# 人類舌鱗狀細胞癌中銅鋅超氧化物歧化酶之表現

# Expression of Copper-Zinc Superoxide Dismutase in Squamous Cell carcinoma of the Human Tongue

## 賴銘堂

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#### 摘要

背景;超氧化物自由基(superoxide radical: O2)被認爲何癌症的發生及病理機制有關係。在研究細胞的發生及治療機轉方面,O2 及具有消除 O2 能力的超氧化物歧化酶(superoxide dismutase; SOD)遂成爲受矚目的課題。若干文獻曾報告癌組織細胞可測出不同活性之 SOD;然而 SOD 在各類癌組織細胞內的實際存在情形及其含大量都未明。本研究之目的在初步檢測人類頭頸部癌組織細胞內 SOD 的可能存在情形及其分布特徵;並比較嚴組織內部同細胞之 SOD相對含量。方法:本研究以免疫組織化學法檢測16件人類分化良好舌鱗狀細胞癌組織內銅鋅超氧化物歧化酶(copper, zine-superoxide dismutase; CuZnSOD) 的表現情形,病檢測舌鱗狀細胞癌組織內各類細胞之CuZnSOD 的相對含量。 結果:人類分化良好舌鱗狀細胞癌組織內各類細胞之CuZnSOD 的相對含量。 結果:人類分化良好舌鱗狀細胞癌之癌細胞、舌之鱗狀上皮細胞、横紋肌細胞及血管內細胞內可檢測出高含量之 CuZnSOD,舌之其他組織細胞所檢測之 CuZnSOD 含量則相對偏低。 結論:人類舌鱗狀細胞癌之癌細胞所含CuZnSOD 之量較其他組織細胞爲高,除表現出其代謝率較高的特性外,亦表示其消除 O2 之能力較強,因而可能對 O2 所產生之傷害具較高之耐受性。

#### **Abstract**

BACKGROUND: Superoxide radicals(O2), by virtue of their ability to trasform proca-cinogen to carcinogen and can caus chemical modification of DNA, are delieved to be mutagenic and carcinogenic. Superoxide dismutases (SODs) witch can catalyze the dismutation of O2 are therefore considered to be important factors in the pathogenesis and treatment of cancer. Several types of cancer cell have been demonstrated to possess different levels of SODs. The localization and levels of SODs in most carcinomas of the

human head and neck not benn extensively investigated. METHODS: Using immunohistochemical methods this study was designed to detect the expression of copper-zinc SOD (CuZnSOD) in 16 specimens of well-differentiated squa-mous cell carcinoma of human tongue that received neither radiotherapy nor chemother-apy. The relative CuZnSOD levels in different cell types making up the carcinoma tissues were also evaluated. BESULTS: The results showed that the squamous carcinoma cells, squampus epithelial cells, striated muscule cells and vasular endothelial cells strong positive CuZnSOD immunostaining. CONCLUSIONS: These results may reflect the higher metabolism of squamous carcinoma cells, and may idicate a higher ability of aquamous carcinoma cells to scavenge O2, thereby enhancing their resistance to the adverse effects of O2.