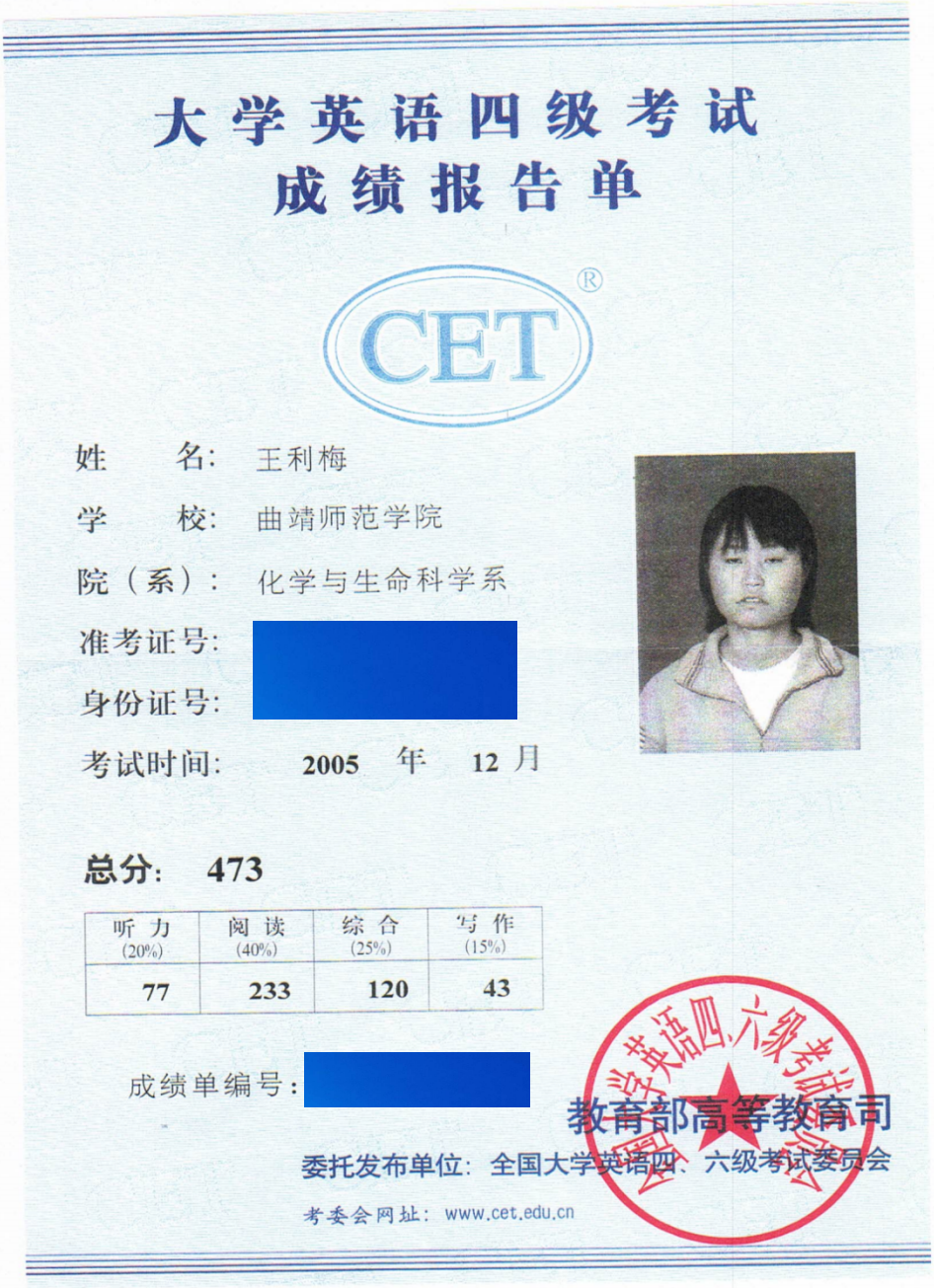
4、 外语能力证书



大学英语四级考试
成绩报告单

CET®

姓　　名：王利梅

学　　校：曲靖师范学院

院（系）：化学与生命科学系

准考证号：[redacted]

身份证号：

考试时间：2005 年 12 月

总分：473

听力 (20%)	阅读 (40%)	综合 (25%)	写作 (15%)
77	233	120	43

成绩单编号：[redacted]

教育部高等教育司

委托发布单位：全国大学英语四、六级考试委员会

考委会网址：www.cet.edu.cn

5、 学术能力证明材料

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SYNAPSE WILEY

RESEARCH ARTICLE

Mechanism of cognitive impairment induced by D-galactose and L-glutamate through gut–brain interaction in tree shrews

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Limei Wang and Jiangli Lu contributed equally to this work.

