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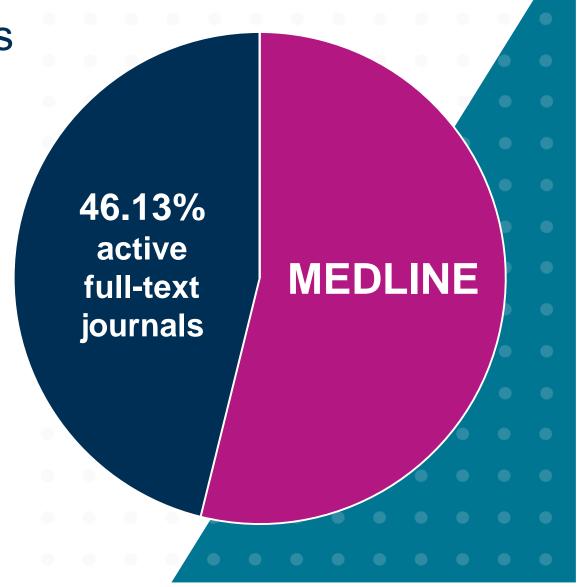
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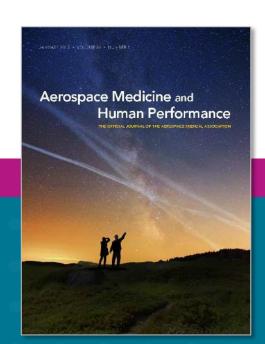
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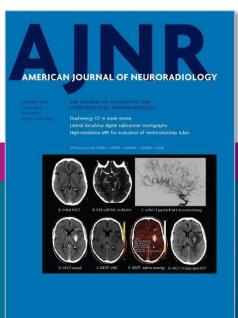
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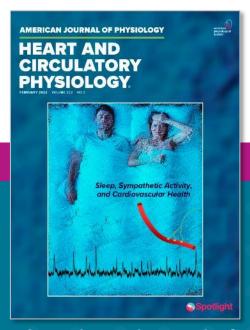
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South Asia & Southeast Asia	13	29	64
Spain	1	9	15
Sub-Saharan Africa	2	9	11
United Kingdom & Ireland	414	610	931
United States	82	247	467



Aerospace Medicine & Human Performance



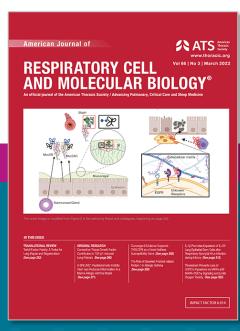
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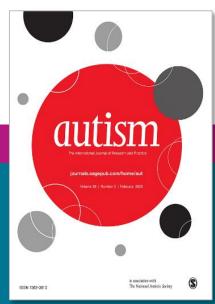
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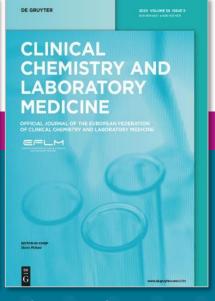
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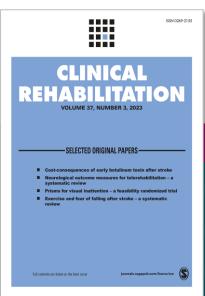
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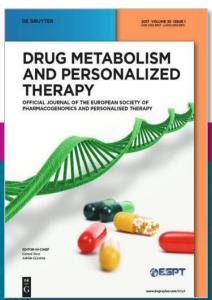
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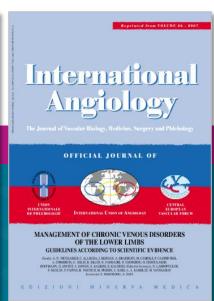
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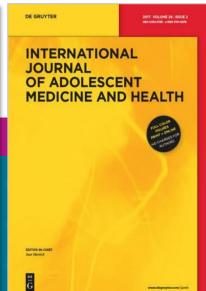
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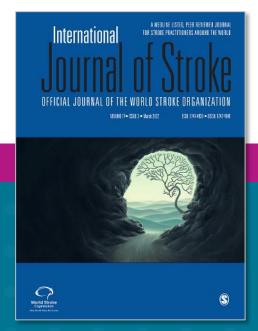
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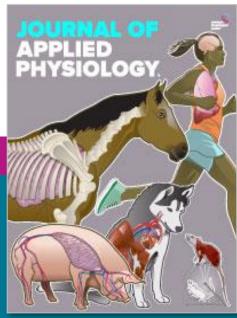
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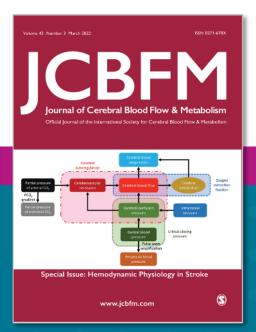
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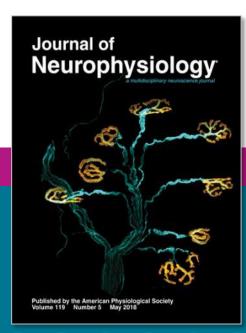
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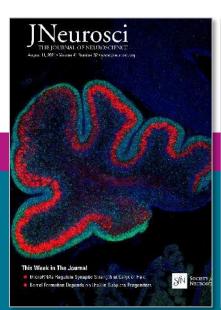
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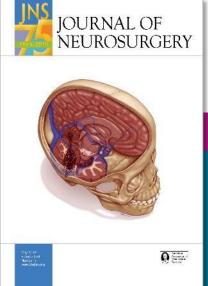
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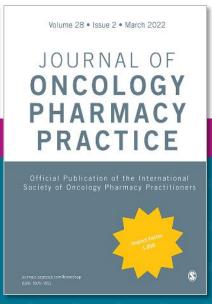
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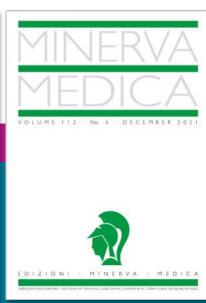
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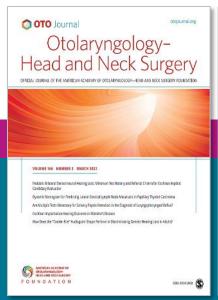
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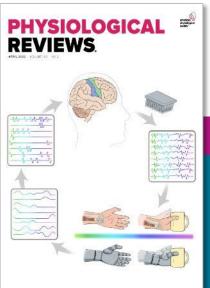


