Vitiligo and Oxidative Stress

A plain-language summary of: *Targeting Oxidative Stress to Treat Vitiligo: Clinical and Molecular Evidence*

Aprile et al., *Biomolecules*, 2026, 16, 612 -- doi:10.3390/biom16040612

1. What Is Vitiligo?

Vitiligo is a skin condition where patches of skin lose their color and turn white or light pink. This happens because special cells called melanocytes stop working and die. Melanocytes are the cells that make the pigment (color) in our skin, hair, and eyes. About 1 or 2 people out of every 100 get vitiligo. There are two main types. Non-segmental vitiligo (NSV) is the most common. It shows up on both sides of the body at the same time, like on both hands or both knees. Segmental vitiligo (SV) only appears on one side of the body. Vitiligo does not hurt, but it can make people feel embarrassed or sad, especially when patches appear on the face or hands where others can see them.

2. The Root Cause: Too Many Harmful Particles

Every cell in your body makes tiny harmful particles every day as a natural side effect of producing energy. Scientists call these reactive oxygen species (ROS). Think of them like sparks from a campfire -- a few sparks are normal and harmless, but too many can start a fire. The body has a 'cleanup crew' of helper proteins called enzymes that normally destroy these sparks before they cause damage. The most important cleanup enzyme is called catalase (CAT). Other helpers include glutathione peroxidase (GPx) and superoxide dismutase (SOD). Together they form a defense team that keeps harmful particles under control. This harmful buildup is called oxidative stress (OS) when there are too many harmful particles for the cleanup team to handle.

In people with vitiligo, this cleanup team breaks down. One harmful chemical in particular, hydrogen peroxide (H_2O_2), builds up to very high levels in the skin. It is the same chemical used to bleach hair, and inside a cell it acts like a poison. It breaks the enzymes that make skin color, damages the cell's energy source, and eventually kills the color-making cell.

Two other parts of the defense team also fail. A natural sponge chemical called glutathione (GSH) gets used up too fast. And a 'master switch' protein called NRF2 fails to turn on when it should. NRF2 normally acts like a fire chief, calling in more cleanup helpers when danger rises. Its inhibitor protein KEAP1 should release NRF2 when things go wrong, but in vitiligo this does not happen properly. NRF2 normally plugs into special spots on the DNA called antioxidant response elements (ARE) to switch on cleanup genes like HO-1. When NRF2 fails, the cell has no way to fight back.

3. Genes Play a Role

Some people are born with gene versions that make vitiligo more likely. Genes are like instruction manuals written in your DNA that tell your body how to work. Scientists have studied the DNA of large groups of people in studies called genome-wide association studies (GWAS) and found more than 50 spots in the genome where certain versions make vitiligo more likely. Some risky gene versions reduce the amount of cleanup enzymes a person makes. For example, some people are born with broken versions of the GSTM1 and GSTT1 genes, leaving them with fewer tools to remove harmful particles. Other gene variations, like those in PTPN22 and FOXD3, affect how the immune system behaves.

The strongest gene-based risk comes from human leukocyte antigen (HLA) genes. These genes control what the immune system learns to recognize as an 'enemy.' Certain HLA versions make it much more likely that the immune system will see melanocytes as a threat and attack them.

4. How Harmful Particles Destroy Color Cells

4a. Damaged Batteries

Mitochondria are tiny structures inside every cell that act like batteries, turning food into energy. In vitiligo melanocytes, harmful particles attack these batteries and the mitochondrial DNA (mt-DNA) inside them. A damaged battery produces even more harmful particles, which damage it further in a vicious cycle. Eventually the damaged battery releases a signal that tells the whole cell to self-destruct, using proteins called caspases as the scissors that cut the cell apart.

4b. The Protein Factory Breaks Down

The endoplasmic reticulum (ER) is the protein factory inside each cell. It is where tyrosinase (TYR), the enzyme that makes skin color, gets built. When harmful particles fill the cell, the factory starts producing broken proteins. The cell sends out an alarm called the unfolded protein response (UPR) through three sensors named PERK, IRE1, and ATF6. At first the alarm tries to fix things, but if the damage goes on too long, the alarm switches from 'fix it' to 'shut it all down,' and the cell dies. Interestingly, tyrosinase proteins that come out broken can leak out of the dying cell and become targets for the immune system.

