

♥ Initial ideas ♥ (CRSF)

What do I want my project?

↳ Biology / Health

↳ What topics do I specifically want to explore?

↳ Skin

↳ Dermatology

↳ Eczema

Why do I want to do this?

↳ I have personal experience with Eczema

↳ There are so many factors that affect it

Jan 31st / 2026 February 4th 2026

- Explore the interactions between external & internal factors

Internal Factors examples

Biological predisposition = increased likelihood or susceptibility to developing a particular disease

↳ examples = genetics, immune sensitivity, skin barrier dysfunction

Physiological regulation

↳ stress response

↳ Gut / skin

External Factors

↳ Environmental

(climate, water, pollution, humidity)

↳ Lifestyle

• stress & sleep

February 4th 2026

Main Ideas

(biological predisposition)

Internal factors → immune sensitivity & skin barrier dysfunction

Internal factors → stress response and relation between skin and gut health (microbe, diet & gut permeability)

External factors → climate, humidity, water, pollution, geography (environmental)

External Factors → lifestyle (what you eat, sleep, stress)

Main idea

↳ explain interactions between internal / external factors and incorporate my own data by

sharing my personal experience in India, experiences of other students and dermatologists' POV

(if I can get an interview with one closely)

Harmon =
dry clondruff

Cleanser, serum,
aloe vera,
moisturizer
SUNSCREEN

Introduce → Idea that every year eczema is increasing among children and adolescents. Introduce my goal of explaining eczema and the interactions between internal/external factors.

Explain → start by explaining a normal skin barrier along with the key components of the skin

Internal Factors

↳ explain each individual factor in detail

1. Explain the Skin Barrier Dysfunction of a individual with atopic dermatitis

2. Explain immune sensitivity of an individual with eczema

3. Explain how overall gut health impacts an individual with eczema (recognize is is new science & new studies are being done on this)

External Factors

1. Environmental

↳ go into depth about how climate, humidity and water quality influences / triggers one's eczema

↳ include data about my summer trip to India / personal experience

2. Lifestyle

↳ explore how lifestyle, stress, sleep all correlate to eczema

External Factors / Internal Factors

↳ include now go into detail about how external & internal factors interact in Atopic dermatitis

↳ tie everything together

↳ present data of students experiences in my school

POTENTIALLY I CAN TRY TO
INTERVIEW A DERMATOLOGIST

↳ talk about this data & people's diff experiences & how things relate

Conclusion

Speak about the interaction of the factors and how each individual is different and has different experiences with eczema → eczema is different for everyone

CYSF brainstorming

Feb 4 2026

1. Perform Background Research
2. How does internal health and external triggers interact in Atopic Dermatitis?
 - problem/testable question
3. Thesis = Immune sensitivity, skin barrier dysfunction and overall gut health are some internal health factors that interact with environmental and lifestyle related triggers, impacting an individual with Atopic Dermatitis.
 - = Immune sensitivity, skin barrier dysfunction and overall gut health are just a few internal health factors that interact with environmental and lifestyle related triggers. these interactions have an impact on the severity of an individual's ~~ecz~~ Atopic Dermatitis.

Improved thesis

~~Immunes~~ Immune sensitivity, skin barrier dysfunction overall gut health are internal health factors that interact with environmental and lifestyle related triggers. these interactions impact the severity of an individual's Atopic Dermatitis

Feb 4 2026

Important

- ↳ use research from multiple sources / textbooks
- ↳ if sources disagree (ex gut health) state why & state disagreements

Scientific Principles

- ↳ Don't just say the facts
- ↳ explain how immune system responds, how skin barrier works, how inflammation works
- ↳ Say this at the start when explaining skin / normal barrier

Concepts

- ↳ use graphs / tables not just into the show the concepts
- ↳ cite sources

Results → logbook, summaries

Conclusion → conclude about the info & go to the underlying question. Talk about the interactions and how research has proven this

Further → Explain how this research can be used to experiment & create methods to deal with eczema.

Science Fair Notes

Feb 7th

(general information / problem)

→ Atopic dermatitis

can be characterized by itchiness, redness and intense itching

General Skin Information

↳ The skin is the heaviest and largest organ that has a role as the central sensory organ. The skin performs multiple tasks like protecting your body from germs, toxic materials, the cold and sun rays. It helps with regulating the body temperature and with the storage of water, fat and metabolic processes. There are 3 sections to the skin → the outer layer is called

Outer layer = is called the epidermis

middle layer = dermis

Inner most layer = Subcutis

The epidermis is the outermost layer of the skin that varies in thickness depending on the part of the body. It is mainly made up of keratinocytes that produce keratin. This layer of the skin is constantly renewing itself. New cells are made in the lower layers and are moved eventually moved to the top. When skin cells die off, they are shed resulting in a continuous cycle. The epidermis also contains cells like Merkel cells, lymphocytes, langerhans and melanocytes.

