INTRODUCTION

Diabetes is a common chronic disorder and metabolic disease in which body cannot effectively control glucose homeostasis. Diabetes can lead to severe damage to the heart, eyes, kidneys, blood vessels, and nerves. According to the data of the International Diabetes Federation, it is estimated that 463 million people have diabetes in 2019 and this number is projected to reach 578 million by 2030 and 700 million by 2045 (Agrawal et al., 2011; Thomas et al., 2019). China, which is the most populous country, ranks number one with an estimate of 109.6 million adults with diabetes (Hu & Jia, 2018).

Type 2 diabetes mellitus (T2DM) is a widely prevalent form, classified by metabolic disorders with hyperglycemia, and considered as a main health concerned issue comprising higher morbidity and mortality. As a result, more and more studies focused on the development of antidiabetic medications (Bailey et al., 2016; Delzenne et al., 2015) and functional foods (Beidokhti & Jager, 2017; Martel et al., 2017;
Naveen & Baskaran, 2018), especially for the treatment of T2DM which accounts for about 90% of the diabetes and is preventable and can be cured by medication and health lifestyle (Thomas et al., 2019).

Over the years, many types of traditional food treatments and natural remedies have been used to treat diabetes (Leiherer et al., 2013; Rudkowska, 2009); however, the validity and effectiveness of just a few of them have been evaluated. There is a traditional belief in Africa, Asia, and Middle East that regular consumption of camel milk may aid in prevention and control of diabetes, and epidemiologic study also reported a significant lower incidence of diabetes in people consuming camel milk than those did not have the habit in the same community (0.4% vs. 5.5%) (Agrawal, Budania, et al., 2007; Agrawal, Saran, et al., 2007). The hypoglycemic function of camel milk has been confirmed in streptozotocin-diabetic rats (Abdel-Salam & Al-Damegh, 2018; Al-Numair, Chandramohan, & Alsaif, 2011a, Agrawal, Saran, et al., 2007). It also reported that the hydrolysate of camel whey protein harbored insulin-like and whey protein hydrolysates may be the main ingredients in hypoglycemic effect of camel milk. Insulin-like was proved by the decreased insulin requirement in type 1 diabetic patients (Agrawal et al., 2011; Agrawal et al., 2003a, 2003b; Agrawal, Budania, et al., 2007; Agrawal, Saran, et al., 2007; Mohamad et al., 2009), and T2DM (Agrawal et al., 2011; Ejahed et al., 2015; Korish, 2014; Wang et al., 2009) characterized by reduced fasting glucose and glycosylated hemoglobin. Insulin-like and whey protein hydrolysates may be the main ingredients in hypoglycemic effect of camel milk.

MATERIALS AND METHODS

2.1 Participants and study design

We performed a double-blind, randomized, placebo-controlled pilot study over 4 weeks to investigate the effects of camel milk in type 2 diabetic patients. Type 2 diabetic patients were recruited from subjects attending to the clinic of Beijing Chinese Medicine Hospital Pinggu Hospital. Inclusion criteria were age 35–68 years, absence of gastrointestinal disease, and willingness to abstain from intake of other milk, probiotic food, and fermented milk products during the study but otherwise stick to previous eating habits. Exclusion criteria were pregnancy or lactating in women, cancer, allergy, or intolerance to camel milk or cow milk.

After run-in phase, participants were categorized into two groups and received camel milk powder and cow milk powder (placebo control), respectively, for 4 consecutive weeks (two times per day, 10 g each time). All the samples were provided by Sanhe Fucheng Biological Technology Co. Ltd (Langfang, China). Nutrient content of the camel milk and cow milk powder was shown in Table 1. Blood glucose, lipid profile, serum cytokines content, and collection of fecal samples were performed at the end of the run-in phase and at end of the study. During the study, participants underwent interviews regarding adverse effects, symptoms, or changes in quality of life every week. The study was approved by the local ethics committee of China Agricultural University (CAUHR-2018026) and registered at ClinicalTrials.gov (NCT04296825). All participants provided written informed consent.

2.2 Blood sample collection and plasma glucose measurements

Human peripheral blood was collected in Vacutainer tubes and (Cat #368921, BD Biosciences) and Vacutainer heparin tubes (Cat #367886, BD Biosciences), respectively. Blood samples were centrifuged at 1,500 × g for 30 min at room temperature and samples in for fasting glycemia, 2-hr postprandial glycemia, insulin, and lipid measurements within 1 hr after blood collection, and serum samples in Vacutainer heparin tubes were carefully removed, aliquoted, snap-frozen in liquid nitrogen, and stored in aliquots at −80°C until further analysis.

2.3 Biochemical indexes measurements

Serum insulin were measured using the Architect i2000SRanalyzer (Abbott Diagnostics), and serum content of glucose, uric acid, total cholesterol (TC), total triglyceride (TG), high-density lipoprotein

| Table 1 | Nutrient content of the camel milk and cow milk powder |
|----------|-----------------|-----------------|
| Variables | Camel milk powder | Cow milk powder |
| Fat (%)   | 32.6            | 28.2            |
| Total protein (%) | 30.3    | 25.1            |
| -Whey protein (%) | 8.0      | 4.5             |
| -Casein (%) | 20.8         | 20.2            |
| Carbohydrate (%) | 37.1     | 42.0            |
| H₂O (%)   | 4.3             | 2.1             |
cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were measured using a Roche cobas® e 411 analyzer (Roche) according to the manufacturers’ protocol by the certified core clinical laboratory at the Beijing Chinese Medicine Hospital Pinggu Hospital. To evaluate insulin resistance, the HOMA-IR formulation was applied (Wallace et al., 2004) according to the following equation: HOMA-IR = Fasting blood glucose (mmol/L) × fasting insulin concentration (μU/ml) ÷ 18.405.

2.4 | Gut hormones and cytokines assays

For determination of appetitive hormones (amylin [active], ghrelin [active], glucagon-likepeptide 1 [GLP-1, active], and pancreatic polypeptide [PP]), inflammation cytokines (TNF-α, IL-6, MCP-1), myokines (FGF-21, irisin, osteocrin, osteonectin), and adipokines (adiponectin, resistin, lipocalin-2, adipin), human cytokine immunobead panels (Milliplex, Millipore, Cat # MHEMAG-34K, HMYOMAG-56K, and HADK1MAG-61K-04) coupled with a multiplex assay (involving xMAP technology, Luminex) were used according to the manufacturers’ protocol.

2.5 | Gut microbiota analysis

A total of 54 cecal samples were collected at the end of the run-in phase and at end of the study. All samples were collected into sterile tubes containing RNA later and stored at −20°C. DNA was extracted from fecal samples using the phenol–chloroform extraction method (Köchl, 2005) and quantified using a Nano Drop spectrophotometer (OneC, Thermo Fisher Scientific) and stored at −80°C until further analysis.

DNA was amplified using the universal primers 338F (5′- ACTCCTACGGGAGGCAGCAG-3′) and 806R (5′- GGACTACHVGGGTWTCTAAT-3′) to target the V3-V4 region of bacterial 16S rRNA. The resulting 468-bp-sized products were assessed, quantified, pooled, and sequenced on an Illumina Miseq PE300 platform (Illumina) at Shanghai Majorbio Bio-pharm Technology Co. Ltd. (Shanghai, China) using a paired-end sequencing strategy. Raw data were spiced, filtered, and then used to select the operational taxonomic units (OTUs) with USEARCH software (version 7.0) and a default cutoff of 97% sequence similarity.

Operational taxonomic units were further subjected to the Ribosomal Database Project classifier software for taxonomic identification with an 80% confidence threshold at the phylum, class, order, family, genus, and species levels. Further analysis such as abundance and pairwise comparison of gut microbiome was analyzed on the platform of Majorbio I-Sanger Cloud Platform (www.i-sanger.com). Pairwise comparison of the microbiome communities of the different groups was performed at genus levels.

2.6 | Statistical analysis

Statistical significance was determined by paired two-tailed Student's test analysis for end and baseline comparison and one-way ANOVA tests for differences between treatments using GraphPad Prism version 7.0 software (San Diego, CA, USA). \( p < .05 \) was considered statistically significant.
In this study, there was a decrease in fasting blood glucose (p < .05) in patients intervened with camel milk (group C). It was reported that one of the potential molecular basis of the antidiabetic properties of camel milk was related to the higher insulin-like amino acid composition of whey proteins (Beg et al., 1986). Although only few quantitative studies about the insulin content of camel milk were reported (Wernery et al., 2006), but evidence for the significant reduction in insulin dose required in type I diabetic patients still to be brought (Agrawal et al., 2005, 2011; Agrawal, Budania, et al., 2007; Agrawal, Saran, et al., 2007; Agrawal et al., 2003a, 2003b; Mohamad et al., 2009; Wang et al., 2009). In this study, serum content of insulin was not affected (Figure 2e p > .05) Probiotics intervention did not improve the insulin resistance (HOMA-IR) of the patients (Figure 2f, p > .05).

Our result is in accordance with previous studies in type I diabetic patients (Agrawal et al., 2005, 2011; Agrawal, Budania, et al., 2007; Agrawal, Saran, et al., 2007; Agrawal et al., 2003a, 2003b; Mohamad et al., 2009; Wang et al., 2009). It was reported that the antidiabetic activity of camel milk is mediated by an insulin-like effects on beta-cells of the pancreas (Abdulrahman et al., 2016), which may lead to the different results between type I and type II diabetes mellitus. As expected, the changes in plasma insulin were inconsistency in previous type II diabetes mellitus (Agrawal et al., 2003a, 2003b; Brezovecki et al., 2015).

3.3 Changes in lipid profile and cardiovascular risk

Total cholesterol, total triglyceride, and the indicators of vascular risk (LDL/HDL cholesterol ratio and TC/HDL-C) were shown in Figure 3, and at baseline and postintervention, there were both no significant differences between groups (p > .05). After a 4-week intervention, the total cholesterol of patients in group C decreased (p = .0225, Figure 3a), although there were no significant effects in the total triglyceride, patients in the placebo group (Group P) had a significantly higher total triglyceride (p = .0264, Figure 3b). There were no significant changes in LDL-C/HDL-C before and after the interventions (p > .05, Figure 3c), but an nonsignificant decrease in the atherogenic index in group C (TC/HDL-C, Figure 3d).

The effect of camel milk on lipid profile was mainly studied in diabetic rats (Khan et al., 2013; Korish, 2014; Wang et al., 2009), which was consistent with our findings that there was a decrease in total cholesterol after the intervention in group C (p = .0225, Figure 3a). However, in the limited studies in type II diabetic patients, one study (Wang et al., 2009) was in accordance with ours while another one reported that there were no changes in lipid profile (Ejtahed et al., 2015). Furthermore, there was also a decrease in the ratio of total cholesterol and HDL-C (Figure 3d), indicating a decreased vascular risk by camel milk. It was interested to see the significant increase in total triglyceride in group P, the similar effect was found in alloxan-induced diabetic dogs with a significant increase total cholesterol when treated with cow milk for 5 weeks (Sbou, Djegham, et al., 2010; Sbou, Khorchani, et al., 2010).

3.4 Changes in inflammatory cytokines

Serum contents of inflammatory markers (TNF-α, IL-6, MCP-1) in patients before and after 4-week intervention were shown in Figure 4. After the 4-week intervention, there were an nonsignificant decrease in the content of IL-6 in group C, which was significant in patients in group P (p = .0348, Figure 4b). Furthermore, the decrease
in TNF-α (Figure 4a) and MCP-1 (Figure 4c) was also more obvious in group P than in group C.

It was reported that the antidiabetic mechanism of camel milk may be also related to its greater antioxidant and immunomodulatory activities than bovine and other whey proteins (Ayoub et al., 2018; Badr, Ramadan, et al., 2017). Meanwhile, it is widely accepted that defects in both redox and the immune systems resulted in the damage and the destruction of the pancreatic beta-cells and linked to diabetes mellitus (Newsholme et al., 2019). And the treatment of diabetic rats with camel milk or camel milk whey proteins was shown to reduce the proinflammatory IL-1β, IL-6, and TNF-α (Badr, Sayed, et al., 2017; Korish, 2014; Mahmoud et al., 2016). Here in our study, we only found a significant decrease in IL-6 ($p = .0103$, Figure 4b).

### 3.5 Changes in adipokines and myokines profile

Serum contents of adipokines (adiponectin, resistin, lipocalin-2, adipsin) and myokines (FGF-21, irisin, osteocrin, osteonectin)
in patients before and after 4-week intervention were shown in Figure 5. There was only a significant decrease in serum content of resistin \((p = .0388, \text{Figure } 5b)\) and lipocalin-2 \((p = .0435, \text{Figure } 5c)\) in patients of group C. Interestingly, a significant decrease in serum content of osteonectin was found in group P \((p = .0091, \text{Figure } 5h)\).

Recent evidence has identified skeletal muscle and adipocytes as secretory organs, which communicate with each other to regulate energy homeostasis and insulin sensitivity through the cytokines called myokines and adipokines (Galic et al., 2010; Pedersen & Febbraio, 2012), respectively. As we can see from Figure 5, intervention with camel milk significantly decreased the content of resistin and lipocalin-2 levels, which was reported to be good for the improving of diabetes. Elevated serum lipocalin-2 is closely and independently associated with impaired glucose regulation and type 2 diabetes in Chinese people (Huang et al., 2012), and the lipocalin-2 deficiency attenuates insulin resistance associated with obesity in mice (Law et al., 2010). Resistin promotes insulin resistance in mice, whereas whether it does so in humans is unclear (Heilbronn et al., 2004; Steppan et al., 2001) because it was synthesized in adipocytes in mice whereas in humans it is generated by macrophages and monocytes, but not adipocytes (Oh et al., 2017). According to our results, there was a significant decrease in resistin after the intervention accompanied by the decreased fasting blood glucose, and it maybe also positively correlated with the hyperglycemia. There were no significant changes in myokines when compared serum contents before and after the intervention \((p > .05)\); however, there was a significant elevation in osteocin in patients intervened with camel milk than those took cow milk (Figure 5g, C-W4 vs. P-W4, \(p = .0185\)), which is an indicator for the improvement in skeletal muscle (Subbotina et al., 2015). Furthermore, intervention with cow milk significantly decreased serum content of osteonectin \((p = .0091, \text{Figure } 5h)\), which is concerned with normal remodeling and maintenance of bone mass (Zhu et al., 2019). The decrease may be related to the unimproved hyperglycemia.

### 3.6 Changes in appetitive hormones

Serum contents of appetitive hormones (amylin, ghrelin, GLP-1, PP, PYY) in patients before and after 4-week intervention were shown in Figure 6. There were no significant changes before and after the intervention with either camel milk or cow milk. However, it was note-worthy to see that there was a significant higher concentration of amylin (Figure 6a, \(p = .0469\)) and GLP-1 (Figure 6c, \(p = .0538\)) in the serum of patients intervened with camel milk than those intervened with cow milk.

Camel whey protein was also found to have an inhibitory effect on dipeptidyl peptidase IV (Ayoub et al., 2018; Mudgil et al., 2018),
the key enzyme that cleaves and inactivates incretins such as GLP-1 and then indirectly control the secretion of insulin (Andersen et al., 2018). As we can see the changes in appetite hormones from Figure 6, there was only a nonsignificant increase of GLP-1 and PP after the intervention of camel milk (Group P, placebo) and camel milk (Group C).

Although the elevation of GLP-1 was nonsignificant before and after the intervention, the increase was obvious when compared patients in different group after the intervention (C-W4 vs. P-W4, \( p = .0538 \)). Amylin, a hormone colocalized, copackaged, and cosecreted with insulin from adult pancreatic islet \( \beta \) cells, was reported to be deficient in diabetic patients (Schmitz et al., 2004). Although the increase in amylin was nonsignificant before and after the intervention with camel milk (C-W4 vs. C-W0, \( p > .05 \)), its content in patients supplemented with camel milk for 4 weeks was significantly higher than patients supplemented with cow milk (Figure 6a, C-W4 vs. P-W4, \( p = .0469 \)).

3.7 | Changes in gut microbiota

The different gut bacteria at the genus level before and after the intervention or between different groups analyzed by two-tailed
FIGURE 5  Serum contents of adipokines and myokines in each group before (W0) and after (W4) the intervention. (a) adiponectin; (b) resistin; (c) lipocalin-2; (d) adipsin; (e) FGF-21; (f) irisin; (g) osteocrin; (h) osteonectin in patients intervened with cow milk (Group P, placebo) and camel milk (Group C)
Student’s t test were shown in Figure 7. The community abundances on genus levels of gut microbiome composition were shown in Figure 7a. The microflora composition of the group C and P was very similar before the intervention began. However, after probiotics intervention, there were some differences in flora composition. In order to further clarify the specific differences, we performed pairwise comparison of the microbiome communities of the four groups at genus levels. There were no different genera before and after the intervention in group C. But for group P, there was a significant increase in *Phascolarctobacterium* (*p* = .04225) and a decrease in unclassified_f__Micrococaceae (*p* = .02046, Figure 7a). When compared gut microbiota between patients in group C and P after the 4-week intervention, there was a significant increase in the relative abundance of *Clostridium_sensu_stricto_1* and *Eubacterium_eligens_group* in group C (Figure 7b, *p* < .05, C-W0 vs. P-W4), which was not due to the difference between individuals before the intervention (Figure 7c *p* > .05, C-W0 vs. P-W0).

More and more evidence suggests a close relationship between gut microbiota and diabetes (Tilg & Moschen, 2014). *Phascolarctobacterium* can produce short-chain fatty acids, including acetate, propionate, and butyrate, and can be dramatically increased by berberine and metformin (Zhang et al., 2015). Fermented cow milk was reported to significantly enrich the relative abundance of *Phascolarctobacterium* (Rettedal et al., 2019), and there was no

**Figure 6** Serum contents of appetitive hormones in each group before (W0) and after (W4) the intervention. (a) amylin; (b) ghrelin; (c) GLP-1; (d) PP; (e) PYY in patients intervened with cow milk (Group P, placebo) and camel milk (Group C).
significant difference between the two groups both before and after the intervention (Figure S1, \(p > .05\)), so this was not the reason for the different effect in glycemic index between camel milk and cow milk. Clostridium_sensu_stricto_1 is one of the most important anaerobic, fermenting bacteria in human gut which may metabolize various compounds such as carbohydrates and amino acids (Wiegel et al., 2006). There was a significant enrichment in this genus in patients of group C (Figure 7b); however, its proportion decreased significantly when compared gut microbiota before and after the intervention. In addition, the other genus changed significantly between the different intervention, and [Eubacterium]_eligens_group was found at low levels in individuals with type 2 diabetes mellitus (Karlsson et al., 2013; Viciani, 2017). We can also see from Figure 7c and b that its relative abundance decreased after the intervention with both milks. Interestingly, our study also found that there was a significant lower abundance of [Eubacterium]_eligens_group in patients intervened with camel milk which exhibited an improvement in the symptom of diabetes.

**4 | CONCLUSION**

In conclusion, the present work demonstrates that camel milk powder can also exhibited an antidiabetic activity in type 2 diabetic patients. After a 4-week intervention with 10 g of camel milk powder twice a day, there was a decrease in fasting blood glucose and 2-hr postprandial blood glucose, as well as serum content of total cholesterol. Meanwhile, supplement with camel milk powder also significantly decreased serum content of resistin and lipocalin-2, adipokines which was reported to be positively associated with diabeties. Patients intervened with camel milk also exhibited a significant higher content of amylin and GLP-1 than patients intervened with cow milk. These results in combination suggested that camel milk powder can be used as part of the treatment of type 2 diabetes.

**INFORMED CONSENT**

Study procedure was explained for participants, and all participants provided written informed consent.

**ACKNOWLEDGMENTS**

This work was supported by the National Key Research and Development Program of China (2018YFD0400305), Scientific Research Project of Beijing Municipal Education Commission (KZ201910011014), and Beijing Municipal Science and Technology Commission Project (Z18110009318005). Additionally, we would like to thank all study participants and all the nurses in Beijing Chinese Medicine Hospital Pinggu Hospital for their assistance in helping in running the study visits and in processing of blood samples and in blood biochemical items analysis.

**CONFLICT OF INTEREST**

The authors declare no conflict of interest.

**AUTHOR CONTRIBUTIONS**

Yajie Zheng: Conceptualization (lead); Data curation (lead); Formal analysis (lead); Investigation (lead); Writing-original draft (lead). Fang Wu: Conceptualization (equal); Data curation (equal); Investigation (equal); Writing-original draft (equal); Writing-review & editing (equal). Ming Zhang: Data curation (equal); Investigation (equal); Writing-review & editing (equal). Bing Fang: Data curation (equal); Methodology (equal). Lijie Dong: Investigation (equal); Shaoyang Ge: Methodology (equal).

**ETHICAL APPROVAL**

The clinical experiment was registered in Clinical Trials Protocol Registration System (ClinicalTrials.gov Identifier: NCT04296825).
DATA AVAILABILITY STATEMENT
All authors confirm that the data supporting the findings of this study are available within the article.

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