

Cosmic Death Fungus

A Primer On An Ancient Enemy

Document Version 2.02

Table Of Contents

I. Overview

1.1 Introduction	9
1.2 Defining The Enemy	11
1.3 Common Yeast	
1.3.1 Candida & Saccharomyces	12
1.3.2 Alternative Metabolism	13
1.3.3 You Are The Food	14
1.4 Aspergillus	15
1.5 Cryptococcus	16
1.6 Conclusions	17

II. The Fall Of Man

2.1 Taforalt: Origins	19
-----------------------------	----

2.2 Taforalt: Migration	22
2.3 Familial Candidiasis	
2.3.1 How Errors Occurred	25
2.3.2 Immunodeficiencies	28
2.3.3 The Pharoah's Curse	31
2.4 From Womb To Childhood	
2.4.1 APECED	36
2.4.2 Miscarriages & Defects	37
2.4.3 Thymus: Immune Failure	38

III. Origins Of Disease

3.1 Inflammation	40
3.1.1 Candida: A Common Trigger	41
3.1.2 Mycotoxins	42

3.1.3 CDF Associated Interleukins	43
3.2 Connections With Common Diseases	
3.2.1 Tooth Decay	47
3.2.2 Cancer	49
3.2.3 Sclerosis	50
3.2.4 Heart Disease	52
3.2.5 Diabetes & Weight Gain	53
3.2.6 Joint Pain, Back Pain, Arthritis	54
3.2.7 Diseases Of The Brain	55
3.2.8 Accelerated Aging	58
3.3 Conclusions	59

IV. The NAC Protocol

4.1 Introduction and Backstory	61
4.2 Methodology	64

4.3 The Protocol	69
4.4 Brushing and Upper Respiratory	74
4.5 Common Die Off Symptoms	75
4.6 Common Experiences	78
4.7 Diet Considerations	79
4.8 Conclusion	80

V. The Maintenance Protocol

5.1 Introduction	82
5.2 Methodology	84
5.3 Die Off Symptoms	88
5.4 Common Experiences	89
5.5 Conclusion	91

VI. History, Mythical & Religious References

6.1 Origin Of The Tribes	95
6.2 Egypt	
6.2.1 Egypt: The Ennead	100
6.2.2 Egypt: The Pharoahs	113
6.3 Sumerian Legends	134
6.4 Abrahamic Legends	140
6.5 Scandinavian Legends	147
6.6 Irish Legends	151
6.7 Greek Legends	156
6.8 Interplanetary Ordeal	160

VII. Science Behind The Protocol

The NAC Protocol

7.1 NAC	175
7.1.1 Primary Benefit & Methodology	176

7.1.2 Antibiofilm Activity	182
7.1.3 Protocol Synergy	186
7.1.4 Safety Studies	188
7.2 Oregano Oil	191
7.2.1 Primary Benefit & Methodology	192
7.2.2 Antibiofilm Activity	195
7.2.3 Antifungal Activity	198
7.2.4 Protocol Synergy	201
7.2.5 Immune Modulation	203
7.2.6 Safety Studies	205
7.3 Black Seed Oil	209
7.3.1 Primary Benefit & Methodology	210
7.3.2 Antibiofilm Activity	212

1.1 Introduction



The rabbit hole you are about to jump down is going to merge disciplines rarely seen together; esotericism, history, archaeology and science.

This information is considered undisclosed, hidden, forbidden, avoided, sometimes challenged and frequently ridiculed.

Cosmic Death Fungus (CDF) isn't one specific fungi. It is several genera of fungi that can shape shift at will, play with your immune system as food and cause a lifetime of illness and despair.

Cosmic signifies that the fungi in question has a cosmic origin, otherwise known as Panspermia theory. Death signifies the Entropy that CDF causes by continual damage to our DNA.

CDF can cause brain disorders, heart problems, tooth decay, cancer, diabetes, sclerosis, back and joint pain, anxiety, depression and accelerated aging.

It is also responsible for multiple defects in our genome which predispose us to serious diseases, then we pass these defects on to our children.

It doesn't stop at causing suffering and disease. It also influences our thoughts, behaviors and eating habits. It can prompt deviant or impulsive behavior, thrives on conflict and stress, and creates hormone imbalances for its own benefit. It wreaks havoc throughout your body and works intelligently to get what it wants.

You are its food. You are not even fully human. In fact, you won't experience your true potential until you remove it from your body and heal the damage it has caused.

1.2 Defining The Enemy

Typically fungi is classified as a mold or a yeast.

Dimorphic fungi can be both, changing shape and purpose based on many different factors.



This type of fungi is not symbiotic and provides no benefit to the host.

Thermally dimorphic fungi can maintain mycelial form in the environment, but when entering the human body they become pathogenic. (source)

Even perfectly healthy individuals can trigger disease by simple things such as taking antibiotics. (source)

1.3.1 Common Yeast : Candida and Saccharomyces

This form of fungi can exist in single cell and hyphal form. Initial infection starts in the gastrointestinal tract, oral cavity and upper respiratory tract. Both create biofilms.



Candida gets most of the attention and is the most widely studied as a fungal pathogen, but common bread and brewers yeast can also be pathogenic and cause disease. ([source](#))

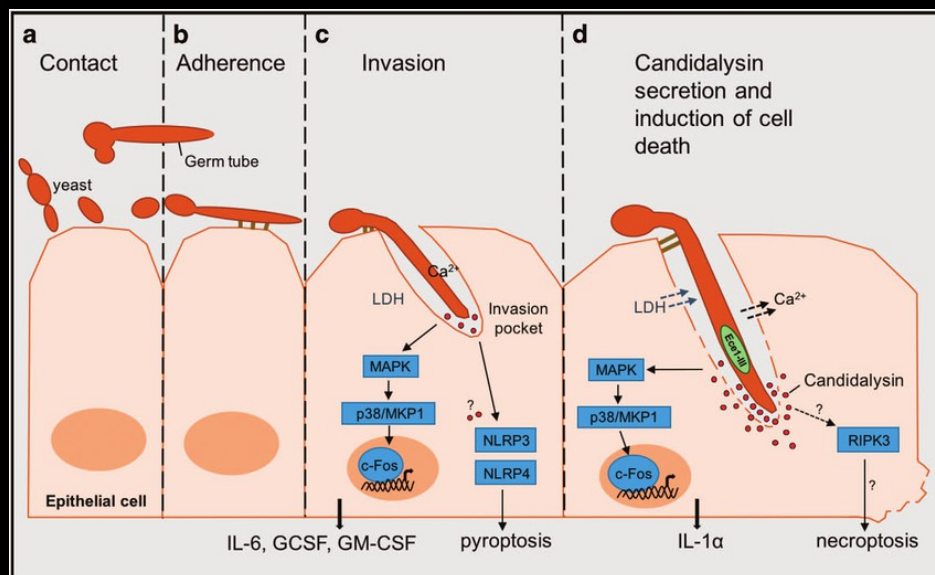
Surprisingly, many supplements claim this yeast is beneficial for ingestion, even though it can filament and invade organ and tissue causing serious problems ([source](#)). It's also commonly used to fatten cattle ([source](#)) and can directly trigger diabetes. ([source](#))

1.3.2 Common Yeast : Alternative Metabolism

Candida utilizes glucose sensing receptors to detect when sugar is consumed. ([source](#)) Attempting to eliminate sugar will not work, however.

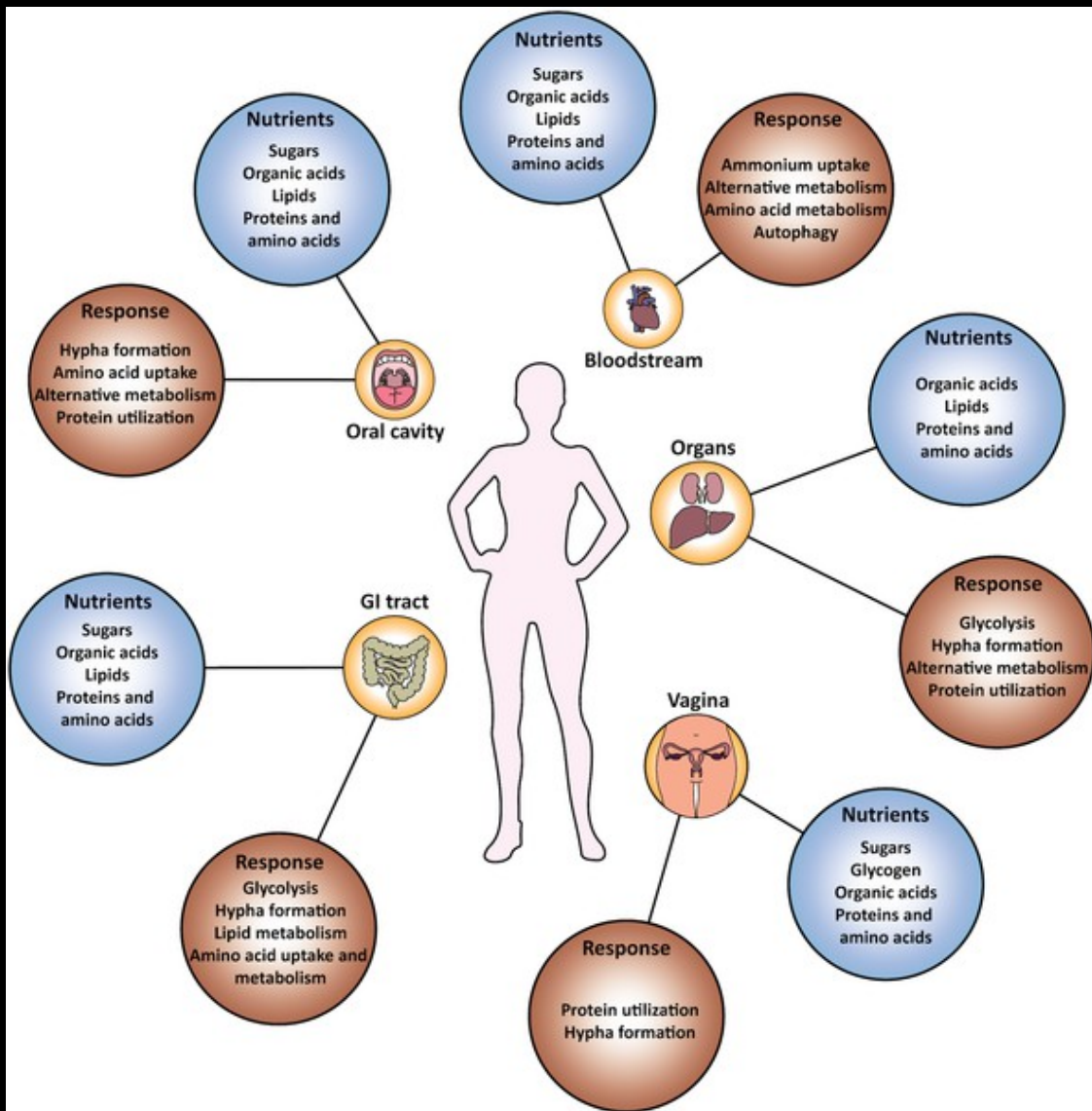
It can utilize alternative metabolism at any time. one regular way it feeds off of us is using the peptide toxin candidalysin to penetrate membranes and tissue then feeds off of what is available.

([source](#))



1.3.3 Common Yeast : You Are The Food

Depending on where in the body it resides, it can choose intelligently to utilize fatty acids, protein, glucose or LDH as food sources. ([source](#))



1.4 Aspergillus

This pathogenic fungi is particularly dangerous because it can be inhaled and quickly infect the lungs, brain and modify immune and gene expression. (source)



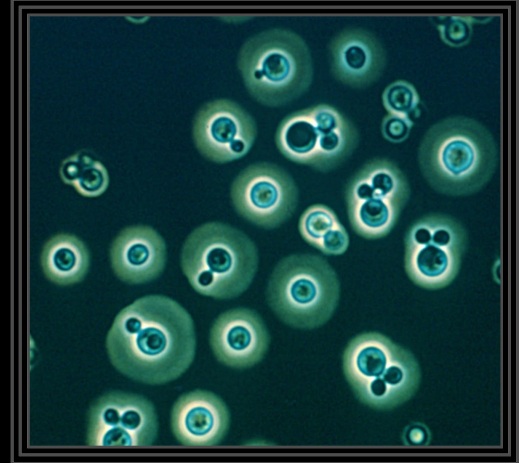
Exposure typically happens in the home in a closed environment. This particular fungi can become systemic and be the cause of brain lesions, nervous system damage (source) and even autism. (source)

Once inhaled, this human pathogenic mold uses aflatoxin and gliotoxin to suppress immune response and increase virulence. (source)

In immune competent people it can remain in the lungs for years and heavily contributes to asthma. (source)

1.5 Cryptococcus

This dimorphic pathogen is particularly dangerous to humans since it has an additional layer of defense using a chitin formed membrane similar to crustaceans. ([source](#))



This dangerous fungi with it's own mobile biofilm attacks various body systems, including the brain ([source](#)), lungs ([source](#)), prostate and central nervous system. ([source](#))

This insidious fungi primarily targets the brain and central nervous system via inhalation. It has several advanced techniques to enter the brain, including traversing the blood brain barrier inside of our own immune cells as a Trojan horse technique. ([source](#))

1.6 Conclusion



This was a quick overview covering the 3 specific genera we focus on: *Aspergillus*, *Cryptococcus* and *Candida*.

We've shown how it goes well beyond a gut issue, can feed on more than just sugar, and can infect various body systems. The average person is infected with at least 3 pathogenic fungi, and the above mentioned are the most common.

The failure of modern medicine is based on symptom management and ignoring fungi as an origin of disease. Even though there are decades of research linking fungi to all types of disease, the focus still remains on viruses and bacteria.

Most studies focus on fungi as being a problem only when you are immune compromised. This is an abysmal failure in understanding of how fungal metabolites contribute to disease and suffering.

Section II : The Fall Of Man



2.1 Taforalt: Origins

Taforalt, Morocco

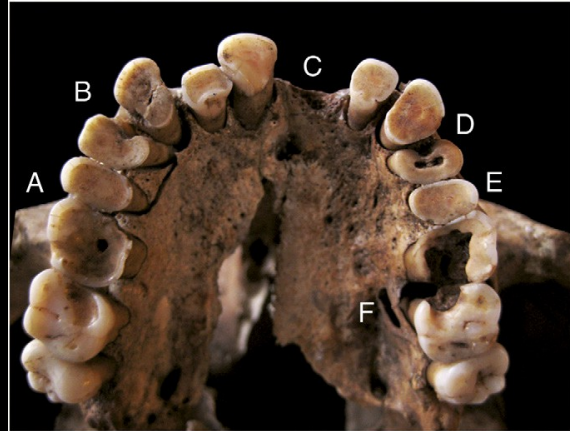
These ancient cave systems were discovered in 1961, containing burial sites for early Homo Sapiens spanning thousands of years.



As archeologists dug through layers representing different time periods, the period between 35,000 to 15,000 BC harbored a unique trait that other periods did not. This period showed the earliest evidence of dental caries in the population. ([source](#))

Claims were made that new agricultural practices lead to this evidence of dental caries, but evidence shows that these new practices came approximately 10,000 years later. ([source](#))

Thee real cause of the dental caries was based on thousands of years of raising pigeons.



These birds are known to harbor and spread several species of pathogenic fungi. ([source](#))

We also know that tooth decay and biofilm formation is the interplay between bacteria and fungi. ([source](#)) ([source](#))

We also know due to modern science that when constantly subjected to fungal pathogens, the body suffers from constant DNA damage. ([source](#))

This mostly results from their metabolites and toxins used to suppress immune function. ([source](#))

After many generations of exposure to multiple fungal pathogens from pigeon excreta and constant inhalation of spores, this genetic damage lead to mutations and the eventual formation of hereditary defects known today as Familial Candidiasis. ([source](#))

We believe that other familial defects will be discovered for Cryptococcus as well.

These defects in our genome are specific to our response to fighting pathogenic fungi, and we believe these defects lead to the first evidence of dental caries as our immune response began to malfunction.

This cradle of civilization was populated by the Amazigh people, a fair skinned and red haired population that later began their migration to Egypt, Europe and beyond.

2.2 Taforalt: Migration

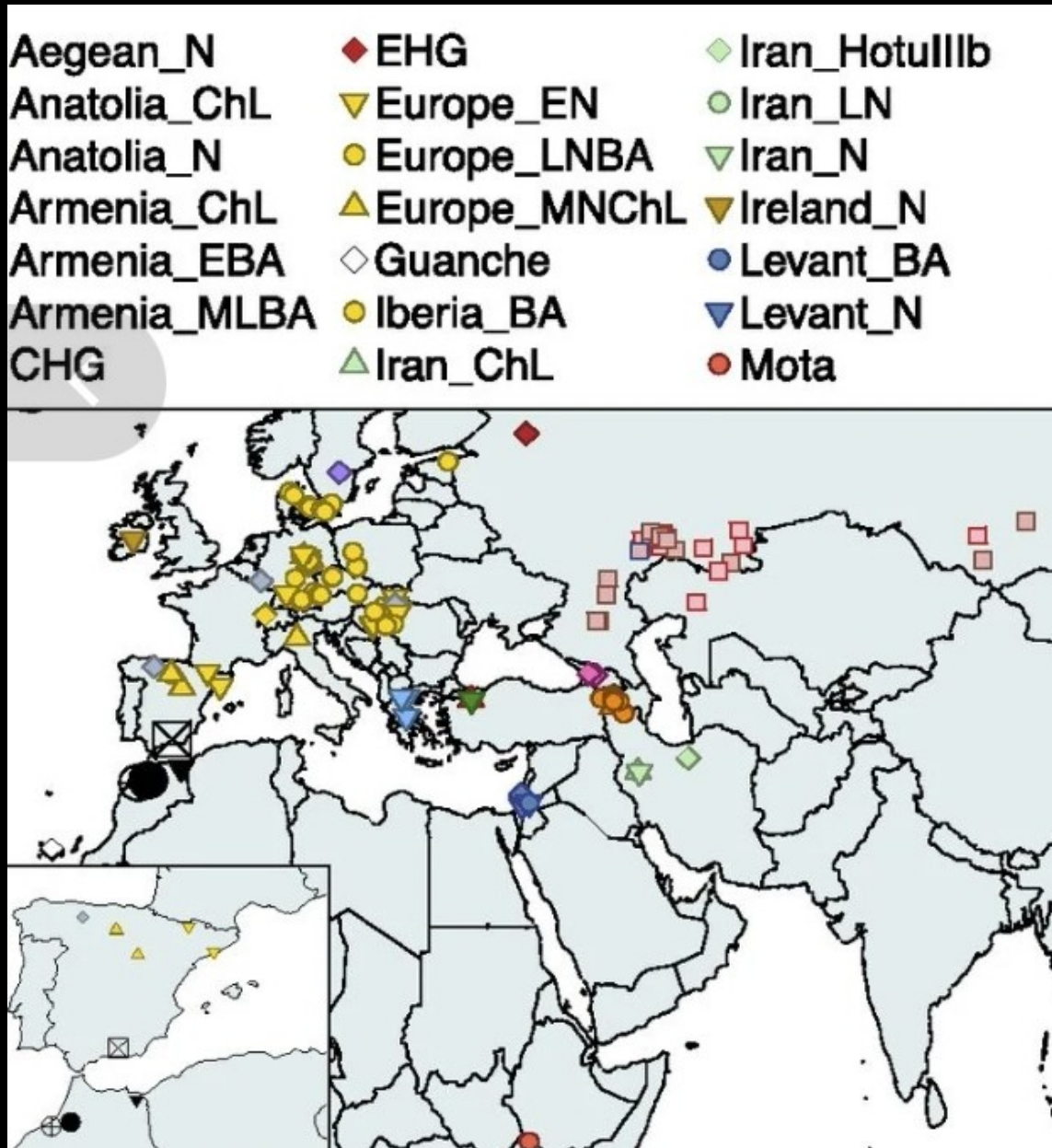
The Amazigh people who occupied Taforalt in Northern Africa had distinct features for the area, including light skin and red hair.

The MC1R Genome has some roots in Taforalt and is featured heavily in the genetic breaks we will discuss later.



Details of the Taforalt migration shake the current version of history and civilization at its foundation. Yet the genetic records of the migrations have been verified. Also known as the Berbers, they populated what is now known as Europe, as well as Iran, Egypt and Russia. ([source](#))

Migratory Path From Taforalt (bottom left)



The Berbers became the root of what we now call European civilization. They were Pharoahs of Egypt. They populated England, The Netherlands, Ireland and surrounding areas.



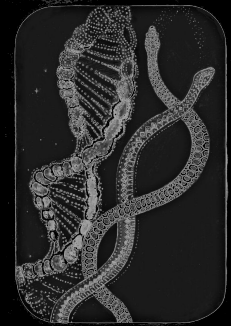
It's therefore no great surprise that red haired people or people with genetic lines sharing these characteristics also have the most genetic breaks. Their ancestors in Taforalt spread these gene errors far and wide.

We have further archeological evidence to share on this ancient Pharoah line in later chapters under The Pharoah's Curse.

We've been gathering personal reports for over a year now online, and we've seen plentiful evidence that people from migration areas are more effected by fungal infections than others.

2.3.1 How Genetic Errors Occurred

Gene mutations are the reason for these errors found in Familial candidiasis, and even a function of evolution itself.



One good example would be fish subjected to PCBs in their water supply. the eventual gene mutations that make them more resistant to PCBs gets passed on via natural selection and becomes commonplace throughout the population. ([source](#))

Since Familial Candidiasis doesn't have an immediate impact on survival, this devolution was carried on and survived to modern day.

Just as the mutations of fish created resistance to PCBs, the mutations in humans caused by fungus created mutations beneficial to fungal infection.

The fungi we call CDF have many harmful metabolites that damage DNA and also the body's repair functions.

Starting with Candida species, a toxic metabolite of concern is acetaldehyde. It damages the guanine enzymes responsible for repairing DNA ([source](#)) and also directly damages the DNA using reactive oxygen species. ([source](#))

Aspergillus uses different mycotoxins based on the species, the most common being aflatoxin and gliotoxin, used to suppress immune response to increase virulence. These toxins also directly damage DNA. ([source](#)) ([source](#))

Cryptococcus is far more complex, and could be considered the evolution of CDF, having a mobile biofilm and over 30 different metabolites. ([source](#))

Cryptococcus interacts with its food using more advanced techniques, tailoring release of enzymes depending on environment. Urease is one of the known metabolites that can cause molecular damage, but it can use protease reactions and other techniques. ([source](#))

The DNA and nuclear damage these fungi cause are only part of the problem. When combined with genetic breaks, dysfunctional immune response due to genetic errors can cause a never ending autoimmune loop, where the fungus cannot be eradicated and it continues to damage host cells and DNA.

Since the immune system cannot do the job on its own, additional antifungals and strategies are required.

At this point you've seen how it can damage our bodies and send us into a downward spiral. It doesn't require any preset conditions to damage your body.

2.3.2 Specific Immunodeficiencies

Searching the OMIM database ([source](#)) will show many entries related to Familial Candidiasis, the collection of genetic breaks we discuss here. This section will cover only a handful to demonstrate how relevant they are to chronic infections.

CANDF2 (Immunodeficiency 103)

A defect in CARD9 gene, 342 variants. Primary immunodeficiency characterized by increased susceptibility to fungal infections. Predisposition to Systemic and Invasive forms.

CANDF3

Chronic fungal nail infections.

CANDF4

Vaginal and Nail Candidiasis. Immune defect. Immune response 15% or less versus normal.

CANDF5 (Immunodeficiency 51)

Immune dysfunction in interleukin 17A,17F, 17A/F, 17E, responsible for responding to fungal infection. Frequent bacterial infections in the lungs. Connected to pancreatic cancer.

CANDF6

Gain of Function mutation in STAT1. Recurrent bacterial, viral, fungal, and mycoplasmal infections, disseminated dimorphic fungal infections, enteropathy with villous atrophy, and autoimmune disorders, such as hypothyroidism or diabetes mellitus. A subset of patients show apparently nonimmunologic features, including osteopenia, delayed puberty, and intracranial aneurysms. Laboratory studies show increased activation of gamma-interferon (IFNG; 147570)-mediated inflammation

CANDF7

A primary immunodeficiency disorder with altered immune responses and impaired clearance of fungal infections, selective against Candida. It is

characterized by persistent and/or recurrent infections of the skin, nails and mucous membranes caused by organisms of the genus Candida, mainly Candida albicans.

