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Letter to the Editor

RESPONSE TO THE REPORT BY DREXLER ET AL.

Dear Editor:

In their report, Drexler et al. took the pretext of our recent publication (Suda et al., 2001) to make some fundamental statements on the danger of cross-contamination between cell lines. We fully agree with their analysis. It is a fact that even cell lines from cell banks do not automatically guarantee the identity of the cells as has been recently demonstrated with the ECV304 cell line. With the advent of the genotype fingerprinting, it can be expected that in the near future the identity of cells from cell line banks will be checked and certified.

In our publication we clearly state from the beginning that fingerprinting has revealed the genotypical identity of ECV304 with the T24 bladder carcinoma cell line (Dirks et al., 1999). The aim of our study originally was to establish a blood–brain barrier (BBB) model for permeation studies screening different cell lines, one of them being the ECV304 (acquired directly from the American Type Culture Collection [ATCC]). As it stands, no cell culture system of endothelial origin is available for Ussing chamber-type permeation studies, which rely on the formation of tight monolayers of the cells under investigation. Neither primary cultures of the rat brain endothelial cells nor PBMEC/C1–2, a porcine brain microvascular endothelial cell line (Teifel and Friedl, 1996), form tight monolayers as judged by transendothelial electrical resistance (TEER) measurements and permeation studies with mannitol (Suda et al., 2001). This is the reason that before the identification of ECV304 as a cross-contaminant, these monolayer-forming cells with high TEER values were widely used as a BBB model. Different suggestions have been made during the ongoing discussion regarding relevant BBB models for permeation studies in which the tightness of the cell layer plays a fundamental role. In this context, even the use of the well-characterized Madin-Darby canine kidney (MDCK) (McRoberts et al., 1981) has been proposed (Veronesi, 1996), despite its epithelial origin, on the basis of its high electrical resistance. Seen in this background, we found it appropriate to present a phenotypical comparison of ECV304 and the authentic T24 cell line with a typical endothelial cell line (PBMEC/C1–2) and a typical epithelial cell line (MDCK) (Suda et al., 2001). It is important to note that phenotypical differences between ECV304 cells from different sources, namely, the ATCC and the ECACC, have previously been reported (Scism et al., 1999). This may explain the discrepancies to our results pointed out by Drexler et al., who did not specify the source of the cells used for their unpublished results mentioned in their report. In summary, it is up to every researcher to make the choice of adequate experimental systems under the conditions that it is well defined and declared.

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