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Abstract

D-Galactose (D-gal) and L-glutamate (L-glu) impair learning and memory. The mechanism of interaction between the gut microbiome and brain remains unclear. In this study, a model of cognitive impairment was induced in tree shrews by intraperitoneal (ip) injection of D-gal (600 mg/kg/day), intragastric (ig) administration with L-glu (2000 mg/kg/day), and the combination of D-gal (ip, 600 mg/kg/day) and L-glu (ig, 2000 mg/kg/day). The cognitive function of tree shrews was tested by the Morris water maze method. The expression of Aβ1-42 proteins, the intestinal barrier function proteins occludin and P-glycoprotein (P-gp), and the inflammatory factors NF-κB, TLR2, and IL-18 was determined by immunohistochemistry. The gut microbiome was analyzed by 16SrRNA high-throughput sequencing. After administering D-gal and L-glu, the escape latency increased ($p < .01$), and the times of crossing the platform decreased ($p < .01$). These changes were greater in the combined administration of D-gal and L-glu ($p < .01$). The expression of Aβ1-42 was higher in the perinuclear region of the cerebral cortex ($p < .01$) and intestinal cell ($p < .05$). There was a positive correlation between the cerebral cortex and intestinal tissue. Moreover, the expression of NF-κB, TLR2, IL-18, and P-gp was higher in the intestine ($p < .05$), while the expression of occludin and the diversity of gut microbes were lower, which altered the biological barrier of intestinal mucosal cells. This study indicated that D-gal and L-glu could induce cognitive impairment, increase the expression of Aβ1-42 in the cerebral cortex and intestinal tissue, decrease the gut microbial diversity, and alter the expression of inflammatory factors in the mucosal intestines. The dysbacteriosis may produce inflammatory cytokines to modulate neurotransmission, causing the pathogenesis of cognitive impairment. This study provides a theoretical basis to explore the mechanism of learning and memory impairment through the interaction of microbes in the gut and the brain.

KEYWORDS
cognitive impairment, D-galactose, gut microbes, intestinal mucosal barrier, L-glutamate, tree shrew

项目批准号	
申请代码	
归口管理部门	
依托单位代码	

824601

国家自然科学基金

资助项目计划书

（预算制项目）

资助类别：地区科学基金项目

亚类说明：

附注说明：

项目名称：石斛多糖维持糖尿病树鼩葡萄糖稳态的作用机制

直接费用：32万元　　执行年限：2025.01-2028.12

负 责 人：王利梅　　BRID：03871.00.73029

通讯地址：云南省昆明市呈贡区雨花街道春融西路1168号

邮政编码：650500　　电　　话：18787456226

电子邮件：wlmeiaa@163.com

依托单位：昆明医科大学

联 系 人：杨海龙　　电　　话：0871-65922623

填表日期：2024年08月26日

国家自然科学基金委员会制

102

Version：1.009.814

国家自然科学基金项目负责人、依托单位承诺书

国家自然科学基金项目负责人承诺书

本人郑重承诺：我接受国家自然科学基金的资助，严格遵守中共中央办公厅、国务院办公厅《关于进一步加强科研诚信建设的若干意见》《关于进一步弘扬科学家精神加强作风和学风建设的意见》《关于加强科技伦理治理的意见》《科技伦理审查办法（试行）》等规定，和国家自然科学基金委员会关于资助项目管理、项目资金管理等各项规章，在《计划书》填写及项目执行过程中：

