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Detection of cervical lesions via cytological examination in patients in Erbil city, Kurdistan

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Background and Objectives: Cervical cytological examination is the standard screening test for cervical cancer. The objective of this study is to investigate cervical cytological lesions incidence in patients in Erbil city, Kurdistan, Iraq.

Patients and Methods: This study of cervical cytology was carried out on 1763 patients at private clinic in Erbil, Kurdistan, Iraq. The age of women ranged from 17–87 years.

Results: Out of the total patients (1763), the number of women diagnosed with CIN I, CIN II, CIN III and carcinoma of cervix were 58 (3.3%), 2 (0.1%), 5 (0.3%) and 4 (0.2%), respectively. Severe cervicitis was detected in 1552 (88.8%) women while mild and moderate cervicitis were found in 47 (2.7%) and 164 (9.3%) patients. The results revealed that highest percentages of patients with CIN, moderate and severe cervicitis were diagnosed in women of 30-50 years old. While all patients with carcinoma of cervix were detected in women of ≥50 years old; more than 96% of CIN and all carcinoma cases were associated with severe cervicitinds.

Conclusion: It was found that most cases of cervical inflammatory and CIN lesions were seen among women of 30-50 years old. Most CIN and SCC patients were found to be associated with squamous metaplastic cells and severe cervicitis.

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Nischarin, a novel tumor suppressor affects AMP kinase pathway to regulate breast tumorigenesis

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The AMP activated kinase protein kinase (AMPK) is a key cellular energy sensor and functions to maintain cellular energy homeostasis. Activation of AMPK allows cells to survive under conditions of energy stress by turning on ATP-producing catabolic pathways and inhibiting ATP-consuming anabolic processes. Mammalian target of rapamycin (mTOR1) is a central growth regulator which is inhibited by AMPK. mTOR1 promotes anabolic processes leading to cell growth. We previously identified and cloned a novel protein that we termed Nischarin, and we have shown that this protein inhibits cell migration, cell invasion and tumor growth in mouse xenograft models. The long term goal of our work is to elucidate the mechanism by which Nischarin regulates breast tumor progression and to identify novel therapeutic approaches to suppress tumor progression in Nischarin lacking breast tumors. In humans, Nischarin is underexpressed in breast cancers. Based on these observations, we hypothesize that Nischarin suppresses breast cancer development and progression. To test this hypothesis we have generated Nischarin conditional knockout mice. Our preliminary data revealed that cells derived from Nischarin null mice exhibited increased migration and invasion. Also, we show that Nischarin lacking tumor cells have low levels of active AMP kinase suggesting that AMPK plays an important role in Nischarin regulation of tumorigenesis. Furthermore, our data indicate that downstream of mTOR1 is affected by manipulation of Nischarin expression. Currently, we are finding ways to up-regulate AMPK in animal models. By gaining a better understanding of the AMPK-mTOR1-Nischarin signaling and their roles in the regulation of mammary growth and metastasis, we will be better equipped to bring cancer therapies to the bedside.

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