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Original Research Article

Preclinical haematological profile studies of an ayurvedic medicine ajowan after chronic administration to male Sprague-Dawley rats

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ABSTRACT

Background: Ayurvedic medicines are widely utilized for their therapeutic potential, yet many remain insufficiently evaluated for safety. Ajowan (*Trachyspermum ammi* Linn.), a traditional remedy for gastrointestinal disturbances, is commonly used in South Asia. However, data on its haematological safety following prolonged use are scarce. This study aimed to assess the haematological effects of chronic ajowan administration in male Sprague-Dawley rats.
Methods: Eighteen healthy male Sprague-Dawley rats were randomized into three groups: control (n=8), low-dose ajowan (JAN) (50 mg/kg; n=5), and high-dose ajowan (JAN) (400 mg/kg; n=5). The extract was administered orally for 28 days. On day 29, blood samples were collected and haematological parameters- including red and white blood cell indices, platelet counts, and erythrocyte sedimentation rate (ESR), were analysed using a CELL-DYN 3700 haematology analyser.
Results: Chronic ajowan administration produced dose-related, though mostly statistically insignificant, changes in red cell indices, with slight increases in RBC, haemoglobin, and haematocrit values. White blood cell counts increased by 21.1% (low dose) and 32.8% (high dose; p=0.039), suggesting mild immunostimulation. Differential counts revealed decline in eosinophil and neutrophil, while lymphocytes and monocytes rose slightly. Platelet counts fell modestly (−2.5% at low-dose and −10.2% at high-dose), but platelet indices and ESR largely remained within normal ranges. No marked hemotoxic effects were observed.
Conclusions: Chronic ajowan administration did not induce significant haematological toxicity in male rats, though elevated WBC counts and mild platelet reductions warrant further study.

Keywords: Ajowan, Ajwain, Trachyspermum ammi, Hematological profile, Ayurvedic medicine, Preclinical safety

INTRODUCTION

Ayurveda, one of the oldest medical systems in the world with origins in the Indian subcontinent, has been a cornerstone of the traditional health practices in South Asia for centuries. Blending ayurveda with moder medical practices aim to draw on the strengths of both approaches. This includes incorporating time-tested natural remedies from Ayurveda into contemporary healthcare, as well as applying modern scientific methods to enhance Ayurvedic treatments.[1]Ayurvedic medicines, which originate from plants and plant-based substances, contain a diverse range

of bioactive compounds that may provide valuable therapeutic effects.[2] Among the widely used Ayurvedic herbs, ajowan (*Trachyspermum ammi*) holds prominence due to its use in colic pain (Shula), flatulence, and gastrointestinal disturbances. *Trachyspermum ammi* Linn. (ajowan) is an aromatic annual herb from the Apiaceae family, commonly valued for its dual role as a household spice and a medicinal plant. Owing to these properties, it has been utilized for centuries in traditional medicine systems around the world.[3]

Ajowan is traditionally used to address various digestive issues. For colic pain, a paste of its crushed seeds is applied to the affected area, while a warm poultice prepared from the seeds is sometimes used on the chest to relieve respiratory complaints such as asthma. The seeds yield 2-4.4% of a brownish essential oil called ajowan oil, which is rich in thymol a compound commonly used to treat digestive disorders, stimulate appetite, and manage respiratory conditions like bronchitis.[4] The fruit is known for its ability to stimulate bodily functions, relieve muscle spasms, and aid in digestion by relieving flatulence.[5] According to the World Health Organization (WHO), a significant proportion of the global population, estimated at nearly 80%, uses herbal remedies to meet at least part of their primary healthcare requirements.

Recognizing its importance, the WHO has endorsed the widespread use of herbal remedies as an alternative approach to providing primary healthcare services.[6] In Bangladesh and neighbouring countries, ayurvedic medicines are not only cost-effective but also trusted due to their natural origin. However, with increasing global interest in evidence-based traditional medicine, establishing the safety profile of such treatments is essential. The current study was conducted to investigate the haematological effects resulting from the chronic oral administration of ajowan (JAN) in male Sprague-Dawley rats.

