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Schisandra chinensis Fructus (Bei Wuweizi), Rather Than *Schisandra sphenanthera* Fructus (Nan Wuweizi), Is Responsible for Producing the Qi-Invigorating Effects in the Five Visceral Organs

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Abstract

Schisandra chinensis Fructus (Bei Wuweizi) and *Schisandra sphenanthera* Fructus (Nan Wuweizi) are traditional Chinese herbs known for their tissue-protective and adaptogenic properties. This study investigates the Qi-invigorating effects of schisandrin A (Sch A), the primary active lignan in Nan Wuweizi, and compares it to our previous findings for schisandrin B (Sch B) from Bei Wuweizi. We measured mitochondrial ATP generation capacity (ATP-GC) and glutathione redox status in various tissues of Sch A-treated mice. Results showed that Sch A treatment increased mitochondrial ATP-GC in several tissues, particularly the liver, but was less effective than Sch B. Additionally, Sch A improved mitochondrial glutathione redox status in the brain, kidney, and lung, but did not significantly affect innate or adaptive immunity. These findings suggest that Sch A may provide limited tissue protection compared to Sch B, indicating that the Qi-invigorating effect on visceral organs attributed to Wuweizi likely pertains more to Bei Wuweizi. Our results support the traditional use of Bei Wuweizi in herbal formulations.

Keywords

Wuweizi, *Schisandra chinensis* Fructus, *Schisandra sphenanthera* Fructus

1. Introduction

Schisandra chinensis Fructus (Bei Wuweizi) and *Schisandra sphenanthera* Fruc-

tus (Nan Wuweizi) are two closely related Chinese herbs widely used in herbal formulas for their tissue-protective, astringent, and calming properties. Both herbs serve as adaptogens to enhance strength, stamina, and resilience to environmental and emotional stress [1]. The renowned herbalist Sun Si Miao in the Tang Dynasty noted that "Taking Wuweizi can invigorate the Qi in the five visceral organs, especially in summer." [2] However, it remains unclear whether this Qi-invigorating effect is attributed to Bei Wuweizi, Nan Wuweizi, or both. Since Qi-invigoration is associated with mitochondrial ATP production [3], our recent laboratory study demonstrated that schisandrin B (Sch B), the primary active lignan in Bei Wuweizi, enhances mitochondrial ATP generation in various tissues of mice [4]. As Qi-invigorating Chinese tonifying herbs invariably increase mitochondrial ATP generation [5], mitochondrial ATP generation capacity (ATP-GC) can be adopted as a primary endpoint to assess Qi-invigorating action in *ex vivo* and *in vitro* experiments [5] [6]. In this study, we aimed to investigate whether schisandrin A (Sch A), the main active lignan in Nan Wuweizi, produces a similar Qi-invigorating effect compared to Sch B. The mitochondrial glutathione redox status was measured in various tissues of Sch A-treated mice. The impact of Sch A treatment on innate and adaptive immunity was also assessed.

2. Materials and Methods

2.1. Reagents

Fetal bovine serum (FBS) was obtained from Life Technologies Corporation (Carlsbad, CA). Reduced glutathione (GSH), oxidized glutathione (GSSG), glutathione reductase (GR), adenosine triphosphate (ATP), adenosine diphosphate (ADP), RPMI-1640 medium (without phenol red), penicillin, streptomycin, MTT (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyl tetrazolium bromide), nitroblue tetrazolium chloride (NBT), lithium lactate, phenazine methosulfate (PMS), β-nicotinamide adenine dinucleotide hydrate (β-NAD), and lipopolysaccharide (LPS) were purchased from Sigma Chemical Co. (St. Louis, MO). The luciferase kit was obtained from Perkin Elmer (Boston, MA). Concanavalin A (Con A) was obtained from Santa Cruz Biotechnology Inc. (Santa Cruz, CA). Sch A was isolated from the petroleum extract of *Schisandra Sphenanthera* Fructus by silica gel column chromatography as previously described [7]. The purity of Sch A, as assessed by HPLC analysis, was larger than 95% (w/w).

