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Chitosan Redifferentiates Dedifferentiated Chondrocytes

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When chick limb bud mesenchymal cells were cultured at confluent density, they became to obtain ability to synthesize type II collagen and proteoglycan. On day 7 of culture, most of cells were immunostained with anti type II collagen antibody but not with type I collagen indicating that these cells are differentiated chondrocytes. These chondrocytes dedifferentiated and lost their ability to synthesize type II collagen when they were passaged every 2-3 days until passage 4. The dedifferentiated chondrocytes were seeded on culture dishes coated with 1% chitosan solution. Expression of type II collagen was analyzed by western blot and immunostaining after the cells were cultured on chitosan film for 5 days. The cells regained the ability to synthesize type II collagen and kept differentiated until 15 days. Possible involvement of PKC and p38 MAPK in redifferentiation of dedifferentiated chondrocytes was examined and they were found to play a positive role in redifferentiation. These results suggest that chitosan has capacity to redifferentiate dedifferetiated chondrocytes and also showed that PKC and p38 MAPK perform a part of redifferentiation of chondrocytes.

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Measurement of Toxicity of Nine Insecticides to *Hylyphantes graminicola* (Araneae: Linyphiidae)

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The authors investigated the toxicity of nine common insecticides to *Hylyphantes graminicola* by dipping method. The results showed that three insecticides including monosultap, imidacloprid and Bt are no harmful to the spider. Avermectin had the highest toxicity to the spider, the orders of relative toxicity of other five insecticides were as follows: fipronil > deltamethrin > fenvalerate > phoxim > methamidophos. The report showed that biological insecticides were all not safe to spiders. *Hylyphantes graminicola* is the dominant species of spiders in the cotton fields in China (Dong et al., 1994). It feeds on the larva and imago of cotton aphid, bollworm, plant hopper, leafhopper and other pest insects. It distributes widely in China, including Hubei, Jiangxi, Jilin, Jiangsu, Zhejiang, Anhui, Fujian, Shandong, Hunan, Guangdong, Sichuan, Shanxi, Taiwan and other provinces (Feng, 1992; Zhao, 1993). In order to reduce the harm of pesticides to natural enemy, we should select the pesticides that are high effective to pests but low poisonous or nonpoisonous to natural enemy of pests. Therefore, to measure the toxicity of pesticides to *Hylyphantes graminicola* is very important for the using of pesticides in cotton fields scientifically and to protect the spiders from the harmfulness of pesticides.

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Identification and Functional Analysis of a p73-binding Protein, H-Ras Homolog

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p73 is a nuclear protein that is similar in structure and function to p53. Notably, the C-terminal region of p73 has a regulatory function through interactions with positive or negative regulator.In this binding protein, H-Ras homolog protein similar tostudy, we identified p73 H-Ras by using the yeast two-hybrid technique.We confirmed that H-Ras both in vivo and in vitro. Thishomolog protein binds full length of p73 association is mediated via amino acids 56-87 of H-Ras homolog protein and . Furthermore, H-Ras also interacts with p73.DNA-binding domain of p73 Overexpression of H-Ras homolog and H-Ras lead to increase the transcriptional activity of p73 as well as an apoptosis of p73. Ras proteins are known to be small membrane-localized guanine-nucleotide binding proteins. However, H-Ras homolog proteins and H-RasV12 are localized in the nucleus in addition to cytosol and plasma membrane. Moreover, interaction of p73 and H-Ras homolog protein occurs in nucleus. This data leads to the conclusion that association of represents novel pathway in nucleus where Ras signaling affectsRas and p73 mutiple targets.

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p300 is a Nuclear Integrator of Nuclear Factor-κB and p73β

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p73β, the newly identified p53 homology, is associated B prevents cellswith the induction of apoptosis or growth arrest, while NF- from apoptosis. One stimuli which induce two proteins suggests the possible regulatory mechanism between p73β and p65. In this report showed that both p73β and p65 could affect on each other's transcriptional activity. Endogenous tumor necrosis factor alpha (TNF-α) activated NF-κB inhibits endogenous wild type p73β transactivation. Conversely, treatment with NA (Vitamin K analog, 2, 3-dichloro-5, 8-dihydroxy-1, 4 naphtoquinone: NA) which is capable of induction of endogenous p73β down-regulate NF-κB activity. Both of p73β and p65 interact with the transcriptional protein p300. We showed that inhibitory effect of p73β and p65 was dependent on the binding to limited amount of p300 and overexpression of p300 diminished the competition between p73 and p65. Furthermore, this result showed that transcriptional activity these proteins were recovered by enough amount p300. Competition of binding of p73β and p65 for p300 has significance for transcriptional activity both of them. This process may be important in determining the fate of cell, in which both p73β and NF-κB.