

The Stress Response in Vitiligo

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In our current line of research, we want to understand the factors that guide the onset of a bout of depigmentation. Why do some people develop white skin after sunbathing or others after coming in contact with rubber? What is it that triggers skin whitening after someone has made an ugly fall or cuts the skin by broken glass? From earlier research, we know that such events are best summarized as “stress” and that cells respond by making “stress proteins”. These proteins are made to protect cells against the consequences of mishaps by protecting cellular contents from enzymatic breakdown. The same mechanism, with the same stress proteins is used throughout the living world to protect cells against temporary danger, putting the cell on hold until the stress has passed. How do these events affect the skin? And how can this lead to pigment loss?

We are investigating the expression of stress proteins in skin from vitiligo patients, comparing it to what can be observed in control skin. Also, we are looking for responses of different cell types to purified stress proteins. Stress proteins can help cells survive, but if the cell cannot be saved, the stress proteins spill out into the surroundings and can help trigger a skin immune response. This is because stress proteins can activate cells that aggressively support such immune response: the dendritic cell. These cells become active when they come in contact with stress proteins, recruiting other immune cells to the scene that can kill pigment cells and cause depigmentation. Our work is focused on defining why this extra function of stress proteins leaves pigment making cells especially vulnerable to an immune attack.

We presented our data at the May 2002 meeting of the Society for Investigative Dermatology in Los Angeles, California. This will help to get the word out that stress can lead to full-blown vitiligo in individuals with a tendency to develop autoimmune responses.