METHODS Replicative Senescence model was established by serial subcultivation of primary HUVECs in vitro . Then, Cells were divided into control group, model group and drug group, and after cultured 48 hour, the levels of the interleukin (IL-6) and the tumor necrosis factor α (TNF-α) in cellular supernatant are to be measured via ELISA; and the senescent cells was examined by the senescence-related beta galactosidase (SA-β-gal) staining.

RESULTS Compared with the control group, the levels of IL-6 and TNF-α significantly increased, meanwhile the number of SA-β-gal-positive cells significantly increased in the model group, However, after treated with 80μmol/L Ginsenoside Rb1, the levels of IL-6 and TNF-α significantly decreased, meanwhile the number of SA-β-gal-positive cells significantly decreased.

CONCLUSIONS Ginsenoside Rb1 could reverse HUVECs replicative senescence. The anti-aging mechanism of ginsenoside Rb1 is closely related to its resistance against oxidative damage.

GW27-e0266

Ginsenoside Rb1 improves aged-related cognitive function and mitigates the intrinsic aging of mouse brain

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OBJECTIVES To testify whether ginsenoside Rb1 could improves aged-related cognitive decline and mitigates the intrinsic aging of mouse brain.

METHODS Female C57BL/6 mice were divided into three groups randomly: control group(4 months, n=12); model group(24 months, n=12) and drug group(24 months, n=12). In drug group, ginsenoside Rb1(20mg·kg-1·d-1) was injected into the mice abdominal cavity for 8 weeks, Control group and model group were peritoneal injection with the same amount of normal sodium. The effect of ginsenoside Rb1 on the learning and memory of C57BL/6J mice was tested by Morris water maze. The senescent neuron was examined by the senescence-related beta galactosidase (SA-β-gal) staining.

RESULTS Compared with the control group, the aged-related cognitive function significantly declined, and the number of SA-β-gal-positive cells significantly increased in the model group, However, after treated with Ginsenoside Rb1, the aged-related cognitive function were improved significantly, meanwhile the number of SA-β-gal-positive cells significantly decreased.

CONCLUSIONS Ginsenoside Rb1 could improve aged-related cognitive function and mitigate the intrinsic aging of mouse brain.

GW27-e0268

A study on the mechanism of the SIRT1 in Ginsenoside Rb1 against the intrinsic aging of mouse brain

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OBJECTIVES To study the mechanism of the SIRT1 in Ginsenoside Rb1 against the intrinsic aging of mouse brain.

METHODS Female C57BL/6 mice were divided into three groups randomly: control group(4 months, n=12); model group(24 months, n=12) and drug group(24 months, n=12). In drug group, ginsenoside Rb1(20mg·kg-1·d-1) was injected into the mice abdominal cavity for 8 weeks, Control group and model group were peritoneal injection with the same amount of normal sodium. The SIRT1 mRNA and protein expression in hippocampus in each group are to be detected respectively in the methods of RT-PCR and Western blot. The senescent neuron was examined by the senescence-related beta galactosidase (SA-β-gal) staining.

RESULTS Compared with the control group, the SIRT1 mRNA and protein expression significantly decreased, but the number of SA-β-gal-positive cells significantly increased in the model group, However, after treated with Ginsenoside Rb1, the SIRT1 mRNA and protein expression significantly increased, but the number of SA-β-gal-positive cells significantly decreased.

CONCLUSIONS Ginsenoside Rb1 could mitigate the intrinsic aging of mouse brain. The anti-aging mechanism of ginsenoside Rb1 is closely related to its regulation of the SIRT1.

GW27-e0269

A study on the mechanism of the eNOS/NO system in Ginsenoside Rb1 against the intrinsic aging of mouse brain

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OBJECTIVES To study the mechanism of the eNOS/NO system in Ginsenoside Rb1 against the intrinsic aging of mouse brain.

METHODS Female C57BL/6 mice were divided into three groups randomly: control group(4 months, n=12); model group(24 months, n=12) and drug group(24 months, n=12). In drug group, ginsenoside Rb1(20mg·kg-1·d-1) was injected into the mice abdominal cavity for 8 weeks, Control group and model group were peritoneal injection with the same amount of normal sodium. The eNOS and NO protein expression in hippocampus in each group are to be detected respectively in the methods of RT-PCR and Western blot. The senescent neuron was examined by the senescence-related beta galactosidase (SA-β-gal) staining.

RESULTS Compared with the control group, the eNOS and NO mRNA and protein expression significantly decreased, but the number of SA-β-gal-positive cells significantly increased in the model group, However, after treated with Ginsenoside Rb1, the eNOS and NO mRNA and protein expression significantly increased, but the number of SA-β-gal-positive cells significantly decreased.

CONCLUSIONS Ginsenoside Rb1 could mitigate the intrinsic aging of mouse brain. The anti-aging mechanism of ginsenoside Rb1 is closely related to its regulation of the eNOS/NO system.

GW27-e0270

A study on the mechanism of the inflammatory response in Ginsenoside Rb1 against the intrinsic aging of mouse brain

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OBJECTIVES To study the mechanism of the inflammatory response in Ginsenoside Rb1 against the intrinsic aging of mouse brain.

METHODS Female C57BL/6 mice were divided into three groups randomly: control group(4 months, n=12); model group(24 months, n=12) and drug group(24 months, n=12). In drug group, ginsenoside Rb1(20mg·kg-1·d-1) was injected into the mice abdominal cavity for 8 weeks, Control group and model group were peritoneal injection with the same amount of normal sodium. The malonaldehyde(MDA) level in the mouse serum is to be measured via TBA; the superoxide dismutase(SOD) activity is to be determined in the method of xanthine oxidase; The senescent neuron was examined by the senescence-related betagalactosidase (SA-β-gal) staining.

RESULTS Compared with the control group, the activities of SOD significantly decreased, but the MDA level and the number of SA-β-gal-positive cells significantly increased in the model group, However, after treated with Ginsenoside Rb1, the activities of SOD significantly increased, but the MDA level and the number of SA-β-gal-positive cells significantly decreased.

CONCLUSIONS Ginsenoside Rb1 could mitigate the intrinsic aging of mouse brain. The anti-aging mechanism of ginsenoside Rb1 is closely related to its resistance against inflammatory response.

GW27-e0271

A study on the mechanism of the oxidative damage in Ginsenoside Rb1 against the intrinsic aging of mouse brain

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OBJECTIVES To study the mechanism of the oxidative damage in Ginsenoside Rb1 against the intrinsic aging of mouse brain.

METHODS Female C57BL/6 mice were divided into three groups randomly: control group(4 months, n=12); model group(24 months, n=12) and drug group(24 months, n=12). In drug group, ginsenoside Rb1(20mg·kg-1·d-1) was injected into the mice abdominal cavity for 8 weeks, Control group and model group were peritoneal injection with the same amount of normal sodium. The levels of the interleukin