CANDF8

Affiliated tissues include skin, and related phenotypes are macroglossia and seborrheic dermatitis

CANDF9

Important gene associated with Candidiasis, Familial, 9 is IL17RC (Interleukin 17 Receptor C). Affiliated tissues include skin, and related phenotypes are recurrent aphthous stomatitis and chronic mucocutaneous candidiasis

2.3.3 The Pharaoh's Curse



(Pharaoh Ramses II, son of Seti I)

One of the landing points for the Amazigh people was Northern Egypt. Our research lead to a well hidden document from France that the Egyptian Antiquities would rather you not see.

Pharaoh Ramses II was quickly moved to France after being discovered, as he was decaying from a fungus. This fungus remained active for thousands of years in his tomb.

He lived as most Amazigh lived. Chased and tortured by the fungi we refer to as CDF. In the documents, later translated, they found all of the classic symptoms of FC. He had posture issues, his legs were bowed, he was balding, and the fungus literally consumed his nose. He wore a prosthetic nose to cover up this fact, which is also described in the documents.

There is a big connection to history and mythology here that we will expand upon more in coming sections. But for now, let's clarify that Ramses II followed Seti (Seth) and moved his people to monotheism. The decision to worship Seth over Osiris was indicative of the battle his people faced. Osiris promoted bread and beer, and Seth fought against it. They knew even then how dangerous this fungus was.

The following are quoted excerpts from the documents, translated to English.

"for the skull:

loss of material in the region of the cribriform plate of the ethmoid bone (evisceration gap); calcification of the falx cerebri, with atheroma of the carotid sinuses and temporal arteries; wear of the dental crowns in the upper and lower jaws, with geodes at the level of the root apex, due to abscesses; a kind of nasal prosthesis composed of exogenous elements, of circular or oval outline."

"The radiochromo densitographic study revealed in particular that the cervical spine and the skull present an aspect reflecting a post-mortem fracture of the spine, certainly straightened during the mummification. As for the jaws, all methods then combined or confused, their examination makes it possible to affirm that it is about an individual carrier of numerous and important dental lesions, due to cavities often

complicated by septic bone lesions, that in a very old man, reached of a very alveolysis. marked and an osteitis of the mandible linked to an abscess formation towards the chin."

"And this is how we were finally able to reach an approximation: height, by bone measurements: 1.72 m, age, by the state of the bones and teeth: 80 years, race, by the diameters, indices, angles and cranial or facial profiles: a type a priori Berber.

There remained the hair, of exceptional interest because of its state of conservation fine, supple, slightly wavy in places, of a blond-red pulling on the yellowish. Oval in section, and overlapping all the other anthropometric observations, they are characteristic of the hair of a cymotrichous leucoderma, close to prehistoric Mediterraneans, such as a Berber, with white skin and not a Nubian, with black skin. , contrary to what had been maintained"

Recherches sur les momies

Ramsès II

Pierre-Fernand CECCALDI *, Colette ROUBET **

Les recherches sur les momies pharaoniques ont trouvé leur aboutissement à propos du « traitement » de la momie de Ramsès II.

Préalablement à toute décision, elle a été l'objet d'une étude étendue et approfondie, avec tous examens et analyses propres, bien sûr et surtout, à établir « *diagnostic, pronostic et traitement* », mais aussi, et à la faveur de cette étude même, à en extraire le maximum de renseignements, cela sous la haute direction du doyen L. Balout, au Musée de l'Homme à Paris (1976-77).

L'autopsie de la momie a été remplacée par des techniques radiologiques et dérivées.

Le bilan radiographique et xérographique a montré notamment

pour le crâne :

une perte de matière dans la région de la lame criblée de l'ethmoïde (brèche d'éviscération); une calcification de la faux du cerveau, avec un athérome des sinus carotidiens et des artères temporales; une usure des couronnes dentaires aux maxillaires supérieur et inférieur, avec des géodes au niveau de l'apex radiculaire, dues à des abcès; une sorte de « prothèse » nasale composée d'éléments exogènes, de contour circulaire ou ovalaire.

pour le tronc et les membres :

une néo-arthrose acromio-humérale supérieure droite; une cypho-scoliose dorsale haute, à convexité droite; une pelvi-spondylite rhumatismale; une dysplasie des deux hanches; une rétraction prononcée des parties ex-molles des membres inférieurs, avec un état athéromateux très calcifié; d'importantes craquelures du bouclier résineux au niveau du bassin et des genoux.

L'étude radio-chromo-densitographique a révélé notamment que le rachis cervical et le crâne présentent un aspect traduisant une fracture post-mortem du rachis, redressée certainement lors de la momification.

Quant aux *maxillaires*, toutes méthodes alors combinées ou confondues, leur examen permet d'affirmer qu'il s'agit d'un individu porteur de nombreuses et

* Directeur du Laboratoire de l'Identité judiciaire, Paris.

** Sous-directeur au Muséum d'Histoire naturelle, Paris.

2.4.1 APECED

Autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED) is an immune disorder due to the genetic breaks we've discussed. It allows fungal infection of the ectoderm layer in the womb and results in failure of t-cell tolerance, autoimmunity against various organs, lifetime predisposition to candidiasis and damage to the Thymus, which regulates T cell function and differentiation. ([source](#))

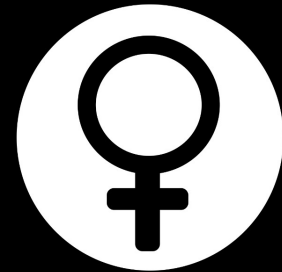
Other features of this genetic disorder are hypoparathyroidism, and primary adrenal insufficiency (Addison's disease). ([source](#))

This can manifest as tongue thrush, nail fungus, balding, auto immune skin disorders, and various other candida related symptoms.

This genetic break via mutation is painted as a rare condition, but we believe it is much more common than expected.

2.4.2 Miscarriages & Birth Defects

Candida is usually the primary culprit when associating fungi with miscarriages. ([source](#))



It kills our children before they are born. But it doesn't stop there. It also attacks male sperm ([source](#)) and even becomes more virulent during pregnancy. ([source](#))

If the child survives this process there is now a possibility of birth defects and permanent neurological damage if they carry the infection post birth. ([source](#))

Mainstream thinking claims that candida is harmless and only causes yeast infections and nail fungus. They were certainly wrong on that as well.

2.4.3 The Thymus: Early Immune Failure



The Thymus plays a critical role in our body's defenses against pathogenic fungi. It is the origin point of new T-cells and also where the cells are differentiated.

For most people this organ shrinks and becomes atrophied early in life, right around puberty, but the damage starts much earlier. ([source](#))

It is one of the most common targets of pathogens, ([source](#)) and fungi in particular. ([source](#))

This causes dysfunction in critical interleukins 17 and 22 in response to fungal pathogens, and is also tied to the genetic breaks under Familial Candidiasis. ([source](#))

Section III: Origins Of Disease



3.1 Inflammation

"Inflammation is the cause of all disease."

Stop right there. Nobody questioned where the inflammation was coming from.



Furthermore, if it's from a commonly occurring source, we can address the real problem.

In order to impose a unified theory where CDF is the primary cause of inflammation, thus the primary cause of most disease, you would need to show that there is a chronic issue regarding exposure, and a chronic issue in the way the immune system handles the response.

The next few sections will dive a bit deeper into how CDF causes inflammation, and how it can be connected to various disease states in the body.

3.1.1 Candida: A Common Trigger

Candida Albicans does immense damage to the body and is a constant source of inflammation.

A primary cause of chronic inflammation from Candida is from its metabolite, Acetaldehyde. When it metabolizes glucose it produces this toxic substance, which constantly damages your DNA and is carcinogenic ([source](#)).

Acetaldehyde directly induces pro inflammatory cytokines ([source](#)). When you combine it with immune defects from Familial Candidiasis, now you have an out of control situation. The immune system cannot remove the infection, and it continues to damage your DNA and cause inflammation indefinitely.

This form of autoimmune loop has been linked to various diseases that we discuss in coming sections.

3.1.2 Mycotoxins

Candida isn't the only source of chronic inflammation. The other abundant source is Aspergillus. Before we explain these toxins, keep in mind that it is in your food (source) and you breathe in the spores regularly, (source) usually from your home environment.



Aspergillus Fumigatus produces Gliotoxin. This potent toxin triggers inflammation, destroys brain cells, eats holes in your brain and is connected to diseases like Multiple Sclerosis (source).

Aspergillus Flavus produces Aflatoxin. This is also prevalent in the food we eat. This toxin damages DNA, is also considered carcinogenic, damages the immune and central nervous system. (source)

3.1.3 CDF Related Interleukins

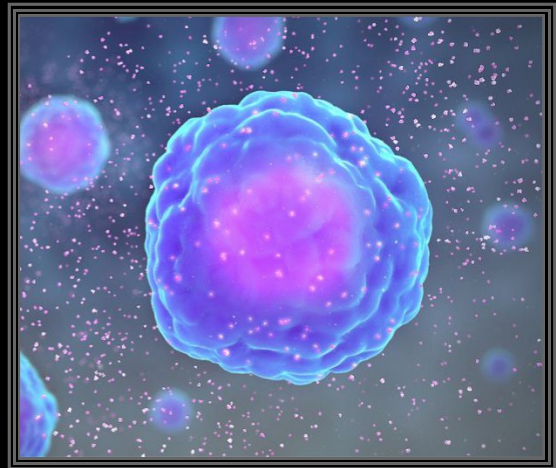
Immune dysfunction against CDF is a potent source of chronic inflammation and autoimmune disease. As mentioned earlier, a common target of CDF is the Thymus, since it plays an essential role in adaptive immune response.

Our immune response is neutered at a young age. Thymus function crashes. Most have their tonsils removed, which is full of both b and t cells used by adaptive immune response. Then the relentless attack against our DNA begins.

Involution of the thymus is one of the most ingenious attacks by CDF. It's connection to APACED and Familial Candidiasis has been made previously ([source](#)).

Chronic inflammation due to chronic infection and genetic breaks show the same responses.

Typically CDF will trigger Interleukin 6 (IL-6) and Tumor Necrosis Factor Alpha (TNF-a). Consistently higher response is seen with Candida ([source](#)) and Aspergillus. ([source](#))



Cryptococcus is unique because it can suppress IL-6 response ([source](#)) as a method of survival.

So what exactly would chronic IL-6 and TNF-a response do to the body?

Interleukin 6 is not only directly responsible for inflammation, but also for triggering the change from acute to chronic inflammation. ([source](#)).

TNF-a induces inflammatory gene expression and apoptosis in healthy cells, also directly contributing to diseases like cancer. ([source](#))



Now you have a direct path. All 3 CDF genre induce IL-6 and TNF-a. Chronic infection that the body cannot clear will create chronic inflammation. Chronic inflammation is viewed as the source of most disease. ([source](#))

Now you understand a cause of chronic inflammation and disease, but more importantly a true source.

The next section will focus on common forms of disease, and the myriad connections that have been made to fungus. The evidence has been there for decades, hidden in studies waiting for someone to make the connections.

3.2 Connections With Common Diseases



3.2.1 Tooth Decay

Before we get directly into tooth decay, let's start with gingivitis. The interplay with fungus is once again required.



The bacterium *P. gingivalis*, responsible for disease, has issues adhering to epithelial cells. Candida is commonly found in pockets of infection due to dental work like root canals, and it directly contributes to adhesion and virulence by triggering a pro inflammatory response. ([source](#))

Candida also lays the groundwork for tooth decay through biofilms on the teeth. In biofilms that Candida forms, any cohabiting bacteria have shown dramatically increased virulence and pathogenicity. ([source](#))

The most recent science shows that Candida can utilize Candidalysin, it's peptide toxin, to penetrate tissue and begin adherence to create biofilms. [\(source\)](#)



Candida causes the initial penetration of tissue and adherence, then forms a multi-species biofilm with *P. gingivalis*.

Without the presence of Candida, *P. gingivalis* has a hard time adhering to tissue.

Fennel oil has been scientifically proven to be safe and effective against biofilm, bleeding gums, halitosis, mouth ulcers, and preventing tooth decay [\(source\)](#) and is one of the solutions provided later in this document.

3.2.2 Cancer

One of the most recent and eye opening studies of cancer was released recently. The study included 17,000 people and 35 different types of cancer. Fungus was detected in the cancer cells of all 35 types. ([source](#))

To make matters worse, CDF sets up this condition using it's own metabolite, Acetaldehyde. ([source](#))

Because CDF is a systemic issue, it can set up cancer virtually anywhere in the body. More recently there has been a direct association with oral cancers as well. ([source](#))

Don't discount Aspergillus, however. One of the most frequently encountered, *A. fumigatus*, produces gliotoxin. This toxin is directly carcinogenic and known to cause cancer of various types ([source](#))

3.2.3 Sclerosis

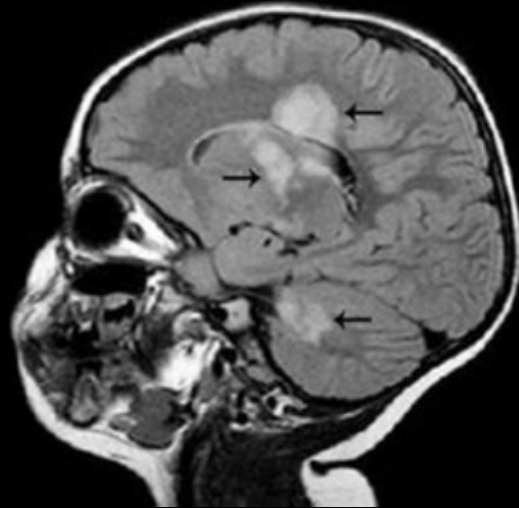
Nothing puts young people in wheelchairs like Multiple Sclerosis. It's the #1 disability in younger people. The level of suffering compounds as loved ones must give up their incomes to care for them.



Science is now catching on to what we've been saying. ([source](#)) The newest research is now making a link to Aspergillus exposure ([source](#)) and the onset of MS. Even back in 2010 they were raising the alarm ([source](#)) but nobody listened.

Science calls it incurable, but we know much about this disease. Aspergillus can be responsible for infecting the brain causing the characteristic lesions ([source](#)) or it can be cryptococcus via infection of microglia cells,

preventing their normal function of clearing abscesses of debris and toxins. ([source](#)) These reactive microglia can directly cause lesions via ROS generation.



It's also possible that both fungi, *Aspergillus* and *Cryptococcus*, can contribute to the disease concurrently. There is evidence that they can increase virulence while cohabiting. ([source](#))

Characteristic brain lesions in combination with damage of the myelin sheath around nerves are the telltale signs of MS.

Remyelination is possible once the source of infection is fully cleared. ([source](#))

3.2.4 Heart Disease

There is a misconception that high cholesterol causes heart disease. The truth on the matter is that high cholesterol is a symptom, not a cause.

The body protects itself from mycotoxins in the blood by binding with a cholesterol or lipid. ([source](#)) The same process is used in the brain to protect it from free roaming mycotoxins. ([source](#))

Recent research has shown that cryptococcus uses a unique mechanism to trigger atherosclerosis via lipid peroxidation. ([source](#))

Not surprisingly, cholesterol drugs were originally tested as antifungals. On the flip side, many drugs derived from mycotoxins can directly trigger atherosclerosis as seen in the above source.

3.2.5 Diabetes and Weight Gain

Diabetes derived from pancreas dysfunction is a hallmark of fungal infection. IL-6 and TNF-a are regular inflammatory reactions to fungal infection, and also show up in pancreatic cancer. (source)

Damage is caused by inflammation of the pancreas, which is responsible for controlling blood sugar via insulin secretion and also releasing enzymes for digestion. (source) Eventually the body cannot regulate insulin correctly, and weight gain rapidly increases. (source)

Cryptococcus (source) and Candida (source) are both known to attack the organ but background inflammation from mycotoxins is likely the major contributor. (source) Candida's production of acetaldehyde plays a major role, as well as brewer's yeast. (source) (source)

3.2.6 Joint Pain, Back Pain & Arthritis

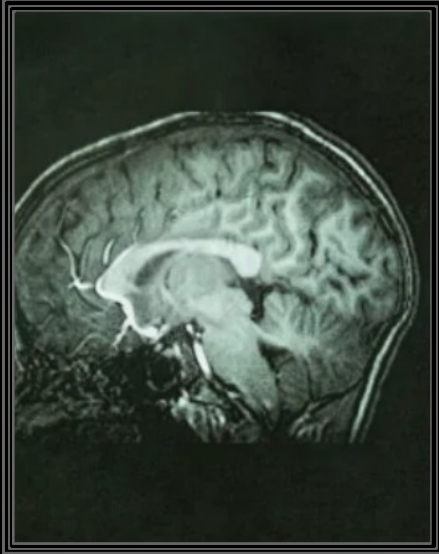
All of these symptoms of suffering usually have the same cause; inflammation that is triggered by long term infection. IL-6 plays a significant part.

Rheumatoid arthritis (considered an autoimmune disease) is directly linked to overactive IL-6 ([source](#)) and Osteoarthritis cartilage and tissue degeneration is as well. ([source](#))

Keep in mind that IL-6 is a hallmark of longterm CDF infection and the associated mycotoxins, covered previously.

Lower back pain is a common trigger when carrying a longterm infection. Sciatica is a good example. IL-6 and TNF-a are commonly found, but also IL-17 and IL-22, specific to fighting fungal infection. ([source](#))

3.2.7 Diseases Of The Brain



This information is going to be shocking to some people. Various brain related diseases all have common causes. Neuroinflammation gets blamed often, but once again, we question where it comes from.

We're seeing multiple angles of attack. More than one species can work together to cause these issues, and even increase virulence in combination. ([source](#))

Amyloid beta plaqueing is associated with Parkinson's Disease ([source](#)), Alzheimer's Disease ([source](#)) and Multiple Sclerosis ([source](#)) to name a few. So how does this slime accumulate on the brain and in lesions, preventing the body from repairing itself?

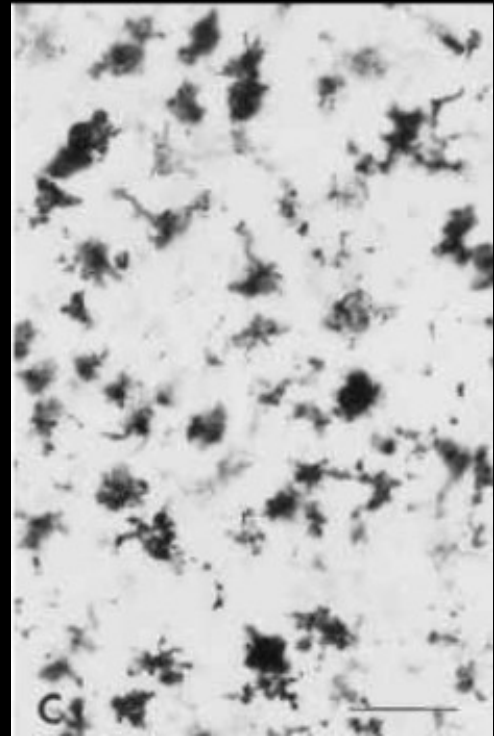
Unfortunately there are multiple ways.

The first is through mycotoxin mitigation, causing hypercholesterolemia and amyloid beta buildup ([source](#)).

This is a similar defense mechanism that A.V. Constantini discovered in his research of Atherosclerosis, with lipids binding to mycotoxins in the blood stream mentioned earlier.

The second method of amyloid plaquing is cryptococcus. This fungus is very advanced. One of the reasons Multiple Sclerosis is difficult to treat is because cryptococcus zombify microglia, which are the cells responsible for clearing debris and toxins from lesions. They then become reactive microglia ([source](#)) and directly produce amyloid beta, preventing the lesions from healing while also further damaging the brain with ROS/NOX2 ([source](#)) from glial dysfunction.

Cryptococcus is also known to directly infect astrocytes, ([source](#)) which normally assist in plaque removal. Once cryptococcus invades astrocytes, this not only weakens the blood brain barrier but increases plaquing. ([source](#)).



The third method of infectious disease progression is yet to be defined publicly, but to summarize we believe it is a gene defect related to cryptococcus that is Familial, and interferes with normal ability to prevent NOX2 oxidation. More information will be available on this in future updates.

3.2.8 Accelerated Aging

How is aging defined? It's generally accepted that genome damage, telomere shortening and mitochondrial dysfunction are major contributing factors.

We've already discussed how CDF is constantly damaging our DNA, and puts us into a state of chronic inflammation. Part of this cellular damage is also mitochondrial dysfunction.

To get technical for a moment, a structure called a g-quadruplex is responsible for performing many genome repairs in the cell. When the body contains active fungal infections, DNA damage occurs. Also the ability of g-quadruplexes to repair DNA errors is impeded. CDF directly causes an oxidative reaction in the cell (ROS) and destroys the g-quadruplex. Additional damage to the genome happens via oxidative lesions to telomeres. ([source](#))

3.3 Conclusions

We've covered a lot of material in this section. We've shown how chronic inflammation is triggered, what pathologies are responsible, and how CDF genera contribute to the most common diseases.

Considering the contribution of CDF and its mycotoxins to chronic inflammation and the top diseases by mortality, the problem becomes so overblown that it's difficult to understand why we are in this situation.

The death toll is minimized by focusing on pathologies featuring chronic overgrowth, like cryptococcal meningitis. But what if we add up the numbers based on cancer, heart disease and the other diseases mentioned?

What level of suffering and misdiagnosis will it take before this issue is given the gravity it deserves?

SECTION IV:
THE NAC PROTOCOL



4.1 Introduction And Back Story

Peribsen and MacLir are pen names for the creators of The NAC Protocol. It began when they started sharing improvements from supplements they found.

MacLir suffered from sciatica and constant lower back issues. After taking curcumin for a few months and realizing it helped, he passed on the information to Peribsen. Shortly after, Peribsen also saw great benefit from such a simple solution.

Peribsen then noticed he was having thrush issues and went to the doctor. A round of fluconazole and nystatin was provided, but what happened was a chain reaction. Peribsen suddenly realized that these medications were helping with various other issues he'd had for a lifetime. Soon the conversations began. Things began to click. The curcumin that helped both of them was also a potent antifungal.

Was it really possible that all of these issues were related to fungus? Both began pouring through research and realized that the answers were there all along.

Things moved very quickly at this point.

Soon they began working with a geneticist, doctors and academia. It was decided that they would work out a natural protocol that could benefit others, and they made a commitment to research and release the protocol, and do it freely at no cost.

Many months passed as they continually became more healthy and lifetime symptoms disappeared. They shared research on hundreds of different antifungals and combinations. They eventually found the perfect combination based on many months of research and experimentation. They dedicated the next period to simply following the protocol.

The results were astounding. The combined compounds truly worked magic in the body. They began sharing it with friends and family and they had the same positive results.

In December of 2021, they released the protocol into the wild. With help of some friends (including myself) an online presence was formed on forums and the first pdf was created. Since those humble beginnings, we have been tracking hundreds of positive reports online.

We sincerely hope you find it as beneficial as we did.

4.2 Protocol Methodology

If you've read through the first 3 sections, you understand the nature of the problem. The NAC Protocol was designed to be safe, effective, and able to be used by anyone of any age. There are a few caveats.

The protocol will naturally lower blood sugar and blood pressure. It can also have a blood thinning effect. You may notice lower Iron, Calcium and Zinc levels. You may want to consider a quality multi vitamin as a daily routine.

Even though the protocol is considered safe and effective, if you are suffering from insulin issues, high blood pressure, asthma or have pre existing health issues or use prescription medications, please consult with your doctor before starting the protocol.

There are 3 components to The NAC Protocol. Those are Oregano Oil, Black Seed Oil and N-Acetylcysteine (NAC).



Oregano Oil is the heavy hitter.

It was chosen out of hundreds of potential antifungals because it contains several unique properties we were looking for. It will do most of the fungal eradication and also assist in breaking down biofilms.

It also attacks pathogenic bacteria as well, so it's great to deal with dysbiosis issues that can cause irritable bowel syndrome, digestive issues, acid reflux, etc.

It also shuts down inflammatory response from the immune system, so helps in the healing process. It repairs the tight junctions in the gut barrier, correcting any 'leaky gut syndrome' and is safe to use longer term.

N- Acetylcysteine (NAC) provides multiple functions in the protocol. It's an effective biofilm breaker and also an antifungal as well.



It serves as a scavenger to Candida's metabolic products to help with die off symptoms. It helps protect the liver during detox.

It's also crucial to cell and DNA repair. It increases the necessary cysteine and guanine stores to regain proper cell function after years of damage. It boosts glutathione during the process to help with any oxidative damage while you are removing the source of the damage.

It chelates various toxic byproducts and moves them out of the body. During wholesale destruction of fungi and their cell contents, it benefits in the cleanup process.

