meteorological disturbances that entrain pollution from Europe and Asia into westerly winds hit the U.S. west coast between February and May each year (*9*). Another study finds that a tripling of east Asian anthropogenic emissions from fossil fuel combustion by 2010 compared with 1985 levels could increase ground level monthly mean ozone concentrations in the western United States by 2 to 6 parts per billion (ppb), making attainment of the new U. S. ozone standard more difficult (*10*). Present background ozone concentrations in surface air over the United States are in the 25 to 55 ppb range.

Pollutant concentrations in snow, fish, wildlife, sediments, and Arctic inhabitants indicate that some substances transported into and across the Pacific may already be working their way into ecosystems and humans. A study on the Fraser River watershed in British Columbia concluded that toxic airborne pollutants from Asia may be a source of contamination in lake fish and sediments (*11*). Blais *et al.* (*12*) found surprisingly high POP concentrations in the snowpack of high mountains in the Canadian west. Edmonds *et al.* (*13*) found increased nitrates and sulfates in pristine streams in the Olympic National Forest on the coast of Washington State. Other studies document POPs and mercury in wildlife and human populations in the Arctic (*14*), pesticides in bald eagles of the Aleutian Archipelago (*15*), and very high polychlorobiphenyl (PCB) concentrations in some Pacific Northwest orca populations (*16*). In all of these cases, the origin of the pollutants is undetermined, but long-range atmospheric transport across the Pacific Ocean cannot be ruled out.

Increasing recognition of trans-Pacific air pollution is evident in several new international research programs. For example, ACE-Asia (Aerosol Characterization Experiment-Asia) (*17*), its attendant Pacific Rim Aerosol Network, and the TRACE-P experiment (TRAnsport and Chemical Evolution over the Pacific) (*18*) aim to quantify the properties and distribution of aerosols and other atmospheric species in the Asia-Pacific region. The Intercontinental Transport and Chemical Transformation of Anthropogenic Pollution (ITCT) project will focus on atmospheric transport and chemistry over the North Atlantic and North Pacific, and SOLAS (Surface Ocean Lower Atmosphere Study) (*19*) aims to elucidate the influence of pollutants on interactions of the marine biogeochemical system, the atmosphere, and climate in the Pacific and elsewhere. The U.S. Environmental Protection Agency (EPA), the National Oceanic and Atmospheric Administration (NOAA), and other government agencies are planning atmospheric inflow studies to the west coast of the United States.

Research into the dynamics of long-range transport, deposition, and impacts of atmospheric pollutants in the Pacific region is only beginning. The nature, magnitude, and spatial distribution of the pollutants and their effects are largely unknown. Greatly expanded interdisciplinary and international research effort is required before trans-Pacific air pollution and other environmental issues in the Pacific region can be addressed effectively.

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NOTA BENE: CANCER

The Killer Instinct of a p53 Target

About 50% of human cancers have mutations in the tumor suppressor protein, p53. When activated in response to DNA damage, this master transcription factor switches on the expression of target genes that either halt the cell in G_1 phase of the cell cycle so that its DNA can be repaired or induce it to self-destruct through an elaborate process called apoptosis. Now, Oda *et al.* (*1*) have identified a new p53 target gene, *p53AIP1*, that, when activated by p53, alters the mitochondrial membrane potential resulting in cell death.

The investigators isolated *p53AIP1*, which is not homologous to any other known genes, by searching for sequences in the human genome that bind to p53. In cultured fibroblasts exposed to γ irradiation, which damages the DNA and activates p53, expression of *p53AIP1* increased after 12 hours, reaching a maximum at 24 hours. This was surprising given that other p53 target genes such as $p21^{waf1}$ (which causes cell cycle arrest) are expressed 1 to 2 hours after radiation exposure, reaching maximum levels at 12 hours. When overexpressed in cultured glioblastoma cells, p53AIP1 becomes concentrated in the mitochondria and alters the electrochemical gradient across the mitochondrial inner membrane, inducing 50% of the cells to die within 48 hours.

Oda *et al.* next looked at whether the addition of phosphate groups (phosphorylation) to serine (Ser) residues in p53 changed after exposure of cells to different doses of ultraviolet radiation. At lower doses (10 to 15 J/m^2), Ser-15 and Ser-20 in p53 were rapidly phosphorylated, reaching a maximum at the same time as $p21^{waf1}$. In contrast, Ser-46 was phosphorylated only at doses of 20 J/m^2 or higher—intriguingly, the only doses at which p53AIP1 was expressed. Apparently, Ser-46 phosphorylation is a prerequisite for p53AIP1 production because if Ser-46 is replaced by alanine (which cannot be phosphorylated), p53 is unable to bind to the *p53AIP1* gene promoter and, thus, no *p53AIP1* mRNA is made.

The authors propose that cells have a two-tiered system to respond to DNA damage. At lower radiation doses causing DNA damage that can still be repaired, $p21^{waf1}$ and other p53 target genes that halt cell division or repair DNA are switched on; at higher doses causing DNA damage that is irreparable, p53AIP1 is expressed and the cells die. Elucidating the p53 target genes that induce cells to self-destruct is important, particularly given that missense mutations in amino acids close to Ser-46 in p53 have been reported in some bladder and lung cancers, suggesting that the normal mechanism for removing severely damaged cells has been circumvented in these tumors.

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