4c. Leaky Waste Containers

Peroxisomes are tiny compartments in cells that handle hydrogen peroxide. Normally they break it down safely using catalase. But in vitiligo cells, catalase is too low, so hydrogen peroxide leaks out into the rest of the cell and causes damage everywhere. It is like having a leaky waste bin that poisons the whole room.

4d. Color Cells Fall Off the Skin

Melanocytes need to stick to the skin like tiles on a floor. Harmful particles break the 'glue protein' called FAK that holds color cells in place. They also turn on enzymes called MMPs that chew through the skin scaffold (ECM) underneath. Without anything to grip, color cells fall off and die.

4e. Color Production Stops

Even melanocytes that are still alive can stop making pigment. Hydrogen peroxide directly breaks the color-making enzyme TYR by attacking its copper center. It also turns off MITF, the 'boss gene' that tells the cell to make color in the first place. So even cells that have not yet died go quiet and stop producing pigment, along with the helper enzymes TYRP1 and DCT.

4f. Zombie Cells

Not all damaged color cells die right away. Some become 'zombie cells' -- they stop dividing and stop making pigment, but they keep releasing harmful chemicals into the skin around them. Scientists call this state senescence. You can spot these cells by markers like SA-beta-Gal, p16, and p21. The cloud of harmful chemicals zombie cells release is called the SASP. SASP inflames the skin around them and attracts more immune cells to the area, making vitiligo worse over time. The tumor-suppressor protein p53 pushes cells into this zombie state when it detects too much DNA damage.

5. The Immune System Joins the Attack

The damage to color cells does not stay quiet. When a melanocyte is hurt or dying, it releases alarm signals called DAMPs -- including a protein called HSP70 and a signal called HMGB1. These alarms are picked up by immune cells called dendritic cells (DC). The NLRP3 alarm system inside immune cells also fires, releasing IL-1 beta to start the immune response.

Dendritic cells take pieces of the dead color cells and show them to killer T cells (CD8+). These killer cells are now trained to hunt melanocytes. A chemical signal called interferon-gamma (IFN-gamma) spreads the attack. It turns on a pathway inside cells called JAK-STAT (especially STAT1), which causes the skin to release chemical guides called CXCL9 and CXCL10. These guides act like breadcrumbs leading more killer T cells straight to the skin using a sensor on their surface called CXCR3.

Once there, killer T cells look for color-cell proteins displayed on the MHC-I screens on melanocytes and destroy them. Helper T cells (CD4+) encourage the attack, while extra signals from TNF-alpha and IL-17 keep the fire going. Peacekeeping immune cells called Tregs are too few and too weak to stop it. This creates a self-feeding loop: more harm leads to more immune attack, which causes more harm.

6. Treatments

6a. Light Therapy

The most widely used treatment is narrow-band UVB (NBUVB) light therapy. Patients sit under a special lamp a few times a week. This works in several ways: it helps turn on the NRF2 cleanup system, calms down the attacking T cells, grows more Tregs, and wakes up sleeping color cell stem cells in hair follicles using the WNT and cAMP pathways. Combining NBUVB with a man-made version of a natural hormone called alpha-MSH (which binds to the MC1R door on color cells) works even better -- about half of patients see significant color return, compared to only about 1 in 8 with light therapy alone.

6b. JAK Inhibitor Medicines

The biggest treatment advance in recent years is a group of medicines called JAK inhibitors (JAKi). They block the JAK switches that interferon-gamma uses to spread the immune attack, reducing CXCL9 and CXCL10 and stopping killer T cells from reaching the skin. One of these medicines, ruxolitinib cream, was approved by the FDA in 2022 as the first medicine specifically designed to bring color back in vitiligo. In trials measuring F75VASI (75% color return on the face), about 30 out of 100 patients reached this goal after one year. Pill versions are also being tested for widespread vitiligo.