Black Seed Oil is considered a powerhouse of healing. It contains many powerful antifungal and restorative compounds that occur naturally, one being thymoquinone.



This is the restorative part of the protocol. It protects the liver during detox, but most importantly it can dramatically increase the effectiveness of your immune system. It will increase T-cell count and differentiation and also acts as an immune modulator.

The longer you take it, the more effective your immune system becomes. And the results are lasting, even after you stop using it.

Black seed oil also increases the effectiveness of antifungals and decreases fungal resistance.

Now you've seen the 3 components to the protocol, and how all 3 are both antifungal and biofilm breakers. They work together in a carefully balanced synergy that supports the body as you rid yourself from fungus.



Each compound serves a function, but all 3 contribute as a liver protective, antifungal and biofilm buster. These 3 compounds offer a complete solution to the eradication of CDF.

By halting DNA damage and blocking CDF's oxidation process, the body begins to repair itself correctly. For many people this will be the first time functioning at this new level.

The possibilities are now endless. Brain fog lifts, many aches and pains disappear, and a new found motivation begins to take form.

4.3 The NAC Protocol

Morning

1200mg NAC

Oregano Oil (min. 40mg Carvacrol)

Black Seed Oil (1 teaspoon)

Night

600mg NAC

Oregano Oil (min. 40mg Carvacrol)

Black Seed Oil (1 teaspoon)

Continue daily for a minimum of two months and count out 3 weeks with no die off symptoms prior to moving to the next step.

Fungal die off symptoms may include :

Tiredness, exhaustion, muscle soreness, increased chest or nasal discharge, cold or flu like symptoms, cold sores, headaches, rash, acne, irritability, change in stool frequency, volume or color; increased urination, bloated stomach, cramps, increased gas.

Maintenance Protocol

Morning

600mg NAC

500mg Slo Niacin (nicotinic acid)

100mg Pterostilbene

250mg Pomegranate Extract (min. 40% EA)

Black Seed Oil (1 teaspoon)

Night

500mg Slo Niacin

100mg Pterostilbene

250mg Pomegranate Extract

Black Seed Oil (1 teaspoon)

After every 3 weeks on the maintenance protocol, take 1 week off. Continue to use black seed oil during the off cycle.

Sourcing the supplements required requires some diligence. When selecting your oregano oil, you want to focus on products that list the specific carvacrol content on the bottle. Deceptive practices with 10:1 extracts like 'typically contains' is a warning sign and should be avoided. The next page contains a list compiled of every brand we could find (in the US) that actually lists the carvacrol content.

Minimum carvacrol content per morning and night dose is 40 mg. As a general rule, if one capsule contains over 50mg carvacrol, you take 1 morning and 1 night. If one capsule contains less than 40mg carvacrol, you take 2 morning and 2 night.

For POM extract you want 100mg Ellagic acid for each morning and nightly dose. If you buy a Pomegranate extract that has 40% Ellagic Acid, that means a 250mg capsule contains 100mg Ellagic Acid.

Carvacrol amounts listed per capsule

Biotics Research (33mg)
California Natural (32mg)
Carlyle (20mg)
Designs For Health (36mg)
Gaia Herbs (27mg)
Gold Source Labs (32mg)
Herbs Of Gold (80mg)
Island Nutrition (75mg)
Natural Factors (144mg)
Nature's Way (37mg)
NOW Oregano Oil (99mg)
Oreganol Super Strength (102mg)
Pure Mountain Botanicals (32mg)
Pure Synergy (36mg)
Source Naturals (31 mg)
Solaray Aerial Extract (40mg)
The Vitamin Shoppe (super strength) (32mg)
Vitality Works (32mg)
Wellness Resources (55mg)
Zane Hellas (130mg)
Zenement (106mg)

We only recommend cold pressed black seed oil in a larger bottle.

There are two brands in the US that we can recommend, Horbaach and SVA Organics. Both provide a 16oz. bottle for \$25, which will last for months.



European sourcing is more limited and can be more pricey. For black seed oil you can find Kräuterland Black Cumin Oil on amazon.de, or visit a UK supplier located at <https://theblessedseed.com/shop/strong-black-seed-oil-1-litre/>

Oregano oil can be ordered from Europe via vitaminversand24.com or Vit4Ever on Amazon.

4.4 Brushing And Upper Respiratory

Continuous reinfection from CDF happens from inhalation, the oral cavity and the upper respiratory. The mouth and sinuses are especially vulnerable. To reduce this daily while doing the protocol, there are two methods that are effective and should be combined with the protocol.

Xlear makes a nasal spray that contains Xylitol and Grapefruit Seed Extract. Using this in the morning and before bed will break down any biofilms and drain them out of your sinuses. It can be used as a prophylaxis for 4 to 6 hours of protection when around large groups of people or sick people.

The brushing routine involves using 1 to 2 drops of fennel oil on your toothbrush, with or without toothpaste. Fennel is a very strong antifungal and will clear the mouth and any pockets of infection over time, which ends up being another source of reinfection.

4.5 Common Die Off Symptoms

The onset of Die off symptoms will be different for everyone. With a large, active fungal overgrowth, Die off can occur in the first few days. Some people may take weeks to experience any major symptoms. People who are physically fit and follow a good diet may take up to a month before Die off occurs. This is because their body is managing active infection levels, but the protocol is slowly breaking down mature biofilms that have been there for years. When those biofilms release their pathogens, Die off is likely to occur even in healthy individuals.

The most common symptoms based on over a year of reports are bloating, stomach discomfort or temporary sharp pains, headaches and Tiredness. Some of the other Die off symptoms listed in the protocol in Section 4.3 are a result from breakdown of biofilms. When these release you will have viruses (including herpes), bacteria and fungi suddenly trying to replicate and

survive. This is usually where flu like symptoms come from.

During eradication of pathogenic fungi, the protocol disrupts (punctures) their membrane and the cell contents are dumped out. Your body needs to clean up this mess, which will include various toxins, fractured nucleic acids and polysaccharides, just to name a few.

There are several strategies to assist with die off symptoms and make them more manageable. Regular use of probiotics in the form of yogurt, kefir or fermented foods will absorb and decrease the toxins improving your symptoms.

The second option is to use mycotoxin scavengers to eliminate or convert the toxins. This involves introducing compounds that bond molecularly to the toxin and render it harmless.

NAC is already a scavenger for multiple mycotoxins, but there are other options. Molybdenum works well with Candida die off. L-Methionine scavenges Gliotoxin. Glucomannan works on Aflatoxin and Ochratoxin.

Coq10, Vitamin K, Arginine and Glycine can also reduce die off symptoms via chemical bonding to mycotoxins.

One of the simplest ways to decrease detox time is increased water intake, fiber and rest. Your body will tell you when you need rest. Try to get extra sleep and you will find yourself feeling better upon waking.

4.6 Common Experiences

Okay, now we can focus on the positive for a bit. There are many common experiences that we call breakthroughs when using the protocol. These shared experiences give glimpses of what is to come, and some are even more profound.

Decreased brain fog and an increase in vivid or lucid dreaming is commonly reported. At some early point in the protocol, you will beat the fungus back enough and experience an incredible feeling of vitality, where your body feels amazing, and your thoughts and feelings move toward momentary bursts of joy. This is a huge relief for people with depression or long term body pain or stiffness.

These moments are typically fleeting and may only last a day or two before you go into the next round of die off. Remember how good you felt at that moment and use it to overcome the next battles.

4.7 Diet Considerations

There has been and continues to be much debate over candida diets. There is a reason that traditional candida diets fail for many people. First, it's far too restrictive and prone to failure. Second, eliminating sugar won't solve the problem.

The answer that works for the most people, without getting into crazy diets that are prone to failure, is to reduce sugar where possible and avoid refined sugar, processed foods and yeast.

You don't have to go hungry, stop drinking coffee or do anything crazy. Cut out bread and yeast products.

Generally a modified paleo diet with added carbs considered low glycemic is good, but the ideal diet is the one you can stick with.

4.8 Conclusions

This has been a brief overview of The NAC Protocol. It was meant to be easy reading, but if you prefer to dig in deeper into the science behind how it works, jump directly to Section 7.

The NAC Protocol has provided a solution to a lifetime problem for most people. Mitigation of fungal and bacterial pathogens and their dangerous mycotoxins.

We invite you to join in the daily conversation where we answer questions and post the latest research.

Join our [reddit community](#) to get real-time updates at r/cosmicdeathfungus and join the conversation.

SECTION V:
The Maintenance Protocol



5.1 Introduction & Methodology

The Maintenance Protocol is the last step of The Nac Protocol. Each individual is unique in their physical health, level of genetic breaks and level of hard to hit infection in the peripheral nervous system and organs.

The purpose of this stage fulfills 3 different requirements based on individual need. First, it provides vasodilation combined with a systemic antifungal (Pterostilbene or Resveratrol) to hit harder to reach areas like the hands, feet, interstitial and other areas that are only accessible via small capillaries. This helps clear out the last outposts in the body.

Second, by using sirtuin activation and the increased NAD pool provided by niacin, a special process of genome repair is activated that uses master template transcription.

Third, it provides a longterm answer for those with genetic breaks who need regular antifungals to stay healthy. It puts the body into homeostasis and provides relief from autoimmune response or dysfunctioning mitochondria, preventing full healing.

5.2 Maintenance Protocol

Morning

600mg NAC

500mg Slo Niacin (nicotinic acid)

100mg Pterostilbene

250mg Pomegranate Extract (min. 40% EA)

Black Seed Oil (1 teaspoon)

Night

500mg Slo Niacin

100mg Pterostilbene

250mg Pomegranate Extract

Black Seed Oil (1 teaspoon)

After every 3 weeks on the maintenance protocol, take 1 week off. Continue to use black seed oil during the off cycle.

During the maintenance protocol, NAC continues to provide the cysteine and guanine stores required for functional DNA repair in G-quadruplexes. Oregano is removed as the primary antifungal and replaced with Pterostilbene or Resveratrol, providing the additional benefit of sirtuin activation with antifungal activity.

Niacin is introduced to increase NAD pool levels and works directly with the sirt activator to repair DNA on a deeper level.

There are a few options on forms of Niacin. This protocol recommends timed release nicotinic acid (Slo Niacin) as an affordable alternative that prevents the systemic flushing effect of pure Niacin. It's a general solution that works for most people, lasts longer in the body as a timed release and is regarded as safe. There are benefits to the other forms of niacin available as well.

The flush form of niacin does the most dilation of blood vessels and capillaries, pushing active compounds deeply into the CNS, interstitial and tissues. The active time in the body is short, however, and many people don't like the flush effect.

Nicotinamide or Niacinamide is a non flush form and can also be used as an alternative to Slo Niacin or Nicotinic Acid. The downside is the short activity time in blood plasma, which is where a timed or extended release form holds the advantage.

The third Niacin option is Nicotinamide Riboside, which has more unique properties as far as NAD pool uptake and brain benefits. It also suffers from a short active time.

Originally the protocol used Resveratrol as a sirtuin activator, but we moved to Pterostilbene for a few reasons. First, it is 5 times more

bioavailable. Second, it lasts 7 times longer in the body. Combining Pterostilbene with a timed release Niacin was the obvious solution to keep those therapeutics active for as long as possible.

There were concerns over long term niacin use being potentially toxic to the liver. After a review of the literature, this occurred specifically with Sustained Release (SR) formulations. Regular niacin and Extended Release (ER) or timed release are considered safe at the dosages for this protocol. Problems occurred in studies with SR primarily, and at dosages above 2g daily.

5.3 Die Off Symptoms

Symptoms during this maintenance phase of the protocol are harder to predict. People respond differently to sirtuin activation based on genomic traits and also long term infections that have been well protected for years.

For most people without significant genetic breaks or an official Candidiasis diagnosis, starting the maintenance protocol will start with some exhaustion and muscle soreness. This goes away within a few weeks for most people.

The severity of genetic breaks or peripheral infection can lead to surprising die off symptoms after smooth sailing with the initial protocol. This can be as extreme as nausea and vomiting, which typically lasts only a few days, or it could be extreme fatigue and headaches.

5.4 Common Experiences

Maintenance is truly a godsend for many people, especially as they age. After experiencing any initial die off or adjustment to sirt activation, there are many benefits to both young and old.

One of the most commonly reported benefits is a new general feeling of wellbeing, with a body free from any recurring aches and pains, more energy and better sleep.

More long term use of the maintenance protocol has prompted many unique personal reports, likely due to genome repair over time. This includes moles disappearing from the skin, new hair regrowth, correction of ophthalmic issues and much more.

The reports that appear almost miraculous in nature are likely due to a few factors. With a lifetime infection with CDF, destruction of the guanine quadruplexes in cells never gets fixed.

After the initial protocol and the maintenance protocol fueling higher levels of repair, we do not even know the limits yet as far as what can be undone.

5.5 Conclusions

The NAC Protocol and latter maintenance phase provide a real solution to the causes of disease, aging and suffering. By mitigating fungal colonies, we remove the source of most mycotoxins and stop DNA and mitochondrial damage. As cells are regenerated through autophagy and mitophagy, they now have functioning guanine quadruplexes and the body works as intended.

The immune system functions at new levels and is far better at staving off invasion.

Then the maintenance phase begins undoing years of genetic damage, giving our innate abilities as humans back to us. A properly working mind, body and spirit.

With everything working as intended, you can now call yourself fully human. You are no longer a distorted genetic mess under constant attack.

You now hold the keys to unlocking your true potential, the ability to put thought into action in new ways, and increased perception, awareness, and a spiritual connection that is unhindered by invasion. If you'd like to read more on the science or mythology behind the protocol, please keep reading.



SECTION VI:
Historical, Mythical & Religious
References



SECTION 6.1 :

Egypt



6.1 Origin Of The Tribes

The legends of great illnesses and healers are prevalent throughout the verbal and written histories of mankind.



If we sacrifice dogmatism and consider these as more than just fireside stories from past civilizations, we find examples of viable treatment approaches.

Is it an effect of fungal infections over the course of thousands of years that has led to our forgetting ways to heal the body of these diseases? ([source](#))

Warnings of avoiding the underworld are common in nearly every ancient culture's accounts. The earliest genetic records of modern humans in Northwest Africa come from Taforalt in Morocco. ([source](#))

This cave system, referred to as the Grotto of Pigeons, was utilized for over three hundred thousand years as a dumping ground.



Human remains, waste, and animal remains all have been found at the site. As the name suggests there were large populations of pigeons inhabiting the caves. Then during the Younger Dryas period, melting glaciers around the Atlas Mountain range prompted a shift in the level of the water table. ([source](#)) ([source](#))

Higher water levels brought up material from the depths of the cave systems, exposing the population to pathogens that their immune systems could not manage. Illnesses would have naturally increased as a result and prompted a response from the peoples of the area.

As shown in date progressions of genetic data there were migrations eastward from Taforalt.

These settlers reached Mauritania, Libya, Egypt, and even regions of the far North such as Lapland within the arctic circle. (source)



These migrations and genetic records show that a major shift in temperature and water levels prompted a large-scale exodus out of the region. (source)

Some of these settlements predate nearly every other record of human activity in the area. Taforalt is certainly one of the earliest examples, with northern Libyan settlements and western Egyptian settlements also being dated to neolithic timeframes. (source)



Of note is the Siwa Oasis in western Egypt, known as the oldest settlement in the nation, with dates that go back to the 10th millennium BC. ([source](#))

It is still active and is the site of the Temple of Amun, which is still standing today.

With the Imazighen tribes came their gods and legends, upon which were founded the religions of many cultures. Atlas, Hercules, Ash (later absorbed into Seth), Amun, and Osiris all have origins in Berber religious roots. ([source](#))

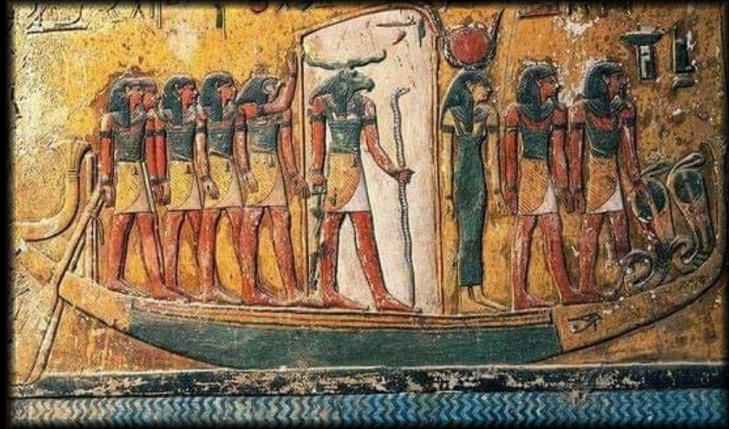
We will use these gods as a point to start an exploration of overlooked medical knowledge that has been set aside in favor of modern approaches.

For what reason have we been taught these records were not to be heeded as bearing any truth?

If we look for parallels between the records of pre-history throughout the Mediterranean, a multi-faceted story of mankind's hindered progress emerges. It should be said the "separate" groups that emerged as time progressed weren't separate at all, and that the pieces of the solution to disease and death were ours all along.

6.2.1 Egypt: The Ennead

Seth has been a god in many pantheons, originating from the Imazighen god Ash who was known as god of the oases.



With the western tribes being known as “people of the oasis” ([source](#)), a way to identify outsiders from the Red Lands, the Berber origin of Ash and Seth becomes clearer. Seth’s nomadic and chaotic tendencies are an integral part of the larger story we are attempting to decipher.

Egyptian attitude towards Seth changed dramatically from a benevolent protector to a villain. This change took centuries, and by the time of Egypt’s New Kingdom period he was known as a jealous murderer. ([source](#))

Should this be considered an example of CDF in ancient times, even affecting the attitude of the populace toward their gods? In his earliest form as a protector, the god Seth was known best for protecting Ra the creator god from the serpent Apep. ([source](#))

This evil serpent in the Egyptian religion was the bringer or representation of death and disease. Every night Ra and Seth would go to the underworld when night fell and battle Apep.



Seth would pierce him with a spear to drive him back to the edge of the world. He was believed to have hid behind Baku mountain to the west of Egypt – also the direction of Taforalt from Egypt. ([source](#)) The same site we propose introduced mankind to destructive levels of pathogenic fungi.

Many aspects of Apep are like the aspects of fungal infection we have been discussing. It was viewed by Egyptians as an incarnation of non-existence, chaos, and darkness itself. Its bite was venomous, and he could shift his size, shape, and anatomy to suit the moment.

In one story he was able to mesmerize the entire Ennead on the solar barge, except for Seth, who was unaffected and drove him back. ([source](#))



Is this a reference to the confusion caused by cryptococcus when it has taken hold in our brain? There is even reference to Seth eventually taking on all aspects of Apep – was this an early explanation of infection from exposure after those nights on the barge of Ra?

In most Egyptian artwork that represented the struggle against Apep, we see Seth on the barge ahead of Ra. Spear poised to strike the serpent and hold it down while Ra, in the form of a cat, cut off Apep's head. In nearly every representation of this part of the legend the serpent god is barring access to the Acacia tree – considered by ancient Egyptians to be the tree of life. ([source](#))

Another recurrent theme in early Ennead stories is conflict between Seth and his brother Osiris. A centerpiece of this struggle was disagreement on staying nomadic like Seth and those from the Red Lands, or building great cities like Osiris and those from the Black Lands. These stories served the purpose of explaining the attacks from Red Lands outsiders on the cities of Lower Egypt.

They also have a deeper connection to the concepts that were prevalent around the Mediterranean that mankind should not settle – cursed to instead wander the Earth.

The settling of Egypt by Osiris led to issues that show connections to yeast and its damages. Known as the first builder he was revered greatly in the settled regions of Egypt.

Alongside his sisterwife Isis he was worshipped for founding the greatest city known to Egyptians of the era.

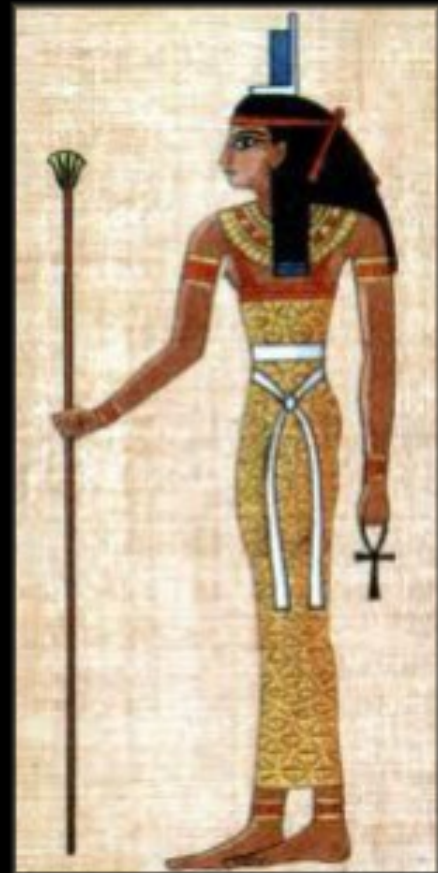
A backbone of their gifts to the people was that of bread and beer.



Even in ancient times, the yeast that was most prevalently used was *Saccharomyces Cerevisiae* ([source](#)), a close relative of other pathogenic yeast and in particular *Cryptococcus Neoformans* ([source](#)).

Isis, called as the Lady of Bread and Lady of Beer, was worshipped as one of the most important gods of the Egyptian culture. ([source](#))

She was a devious goddess who was considered very wise, and not above utilizing complicated machinations to achieve her goals. Her introduction of bread and beer to the people of Egypt can be taken as a method of controlling her subjects more fully.



At first harvest the workers would lay their tools down and pray for Isis to bless the crops that have already been trimmed. Wheat that has been trimmed is naturally inoculated by yeast, leading to what we now call sourdough leavening. ([source](#))

Was this a prayer for Isis to bless the dead wheat with “life” and in turn make it viable for bread and beer production?

In many situations these two items were the only pay that the commoner would receive, a meta-connection to the concepts of prosperity in the minds of Egyptians.

This gift of life, or leavening, is represented religiously as the Ankh – also known as Isis' girdle.



This abundance of bread and beer throughout the cities of Egypt reflects one of the initial changes brought about by pursuing agriculture, and was another point of conflict between Seth's nomadic tribes and Osiris' builders.

A more widely known story of these brothers' infighting is the murder of Osiris. This entire series of events was prompted by a factor that isn't always mentioned as important – Seth's infertility.

This drove his sisterwife Nephthys to seduce Osiris by posing as Isis resulting in the birth of Anubis. Although he had been seduced, and had no blame in the matter, this was unknown to Seth who plotted to murder his brother. ([source](#))

Seth's method of choice for murder varies among Egypt's many dynasties, the most famous being the sarcophagus banquet, where Osiris is trapped in a custom-made coffin and drowned in the river.

After much searching, Isis locates her dead husband's body grown into a tree that had sprouted around it far downriver from the banquet. In most accounts his body is then chopped into many pieces and spread across Egypt by Seth to prevent any resurrection attempts. ([source](#))

Isis reclaims his body parts excepting his phallus. By burying each part where it was found and making a tomb, she is able to resurrect Osiris who becomes god of the underworld. ([source](#))

Duat, as the underworld was known, represented a gateway of sorts more than a direct punishment.



A soul could be denied an afterlife altogether if the person had done more chaotic than good in their life, referred to as the second death. Otherwise, Osiris would bring them to the field of reeds where they would live out a new life in a perfected world.

A reasonable interpretation of Duat is as a means to keep the dual soul in a state of death and darkness or subjective illusion, not allowing full immortality to be achieved. ([source](#))

In the Papyrus of Ani, The Chapter of Not Dying a Second Time, there is specific mention of a peculiar way to avoid the second death:

“Let quietness of heart be given unto me instead of cakes and ale”. ([source](#))

Another reference to yeast preventing a connection to the beyond, possibly even interfering directly with the soul’s ability to move on.

In this section of the instructions for gaining eternal life, forsaking bread and beer is part of the path to ascension.

Continuing the theme of confusion and deception related to the serpent and its venom, we now will look to an interaction between Ra and Isis.

Having tired of Ra’s rule as an aging god, Isis set an intricate trap to steal his secret name.

With this power she would be able to install her son Horus the Younger as the ruler of Egypt. A god’s secret name in Egypt was considered the seat of their true power, in this case Ra’s control over life. ([source](#))

Isis knows the route that Ra travels every day to take in his creation and created a snake out of Ra's spittle and dust from the ground.

She gave life to this creation and set it along his path with the intent of biting Ra to poison him.

As intended the snake bit Ra's ankle and he cried out for the Ennead to aid him, with Isis being one of the first to offer her assistance.