（一）按照《批准通知》《国家自然科学基金资助项目计划书填报说明》的要求填写《计划书》，未自行降低、更改目标任务或约定要求，或缩减研究（研制）内容；

（二）树立“红线”意识，严格履行科研合同义务，按照《计划书》负责实施本项目（批准号：82460164），切实保证研究工作时间，按时报送有关材料，及时报告重大情况变动，不违规将科研任务转包、分包他人，不以项目实施周期外或不相关成果充抵交差；

（三）遵守科研诚信、科技伦理规范和学术道德，认真开展研究工作，对资助项目发表的论著和取得的研究成果按规定进行标注，不在非本项目资助的成果或其他无关成果上标注本项目批准号，反对无实质学术贡献者“挂名”，不在成果署名、知识产权归属等方面侵占他人合法权益，并如实报告本人及项目组成员发生的违背科研诚信要求的任何行为；

（四）尊重科研规律，弘扬科学家精神，严谨求实，追求卓越，反对浮夸浮躁、投机取巧，不人为夸大学术或技术价值，不传播未经科学验证的现象和观点；

（五）将项目资金全部用于与本项目研究工作相关的支出，并结合科研活动需要，科学合理安排项目资金支出进度；

（六）做好项目组成员的教育和管理，确保遵守以上相关要求。

如违背上述承诺，本人愿接受国家自然科学基金委员会和相关部门做出的各项处理决定。

项目负责人（签字）：王利杨

2024年 9 月 11 日

依托单位科研管理部门：	依托单位财务管理部门：
负责人（签章）： 2024年 9 月 20 日	负责人（签章）：卢宏坤 2024年 9 月 20 日

国家自然科学基金项目依托单位承诺书

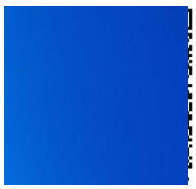
我单位同意承担上述国家自然科学基金项目，将保证项目负责人及其研究队伍的稳定和研究项目实施所需的条件，严格遵守中共中央办公厅、国务院办公厅《关于进一步加强科研诚信建设的若干意见》《关于进一步弘扬科学家精神加强作风和学风建设的意见》《关于加强科技伦理治理的意见》《科技伦理审查办法（试行）》等规定，和国家自然科学基金委员会有关资助项目管理、项目资金管理、科研诚信管理和科技伦理管理等各项规定，并督促实施。

依托单位（公章）

2024 年 9 月 10 日

计划类别：基础研究计划

项目编号：202401AY070001-207



云南省科技厅科技计划项目合同书

项目名称：石斛碱通过肠道菌群激活IR/AKT/FoxO1通路调控能量代谢改善糖尿病认知障碍的作用机制

甲方（项目管理部门）：昆明医科大学

乙方（项目承担单位）：昆明医科大学

丙方（项目推荐部门）：昆明医科大学科学技术处

项目负责人：王利梅　　电话：0871-65939180

项目起止年限：2024年05月至2027年04月

云南省科学技术厅制

十、合同书各方

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开户银行：中国银行股份有限公司昆明市人民西路支行 账号名称：昆明医科大学 银行账号：135601927831 （承担单位公章） 2024年 7 月 1 日		
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计划类别：基础研究专项

项目编号：202201AT070296

云南省科技厅科技计划项目合同书

项目名称：石斛多糖通过肠道菌群靶向ADH1B/C基因调控PI3K/AKT信号通路改善糖尿病糖代谢的机制研究

承担管理单位（甲方）：云南省科学技术厅

项目承担单位（乙方）：昆明医科大学

项目推荐部门（丙方）：昆明医科大学

项目负责人：王利梅　　电话：0871-65939180

项目起止年限：2022年06月至2025年05月

云南省科学技术厅制

九、合同书各责任方

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（单位公章） 年　月　日	

生命科学基础研究入门

Shengming Kexue Jichu Yanjiu Rumen

主　编　王利梅　卢敏南　曹　雪
副主编　杨学芳　李盛安　张会香　刘建军　罗夙医

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WEISHENGWU YU
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微生物与动植物健康

曹雪　王利梅　张会香◎主编

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昆 明 市 人 民 政 府

昆政笺〔2025〕7号

昆明市人民政府关于入选第二十一批昆明市中青年学术和技术带头人及后备人选的通知

各有关单位:

