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The cardiovascular implications of low energy availability in physically active females: a systematic review

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Low energy availability (LEA) is a state in which the body lacks sufficient energy to support normal bodily functions required to maintain health[1]. While LEA is frequently associated with a plethora of cardiovascular abnormalities, supporting research is limited and largely comprised of studies that are small and preliminary in nature[2]. To date, there has been no comprehensive synthesis of the evidence regarding the cardiovascular implications of LEA in female athletes and our review aims to address this.

This review followed the Cochrane Collaboration PRISMA protocol[3]. Various online databases (PubMed, One Search, Web of Science, Google Scholar) and manual searches were used, with a cut-off publication date set at November 2023. Search keywords were devised using the PICO framework[3]. Medical Subject Headings (MeSH) terms were used in PubMed. Boolean logic was applied to filter relevant results. Eligibility criteria included physically active to elite-trained female athletes, LEA risk determination, and cardiovascular measures (including cardiovascular disease (CVD) risk, biomarkers and/or non-invasive measures). Articles were screened, extracted, and quality assessed using the NIH Quality Assessment Tool. Three researchers were blinded to each other's decisions to reduce potential biases and queries were resolved through reviewer discussion.

A total of nine studies were included. After evaluating the studies using the quality assessment tool, five studies were found to be moderate quality and four low-quality, due to factors including the inability to blind subjects or testers. The studies examined multiple cardiovascular health indicators and had mixed findings regarding the connection between LEA and potentially increased cardiovascular risks, aligning with previous research. Two studies suggested LEA leads to unfavourable blood lipid profiles which are known to lead to an increased risk of CVD[4], while three others found no significant difference in blood lipids due to LEA. One study identified twenty detrimental associations between lipids and clinical markers of hormone imbalance, dyslipidemia, and altered body composition, which are characteristic issues of LEA. These associations reflect the underlying metabolic disruptions caused by LEA typically involved with cardiovascular issues. Regarding autonomic control, vascular function and morphology, two research studies showed no significant association in heart rate variability, brachial flow-mediated dilation, pulse wave velocity, carotid artery reactivity, carotid-intima media thickness, or biomarkers reflecting endothelial activation with LEA. Although one study indicated abnormalities in flow-mediated dilation among those with LEA. Additionally, one study determined female athletes with LEA were more than 2.5 times more likely to experience cardiovascular issues (e.g., arrhythmias, valvular heart disease, coronary artery disease, and cardiomyopathy) as determined by the preparticipation examination.

This review highlights LEA's potential to elevate CV risks in female athletes. To validate these implications, further future larger-scale longitudinal studies with robust study designs are necessary to confirm the cardiovascular implications of LEA in female athletes.

References

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