While Ra is slowly dying from poisoning, she pressures him for his secret name, explaining only this has the power to remove the venom.

Ra relents and is sent on to rule the heavens and leave the earth to Isis and Horus. ([source](#))

In this story we see an example of an ancient curse that relies on spittle and dirt. Both items contain many microbes, and cryptococcus and candida are both extremely common in soils, droppings and wood detritus. ([source](#))

There are many elements of the Ennead legends that point to an early knowledge of yeast as a double-edged sword. Both a requirement of living in the era, and a means of keeping commoners in check via pathogenic exposure in the food supply.

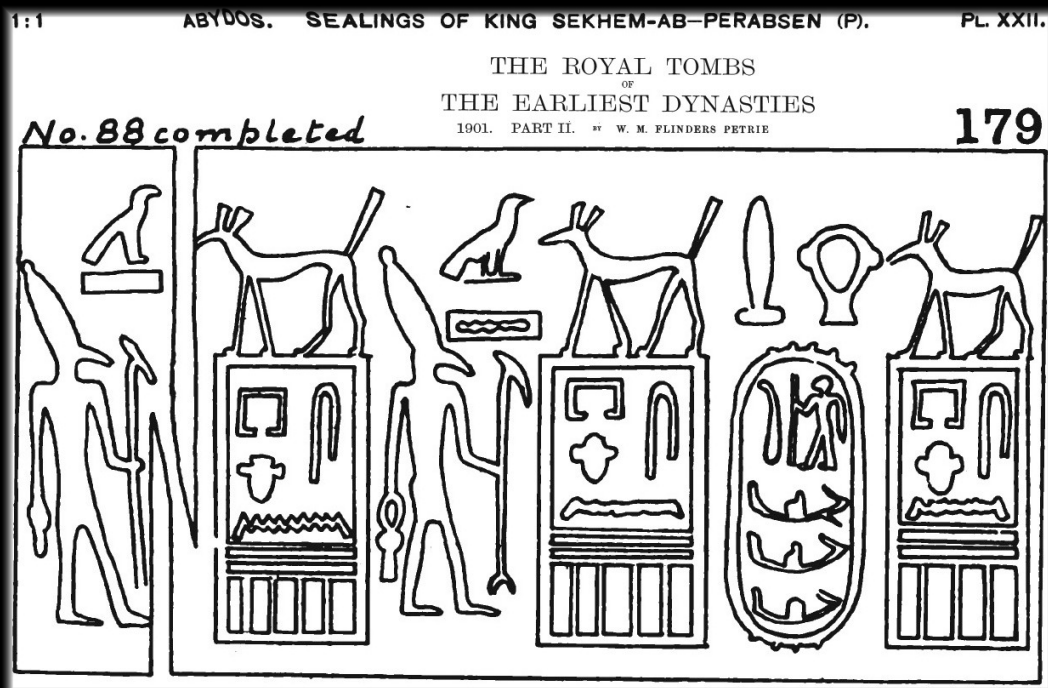
Seth's conflict with his Ennead family should be taken as a warning against the progression of these invasive fungal diseases in a civilization. At first Seth and the others coexisted amicably then issues that have proven related to fungal infection begin showing up in the legends.

Infertility, confusion, poisoning from exogenous toxins are all shown to have had a negative effect on the Ennead.

It should also be considered that Seth was rewritten negatively during later periods to promote the bringers of yeast to the populace. At some points in Egypt's history those who looked like Seth, the red-headed god, were sacrificed to Osiris.

This was in honor of Osiris being opposed to Seth and was also a means to remove foreigners who had come from the Red Lands, likely the Siwa Oasis mentioned previously. ([source](#))

6.2.2 Egypt: The Pharaohs



There are close ties between the Pharaohs of Egypt and the gods we have discussed so far. One of the earliest is Seth Peribsen, 2nd dynasty, who installed Seth as primary deity and took his name to the throne instead of Horus'.

He was a ruler who came from the Upper Egypt area associated with the Imazighen rooted tribes from the oases.

The knowledge of avoiding settlements and thereby exposure to fungal disease would have been common to pastoral tribes from the west. The attachment to Seth has deep ties to the Taforalt origins of gods and life in the region.

During his reign all of Egypt advanced greatly in both the religious and learning arenas. Even writing itself was perfected to the point that his tenure saw the first complete Egyptian sentence known.



It read: “The golden one, he of Ombos, hath unified and handed over the two realms to his son, the king of Lower and Upper Egypt, Peribsen”. This was found as a clay seal in the proposed site of Peribsen’s tomb and reflects well on his cultural advancements during the 2nd dynasty. ([source](#))

When Egypt's territories consisted of much smaller populations, one of the duties of the Pharaohs' priests was medicine. The change to Seth worship would have brought with it the diet and medicine of Upper Egypt and the Red Lands.

With the known impact of fungi on brain structures and function, these changes in Egyptian culture could be considered a reflection of broad health improvements. Possibly enough to prompt a golden age in the Old Kingdom.

In the mentions of medicine from the Old Kingdom, Imhotep cannot be overstated and is widely considered the first true physician in Egypt.



He gathered a series of case reports and treatments into a manual of medical care accurate enough they are sometimes referred to in modern medicine.

A copy of this treatise was preserved as the Edwin Smith papyrus, although it is not considered complete as the last case ends prematurely. ([source](#))

Imhotep was respected for his expertise in many fields that ranged from medicine, to engineering, to priesthood. His most famous construction was the temple of Djoser which is the oldest stone building in history.

He oversaw not only the long-term building project but also the medical care of the entire work crew. He was the first to be called a physician, the first to be called an architect, and eventually became recognized as a god of healing. ([source](#))

This movement of a great healer from commoner to god may also give insight to the benevolent form of Seth as a healer in earlier Ennead frameworks.

Considering these achievements in the framework of the golden age, they present as an example of a mind not bogged down with pathogenic fungi. With his knowledge of fighting infections, and the many plants with antimicrobial properties used in this period ([source](#)), Imhotep most likely removed the fungal sources of disease from himself.

As a result, the works that he was able to complete shadowed everything that had come before – still admired today as the beginnings of entire disciplines of thought.

As Egypt grew in population and influence in the region, there were new immigrants to the area, most noteworthy being the Hyksos.



They were a group of outsiders from the perspective of the indigenous Egyptians, but integrated into Egyptian culture throughout the centuries.

Levantine in origin, name, and religious aspect the Hyksos eventually came to rule Lower Egypt in the 15th dynasty. ([source](#))

The previous two dynasties were marked by a potential famine leading to great declines in population and power structures. This improved once the Hyksos kings came to power and we see another period of improvements to Egyptian culture.

Contrary to other rulers of the nation, one of the better-known pharaohs from this dynasty was recorded under an odd name. He carried the name of the serpent god of chaos and death Apophis. During his reign struggles between the divided Upper and Lower Egypt occurred more frequently than previously.

This culminated in the peace between the Hyksos and Thebans deteriorating and the Theban pharaoh Seqenenre dying on the battlefield. By the end of the 15th dynasty the Hyksos were displaced from rule. ([source](#))

Some consider this displacement a potential explanation for the legend of Exodus that is recorded in the Torah.



While he is known as Apophis, in truth he was a worshipper of the god Seth. Were some of the old religious ways of the Levant brought along with the Palestinian roots of the Hyksos that settled Egypt?

Seth has a strong connection to the earliest Levantine tribes with his being part of their story of the creation of the world. ([source](#)) This dramatic change of worship that upset the norms of the era is worthy of consideration, as Seth eventually became synonymous with Apep.

Once the Hyksos were routed at the end of the 15th dynasty, they were primarily removed from the records.

Said another way, marring their record by associating negative traits of Seth as Apep to their rule would be propaganda to support Theban rule and their preferred Osirian worship.

Worship of the god of the underworld is maintained steadily until the 18th dynasty, which brought with it the most dramatic shift in religion seen by ancient Egypt.

Akhenaten – whose name means “Effective for Aten”. Known primarily as the heretic pharaoh. In a land where polytheism had always been the prevailing religious structure, Akhenaten changed worship to a monotheistic system with the Aten at its core. ([source](#))

A type of singular creator worship in the form of life-giving Sun with its hands that reached the entirety of the world, touching all with its power.



The Amarna period as it is known today was another time of change far beyond just the religious component.

The capital was moved to Akhetaton, and depictions of the pharaoh's family were like none from previous dynasties, with exaggerated features that are still unsatisfactorily explained. ([source](#))

Some iconography of Akhenaten shows him offering gifts to the Aten with no sign of the ankh to be found.

There is a possible explanation for the unique features of the pharaoh and his turning away from Isis and her gift of bread and beer.

Might Akhenaten have linked an unexpected healing process, the issue of yeast in the food supply, and the effect of the light of the sun on fungi together? His family showed many signs of inborn endocrine disorders, and this applied to his son Tutankhamun's famous medical symptoms.



This was apparent in Tutankhamun's bone necrosis and suggested by the presence of a walking cane and pharmacy in his resting place. (source)

A prominent theory is that they may have suffered from Antley-Bixler syndrome, a rare endocrine disorder that presents as bone and other systemic issues ([source](#)). This syndrome shows errors in mitochondrial synthesis of steroids, which is a process handled by cytochrome P450 enzymes ([source](#)). As with most endocrine disorders phosphate imbalances are present affecting NADPH transfer, directly impacting steroid hormone synthesis and endocrine function ([source](#)).

These symptoms are also part of a large-scale immune system condition called APECED – Autoimmune Polyendocrinopathy Candidiasis Ectodermal Dysplasia, which affects bone health and formation by dysregulation of calcium and phosphorous stores in the body ([source](#)). There is increased risk of bone failures which may also help explain the catastrophic bone break not healing as healthy bone should have for Tutankhamun ([source](#)). APECED is also a reasonable fit due to its implication in many

cytochrome P450 errors related to steroid synthesis (source).

To further stress the likelihood of APECED's place in the Amarna lineages we should recall that it is a candidiasis related dysplasia. It means easier infection via all exposure routes that fungi can access our systems by, allowing fungi to leverage immune response in the body to its benefit. Due to the frequency of infections and ease of inhaling or ingesting yeast spores, people with this syndrome have higher mortality and malignancy rates (source). These immune errors can affect fetal development and prenatal thymic function usually resulting in miscarriage (source).

Tutankhamun had an early death, and in his tomb were discovered two stillborn female fetuses, assumed by most to have been his daughters. His family's medical history indicates that the possibility of unrecognized autoimmune disease related to fungal pathogens must be considered (source).

Therefore, invasive fungal disease in a large, unprepared population could easily connect the decline of the 14th and 15th dynasties to the health issues of the 18th dynasty. If there were an instance of a virulent species of pathogenic fungi that swept through, the exposure could have leveraged existing genetic susceptibilities to set in and affect immune systems of later generations. ([source](#))

With systemic fungal infections still affecting the Egyptian populace to this day, the prevalence of the same occurrences in ancient Egypt should be considered ([source](#)).

Although Akhenaten and the worship of his favored deity the Aten were vilified by priests from his own cabinet, his aims may have been in the interest of his people's health. The lack of the Ankh, a representation of Isis' girdle and her gift of bread and beer, points to some change in at least the health approaches of the pharaoh.

The advancements in medicine for the commoner were also noteworthy, with medical inscriptions found on pottery and stones from the Amarna period onward ([source](#)).

Akhenaten as the purveyor of a coordinated push away from disease, and primarily ones that were part of the existing populace control, is very in line with the rest of his generally accepted impacts on the politics of his era.

He indirectly set the stage for one of the greatest world leaders in recorded history, Ramses the Great.

The end of the 18th dynasty was a tumultuous progression towards the start of the Ramessid era of the 19th dynasty. Horemheb's lack of an heir during his reign as the last pharaoh left him to appoint a trusted advisor, and his choice was a non-royal with connections to the Hyksos city Avaris ([source](#)).

Previously known in the kingdom as his vizier Paramessu, upon ascension to the throne he took the name Ramses I – “Ra has birthed him”.

The reign of Ramses I was short, at around two years, but he had a young heir who would continue the succession of the lineage. His ascended name was Seti I which translates to “Of Seth” and his favor toward this god would carry through the entirety of the 19th dynasty.

This signified another return to the influences of Upper Egypt and the Amazigh roots that had brought Seth to the region in antiquity. He unexpectedly died younger than others from his line, in his early 40s, and researchers have assumed long-term disease of the heart as the cause ([source](#)) ([source](#)).

If this is correct it points to the possibility of heart disease and its connection to fungal infections ([source](#)).

After Seti I's death, Ramses II, also known as Ramses the Great, ascended the throne to begin one of the longest reigns in Egyptian History. During his time ruling Egypt he expanded the empire considerably, and peace treaties enacted with the Hittites led to a time of peace.

Ramses married a Hittite princess named Nefertari to reinforce this alliance. Many new temples and tombs were built in his kingdom, including a great library to hold scrolls telling of his exploits. ([source](#))

Although males of the commoner populations averaged forty years of age at death, Ramses' lived over twice that, with the consensus being around ninety years. Due to this advanced age signs of progressive disease pointing to a fungal nature had time to emerge.

Atherosclerosis and vascular calcification were present in radiological examinations of several Ramessid pharaohs and Ramses was included in those affected ([source](#)). Bone degradation in the

hips and knees indicating arthritic issues or spondylitis, is most likely a symptom of undiagnosed APECED condition ([source](#)). Arterial calcifications in the head and neck were noted in examinations further supporting the position of fungal disease that appears to have plagued Ramses' line ([source](#)). Indications of dental lesions, osteitis of the mandible, and an abscess near the chin area in Ramses jaw are to be noted. In modern medicine these complications show direct relation to fungal co-infections of the oral mucosa ([source](#)).

Ramses the Great's advanced age is a clear indication of the highest available medical knowledge being applied throughout his life. Not only was he able to rule for decades, he was able to sire many heirs to the point where a majority died before they could become pharaohs themselves. The total number of children he had is impossible to determine in our era, but the accepted range is between eighty and a hundred ([source](#)).

Fertility issues have known connections to pathogenic diseases, from fungal sources in particular, so this large number of offspring reinforces the level of care provided during his life.



With multiple descriptions of his features from medical examinations indicating “Berber”, “prehistoric Mediterranean”, and “real blonde-red haired leucodermic” it is likely that his family carried the immune predispositions we are concerned with.

Red hair has long been associated with the god Seth, foreign tribes from the Red Land, and Amazigh from Siwa Oasis. Ramses' had Upper Egypt which lineage ties to the Amazigh tribes that preceded other civilizations, connecting his genetics distantly to the Taforalt and the grotto.

As to the question of whether exposure to spores of pathogenic fungi would have occurred during his lifetime, his autopsy by the French in 1976 showed there were still active fungi on the mummy. Pollen, sand, and other detritus were found – including 60 species of fungi damaging his body ([source](#)).

In modern mycology it is well known that both candida and cryptococcus are so prevalent to be considered ever-present in our environment. Human fungal pathogens have existed for millions of years, there is no preclusion that ancient Egypt and Ramses would not have been just as exposed to these fungi as we are today.

Ancient Egypt has deep connection to the concepts of death, loss of spiritual life, and chaos being a part of disease.

The god Apophis represents the legendary reference to this, with his chasing Ra and the Ennead gods throughout their history.

The mention of forsaking bread and beer in the Book of the Dead to avoid the second death, or death of the soul indicates a hidden knowledge of yeast's impacts on body and mind.



With many pharaohs worshipping Seth throughout Egyptian history, a god who was the earliest story of a protector against disease, a pattern emerges of their culture pushing back against this deadly enemy.

The greatest kingdom of its time, Egypt would have been a destination for many disparate groups of people.

Regardless of how far modern man spread in the time from Taforalt, 18,000 years ago, through the current age, it can be nearly guaranteed their bloodline passed through Egypt.

Through its many periods, the mixing of tribes resulted in shared gods and changes in worship. We will examine some legends from those areas and groups in the following sections to show that Egypt was not unique in its struggles against disease.

6.3 Sumerian Legends

The pantheon of Sumerian gods is commonly referred to as the Anunnaki, a name derived from their father Anu, god of the sky. They were associated with fertility and became the judges of the underworld after their time as gods over earth. ([source](#))



Two of the gods in particular concern us here, the brothers Enlil and Enki – their story implies the knowledge of biology and the impact of illness on humankind.

Enlil was responsible for the creation of the earth and mankind, his name translating to “Lord of the Air”. His role in Mesopotamian beliefs was very similar to that of Yahweh from the later Israelite religion.

The stories tell of his many abilities; making crops plentiful, controlling the winds, even sending a great flood to end mankind's overpopulation. He is described as having the form of a dragon and varying from benevolent to wrathful in his nature.



His banishment is an early record of a crop infestation causing disease in the Sumerian population. Disguised as the story of taking a consort, Ninlil – the grain goddess, before she was ready for marriage.

This crop failure was most likely of fungal origin, as he used the “seed of the moon” to poison the crop. The children that resulted from this were the moon god, the god of death, and the god of rivers and canals.

As a punishment he was banished to the underworld where he fathered the god of diseases and demons with his consort Ereshkigal. ([source](#))



The second god we will examine is Enki, whose appearance is also that of a dragon or serpent.

He was credited for giving knowledge to mankind, as well as being the god of semen and amniotic fluid – a rudimentary definition for biology itself.

It is quoted in legends that it was for him to decide if a body should be good or bad. ([source](#)) ([source](#))

Enki is known for creating disease itself as the story of Enki and Ninhursag explains.

After a series of infidelities with various other gods, Enki's spouse Ninhursag removes his sperm from his latest consort. She uses this to create plants in the swamps that Enki then eats, and he is struck with illnesses. His nose, mouth, throat, arm, ribs, sides are all noted in the list of pains. ([source](#))

These are healed by his wife one at a time, resulting in the birth of many lesser gods.

Enlil and Enki are attached to creation, knowledge, and biology through many stories that survived. The impacts of pathogenic fungi like candida and cryptococcus are deeply connected to the reproductive system, disruption of brain processes, and altering of genetics in both sperm and egg.

These events are shared by other religions of later eras but from a slightly different tone, indicating a shared genesis of plot and icons.

To reinforce the idea of these gods affecting mankind and nature, we move now to the story of Gilgamesh and Enkidu.

The goddess Aruru creates Enkidu out of water and clay and places him in the wilderness. He is described as having long hair and a goat-like, hairy body with no knowledge of man's ways. He is strong and aggressive, having been created to challenge Gilgamesh's strength. A beast man by all definitions. They eventually clash and Gilgamesh and Enkidu become equals, and friends, facing many trials together. ([source](#))

There is an important detail hidden in Enkidu's name. The translation of his name is "Enki made this" which seems odd considering he was created by Aruru who oversaw fertility. The implication of Enkidu having what could be taken as a series of neonatal infection issues is that Enki "chose" that Enkidu's body should be bad. Again, the impact of fungi in development arises as a potential explanation.

In one of the final stories in the legends, after Enkidu has died, Gilgamesh sets out to gain immortality. After a series of events, including a recounting of the flood myth including the building of the ark, he is told that there is a way to achieve immortality. It is a plant that removes disease and resides at the bottom of the ocean. This is related to him by a very old immortal human named Utnapishtim and Gilgamesh sets out to retrieve this plant for himself.

After obtaining it he delays taking it because he wants to test it on a human first. This plan fails because while asleep a snake comes and eats the plant, which could be taken as another serpent reference of disease keeping him from immortality.

The Sumerian legends echo similar tones regarding disease, crops, plants, and the underworld – is this a similar record to our attempts to rout out fungal disease at its core?

(source)

6.4 Abrahamic Legends

Shared lineage between the Egyptian legends and the Abrahamic legend of the Garden of Eden. Both records have a connection through their primary participants.



The story of fighting against a serpent that represented disease is one of the clearest examples. Atum serves as a top-level creator god; he matches the Abrahamic concepts of a One Above All.

He is even split into aspects of himself, like the concept of El and his many aspects that interacted with mankind in the Israelite texts ([source](#))

Ra and his place in the pantheon can be seen as a reflection of Yahweh's stewardship of the first humans, at the command of El.



We can also find similarities in the humans that were created. Geb and Nut, the parents of the primary Ennead gods, can be compared to Adam and Eve from the Hebrew texts.

Osiris the builder is Cain the builder. His sisterwife Isis matches the role of Awan. Seth and his nomadic Red Land roots echo the command from Seth's creator god to not settle and stay pastoral.

His sisterwife Nephthys is similar to Azura from the creation story. Horus the Elder, whose role changes dramatically early in the stories, fits as Abel and his early death.

And even Osiris' son Horus the Younger is a retelling of Cain's son Enoch the builder. ([source](#)) ([source](#))

Besides the Israelite record matching the Egyptian legends, similarity with Sumerian stories exist as related to the God of creation.



Enlil and the flood share nearly an exact set of events between both religions, an angered god destroying humanity.

Follow this with the story of Enki and his bringing disease into existence by seeking errant knowledge, which then passed on to the world.

This shows a common root that points to the region's floods that drove human migrations at the end of the last ice age. ([source](#))

In the Tanakh, or Old Testament, there are many warnings against yeast, and it is a symbol of sin in most mentions there. Sacrifices were barred from containing yeast, and is not to be on the grain offerings ([source](#)) or on the same altar as a blood sacrifice ([source](#)).

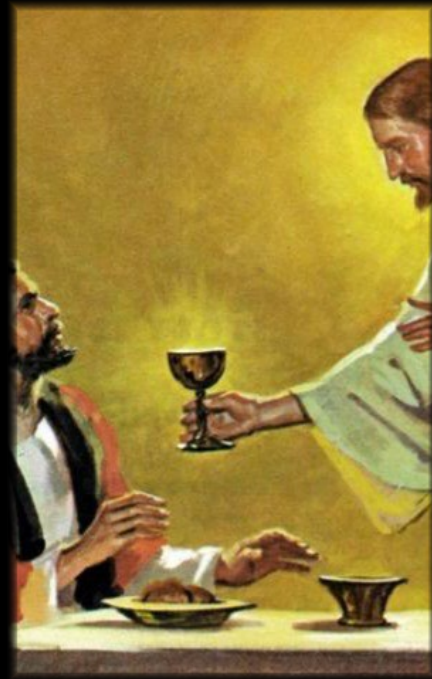
Denying yeast was seen as a way of rejecting Egyptian religion, which in nearly every kingdom relied heavily on bread and beer to control the populace. There are also periods that leavening must be cleaned from the house and many cleansing rituals and rites in the Jewish faith center around controlling exposure to yeast, or sin, by removing uncleanness.

This is further reinforced by the Christian religion in many stories of Jesus Christ that are found in the Bible's new testament. The physical aspects of what he was teaching the people of his era were an example of removing sin from the body.

Applying definition of sin as yeast and the act of removing it as healing the body, Jesus' own explanations of his message become a lesson avoiding fungi.

He was known for fasting great periods of time, for eating unleavened bread, and drinking wine.

Fasting has a natural antifungal effect, but with some strain on body systems if maintained too long ([source](#)).



Avoiding yeast by not eating leavened bread is an obvious opportunity for reduction in pathogenic exposure. And red wine is a natural source of resveratrol, which exerts change against immune function and is active against fungi very effectively ([source](#)).

We can see a set of many clear lessons from the story of Jesus Christ that point to avoiding damaging the body by avoiding sin, which he noted also corrupted the spirit, much like the Egyptian promises to forsake bread and beer to join the afterlife.

In the Islamic faith there is a ruling that pathogenic yeast, and yeast that come from alcohol or wine are impermissible. There is specific mention of brewer's and torula yeast – which are just other names for *Cryptococcus* and *Candida* species, respectively.

They share the same warnings about uncleanness, and this relates back to the idea from the other Abrahamic religions that sin and disease are closely tied.

In fact, a major component of the chemistries that work well against pathogenic fungi is taught directly by Prophet Muhammad and is mentioned in the Holy Quran.



Of nigella Sativa, the source of black seed oil, he states "The black seed can heal every disease, except death".

There are many stories of healing the sick, the blind and lame. Knowledge of medicine and it leading to helping clear the body of sin as disease is reflected in the plants that he mentioned as holy ([source](#)).

The Abrahamic religions share certain goals, and one of the easiest to see regardless of personal belief is that of clearing the body of ills.

Deeper knowledge of that which is above comes through that which is below, and clearing the connection to your own consciousness, or spirit as some say, is a worthy goal not reserved for ancient stories. With the available examples throughout the Mediterranean faiths, it presents opportunity to learn from our shared pasts.

6.5 Scandinavian Legends

The earliest culture in Scandinavia known to date are the historically nomadic Sami populations, with settlements as far north as the Arctic



Circle. The oldest habitation in the area, most likely related to Sami activity, is over 11,000 years old ([source](#)).

