COMMENTS

Another Crack in the Edifice of the Brain-Blood Interface

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The paper by Cosgrove *et al.*, "The choroid plexus removes β-amyloid from brain cerebrebrospinal fluid" (1), selected for the Best Paper Award in the Experimental Biology Category for 2005, represents early progress in the understanding of the flux of soluble amyloid monomeric amyloid in and out of the brain. The authors make a case for the presence of specific, possibly saturable mechanisms that regulate the passage of this interesting molecule across two models of rodent choroid plexus. Amyloid is a common molecule that is found throughout the body; however, because the disease associated with excess amounts of insoluble amyloid in the brain is Alzheimer dementia (2), the report is of special interest.

This is welcome progress in illustrating the complexity of traffic at the brain-blood interface. This previously monolithic border is now increasingly understood to have separate cellular and micro environmental layers and to perform previously unsuspected functions (3–5). To secure their claims, the authors will have to determine which cells are interacting with amyloid as it is taken up by the *ex vivo* choroid plexus, confirm whether these cells are present and functioning in the same manner in the cells that they used for tissue culture, and determine whether the remainder of the blood vascular system acts in the same way when confronted by soluble monomeric amyloid. However, this report by Cosgrove *et al.* exposes further the variety of mechanisms that may regulate the transfer of substances across borders in the brain. Its findings should serve as a stimulus for continuing study of the brain-blood interface.

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