Summarization Feb 8th (2026)

From Sources

The epidermis is the outermost layer of the skin and varies in thickness depending on the part of the body. The role of the epidermis is to provide an effective barrier that separates the individual from the surrounding environment. It is primarily composed of keratinocytes which are cells that produce keratin. These cells establish a protective barrier, preserve moisture and prevent pathogens from entering the skin. This layer of the skin is always renewing itself. When new cells are built in the lower layers of the epidermis. When skin cells

The Dermis

The Dermis is the middle layer of the skin that is made up of a network of elastic collagen fibers. These fibers allow the skin to be strong as well as elastic. The Dermis contains connective tissue that attaches to muscles and bones. It also consists of a network of capillaries. Capillaries are the body's smallest blood vessels that ~~per~~ deliver nutrients and oxygen to surrounding cells. The dermis is also involved in temperature regulation, helping your body to cool down or preserve heat. This happens because of the blood vessels in the dermis that use the processes of vasodilation & vasoconstriction to regulate body temperature. This layer also contains sweat glands and sensory cells

Summaries for Background info (Feb 8th 2026)

The subcutis is mostly made up of fat and connective tissue. Just like the dermis, the subcutis contains lymphatic vessels, blood vessels, nerves, sweat glands, sebaceous glands and hair follicles. Between the dermis and subcutis are tiny cavities that contain tissue made out of fat and water. This fat layer ~~also~~ protects bones from injuries and sudden shock. Overall, the subcutis has the primary role of protecting the skeletal system, organs and muscles from harm.

Other components include

Macrophages: These are a type of white blood cell that use the process of phagocytosis to engulf foreign pathogens and present the antigens.

Lymphatic vessels: These are a network of vessels that are closely associated with the ~~own~~ circulatory system. They help to regulate substances fluid in the body and also ~~contain~~, contain a fluid called lymph and contain different type of white blood cells (example = lymphocytes (T cells, B cells, macrophages, monocytes or more).

Nerves = The nerves in this layer send electric signals to and from cells, glands and muscles. They are responsible in the transfer of info.

Fibroblasts = type of connective tissue cell which releases collagen

February 13th background research

Summary on Immune System's Role in Skin

The skin is a layer of protective barrier that guards your internal body from foreign pathogens. To achieve this, the skin harbours several immune cells that are involved in innate and adaptive immune response. The skin itself is part of the innate immune system ~~because~~ due to the fact that it is the first line of defense.

↳ the skin has a ^{complex} immune system that is represented by skin associated lymphoid tissue (SALT). This includes dendritic cells, mast cells, B lymphocytes, T lymphocytes and keratinocytes. The SALT system protects against foreign pathogens and it also plays a role in inflammatory diseases like autoimmune and hypersensitivity disorders.

The epidermis contains three major types of immune cells including keratinocytes, melanocytes & langerhans cells. The epidermis itself has four layers: the stratum basale, stratum spinosum, stratum granulosum and stratum corneum.

1st layer = This layer consists of non-specialised cells which are constantly involved in dividing into new cells (This is known as mitotic activity)

February 12th research

2nd layer = In this layer, the undifferentiated cells from the stratum spinosum the stratum basale become specialized to produce keratin

3rd layer = The third layer (stratum granulosum) is composed of keratinocytes which produce keratin forming proteins and lipids, these are crucial for forming the skin's protective barrier

4th layer → This layer also contains keratinocytes (stratum corneum) but they perform a different function. These keratinocytes are more mature and help to get rid of potentially toxic pathogens and maintain the levels of water and electrolytes (this is known as hydroelectrolytic homeostasis)

→ The stratum corneum contains fully matured keratinocytes and these cells perform a separate function. They help protect the individual by eliminating potentially toxic pathogens. These cells also maintain stable levels of water and electrolytes (this is known as hydroelectrolytic homeostasis)

As

In the stratum basale, melanocytes protect DNA from sun damage by producing a pigment called

Feb 12th research

Keratinocytes

These cells play an important role in maintaining the functions of the Epidermis and they also contribute to inflammatory processes by producing cytokines and expressing MHC class I & II glycoproteins. Keratinocytes can recognize the differences between regular microorganisms and foreign pathogens. They also recognize structures called Pathogen-Associated Molecular Patterns (PAMPs). These structures are important because they recognize several foreign pathogens. These PAMPs are usually composed of flagellins, nucleic acids, teichoic acids, lipopolysaccharides (LPS) and more. There are also molecules that attach to PAMPs, these molecules are known as Pattern Recognition Receptors (PRRs) and include Toll-Like receptors.

