DARK DEPRIVATION AND MECHANISMS OF ATHEROSCLEROSIS DEVELOPMENT IN RATS

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Background

Constant dark deprivation disrupts circadian rhythms and central biological clocks, leading to systemic metabolic dysregulation and neuroendocrine disorders (Nelson R.J., Chbeir S., 2018).

Disruption of light-dark cycles is a significant environmental risk factor that contributes to dyslipidemia, vascular endothelial dysfunction, and accelerated atherogenesis.



Circadian rhythm disruption → Metabolic & neuroendocrine disorders → Dyslipidemia + endothelial dysfunction → atherosclerosis

Materials and Methods

Experimental Animals
40 male laboratory rats (180–220 g) divided into:
- **Experimental** group – constant light/dark deprivation
- **Control group** – 12:12 h light/dark cycle

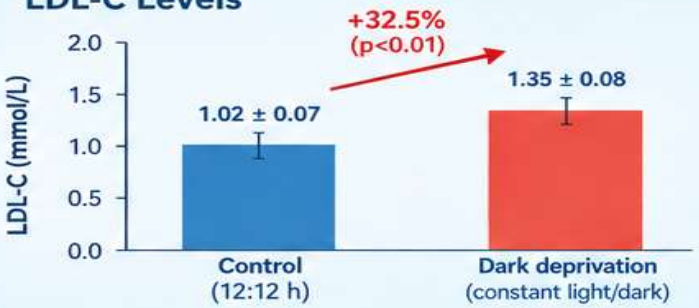
Lipid Profile Determination
Serum levels of LDL-C and HDL-C were quantitatively determined.

Reagents
Commercial diagnostic reagent kits by "Phidisit-Diagnostics" (Ukraine) were used according to the manufacturer's precipitation and spectrophotometric protocols.

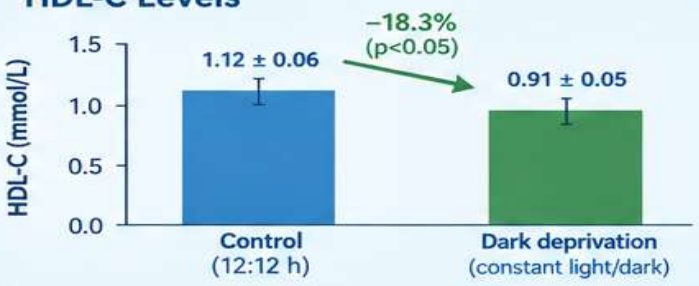
Statistical Analysis
Data were processed using standard parametric/ non-parametric methods (Mean±SEM).
Significance threshold: $p<0.05$.

Results

LDL-C Levels



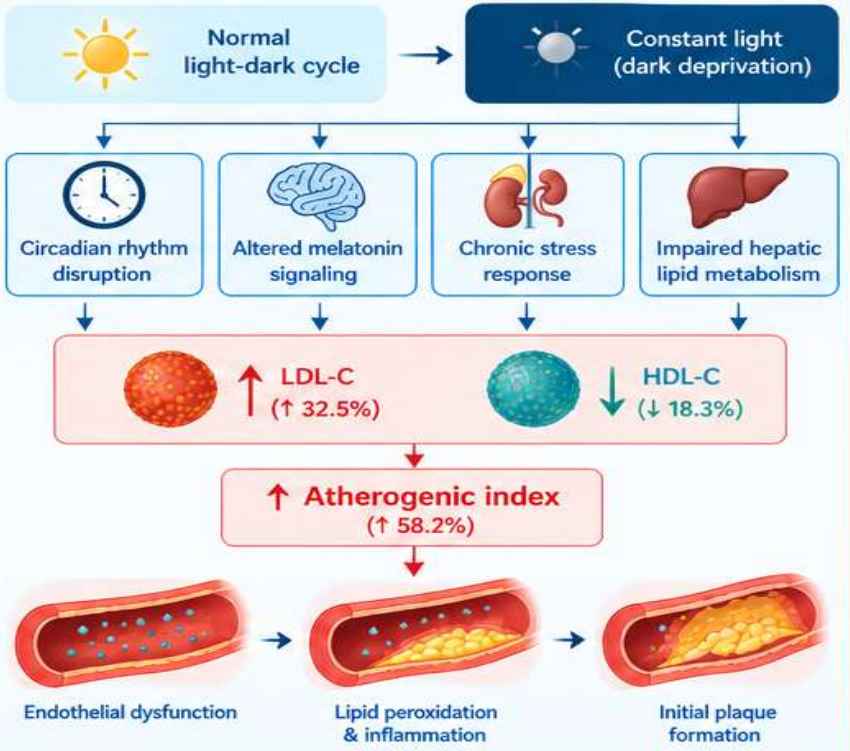
HDL-C Levels



Atherogenic Index (LDL-C/HDL-C)



Mechanisms of Atherosclerosis Development



Conclusion

Dark deprivation serves as a prominent environmental factor promoting lipid dysregulation characterized by elevated LDL-C and reduced HDL-C levels.

The findings confirm that circadian disruptions trigger metabolic pathways that significantly accelerate the development of atherosclerosis in rat models.