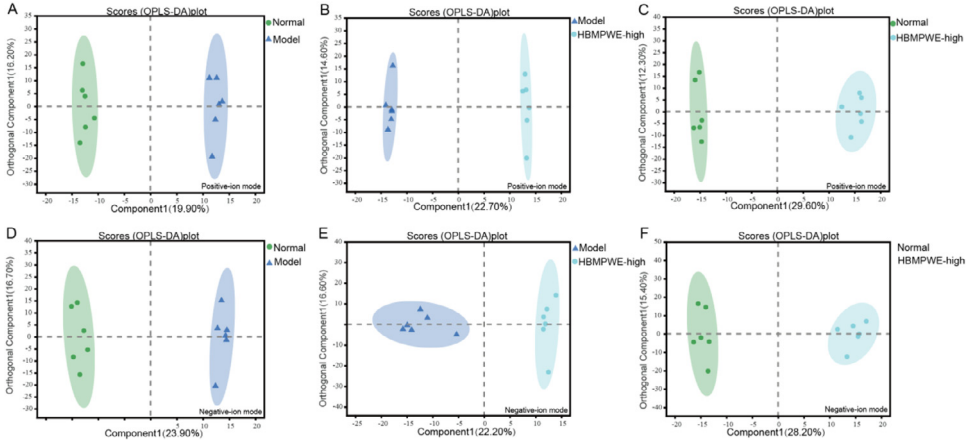

Fig. 1. OPLS-DA score plots in different groups under positive and negative-ion modes. (A) OPLS-DA score plots between normal and model in positive-ion mode. (B) OPLS-DA score plots between model and HBMPWE-high in positive-ion mode. (C) OPLS-DA score plots between normal and HBMPWE-high in positive-ion mode. (D) OPLS-DA score plots between normal and model in negative -ion mode. (E) OPLS-DA score plots between model and HBMPWE-high in negative -ion mode. (F) OPLS-DA score plots between normal and HBMPWE-high in negative -ion mode.

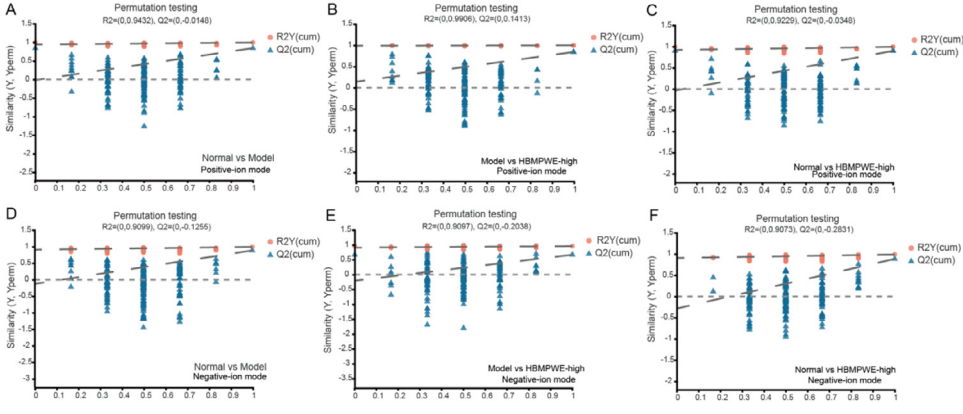

Fig. 2. OPLS-DA replacement test of liver metabolite in different groups under positive and negative-ion modes. (A) OPLS-DA replacement test of liver metabolite between normal and model in positive-ion mode. (B) OPLS-DA replacement test of liver metabolite between model and HBMPWE-high in positive-ion mode. (C) OPLS-DA replacement test of liver metabolite between normal and HBMPWE-high in positive-ion mode. (D) OPLS-DA replacement test of liver metabolite between normal and model in negative-ion mode. (E) OPLS-DA replacement test of liver metabolite between model and HBMPWE-high in negative-ion mode. (F) OPLS-DA replacement test of liver metabolite between normal and HBMPWE-high in negative-ion mode.

groups (Fig. 1) [2]. There were obviously separated clusters between two groups (model group versus normal group, HBMPWE-high group versus model group, HBMPWE-high group *vs* normal group). The validation parameters R^2Y values were close to 1, and Q^2 values were greater than 0.5 (Fig. 2 and Table 1), indicating that model had good reliability and predictability [3]. Under the OPLS-DA model, differentially regulated lipid species were identified in model group versus normal group and HBMPWE-high group versus model group using following criteria: $P < 0.05$, VIP (variable importance in the projection) > 1. The metabolic set was created, and a Venn diagram was made to screen common differential metabolites (Fig. 3) [4].

