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Novel anticancer agent, SQAP, binds to focal adhesion kinase and modulates its activity

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SQAP is a novel and promising anticancer agent that was obtained by structural modifications from a natural compound. Previous assays demostrated that SQAP inhibits angiogenesis *in-vivo* resulting in increased hypoxia and reduced tumor volume with low side effects. In this study, the mechanism by which SQAP modifies the tumor microenvironment was revealed through the application of a T7 phage display screening. This approach identified five SQAP-binding proteins including sterol carrier protein 2, multifunctional enzyme type 2, proteasomal ubiquitin receptor, UV excision repair protein and focal adhesion kinase (FAK). All the interactions were confirmed by surface plasmon resonance analysis. Since FAK plays an important role in cell turnover and angiogenesis, the influence of SQAP on FAK was the principal goal of this study. We analyzed FAK-SQAP binding through a docking assay. Moreover, SQAP decreased FAK and FAK's downstream signaling phosphorylation in addition to cell migration in human umbilical vein endothelial cells and A549 cancer cells. These findings suggest that inhibition of FAK phosphorylation works as the mechanism for the anti-angiogenesis activity of SQAP.

Biography

Jesus Izaguirre Carbonell obtained his degree in Pharmacy at Universidad Miguel Hernandez in Spain. Thereafter, he moved to Japan, where he is currently completing his PhD at Tokyo University of Science under Prof. Sugawara supervision, and where he also obtained his Master degree. He has published 4 papers to receive his PhD degree in renowned journals.

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