METHODS

Drug collection and preparation

For this study, ajowan (JAN) was sourced from Sri Kundeswari Aushadhalaya Limited, Chittagong, Bangladesh. One kilogram of ajowan was extracted using ethyl alcohol, producing approximately 55.94 grams of extract. A suspension of this resinous extract was prepared in a volume that allowed for precise dosing while minimizing any significant impact on the animal's total body fluid volume. The extract was given orally at doses of 50 mg/kg and 400 mg/kg of body weight. Additionally, to induce anaesthesia, ketamine was administered intraperitoneally at a dose of 500 mg/kg.

Experimental animals

In this study, healthy male Albino rats (Rattus norvegicus, Sprague-Dawley strain), aged eight weeks and weighing between 50-70 grams, were used. The rats were kept in the Animal House of the Department of Pharmacy at Jahangirnagar University. Before the experiment, rats were randomly assigned to 2 groups, with five rats in Low-dose JAN group (50 mg/kg; n=5) and five rats in High-dose JAN group (400 mg/kg; n=5). Additionally, a separate group of eight rats, not given the test substance, was included as the control group (n=8). These control animals received distilled water as a placebo, matched in volume and duration to the treatment given to the experimental groups.

Rats were housed in 30×20×13 cm plastic cages with soft wood shavings, given food and water ad libitum, and kept under a natural light–dark cycle in a clean, well-ventilated facility. They were fed mouse chow prepared according to the Bangladesh Council of Scientific and Industrial Research (BCSIR) formulation. All experimental procedures involving rats were conducted in strict accordance with the ethical guidelines for the care and use of laboratory animals, as approved by the Ethical Review Committee of the Faculty of Life Sciences, Department of Pharmacy, Jahangirnagar University.

Experimental design

Dosing and sample collection

Eighteen healthy male Sprague-Dawley rats (50-70 g, 8-10 weeks old) were housed under standard conditions (12-hour light/dark cycle, with ad libitum food and water). After a 7-day acclimatization period, the rats were randomly assigned into three groups:

Group I (control, n=8): Received distilled water

Group II (low-dose JAN group, n=5): Received JAN 50 mg/kg body weight

Group III (high-dose JAN group, n=5): Received JAN 400 mg/kg body weight

The drug was administered orally once daily between 10 AM and 12 PM, for 28 consecutive days via intra-gastric syringe. All procedures involving the rats strictly followed ethical guidelines for the care and use of laboratory animals. Each rat was marked on the tail for identification purposes to monitor and record individual responses both before and after treatment.[7]

Blood samples collection and preparation of serum

At the end of 28-day treatment period, on day 29, after 18 hours of fasting, blood was collected from rats under ketamine induced anaesthesia (500 mg/kg body weight, i.p.). Whole blood was drawn from the post vena cava and immediately placed into tubes containing Ethylenediamine tetra acetic acid (EDTA). All analyses were completed within 12-hour of sample collection.[8]

Determination of haematological profile studies

Haematological evaluation included red blood cell and platelet counts, determined by the electrical impedance method using the CELL-DYN 3700 system, an automated haematology analyzer.[9] The system applied four measurement methods: WBC Optical Count (WOC) and WBC Differentials via the Optical Flow channel; WBC Impedance Count (WIC) via one Electrical Impedance channel; RBC and platelet counts via a second Electrical Impedance channel; and haemoglobin levels via the Spectrophotometric channel.[10]

In each cycle, samples were aspirated, diluted, mixed, and measured for all parameters. Mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC) were calculated using established formulas as described by Wintrobe.[11]

$$MCV(fL)=(HCT(\%)\times 10)/RBC(\times 10^6/\mu L)$$

$$MCH(pg)=(Hemoglobin(g/dL)\times 10)/RBC(\times 10^6/\mu L)$$

$$MCHC(g/dL)=(Hemoglobin(g/dL)\times 100)/HCT(\%)$$

Haematocrit (HCT) was determined by multiplying the red blood cell (RBC) count by the mean corpuscular volume (MCV), as shown below:

$$HCT(\%) = RBC(\times 10^6/\mu L)\times MCV(fL)/10$$

Red cell distribution width (RDW) is a measurement parameter used to assess the heterogeneity of the RBC population. Red cell distribution width (RDW) was calculated using the formula: RDW = (standard deviation of MCV ÷ mean MCV)×100.[8] Platelet indices including platelet count (PLT), mean platelet volume (MPV), and platelet distribution width (PDW) were assessed in all samples through the electrical impedance technique, utilizing CELL-DYN 1700 and GENS analyzers.[12] The Westergren Method was used to measure the Erythrocyte Sedimentation Rate (ESR).[13]

Statistical analysis

Group data were presented as Mean ± SEM (Standard Error of the Mean). To assess statistical significance, unpaired "t" tests were performed. Data analysis was carried out using the Statistical Package for Social Sciences (SPSS), version 15. Differences between groups were deemed statistically significant at p-values: <0.05 (Significant), <0.01 (Highly Significant), and <0.001 (Very Highly Significant).