根据《昆明市中青年学术和技术带头人及后备人选选拔培养考核办法》(昆明市人民政府令第142号)和《昆明市中青年学术和技术带头人及后备人选选拔培养考核实施细则》有关规定，经专家评审、部门审核并向社会公示，市人民政府研究决定，王娓娓等20人入选第二十一批昆明市中青年学术和技术带头人，丁文云等30人入选第二十一批昆明市中青年学术和技术后备人选。

希望入选的第二十一批昆明市中青年学术和技术带头人及后备人选进一步增强责任感、使命感，充分发挥学术技术带头人及后备人选作用，立足本职、潜心研究，继续发扬求真务实、勇于创新的精神，为推动昆明市经济社会高质量发展提供坚强的科技支撑。

附件：第二十一批昆明市中青年学术和技术带头人及后备人选名单

昆明市人民政府

2025年1月26日

(此件公开发布)

附件

第二十一批昆明市中青年学术和技术带头人及后备人选名单

一、第二十一批昆明市中青年学术和技术带头人

王娓娓　昆明医科大学第二附属医院

卢红波　云南锡业新材料有限公司

付　俊　中国有色金属工业昆明勘察设计研究院有限公司

冯　阳　云南省交通科学研究院有限公司

刘彦章　昆明中天达玻璃钢开发有限公司

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李　浩　云南省交通规划设计研究院股份有限公司

时晓菲　云南民族大学

吴弦骏　中国电建集团昆明勘测设计研究院有限公司

宋　杰　云南省农业科学院花卉研究所

陈亚娟　昆明医科大学

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戴静毅　昆明市第三人民医院

魏　宾　新坐标科技有限公司

二、第二十一批昆明市中青年学术和技术后备人选

丁文云　中铁二院昆明勘察设计研究院有限责任公司

刁玉华　昆明市食品药品检验所（昆明市食品药品检验研究院）

马江丽　昆明贵研催化剂有限责任公司

马殿普　云南锡铟实验室有限公司

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文章楠　昆明理工大学

华建豪　云南贵金属实验室有限公司

刘　涵　中国电建集团昆明勘测设计研究院有限公司

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李建兴　中国电建集团昆明勘测设计研究院有限公司

李森茂　昆明医科大学第二附属医院

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陈子豪　昆明医科大学第一附属医院

杨烨鹏　昆明学院

证书号第14165556号

实用新型专利证书

实用新型名称：一种蛋白质溶解装置

发　明　人：王利梅;罗梅;杨云凤;王凌霄;杨庆;刘越;何忠银

专　利　号：ZL 2020 2 3028795.7

专利申请日：2020年12月16日

专利权人：昆明医科大学

地　　址：650500 云南省昆明市呈贡区雨花街道春融西路1168号

授权公告日：2021年09月14日　　授权公告号：CN 214182789 U

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Research Letter

RESEARCH LETTER – Environmental Microbiology

Improving Alzheimer's disease by altering gut microbiota in tree shrews with ginsenoside Rg1

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One sentence summary: Ginsenoside Rg1 may improve Alzheimer's disease symptoms in tree shrews.

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ABSTRACT

Ginsenoside Rg1 (GRg1) has neuroprotective effects on Alzheimer's disease (AD). The occurrence and progression of AD are closely related to gut microbiota. Few studies have learned the direct relationship between GRg1 and gut microbiota. In this study, we found an original way to research this relationship by using GRg1 in the AD model of tree shrews. Morris water maze and immunohistochemistry were performed to test the cognition repairing function of GRg1 by tree shrews and 16S ribosomal RNA sequencing was used to explore the composition and abundance of gut microbiota. After GRg1 treatment, the result of Morris water maze showed an improvement in cognitive function, and immunohistochemistry revealed a decrease in tau protein. Moreover, 16SrRNA sequencing results showed the abundances of *Proteobacteria* and *Verrucomicrobia* were significantly different, and *Lactobacillaceae* was significantly increased in the GRg1 treatment group. It also showed that the gut microbiome with middle and high doses of GRg1 was close to the normal group. In conclusion, this study suggests that GRg1 at middle and high doses may change the abundance of gut microbiota to improve AD, and that *Proteobacteria* and *Verrucomicrobia* are key microbiota. This is the first report that has ever studied the relationship between GRg1 and gut microbiota in tree shrews.