Table 1
OPLS-DA model parameter.

	Positive-ion mode		Negative-ion mode	
Group	R^2Y	Q^2	R^2Y	Q^2
Model vs Normal	0.995	0.844	0.995	0.887
HBMPWE-high vs Model	0.999	0.841	0.959	0.669
HBMPWE-high vs Normal	0.995	0.900	0.990	0.885

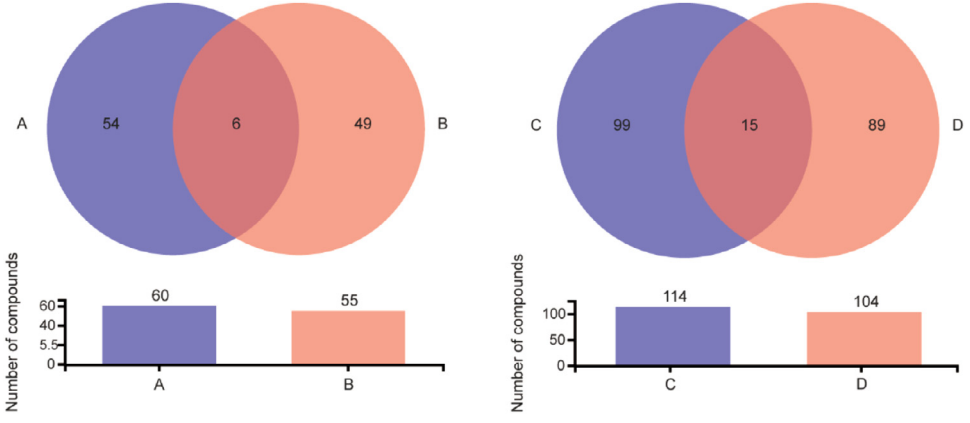

Fig. 3. Venn diagram of metabolic set. (A) The number of down-regulated metabolites in the model group, compared with the normal group; (B) The number of up-regulated metabolites in the HBMPWE-high group, compared with the model group; (C) The number of up-regulated metabolites in the model group, compared with the normal group; (D) The number of down-regulated metabolites in the HBMPWE-high group, compared with the model group.

1.2. Analysis of liver differential metabolites of HBMPWE on NAFLD

After the male golden hamsters were fed with HFFCD, the amount of 15 common differential metabolites increased significantly, and the amount of 6 common differential metabolites decreased significantly, compared with the normal group. In contrast, when the golden hamsters were fed with HFFCD were treated with HBMPWE, these common differential metabolites were reversed (Table 2). Among these 21 common differential metabolites, there are few studies on the biological functions of some metabolites, such as PE (16:1(9Z)/20:1(11Z)), PE (16:1(9Z)/20:2 (11Z,14Z)), PC (18:0/18:2 (9Z,12Z)), PE (20:3 (8Z,11Z,14Z)/0:0), and their biological functions and their role in NAFLD need to be further studied and verified. However, the biological functions of some metabolites have been reported [5]. The glucose ceramide is a metabolite of ceramide, and its accumulation will lead to metabolic disorder [6–8]. In addition, the glycerol-3-phosphate generates phosphatidylic acid after continuous acylation (PA), which generates diacylglycerol (DG) after further dephosphorylation. The DG synthesizes triglyceride (TG) is the storage form of fatty acids [9,10]. Other studies have shown that lipid peroxidation can be caused by the disorder of fatty acid metabolism and the decrease of the content of 1-palmitoylglycero phosphoinositol [11–13]. These reports are consistent with this research results, the golden hamsters in the model group caused lipid metabolism disorder after feeding HFFCD, the amount of glucosylceramide (d18:1/16:0), glycorol-3-phosphate, and DG (18:0/18:2(9z,12z)/0:0)) in the liver increased significantly, and the amount of 1-palmitoyllycero phosphoinositol decreased significantly. After the treatment of HBMPWE, these abnormally increased or decreased differential metabolites could be reversed, which was close to the expression level of normal group. NAFLD severity and treatment outcome can be quantified by differential metabolite analysis.

Table 2
Common differential metabolites in each group.

Mode	Metabolite	M/Z	Adducts	Formula	RT/min	Model vs Normal	TMPWE-high vs Model	HMDB ID
Pos	Glucosylceramide (d18:1/16:0)	722.5552	M+H-H_2O, M+H, M+Na	$C_{40}H_{77}NO_8$	10.62	↑###	↓*	HMDB0004971
Pos	PE(16:1(9Z)/20:1(11Z))	744.5550	M+H, M+2Na-H, M+Na	$C_{41}H_{78}NO_8P$	10.99	↑##	↓***	HMDB0008966
Pos	PE(16:1(9Z)/20:2(11Z,14Z))	742.5393	M+H, M+Na, M+2Na-H	$C_{41}H_{76}NO_8P$	10.55	↑#	↓***	HMDB0008967
Pos	PE(P-18:0/20:4(6E,8Z,11Z,14Z)(5OH[S]))	768.5550	M+H, M+Na	$C_{43}H_{78}NO_8P$	10.85	↑#	↓*	LMGP20020013
Pos	PC(18:0/18:2(9Z,12Z))	786.6019	M+H, M+Na	$C_{44}H_{84}NO_8P$	10.81	↑#	↓***	HMDB0008039
Pos	PE(20:3(8Z,11Z,14Z)/0:0)	504.3093	M+H-H_2O, M+H	$C_{25}H_{46}NO_7P$	8.06	↑#	↓**	LMGP02050022
Pos	DG(18:0/18:2(9Z,12Z)/0:0)	603.5356	M+H-H_2O	$C_{39}H_{72}O_5$	10.99	↑##	↓**	HMDB0007161
Pos	C16 Sphingosine	272.2586	M+H	$C_{16}H_{33}NO_2$	7.84	↑##	↓*	LMSP01040008
Pos	S-(2-carboxypropyl)-Cysteamine	164.0741	M+H	$C_6H_{13}NO_2S$	0.99	↑##	↓**	HMDB0002169
Pos	Tetrahydrodipicolinate	154.0499	M+H-H_2O	$C_7H_9NO_4$	0.86	↑###	↓*	HMDB0012289
Neg	Glycerol 3-phosphate	171.0057	M-H, M+K-2H, M-H_2O-H	$C_3H_9O_6P$	0.58	↑#	↓*	HMDB0000126
Neg	LysoPC(20:2(11Z,14Z))	592.3636	M+Cl, M+FA-H	$C_{28}H_{54}NO_7P$	8.37	↑#	↓**	HMDB0010392
Neg	LysoPE(20:3(11Z,14Z,17Z)/0:0)	502.2951	M-H	$C_{25}H_{46}NO_7P$	8.05	↑#	↓**	HMDB0011514
Neg	LysoPE(20:1(11Z)/0:0)	506.3261	M-H	$C_{25}H_{50}NO_7P$	8.94	↑##	↓**	HMDB0011512
Neg	LysoPE(20:2(11Z,14Z)/0:0)	504.3111	M-H	$C_{25}H_{48}NO_7P$	8.47	↑#	↓**	HMDB0011513
Pos	1-Palmitoylglycerophosphoinositol	555.2939	M+H-2H_2O, M+2Na-H, M+Na, M+H-H_2O	$C_{25}H_{49}O_{12}P$	8.11	↓##	↑***	HMDB0061695
Pos	PE-NMe2(16:0/22:6(4Z,7Z,10Z,13Z,16Z,19Z))	814.5369	M+H, M+Na	$C_{45}H_{78}NO_8P$	10.07	↓##	↑*	HMDB0113959
Pos	13-Tetradecene-1,3-diyne-6,7-diol	482.3251	2M+ACN+H	$C_{14}H_{20}O_2$	7.63	↓##	↑**	HMDB0039539
Neg	LysoPC(22:6(4Z,7Z,10Z,13Z,16Z,19Z))	612.3322	M+Cl, M+FA-H	$C_{30}H_{50}NO_7P$	7.54	↓#	↑*	HMDB0010404
Neg	LysoPC(22:4(7Z,10Z,13Z,16Z))	616.3631	M+FA-H	$C_{30}H_{54}NO_7P$	8.19	↓##	↑***	HMDB0010401
Neg	LysoPE(0:0/22:5(7Z,10Z,13Z,16Z,19Z))	572.3005	M+FA-H	$C_{27}H_{46}NO_7P$	8.24	↓##	↑*	HMDB0011495

# $P < 0.05$
$P < 0.01$
$P < 0.001$, *vs* the normal group.
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$, *vs* the model group.

2. Experimental Design, Materials and Methods

2.1. Chemicals and regents

Solvents (methanol, acetonitrile, formic acid, isopropanol; chromatographic grade), chemical reagents, and biological reagents were obtained from ThermoFisher Scientific (Shanghai, China).

2.2. Plant materials, extract preparation

HBMPW was provided by Tibet Yuewang Medicine Diagnosis Ecological Tibetan Medicine Science and Technology Co., Ltd. (Lot Number: 20190408, Lhasa, China). HBMPW powder was soaked in 70% EtOH for 12 h and added into percolation tube layer by layer. The percolation extract was placed in a large rotary evaporator for vacuum concentration to obtain a dry extract [14].

2.3. Liver metabolomic analysis

Male golden hamsters (110−130 g, SPF) were divided into three groups ($n = 8$ per group): Normal, NAFLD model, and NAFLD+HBMPWE groups, NAFLD model, and NAFLD+HBMPWE groups were fed with HFFCD for six weeks. From the third week of feeding HFFCD, the animals in NAFLD+HBMPWE group received 168 mg/kg/d HBMPWE by intragastric administration. After 4 weeks of continuous administration, all the male golden hamsters were fasted but given water for 12 h. Subsequently, the male golden hamsters were anesthetized and sacrificed to obtain the livers, which were flash-frozen in liquid nitrogen and stored at −80 °C for further metabolomics analysis [15].

Liver samples were extracted using methanol and water with homogenization. The mixture was allowed to settle at −20 °C and treated by high throughput tissue crusher Wonbio-96c (Shanghai Wanbo Biotechnology Co., Ltd., China) at 50 Hz for 6 min, then followed by vortex for 30 s and ultrasound at 40 kHz for 30 min at 5 °C. The samples were placed at −20 °C for 30 min to precipitate proteins. After centrifugation at 13,000 g at 4 °C for 15 min, the supernatant was carefully transferred to sample vials for LC-MS/MS analysis. The mass spectrometric data was collected by a Thermo UHPLC-Q Exactive Mass Spectrometer equipped with an electrospray ionization (ESI) source operating in either positive or negative ion mode.

2.4. Statistical analysis

The original data from UPLC-QTOF/MS was processed by Progenesis QI (Waters Corporation, Milford, USA). These data were analyzed by the platform of Majorbio Cloud Platform (www.majorbio.com) for principal component analysis (PCA) and orthogonal partial least squares discriminant analysis (OPLS-DA). The differential metabolites were selected by using the VIP value and the P value (VIP $>$ 1.0 and $P < 0.05$) [16].

Ethics Statement

The experiments with male golden hamsters complied with the National Institutes of Health guide for the care and use of Laboratory animals (NIH Publications No.8023, revised 1978, https://nihpublications.od.nih.gov/).

Declaration of Competing Interest

All authors have no conflicts of interest to declare.

CRediT Author Statement

Mei-Ning Zhu: Data curation, Investigation, Supervision, Writing – original draft; **Cui-Zhu Zhao:** Data curation, Investigation, Supervision, Writing – original draft; **Chong-Zhi Wang:** Writing – review & editing, Methodology; **Jian-Bo Rao:** Conceptualization, Visualization; **Yong-Wei Qiu:** Conceptualization, Visualization; **Yan-Ping Gao:** Visualization, Investigation, Software, Validation; **Xiao-Yun Wang:** Visualization, Investigation, Software, Validation; **Ya-Mei Zhang:** Visualization, Investigation, Software, Validation; **Guang Wu:** Software, Data curation; **Jie Chen:** Software, Data curation; **Qin-Ge Ma:** Formal analysis, Writing – review & editing; **Guo-Yue Zhong:** Formal analysis, Writing – review & editing; **Rong-Rui Wei:** Formal analysis, Writing – review & editing.

Acknowledgments

This work was supported by the Science and Technology Project of Jiangxi Health Commission (202211413), the Research Project on the Construction of Information Sharing Platform for Tibetan, Mongolian, Uygur Ethnic Medicine Resources, Variety Systematization, and Breeding Protection Technology (2019YFC1712300), the Special Fund for Graduate Innovation of Jiangxi University of Traditional Chinese Medicine (JZYC21S75), and the State Scholarship Fund of China Scholarship Council (202008360033).

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.dib.2021.107773.