RESULTS

Effect of chronic administration of ajowan on haematological profiles of male rats

As summarized in Tables 1 and 2, in male rats, red blood cell (RBC) count increased by 4.88% at the low dose and 5.70% at the high dose, changes that were prominent but not statistically significant (p=0.395, 0.293). Haemoglobin (HGB) rose by 6.39% and 5.89% at low and high doses (p =0.224, 0.273), while haematocrit (HCT) increased by 3.99% and 5.71% (p=0.527, 0.249), suggesting dose-related prominent, but insignificant trends.

Table 1: Hematological profiles after chronic administration of ajowan (JAN) in 50 mg/kg dose to the male rats for 28 days.

Parameters	Control (mean±SEM)	Low-dose JAN group (mean±SEM)	P value	% change
RBC	6.58±0.12	6.90±0.21	0.395	↑ 4.88
HGB	12.05±0.25	12.82±0.38	0.224	↑ 6.39
HCT	34.81±0.69	36.20±0.75	0.527	↑ 3.99
MCV	52.98±0.47	53.72±0.48	0.498	↑ 1.41
MCH	18.33±0.19	18.60±0.12	0.522	↑ 1.50
MCHC	34.61±0.15	34.64±0.14	0.989	↑ 0.08
RDW-SD	43.26±0.87	43.02±0.97	0.976	↓ 0.56
RDW-CV	16.81±0.39	16.68±0.65	0.973	↓ 0.79
WBC	6.33±0.52	7.66±0.46	0.207	↑ 21.11
Neutrophil (%)	14.38±0.89	14.00±1.87	0.968	↓ 2.61
Eosinophil (%)	3.00±0.87	1.40±0.40	0.235	↓ 53.43
Basophil (%)	0.75±0.25	1.00±0.55	0.834	↑ 33.33
Lymphocyte (%)	81.63±1.16	82.00±2.32	0.980	↑ 0.46
Monocyte (%)	1.00±0.27	1.00±0.27	0.417	↑ 40.00
Immature granulocyte (%)	0.21±0.12	0.22±0.20	0.999	↑ 3.53
Platelet (PLT)	548.71±19.97	535.20±21.66	0.867	↓ 2.46
MPV	6.33±0.04	6.30±0.04	0.908	↓ 0.40
PCT	0.34±0.02	0.35±0.01	0.902	↑ 2.39

Continued.

Parameters	Control (mean±SEM)	Low-dose JAN group (mean±SEM)	P value	% change
PDW	15.88±0.66	15.62±1.11	0.967	↓ 1.61
P-LCR	23.76±0.69	19.54±2.32	0.059	↓ 17.77
ESR	2.00±0.38	1.80±0.37	0.914	↓ 10.00

Data were presented as Mean±SEM (Standard Error of the Mean), p-values: <0.05 (Significant), <0.01 (Highly Significant), and <0.001 (Very Highly Significant), ↑: increase, ↓: decrease.

Table 2: Hematological profiles after chronic administration of ajowan (JAN) in 400 mg/kg dose to the male rats for 28 days.