This matches the dates of other migrations out of the Taforalt and Mediterranean regions after the last glacial period ended.

The Sami are genetically linked to the Berber population and share a link that is traced back over 9,000 years via traits ([source](#)).



Eventually other populations from the southern areas settled regions close to the Sami, and religion and family lines were shared.

The primary god of the Scandinavian region is Odin. Known as a great healer and a human who gained enlightenment by way of removing disease from himself. This story involves a tree, a cauldron, and an eye on the surface, but should be taken as a very direct lesson against fungal disease of the body.

A god from a different age of the pantheon, who was named Mimir, had a cauldron that was kept boiling under a tree. Odin wanted a drink from this cauldron which was also called a well in some versions of the tale.

He was denied by Mimir and told that to gain enlightenment he would have to hang on the tree and sacrifice his eye.

Odin does so, hanging on the tree for 9 days and then plucking out his eye to throw in Mimir's well. Once he has done this, he is given a drink from the cauldron and gains enlightenment ([source](#)).

This tree was said to be a holy tree which in most references is accepted to be a Holy Yew. When Odin removed his eye was he seeking to gain knowledge of a disease he was plagued with, preventing his enlightenment?

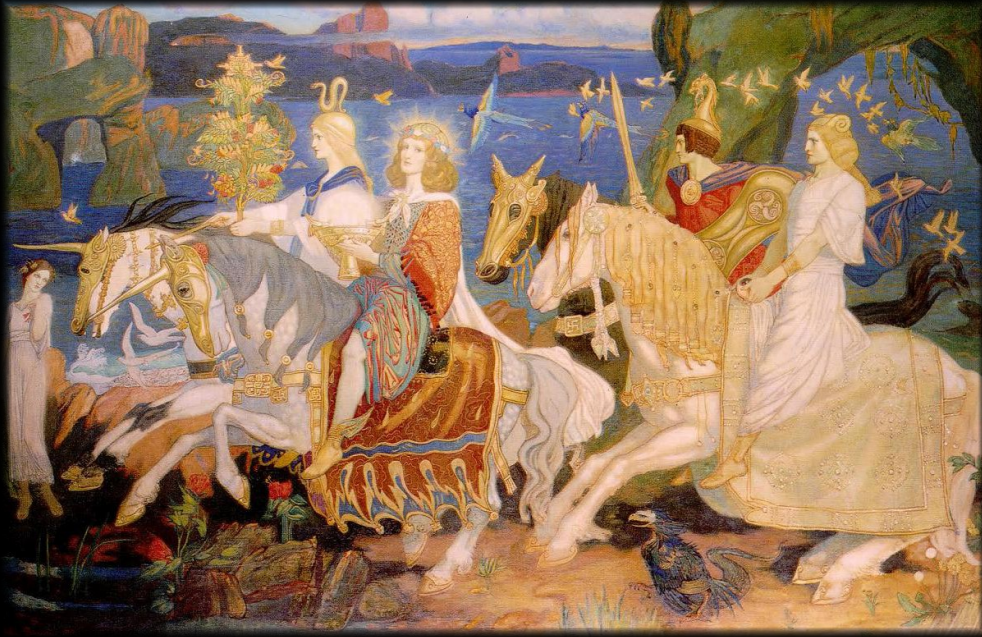
This story takes on a different tone in light of modern medicine's advancements in plant-based pharmacology, and our better understanding of disease – particularly fungal disease. The Yew that Mimir's cauldron was under contains a chemical called taxane.

Taxane is used in a drug called paclitaxel that has been used in cancer treatment and has antifungal properties. ([source](#)) ([source](#))

Odin's removing of his eye can be seen as an attempt to self-diagnose a disease of the eye that would have been affected by the Yew's antifungal and anti-cancer properties.

In this case we propose that he was suffering from orbital fungal cellulitis, a disease of the eye that presents in advanced fungal infections that have turned systemic ([source](#)).

6.6 Irish Legends



Now we will examine origins of a tribe of travelers from the north who landed in Ireland in their great ships. When they landed by crashing into the side of a mountain they set fire to their ships which burned for three days straight, blotting out the sun.

Interestingly, recent genetic data has identified a majority of European genetics in the Sami population and particularly close to Irish genetics ([source](#)).

This group was known as the Tribe of D'Anu or the Tuatha de Danann, and they were known for many great battles against the Fomorians. Fomorians were the inhabitants of the land before the D'Anu arrived, and are generally described as cave dwellers, with many aspects of their appearance and fearsome attitude lining up with long term fungal infection. The connection to the caves and disease makes an appearance again here. ([source](#))

The name Tribe of D'Anu is a reference to the Anunnaki, as it essentially means tribe of the Anu. Here we see another version of the shared creation story and legends of gods.

Their group was said to have descended from the sky in some interpretations of the story, and they had deep connections to the underworld of the region, much like the gods of the Egyptian and Sumerian pantheons.

Although not directly connected to the D'Anu lineage, Scota the progenitor of the Gaels, also was rumored to be the daughter of a great pharaoh.

When this story is told it is usually Ramses the Great who is attached to her lineage.

One of the primary gods of Tribe of D'Anu was known by the name Lugh, which translates to "light". He was known for being able to perform any task required successfully and had a spear that never missed its mark.

Here we see a reference to Seth far removed the Amazigh origins and interestingly Lugh was also known as a great healer.



He eventually routed the Fomorians and ruled over Ireland now as king of the Tribe of D'Anu. After his death he took to the underworld to protect Ireland from that which lie below ([source](#)).



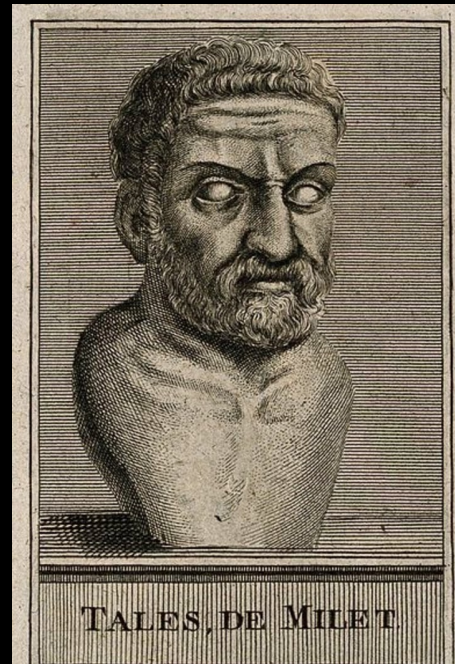
Before we move to the last group of legends, Miach should be mentioned. He was the greatest healer of the D'Anu and could heal any wound, even reversing death itself.

His father Dian Cecht became extremely jealous of this skill after Miach healed the king Nuada and killed him over it.

This caused 365 healing plants to sprout where his body had fallen. Out of further jealousy, Dian scattered those plants so no one would remember what illnesses they remedied. ([source](#))

The Tribe of D'Anu was eventually defeated by the Milesians. They gave up the surface world to go to the underworld and protect mankind from what lay below.

Their goddess Danu passed them on to live in Tir na nOg – the land of the young. The idea of the healer, disease, and immortality that has been prevalent among these legends is apparent here as well ([source](#)).



6.7 Greek Legends

Greece has a strong connection to the prior Mediterranean rooted religions, and one story from these Greek legends centers on the struggle to defeat time's hold over life. Although they had advanced their knowledge of medicine and disease greatly compared to previous civilizations, the deterioration of the body was attributed to Cronus. Taken another way, he was death itself ([source](#)).



A primary theme in Greek religion is a power struggle between Titans and the gods, resulting in the gods' victory and them ruling over the humans of Earth. Titans are explained as being the second generation of living beings, and their prime god was Cronus.

He was called several names through different eras – Gurzil by the Berber, Saturn by the Romans, and Geb by the Egyptians. But he represented time and life's destructive progress towards death.

Cronus has eaten each of Zeus' brothers and sisters, out of fear that they would unseat him. He had attempted to devour Zeus as well, but he was fooled by his wife Rhea.

He grew to adulthood and returned to force his father to return his brothers and sisters, one kick to the stomach after another. One interpretation of the legend is this was Zeus defeating time as a representation of disease.

In a later story, of Zeus and his interactions with the Titan Prometheus, we are shown how the origins of disease in humanity came from Zeus. ([source](#))

At one point Zeus withheld fire from mankind, prompting Prometheus to steal it from the heavens and give it back to man.

He does this in the form of a fennel stalk. An interesting mention since fennel is known to carry active compounds that are effective at extremely low dosages against pathogenic fungi.

(source)



For this theft Zeus gives man a new punishment in the form of Pandora, who brings all diseases that Zeus controls down upon man.

The punishment that was meted to Prometheus was to have his liver eaten by a bird every morning, and as he was immortal it would regenerate and start the cycle again (source).

In other Greek legends, silphium, pomegranate, and golden apples are all mentioned as known medicines.

They are markers of health and avoiding death and disease in Greek legends. Nearly all plants mentioned in reference to healing ability are antifungal or antimicrobial in nature ([source](#)).

We have shown the knowledge of medicine and plants being split between different civilizations, religions, and legends. It is knowledge that should be recouped and applied in the framework of modern disease if we are to progress past symptom based medicine that has become standard.

The idea of these pathogenic fungi and disease they bring having been set against mankind is unsettling and now we will outline a real world explanation for their presence in our environment.

6.8 Interplanetary Ordeal



Throughout the legends we have considered, we have seen references to fungal disease and warnings against yeast repeated. In some directions of research there is a pathogenic control system discussed as part of what past civilizations saw as gods imposing themselves on our reality. Our consciousness, the length of biological timeline, and even affecting the environment and weather have been mentioned as possible targets of effect.

Is an ultraterrestrial or interdimensional civilization the explanation for thousands of years of recorded interactions with supernatural beings and events? When first presented with this, the mind struggles with implications and rejections.

First and foremost, we need to set the correct understanding of this arm of the fungal kingdom. The classification of fungi is debatable, and one could argue they're closer to a micron-scale insect than fungi.

They can utilize multiple substrates to build the material they use in biofilms and other organic structures they synthesize.

They can also show auxotrophic mutations - meaning that they can no longer synthesize their own compounds and exclusively steal them from the host within a few mutations ([source](#)).

Also noteworthy is the use of neuron-like structures by these fungi. There are several peptides expressed in their mitochondrial processes that the human genome has no coding for. ([source](#))

One note to take here is that the primary structures in your nervous system and body involve similar membrane constructions and other functions, and electrical potentials for nerve messaging etc. These are constructed of tubulin at the smallest levels, building up to bigger structures of the cells ([source](#)).

When discussing fungi as part of disclosure we haven't confirmed life anywhere else in the system. I'll counter with the fact that we have multitudes of examples to pull from of fungi in extreme environments and thriving.

Let's consider that a fungus evolved in the environment of another planet like Saturn, it would most likely be very different than pathogenic fungi at the same evolution timescale that originated on Earth. Paul Stamets has inferred that we should expect to find fungi on every planet in the system. ([source](#))

Now let's move to the next piece. What connections could these fungi have to Saturn and its rings?



This is an origin theory on the most plausible version of panspermia within current science. And there is a stronger case for this possibility with recent astronomical research considered. There is an event estimated to have occurred approximately 165 million years ago whereby Saturn lost one of its larger moons in a gravity correction of sorts. This led to the remnants of the moon being scattered across our system ([source](#)).

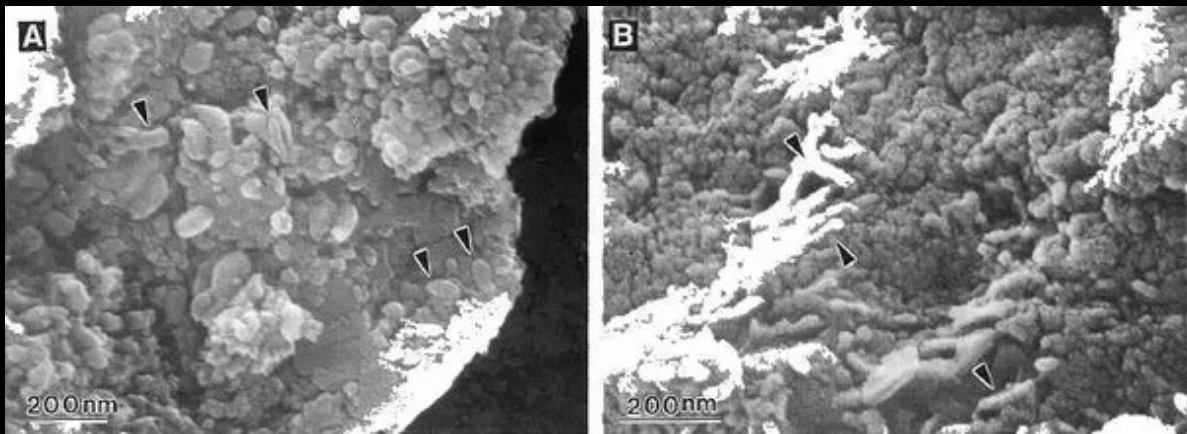
Bear in mind we have a corkscrew path through the galaxy and that we would get dragged through this debris field on a cyclical pattern.

We should assume this was a high energy event, as it was a large moon getting torn apart by its host planet. This means the path and impacts of the debris as meteors could have reached the earth easily post-event.

There is evidence of these fungi and their effect on the dinosaur population in pre-history. These infections as fungal origin leading to osteosarcomas and sepsis are well documented in the fossil record ([source](#)).

There are several referenced dates in that article, but the fungal infection deaths average around the 200-150-million-year range. Some were even as recent as 70 million years ago – and then the dinosaurs went extinct around 65 million years ago.

At this point we need a more modern example of panspermia, one that brings to light the potential impact on recent populations.



Staying with the meteor panspermia theory, a clear piece of evidence to examine is the Alan Hills 84001 meteorite. A meteorite from Mars partially comprised of very small structures that present as organic in nature.

They were embedded in the indigenous material, and they also showed resemblance to colonies and biofilms, basically hyphenations as you can see in the picture in the article below.

There are now at least three known examples of this type of meteorite, with similar microstructures embedded in them ([source](#)).

There is some debate against biogenesis related to the AH84001 meteorite and silica proving to be the material the tubes are constructed from.

However, we have examples of silica being utilized in a similar manner by life on our own planet. One is a *Saccharomyces* species that was easily changed to generating silica-based structures instead of the common calcium-based structures found in standard chitin ([source](#)).

There are glass sponge extremophiles from the deep ocean who also construct their supporting structures from silica ([source](#)).

Now we have set the stage to explain the panspermia angle for more recent global issues around pandemics. We do not suggest the current pandemic is strictly due to fungi, but we do suspect they are the substrate that is getting leveraged by other pathogens.

Fungi establish the immune failures systemically that are genetic. If virulent enough they can break extra parts of the immune system using the original errors that are there.

Now we need to look at a very strange outbreak of *Cryptococcus neoformans* that is tied to systemic brain infections. This is an event that happened over Vancouver, B.C. in 1998 where noteworthy meteors came into the atmosphere ([source](#)).

These were seen by many witnesses and there was no recovery effort due to landing in waterways. There are cases of meteorites which have been found containing liquid water sealed inside.

For our purposes we'll assume a situation where the extremely hot meteor that just entered the atmosphere has now hit far cooler water and cracked or expelled material into the waterways.

Moving ahead to the next year, still in Vancouver, B.C., and not terribly far from the projected impact sites from the last article linked. What we're looking at here though is an example of the worst outbreak of *cryptococcus neoformans* on record by that time, and still unexplained. There are some best guesses, but that's about as far as it's really gone ([source](#)).

Fitting the theory that we're working from, this is a large-scale outbreak of invasive fungal disease from *cryptococcus neoformans* – in an immunocompetent and modern population.

We present the outlay for panspermia of a pathogen that then infected a large population. But what about the controls themselves? What about the issue of an applied force that controls things at a level we wouldn't normally consider possible? The issue is more complicated than just a pathogen on some meteorites.

We have shown the multitude of ways that pathogenic fungi affect the body. Damaging organs via toxins; leading to cancer, dementia, and inflammatory diseases overall. They attack your tissue to prompt chemical responses from your body that eventually boil down to their colony stealing your ATP. They are behind degenerative diseases such as hearing loss, macular degeneration, loss of reflexes. Now let's take a closer look at the way they interrupt your nervous system and your brain chemistry. We need to cover a quick theory lesson here though.

Consciousness in the theory that we subscribe to, is an interface between our internal and external reality. Some would take that to an extension that the physical body and brain are roughly approximate to an antenna that is receiving and interpreting sensor data.

This can be drilled down to the point where you are reviewing quantum state changes in the microtubules of neurons.

That's important to the next couple posts here so keep it in mind. This theory is commonly referred to as Orch-OR theory, which has been put forward by Roger Penrose and Stuart Hameroff for decades. Both are respected in the world of physics and consciousness theory.

Orch-OR was heavily scrutinized and laughed off until more recent data has become available. Check the section on microtubules here before you go on ([source](#)). ([source](#))

The quantum state changes in extended Orch-OR theory can be taken as the "antenna" of the neural network receiving and sending data. What if something interrupts that sending and receiving, in this case with structures of its own? Microtubules that are measured changing quantum states are made up at the smallest level of a peptide called tubulin. These make up the basis of many structures in the body, but especially in neurons, glia, and other nervous system components.

Pathogenic fungi also have their own tubulins that they construct their cellular structures out of. Remember, these have been considered commensal to the body since before modern medicine started.

First, here's *Cryptococcus neoformans*, and a couple other species, being damaged when their tubulin process is interrupted ([source](#)).

This is how a cryptococcal infection looks after starting its process of pseudo hyphenation in the body. This happens in the lungs, spinal fluid, and nervous system. These are the tubulin-based structures referenced above in the antenna explanations. ([source](#))

And here's a solid explanation of how it uses these processes we've been discussing to change your nervous system to benefit itself. It even does it from inside the cells you need control of to send your own nervous system messaging around the body.

Figure 1 in the second article shows the damage to microglia very clearly ([source](#)). ([source](#))

Now you see the full picture, at least for the physical and the quantum-level structures that are being built during these infections. As to why the fungal structures mimic and interrupt the human ones is not something that I could answer now. It certainly adds up uncomfortably, and added to the panspermia theory being this obvious now it brings questions to mind that demand further explanation.

How to join the daily conversation, follow the latest news and discoveries and read direct reports from those on the protocol:

Join our new Reddit Community

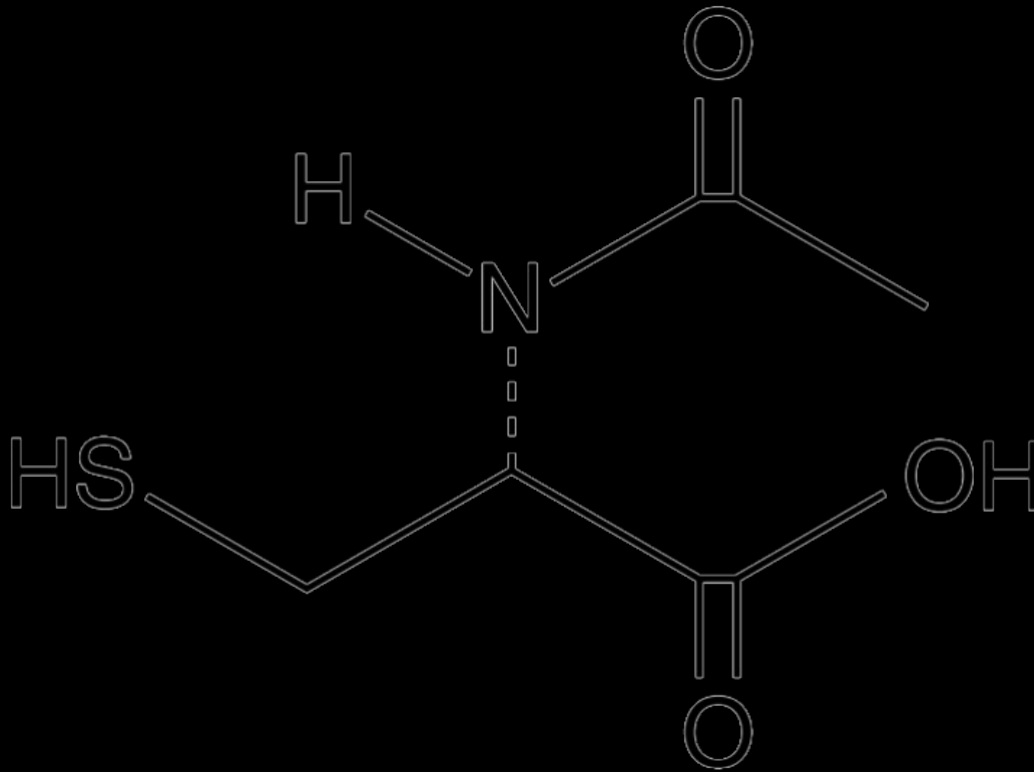
<http://www.reddit.com/r/cosmicdeathfungus>

SECTION VII: Science Behind The Protocol

The NAC Protocol

*fungus mitigation and
immunomodulatory protocol*

7.1 N-Acetyl Cysteine



N-Acetyl Cysteine (NAC) is a derivative of amino acid L-cysteine, used clinically to treat acetaminophen overdose and associated hepatic injury. It's commonly used off label in the treatment of lung conditions, including COPD and cystic fibrosis.[1] The sulfhydryl grouping confers antioxidant effect, and NAC acts as a precursor to glutathione(GSH) production [2]

7.1.1 Primary Benefit And Methodology

N-Acetyl Cysteine (NAC) features a restorative and protective role in The NAC Protocol, both by ameliorating the genomic damage caused by fungal toxins and restoring excision and chemical repair of DNA.

The specific metabolites studied were Aflatoxin, Gliotoxin, Ochratoxin and Acetaldehyde.

Aflatoxin is a secondary metabolite of *Aspergillus*, specifically *A. flavus* and *A. parasiticus*. [3] Aflatoxin B1 (AFB1) is considered hepatotoxic, teratogenic and immunotoxic in humans. [4]

Studies on a human epidermal cell line showed that concentrations of AFB1 >10 μ M are toxic to HaCaT cells and induce oxidative stress

via ROS and NO generation. [4]

Substantial damage to IMR32 neuronal cell lines was also observed, upregulating NOX2 and triggering DNA damage via downregulation of PARP1, BRCA2, and RAD51. [5]

Gliotoxin is also a toxic metabolite of *Aspergillus*, species *A. fumigatus*, and works via uptake of the disulfide bridge, which cycles between oxidized and reduced state, in turn generating ROS and destroying plasmid DNA.[6] Gliotoxin is also responsible for activating ROS-mediated apoptosis, and disrupting the integrity of the epithelial and endothelial barriers to enhance systemic fungal invasion. [6]

Ochratoxin (OTA) is produced by multiple species of *Aspergillus*. [7] It is capable of inducing oxidative DNA damage and apoptosis, starting with glutathione depletion. Animal

studies suggest that OTA-dependent oxidative stress is the precursor to cell lysing. [8] OTA concentrations were tested on a human renal proximal tubular epithelial cell line (HK-2), further confirming the role of oxidative stress in genotoxicity. [9] A study of OTA genotoxicity on porcine ovarian granulosa cells showed similar response to Aflatoxin, damaging repair related genes PARP1 and RAD51. [10]

Acetaldehyde is a metabolite of *Candida Albicans* resulting from glycolysis.[10] Both ROS and Ca²⁺ pathways are involved in Drp1 phosphorylation and mitochondrial fragmentation. Elevation of Drp1 phosphorylation was partly dependent on ROS-mediated activation of c-Jun-N-terminal kinase (JNK) and p38 mitogen-activated protein kinase (MAPK). [11] Acetaldehyde chemically induced DNA adducts follow a dose-response relationship, with mutagenicity

frequently occurring as aldehyde dehydrogenase reductions become overwhelmed. [12,13]

Guanine is the most frequently oxidized DNA base, causing transversions in DNA replication. [14] O⁶-methylguanine (O⁶mG) is a common mispairing, causing GC to AT transversion. Repair of O⁶mG to guanine is done by O⁶-alkylguanine-DNA alkyltransferase (AGT), which requires cysteine. [15] 8-Oxo-7,8-dihydroguanine (8-oxoG) is also frequently oxidized, causing GC to TA transversions. [16]

A study on mice containing Ataxia telangiectasia, which show continuous oxidative stress, showed that Thiol-containing NAC counteracts 8-OH deoxyguanosine, a marker for DNA deletions and genome instability. [17] Further, NAC was also shown to restore O⁶mG, likely by

preventing modification of essential thiol groups. [18]

By modulating the intracellular redox state, NAC can directly reduce oxidative-mediated apoptosis and DNA damage, acting as a scavenger for ROS and maintaining reduced glutathione (GSH) production in the liver. [19] Increased levels of GSH from supplementary NAC intake act as a catalyst with Glutathione S-transferases (GSTs) to reduce AFB1 by metabolizing and excreting it. [20]

NAC inhibits Gliotoxin-induced apoptosis by blocking activation of caspase-3-like proteases and also scavenging intracellular ROS. [21] With OTA it inhibits apoptosis by preventing glutathione depletion. [8]

Finally, NAC binds to Acetaldehyde acting as a scavenger, attenuating ROS and further

carcinogenic or genotoxic effect. [22]

7.1.2 Anti-Biofilm Activity

The extracellular matrix of biofilms must be considered a target when eliminating fungal infections due to antimicrobial drug resistance and persistence of infections.

