Our clinical and mechanistic understanding of vitiligo has progressed significantly in the past 5-10 years, and this has led to an improved approach to patient management. Recent discoveries include lesion patterns that indicate disease activity, chemicals that induce or exacerbate disease, and novel treatment targets that may soon lead to new, targeted therapeutic options.

Severe forms of vitiligo
- Vogt-Koyanagi-Harada syndrome – vitiligo, poliosis, alopecia, uveitis, meningitis
- Alezzandrini syndrome (7 published cases) – segmental vitiligo, poliosis, ipsilateral hearing loss and retinal detachment

Chemicals that induce vitiligo
- Monobenzone
- Rhododendrol, ingredient in Japanese skin lightening cream, recalled in 2013
- Permanent hair dyes, maybe others

Treatment targets in vitiligo – keys to success
- Melanocyte stress – antioxidants, but little convincing data
- Autoimmunity – blocking cytokines and cytokine signaling
- Melanocyte regeneration, growth, and migration – poliosis indicates lack of melanocyte reservoir for repigmentation

Simple treatment algorithm

Future Targeted Immunotherapy – focus on the IFN-γ-chemokine signaling pathway
- Cytokine and cytokine receptor antibodies, small molecule inhibitors
  - IFN-γ
  - CXCL10
  - CXCR3
- Cytokine signaling blockade – i.e. Janus Kinase (JAK) inhibitors
  - Tofacitinib (pan-JAK inhibitor) oral
  - Ruxolitinib (JAK 1/2 inhibitor)
    - Oral
    - Topical
References:

Website:
Umassmed.edu/vitiligo

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@HarrisVitiligo