Expression of formyl-peptide receptors in human lung carcinoma

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The human formyl-peptide receptors FPR1, FPR2 and FPR3 belong to the G-proteincoupled receptor (GPCR) family and were first detected in phagocytic leukocytes and in monocytes. Their expression has also been demonstrated in several tissues and cell types at the protein or mRNA level and their relevant biological functions have emerged through the identification of high affinity host-derived agonists. Lung cancer remains the leading cause of cancer death worldwide. Tobacco smoking and air pollution exposure are mainly implicated in lung cancer development. The current use of molecular profiling technologies to assess DNA, RNA, protein and metabolites have provided substantial advances in our understanding of the molecular basis of cancer, leading to the potential development of more effective targeted therapies. The identification of markers involved in cell growth may further allow the molecular profiling of lung cancer. We investigated the possible role of FPRs as molecular markers in several types of lung carcinoma. We collected tumour tissue samples from six groups of patients (48 total) affected by lung cancer. Surgical biopsies were analyzed for expression of FPR isoforms both in tumoral and peritumoral tissue by real-time Polymerase Chain Reaction (PCR), western blot and immunofluorescence. Real-time PCR, western blot and immunofluorescence analyses showed that FPR expression is lower in types of human lung cancer tissues when compared to the surrounding peritumoral tissues. Since FPRs are involved in EGFR transactivation, these results suggest a potential role of the cross-talk mechanisms in tyrosine kinase inhibitors resistance observed in lung cancer treatment. The study of the mechanistic basis for the control of FPR expression in normal peritumoral versus tumoral tissues could provide the basis for new diagnostic and therapeutic interventions.

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References

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Keywords

Formyl-peptide receptors (FPRs); Lung Cancer; Biopctic Samples; real-time PCR.