Check for updates

OPEN ACCESS

EDITED AND REVIEWED BY Einar M. Sigurdsson, New York University, United States

*CORRESPONDENCE Peter C. Searson ⊠ searson@jhu.edu

[†]These authors have contributed equally to this work and share first authorship

RECEIVED 27 October 2023 ACCEPTED 20 November 2023 PUBLISHED 21 March 2024

CITATION

Zhao N, Chung TD, Guo Z, Jamieson JJ, Liang L, Linville RM, Pessell AF, Wang L and Searson PC (2024) Corrigendum: The influence of physiological and pathological perturbations on blood-brain barrier function. *Front. Neurosci.* 17:1328902. doi: 10.3389/fnins.2023.1328902

COPYRIGHT

© 2024 Zhao, Chung, Guo, Jamieson, Liang, Linville, Pessell, Wang and Searson. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.

Corrigendum: The influence of physiological and pathological perturbations on blood-brain barrier function

Nan Zhao^{1†}, Tracy D. Chung^{1,2†}, Zhaobin Guo^{1†}, John J. Jamieson^{1,3†}, Lily Liang^{1,2†}, Raleigh M. Linville^{1,2†}, Alex F. Pessell^{1,2†}, Linus Wang^{1,2†} and Peter C. Searson^{1,2,4*}

¹Institute for Nanobiotechnology, Johns Hopkins University, Baltimore, MD, United States, ²Department of Biomedical Engineering, Johns Hopkins University, Baltimore, MD, United States, ³Department of Chemical and Biomolecular Engineering, Johns Hopkins University, Baltimore, MD, United States, ⁴Department of Materials Science and Engineering, Johns Hopkins University, Baltimore, MD, United States

KEYWORDS

blood-brain barrier, perturbations, dysfunction, brain health, neurovascular unit, brain pathologies

A corrigendum on

The influence of physiological and pathological perturbations on blood-brain barrier function

by Zhao, N., Chung, T. D., Guo, Z., Jamieson, J. J., Liang, L., Linville, R. M., Pessell, A. F., Wang, L., and Searson, P. C. (2023). *Front. Neurosci.* 17:1289894. doi: 10.3389/fnins.2023.1289894

In the published article, there was an error in Table 1 as published. In row 6, column 3, "Ab" should be "A β ". In row 5, column 3, "Ab" should be "A β ". The corrected Table 1 and its caption Blood-brain barrier (BBB) components and examples of dysfunction appear below.

There was also an error in Section 3. Temperature, paragraph 1, "when CBF is lowest" should be "when CBF is highest". The corrected paragraph appears below:

The average core body temperature (Tc) of healthy individuals is around 36°C (Mackowiak et al., 1992; Mackowiak and Worden, 1994; Sund-Levander and Grodzinsky, 2009; Protsiv et al., 2020). The average temperature of the brain is typically 1–2°C higher than Tc due to its high metabolic rate (Bain et al., 2015). Recent studies suggest that brain temperature varies with brain region and age, with temperatures as high as 40°C measured in the thalamus of healthy adults (Rzechorzek et al., 2022). The average brain temperature shows diurnal cycles, with the lowest temperature at night when CBF is highest, although these cycles are compromised with aging (Rzechorzek et al., 2022).

The authors apologize for these errors and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

BBB function	Molecular constituents	Dysfunction	Examples
Paracellular transport	Tight junctions (TJs) expressed by BMECs (e.g. claudin-5, occludin, ZO-1,)	 ↓ TJs - ↑ paracellular transport cell loss - ↑ paracellular transport mechanical disruption - ↑ paracellular transport 	Leaky brain:microbleeds or microhemorrhages (AD, CAA, stroke, TBI, healthy aging)
Passive transport	Small (< 500 Da) lipophilic molecules	see ATP-binding cassette efflux pumps (ABCs)	
Carrier mediated transport: solute carrier transporters (SLCs)	Energy transport (e.g. GLUT-1), amino acid transport (e.g. LAT-1), organic anion/cation transport (e.g. OATP1A2), nucleotides	↓ GLUT-1 - ↓ nutrient transport ↓ LAT-1 - ↓ protein & nucleotide synthesis, metabolism	Changes in respiration of cells in NVU
ATP-binding cassette efflux pumps (ABCs)	P-glycoprotein (P-gp, ABCB1), BCRP (ABCG2), MRP1 (ABCC1)	\downarrow P-gp - \uparrow passive transport of substrates, \downarrow clearance of A\beta	
Vesicular trafficking I receptor-mediated transport	Transferrin receptor (TfR), insulin receptor (IR), leptin receptor (LEP-R), low density lipoprotein receptor 1&2 (LRP1/2), receptor for advanced glycation end products (RAGE)	\downarrow LRP1 - \downarrow clearance of AB and APOE 2/3	
Vesicular trafficking II adsorption mediated transport	Histone, albumin	↓ MFSD2A - ↑ caveola-mediated vesicular transport	Shift to non-specific transport in aging
Ion transporters	Sodium pumps, calcium transporters, and potassium channels	changes in ionic homeostasis	
Other processes involving BMECs	Wound healing response, activation	activation - ↑ adhesion molecules (e.g. ICAM-1)	
Supporting cells	Loss or degeneration of SMCs, PCs; detachment of astrocytic end-feet	↓ signaling between BMECs and supporting cells	AD
Basement membrane	Endothelial, parenchymal (collagen IV, laminin, perlecan, agrin, nidogen)	↑ thickness in aging	Aging, AD

TABLE 1 Blood-brain barrier (BBB) components and examples of dysfunction.