Keywords: Alzheimer's disease; ginsenoside Rg1; tree shrew; gut microbiota; Morris water maze; tau protein

INTRODUCTION

Alzheimer's disease (AD), which plays a critical role in causing memory loss, cognitive impairment and behavior changes, leads to death about 9 years after diagnosis. Studies have shown the pathological features of AD including senile plaques and neurofibrillary tangles. The major constituent of senile plaques is amyloid-β peptides (Aβ), which are generated as cleavage fragments by the action of γ and β secretase on amyloid precursor protein (APP) metabolism (Xing *et al.* 2013). So far, numerous neuroprotective drugs have been discovered in preclinical studies (Ji *et al.* 2017; Li *et al.* 2016). However, no drugs have shown the effect of inhibiting the deterioration of AD significantly.

Ginseng is a traditional medicinal herb that has been applied widely for the treatment of memory loss in Asia (Hu *et al.* 2014). Ginsenoside Rg1 (GRg1) is the major pharmacologically active ingredient of Panax notoginseng, and plays a neuroprotective role in AD (Bao-Sheng *et al.* 2014). GRg1 ameliorates the impairment of learning and memory in SAMP8 mice (Shi *et al.* 2010). Fuzheng Quxie Decoction (FQD) at a medium dose not only

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Original Article

MOLECULAR METABOLISM

Aregs-IGFBP3-mediated SMC-like cells apoptosis impairs beige adipocytes formation in aged mice

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ABSTRACT

Aging is associated with a decline in the browning capacity of white adipose tissue (WAT), contributing to metabolic dysfunction. Beige adipocytes, which dissipate excess energy as heat, are a key feature of this process. In this study, we investigate the role of adipose stem and progenitor cells (ASPCs), specifically the Aregs (CD142+) subpopulation, in regulating beige adipocyte formation in aged mice under cold stimulation. Our findings reveal that Aregs significantly increase in the subcutaneous WAT (sWAT) of aged mice following cold exposure. We further demonstrate that Aregs secrete insulin-like growth factor binding protein 3 (IGFBP3), which appears to play a pivotal role in the cross-talk between adipogenesis-regulatory cells (Aregs) and smooth muscle cell-like (SMC-like) cells, thereby leading to the inhibition of beige adipocytes formation. Functional enrichment analysis highlighted the activation of TGFβ, MAPK and p53 signaling pathways in SMC-like cells, all of which are known to induce cell apoptosis and fibrosis. Moreover, IGFBP3 was found to interact with receptors and signaling molecules, including Egfr, Irf1 and Cdkn1a, in SMC-like cells, enhancing their apoptosis. Co-culture experiments confirmed that IGFBP3 significantly suppressed the formation of beige adipocytes, further corroborating its role in impairing browning. Overall, our study provides novel insights into the molecular mechanisms by which Aregs and IGFBP3 contribute to the age-related decline in WAT browning. These findings suggest potential therapeutic targets for reversing impaired WAT browning in aging and related metabolic disorders.

Keywords Aregs; IGFBP3; SMC-Like cells; Browning; Aging

1. INTRODUCTION

Adipose tissue plays a pivotal role not only in energy storage but also as an active endocrine organ regulating systemic metabolism. Among its subtypes, brown adipose tissue (BAT) and beige adipose tissue have garnered attention for their unique ability to dissipate energy via thermogenesis, mediated by mitochondrial uncoupling through uncoupling protein 1 (UCP1) [1,2]. Beige adipose tissue, in particular, has drawn significant interest due to its remarkable plasticity. It can transdifferentiate from widespread white adipose tissue (WAT) or de novo differentiate from beige progenitor cells scattered within WAT under specific stimuli, such as classical cold exposure. De novo differentiated beige progenitor cells exhibit smooth muscle-like (SMC-like) characteristics [3–5]. This entire process, referred to as "browning" [6,7], holds great potential for combating obesity, cardiovascular diseases (CVD), and other metabolic disorders [8–10].