→ The binding of PAMPs and TLRs are extremely important in adaptive and innate immunity.

There are also (NOD) like receptors (NLRs) which recognize PAMPs. These receptors are involved in the recognition of substances like irritants and toxins. As shown, keratinocytes play an important role in the immune system because they can recognize foreign pathogens through the use of NLRs, TLRs and PAMPs. They also produce antimicrobial peptides (AMPs). These can perform several functions like killing pathogens and signaling immune cells. In conclusion, keratinocytes are not just cells which produce protective keratin. They are cells which play an important role in the immune system.

Feb 12th Research

The skin contains T lymphocytes which are mainly composed of memory T cells. T lymphocytes are found in the ~~base~~ stratum basale layer of the epidermis and in clusters around capillaries in the dermis. T cells participate in immune response and include several types like Th1, Th2, Th3 and Th17 & regulatory / suppressor T cells. These types have been found most particularly in individual with infectious and inflammatory diseases. Th1 lymphocytes are involved in infections & autoimmune or immunomediated diseases like psoriasis. Th2 lymphocytes participate in response to foreign pathogens in allergic diseases such as atopic dermatitis and psoriasis. Th17 cells are also involved in the development of psoriasis and atopic dermatitis. These cells are the body's first line of defense against infection by fungi and bacteria. Th2 + cells release various cytokines like IL-4, IL-5 and IL-13. Cytokines can be defined as proteins that help with the activity of these immune cells. There are also regulatory T cells which suppress immune responses to ensure that healthy tissue isn't affected. These cells are extremely important because they regulate the immune responses which cause inflammation. In conclusion, T cells are important immune cells which are involved in the regulation of immune response of the skin.

Feb 13th

Skin Barrier Dysfunction

↳ relates to the fact that skin with Atopic Dermatitis isn't as effective at keeping irritants and pathogens out of the skin. Skin suffering from Atopic Dermatitis is also weak at controlling moisture loss. EczemaHelp.ca refers to the skin as a "brick-and-mortar wall" that prevents pathogens from entering. Eczema prone skin is more damaged so pathogens can easily enter, cause inflammation & increased moisture loss.

~~epidermis~~
~~skin~~

↳ In normal skin, the barrier helps to prevent the entry of irritants through several components. This layer of the skin is constantly renewing itself because of the keratinocytes that are constantly evolving. Keratinocytes migrate from the deepest layers of the epidermis to the stratum corneum. Keratinocytes become corneocytes through a process called cornification. Corneocytes are keratinocytes which are dead and strong. During this constant cycle, keratinocytes produce lipids which help form extracellular lipid enriched layers. These stages of differentiation have expression proteins like FLK (filaggrin), transglutaminases (TGM), loricrin and much more. The AMPs that keratinocytes produce kill microorganisms and help maintain skin homeostasis.

Feb 18th research

Skin Barrier Dysfunction

1. ~~this~~ the layer that contains corneocytes also has a lipid barrier which prevents water loss and retain moisture. ~~the~~ ceramides, fatty acids and cholesterol are 3 types of lipids that help retain moisture.

Skin with Atopic Dermatitis has several factors that contribute to the overall abnormal composition of the epidermis. For instance, overexpression of Th1 and Th2 cytokines contribute to skin barrier dysfunction because it alters lipid and protein content. FLN (filaggrin) is an essential protein that helps maintain normal skin pH and lock in moisture. Atopic Dermatitis skin often has a lack of FLN which changes the shape of corneocytes. This causes more skin inflammation. The deficiency of FLN also causes an individual to be less tolerable to different irritants. The deficiency of FLN is usually decreased over by the expression of IL-4, IL-13, IL-25, IL-17A, IL-22 cytokines from T lymphocytes. Other essential proteins are also impacted by the overexpression of Th2 cytokines. Th2 cytokines also impacts the abundance of CPSN (a corneodesmosome) an intercellular protein which helps maintain a normal skin barrier. According to the national library of medicine, a study shows that the deficiency of CPSN resulted in more increased pathogenic entry in a organ like skin model. The cytokines which ~~the~~ contributed to this defect included several types like IL-4, IL-13, IL-22, IL-25, IL-31.

