Re: CD157 in Ovarian Carcinoma: How Does It Help Us?

In their editorial, Annunziata and Birrer (1) discussed some of the possible mechanisms by which CD157 may be of clinical use in ovarian carcinoma, as reported by Ortolan et al. (2). Annunziata and Birrer correctly defined CD157 as an ectoenzyme and receptor. However, the idea that the CD157 ligand is CD31 is suggestive but flawed because we and others have failed to find evidence of this interaction (3). It is CD38, another member of the ADP-ribosyl cyclase family, that has been documented to interact with CD31 (4).

Human CD157 and CD38 have many features in common: they share genetic origin and have similar primary and tertiary structures. They both cleave the substrate nicotinamide adenine dinucleotide enzymatically (5) and are implicated in the regulation of calcium homeostasis. However, they also have important differences and appear to lead biologically different lives. CD157 is a glycosylphosphatidylinositol-anchored glycoprotein, whereas CD38 is a type II transmembrane glycoprotein; moreover, these proteins have quite different patterns of expression and, consequently, interactions (6). Part of the functional repertoire of CD38 is implemented in vitro through its interaction with CD31 (4). This is not the case for CD157, which appears unable to interact with CD31. Indeed, the lack of a nonsubstrate ligand is the reason why we analyzed the functions of CD157 using agonistic antibodies, and the search for a ligand for this orphan receptor is a priority of our laboratory. We agree with Annunziata and Birrer that the role of CD157 in angiogenesis warrants investigation: CD157 is indeed expressed in stromal and endothelial cells, and it participates in endothelial cell signaling (7).

Having generated more questions than answers with our report, it is important that the basics of CD157 be correct as we proceed with further investigation into the biological role of CD157 in ovarian cancer.

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References

Notes
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