1 Title

A Turkish police officer stands guard outside a Turkish police station in Istanbul November 8, 2007. REUTERS/Efkan Ala

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The study was carried out in accordance with the manufacturer's instructions (N., Western Sydney, Australia).

Dichalamine oxidase inhibitors (DIMs) (9, 10, and 14) had a significant effect on the expression of the putative carcinogenic caspase-2 and putative pNK-3 (pNK-3) genes in genotypes that had previously been identified as having the highest expression.

The same group of DIMs (12, and 16) had shown that in vitro expression of the putative pNK-3-

genotype was significantly decreased in the control genotype (Fig. 1A), indicating that the phenotype of the carcinogenic caspase-2/pNK-3 gene was not affected.

The expression of the putative pNK-3-
genotype was up-regulated in the control genotype (Fig. 1B), which
reveals that the putative carcinogenic caspase-2/pNK-3
genotype was not affected by DIMs (Fig. 1C). These results
suggest that the putative carcinogenic caspase-2/pNK-3
genotype was not affected by DIMs.
The activation of pNK-3 was also suppressed by DIMs (Fig. 1D).
The expression of the putative pNK-3-
genotype was up-regulated in the control genotype in response to DIMs (Fig. 1E),
which is consistent with the finding that the putative carcinogenic
caspase-2/pNK-3
genotype was not affected by DIMs (Fig. 1F), which
suggests the presence of a putative carcinogenic
caspase-2/pNK-3
genotype.
The restriction of the putative pNK-3-
genotype and the suppression of the expression of the putative
bactein-2/pNK-3
genotype was significant (Fig. 1G), indicating that DIMs
were not able to inhibit the expression of the putative
caspase-2/pNK-3
genotype, which was consistent with the observation that
the putative carcinogenic caspase-2/pNK-3
genotype was not affected by DIMs (fig. S4A).
The expression of the putative carcinogenic caspase-2/pNK-3

genotype was significantly decreased in the control genotype (Fig. 1G), which is consistent with the finding that the phenotype of the carcinogenic caspase-2/pNK-3 genotype was not affected by DIMs (Fig. 1G). The expression of the putative caspase-2/pNK-3 genotype was up-regulated in the control genotype (Fig. 1H), which remains consistent with the finding that the phenotype of the carcinogenic caspase-2/pNK-3 genotype was not affected by DIMs (Fig. 1I). These results suggest that the putative carcinogenic caspase-2/pNK-3 genotype was not affected by DIMs.

Discussion

The present study demonstrated that DIMs (9, 10, and 14) did not inhibit the expression of the putative caspase-2/pNK-3 genotype or the expression of the putative carcinogenic caspase-2/pNK-3 genotype. The putative carcinogenic caspase-2/pNK-3 genotype was up-regulated in the control genotype (Fig. 1K), which remains consistent with the observation that DIMs (9, 10, and 14) did not inhibit the expression of the putative caspase-2/pNK-3 genotype (Fig. 1L). This suggests that DIMs were not able to inhibit the expression of the putative caspase-2 genotype, which was consistent with the observation that the phenotype of the carcinogenic caspase-2/pNK-3 genotype was not affected by DIMs (Fig. 1M). DIMs were able to inhibit the expression of the putative caspase-2/pNK-3 genotype, which was consistent with the observation that the phenotype of the carcinogenic caspase-2 gen