Skin Barrier Dysfunction Feb 18th research

Final Summary

A normal skin barrier contains several important proteins which help to maintain a normal barrier through regular function. Individuals with Atopic Dermatitis skin isn't as effective at keeping irritants and pathogens out of the skin from entering the skin. Skin suffering from Atopic Dermatitis is also weak at controlling moisture retention. EczemaHelp.ca refers to the skin as a "brick-and-mortar wall" that prevents pathogens from entering. Eczema prone skin is more damaged which allows irritants to enter easily and causing inflammation and moisture loss. In normal skin, in the epidermis, there is constant renewal of cells because the keratinocytes are constantly evolving. Keratinocytes migrate from the deepest layers of the epidermis to the stratum corneum. Here, keratinocytes become corneocytes through a process called cornification. Corneocytes are basically keratinocytes which are dead and provide a strong barrier. During this cycle keratinocytes produce expression proteins like filaggrins (FLNs), loricrin, transglutaminases (TGMs) and much more. Additionally, the AMPs that keratinocytes produce help maintain skin homeostasis and kill harmful microorganisms. The stratum corneum also contains 3 types of lipids which prevent water loss in the epidermis. These include cholesterol, free fatty acids and ceramides.

Skin with Atopic Dermatitis has several factors that contribute to the overall abnormal composition of the epidermis. In fact, the exact cause of Atopic Dermatitis is still not concluded because this involves several variables. For instance, the overexpression of Th2 and Th22

Feb 12th

Skin Barrier Dysfunction Final Summary

contribute to skin barrier dysfunction because it alters lipid and protein content. the overexpression of these cytokines impact multiple essential proteins. For instance, FLH is a protein which helps maintain ~~control~~ normal skin pH levels and lock in needed moisture. Atopic Dermatitis skin often lacks FLH which changes the shape of corneocytes. This results in increased inflammation. The lack of FLH can be caused by genetic factors, overexpression of cytokines and much more. The deficiency of FLH causes an individual to be less tolerant to different irritants.

FLH is usually decreased by the overexpression of IL-4, IL-13, IL-25, IL-17A, IL-22 cytokines from T lymphocytes. Th2 cytokines also impacts the abundance of Corneodesmosin (CDSN). This is a intercellular protein which maintains a regular skin barrier. According to 'Significance of Skin Barrier Dysfunction in Atopic Dermatitis

by Kim and Leung', a study shows that the lack of CDSN resulted in increased pathogenic entry in a organ like skin model. The cytokines which contributed to this defect included several types like IL-4, IL-13,

IL-22, IL-25 and IL-31. The AMPs produced by keratinocytes are also impacted by Th2 cytokines.

They suppress the production of AMPs which leads to a weakened barrier that is more prone to infections. For instance, deficiency in AMPs led to more *S. aureus*

infection. *Staphylococcus aureus* is a type of bacterium which is able to grow on ~~the~~ AD skin because of a weakened skin barrier.

Feb 13

Skin Barrier Dysfunction - Final Summary

Additionally, skin with Atopic Dermatitis has a defective lipid barrier in the corneated layer due to abnormalities in the expression of enzymes. These enzymes are usually involved in the synthesis of fatty acids and ceramides for the lipid matrix. However they are expressed differently in AD skin. This results in changes of lipid composition.

This results in an abnormal skin barrier and increased permeability of the stratum corneum. This means eczema prone skin is more susceptible to inflammation. Overall, an already damaged barrier allows allergens to activate cytokines and T lymphocytes causing inflammation. The overexpression of these cytokines also further damage the skin barrier.

Introduction to Immune Sensitivity. Feb 14th

As we know, the immune system plays a role in protecting the body from irritants by reacting to them when in contact. The dysfunction of the skin barrier allows irritants to enter more easily in Atopic Dermatitis (AD) skin. Individuals with Atopic Dermatitis have an overreactive immune system and this hypersensitivity results in eczema flares. Additionally, eczema is worsened by the scratch/itch cycle. This occurs when the nervous system sends out signals that to scratch an itchy eczema lesion. Scratching an eczema flare worsens the situation and makes it more prone to infections and pathogenic entry. Eczema help.ca suggests that Atopic Dermatitis is due to immune mediated inherited abnormalities in the skin barrier. Often, the genetics of an individual plays a huge part. In AD skin, there is an imbalance of Th1 and Th2 cells and their corresponding cytokines. There are also increased levels of the antibody immunoglobulin E (IgE), antibodies and eosinophils. Also, the innate immune system does not function normally. When the innate physical barrier is broken, the innate immune system is less effective and inefficient. The activation of adaptive immune responses like T cell cytokines further damages the skin.

Introduction to Immune Sensitivity Feb 14th

The immune system plays a role in protecting the body from irritants by reacting to them when in contact. Individuals with Atopic Dermatitis have an overreactive immune system and this hypersensitivity results in eczema flares. Additionally, eczema is worsened by the scratch/itch cycle. This occurs when the nervous system sends out signals to scratch an itchy eczema lesion. Scratching an eczema flare makes the skin more prone to infections and pathogenic entry. Atopic Dermatitis is heavily impacted by immune mediated inherited abnormalities in the skin barrier. Often, genetics of an individual play a huge part. Immune sensitivity is illustrated by increased levels of immunoglobulin E (IgE), antibodies & eosinophils.