6c. Antioxidant Helpers

Because too many harmful particles are the root cause, researchers have tested treatments that act as cleanup helpers. N-acetylcysteine (NAC) helps the body make more GSH (the natural sponge). Compounds that switch on the NRF2 master switch (like sulforaphane from broccoli sprouts) are being studied. A cream that mimics catalase to break down hydrogen peroxide in the skin has also been tested, with mixed results.

6d. Creams That Calm the Immune System

Steroid creams (TCS) and calcineurin inhibitor creams (TCI -- like tacrolimus) are usually the first treatments tried for small patches of vitiligo. They calm the immune attack on color cells but do not fix the underlying hydrogen peroxide problem. They work best when combined with light therapy.

7. How Doctors Track the Disease

Doctors can measure certain chemicals in the blood to see how active vitiligo is. The homing signals CXCL9 and CXCL10 are higher when vitiligo is spreading fast. They can also predict whether a patient will respond to JAK inhibitor treatment. Damage markers like MDA (from fatty cell wall damage) and 8-OHdG (from DNA damage) show how much harm is ongoing. Doctors use scoring tools like VASI to measure how much skin is affected and VIDA to track whether the disease is getting better or worse.

8. Conclusions

Vitiligo is caused by a chain reaction that starts with too many harmful particles in color cells. When the cleanup systems fail, hydrogen peroxide builds up and damages or kills melanocytes. Dying cells send alarm signals that pull the immune system into the attack. The JAK-STAT1 and IFN-gamma pathway keeps the attack going. This creates a loop that is hard to break.

The good news is that scientists now understand this process much better, and treatments are improving fast. JAK inhibitor creams can already bring color back. Combining them with antioxidant treatments (to fix the harmful particle problem) and NBUVB light therapy (to restart color cell growth, guided by alpha-MSH) is the most promising path forward. More large randomized controlled trials (RCTs) using standard scoring tools like VASI and F75VASI will help doctors figure out the best combination and order of these treatments.

Reference

Aprile, N., Scano, S., Bellei, B., Marini, A., & Filoni, A. (2026). Targeting oxidative stress to treat vitiligo: Clinical and molecular evidence. *Biomolecules, 16*(4), 612. https://doi.org/10.3390/biom16040612

Glossary of Acronyms

The Disease

NSV -- Non-Segmental Vitiligo [Return] [Wikipedia]

The most common type of vitiligo. White patches show up on both sides of the body at the same time, like on both hands or both cheeks. It is linked to the immune system attacking the wrong cells.

SV -- Segmental Vitiligo [Return] [Wikipedia]

A type of vitiligo that only appears on one side of the body. It usually stops spreading after a while and is less tied to the immune system.

GV -- Generalized Vitiligo [Wikipedia]

Another name for non-segmental vitiligo (NSV). White patches show up in many places across the body.

UP -- Universal Vitiligo [Wikipedia]

A very severe form of vitiligo where more than 80% of the skin loses its color.

UVB -- Ultraviolet B Light [Wikipedia]

A type of invisible light from the sun. Doctors use a safe, controlled version to treat vitiligo. It helps color cells grow back.

NBUVB -- Narrow-Band UVB [Return] [Wikipedia]

A special, narrow beam of UVB light used to treat vitiligo. It works well and has fewer side effects than older light treatments.

Oxidative Stress & Antioxidant Systems

ROS -- Reactive Oxygen Species [Return] [Wikipedia]

Tiny harmful particles that cells make as a normal side effect of producing energy. Think of them like sparks from a fire. A few sparks are fine, but too many can burn things down.

OS -- Oxidative Stress [Return] [Wikipedia]

What happens when there are too many harmful particles (ROS) and not enough cleanup helpers to get rid of them. Like having too much rust and not enough rust remover.

H2O2 -- Hydrogen Peroxide [Return] [Wikipedia]

A harmful chemical (the same one used to bleach hair) that builds up to dangerous levels in vitiligo skin. Too much of it breaks the color-making enzymes and poisons melanocytes.

CAT -- Catalase [Return] [Wikipedia]

A cleanup enzyme that breaks down hydrogen peroxide (H_2O_2) before it can hurt cells. In vitiligo skin, there is not enough of it, so H_2O_2 piles up.

GPx -- Glutathione Peroxidase [Return] [Wikipedia]

Another cleanup enzyme that destroys harmful particles inside cells. Often low in people with vitiligo.