Biofilm formation by fungi and bacteria contribute to various pathogenic processes including gastrointestinal diseases, systemic autoimmune diseases, and neurodegenerative diseases. [23] Until more recently it was assumed that biofilms were formed exclusively by bacteria. Various pathogenic fungi can also form biofilms, including *Candida Albicans*, *Cryptococcus neoformans*, *Cryptococcus gatti*, *Aspergillus fumigatus* and *Saccharomyces cerevisiae*. The persistence of fungal infections is greatly enhanced by it's ability to form biofilms. [24,25]

NAC is a powerful mucolytic antioxidant that efficiently inhibits and disrupts biofilms.

Pseudomonas aeruginosa is an encapsulated bacterium that frequently causes infections in humans that are difficult to treat due to quick formation of biofilm.

At a concentration of 0.5 mg/ml NAC can detach mature *P. aeruginosa* biofilms, and at 10mg/ml biofilms were completely disrupted. [26] A study on treatment of endodontic multi-species biofilms using NAC showed minimum inhibitory concentration (MIC) of 0.78–3.13 mg/ml. Multi-species culture consisted of *Actinomyces naeslundii*, *Lactobacillus salivarius*, *Streptococcus mutans*, and *Enterococcus faecalis*. [27]

A study of NAC on *Candida Albicans* biofilm adhesion and disruption showed that NAC works

effectively on mature biofilms (50-95% disruption) but less effectively on adhesion ($\geq 32.8\%$). The study also showed increased efficacy when combined with ketoconazole, an antifungal. [28]

Cryptococcus Neoformans requires capsular polysaccharide for biofilm formation, which primarily consists of Glucuronoxylomannan (GXM) and is also a constituent of cryptococcal biofilm. These biofilms are composed of 80% GXM which provides a unique challenge. [29] The trans-cell wall vesicular transport system of *Cryptococcus* is dependent on laccase [30], which is susceptible to NAC via a superoxide reaction to copper, converting it to H₂O₂ [31]. This reaction in the laccase containing vesicle and corresponding membrane disruption appear to prevent further virulence and tissue adhesion.

A study on wound biofilm formation treated with NAC showed interference with bacterial cellular redox states (NADH) and interference with ECM. Disruption of biofilms was primarily due to the molecular structure of NAC with acetyl and carboxyl groups. [32]

7.1.3 Protocol Synergy

NAC has shown a synergistic effect with many antifungals, decreasing the MIC values significantly.[33] It is believed that this is due to better penetration through membranes and biofilms due to its mucolytic effect, hydrolyzing glycoproteins and lipids via disulfide bonds and decreasing viscosity. [34]

Additional benefit is provided by correcting the imbalance between reactive oxygen species (ROS) and glutathione depletion, which offers a protective effect combined with antifungals. As an inhibitor to c-Jun N-terminal kinase (JNK) it can also reduce endothelial dysfunction, inflammation and invasion.[35]

The antifungal activity of Carvacrol induces

ROS [36] which is ameliorated by NAC, as it is commonly used in clinical settings to identify and test ROS inducers. [37]

NAC plays a supportive role, including ROS scavenging, disulfide reduction and glutathione replenishment.

A recent study on NAC further investigated the method of action and proposed an alternative function for antioxidant activity, suggesting that NAC uptake and deacetylation decelerate and prolong Cys delivery, releasing hydrogen sulfide(H₂S), a product of Cys catabolism. A further product of H₂S, sulfate sulfur species, is also proposed to contribute to NAC's beneficial effects as a cytoprotective. [38]

7.1.4 Safety Studies

NAC has a well-established safety profile, and its toxicity is rare. Elimination of NAC occurs through the renal system, with approximately 30% excreted through urine. In oral administration the most reported adverse effects are gastrointestinal symptoms such as nausea, vomiting or diarrhea. [39]

Intravenous or oral inhalation can cause more serious adverse effects, including anaphylactoid reactions of flushing, itching, and angioedema, and systemic symptoms, such as bronchospasm and hypotension. [40]

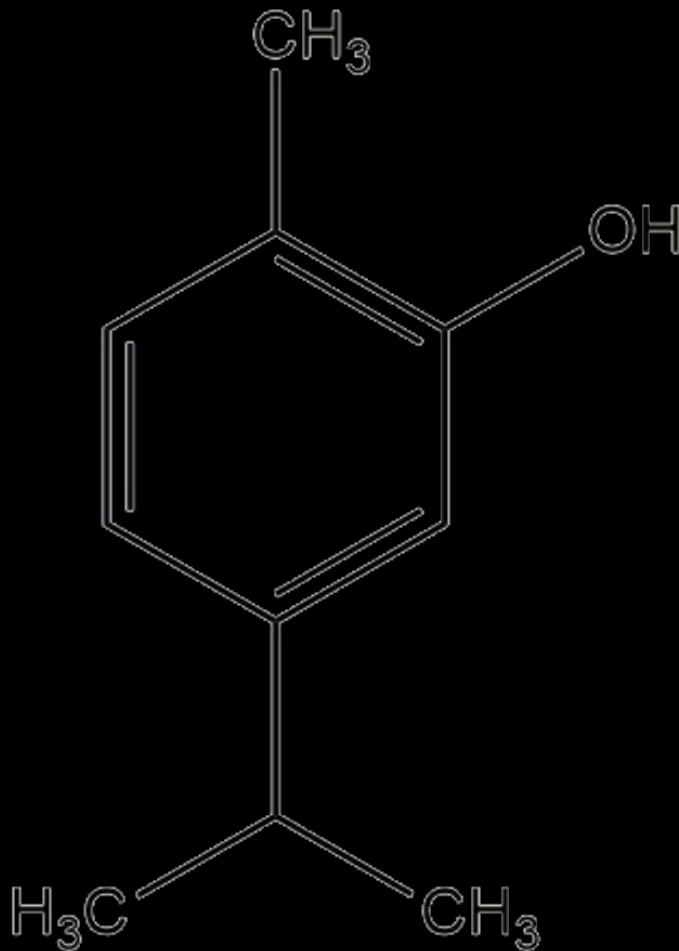
Oral dosages of 600mg and 1200mg daily showed no significant increase in adverse effects. Dosages as high as 3000mg daily resulted in minor gastrointestinal symptoms. [41]

There was 1 fatal case of anaphylactoid reaction in a 40 year old woman with chronic asthma who received intravenous treatment. [42] A potential histamine response with asthmatic patients increases susceptibility to anaphylactoid reactions, and can potentially occur via oral administration. [43] Additionally, review of all available literature found no incidence of sulfa or sulfonamide allergy reactions with administration of NAC.

Reports of NAC preventing apoptosis have been a subject of debate. As an example, NAC can be beneficial to neuronal cells by preventing apoptosis caused by trophic factor deprivation [44] but in other cases can promote tumor progression by downregulating tumor antigen P53. [45] Thymoquinone provides a counter to this with p53-mediated apoptosis [46], but more importantly is the action of

Origanum Vulgare (Oregano) as it binds to sterols on the fungal membrane, specifically ergosterols and disrupts the permeability of the membrane leading to apoptosis. The two primary active compounds, Carvacrol [47] and Thymol [48] both contribute to this process.

7.2 Oregano Oil



Origanum Vulgare (Oregano) contains two active compounds in abundance, Carvacrol and Thymol. Carvacrol is the primary constituent, a p-menthane monoterpene derived from cymene that provides many benefits to the human body. [49]

7.2.1 Primary Benefit & Methodology

Oregano serves multiple purposes as part of The NAC Protocol, including antifungal, anti-inflammatory and immunomodulatory roles.

Several additional benefits are obtained by using Oregano over other natural antifungals. One instance is dysbiosis, where imbalances in the mycobiota can influence homeostasis and disease progression. [50] Carvacrol works effectively against pathogenic bacteria and fungi [47,51,52,53] to ameliorate dysbiosis. In one study of mice with *C. difficile* infection, Oregano Oil positively altered the microbiome composition, as revealed by an increased abundance of beneficial bacteria and reduced the proportion of detrimental flora. [54]

A similar study on weaned piglets found that Oregano Oil supplemented in chow (25 mg/kg)

showed a lowered population of *Escherichia coli* in the jejunum, ileum, and colon. They found that Oregano Oil promotes intestinal barrier integrity by correcting dysbiosis and lowering inflammation by measuring mitogen-activated protein kinase (MAPK), protein kinase B (Akt), and nuclear factor κ B (NF- κ B) signaling pathways. [55]

A study on oregano oil's effect on the intestinal barrier integrity of Hyland rabbits revealed that oregano essential oil increased significantly the gene expression of junctional adhesion molecule 2 (JAM2) and JAM3 in jejunum ($p < .05$), showing a direct improvement in intestinal barrier function. [56]

A study on broiler chickens fed dietary oregano in their feed showed a reduction in *Campylobacter* spp. and *E. coli*, with a significant increase in *Lactobacillus* spp. [57] while another broiler study showed similar results, with *Lactobacilli* raised ($P < .001$) in ileum and cecum of all groups

supplemented with Oregano. [58]

These additional benefits to dysbiosis and intestinal barrier function were considered when choosing Oregano as the primary antifungal. Altered microbial composition, termed dysbiosis, has been implicated in mucosal barrier dysfunction and inflammatory responses. Restoring the epithelial barrier can potentially prevent autoimmune response and systemic infection. [59]

7.2.2 Antibiofilm Activity

The two primary compounds of *Origanum Vulgare*, Carvacrol and Thymol, [49] show both powerful inhibitory and disruptive activity against biofilms. Pathogenic fungi can make their own biofilms [60] or cohabitate in multi-species bacterial biofilms where they arrange micro colonies with distinct features. [61]

Therefore it is important to address mixed biofilms to effectively treat fungal infections. In a study on *Staphylococcus aureus* and *Candida albicans* in single and mixed cultures, Carvacrol showed a strong decrease of cell count, biomass, metabolic activity, and vitality of established 24- and 48-h biofilms. [62] A synergy was also shown between Carvacrol and Thymol in a similar study on *Candida albicans* and *Staphylococcus epidermidis*, where this combination killed highly tolerant persister cells of mono-species and

mixed-species biofilms and demonstrated less risk of resistance development. [63] Effectiveness against *Salmonella Enteritidis* biofilms also showed Carvacrol and Thymol as effective, showing inhibition of biofilm formation at sub-minimum inhibitory concentration and effectiveness against preformed biofilms. [64]

Effectiveness was also found against biofilms produced by pathogenic fungi. In a study on oral candidiasis, carvacrol and thymol significantly reduced both mature biofilm biomass and metabolic activity. [65] A study on the antibiofilm and antifungal activity against *Cryptococcus neoformans* and *Cryptococcus laurentii* compared Oregano oil (Carvacrol), Cinnamon oil (Cinnamaldehyde), Lemongrass oil (Citral), Clove oil (Eugenol), Peppermint oil (Menthol) and Thyme oil (thymol). The top 2 compounds for antibiofilm activity were Thymol and Carvacrol, respectively. [66]

Method of inhibitory action on biofilms was elucidated in a *Salmonella typhimurium* biofilm study. Proteomic analysis showed changes in the proteins DsbA (thiol: disulfide interchange protein DsbA), LuxS (S-ribosylhomocysteine lyase), DksA (RNA polymerase binding transcription factor DksA), and SODs (superoxide dismutases) A, B and C showed inhibited synthesis. [67]

7.2.3 Antifungal Activity

Origanum Vulgare (Oregano) and its primary constituents Carvacrol and Thymol have shown anti-oxidant, antiseptic, anticarcinogenic, anti-inflammatory, antidiabetic, immunomodulatory, antimicrobial, antispasmodic and antibacterial benefits. Effectiveness against a wide variety of pathogenic fungi and bacteria have been observed. [68] Carvacrol and Thymol are effective antifungal compounds that directly disrupt membrane integrity and ergosterol synthesis against *Candida* isolates. [69]

Inhibitory activity against *Candida globosa*, *Candida albicans*, *Cryptococcus laurentii*, *Trichosporon asahii*, *Kodamaea ohmeri* and *Saccharomyces* using an Oregano ethanolic extract showed a MIC value of 1.56 mg/mL. [74]

A study of Oregano against *Aspergillus flavus* and

Penicillium commune as possible alternatives for food preservation showed a MIC of 4 mg/mL. [70] Effectiveness against Aspergillus niger and Aspergillus flavus was compared between oregano (Origanum vulgare), thyme (Thymus vulgaris) and clove (Syzygium aromaticum), with Oregano showing the highest inhibitory levels. [71]

Tests of essential oils against heat resistant molds Aspergillus fumigatus and Paecilomyces variotii using citrus (Citrus sinensis L. Osbeck), laurel (Laurus nobilis L.), myrtle (Myrtus communis L.), oregano (Origanum vulgare L.), and savory (Satureja thymbra L.) showed Oregano as the most effective inhibitor of growth. [72] Another study on Aspergillus niger, Aspergillus carbonarius, and Aspergillus wentii showed inhibitory effect of 95.6%, 45.6%, and 100% at 2.5mL/100mL, respectively. [73]

Effectiveness of essential oils tested against Cryptococcus neoformans and Cryptococcus

laurentii showed Carvacrol and Thymol as most effective (16 and 32 $\mu\text{g/mL}$) as planktonic inhibitors, compared to Cinnamon oil (Cinnamaldehyde), Lemongrass oil (Citral), Clove oil (Eugenol), Peppermint oil (Menthol) and Thyme oil (thymol). [66]

7.2.4 Protocol Synergy

There are a number of likely synergies between NAC, Oregano Oil and Black Seed Oil based on available studies.

Thymoquinone (TQ) is the active compound in Black Seed Oil (*Nigella Sativa*). In a study of oral candidiasis, TQ was tested against *Candida albicans*, *Candida tropicalis*, *Candida glabrata* and *Candida krusei* strains and the synergistic antifungal activity of these strains in combination with nystatin. With TQ alone *C. albicans* was significantly inhibited at 7.5 $\mu\text{g}/\text{mL}$. Nystatin showed inhibition against *C. albicans* at 1.875 $\mu\text{g}/\text{mL}$, but when combined with TQ it lowered MIC to 0.234 $\mu\text{g}/\text{mL}$ showing a strong synergy. [74] TQ has also shown a synergistic effect against multi-drug resistant bacteria and fungi when combined with antibiotics [75] or antifungal treatments. [76] We believe there will

be a similar synergy between Carvacrol, Thymol, and Black Seed Oil compounds, decreasing inhibitory concentrations and increasing effectiveness against multi-drug resistant fungi.

Determining chemical composition of *N. sativa* shows many potential synergies. Some of the additional active compounds found by GC and GC/MS analysis were trans-anethole, p-cymene and limonene. [77] Carvacrol and p-cymene have shown synergy as compounds, reducing the minimum inhibitory concentration of Carvacrol. [78] Studies on Limonene-Carvacrol (Lim-Car) have also shown synergy in inhibitory concentrations. [79]

Synergy between NAC and antifungals has been shown previously with Fluconazole and Caspofungin. [80] Data infers that the mucolytic activity of NAC combined with the antifungal activity of Oregano provide an effective treatment against eukaryotic and sessile forms of pathogenic fungi.

7.2.5 Immune Modulation

Oregano as part of The NAC Protocol acts as an immunomodulatory compound through several mechanisms. Both Carvacrol and Thymol play a role, with Thymol suppressing expression of iNOS and COX-2, blocking the phosphorylation of I κ B α , NF- κ B p65, ERK, JNK, and p38 MAPK. [81] Carvacrol showed similar effect against pro inflammatory IL-1b, COX-1 and COX-2, while upregulating IL-10 [82, 83] and demonstrating tissue healing ability against gastric ulcers and remodeling ability in a skin disease study. OEO significantly inhibited several inflammatory biomarkers, including monocyte chemoattractant protein 1 (MCP-1), vascular cell adhesion molecule 1 (VCAM-1), intracellular cell adhesion molecule 1 (ICAM-1), interferon gamma-induced protein 10 (IP-10), interferon-inducible T-cell alpha chemoattractant (I-TAC), and monokine induced by gamma interferon (MIG) [84,85]

Oregano oil supports the immune system overall by also reducing mycotoxin burden via fungicidal action against susceptible pathogens like *Aspergillus* and *Candida*, as shown in Sec. 7.2.3.

Additional immune modulatory benefit was demonstrated in Section 7.2.1, showing how Oregano can ameliorate dysbiosis issues which disrupt homeostasis and promote disease progression.

Finally, the repair of the intestinal epithelial barrier by reducing inflammation and stabilizing dysbiosis promotes further immune enhancing effect. In all, Oregano is a powerful tool against pathogen-derived disruption of homeostasis and acute inflammatory response.

7.2.6 Safety Studies

Oregano is one of the most widely studied natural antimicrobials, with animal studies in vitro and in vivo, as well as human clinical trials, approval as a food additive by the FDA, and used extensively as a food preservative to prevent spoilage.

A Phase I clinical study on the safety of Carvacrol studied 1mg/kg and 2mg/kg groups in a human trial for one month, finding all post-treatment measured parameters were within normal range. The results of this phase I study regarding carvacrol effects on healthy subjects, showed clinical safety and tolerability. [86]

A Phase II clinical trial on the possible therapeutic effect of Carvacrol on asthmatic patients also showed no adverse outcomes. [87]

Due to strong interest by the food industry for natural options for food preservatives, animal studies are also plentiful. A study on in vivo genotoxic effects produced in rats orally exposed to 81, 256 or 810 mg carvacrol/kg body weight (bw) at 0, 24 and 45 h found that carvacrol (81-810 mg/kg bw) did not induce in vivo genotoxicity or oxidative DNA damage in any of the tissues investigated. [88]

Studies on Oregano Oil (OO) and Oregano Essential Oil (OEO) showed similar safety profiles. A study on OEO's oxidant effect (DPPH and ABTS assays) and cytotoxicity found OEO to be nontoxic. [89] A similar study on Wistar rats tested for genotoxicity over a 90 day trial, using 50, 100 and 200 mg/kg administered daily. Results obtained in the genotoxicity assays indicated lack of effect in micronucleus and standard comet assay under the conditions tested, showing no genotoxicity or oxidative damage to tissue. [90]

The evidence currently suggests that Oregano Oil is safe for more long-term use, showing no indicators of oxidative damage, genotoxicity, mitochondrial dysfunction or morphological changes in healthy cells. Oregano Oil and its active compounds do show cytotoxicity against cancerous cells, however.

In a study on Acute myeloid leukemia cell lines (AML) carvacrol and thymol showed powerful synergy, inducing tumor cell death with low toxicity on normal cells. Cell death induced by the carvacrol and thymol combination is caspase-dependent in the HL60 cell line and caspase-independent in the other cell lines tested. [91] Furthermore, a study on F1 DBA C57 Black hybrid mice studied OEO effect on Lewis carcinoma tumor engraftment. Mice were fed a low uptake dose of oregano essential oil with drinking water for three months, showing a tumor engraftment decreased by 1.8 times, its size decreased by 1.5 times, and the development of tumor was

significantly suppressed. Interestingly, activity of antioxidant enzymes was found to increase after three months of essential oil uptake (by 1.5–3 times) as compared to the control group.[92]

Oregano oil as part of The NAC Protocol is recommended at 40mg Carvacrol twice daily, or 80mg total intake per day. Safe levels were tested up to 600mg per day in the above referenced Phase I trial (2mg/kg), and up to 800mg/kg daily in animal trials showed no cytotoxic effects.

7.3 Black Seed Oil



Thymoquinone, derived naturally from *Nigella Sativa*, is a natural compound with widespread protective effects, including anti-oxidative, anti-inflammatory, immunomodulatory, anti-cancer, and anti-microbial. [93]

7.3.1 Primary Benefit & Methodology

Black seed oil is typically produced using a cold press process, extracting the active compounds from the *Nigella Sativa* seed. A GC-MS analysis revealed more than 30 active compounds, including thymoquinone, fenchone, p-cymene, trans-anethole, limonene, carvone, carvacrol, longifolene and many additional active compounds. [94]

The effect of Black seed oil (BSO) as part of The NAC Protocol is multi-purpose, serving as hepatoprotective, a potentiator for antifungal activity, a biofilm disruptor, immune modulator, and a restorative which can increase t-cell count and differentiation. [95,78,96,97,98]

A study of doxorubicin-induced cardiotoxicity in rats using 10mg/kg daily TQ in drinking water showed amelioration of induced cardiotoxicity. TQ proved to be a potent superoxide radical

scavenger, with scavenging power being as effective as superoxide dismutase against superoxide. [99] A reduction of TQ in the liver to dihydrothymoquinone is part of this antioxidant mechanism, and combined they appear to mediate this protective action [100] and also act as effective OH radical scavenging agents. [101] TQ is known as a scavenger for hydroxyl and carbon centered radicals. It also shortens ROS-facilitated stress by yielding glutathionylated-dihydrothymoquinone via non-enzymatic reaction. [102]

Antioxidant and Anti-inflammatory actions of TQ are the primary mechanisms that protect hepatocytes from injury. Myeloperoxidase activity in the liver tissue is an aggravating factor by increasing lipid peroxidation and free radical formation. [103]

The hepatoprotective role of BSO is crucial as part of The NAC Protocol.