Immune Sensitivity Feb 19th

T regulatory cells (Tregs)

Regulatory T cells play a crucial role in the suppression of immune responses. They shut down immune responses to ensure that healthy tissue isn't affected by other T cells like cytotoxic T cells. Individuals with Atopic Dermatitis (AD) skin are seen to have an abnormally functioning T cell population which shifts into an overreactive Th2 response. Many Tregs express CD25 and the transcription factor called Foxp3. These can be described as essential markers for Tregs in order to function. In fact, Tregs that express CD25 have been observed to carry increased suppressive properties.

In skin, Tregs play an important role in surveying the immune system at the location. There are several types of Tregs such as natural Tregs and adaptive Tregs. Adaptive Tregs include regulatory cells known as Tr1. These secrete IL-10 and suppress antigen-specific responses.

In some individuals In atopic Dermatitis, the Foxp3 gene often goes through a mutation and this results in a congenital disorder known as IPEX. This syndrome means that there is a deficiency in CD25⁺ Tregs which results in immune dysregulation. Many individuals with this mutation have inflammatory conditions like airway inflammation and eczema.

IPEX
patients
can often
develop
infect

Tregs

The Role of Regulatory T cells in Atopic Dermatitis by Agrawal, Wisniewski, Wootton (2015) suggests that adults with established Atopic Dermatitis actually had increased levels of Tregs which express CD25 and Foxp3. Now, this might seem odd but studies show that Tregs are not necessarily reduced in AD but they function abnormally. They function the same as in a normal skin. Individuals with AD are often often have colonies of *Staphylococcus* on their skin and more IgE antibodies. This impacts the ability of CD25⁺ Tregs to function normally. Tregs can also transform into several different types of T cells. This is known as "plasticity". Tregs can turn into Th2 cells as well as Th17 cells. These are the same cells involved in the overreaction of the immune system. To conclude, the abnormality of Tregs can impact the development of inflammatory conditions like Atopic Dermatitis. There is still further research to be done on Tregs because they inhibit confusing properties.

Feb 14th - Research

1. Immune Sensitivity

As we know, the immune system plays a role in protecting the body from irritants by reacting to them when in contact. Skin Barrier Dysfunction allows irritants to enter more easily in Atopic Dermatitis (AD) skin. Individuals with Atopic Dermatitis have an overreactive immune system and this hyperactivity results in eczema flares. Atopic Dermatitis is heavily impacted by a overactive immune system and hypersensitive nerves in the skin.

Additionally, eczema is worsened by a cycle known as the itch/scratch cycle. This occurs when the nervous system sends out signals to scratch an itchy flare that has happened due to an overreactive immune system. Scratching an eczema flare worsens the skin, makes it more prone to infection and provides an easier entry for pathogens.

↳ There is evidence that suggest that Atopic Dermatitis is due to immunity mediated an inherited abnormalities in the skin barrier. Often, the genetics of an individual plays a huge part in the immune system.

↳ Usually, a normal skin barrier ^{has} ~~there~~ a balance of the number of Th1 cells & Th-2 cells. These are T helper lymphocytes. In Atopic Dermatitis, there is often an imbalance. Usually, there are way more Th2 cells and their corresponding cytokines.

Feb 14th research

Zara =
eczema

There are also some higher levels of the antibody immunoglobulin E (IgE), antibodies and eosinophils.

The innate immune system can be thought as a first responder which non-specifically targets pathogens and irritants. The adaptive immune system is slower but it can specifically and accurately eliminate pathogens.

In AD skin

↳ Several components of the innate immune system do not function normally. When the physical skin barriers are disrupted the innate immune system is inefficient and less effective. This also irritant like *S. aureus* to colonise the skin. The activation of adaptive immunity response like T lymphocyte cytokines further damage the skin. The overreaction of Th2 cytokines and other types impact the severity of Atopic Dermatitis.

Immunity Sensitivity Feb 15th

Tregs Final Summary

Regulatory T cells (Tregs) play a crucial role in the suppression of immune responses. They gradually slow down immune processes (like cytotoxic T cells) to ensure that healthy tissue isn't damaged. Many Tregs express CD25 and the transcription factor called Foxp3. These can be described as ^{essential} markers ~~to~~ needed for Tregs to function normally. Tregs that express CD25 have been observed to carry increased suppressive properties. There are several type of Tregs such as natural and adaptive Tregs. Adaptive Tregs include type-1 regulatory cells known as Tr1. These ~~are~~ ^{release} IL-10 to suppress specific specialised immune responses. Individuals with Atopic Dermatitis (AD) skin are seen to have an abnormally functioning T cell population which transforms into an overreactive Th2 response. In some individuals, the Foxp3 gene often goes through a mutation and this results in a congenital disorder known as IPEX. This syndrome causes a deficiency in CD25⁺ Tregs. This results in immune dysregulation. Many individuals with this mutation develop inflammatory conditions like airway inflammation and Eczema. The Role of Regulatory T Cells in Atopic Dermatitis by Ag Rawal, Wishresh, Woodcock (2015) suggests that adults ~~with~~ with established Atopic Dermatitis actually had increased levels of Tregs which express CD25 and Foxp3. Now, this might seem odd but studies show that Tregs are not necessarily reduced in AD but they function abnormally. Individuals with AD have colonies of *S. aureus* and increased IgE antibodies. This impacts the ability

