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When Heat and Acaricides Collide: How Spider Mites Survive Two Stresses at Once

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A spider mite exposed to scorching heat and an acaricide appears to face two completely different threats. At the cellular level, however, the two stresses may partly activate the same survival machinery. Recent research on *Tetranychus truncatus* has identified heat shock protein 70 (Hsp70) genes that respond not only to high temperature but also to sublethal exposure to several acaricides. When two of these genes were silenced, the mites became more vulnerable to both heat and chemical stress. This does not mean that hot weather automatically creates acaricide resistance. Instead, it reveals an important biological connection between climatic stress and chemical tolerance and suggests that future mite management may need to consider both together.

A Mite Under Double Pressure

For a tiny spider mite living on a crop leaf, a hot afternoon can be a severe physiological challenge. Its body temperature closely follows the surrounding environment, and prolonged heat can damage proteins, disturb cellular processes and increase oxidative stress. At the same time, a mite in a treated crop may encounter acaricide residues that place another kind of stress on its cells. Traditionally, these problems have often been considered separately: temperature belongs to ecology and climate, whereas acaricide tolerance belongs to toxicology and pest management. The mite itself, however, experiences them simultaneously. Research on *T. truncatus*, a polyphagous tetranychid, now suggests that some of the cellular tools used to survive heat may also help the mite cope with acaricide exposure (Song *et al.,* 2024a; Sun *et al.,* 2026).

The Mite's Molecular Repair Crew

One important part of this response involves heat shock proteins, commonly abbreviated as HSPs. Despite their name, these proteins do more than respond to heat. They act as molecular chaperones, helping other proteins fold correctly, preventing damaged proteins from forming harmful aggregates and assisting cells in recovering after stressful events. Hsp70 is one of the best-known families in this cellular protection system. A useful way to imagine Hsp70 is as an emergency maintenance crew inside the cell. When heat begins to distort proteins, the crew becomes more active, stabilizing cellular functions and helping damaged proteins recover. Similar Hsp70 responses to thermal stress have been reported in mites, showing that this protective system is an important component of their environmental adaptation (Wang *et al.,* 2021).

Five Genes, but Two Stood Out

Sun *et al.* (2026) identified five Hsp70 genes in *T. truncatus*, designated TtHsp70-1 to TtHsp70-5. Under controlled thermal stress at 38-42 °C, TtHsp70-1, TtHsp70-2 and TtHsp70-3 were strongly upregulated, indicating their involvement in heat adaptation. The more surprising result came when mites were exposed to sublethal concentrations (LC30) of three acaricides:

propargite, abamectin and fenpropathrin. TtHsp70-1 and TtHsp70-2 were induced by these chemical treatments as well. The same two genes were therefore responding to both sides of the problem, heat and acaricide exposure. This overlap suggested a possible mechanism of cross-tolerance, but gene expression alone could not prove that the proteins were actually helping the mites survive.

What Happened When the Defence Was Switched Down?

To test the function of these genes, the researchers used RNA interference (RNAi) to reduce the activity of TtHsp70-1 and TtHsp70-2. After the genes were silenced, heat-induced mortality increased by about 12.5-35.0%. The mites also became more susceptible to the tested acaricides, with chemical-induced mortality increasing by about 7.5-38.8%, depending on the treatment (Sun *et al.,* 2026). This is important because it provides functional evidence rather than a simple correlation. Disabling part of the mite's stress-protection machinery reduced its ability to handle both thermal and chemical stress. In this experimental system, Hsp70 therefore acted as part of a shared survival mechanism.

Heat Tolerance and Acaricide Tolerance May Share More Than Hsp70

Hsp70 may be only one part of a broader network. Li *et al.* (2023) showed that two cytochrome P450 genes, TtCYP3A2 and TtCYP4V2, contributed to high-temperature tolerance in *T. truncatus*. In another study, pyridaben-resistant and susceptible strains differed in antioxidant-enzyme responses under heat stress, and silencing the TtPOD4 gene increased mortality of the resistant mites at high temperature (Song *et al.,* 2024b). Together, these studies are building a more complicated picture of acaricide resistance and climate adaptation. The molecular systems associated with chemical tolerance do not necessarily operate independently from the systems mites use to withstand environmental stress.

But Heat Does Not Automatically Create Resistance

This distinction is essential. The Hsp70 experiment provides evidence for cross-tolerance at the molecular level under controlled conditions. It does not show that a period of hot weather will automatically transform a susceptible field population into an acaricide-resistant one. Poor control during hot weather should also not be diagnosed immediately as resistance. Application quality, dose, spray coverage, pest stage, population density and environmental conditions can all influence field performance. Before resistance is blamed, these operational factors should be checked. The useful lesson is more precise: temperature can be part of the biological context in which acaricide tolerance operates.

What Does This Mean for Mite Management?

The discovery does not justify a new pesticide dose or an immediate change in acaricide recommendation. Its value is in showing why mite management should become more biologically informed. Hot and dry periods deserve careful surveillance, and control decisions should be based on actual mite populations and crop damage rather than routine calendar spraying. When acaricides are needed, the recommended dose, spray volume and application method should be followed so that avoidable sublethal exposure caused by poor coverage or incorrect application is minimized. Repeated selection with the same mode of action should be avoided. Rotation of acaricide modes of action, conservation or augmentation of predatory mites where appropriate, cultural practices and threshold-based intervention can reduce dependence on repeated chemical treatments. Temperature should increasingly be interpreted alongside mite density, crop stage, treatment history and natural-enemy activity. Such integrated information is more useful than treating weather and acaricide resistance as unrelated problems.

Could the Mite's Shield Become Its Weak Point?

The most futuristic part of this research is RNA interference. If silencing TtHsp70-1 and TtHsp70-2 makes the mite more vulnerable to both heat and acaricides, these genes become interesting potential molecular targets. In principle, weakening a pest's stress-protection system

could create new management opportunities. However, this remains a research prospect, not a field recommendation. Efficient delivery of RNA to mites on crops, stability under field conditions, target specificity, environmental safety and cost all require further study. The immediate importance of the experiment is therefore the biological insight: a single protective system may help a crop pest cope with multiple stresses at the same time.

Conclusion

A spider mite does not experience climate and crop protection as separate chapters. Heat, host-plant condition, natural enemies and acaricides all act on the same small organism at the same time. Research on *Tetranychus truncatus* now shows that two Hsp70 genes can contribute to tolerance of both high temperature and acaricide exposure. Silencing these genes weakens the mite against both stresses, revealing a molecular bridge between thermal adaptation and chemical tolerance. For the farming community, the message is not that hot weather inevitably makes acaricides ineffective. The message is that mite management under a warmer and more variable environment cannot depend on chemicals alone. Careful monitoring, correct acaricide use, rotation of modes of action, protection of biological control agents and integrated mite management remain the most practical safeguards. At the same time, understanding the mite's own stress-survival machinery may reveal new targets for future crop protection.

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