7.3.2 Antibiofilm Activity

As part of The NAC Protocol BSO acts primarily as hepatoprotective, immune modulatory and an antifungal potentiator. Anti-Biofilm activity is also robust due to the abundant monoterpenes and sesquiterpenes. [94] Minimum biofilm inhibitory concentration (MBIC) for Thymoquinone ranges from 25-100 µg/mL, with *Candida Albicans* being highly susceptible using in vitro assays. [104] *Staphylococcus aureus* and *Staphylococcus epidermidis* minimum biofilm inhibition concentration (BIC50) was reached with 22 and 60 µg/ml, respectively. TQ also prevented cell adhesion.[105] *N.sativa* oil (BSO) showed highest microbial activity when compared to aqueous and methanolic extracts. BSO was also shown to reduce preformed biofilms of multi-drug resistant MRSA 1294, MRSA 1295 and MRSE 1297 effectively. [106]

The complexity of bioactive ingredients plays a

major role. In a study testing BS0 against *Listeria monocytogenes*, a common food contaminant, 30 ligands were tested. α -longipinene was selected based on in silico docking studies. Further in vitro studies demonstrated the anti-biofilm activity of α -longipinene. [107] The complexity of terpenes in the volatile oil likely contributes to its broad spectrum effectiveness. This complexity leads to many potential synergies. p-cymene, a major constituent of BS0 based on GC-MS analysis [94] has shown to have a synergistic effect with γ -terpinene, carvacrol and other active compounds in BS0 to increase anti-biofilm activity. [108]

Studies on the individual active compounds in BS0 show several unique anti-biofilm qualities. Limonene interferes with *C. albicans* biofilm adhesion, while trans-Anethole shows synergy with biofilm inhibition against *S. aureus*. [109,110]

7.3.3 Antifungal Activity

Nigella sativa has been studied extensively for its pharmacological benefits, but antifungal research is limited. In a study of *N. sativa* in a methanolic extract, it was found effective against 20 different strains of *Candida*. [111]

A further study on candidiasis of mice using an aqueous extract of *N. sativa* (6.6 mL/kg) showed significant inhibitory effect, only 24 hours after inoculation. A 5-fold decrease in *Candida* in kidneys, 8-fold in liver and 11-fold in spleen was observed. [112] Inhibitory effect on *Aspergillus parasiticus* (CBS 921.7) and *Aspergillus flavus* (SQU 21) was also demonstrated (1-3mg/100ml) using *N. sativa* oil (BSO) with potential metabolic effects on biosynthesis pathways for aflatoxin. [113]

Investigating the composition of *N. sativa*

volatile oil yields several active compounds, including thymoquinone, p-cymene, α -thujene, limonene, trans-anethole, fenchone and carvacrol. [94]

Thymol, thymoquinone (TQ) and thymohydroquinone (THQ), all constituents of *N. sativa*, were tested against 30 pathogens acquired from patients at a concentration of 1mg/mL. 100% inhibition was demonstrated against eight dermatophyte, five yeast and five mold isolates. TQ was found to be the strongest antifungal compound against dermatophytes and yeasts. Thymol was the most effective against molds. [114]

A study on human infection by *Fusarium solani*, a filamental fungi from the Nectriaceae family, was performed comparing Thymoquinone to Amphotericin B. A 10 day inhibition test using 1 mg/mL was performed. TQ demonstrated 100% inhibition by day 10, however Amphotericin B only inhibited 72.4% of growth in the same time range. [115]

P-cymene has shown to be effective against drug-resistant forms of *Candida*, showing synergy when combined with Thymol. [116]

Trans-anethole also has strong antifungal properties. Fennel is known as a strong antifungal, which is composed primarily of trans-anethole. [117] Trans-anethole has demonstrated effect with other drugs as it exhibits synergistic activity against several fungi. [118]

Fenchone was also shown to inhibit fungal growth (32-64 $\mu\text{g}/\text{mL}$) testing against *Candida albicans* ATCC-76645 and LM-05, *Candida tropicalis* ATCC-13803 and LM-20, and *Candida Krusei* ATCC-6258. [119] Limonene was also shown effective against *C. tropicalis* (20-40 $\mu\text{L}/\text{mL}$) using potato dextrose broth. [120]

7.3.4 Protocol Synergy

N. sativa oil (BSO) has shown synergy with both antifungals and antibacterials as a potentiator. [75,76] Active compounds in BSO have also shown direct synergy with Carvacrol, including p-cymene and limonene. [78,79] Carvacrol and Thymol, the primary active compounds in Oregano are also featured in BSO. [94] BSO has been found effective against multi-drug resistant *S. aureus*, *P. aeruginosa* and *C. albicans*, and Carvacrol performs similarly. [121,122]

Carvacrol and Thymol concentrations in BSO are in lower concentrations [94] but when adding Oregano, which contains higher levels of Carvacrol and Thymol, two distinct methods of action are present. [49] Thymoquinone has been observed to disrupt *C. albicans* cell wall synthesis, disintegrate the cytoplasm and act as a pro oxidant inducing oxidative stress via ROS

generation. [123,124] Differentially, Oregano disrupts the cell membrane by interrupting ergosterol synthesis. [69]

N-Acetylcysteine (NAC) was studied on chronic wound biofilms using mice with a 20 day maturation period. Pseudomonas, Staphylococcus, Acinetobacter, and Enterobacter were identified in the wound biofilm. NAC demonstrated effectiveness in disrupting the extracellular matrix of the biofilm, penetrating the bacterial cell membrane, inducing oxidative stress and disrupting protein synthesis. [125]

We believe the mechanism of NAC combined with the antifungal compounds in *N. sativa* and Oregano provides a specific advantage when treating fungal and bacterial infections. This combination is crucial with up to 80% of the targeted pathogens residing in biofilms. [126]

7.3.5 Immune Modulation

Nigella Sativa has been used in Middle Eastern folk medicine since biblical times, with modern research showing *N. sativa* has effect on respiratory problems, dyspepsia, metabolic syndrome, diabetes mellitus, inflammatory diseases, and various types of cancer. [127,128]

The primary purpose of Black Seed Oil (BSO) as part of The NAC Protocol is both hepatoprotective and immunomodulatory. BSO has potent antioxidant effect over several pathways, modulating NF- κ B, inhibiting iron-dependent lipid peroxidation, elevation in total thiol content and (GSH) level, radical scavenging, increasing the activity of quinone reductase, catalase, superoxide dismutase (SOD) and glutathione transferase (GST) and inhibiting COX/LOX. [129,130]

As an anti-inflammatory, Thymoquinone (TQ) inhibits JNK, ERK and P38 phosphorylation and PI3K/mTOR signaling activation. In addition, BSO was shown to decrease lipid profiles (TG, TC, LDL, VLDL), liver enzymes (AST and ALT), hs-CRP inflammatory marker, IL-6 and TNF- α . [131,132,133]

As an immunomodulator, BSO can directly improve immune response to fighting infection. A study of immunostimulation on a murine macrophage cell line showed that *N. sativa* ethanolic extract directly increased macrophage count in a cell proliferation assay, showing up to an 138% increase (25 μ g/ml). [134] An additional study using ethanolic extract on blood derived, splenic and peritoneal macrophages showed a remarkable increase in phagocytic activity. [135]

Immunostimulatory effect has also been demonstrated with peripheral blood mononuclear

cells (PBMCs), LPS-induced doubling in phagocytic activity and upregulation of p-I κ B α and p-NF- κ B p65. [136,137]

N. sativa can also enhance survivability in CD8-Positive T Cells by enhancing cytokine interferon- γ (IFN γ) production. A study on the immunomodulatory effect of BSO on rheumatoid arthritis found a positive Modulation of T lymphocytes as well. [138,139]

BSO strengthens immune response, T Cell proliferation and function, supporting the body's response against infection.

7.3.6 Safety Studies

N. sativa preparations have shown to provide gastroprotective, neuroprotective, anti-cancer, anti-diabetic, cardioprotective, bone regenerative and anti-arthritic effect. [140,141,142,143,144,145,146]

Several acute and subchronic toxicity tests have been carried out on *N. sativa*. Acute oral administration (LD50) was measured in mice (2.4 g/kg) with signs of toxicity being difficulty in respiration and hypoactivity. Acute and sub-acute toxicity was measured in Sprague Dawley rats showing LD50 of 2000mg/kg, with sub-acute dosage of 500mg/kg showing a decrease in AST enzymes. No lethality was observed in all dosage groups (100, 500, 1000 and 2000 mg/kg). Analysis of liver and kidney observed no adverse morphology and BSO was considered safe and non-toxic. [147, 148]

A Phase I human clinical trial on the safety of Thymoquinone (TQ) with Patients with Advanced Refractory Malignant Disease. 21 patients received 1 to 20 weeks treatment (median 3.7 weeks) with no side effects reported. No maximum tolerated dose was identified (75mg/day to 2600mg/day). [149]

An additional randomised, double-blinded placebo-controlled Phase I human clinical trial was carried out on 70 individuals for a period of 90 days. Blood and serum collection tests were performed. Liver function parameters included alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP). Lipid profiles included Total cholesterol (TC), Low density lipoprotein (LDL), High density lipoprotein (HDL), Very low-density lipoprotein (VLDL) and Triglycerides (TG). Renal function markers (creatinine) were also tested. Recruited participants did not exhibit any clinical signs of toxicity or adverse effects. Liver toxicity

and kidney function markers showed no change, however, lipid profiles showed significant decrease but were within safe limits. Change in TC, TG, LDL, VLDL and HDL were 12.1%, 19.66%, 16.33%, 12.76%, 8.21% and 15.27%, respectively.

[150]

Citations

1. Muhammed Ershad, Vearrier D. 2019 Mar 19. N Acetylcysteine. Nihgov. <https://www.ncbi.nlm.nih.gov/books/NBK537183/>.
2. Pedre, B., Barayeu, U., Ezeriņa, D., & Dick, T. P. (2021). The mechanism of action of N-acetylcysteine (NAC): The emerging role of H₂S and sulfane sulfur species. *Pharmacology & Therapeutics*, 228, 107916. <https://doi.org/10.1016/j.pharmthera.2021.107916>
3. GOURAMA, H., & BULLERMAN, L. B. (1995). *Aspergillus flavus* and *Aspergillus parasiticus*: Aflatoxigenic Fungi of Concern in Foods and Feeds†: A Review. *Journal of Food Protection*, 58(12), 1395–1404. <https://doi.org/10.4315/0362-028x-58.12.1395>
4. Dey, D. K., & Kang, S. C. (2020). Aflatoxin B₁ induces reactive oxygen species-dependent caspase-mediated apoptosis in normal human cells, inhibits *Allium cepa* root cell division, and triggers inflammatory response in zebrafish larvae. *Science of the Total Environment*, 737, 139704. <https://doi.org/10.1016/j.scitotenv.2020.139704>
5. Huang, B., Chen, Q., Wang, L., Gao, X., Zhu, W., Mu, P., & Deng, Y. (2020). Aflatoxin B₁ Induces Neurotoxicity through Reactive Oxygen Species Generation, DNA Damage, Apoptosis, and S-Phase Cell Cycle Arrest. *International Journal of Molecular Sciences*, 21(18), 6517. <https://doi.org/10.3390/ijms21186517>
6. Wu, T. Y., Khorramshahi, T., Taylor, L. A., Bansal, N. S., Rodriguez, B., & Rey, I. R. (2022). Prevalence of *Aspergillus*-Derived Mycotoxins (Ochratoxin, Aflatoxin, and Gliotoxin) and Their Distribution in the Urinalysis of ME/CFS Patients. *International Journal of Environmental Research and Public Health*, 19(4), 2052. <https://doi.org/10.3390/ijerph19042052>
7. Varga, J., Kevei, E., Rinyu, E., Téren, J., & Kozakiewicz, Z. (1996). Ochratoxin production by *Aspergillus* species. *Applied and Environmental*

Microbiology, 62(12), 4461–4464.
<https://doi.org/10.1128/aem.62.12.4461-4464.1996>

8. Kamp, H. G., Eisenbrand, G., Schlatter, J., Würth, K., & Janzowski, C. (2005). Ochratoxin A: induction of (oxidative) DNA damage, cytotoxicity and apoptosis in mammalian cell lines and primary cells. *Toxicology*, 206(3), 413–425.
<https://doi.org/10.1016/j.tox.2004.08.004>

9. Arbillaga, L., Azqueta, A., Ezpeleta, O., & López de Cerain, A. (2007). Oxidative DNA damage induced by Ochratoxin A in the HK-2 human kidney cell line: evidence of the relationship with cytotoxicity. *Mutagenesis*, 22(1), 35–42.
<https://doi.org/10.1093/mutage/gel049>

10. Marttila, E., Bowyer, P., Sanglard, D., Uittamo, J., Kaihovaara, P., Salaspuro, M., Richardson, M., & Rautemaa, R. (2013). Fermentative 2-carbon metabolism produces carcinogenic levels of acetaldehyde in *Candida albicans*. *Molecular Oral Microbiology*, 28(4), 281–291.
<https://doi.org/10.1111/omi.12024>

11. Yan, T., & Zhao, Y. (2020). Acetaldehyde induces phosphorylation of dynamin-related protein 1 and mitochondrial dysfunction via elevating intracellular ROS and Ca²⁺ levels. *Redox Biology*, 28, 101381.
<https://doi.org/10.1016/j.redox.2019.101381>

12. Alamil, H., Galanti, L., Heutte, N., Van Der Schueren, M., Dagher, Z., & Lechevrel, M. (2020). Genotoxicity of aldehyde mixtures: profile of exocyclic DNA-adducts as a biomarker of exposure to tobacco smoke. *Toxicology Letters*, 331, 57–64. <https://doi.org/10.1016/j.toxlet.2020.05.010>

13. Montel, R. A., Munoz-Zuluaga, C., Stiles, K. M., & Crystal, R. G. (2021). Can gene therapy be used to prevent cancer? Gene therapy for aldehyde dehydrogenase 2 deficiency. *Cancer Gene Therapy*.
<https://doi.org/10.1038/s41417-021-00399-1>

14. Kino, K., Hirao-Suzuki, M., Morikawa, M., Sakaga, A., & Miyazawa, H. (2017). Generation, repair and replication of guanine oxidation products. *Genes and Environment*, 39(1).

<https://doi.org/10.1186/s41021-017-0081-0>

15. Shukla, P. K., & Mishra, P. C. (2009). Repair of O6-methylguanine to guanine by cysteine in the absence and presence of histidine and by cysteine thiolate anion: a quantum chemical study. *Physical Chemistry Chemical Physics*, 11(37), 8191.

<https://doi.org/10.1039/b908295f>

16. Kino, K., Hirao-Suzuki, M., Morikawa, M., Sakaga, A., & Miyazawa, H. (2017). Generation, repair and replication of guanine oxidation products. *Genes and Environment*, 39(1).

<https://doi.org/10.1186/s41021-017-0081-0>

17. Reliene, R., Fischer, E., & Schiestl, R. H. (2004). Effect of N-acetyl cysteine on oxidative DNA damage and the frequency of DNA deletions in atm-deficient mice. *Cancer Research*, 64(15), 5148–5153.

<https://doi.org/10.1158/0008-5472.CAN-04-0442>

18. Göder, A., Nagel, G., Kraus, A., Dörsam, B., Seiwert, N., Kaina, B., & Fahrner, J. (2015). Lipoic acid inhibits the DNA repair protein O6-methylguanine-DNA methyltransferase (MGMT) and triggers its depletion in colorectal cancer cells with concomitant autophagy induction. *Carcinogenesis*, 36(8), 817–831.

<https://doi.org/10.1093/carcin/bgv070>

19. Liu, X., Wang, L., Cai, J., Liu, K., Liu, M., Wang, H., & Zhang, H. (2019). N-acetylcysteine alleviates H₂O₂-induced damage via regulating the redox status of intracellular antioxidants in H9c2 cells. *International Journal of Molecular Medicine*, 43(1), 199–208.

<https://doi.org/10.3892/ijmm.2018.3962>

20. Salinas, A. E., & Wong, M. G. (1999). Glutathione S-transferases--a review. *Current Medicinal Chemistry*, 6(4), 279–309.

<https://pubmed.ncbi.nlm.nih.gov/10101214/>

21. Vasdev, S., Mian, T., Longerich, L., Prabhakaran, V., & Parai, S. (1995). N-acetyl cysteine attenuates ethanol induced hypertension in rats. *Artery*, 21(6), 312–316. <https://pubmed.ncbi.nlm.nih.gov/8833231/>

22. Zhitkovich, A. (2019). N-Acetylcysteine: Antioxidant, Aldehyde Scavenger, and More. *Chemical Research in Toxicology*, 32(7), 1318–1319.
<https://doi.org/10.1021/acs.chemrestox.9b00152>
23. Miller, A. L., Bessho, S., Grando, K., & Tükel, Ç. (2021). Microbiome or Infections: Amyloid-Containing Biofilms as a Trigger for Complex Human Diseases. *Frontiers in Immunology*, 12.
<https://doi.org/10.3389/fimmu.2021.638867>
24. Sardi, J. D. C. O., Pitangui, N. D. S., Rodríguez-Arellanes, G., Taylor, M. L., Fusco-Almeida, A. M., & Mendes-Giannini, M. J. S. (2014). Highlights in pathogenic fungal biofilms. *Revista Iberoamericana de Micología*, 31(1), 22–29.
<https://doi.org/10.1016/j.riam.2013.09.014>
25. Martinez, L. R., & Fries, B. C. (2010). Fungal Biofilms: Relevance in the Setting of Human Disease. *Current Fungal Infection Reports*, 4(4), 266–275.
<https://doi.org/10.1007/s12281-010-0035-5>
26. Zhao, T., & Liu, Y. (2010). N-acetylcysteine inhibit biofilms produced by *Pseudomonas aeruginosa*. *BMC Microbiology*, 10(1), 140.
<https://doi.org/10.1186/1471-2180-10-140>
27. Moon, J.-H., Choi, Y.-S., Lee, H.-W., Heo, J. S., Chang, S. W., & Lee, J.-Y. (2016). Antibacterial effects of N-acetylcysteine against endodontic pathogens. *Journal of Microbiology (Seoul, Korea)*, 54(4), 322–329.
<https://doi.org/10.1007/s12275-016-5534-9>
28. Mahmoud Abd El-Baky, R., Mohamed Mohamed Abo El Ela, D., & Fadel Mamoud Gad, G. (2014). N-acetylcysteine Inhibits and Eradicates *Candida albicans* Biofilms. *American Journal of Infectious Diseases and Microbiology*, 2(5), 122–130.
<https://doi.org/10.12691/ajidm-2-5-5>
29. Martinez, L. R., & Casadevall, A. (2007). *Cryptococcus neoformans* Biofilm Formation Depends on Surface Support and Carbon Source and Reduces Fungal Cell Susceptibility to Heat, Cold, and UV Light. *Applied and Environmental*

Microbiology, 73(14), 4592–4601.
<https://doi.org/10.1128/aem.02506-06>

30. Rodrigues, M. L., Nimrichter, L., Oliveira, D. L., Nosanchuk, J. D., & Casadevall, A. (2008). Vesicular Trans-Cell Wall Transport in Fungi: A Mechanism for the Delivery of Virulence-Associated Macromolecules? *Lipid Insights*, 2, LPI.S1000.
<https://doi.org/10.4137/lpi.s1000>

31. Zheng, J., Lou, J. R., Zhang, X.-X., Benbrook, D. M., Hanigan, M. H., Lind, S. E., & Ding, W.-Q. (2010). N-Acetylcysteine interacts with copper to generate hydrogen peroxide and selectively induce cancer cell death. *Cancer Letters*, 298(2), 186–194. <https://doi.org/10.1016/j.canlet.2010.07.003>

32. Li, X., Kim, J., Wu, J., Ahamed, A. I., Wang, Y., & Martins-Green, M. (2020). N-Acetyl-cysteine and Mechanisms Involved in Resolution of Chronic Wound Biofilm. *Journal of Diabetes Research*, 2020, 9589507.
<https://doi.org/10.1155/2020/9589507>

33. Homa, M., Galgóczy, L., Tóth, E., Virágh, M., Chandrasekaran, M., Vágvölgyi, C., & Papp, T. (2016). In vitro susceptibility of *Scedosporium* isolates to N-acetyl-L-cysteine alone and in combination with conventional antifungal agents. *Medical Mycology*, 54(7), 776–779.
<https://doi.org/10.1093/mmy/myw029>

34. Banerjee, S., & McCormack, S. (2019). Acetylcysteine for Patients Requiring Mucous Secretion Clearance: A Review of Clinical Effectiveness and Safety. In PubMed. Canadian Agency for Drugs and Technologies in Health.
<https://pubmed.ncbi.nlm.nih.gov/31503431/>

35. Zafarullah, M., Li, W. Q., Sylvester, J., & Ahmad, M. (2003). Molecular mechanisms of N -acetylcysteine actions. *Cellular and Molecular Life Sciences (CMLS)*, 60(1), 6–20. <https://doi.org/10.1007/s000180300001>

36. Niu, C., Wang, C., Yang, Y., Chen, R., Zhang, J., Chen, H., Zhuge, Y., Li, J., Cheng, J., Xu, K., Chu, M., Ren, C., Zhang, C., & Jia, C. (2020). Carvacrol Induces *Candida albicans* Apoptosis Associated With Ca²⁺/Calcineurin

Pathway. *Frontiers in Cellular and Infection Microbiology*, 10.
<https://doi.org/10.3389/fcimb.2020.00192>

37. Halasi, M., Wang, M., Chavan, T. S., Gaponenko, V., Hay, N., & Gartel, A. L. (2013). ROS inhibitor N-acetyl-L-cysteine antagonizes the activity of proteasome inhibitors. *The Biochemical Journal*, 454(2), 201–208.
<https://doi.org/10.1042/BJ20130282>

38. Pedre, B., Barayeu, U., Ezeriņa, D., & Dick, T. P. (2021). The mechanism of action of N-acetylcysteine (NAC): The emerging role of H₂S and sulfane sulfur species. *Pharmacology & Therapeutics*, 228, 107916.
<https://doi.org/10.1016/j.pharmthera.2021.107916>

39. Tenório, M. C. dos S., Graciliano, N. G., Moura, F. A., Oliveira, A. C. M. de, & Goulart, M. O. F. (2021). N-Acetylcysteine (NAC): Impacts on Human Health. *Antioxidants*, 10(6), 967.
<https://doi.org/10.3390/antiox10060967>

40. Yarema, M., Chopra, P., Sivilotti, M. L. A., Johnson, D., Nettel-Aguirre, A., Bailey, B., Victorino, C., Gosselin, S., Purssell, R., Thompson, M., Spyker, D., & Rumack, B. (2018). Anaphylactoid Reactions to Intravenous N-Acetylcysteine during Treatment for Acetaminophen Poisoning. *Journal of Medical Toxicology*, 14(2), 120–127.
<https://doi.org/10.1007/s13181-018-0653-9>

41. Calverley, P., Rogliani, P., & Papi, A. (2020). Safety of N-Acetylcysteine at High Doses in Chronic Respiratory Diseases: A Review. *Drug Safety*, 44(3), 273–290.
<https://doi.org/10.1007/s40264-020-01026-y>

42. Appelboam, A. V. (2002). Fatal anaphylactoid reaction to N-acetylcysteine: caution in patients with asthma. *Emergency Medicine Journal*, 19(6), 594–595.
<https://doi.org/10.1136/emj.19.6.594>

43. Sandilands, E. A., & Bateman, D. N. (2009). Adverse reactions associated with acetylcysteine. *Clinical Toxicology*, 47(2), 81–88.
<https://doi.org/10.1080/15563650802665587>

44. Ferrari, G., Yan, C., & Greene, L. (1995). N-acetylcysteine (D- and L-stereoisomers) prevents apoptotic death of neuronal cells. *The Journal of Neuroscience*, 15(4), 2857–2866.
<https://doi.org/10.1523/jneurosci.15-04-02857.1995>
45. Sayin, V. I., Ibrahim, M. X., Larsson, E., Nilsson, J. A., Lindahl, P., & Bergo, M. O. (2014). Antioxidants Accelerate Lung Cancer Progression in Mice. *Science Translational Medicine*, 6(221), 221ra15–221ra15.
<https://doi.org/10.1126/scitranslmed.3007653>
46. Dastjerdi, M., Mehdiabady, E., Iranpour, F., & Bahramian, H. (2016). Effect of thymoquinone on P53 gene expression and consequence apoptosis in breast cancer cell line. *International Journal of Preventive Medicine*, 7(1), 66.
<https://doi.org/10.4103/2008-7802.180412>
47. Nóbrega, R. de O., Teixeira, A. P. de C., Oliveira, W. A. de, Lima, E. de O., & Lima, I. O. (2016). Investigation of the antifungal activity of carvacrol against strains of *Cryptococcus neoformans*. *Pharmaceutical Biology*, 54(11), 2591–2596.
<https://doi.org/10.3109/13880209.2016.1172319>
48. Ahmad, A., Khan, A., Akhtar, F., Yousuf, S., Xess, I., Khan, L. A., & Manzoor, N. (2010). Fungicidal activity of thymol and carvacrol by disrupting ergosterol biosynthesis and membrane integrity against *Candida*. *European Journal of Clinical Microbiology & Infectious Diseases*, 30(1), 41–50.
<https://doi.org/10.1007/s10096-010-1050-8>
49. PubChem. (n.d.). Carvacrol. [Pubchem.ncbi.nlm.nih.gov](http://pubchem.ncbi.nlm.nih.gov).
<https://pubchem.ncbi.nlm.nih.gov/compound/Carvacrol>
50. Iliev, I. D., & Leonardi, I. (2017). Fungal dysbiosis: immunity and interactions at mucosal barriers. *Nature Reviews. Immunology*, 17(10), 635–646.
<https://doi.org/10.1038/nri.2017.55>
51. Memar, M. Y., Raei, P., Alizadeh, N., Akbari Aghdam, M., & Kafil, H. S. (2017). Carvacrol and thymol. *Reviews in Medical Microbiology*, 28(2), 63–68.
<https://doi.org/10.1097/mrm.0000000000000100>