Science Fair Research

Feb 15

Eosinophils in Atopic Dermatitis

Eosinophils are a type of white blood cell (leukocyte) which helps defend the body against foreign pathogens and irritants. While they help with protection, Eosinophils have been observed ^{to contribute} ~~in the contribution~~ to the development of inflammatory conditions like Atopic Dermatitis. Individuals with Atopic Dermatitis have been observed to contain a higher level of eosinophils circulating in their blood. This higher concentration of eosinophils is referred to as eosinophilia. Eosinophils are common in inflammatory responses. For instance, in asthma eosinophils are activated by cytokines from Th2 cells. Similarly, eosinophils are ~~high~~ release a high number of cytokines contributing to inflammation. To conclude eosinophils impact ~~the overreactiveness of~~ the hypersensitivity of ~~the~~ the immune system resulting in more inflammation ~~and~~

EOSINOPHILS

17th
Feb ~~17th~~ Research

IgE

Immunoglobulin E (IgE) plays an important role in the development of Atopic Dermatitis. It is an antibody that is used to fight off foreign pathogens and infections. IgE helps trap potentially harmful substances and ~~alert~~^{AD} the immune system. Individuals with Atopic Dermatitis skin often have high serum levels of IgE in their bodies. The term serum level refers to the amount of IgE antibodies produced by your immune system. IgE contributes to the production of several cytokines such as IL-1, IL-3, IL-4, IL-5, ^{and} IL-6. These cytokines trigger an immune response which results in increased inflammation in conditions like Atopic Dermatitis. Increased serum levels of IgE shows how patients with AD skin have a hypersensitive immune system. There is further research to be done on the role of IgE antibodies in Atopic Dermatitis.

Feb 17th Research

Atopic Dermatitis but Health

Atopic Dermatitis is a complex mix of several factors like external triggers, immune sensitivity and skin barrier dysfunction. However, the ^{gut} environment of the gut also influences the health and sensitivity of one's skin. Individuals with Atopic Dermatitis have been observed to lack a diverse microbial population in their gut compared to healthy people. The term microbiome refers to the the variety different types of microorganisms living on certain body parts. This part of our bodies have a huge impact on health and processes such as immunity and metabolism. Individuals with inflammatory conditions like eczema have way less ^{bacteria} ~~gut~~ located in their guts.

The growth of microbes start as soon a baby is born. These microorganisms develop within the gut and establish themselves by the time a child becomes 2-3 years old. Some factors that influence the gut population from birth includes the type of delivery

Vaginal Delivery = The baby is exposed to vaginal microbes which have positive health benefits. ex = *Bifidobacterium* & *Lactobacillus*. This allows the steady regulation of immune responses

Feb 17th

Caesarean Section = *Streptococcus* species, *Staphylococcus* (C-section) & *C. difficile* are found on these babies

Formula feeding & breast feeding $\rightarrow$ *Bifidobacterium*

Bottle feed = *E. coli* & *C. difficile*

The 1st 3 years of an individual and the colonization of bacteria during this time is crucial & determines ~~their~~ health & disease. Intestinal bacteria highly influences immune function.

Vaginal delivery, breast feeding, interaction with siblings & pets $\uparrow$ microbial diversity
 $\uparrow$ Tregs, short-chain fatty acids & immune tolerance

Low biodiversity = affects immune response & increases inflammatory responses

People with AD had less *bifidobacterium*. Patients with severe AD have less *bifidobacterium* than individuals with mild Atopic Dermatitis

AD patients had lots of *Staphylococcus* compared to healthy individuals.