2.2. Animal Care

Twelve adult female ICR mice were randomized into three groups: Control, Sch A (0.1/kg ×4), and Sch A (0.3 g/kg ×4). They were maintained under a 12-hour dark/light cycle at an ambient temperature of approximately 22˚C with ad libitum access to food and water. Experimental protocols were approved by the Research Practice Committee at the Hong Kong University of Science and Technology (AEP-2023-0062).

2.3. Animal Treatment

Animals were randomly divided into groups of four each. A previous study found that having four animals in each experimental group was optimal for detecting differences in endpoint measurements compared to the control group [4]. Preliminary studies also indicated that both male and female mice responded similarly to Sch A and Sch B treatments (data not shown). In the treatment groups, mice were intragastrically administered Sch A (suspended in water) at a daily dose of 0.3 or 1 g/kg for 3 days. Control animals only received water. The pharmacological doses used in this study are consistent with those in the previous study of Sch B. Long-term, low doses of Sch B (0.001 - 0.03 g/kg for 15 days, corresponding to human equivalent doses) were shown to protect against cerebral ischemia-reperfusion injury in rats [8]. Twenty-four hours after the last dose, animals were euthanized by cervical dislocation, and organs (liver, heart, brain, spleen, lung, and kidney) were harvested for biochemical analysis. Biochemical analyses were done in a non-biased manner with established protocols. A blinding approach was not adopted in dosing, tissue processing, and endpoint readouts.

2.4. Preparation of Mitochondrial Fractions and Measurement of Mitochondrial ATP-GC *Ex Vivo*

Tissue samples were excised and rinsed with ice-cold isolation buffer (0.25 M sucrose, 0.1 mM EDTA (free acid), 5 mM Tris base, pH 7.4). Mitochondrial fractions were prepared through differential centrifugation in the isolation buffer at 4˚C. A 10% (w/v) tissue homogenate was produced by homogenizing the minced tissue using a Teflon-glass homogenizer at 2000 - 4000 rpm for 10 - 20 complete strokes. The homogenate was centrifuged at 600 ×*g* for 10 min to remove nuclei and cell debris. The supernatant was further centrifuged at 9200 ×*g* for 30 min to pellet the mitochondria. The pellets were resuspended in 1 mL of respiration buffer (125 mM KCl, 20 mM MOPS, 10 mM Tris base, 5 mM EDTA (free acid), 2 mM KH_2PO_4, pH 7.2) to reconstitute the mitochondrial fractions [4].

The protein concentration of mitochondrial fractions was determined using the Bradford method [4].

2.5. ATP-GC

Before measuring mitochondrial ATP-GC, commercially sourced ADP was enzymatically treated to remove contaminated ATP molecules that could interfere with the accurate measurement of ATP produced by mitochondria. Mitochondrial fractions were prepared by adjusting to 1 mg protein/mL for brain, heart, kidney, and liver fractions, and 0.5 mg protein/mL for lung and spleen fractions. For the assay, 100 μL of these fractions were mixed with 100 μL of a substrate solution (containing 3 mM pyruvate and 3 mM malate) and 50 μL of pretreated ADP solution (15 mM for brain, heart, kidney and liver fractions; 7.5 mM for lung and spleen fractions). The mixtures were incubated for varying durations (0 - 15 min for brain, heart, kidney, and liver fractions; 0 - 5 min for lung and spleen