52. Abass Bnyan, I., Aumaima, T., Abid, H., & Obied. (n.d.). Antibacterial Activity of Carvacrol against Different Types of Bacteria. In *Journal of Natural Sciences Research*. Retrieved February 27, 2023, from <https://www.iiste.org/Journals/index.php/JNSR/article/viewFile/13191/13559>
53. Ferhout, H., Bohatier, J., Guillot, J., & Chalchat, J. C. (1999). Antifungal Activity of Selected Essential Oils, Cinnamaldehyde and Carvacrol against *Malassezia furfur* and *Candida albicans*. *Journal of Essential Oil Research*, 11(1), 119–129. <https://doi.org/10.1080/10412905.1999.9701086>
54. Mooyottu, S., Flock, G., Upadhyay, A., Upadhyaya, I., Maas, K., & Venkitanarayanan, K. (2017). Protective Effect of Carvacrol against Gut Dysbiosis and *Clostridium difficile* Associated Disease in a Mouse Model. *Frontiers in Microbiology*, 8. <https://doi.org/10.3389/fmicb.2017.00625>
55. Zou, Y., Xiang, Q., Wang, J., Peng, J., & Wei, H. (2016). Oregano Essential Oil Improves Intestinal Morphology and Expression of Tight Junction Proteins Associated with Modulation of Selected Intestinal Bacteria and Immune Status in a Pig Model. *BioMed Research International*, 2016, 1–11. <https://doi.org/10.1155/2016/5436738>
56. Li, C., Niu, J., Liu, Y., Li, F., & Liu, L. (2021). The effects of oregano essential oil on production performance and intestinal barrier function in growing Hyla rabbits. *Italian Journal of Animal Science*, 20(1), 2165–2173. <https://doi.org/10.1080/1828051x.2021.2005471>
57. Kelly, C., Gundogdu, O., Pircalabioru, G., Cean, A., Scates, P., Linton, M., Pinkerton, L., Magowan, E., Stef, L., Simiz, E., Pet, I., Stewart, S., Stabler, R., Wren, B., Dorrell, N., & Corcionivoschi, N. (2017). The In Vitro and In Vivo Effect of Carvacrol in Preventing *Campylobacter* Infection, Colonization and in Improving Productivity of Chicken Broilers. *Foodborne Pathogens and Disease*, 14(6), 341–349. <https://doi.org/10.1089/fpd.2016.2265>
58. Franciosini, M. P., Casagrande-Proietti, P., Forte, C., Beghelli, D., Acuti,

G., Zanichelli, D., dal Bosco, A., Castellini, C., & Trabalza-Marinucci, M. (2015). Effects of oregano (*Origanum vulgare*L.) and rosemary (*Rosmarinus officinalis* L.)aqueous extracts on broiler performance, immune function and intestinal microbial population. *Journal of Applied Animal Research*, 44(1), 474–479. <https://doi.org/10.1080/09712119.2015.1091322>

59. Kinashi, Y., & Hase, K. (2021). Partners in Leaky Gut Syndrome: Intestinal Dysbiosis and Autoimmunity. *Frontiers in Immunology*, 12. <https://doi.org/10.3389/fimmu.2021.673708>

60. de Barros, P. P., Rossoni, R. D., de Souza, C. M., Scorzoni, L., Fenley, J. D. C., & Junqueira, J. C. (2020). Candida Biofilms: An Update on Developmental Mechanisms and Therapeutic Challenges. *Mycopathologia*, 185(3), 415–424. <https://doi.org/10.1007/s11046-020-00445-w>

61. Elias, S., & Banin, E. (2012). Multi-species biofilms: Living with friendly neighbors. *FEMS Microbiology Reviews*, 36(5), 990–1004. <https://doi.org/10.1111/j.1574-6976.2012.00325.x>

62. Scaffaro, R., Lopresti, F., D' Arrigo, M., Marino, A., & Nostro, A. (2018). Efficacy of poly(lactic acid)/carvacrol electrospun membranes against *Staphylococcus aureus* and *Candida albicans* in single and mixed cultures. *Applied Microbiology and Biotechnology*, 102(9), 4171–4181. <https://doi.org/10.1007/s00253-018-8879-7>

63. Swetha, T. K., Vikraman, A., Nithya, C., Hari Prasath, N., & Pandian, S. K. (2021). Synergistic antimicrobial combination of carvacrol and thymol impairs single and mixed-species biofilms of *Candida albicans* and *Staphylococcus epidermidis*. *Biofouling*, 1–16. <https://doi.org/10.1080/08927014.2020.1869949>

64. Čabarkapa, I., Čolović, R., Đuragić, O., Popović, S., Kokić, B., Milanov, D., & Pezo, L. (2019). Anti-biofilm activities of essential oils rich in carvacrol and thymol against *Salmonella* *Enteritidis*. *Biofouling*, 35(3), 361–375. <https://doi.org/10.1080/08927014.2019.1610169>

65. Miranda-Cadena, K., Marcos-Arias, C., Mateo, E., Aguirre-Urizar, J. M.,

Quindós, G., & Eraso, E. (2021). In vitro activities of carvacrol, cinnamaldehyde and thymol against *Candida* biofilms. *Biomedicine & Pharmacotherapy*, 143, 112218. <https://doi.org/10.1016/j.biopha.2021.112218>

66. Kumari, P., Mishra, R., Arora, N., Chatrath, A., Gangwar, R., Roy, P., & Prasad, R. (2017). Antifungal and Anti-Biofilm Activity of Essential Oil Active Components against *Cryptococcus neoformans* and *Cryptococcus laurentii*. *Frontiers in Microbiology*, 8. <https://doi.org/10.3389/fmicb.2017.02161>

67. Alves Coelho Trevisan, D., Aline Zanetti Campanerut-Sá, P., da Silva, A. F., Farias Pereira Batista, A., Seixas, F. A. V., Peralta, R. M., de Sá-Nakanishi, A. B., de Abreu Filho, B. A., Machinski Junior, M., & Graton Mikcha, J. M. (2020). Action of carvacrol in *Salmonella Typhimurium* biofilm: A proteomic study. *Journal of Applied Biomedicine*, 18(4), 106–114. <https://doi.org/10.32725/jab.2020.014>

68. Imran, M., Aslam, M., Alsaqaby, S. A., Saeed, F., Ahmad, I., Afzaal, M., Arshad, M. U., Abdelgawad, M. A., El - Ghorab, A. H., Khames, A., Shariati, M. A., Ahmad, A., Hussain, M., Imran, A., & Islam, S. (2022). Therapeutic application of carvacrol: A comprehensive review. *Food Science & Nutrition*, 10(11), 3544–3561. <https://doi.org/10.1002/fsn3.2994>

69. Imran, M., Aslam, M., Alsaqaby, S. A., Saeed, F., Ahmad, I., Afzaal, M., Arshad, M. U., Abdelgawad, M. A., El - Ghorab, A. H., Khames, A., Shariati, M. A., Ahmad, A., Hussain, M., Imran, A., & Islam, S. (2022). Therapeutic application of carvacrol: A comprehensive review. *Food Science & Nutrition*, 10(11), 3544–3561. <https://doi.org/10.1002/fsn3.2994>

70. ALMEIDA, P. de, BLANCO-PASCUAL, N., ROSOLEN, D., CISILOTTO, J., CRECZYNSKI-PASA, T., & LAURINDO, J. (2022). Antioxidant and antifungal properties of essential oils of oregano (*Origanum vulgare*) and mint (*Mentha arvensis*) against *Aspergillus flavus* and *Penicillium commune* for use in food preservation. *Food Science and Technology*, 42. <https://doi.org/10.1590/fst.64921>

71. VIUDA-MARTOS, M., RUIZ-NAVAJAS, Y., FERNÁNDEZ-LÓPEZ, J., & PÉREZ-ÁLVAREZ, J.

A. (2007). ANTIFUNGAL ACTIVITIES OF THYME, CLOVE AND OREGANO ESSENTIAL OILS. *Journal of Food Safety*, 27(1).

<https://doi.org/10.1111/j.1745-4565.2007.00063.x>

72. Gumus, T., Demirci, A. S., Sagdic, O., & Arici, M. (2010). Inhibition of heat resistant molds: *Aspergillus fumigatus* and *Paecilomyces variotii* by some plant essential oils. *Food Science and Biotechnology*, 19(5), 1241–1244.

<https://doi.org/10.1007/s10068-010-0177-9>

73. Kocić-Tanackov, S., Dimić, G., Tanackov, I., Pejin, D., Mojović, L., & Pejin, J. (2012). The inhibitory effect of oregano extract on the growth of *Aspergillus* spp. and on sterigmatocystin biosynthesis. *LWT*, 49(1), 14–20.

<https://doi.org/10.1016/j.lwt.2012.04.013>

74. Blank, D. E., Alves, G. H., Nascente, P. D. S., Freitag, R. A., & Cleff, M. B. (2020). Bioactive Compounds and Antifungal Activities of Extracts of Lamiaceae Species. *Journal of Agricultural Chemistry and Environment*, 9(3), 85–96.

<https://doi.org/10.4236/jacen.2020.93008>

74. Özdal Zincir, Ö., Özdal, U., Ünlü, Ö., Demirci, M., Katiboğlu, A. B., Egil, E., & Altan Şallı, G. (2021). Synergistic effect of thymoquinone and nystatin in the treatment of oral candidiasis; an in vitro study. *Odontology*.

<https://doi.org/10.1007/s10266-021-00667-4>

75. Dera, A. A., Ahmad, I., Rajagopalan, P., Shahrani, M. A., Saif, A., Alshahrani, M. Y., Alraey, Y., Alamri, A. M., Alasmari, S., Makkawi, M., Alkhatami, A. G., Zaman, G., Hakami, A., Alhefzi, R., & Alfihli, M. A. (2021). Synergistic efficacies of thymoquinone and standard antibiotics against multi-drug resistant isolates. *Saudi Medical Journal*, 42(2), 196–204.

<https://doi.org/10.15537/smj.2021.2.25706>

76. Khan, M. A., Aljarbou, A. N., Khan, A., & Younus, H. (2015). Liposomal thymoquinone effectively combats fluconazole-resistant *Candida albicans* in a murine model. *International Journal of Biological Macromolecules*, 76, 203–208.

<https://doi.org/10.1016/j.ijbiomac.2015.02.015>

77. Nickavar, B., Mojab, F., Javidnia, K., & Amoli, M. A. R. (2003). Chemical

Composition of the Fixed and Volatile Oils of *Nigella sativa* L. from Iran. *Zeitschrift Für Naturforschung C*, 58(9-10), 629–631.

<https://doi.org/10.1515/znc-2003-9-1004>

78. Kiskó, G., & Roller, S. (2005). Carvacrol and p-cymene inactivate *Escherichia coli* O157:H7 in apple juice. *BMC Microbiology*, 5(1).

<https://doi.org/10.1186/1471-2180-5-36>

79. Carvalho, R. de C. V. de, Sousa, V. C. de, Santos, L. P., Santos, I. L. dos, Diniz, R. C., Rodrigues, R. R. L., Medeiros, M. das G. F. de, Rodrigues, K. A. da F., Alves, M. M. de M., Arcanjo, D. D. R., & Carvalho, F. A. de A. (2021). Limonene-carvacrol: A combination of monoterpenes with enhanced antileishmanial activity. *Toxicology in Vitro*, 74, 105158.

<https://doi.org/10.1016/j.tiv.2021.105158>

80. Nunes, T. S. B. S., Rosa, L. M., Vega-Chacón, Y., & Mima, E. G. de O. (2020). Fungistatic Action of N-Acetylcysteine on *Candida albicans* Biofilms and Its Interaction with Antifungal Agents. *Microorganisms*, 8(7), 980.

<https://doi.org/10.3390/microorganisms8070980>

81. Liang, D., Li, F., Fu, Y., Cao, Y., Song, X., Wang, T., Wang, W., Guo, M., Zhou, E., Li, D., Yang, Z., & Zhang, N. (2013). Thymol Inhibits LPS-Stimulated Inflammatory Response via Down-Regulation of NF- κ B and MAPK Signaling Pathways in Mouse Mammary Epithelial Cells. *Inflammation*, 37(1), 214–222.

<https://doi.org/10.1007/s10753-013-9732-x>

82. Lima, M. da S., Quintans-Júnior, L. J., de Santana, W. A., Martins Kaneto, C., Pereira Soares, M. B., & Villarreal, C. F. (2013). Anti-inflammatory effects of carvacrol: Evidence for a key role of interleukin-10. *European Journal of Pharmacology*, 699(1-3), 112–117. <https://doi.org/10.1016/j.ejphar.2012.11.040>

83. Landa, P., Kokoska, L., Pribylova, M., Vanek, T., & Marsik, P. (2009). In vitro anti-inflammatory activity of carvacrol: Inhibitory effect on COX-2 catalyzed prostaglandin E2 biosynthesis. *Archives of Pharmacal Research*, 32(1), 75–78.

<https://doi.org/10.1007/s12272-009-1120-6>

84. Silva, F. V., Guimarães, A. G., Silva, E. R. S., Sousa-Neto, B. P., Machado, F. D. F., Quintans-Júnior, L. J., Arcanjo, D. D. R., Oliveira, F. A., & Oliveira, R. C. M. (2012). Anti-Inflammatory and Anti-Ulcer Activities of Carvacrol, a Monoterpene Present in the Essential Oil of Oregano. *Journal of Medicinal Food*, 15(11), 984–991.
<https://doi.org/10.1089/jmf.2012.0102>
85. Han, X., & Parker, T. L. (2017). Anti-inflammatory, tissue remodeling, immunomodulatory, and anticancer activities of oregano (*Origanum vulgare*) essential oil in a human skin disease model. *Biochimie Open*, 4, 73–77.
<https://doi.org/10.1016/j.biopen.2017.02.005>
86. Ghorani, V., Alavinezhad, A., Rajabi, O., Mohammadpour, A. H., & Boskabady, M. H. (2021). Safety and tolerability of carvacrol in healthy subjects: a phase I clinical study. *Drug and Chemical Toxicology*, 44(2), 177–189.
<https://doi.org/10.1080/01480545.2018.1538233>
87. Alavinezhad, A., Khazdair, M. R., & Boskabady, M. H. (2017). Possible therapeutic effect of carvacrol on asthmatic patients: A randomized, double blind, placebo-controlled, Phase II clinical trial. *Phytotherapy Research*, 32(1), 151–159.
<https://doi.org/10.1002/ptr.5967>
88. Llana-Ruiz-Cabello, M., Maisanaba, S., Puerto, M., Prieto, A. I., Pichardo, S., Moyano, R., González-Pérez, J. A., & Cameán, A. M. (2016). Genotoxicity evaluation of carvacrol in rats using a combined micronucleus and comet assay. *Food and Chemical Toxicology: An International Journal Published for the British Industrial Biological Research Association*, 98(Pt B), 240–250.
<https://doi.org/10.1016/j.fct.2016.11.005>
89. Babili, F. E., Bouajila, J., Souchard, J. P., Bertrand, C., Bellvert, F., Fouraste, I., Moulis, C., & Valentin, A. (2011). Oregano: Chemical Analysis and Evaluation of Its Antimalarial, Antioxidant, and Cytotoxic Activities. *Journal of Food Science*, 76(3), C512–C518.
<https://doi.org/10.1111/j.1750-3841.2011.02109.x>
90. Llana-Ruiz-Cabello, M., Puerto, M., Maisanaba, S., Guzmán-Guillén, R.,

Pichardo, S., & Cameán, A. M. (2018). Use of micronucleus and comet assay to evaluate evaluate the genotoxicity of oregano essential oil (*Origanum vulgare* L. Virens) in rats orally exposed for 90 days.. *Journal of Toxicology and Environmental Health, Part A*, 81(12), 525–533.

<https://doi.org/10.1080/15287394.2018.1447522>

91. Bouhtit, F., Najar, M., Moussa Agha, D., Melki, R., Najimi, M., Sadki, K., Boukhatem, N., Bron, D., Meuleman, N., Hamal, A., Lagneaux, L., Lewalle, P., & Merimi, M. (2021). New Anti-Leukemic Effect of Carvacrol and Thymol Combination through Synergistic Induction of Different Cell Death Pathways. *Molecules*, 26(2), 410.

<https://doi.org/10.3390/molecules26020410>

92. Misharina, T. A., Burlakova, E. B., Fatkullina, L. D., Alinkina, E. S., Vorob'eva, A. K., Medvedeva, I. B., Erokhin, V. N., Semenov, V. A., Nagler, L. G., & Kozachenko, A. I. (2013). Effect of oregano essential oil on the engraftment and development of Lewis carcinoma in F1 DBA C57 black hybrid mice. *Applied Biochemistry and Microbiology*, 49(4), 432–436.

<https://doi.org/10.1134/s0003683813040091>

93. Hosseinzadeh, H., Tavakkoli, A., Mahdian, V., & Razavi, B. M. (2017). Review on Clinical Trials of Black Seed (*Nigella sativa*) and Its Active Constituent, Thymoquinone. *Journal of Pharmacopuncture*, 20(3), 179–193.

<https://doi.org/10.3831/kpi.2017.20.021>

94. Gerige, S. J., Gerige, M. K. Y., Rao, M., & Ramanjaneyulu. (2009). GC-MS analysis of *Nigella sativa* seeds and antimicrobial activity of its volatile oil. *Brazilian Archives of Biology and Technology*, 52(5), 1189–1192.

<https://doi.org/10.1590/s1516-89132009000500016>

95. Ebuehi, O. A. T., Olowojaiye, A. A., Erukainure, O. L., & Ajagun - Ogunleye, O. M. (2019). *Nigella sativa* (black seed) oil ameliorates CCl₄ - induced hepatotoxicity and mediates neurotransmitter levels in male Sprague Dawley albino rats. *Journal of Food Biochemistry*.

<https://doi.org/10.1111/jfbc.13108>

96. Younus, H. (Ed.). (2018). *Molecular and Therapeutic actions of Thymoquinone*.

Springer Singapore.

<https://doi.org/10.1007/978-981-10-8800-1>

97. Ojueromi, O. O., Oboh, G., & Ademosun, A. O. (2022). Effect of black seeds (*Nigella sativa*) on inflammatory and immunomodulatory markers in *Plasmodium berghei* - infected mice. *Journal of Food Biochemistry*, 46(11).

<https://doi.org/10.1111/jfbc.14300>

98. Badr, G., Alwasel, S., Ebaid, H., Mohany, M., & Alhazza, I. (2011). Perinatal supplementation with thymoquinone improves diabetic complications and T cell immune responses in rat offspring. *Cellular Immunology*, 267(2), 133–140.

<https://doi.org/10.1016/j.cellimm.2011.01.002>

99. NAGI, M. N., & MANSOUR, M. A. (2000). PROTECTIVE EFFECT OF THYMOQUINONE AGAINST DOXORUBICIN-INDUCED CARDIOTOXICITY IN RATS: A POSSIBLE MECHANISM OF PROTECTION. *Pharmacological Research*, 41(3), 283–289.

<https://doi.org/10.1006/phrs.1999.0585>

100. Nagi, M., Alam, K., Badary, O., Al-Shabanah, O., Al-Sawaf, H., & Al-Bekairi, A. (1999). Thymoquinone protects against carbon tetrachloride hepatotoxicity in mice via an antioxidant mechanism. *IUBMB Life*, 47(1), 153–159.

<https://doi.org/10.1080/15216549900201153>

101. M, B., & F, B. (2000, August 1). Antioxidant Activity of *Nigella Sativa* Essential Oil. *Phytotherapy Research* : PTR.

<https://pubmed.ncbi.nlm.nih.gov/10925395/>

102. Armutcu, F., Akyol, S., & Akyol, O. (2018). The interaction of glutathione and thymoquinone and their antioxidant properties. *Electronic Journal of General Medicine*, 15(4).

<https://doi.org/10.29333/ejgm/89493>

103. Badary, O. A., Abdel-Naim, A. B., Abdel-Wahab, M. H., & Hamada, F. M. A. (2000). The influence of thymoquinone on doxorubicin-induced hyperlipidemic nephropathy in rats. *Toxicology*, 143(3), 219–226.

[https://doi.org/10.1016/s0300-483x\(99\)00179-1](https://doi.org/10.1016/s0300-483x(99)00179-1)

104. Qureshi, K. A., Imtiaz, M., Parvez, A., Rai, P. K., Jaremko, M., Emwas, A.-H., Bholay, A. D., & Fatmi, M. Q. (2022). In Vitro and In Silico Approaches for the Evaluation of Antimicrobial Activity, Time-Kill Kinetics, and Anti-Biofilm Potential of Thymoquinone (2-Methyl-5-propan-2-ylcyclohexa-2,5-diene-1,4-dione) against Selected Human Pathogens. *Antibiotics*, 11(1), 79.
<https://doi.org/10.3390/antibiotics11010079>

105. Chaieb, K., Kouidhi, B., Jrah, H., Mahdouani, K., & Bakhrouf, A. (2011). Antibacterial activity of Thymoquinone, an active principle of *Nigella sativa* and its potency to prevent bacterial biofilm formation. *BMC Complementary and Alternative Medicine*, 11(1).
<https://doi.org/10.1186/1472-6882-11-29>

106. Saleh, F. A., El-Darra, N., Raafat, K., & Ghazzawi, I. E. (2017). Phytochemical Analysis of *Nigella sativa* L. Utilizing GC-MS Exploring its Antimicrobial Effects against Multidrug-Resistant Bacteria. *Pharmacognosy Journal*, 10(1), 99–105.
<https://doi.org/10.5530/pj.2018.1.18>

107. Vanajothi, R., Bhavaniramy, S., Vijayakumar, R., Alothaim, A. S., Alqurashi, Y. E., Vishnupriya, S., Vaseeharan, B., & Umadevi, M. (2022). In silico and In vitro Analysis of *Nigella sativa* Bioactives Against Chorismate Synthase of *Listeria monocytogenes*: a Target Protein for Biofilm Inhibition. *Applied Biochemistry and Biotechnology*, 195(1), 519–533.
<https://doi.org/10.1007/s12010-022-04157-3>

108. Miladi, H., Zmantar, T., Kouidhi, B., Al Qurashi, Y. M. A., Bakhrouf, A., Chaabouni, Y., Mahdouani, K., & Chaieb, K. (2017). Synergistic effect of eugenol, carvacrol, thymol, p-cymene and γ -terpinene on inhibition of drug resistance and biofilm formation of oral bacteria. *Microbial Pathogenesis*, 112, 156–163.
<https://doi.org/10.1016/j.micpath.2017.09.057>

109. OUP accepted manuscript. (2017). *Medical Mycology*.
<https://doi.org/10.1093/mmy/myx074>

110. Kwiatkowski, P., Grygorcewicz, B., Pruss, A., Wojciuk, B., Dołęgowska, B., Giedrys-Kalemba, S., Sienkiewicz, M., & Wojciechowska-Koszko, I. (2019). The

Effect of Subinhibitory Concentrations of trans-Anethole on Antibacterial and Antibiofilm Activity of Mupirocin Against Mupirocin-Resistant *Staphylococcus aureus* Strains. *Microbial Drug Resistance*, 25(10), 1424–1429.

<https://doi.org/10.1089/mdr.2019.0101>

111. Bită, A., Rosu, A. F., Calina, D., Rosu, L., Zlatian, O., Dindere, C., & Simionescu, A. (2012). An alternative treatment for *Candida* infections with *Nigella sativa* extracts. *European Journal of Hospital Pharmacy*, 19(2), 162.2-162.

<https://doi.org/10.1136/ejhpharm-2012-000074.203>

112. Khan, M. A. U., Ashfaq, M. K., Zuberi, H. S., Mahmood, M. S., & Gilani, A. H. (2003). The in vivo antifungal activity of the aqueous extract from *Nigella sativa* seeds. *Phytotherapy Research*, 17(2), 183–186.

<https://doi.org/10.1002/ptr.1146>

113. El-Nagerabi, S. A. F., Al-Bahry, S. N., Elshafie, A. E., & AlHilali, S. (2012). Effect of *Hibiscus sabdariffa* extract and *Nigella sativa* oil on the growth and aflatoxin B1 production of *Aspergillus flavus* and *Aspergillus parasiticus* strains. *Food Control*, 25(1), 59–63.

<https://doi.org/10.1016/j.foodcont.2011.09.033>

114. Taha, M., Azeiz, A., & Saudi, W. (2010). Antifungal effect of thymol, thymoquinone and thymohydroquinone against yeasts, dermatophytes and non-dermatophyte molds isolated from skin and nails fungal infections. *Egyptian Journal of Biochemistry and Molecular Biology*, 28(2).

<https://doi.org/10.4314/ejbmb.v28i2.60802>

115. Randhawa, M. A. (2007). Comparison of Antifungal Activity of Thymoquinone and Amphotericin B Against *Fusarium solani* in-vitro. www.academia.edu.

https://www.academia.edu/63461871/Comparison_of_Antifungal_Activity_of_Thymoquinone_and_Amphotericin_B_Against_Fusarium_solani_in_vitro

116. Chen, F., Guo, Y., Kang, J., Yang, X., Zhao, Z., Liu, S., Ma, Y., Gao, W., & Luo, D. (2020). Insight into the essential oil isolation from *Foeniculum vulgare* Mill. fruits using double-condensed microwave-assisted hydrodistillation and evaluation of its antioxidant, antifungal and cytotoxic activity. *Industrial Crops and Products*, 144, 112052.

<https://doi.org/10.1016/j.indcrop.2019.112052>

117. Tsukuda, Y., Mizuhara, N., Usuki, Y., Yamaguchi, Y., Ogita, A., Tanaka, T., & Fujita, K. (2021). