Data Article

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

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\textbf{A B S T R A C T}

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, 

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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.

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### 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
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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.

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 $R^2_Y$ 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].
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, 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 phosphatidylcholine 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-palmitoylglycerol 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-palmitoylglycerol 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.
| 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-H2O, M+H, M+Na              | C16H27NO8                        | 10.62  | ↑              | ↑↑↑                 | HMDB0004971          |
| Pos      | PE(16:1(9Z)/20:1(11Z))                                                      | 744.5550  | M+H, M+2Na-H, M+Na              | C20H29NO8P                       | 10.99  | ↑              | ↑↑                 | HMDB0008966          |
| Pos      | PE(16:1(9Z)/20:2(11Z,14Z))                                                  | 742.5393  | M+H, M+Na, M+2Na-H              | C20H29NO8P                       | 10.55  | ↑              | ↑↑↑                 | HMDB0008967          |
| Pos      | PE(P-18:0/20:4(6E,8Z,11Z,14Z)(5OH[S]))                                      | 768.5550  | M+H, M+Na                        | C24H34NO8P                       | 10.85  | ↑              | ↑↑↑                 | LMGP20020013         |
| Pos      | PC(18:0/18:2(9Z,12Z))                                                       | 786.6019  | M+H, M+Na                        | C24H34NO8P                       | 10.81  | ↑              | ↑↑↑                 | HMDB0008039          |
| Pos      | PE(20:3(8Z,11Z,14Z)(0:0)                                                    | 504.3093  | M+H-H2O, M+H                    | C18H30NO7P                       | 8.06   | ↑              | ↑                   | LMGP2050022          |
| Pos      | DG(18:0/18:2(9Z,12Z)(0:0)                                                   | 603.5356  | M+H-H2O                         | C18H30O5                         | 10.99  | ↑              | ↑                   | HMDB0007161          |
| Pos      | C16 Sphingosine                                                             | 272.2586  | M+H                             | C16H30NO7                        | 7.84   | ↑              | ↑                   |                      |
| Pos      | S-(2-carboxypropyl)-Cysteamine                                              | 164.0741  | M+H                             | C16H30O7S                        | 0.99   | ↑              | ↑                   | HMDB0002169          |
| Pos      | Tetrahydrodipicolinate                                                      | 154.0499  | M+H-H2O                         | C7H14NO4                         | 0.86   | ↑              | ↑                   | HMDB0012289          |
| Neg      | Glycerol 3-phosphate                                                        | 171.0057  | M-H, M+K-2H, M-H2O-H             | C18H30P                          | 0.58   | ↑              | ↑                   | HMDB000126           |
| Neg      | LysoPC(20:2(11Z,14Z))                                                       | 592.3636  | M+Cl, M+FA-H                    | C20H30NO7P                       | 8.37   | ↑              | ↑                   | HMDB0010392          |
| Neg      | LysoPE(20:3(11Z,14Z,17Z)(0:0)                                               | 502.2951  | M-H                             | C20H30NO7P                       | 8.05   | ↑              | ↑                   | HMDB0011514          |
| Neg      | LysoPE(20:1(11Z))(0:0)                                                      | 506.3261  | M-H                             | C20H30NO7P                       | 8.94   | ↑              | ↑                   | HMDB0011512          |
| Neg      | LysoPE(20:2(11Z,14Z)(0:0)                                                  | 504.3111  | M-H                             | C20H30NO7P                       | 8.47   | ↑              | ↑                   | HMDB0011513          |
| Pos      | 1-Palmitoylglycerophosphinositol                                            | 555.2939  | M+H-2H2O, M+2Na-H, M+Na, M+H-H2O| C24H40O12P                       | 8.11   | ↓              | ↓                   | HMDB0061695          |
| Pos      | PE-NMe2(16:0/22:6(4Z,7Z,10Z,13Z,16Z,19Z))                                   | 814.5369  | M+H, M+Na                        | C24H40NO8P                       | 10.07  | ↓              | ↑                   | HMDB0113959          |
| Pos      | 13-Tetradecene-1,3-diyne-6,7-diol                                           | 482.3251  | 2M+ACN-H                        | C16H34O2                         | 7.63   | ↓              | ↓                   | HMDB0039539          |
| Neg      | LysoPC(22:6(4Z,7Z,10Z,13Z,16Z,19Z))                                        | 612.3322  | M+Cl, M+FA-H                    | C26H40NO7P                       | 7.54   | ↓              | ↑                   | HMDB0010404          |
| Neg      | LysoPC(22:4(7Z,10Z,13Z,16Z))                                                | 616.3631  | M+FA-H                          | C26H40NO7P                       | 8.19   | ↓              | ↑                   | HMDB0010401          |
| Neg      | LysoPE(0:0/22:5(7Z,10Z,13Z,16Z,19Z))                                        | 572.3005  | M+FA-H                          | C27H46NO8P                       | 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.

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Supplementary materials

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

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