fractions) at 37˚C. The reaction was stopped by adding 50 μL of perchloric acid (30%, w/v), followed by centrifugation at 2150 ×*g* for 10 min at 4˚C. An aliquot (120 μL) of the supernatant was neutralized with 90 μL of 1.4 M $KHCO_3$, centrifuged again at 600 ×*g* for 10 min at 4˚C, and the supernatants were analyzed for ATP content using a bioluminescence assay. The mitochondrial ATP-GC for untreated animals was estimated by calculating the area under the curve (AUC1, using GraphPad Prism) of ATP generated (nmol/mg protein) plotted against time (0 - 15 or 0 - 5 min) and expressed in arbitrary units. For samples from Sch A-treated mice, AUC1 values for increasing incubation times (7.5 and 15 min or 3 and 5 min) were normalized to the mean control value from untreated mice and expressed as percent control. The area under the curve (AUC2, using GraphPad Prism) plotting percent control values (AUC1) against incubation time (7.5 - 15 min or 3 - 5 min) was calculated and expressed in arbitrary units. Data from Sch A-treated groups were expressed as a percentage of the untreated control using the formula: [AUC2 (Sch A-treated)/AUC2 (untreated)] × 100%. This two-step data processing aimed to minimize inter-animal and inter-assay variability under the experimental conditions, thus ensuring the experimental results are reproducible [4]. In addition, the *ex vivo* nature of mitochondrial ATP-GC measurement represents a reliable surrogate endpoint for an in vivo effect, and this is supported by the tissue protection afforded by Sch A or Sch B treatment [9] [10].

2.6. Measurement of Mitochondrial Glutathione Redox Status

Mitochondrial GSH and GSSG levels were determined enzymatically using DTNB and GR, as previously described [4]. An aliquot (210 μL) of the mitochondrial fraction was mixed with 90 μL of 10% SSA, and the supernatant was used to measure GSH and GSSG. The mitochondrial glutathione redox status was expressed as the GSH/GSSG ratio.

2.7. Isolation of Splenocytes

Splenic tissue from mice was processed by pressing through a stainless steel mesh sieve with a glass pestle in 25 mL RPMI-1640 medium to achieve a single-cell suspension. This suspension was centrifuged at 400 ×*g* for 5 min, and the resulting pellet was washed once with RPMI medium. Red blood cells in the pellet were lysed using 4.5 mL of water, and the lysis was halted by adding 0.5 mL of 10X Hank's Balanced Salt Solution (HBSS: 1.4 M NaCl, 53 mM KCl, 4.4 mM KH_2PO_4, 55.6 mM Glucose and 3.36 mM Na_2HPO_4) and 5 mL RPMI-medium supplemented with 5% (v/v) heat-inactivated (HI) FBS. After another round of centrifugation, the pellet was resuspended in RPMI-1640 medium with 5% HIFBS for cell counting using 0.4% trypan blue. Finally, the splenocytes were diluted to a final concentration of 1×10^7 cells/mL in RPMI-1640 medium supplemented with 5% HIFBS to serve as effector cells for NK cell activity assays. For the Con A/LPS-induced splenocyte proliferation assay, aliquots of cells were prepared at a final concentration of 5×10^6 cells/mL in RPMI-1640 medium supplemented with 10%

HIFBS [4].

2.8. NK Cell Activity Assay

YAC-1 cells, used as target cells (T), were seeded in 96-well U-bottom culture plates at a density of 2×10^4 cells/well in RPMI-1640 medium supplemented with 5% HIFBS. Splenocytes, prepared as described earlier, served as effector cells (E) and were added at 1×10^6 cells/well to give an E/T ratio of 50:1. The cell mixture was then incubated for 24 h at 37˚C in atmospheric air containing 5% CO_2. Following incubation, lactate dehydrogenase (LDH) activity in the culture medium was measured as previously detailed. NK cell activity was estimated using the following equation and expressed as the percentage of target cells killed [4].

$$\text{NK cell activity (\%)} = [(A_{ii} - A_i - A_{iii})] \times (A_{iv} - A_i)] \times 100$$

where A equals absorbance value of the respective experimental sample at 600 nm; 1) denotes basal LDH release from target cells; 2) denotes LDH release from a mixture of target cells and effector cells; 3) denotes basal LDH spontaneously released from effector cells; 4) denotes total LDH from target cells.

2.9. Con A/LPS-Induced Splenocyte Proliferation *Ex Vivo*

Mouse splenocyte concentration was adjusted to 5×10^6 cells/mL with RMPI medium supplemented with 10% HIFBS. Then, 90 μL of this cell suspension was seeded in each well of a 96-well plate, either with or without Con A/LPS, to a final volume of 100 μL. Con A/LPS was added at final concentrations of 0, 0.5, 1, 2, 4, or 7.5 μg/mL. The splenocytes were cultured for 72 hours at 37˚C in a humidified atmosphere containing 5% CO_2. Cell proliferation was assessed using MTT as previously described [11].