However, the ability of adipose tissue to undergo browning declines with aging. Aged individuals exhibit a reduced capacity for beige fat formation, which is often associated with impaired thermogenesis and metabolic dysfunction [11,12]. This decline is thought to contribute to the increased risk of obesity, insulin resistance, and CVD observed in the elderly [12,13]. The diminished browning capacity in aging fat depots has been linked to changes in the adipose tissue microenvironment, including altered stem cell function [14,15], the accumulation of inflammatory mediators [8] and a reduced response to cold exposure [16]. Understanding the cellular and molecular mechanisms behind this decline in browning ability could provide valuable insights into how aging exacerbates metabolic diseases and identify potential therapeutic targets to reverse these age-related changes.

Adipogenesis-regulatory cells (Aregs), a CD142+ subpopulation of adipose stem and precursor cells (ASPCs), were first identified and molecularly characterized in 2018 by Schwalie et al. These cells are refractory to adipogenesis and exhibit a paracrine capacity to suppress adipocytes formation in vitro and in vivo, suggesting a critical role in regulating adipose tissue plasticity and metabolic functions [17]. A recent study has suggested that Aregs may play a pivotal role in the remodeling of WAT under cold stimulation [18]. Additionally, it has been shown that the anti-adipogenic activity of Aregs increases with

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BIOCHEMISTRY

Study of the Application of Sprague-Dawley Rats for Disease Research Based on Hematological Parameters

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Abstract—This work aimed to obtain hematological parameters of Sprague-Dawley (SD) rats with different ages and genders, which supports the application of SD in drug screening, disease diagnosis, and treatment research. 26 hematological parameters of SD rats with ages of 3-12-21-week-old were obtained, 12-week-old were the least discrete, 3-week-old were obviously clustered together. White blood cell count (WBC), Mononuclear cell count (MONO), Platelet (PLT) and Platelet count (PCT) were significantly discrete at all weekly ages, while Mean corpuscular hemoglobin concentration (MCHC), High fluorescence intensity reticulocyte ratio (HFR), and Red blood cell distribution width-CV value (RDW-CV) were relatively clustered. The LYMPH (%), HFR (%), HCT (%), MCV, NEUT, MONO, RET (%), RDW-CV (%), and MCHC have a significance in different ages, as well as HCT (%), RBC, RDW-CV (%), RDW-SD in different genders. In conclusion, 12 weeks old SD rats should be selected as far as possible when testing hematological parameters, and 3 weeks old rats should be used only for adolescent related diseases research; gender is a great important factor in hematological parameters; MCHC, HFR and RDW-CV may be a marker in disease model of SD rat. These data will provide useful clinical indices for disease research of SD rat.

Keywords: SD rat, hematological parameters, gender, age, disease research

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1. INTRODUCTION

Hematological parameters are an important data in clinical test (Kristensen et al., 2017; Ying et al., 2017). According to the quantity changes and morphological distribution of red blood cells, white blood cells and platelets, early signs of many diseases can be found (Hildebrand et al., 2012; Stotz et al., 2013). Blood routine is an important reference for the study of toxicology and toxic reaction, and it can be used to determine whether the exogenous subjects have toxic effects on blood of the organism (Chen et al., 2017). Because most of the biomedical researches can't be conducted directly on humans, it is particularly important to select reliable laboratory animals. The data of hematological parameters which were found in early research might differ in different degrees as SD rats grew older (He et al., 2017; Jakob et al., 2014). This suggests that different studies may need rats with different ages.

SD rat is a laboratory animal of outbred stocks and clear genetic background, which are most commonly used for pharmacodynamics and toxicology research (Crincoli et al., 2016; Hancı et al., 2018). Therefore, it is a great significance to select SD rat for disease research by understanding the variation of hematological parameters with different ages and genders. However, in previous studies, the hematological parameters were not comprehensive enough and may be not 100% accurate, or the values might be affected by nutrition, animal housing, and experimental methods (Cora et al., 2012; Heet al., 2017). Despite different hematological parameters for SD rats have been provided in some studies, the values are more than 5 years. The recent study of hematological parameters lack reference values of age (Delwatta et al., 2018). In addition, these publications lack sufficient data for a detailed analysis for SD rats by sex and age.

The hematological parameters of normal SD rat are very important for laboratory animal selection. Jacob Filho et al. research the hematological parameters of different ages on Wistar rats (Jacob Filho et al., 2018), whereas researchers neither tested enough

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