

The scientists initially expected to demonstrate that PBDEs displaced thyroid hormone from the TR site. Instead they found PBDEs may prevent the TR from interacting with TREs, possibly through effects on the TR DNA-binding domain that normally binds to TREs in thyroid hormone–regulated genes.

The team identified two PBDE compounds, BDE-209 and BDE-100, as playing important roles in suppressing transcription from several TREs. None of the hydroxylated PBDE metabolites evaluated significantly suppressed TR-mediated transcription.

Decreases in thyroid hormone levels have been shown to alter the complex treelike branching of Purkinje cell dendrites, which is critical to normal brain development. The authors previously reported effects of polychlorinated biphenyls on dendrites that they determined were mediated by effects on the TR. The current study showed the action

of BDE-209 on TR-mediated transcription also inhibited the growth and branching, or arborization, of Purkinje cell dendrites. These effects may disrupt other aspects of brain development, given that TR-mediated gene expression occurs in many other cells.

The scientists say their work also suggests the effects they observed are associated with additional pathways, such as disruption of intracellular signaling pathways that rely on calcium ion homeostasis. It is unknown how many genes have TREs; therefore, the new work indicates an important next step will be to unearth the mechanism by which PBDEs can disrupt or suppress those TRE-mediated development genes.

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Heat Effects Are Unique

Mortality Risk Depends on Heat Wave, Community Characteristics

During heat waves, higher-than-normal temperatures can present a deadly threat, with mortality occasionally doubling. Recent studies have demonstrated that heat-related mortality risk is influenced by the characteristics of the individual heat wave (such as heat intensity, duration, and timing in season). Researchers explored this relationship more fully in one of the largest multicity studies to date of heat wave impacts in the United States [*EHP* 119(2):210–218; *Anderson and Bell*].

The authors identified heat waves in 43 U.S. communities during the years

1987–2005. A heat wave was defined as 2 or more days in which temperatures exceeded the 95th percentile of warm season (May–September) temperatures for that community during the 19-year period. Each heat wave was characterized according to heat intensity (average mean temperature), duration in days, and the point in the season when the heat wave occurred.

The investigators estimated a 3.74% increase in average daily risk of nonaccidental death during the heat waves compared with non-heat wave days. Although longer and more intense heat waves were more common in the South, estimated effects of heat waves on mortality were greater in the Midwest and greatest of all in the Northeast. The authors attribute this phenomenon to Southern residents being perhaps more physiologically and behaviorally adapted to extreme temperatures. Nationwide, heat waves that occurred

earlier in the warm season appeared to have a greater effect on mortality than heat waves occurring later (an average 5.04% increase compared with an average 2.65% increase), as did hotter or longer heat waves.

Considering that heat waves are expected to become more common and intense in some areas as the Earth's climate changes, it is important to understand the factors that make individual communities vulnerable to heat-wave effects and that make individual heat waves more likely to cause excess deaths. The authors conclude it is important for officials to develop local response plans on the basis of heat-wave mortality trends in their own communities; when it comes to planning for health effects of heat waves, one size does not fit all.

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This Queens, New York, resident was photographed in the middle of a summer 2006 heat wave that ultimately would cause an 8% increase in nonaccidental deaths, including 40 heat-stroke deaths.