SOD -- Superoxide Dismutase [Return] [Wikipedia]

An enzyme that turns one type of harmful particle into hydrogen peroxide (H_2O_2), which catalase then cleans up. Part of the body's cleanup team.

GSH -- Glutathione [Return] [Wikipedia]

A natural chemical inside cells that acts like a sponge, soaking up harmful particles. It is used up too fast in vitiligo skin and runs low.

Trx -- Thioredoxin [Wikipedia]

A helper protein that fixes other proteins damaged by harmful particles. It does not work well when hydrogen peroxide (H_2O_2) is too high.

NRF2 -- NRF2 (Master Antioxidant Switch) [Return] [Wikipedia]

A 'master switch' inside cells. When it is turned on, it tells the cell to make more cleanup helpers. In vitiligo, this switch often stays off when it should be on, leaving the cell defenseless.

KEAP1 -- KEAP1 (NRF2 Lock) [Return] [Wikipedia]

A protein that normally keeps NRF2 turned off. When there is too much damage, KEAP1 releases NRF2 so it can go to work. In vitiligo, this release does not happen properly.

ARE -- Antioxidant Response Element [Return] [Wikipedia]

A spot on the DNA where NRF2 plugs in to turn on cleanup genes. Like a socket that NRF2 uses to switch on the cell's defenses.

HO-1 -- Heme Oxygenase-1 [Return] [Wikipedia]

A protective enzyme that NRF2 turns on. It reduces damage inside cells. Lower than normal in vitiligo skin.

MDA -- Malondialdehyde [Return] [Wikipedia]

A chemical left behind when harmful particles attack the fatty walls of cells. Doctors measure it to see how much damage has occurred.

8-OHdG -- 8-Hydroxydeoxyguanosine [Return] [Wikipedia]

A marker that shows harmful particles have damaged the DNA inside cells. Found at higher-than-normal levels in vitiligo skin.

PTPN22 -- PTPN22 Gene [Return] [Wikipedia]

A gene that helps control how active the immune system is. Certain versions of this gene make vitiligo more likely to develop.

Cellular Machinery & Organelles

ER -- Endoplasmic Reticulum (Protein Factory) [Return] [Wikipedia]

The protein factory inside each cell. It builds and folds proteins correctly. When harmful particles flood the cell, this factory breaks down and can trigger cell death.

UPR -- Unfolded Protein Response [Return] [Wikipedia]

The emergency alarm the protein factory (ER) sends out when too many proteins come out broken. If the alarm runs too long, the cell shuts down and dies.

PERK -- PERK (ER Stress Sensor) [Return] [Wikipedia]

One of three alarm sensors inside the protein factory. It helps the cell slow down protein-making when things go wrong.

IRE1 -- IRE1 (ER Stress Sensor) [Return] [Wikipedia]

A second alarm sensor in the protein factory. It also activates inflammation signals when the factory is in trouble.

ATF6 -- ATF6 (ER Stress Sensor) [Return] [Wikipedia]

A third alarm sensor in the protein factory. When activated, it moves to another part of the cell and turns on emergency genes.

MITF -- MITF (Color Cell Boss Gene) [Return] [Wikipedia]

The main 'boss gene' for color cells. It tells the cell to make pigment. Harmful particles turn it off, so even living cells stop making color.

TYR -- Tyrosinase (Color-Making Enzyme) [Return] [Wikipedia]

The key enzyme that makes melanin (skin color). Hydrogen peroxide (H_2O_2) directly breaks it. It is also a main target of the immune system's attack in vitiligo.

TYRP1 -- Tyrosinase-Related Protein 1 [Return] [Wikipedia]

A helper enzyme that works alongside TYR to make melanin. The immune system sometimes attacks it too in vitiligo.

DCT -- Dopachrome Tautomerase (TYRP2) [Return] [Wikipedia]

Another helper in the melanin-making process. Like TYR and TYRP1, it can be attacked by the immune system in vitiligo.

HSP70 -- Heat Shock Protein 70 [Return] [Wikipedia]

A rescue protein that stressed cells make to protect themselves. When it leaks outside a dying cell, it acts like a fire alarm for the immune system.

