Spontaneous and induced sister chromatid exchanges and delayed cell proliferation in peripheral lymphocytes of Bowen's disease patients and matched controls of arseniasis-hyperendemic villages in Taiwan.

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Abstract

A total of 15 newly-developed Bowen's disease patients and 34 age-sex-residence-matched controls were recruited from three arseniasis-hyperendemic villages in Taiwan to compare spontaneous and arsenic-induced sister chromatid exchanges (SCEs), proportion of cells with high frequencies of SCEs (HFCs), and replication index (RI) in their peripheral lymphocytes. Arsenic-induced Bowen's disease patients were found to have significantly higher spontaneous SCEs and HFCs and a lower spontaneous RI than in matched controls without or with adjustment for age, gender, cigarette smoking, alcohol drinking, tea drinking, status of major diseases, HBsAg carrier status and arsenic exposure indices through multivariate analysis. Sodium arsenite was found to increase SCEs and HFCs and to decrease RI in a dose-response pattern for both cases and controls. The arsenic-induced decrease in RI was significantly greater in arsenic-induced Bowen's disease patients than in matched controls. The arsenic-induced increases in SCEs and HFCs were also consistently, but not statistically significantly, higher in arsenic-induced Bowen's disease patients than in matched controls at all arsenite treatment levels of 0.5, 1.0 and 2.0 microM. The arsenic-induced increase in cytogenetic damages and decrease in cell proliferation among arsenic-induced Bowen's disease patients compared with matched controls may result from their long-term exposure to inorganic arsenic through consumption of high-arsenic artesian well water, elevated individual genetic and acquired susceptibility to arsenic-induced damage, or both.