Gut Microbe & Skin

Intestinal and the microorganisms in the gut impacts the immune function of one's body. Many infants with higher risk of developing atopic conditions like Atopic Dermatitis

- $\hookrightarrow$ low levels of *Bifidobacterium*, *Alkermatia* & high quantities of *Candida* & *Rhodospirillum*
- $\hookrightarrow$ reduced *Bifidobacterium* at 3 weeks stool
- $\hookrightarrow$ diverse gut microbiota = less risk for AD
- $\hookrightarrow$ gut bacteria established at young age influences performance of the immune system / development
- $\hookrightarrow$ gut microorganisms influence the development and specializations of T cells into $Th1$, $Th2$, $Th17$ $Foxp3$ & Treg cells
- $\hookrightarrow$ Treg cells suppress inflammation & prevent the immune system from becoming overreactive. They control the specializations of T cells spilling into Th cells
- $\hookrightarrow$ downregulate eosinophils, mast cells, basophils
- $\hookrightarrow$ suppresses IgE production

Bifidobacterium, *Lactobacillus*, *Clostridium*, *Bacteroides* & *Streptococcus* have metabolic products like butyric acid & propionic acid $\rightarrow$ these activate Treg cells. They also promote the generation of Treg cells formed in the peripheral body parts

Gut microbe & skin Feb 17th

- ↳ 2 Korean metagenomic studies → imbalance of gut microbes & decrease of short chain fatty acids influenced the gut
- ↳ SCFA helps keep epithelial barrier in the gut intact & has anti-inflammatory effect
- ↳ AD ppl = leaky gut
- ↳ penetration of toxins, poorly digested food & microbes into systemic circulation
- ↳ As it reaches skin, Th2 response happens = tissue damage

- ↳ mode of delivery has impacts on AD
- ↳ older siblings = beneficial influence on gut microbiota development = less AD
- ↳ mode of delivery / presence / absence of siblings

SCFA

- ↳ play role in keeping immune response regular

Feb 18th

Gut microbe & Skin final summary

Atopic Individuals

Atopic Dermatitis is a complex mix of several factors like external triggers, immune sensitivity and skin barrier dysfunction. However, the environment of the gut also influences the health and sensitivity of one's skin. Individuals with Atopic Dermatitis have been observed to lack a diverse microbial population in their gut compared to healthy people. The term microbiome refers to the variety of different types of microorganisms living on certain ~~part~~ body parts. The microorganisms of the body have a huge impact on health and processes such as immunity and metabolism. Individuals with inflammatory conditions like Eczema have way less beneficial bacteria located in their guts.

The growth of microbes often ~~start~~ start as soon as a child is born. These microorganisms start developing and eventually become established by the time the child is 2-3 years old. One factor that influences the gut population from birth includes the type of delivery. During vaginal delivery, the child is exposed to microbes from the mother. For instance, vaginal delivery allows ~~for~~ exposure to *bifidobacterium* and *bacteroides*. ~~This allows~~ This allows the steady regulation of immune response. These microorganisms

Atopic Dermatitis

In comparison, Caesarean section (C-section) allows the baby to become exposed to streptococcus species, staphylococcus and C. difficile. The beneficial bacteria are way less on babies delivered by C-sections. The way a baby is fed during early childhood also impacts the population of microorganisms. Formula feeding and breast-feeding increases populations of Bifidobacterium. Bottle fed babies have higher concentrations of E. coli & C. difficile. The early childhood of a baby deeply impacts the development of intestinal bacteria. Intestinal bacteria influences immune function, health and disease. Experiences such as vaginal delivery, breast feeding, interaction with siblings and pets ~~also~~ increase Tregs, Short chain Fatty Acids (SCFA) and immune tolerance.

(AD) Skin

Individuals with Atopic Dermatitis have low biodiversity in microorganisms. As a result, ~~this~~ this affects immune responses and inflammatory response. People with AD skin have less bifidobacterium and other beneficial bacteria. It is important to note that patients with severe AD have less bifidobacterium than individuals with mild AD. Additionally, AD patients have lots of staphylococcus populations compared to healthy individuals. Many infants with higher risk of developing Atopic Dermatitis also have high levels of bacteria such as Candida & Rhodotomula.

Anal summary - Gut microbes

How do gut bacteria help?

A diverse population of beneficial gut microorganisms influence the development of important immune response cells such as Treg cells and the Foxp3 factor. They also play role in the development of specialized T cells such as Th1, Th2, Th17. As we know, Treg cells play an important role in regulating immune responses and preventing an overreactive response. They regulate eosinophils, mast cells, basophils, IgE production and T cell specializations. Gut bacteria like Bifidobacterium, Lactobacillus and clostridium ~~release~~ have metabolic products ~~like~~ such as butyric acid and propionic acid. These products help activate Treg cells and promote generation.

2 Korean metagenomic studies show how an imbalance of gut bacteria and ~~the reduction~~ reduced SCFA influences the gut. SCFA helps keep the barrier in gut and has anti-inflammatory effects.

AD people often have "leaky gut" which leads to ~~poorly~~ ^{absorption of} toxins, poorly digested food and microbes into systemic circulation. As it reaches the skin, Th2 response happens = damage.