DAMP -- Damage-Associated Molecular Pattern [Return] [Wikipedia]

Alarm signals released by damaged or dying cells. They tell the immune system that something is wrong and start an immune response.

FAK -- Focal Adhesion Kinase (Cell Glue Protein) [Return] [Wikipedia]

A protein that acts like glue, holding color cells to the skin. Harmful particles break this glue, and the color cells fall off and die.

ECM -- Extracellular Matrix (Skin Scaffold) [Return] [Wikipedia]

The natural scaffold (like a mesh or floor) that holds skin cells in place. Harmful particles can damage this mesh.

MMPs -- Matrix Metalloproteinases [Return] [Wikipedia]

Enzymes that cut through the skin scaffold (ECM). When there are too many of them, color cells lose their grip and fall off.

RNS -- Reactive Nitrogen Species [Wikipedia]

Harmful particles that contain nitrogen. Like ROS, they can damage cells in vitiligo skin.

NO -- Nitric Oxide [Wikipedia]

A gas made by cells. In large amounts it combines with other particles to form something even more harmful that can damage color cells.

Cell Death Pathways

NLRP3 -- NLRP3 Inflammasome [Return] [Wikipedia]

An alarm system inside immune cells. Harmful particles set it off, and it releases chemicals that start an immune attack against color cells.

HMGB1 -- HMGB1 (Cell Distress Signal) [Return] [Wikipedia]

A protein that leaks out of dying cells and acts like a distress signal, pulling more immune cells into the area.

p53 -- p53 (DNA Damage Detector) [Return] [Wikipedia]

A protein that checks a cell's DNA for damage. If there is too much damage, it tells the cell to stop dividing or die.

BCL2 -- BCL-2 (Cell Survival Protein) [Wikipedia]

A protein that helps cells stay alive. When it is low in color cells, those cells are easier to kill with stress.

caspase -- Caspases (Cell Death Scissors) [Return] [Wikipedia]

Proteins that act like scissors inside a dying cell. When turned on, they cut the cell apart in an orderly way, destroying it.

SA-b-Gal -- SA-beta-Galactosidase (Zombie Cell Marker) [Return] [Wikipedia]

A chemical marker found in zombie cells -- cells that are old and stuck but not quite dead. High levels in vitiligo color cells.

SASP -- Senescence-Associated Secretory Phenotype [Return] [Wikipedia]

The cloud of harmful chemicals that zombie cells release. These chemicals inflame nearby skin and attract more immune cells, making vitiligo worse.

p16 -- p16 (Cell Brake Protein) [Return] [Wikipedia]

A protein that stops cells from dividing. High levels are a sign that a cell has become a zombie cell.

p21 -- p21 (Cell Brake Protein) [Return] [Wikipedia]

Another protein that stops cells from dividing. Works together with p16 to keep zombie cells from multiplying.

RB -- Retinoblastoma Protein (RB) [Wikipedia]

Another braking protein. Works with p16 and p21 to keep zombie cells from dividing.

Immune System

CD8+ -- CD8+ Killer T Cells [Return] [Wikipedia]

White blood cells that can directly attack and destroy other cells. In vitiligo, they wrongly attack and kill color cells.

CD4+ -- CD4+ Helper T Cells [Return] [Wikipedia]

White blood cells that coach and encourage the killer T cells (CD8+), telling them where to go and keeping the attack going.

NK -- Natural Killer Cells [Wikipedia]

Immune cells that attack stressed or damaged cells without needing special instructions first. They may help destroy melanocytes in vitiligo.

DC -- Dendritic Cells [Return] [Wikipedia]

Immune cells that pick up pieces of dead color cells and show them to killer T cells, training those T cells to attack melanocytes.

IFN-g -- Interferon-Gamma [Return] [Wikipedia]

A chemical signal (called a cytokine) released by immune cells that tells other cells to ramp up the attack. It is the main troublemaker in vitiligo.

TNF-a -- Tumor Necrosis Factor-Alpha [Return] [Wikipedia]

Another attack signal that causes inflammation and can directly trigger color cells to die.

IL-17 -- Interleukin-17 [Return] [Wikipedia]

A signal chemical that helps keep the inflammation going in vitiligo skin.

