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Personality traits are important predictors of glycemic control in patients with diabetes

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Objective: Personality traits are important predictor of health behavior, correlated to patients compliance that effected to glycemic control. The aim of the present project was to explore the relationship between personality traits, and glycemic control in type two diabetes patients.

Methods: The measurements used were Chinese version Big-5 personality traits scale. 214 type two diabetes patients were middle aged to elderly, were recruited from the teaching hospital in North of Taiwan.

Result: Glucose values were associated with factor of age, diabetes duration (years), self-rated health status, level of fasting glucose and triglycemic and domain of personality traits. In analytic of poor glycemic control as HbA1C \geq show significant results related to: In additional year can increase 1.199 times; self-rated health status as poor had 40.44 times; increase in 10 mg/dl of fasting blood glucose increases 59%, and 39% of triglyceric and; neuroticism was a significant predictor (B=2.017) had 7.52 times resulting hyperglycemia.

Conclusion: Personality traits can offer new insights into variation of glycemic control among diabetes regimens. Negative affectivity as a facet of neuroticism is correlated to hyperglycemia. Thus, determining personality traits is an important cornerstone of education plan for clinical staffs and may have effectiveness in resulting the level of glucose, in addition to improve the quality of nursing care.

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Overcoming resistance to TRAIL-induced apoptosis in solid tumor cells by simultaneously targeting death receptors, c-FLIP and IAPs

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The discovery of the tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) and its death receptors DR4/5 changed the horizon of cancer research because TRAIL specifically kills cancer cells. However, the validity of TRAIL-based cancer therapies has yet to be established, as most cancer cells are TRAIL-resistant. In our research, we demonstrate that TRAIL-resistance of many cancer cell lines can be overcome after siRNA- or rocaglamide-mediated downregulation of cellular Fas-associated death domain-like interleukin-1 β converting enzyme inhibitory protein (c-FLIP) expression and simultaneous inhibition of inhibitors of apoptosis protein (IAP) activity using AT406, a pan-antagonist of IAPs. Combined triple actions of the TRAIL, the IAPs inhibitor, AT406, and the c-FLIP expression inhibitor, rocaglamide (ART), markedly improve TRAIL-induced apoptotic effects in most solid cancer cell lines through the activation of an extrinsic apoptosis pathway. Furthermore, this ART combination has no harm to normal cells. Among the 18 TRAIL-resistant cancer cell lines used, 15 cell lines become sensitive or highly sensitive to ART, and two out of three glioma cell lines exhibit high resistance to ART treatment due to very low levels of procaspase-8. However, c-FLIP downregulation by c-FLIP-siRNA or rocaglamide was cytotoxic to normal cells. Therefore, it is highly desirable to develop a specific disruptor or antagonist of c-FLIP-FADD interactions to avoid the cytotoxicity caused by changes in the cellular levels of c-FLIP in normal cells. Furthermore, based on the interaction of c-FLIP with FADD, we investigated on the specific antagonist of c-FLIP. We found that one peptide containing six amino acids could bind c-FLIPs (180aa) protein strongly by affinity chromatography. This finding will help us to get the specific antagonist to c-FLIP. Our study provides a rationale for the development of TRAIL-induced apoptosis-based cancer therapies.

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