Parameters	Control (mean±SEM)	High-dose JAN group (mean±SEM)	P value	% change
RBC	6.58±0.12	6.95±0.27	0.293	↑ 5.70
HGB	12.05±0.25	12.76±0.46	0.273	↑ 5.89
HCT	34.81±0.69	36.80±1.35	0.249	↑ 5.71
MCV	52.98±0.47	52.98±0.57	1.000	↑ 0.01
MCH	18.33±0.19	18.38±0.22	0.972	↑ 0.30
MCHC	34.61±0.15	34.68±0.16	0.934	↑ 0.20
RDW-SD	43.26±0.87	43.34±0.91	0.998	↑ 0.18
RDW-CV	16.81±0.39	16.56±0.40	0.905	↓ 1.50
WBC	6.33±0.52	8.40±0.72	0.039*	↑ 32.81
Neutrophil (%)	14.38±0.89	11.60±1.12	0.229	↓ 19.30
Eosinophil (%)	3.00±0.87	1.00±0.32	0.121	↓ 66.67
Basophil (%)	0.75±0.25	0.40±0.25	0.706	↓ 46.67
Lymphocyte (%)	81.63±1.16	86.00±1.34	0.115	↑ 5.36
Monocyte (%)	1.00±0.27	1.00±0.27	1.000	0.00
Immature granulocyte (%)	0.21±0.12	0.06±0.06	0.661	↓ 71.76
Platelet (PLT)	548.71±19.97	492.60±21.88	0.998	↓ 10.23
MPV	6.33±0.04	6.48±0.06	0.063	↑ 2.45
PCT	0.34±0.02	0.34±0.02	1.000	↑ 0.06
PDW	15.88±0.66	17.20±0.76	0.439	↑ 8.35
P-LCR	23.76±0.69	20.32±0.80	0.132	↓ 14.49
ESR	2.00±0.38	1.60±0.40	0.707	↓ 20.00

Data were presented as Mean±SEM (Standard Error of the Mean), p-values: <0.05 (Significant), <0.01 (Highly Significant), and <0.001 (Very Highly Significant), ↑: increase, ↓: decrease.

MCV rose 1.41% at the low dose (p=0.498) with no change at the high dose; MCH increased 1.50% and 0.30% (p=0.522, 0.972). MCHC changed negligibly (0.08%, 0.20%; p=0.989, 0.934). RDW-SD fell 0.56% at low dose and rose 0.18% at high dose (p=0.976, 0.998), while RDW-CV declined 0.79% and 1.50% (p=0.973, 0.905). WBC count increased by 21.11% at the low dose which was not significant, yet it was prominent (p=0.207) and increased statistically significantly by 32.81% at the high dose (p=0.039).

Conversely, neutrophils declined 2.61% and 19.30% (p= 0.968, 0.229), and eosinophils decreased sharply at both doses (53.43%, 66.67%; p=0.235, 0.121). Basophils rose 33.33% at low dose and fell 46.67% at high dose (p= 0.834, 0.706). Immature granulocytes increased 3.53% at low dose (p=0.999) but dropped 71.76% at high dose (p= 0.661). Lymphocytes rose 0.46% and 5.36% (p=0.980, 0.115), and monocytes increased 40.0% at low dose (p =0.417) with no change at high dose. Platelet (PLT) counts fell at both doses (2.46%, 10.23%; p=0.867, 0.998). MPV dropped 0.40% at low dose (p=0.908) and rose 2.45% at high dose (p=0.063). Platelet-large cell ratio (P-LCR) declined 17.77% and 14.49% (p=0.059, 0.132), with a more prominent decrease at the higher dose. Plateletcrit (PCT) rose 2.39% at low dose (p=0.902) with no change at high dose. Additionally, platelet distribution width (PDW) fell 1.61% at low dose (p=0.967) and rose 8.35% at high dose (p=0.439). Finally, erythrocyte sedimentation rate (ESR) dropped by 10.0% and 20.0% at low and high doses, respectively (p=0.914 and 0.707), both considered statistically insignificant.

DISCUSSION

Hematological evaluation is an essential component of preclinical and clinical safety research, forming a standard approach to monitoring the effects of new and established therapies in humans and animals. These studies examine blood samples to measure key indicators such as red blood cells, white blood cells, and platelets, which provide important information on the safety characteristics of a

drug. In preclinical experiments, such assessments are crucial for detecting potential toxicities before human trials, while in clinical settings they track how a medication influences blood parameter. Assessing the haematological profile of laboratory animals, especially rodents, is vital when investigating potential hemotoxicity, as researchers can predict early signs of toxicity that may later emerge in humans.[14] Hematological changes in animal studies often indicate how a substance might affect humans, offering valuable insight into potential toxicity. Marked shifts in blood parameters across all tested doses of a compound suggest safety concerns and reduce its likelihood as a viable candidate for further development.[15]

Blood is one of the first systems influenced by systemic drug exposure, making it a crucial target in toxicity evaluations. This vulnerability stems from the rapid renewal of hematopoietic cells, constant blood movement through tissues, and the widespread impact that any disruption to these cells can have on overall body function. Shifts in blood parameters can signal wider systemic issues, such as bone marrow suppression or impaired immune responses.[16] This study aimed to examine how the test compound influenced haematological parameters in male rats at two different doses, with the objective of detecting signs of hemotoxicity. While most changes were not statistically significant, some dose-related patterns and noticeable variations were prominent, offering insights into the compound's biological effects.

