expressions in thoracic aorta of high-fat diet groups but did not occur in the normal diet group.

Conclusions The results suggest that G-CSF exacerbates lipid abnormity and endothelium damage in hyperlipidemia rabbits, thereby resulting in the deterioration of atherosclerosis. ET-1/eNOS system and MMP-9/TIMP-2 family may regulate the progression.

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THE GRANULOCYTE COLONY-STIMULATING FACTOR PROMOTES ATHEROSCLEROSIS IN HIGH-FAT DIET RABBITS

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Background Granulocyte-colony stimulating factor (G-CSF) has been reported to improve the function of infarcted heart, but its effects on atherosclerosis are unclear.

Methods and Results We examined these and the potential mechanisms in the high-fat diet rabbit model. Six-month-old male New Zealand white rabbits, fed the high-cholesterol (1%) diet or not for 10 weeks, were injected with vehicle or G-CSF (15 μg/kg/day, five days/week for three weeks) subcutaneously daily. G-CSF similarly increased the number of circulating white blood cells (WBC) but not that of platelet both in normal and high-fat diet groups although platelet aggregation function was increased by G-CSF only in normal diet group. G-CSF further increased the plasma levels of total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-C) at an early phase and atherosclerotic lesions area in the thoracic aorta compared with the vehicle treatment only in high-fat diet group. High-fat diet-induced arterial endothelium damage and collagen hyperplasia were greatly aggravated by G-CSF. G-CSF up-regulated endothelin-1 (ET-1), matrix metalloproteinase (MMP-9) and tissue inhibitor of metalloproteinase-2 (TIMP-2) and down-regulated endothelial nitric oxide synthase (eNOS)