The extent of Con A/LPS-stimulated proliferation of isolated splenocytes was determined by calculating the area under the curve (AUC) from a graph that plotted the percentage of initial absorbance (mean absorbance of cells stimulated with Con A or LPS/mean absorbance of cells not stimulated with Con A or LPS × 100%) against the Con A or LPS concentration. The increase in Con A or LPS-stimulated splenocyte proliferation in Sch A-treated mice was estimated by comparing it with the untreated control and expressed as a percentage of the control [4].

2.10. Statistical Analysis

Data, expressed as mean ± SD, were analyzed using one-way ANOVA followed by Tukey's range test to detect the inter-group difference. Differences are considered significant when $p < 0.05$.

3. Results and Discussion

Sch A treatment significantly increased mitochondrial ATP-GC values by 8% - 23% in various tissues, except for the brain, when compared to untreated control

mice. The stimulation levels were highest in the liver, followed by the heart, kidney, lung, and spleen (Table 1). Additionally, mitochondrial glutathione redox status improved in the brain (14%), kidney (31%), and lung (24%) of Sch A-treated mice compared to untreated controls (Table 2). No significant changes were observed in the heart, liver, or spleen tissues. Furthermore, there were no detectable changes in NK cell activity or ConA/LPS-induced splenocyte proliferation in Sch A-treated mice (Table 3). The biphasic dose response of mitochondrial ATP-GC following Sch A treatment may be linked to the fact that increased ATP production in the mitochondria is constrained by excessive reactive oxygen species generated from heightened mitochondrial electron transport [12]. Additionally, higher doses of Sch A treatment also influence the mitochondrial glutathione redox status in a similar manner.

Table 1. Effects of Sch A treatment on mitochondrial ATP-GC in various tissues of mice. Values given are mean % control ± SD (n = 4). Control values (AUC2): Brain, 500 ± 16.3; Heart, 500 ± 18.9; Liver, 500 ± 26.3; Kidney, 500 ± 19.2; Lung, 200 ± 8.08; Spleen, 200 ± 9.38. *p < 0.05, **p < 0.01 when compared to the control value, using one-way ANOVA followed by Tukey's range test.

Sch A	0.3 g/kg			1 g/kg		
Brain	100	±	3.98	104	±	0.50
Heart	116	±	6.13*	103	±	6.41
Liver	123	±	7.18**	107	±	5.02
Kidney	111	±	5.04**	102	±	4.84
Lung	113	±	4.99**	111	±	0.70**
Spleen	103	±	4.59	108	±	2.49*

Table 2. Effects of Sch A treatment on mitochondrial glutathione redox status in various tissues of mice. Values given are mean % control ± SD (n = 4). Control values: Brain, 30.5 ± 3.34; Heart, 20.5 ± 2.08; Liver, 16.5 ± 4.41; Kidney, 24.1 ± 3.25; Lung, 7.80 ± 0.65; Spleen, 16.2 ± 1.36. *p < 0.05, **p < 0.01 when compared to the control value, using one-way ANOVA followed by Tukey's range test.

Sch A	0.3 g/kg			1 g/kg		
Brain	102	±	3.88	114	±	0.50**
Heart	91.5	±	12.7	94.8	±	6.41
Liver	91.5	±	8.19	102	±	8.22
Kidney	131	±	7.96**	103	±	6.33
Lung	124	±	10.1**	97.2	±	7.66
Spleen	104	±	4.27	93.3	±	11.6