An increase in RBC count, HGB and HCT values was observed at both doses. Though not statistically significant, the upward trend may suggest mild erythropoietic stimulation or physiological adaptation. Indices such as MCV, MCH, and MCHC showed minimal changes, indicating stable red cell morphology and haemoglobin content. Red cell distribution parameters (RDW-SD and RDW-CV) showed minor, insignificant shifts, implying no substantial alteration in red cell size heterogeneity. WBCs are a vital part of the immune system, serving as the main line of defence against microbes. They locate, recognize, and attach to bacteria, fungi, or viruses to neutralize them and protect the body.[17] When injury or infection occurs, the inflammatory response triggers signals that mobilize neutrophils, lymphocytes, monocytes, and mast cells to the affected area to support healing and clot formation. However, prolonged elevated WBCs can harm healthy tissue, increasing the likelihood of chronic disease.[18]

In this study, more prominent effects were observed in WBC counts, particularly at the higher dose where a significant increase was noted. The elevated WBC count could imply a mild immunostimulatory effect or a transient immune response triggered by the compound. Differential counts showed a complex pattern: lymphocytes and monocytes exhibited mild increases, while neutrophils and eosinophils declined, most notably eosinophils, which decreased at both doses. Such changes, though subtle, may be linked to early immunomodulation or inflammatory response. Total PLT counts declined in a dose-dependent manner, with a more prominent reduction at the higher dose (10.23%). MPV and platelet-large cell ratio (P-LCR) demonstrated some fluctuations but remained within a physiological range. The P-LCR reduction approached significance at the low dose ($p=0.059$), possibly indicating subtle alterations in platelet activation or production. Since platelets play a key role in clotting and help regulate immune and inflammatory processes, any alteration in number or size can have significant consequences.[19] Hence, the downward trend in platelet count, especially at higher exposure, should be monitored in extended or repeated-dose studies. Plateletcrit (PCT) and platelet distribution width (PDW) remained largely unchanged. ESR, an indirect and nonspecific marker of inflammation, showed a dose-dependent decline, potentially indicating an absence of systemic inflammation under the test conditions.[20]

Ayurvedic preparations, including the one examined, are widely used but often not subjected to rigorous safety evaluations. Differences in formulation methods, ingredient composition, and dosage make it difficult to establish clear safety standards. Without strict regulatory standardization, the possibility of adverse effects cannot be entirely dismissed.[21] The findings of this study indicate that the test compound did not cause any obvious hemotoxicity at the given doses in male rats. Nevertheless, while major changes in haematological parameters were not observed, certain patterns, particularly shifts in WBC and platelet counts, underscore the need for further investigation, especially at higher doses and with extended exposure periods.

CONCLUSION

The study demonstrates that the test compound does not induce overt haematological toxicity in male rats at the administered doses, as most parameters remained within physiological limits without any statistically significant alterations. The observed increases in RBC count, hemoglobin, and hematocrit suggest a possible mild stimulatory or adaptive effect on erythropoiesis without compromising red cell integrity. The rise in total WBC count at the higher dose, along with shifts in differential counts, was significant and indicated a potentially mild immunomodulatory response. Overall, the findings suggest a favorable haematological safety profile of the test compound. However, dose-related subtle changes in immune and platelet parameters highlight the need for further long-term investigations to fully establish its safety profile.