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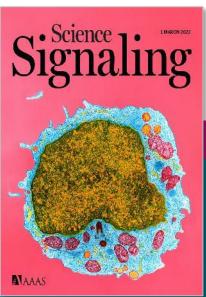
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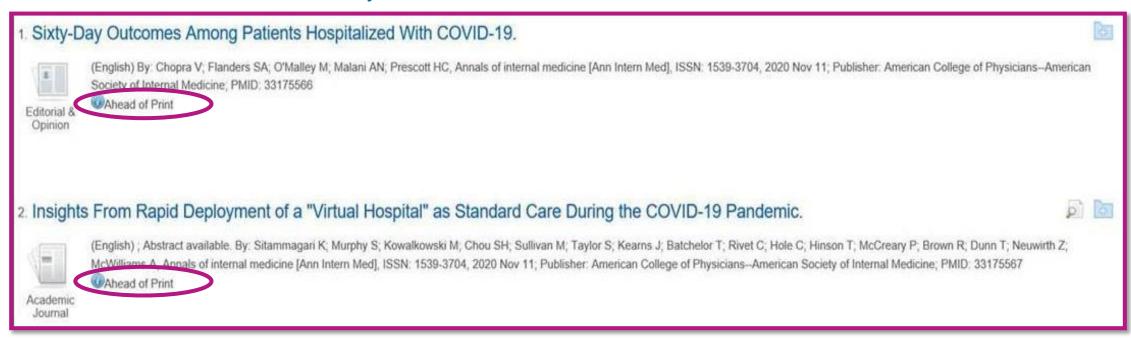
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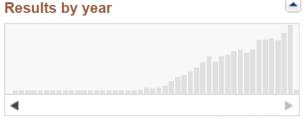
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<u>J Thromb Haemost.</u> 2017 Mar;15(3):575-585. doi: 10.1111/jth.1

Association of ficolin-3 with abdo

Full Records (Abstract Views)

Via PubMed

Fernandez-García CE¹, Burillo E¹, Lindholt JS², Martinez-Lopez D¹, Pilety K³, Mazzeo C⁴, Michel JB⁵, Egido J^{1,6}, Garred P³, Blanco-Colio LM^{1,7}, Martin-Ventura JL^{1,7}.

Author information

Abstract

clear

Essentials Abdominal aortic aneurysm (AAA) is asymptomatic and its evolution unpredictable. To find novel potential biomarkers of AAA, microvesicles are an excellent source of biomarkers. Ficolin-3 is increased in microvesicles obtained from activated platelets and AAA tissue. Increased ficolin-3 plasma levels are associated with AAA presence and progression.

SUMMARY: Background Abdominal aortic aneurysm (AAA) patients are usually asymptomatic and AAA evolution is unpredictable. Ficolin-3, mainly synthesized by the liver, is a molecule of the lectin complement-activation pathway involved in AAA pathophysiology. Objectives To define extra-hepatic sources of ficolin-3 in AAA and investigate the role of ficolin-3 as a biomarker of the presence and progression of AAA. Methods Microvesicles (exosomes and microparticles) were isolated from culture-conditioned medium of ADP-activated platelets, as well as from AAA tissue-conditioned medium (thrombus and wall). Ficolin-3 levels were analyzed by western-blot, real-time PCR, immunohistochemistry and ELISA. Results Increased ficolin-3 levels were observed in microvesicles isolated from activated platelets. Similarly, microvesicles released from AAA tissue display increased ficolin-3 levels as compared with those from healthy tissue. Moreover, ficolin-3 mRNA levels in the AAA wall were greatly increased compared with healthy aortic walls. Immunohistochemistry of AAA tissue demonstrated increased ficolin-3, whereas little staining was present in healthy walls. Finally, increased ficolin-3 levels were observed in AAA patients' plasma (n = 478) compared with control plasma (n = 176), which persisted after adjustment for risk factors (adjusted odds ratio [OR].

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