References

[1] C.Z. Zhao, W. Jiang, Y.Y. Zhu, C.Z. Wang, W.H. Zhong, G. Wu, J. Chen, M.N. Zhu, Q.L. Wu, X.L. Du, Y.Y. Luo, M. Li, H.L. Wang, H. Zhao, Q.G. Ma, G.Y. Zhong, R.R. Wei, Highland barley *Monascus purpureus* went extract ameliorates high-fat, high-fructose, high-cholesterol diet induced nonalcoholic fatty liver disease by regulating lipid metabolism in golden hamsters, J. Ethnopharmacol. (2021), doi:10.1016/j.jep.2021.114922.
[2] B. Worley, R. Powers, PCA as a practical indicator of OPLS-DA model reliability, Curr. Metabol. 4 (2016) 97–103, doi:10.2174/2213235X04666160613122429.
[3] B. Worley, R. Powers, Multivariate analysis in metabolomics, Curr. Metabol. 1 (2013) 92–107, doi:10.2174/2213235X11301010092.
[4] S.C. Kalhan, L.N. Guo, J. Edmison, S. Dasarathy, A.J. McCullough, R.W. Hanson, M. Milburn, Plasma metabolomic profile in nonalcoholic fatty liver disease, Metabolism 60 (2011) 404–413, doi:10.1016/j.metabol.2010.03.006.
[5] C.D. Fuchs, T. Claudel, M. Trauner, Role of metabolic lipases and lipolyticmetabolites in the pathogenesis of NAFLD, Trends Endocrinol. Metab. 25 (2014) 576–585, doi:10.1016/j.tem.2014.08.001.
[6] Y. Guri, M. Colombi, E. Dazert, S.K. Hindupur, J. Roszik, S. Moes, P. Jenoe, M.H. Heim, I. Riezman, H. Riezman, M.N. Hall, mTORC2 promotes tumorigenesis via lipid synthesis, Cancer Cell 32 (2017) 807–823, doi:10.1016/j.ccell.2017.11.011.
[7] P.K. Luukkonen, Y. Zhou, S. Sädevirta, M. Leivonen, J. Arola, M. Orešič, T. Hyötyläinen, H. Yki-järvinen, Hepatic ceramides dissociate steatosis and insulin resistance in patients with non-alcoholic fatty liver disease, J. Hepatol. 64 (2016) 1167–1175, doi:10.1016/j.jhep.2016.01.002.
[8] J.A. Chavez, M.M. Siddique, S.T. Wang, J.H. Ching, J.A. Shayman, A. Scott, S.A. Summers, Ceramides and glucosylceramides are independent antagonists of insulin signaling, J. Biol. Chem. 289 (2014) 723–734, doi:10.1074/jbc.m113.522847.
[9] R.J. Perry, V.T. Samuel, K.F. Petersen, G.I. Shulman, The role of hepatic lipids in hepatic insulin resistance and type 2 diabetes, Nature 510 (2014) 84–91, doi:10.1038/nature13478.
[10] J. Lee, N.D. Ridgway, Substrate channeling in the glycerol-3-phosphate pathway regulates the synthesis, storage and secretion of glycerolipids, BBA Mol. Cell Biol. Lipids 1865 (2020) 158438, doi:10.1016/j.bbalip.2019.03.010.

[11] H. Wu, Y. Chen, Z.G. Li, X.H. Liu, Untargeted metabolomics profiles delineate metabolic alterations in mouse plasma during lung carcinoma development using UPLC/QTOF/MS in MSE mode, R. Soc. Open Sci. 5 (2018) 181143, doi:10.1098/rsos.181143.
[12] J. Li, C. Guo, J. Wu, Fucoidan: biological activity in liver diseases, Am. J. Chin. Med. 48 (2020) 1617–1632, doi:10.1142/S0192415X20500809.
[13] I.C. Yen, J.C. Lin, Y. Chen, D.W. Tu, S.Y. Lee, *Antrodia cinnamomea* attenuates non-alcoholic steatohepatitis by suppressing NLRP3 inflammasome activation *in vitro* and *in vivo*, Am. J. Chin. Med. 48 (2020) 1859–1874, doi:10.1142/S0192415X20500937.
[14] E. Ko, M.Y. Um, M. Choi, T. Han, I.H. Kim, S. Shin, *Cassia tora* seed improves pancreatic mitochondrial function leading to recovery of glucose metabolism, Am. J. Chin. Med. 48 (2020) 615–629, doi:10.1142/S0192415X20500317.
[15] M.X. Ge, W.X. Niu, J.F. Ren, S.Y. Cai, D.K. Yu, H.T. Liu, N. Zhang, Y.X. Zhang, Y.C. Wang, R.G. Shao, J.X. Wang, H.W. He, A novel ASBT inhibitor, IMB17-15, repressed nonalcoholic fatty liver disease development in high-fat diet-fed Syrian golden hamsters, Acta Pharmacol. Sin. 7 (2019) 895–907, doi:10.1038/s41401-018-0195-3.
[16] C. Liu, H. Wu, S.J. Liu, S.T. Chai, Q.X. Meng, Z.M. Zhou, Dynamic alterations in yak rumen bacteria community and metabolome characteristics in response to feed type, Front. Microbiol. 10 (2019) 1116, doi:10.3389/fmicb.2019.01116.