External Factors - Feb 27

Many individuals with Atopic Dermatitis experience flares due to extreme hot and cold temperatures. For instance, the harsh winters of Northern North America can trigger and increase dryness / itches in the skin. In general, low humidity and temperatures decrease skin barrier function and increase stress. Extremely high temperatures also can also provoke flares. Cold weather contributes to the activation of cytokines which further increases the scratch-itch cycle and skin barrier dysfunction. Exposure to harsh and extreme temperatures increase stress and the scratch-itch cycle. Current issues such as climate change creates significant and fast changes in temperature and climate. These ~~sudden~~ Heat events and wildfires increase AD and skin sensitivity due to sudden changes. Climate change ~~also~~ ~~these~~

Atopic Dermatitis

Low humidity dries out skin and makes it more susceptible to ~~renew~~ irritation. However, hot and ~~dry~~ ~~weather~~ conditions increase sweating and flares 0-4 days after exposure

External Triggers → Allergens & pollutants

In ~~the~~ urban areas, the risk of developing inflammatory conditions like Atopic Dermatitis is increased due to emissions and air pollutants. Some air pollutants which trigger Atopic Dermatitis include nitrogen dioxide, benzene and sulfur dioxide. Particulate matter from the combustion of fossil fuels also heavily impact the skin's reactivity. Such pollutants penetrate and irritate Atopic Dermatitis (AD) skin due to skin barrier dysfunction.

Components of indoor environment like renovation, painting and furniture could also cause new flare ups, especially in babies. AD patients. The combination of outdoor pollutants, indoor components and allergens like dust mites increase severity of Eczema.

and air pollution
Additionally, climate change is currently impacting and altering the ^{Pattern and Composition} composition of common allergens like pollen. Individuals with inflammatory conditions are often sensitive to pollen as they activate IgE antibodies. Climate change impacts pollen season and concentration. For instance, warmer urban areas experience higher and earlier levels of pollen release. Air pollution can alter pollen proteins and increase allergenicity. Increased CO₂ levels and temperature both contribute to this. Finally, climate change ~~may~~ ^{can} promote the growth of non-native plants with unfamiliar pollen

External Triggers - Allergens & Airpollutants Feb 27th

An already compromised skin barrier can often be extra sensitive to pollutants. For example the dysfunction of the FLN proteins increases susceptibility to pollution.

Water Quality

Eczema is highly impacted by water with high mineral content and this disrupts the skin barrier. Domestic hard water is measured by calcium carbonate concentrations which is evident in adults and children with Atopic Dermatitis. Hard water refers to when ~~water~~ ^{calcium} ~~is~~ ^{minerals} are dissolved in water from sedimentary rock filtration. This water contains calcium in the form of calcite and dolomite. Hard water creates dryness, skin irritation and increased barrier dysfunction. For instance, bathing in hard water with AD skin can increase sodium lauryl sulfate deposits which cause irritation and ~~skin~~ transepidermal water loss.

Feb 27th

The irritation and severe itching associated with Atopic Dermatitis can have a profound impact on the ability to sleep peacefully. AD often worsens at night, disrupting sleep patterns which leads to awakenings and worsening sleep quality. Sleep quality is extremely important and Atopic Dermatitis can reduce sleep quality can lead to cognitive decline in areas of memory and attention. ^{disrupts} ^{rem sleep} ^{rapid eye movement}

Food

In Atopic Dermatitis, the diet an individual follows can trigger flare-ups, IgE mediated hypersensitivity or late eczematous reactions. These reactions are allergic and could occur hours or days later after digestion of a certain food. Food allergies have mostly impacted the Atopic Dermatitis of infants and children with moderate to severe AD. Food allergies to ingredients such as nuts, soy and wheat can be described as health effects stemming from overreactive and specific immune responses. These responses include IgE antibodies and other non-IgE mediated responses. Immediate reactions are IgE mediated and ^{can} occur within minutes to hours. These types of reactions increase Atopic Dermatitis due to itchiness, and worsened scratching. Many patients have observed that the elimination of ~~food~~ diet related triggers helped improve Atopic conditions. In comparison, late eczematous reactions could occur after hours or day of digestion.

Feb 28th

♥ *Angie* ♥ ♥ ♥ ♥ *Chuy*

Categories

Name and Age	Type of Delivery	Pets?	Siblings?	Food Allergies

Water used	Genetic Family History	Other medical conditions

categories to fill for Zara, Aaron and Vanessa

Personal Experience in India

Week 1

↳ The moment I landed in India

General Data Info

Name	Type of Delivery	Siblings	Pets	Food Allergies
Siya Shetty	—	N/A	N/A	N/A

Other medical conditions	Where my eczema usually flares
Asthma	—

Genetic Family History	Water used at home	Potential ~~triggers~~ triggers
		