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REFERENCES

1. Verma SK, Pandey M, Sharma A, Singh D. Exploring Ayurveda: principles and their application in modern medicine. Bull Natl Res Cent. 2024;48:77.
2. Chrysant SG, Chrysant GS. Herbs used for the treatment of hypertension and their mechanism of action. Curr Hypertens Rep. 2017;19(9):1-10.
3. Naaz A, Wani GN, Begum W, Fathima S. Therapeutic potential of Nankhawah/Ajwain (Tracyspermum ammi Linn): a comprehensive review article. J Drug Deliv Ther. 2024;14(3):164-9.
4. Ullah MA, Hassan A, Hamza A. Medical Constituents of Ajwain (Trachyspermum ammi) for Human Benefits. J Gen Med Clin Pract. 2024;7(2):1-7.
5. Bairwa R, Sodha RS, Rajawat BS. Trachyspermum ammi. Pharmacogn Rev. 2012;6(11):56-60.
6. Parvin A, Azad MA, Sarker A, Khan MAA, Jahan N, Nadvi MR, et al. Effect on liver function of an ayurvedic medicine Punarnavasava after 28 days chronic toxicity studies on male Sprague Dawley rats. Int J Basic Clin Pharmacol. 2025;14(5):673-8.
7. Stevens KR, Gallo MA. Practical considerations in the conduct of chronic toxicity studies. Principles and Methods of Toxicology. New York, NY: Raven Press. 1989:237-50.
8. Amin MT, Fatema K, Karmakar P, Rahman MA, Haque T, Choudhuri MSK. Investigation of hemotoxicologic potential of an Ayurvedic preparation Kutajarista used in Sprue syndrome after chronic administration to male Sprague-Dawley rats. Afr J Pharm Pharmacol. 2020;14(8):308-15.
9. Wolford ST, Schroer RA, Gohs FX, Gallo PP, Brodeck M, Falk HB, et al. Reference range database for serum chemistry and hematology values in laboratory animals. J Toxicol Environ Health. 1986;18(2):161-88.
10. Suljevic E, Fazlic M, Coric J, Kiseljakovic E. Evaluation of haematology analyzer CELL-DYN 3700 SL. Bosn J Basic Med Sci. 2003;3(2):35-41.
11. Wintrobe MM. The volume and hemoglobin content of the red blood corpuscle: simple method of calculation, normal findings, and value of such calculations in the anemias. Am J Med Sci. 1929;177:513-23.
12. Azad MA, Ohidullah M, Neon MN, Sikder MM, Choudhuri MSK. Preclinical hematological profile studies of an ayurvedic medicine Krishna Chaturmukha after chronic administration to male Sprague-Dawley rats. Int J Basic Clin Pharmacol. 2025;14:154-9.
13. Westergren A. Diagnostic tests: the erythrocyte sedimentation rate range and limitations of the technique. Triangle. 1957;3(1):20-5.
14. Olson H, Betton G, Robinson D, Thomas K, Monro A, Kolaja G, et al. Concordance of the toxicity of pharmaceuticals in humans and in animals. Regul Toxicol Pharmacol. 2000;32(1):56-67.
15. Arika WM, Nyamai DW, Musila MN, Ngugi MP, Njagi ENM. Hematological markers of in vivo toxicity. J Hematol Thrombo Dis. 2016;4(2):1-7.
16. Bloom JC, Lewis HB, Sellers TS, Deldar A. The hematologic effects of cefonicid and cefazedone in the dog: a potential model of cephalosporin hematotoxicity in man. Toxicol Appl Pharmacol. 1987;90(1):135-42.
17. Saidani O, Umer M, Alturki N, Alshardan A, Kiran M, Alsubai S, et al. White blood cells classification using multi-fold pre-processing and optimized CNN model. Sci Rep. 2024;14:1-14.
18. Chan SHT, Tsung Y, Zhang Z, Chang L, Guo C, Bo Y, et al. Total and differential white blood cell count and cause-specific mortality in 436750 Taiwanese adults. Nutr Metab Cardiovasc Dis. 2022;32(4):937-47.
19. Chen Y, Zhong H, Zhao Y, Luo X, Gao W. Role of platelet biomarkers in inflammatory response. Biomark Res. 2020;8:1-7.
20. Navaneethan U. Diagnosis of pouch disorders: laboratory tests. Pouchitis and Ileal Pouch Disorders: A Multidisciplinary Approach for Diagnosis and Management. London: Academic Press. 2019;237-46.
21. Wang H, Chen Y, Wang L, Liu Q, Yang S, Wang C. Advancing herbal medicine: enhancing product quality and safety through robust quality control practices. Front Pharmacol. 2023;14:1-16.

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