Sch A did not enhance mitochondrial ATP-GC levels in mouse tissues as effectively as Sch B, showing only a modest stimulation, especially in heart and liver tissues (Table 4). Unlike Sch B, the stimulation of ATP-GC by Sch A does not appear to be tightly linked to an improvement in mitochondrial glutathione

redox status (Table 5). However, the Qi-invigorating action of Chinese tonifying herbs, as well as Sch B, as manifested by the stimulation of mitochondrial ATP-GC, is paralleled by the enhancement of cellular glutathione redox status [5] [8]. In this regard, Sch A may cause an increase in mitochondrial ATP-GC through a mechanism rather than the enhancement of mitochondrial glutathione redox status. Since maintaining a healthy mitochondrial glutathione redox status is vital for protecting tissues from oxidative damage [13], Sch A may provide less tissue protection compared to Sch B. The differential effects of Sch A and Sch B on mitochondrial ATP-GC and glutathione redox status may stem from their chemical structures; Sch B contains a methylene dioxy ring that is crucial for triggering an antioxidant response through reactive oxygen species production during metabolism [14] [15], while Sch A does not. Additionally, Sch A's limited impact on mitochondrial ATP-GC in various tissues may be related to its inability to stimulate both innate and adaptive immunity, which is an indirect measure of Zheng Qi in Chinese medicine (Table 6) [4]. After all, Sch A and Sch B possess a similar, yet not identical, chemical and pharmacological profile [16] [17].

Table 3. Effects of Sch A treatment on NK cell activity and ConA/LPS-induced splenocyte proliferation in mice *ex vivo*. Values given are mean % control ± SD (n = 4). Control values are: NK cell activity, 4.61 ± 0.43; ConA-induced proliferation (AUC), 2942 ± 764; LPS-induced proliferation, 2062 ± 383. *p < 0.05, **p < 0.01 when compared to the control value, using one-way ANOVA followed by Tukey's range test.

Sch A g/kg	NK cell activity	Con-A induced Splenocyte proliferation	LPS-induced Splenocyte proliferation
0.3	90.8 ± 9.56	85.6 ± 6.56**	97.5 ± 10.6
1	97.3 ± 9.71	99.5 ± 7.00	101 ± 7.13

Table 4. Comparison between Sch A and Sch B in the enhancement of mitochondrial ATP-GC in various tissues of mice. % increase (compared to control values): –, Nil; +, ~10% - 20%; ++, 20% - 30%; +++ 30% - 40%. Data for Sch B is extracted from our previously published work [3].

	Brain	Herat	Liver	Kidney	Lung	Spleen
Sch A	–	+	++	+	+	+
Sch B	+	+++	+++	+	++	+

Table 5. Comparison between Sch A and Sch B in the enhancement of mitochondrial glutathione redox status in various tissues of mice. % increase (compared to control values): –, Nil; +, ~10% - 20%; ++, 20% - 30%; +++ 30% - 40%; ++++, > 40%. Data for Sch B is extracted from our previously published work [3].

	Brain	Herat	Liver	Kidney	Lung	Spleen
Sch A	+	–	–	+++	++	–
Sch B	+	++	+++	++	+++	++++

Table 6. Comparison between Sch A and Sch B in the stimulation of innate and adaptive immunity in mice. % increase (compared to control values): –, Nil; ++, 20% - 30%. Data for Sch B is extracted from our previously published work [3].

	Innate Immunity (NK cell activity)	Adaptive Immunity (T/B cell proliferation)
Sch A	–	–
Sch B	++	++

4. Conclusion

In conclusion, while Sch A can slightly stimulate mitochondrial ATP-GC in most tissues, it does not generally enhance mitochondrial glutathione redox status or boost both innate and adaptive immunity in mice. Our findings suggest that the Qi-invigorating effect of Wuweizi, as described by Sun Si Miao during the Tang Dynasty, likely refers to Bei Wuweizi rather than Nan Wuweizi. According to the 2010 Edition of the Chinese Pharmacopoeia, Bei Wuweizi is widely considered the standard in many traditional herbal formulations [18]. However, based on the comparison between Sch A and Sch B regarding their effects on mitochondrial ATP-GC, we cannot rule out the possibility that the whole herb of Nan Wuweizi, which contains various lignan compounds, may enhance mitochondrial ATP-GC in different tissues at higher doses.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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