IL-6 -- Interleukin-6 [Wikipedia]

A signal chemical that helps the immune system stay active and inflamed.

IL-1b -- Interleukin-1 Beta [Return] [Wikipedia]

A powerful attack signal released by the NLRP3 alarm system. One of the first signals that starts an immune response in vitiligo.

CXCL9 -- CXCL9 (Immune Homing Signal) [Return] [Wikipedia]

A chemical guide released by the skin that acts like a trail of breadcrumbs leading killer T cells to vitiligo patches. Higher in active vitiligo.

CXCL10 -- CXCL10 (Immune Homing Signal) [Return] [Wikipedia]

Another chemical guide that leads killer T cells to the skin. Doctors measure it in the blood to see how active vitiligo is.

CXCR3 -- CXCR3 (T Cell Navigation Receptor) [Return] [Wikipedia]

The 'nose' on killer T cells that sniffs out the CXCL9 and CXCL10 trails and guides the T cells to vitiligo skin.

Treg -- Regulatory T Cells (Peacekeepers) [Return] [Wikipedia]

Immune cells that normally calm down the immune system and stop attacks on the body's own cells. In vitiligo, there are not enough of them to stop the attack on color cells.

MHC-I -- MHC Class I (Cell ID Display) [Return] [Wikipedia]

A display screen on the surface of every cell that shows what proteins are inside. Killer T cells check this screen; if they see color-cell proteins, they attack the cell.

APC -- Antigen-Presenting Cell [Wikipedia]

Any immune cell (like a dendritic cell or macrophage) that captures pieces of proteins and displays them to T cells to decide if they are dangerous.

Signaling Pathways

JAK -- JAK (Cell Signaling Switch) [Return] [Wikipedia]

A family of on/off switches inside cells. They carry messages from interferon-gamma and other attack signals. Blocking JAK is one of the most effective treatments for vitiligo.

STAT -- STAT (Signal Partner to JAK) [Return] [Wikipedia]

Partners to the JAK switches. Once JAK activates them, they move into the cell's nucleus and turn on attack genes.

STAT1 -- STAT1 [Return] [Wikipedia]

The main STAT partner for interferon-gamma. It turns on the genes that make the homing signals CXCL9 and CXCL10.

STAT3 -- STAT3 [Wikipedia]

Another STAT partner. Helps keep the immune response going by supporting certain immune cells.

mTOR -- mTOR (Cell Growth Controller) [Wikipedia]

A controller that decides when a cell should grow or recycle its parts. Harmful particles can throw it off balance in melanocytes.

PI3K -- PI3K (Survival Signal) [Wikipedia]

Part of a chain of signals that help cells survive. When it does not work right, cells are more likely to die from stress.

AKT -- AKT (Survival Signal Partner) [Wikipedia]

Works with PI3K to promote cell survival. Reduced in vitiligo melanocytes, making them more vulnerable to dying.

WNT -- WNT Signaling Pathway [Return] [Wikipedia]

A set of signals that tells color cell stem cells to wake up and grow. Important for repigmentation -- getting color back in white patches.

MAPK -- MAPK (Stress Response Pathway) [Wikipedia]

A set of cell signals triggered by stress and harmful particles. When overactive, they push cells toward death.

NF-kB -- NF-kB (Inflammation Switch) [Wikipedia]

A switch that turns on inflammation genes. Harmful particles and attack signals turn it on in vitiligo skin.

cAMP -- cAMP (Cell Messenger) [Return] [Wikipedia]

A small chemical messenger inside cells. UVB light boosts it in color cell stem cells, helping them wake up and grow.

SCF -- Stem Cell Factor [Wikipedia]

A growth signal that tells color cell stem cells to stay alive and move toward white patches of skin. Lower than normal in vitiligo.

Genetics

GWAS -- Genome-Wide Association Study [Return] [Wikipedia]

A type of research that looks at the DNA of thousands of people to find which gene versions make a disease more likely.

SNP -- Single Nucleotide Polymorphism [Wikipedia]

A tiny difference in one letter of the DNA code between people. Some SNPs make vitiligo more likely to develop.

HLA -- Human Leukocyte Antigen [Return] [Wikipedia]

Proteins on cell surfaces that show pieces of proteins to the immune system. Certain HLA types make it much easier for the immune system to mistake color cells for enemies.

FOXD3 -- FOXD3 Gene [Return] [Wikipedia]

A gene that helps color cells develop properly. Certain versions may mean a person is born with fewer or weaker color cells.

GSTM1 -- GSTM1 Gene [Return] [Wikipedia]

A gene that makes a cleanup enzyme. Some people are born without a working copy, leaving them less able to remove harmful particles from cells.

GSTT1 -- GSTT1 Gene [Return] [Wikipedia]

Another cleanup gene. Like GSTM1, missing or broken copies reduce the body's ability to fight harmful particles, increasing vitiligo risk.

mt-DNA -- Mitochondrial DNA [Return] [Wikipedia]

The DNA found inside the cell's batteries (mitochondria). It is very easily damaged by harmful particles and is often damaged in vitiligo color cells.

Treatments & Clinical Terms

JAKi -- JAK Inhibitor [Return] [Wikipedia]

A medicine that blocks JAK switches to quiet the immune attack on color cells. Ruxolitinib cream was the first JAK inhibitor approved by the FDA to treat vitiligo.

TCS -- Topical Corticosteroids (Steroid Creams) [Return] [Wikipedia]

Steroid creams that reduce inflammation and slow the immune attack on color cells. Often the first treatment a doctor tries for vitiligo patches.

TCI -- Topical Calcineurin Inhibitors [Return] [Wikipedia]

Creams (like tacrolimus or pimecrolimus) that calm T cells in the skin without using steroids. Good for the face or skin folds.

PUVA -- Psoralen + UVA Phototherapy [Wikipedia]

An older light treatment that uses a pill to make skin sensitive to light, then shines UVA light on it. Mostly replaced by the safer NBUVB treatment.

NAC -- N-Acetylcysteine [Return] [Wikipedia]

A supplement that helps the body make more GSH (the natural sponge antioxidant). Sometimes used as an add-on treatment to help color cells survive.

a-MSH -- Alpha-MSH (Pigment Hormone) [Return] [Wikipedia]

A natural hormone that tells color cells to make more pigment. A man-made version called afamelanotide is combined with light therapy to boost color return.

VASI -- Vitiligo Area Scoring Index [Return]

A scoring tool doctors use to measure how much skin has lost color and whether treatment is working.

F75VASI -- F75VASI (75% Repigmentation Goal) [Return]

A treatment goal used in studies. It means 75% of the color has come back on the face after treatment. Used to measure if a medicine is working well.

PD-1 -- PD-1 (Immune Checkpoint) [Wikipedia]

A brake on the immune system. Cancer drugs that release this brake sometimes cause vitiligo as a side effect, showing how important T cell control is.

CTLA-4 -- CTLA-4 (Immune Checkpoint) [Wikipedia]

Another brake on the immune system. Releasing it with cancer drugs can also trigger vitiligo, confirming that killer T cells drive the disease.

MC1R -- Melanocortin 1 Receptor [Return] [Wikipedia]

The 'door' on color cells that alpha-MSH knocks on to tell the cell to make pigment. Activating it also helps protect against harmful particles.

Miscellaneous

QOLINDEX -- Quality of Life Index [Wikipedia]

A measure of how much a disease affects someone's daily life, happiness, and relationships. Vitiligo can cause anxiety and sadness, especially in people with naturally darker skin.

RCT -- Randomized Controlled Trial [Return] [Wikipedia]

The gold-standard way to test a medicine. Patients are randomly placed in either the treatment group or the no-treatment group to fairly compare results.

FDA -- U.S. Food and Drug Administration [Return] [Wikipedia]

The U.S. government agency that decides if medicines are safe enough to sell to patients. It approved ruxolitinib cream for vitiligo in 2022.

SCOPA -- Standardized Classification of Vitiligo

A scoring tool used in research to measure the pattern and spread of vitiligo patches in a standard way.

VIDA -- Vitiligo Disease Activity Score [Return]

A score that shows whether vitiligo is getting worse, staying the same, or getting better, based on recent changes.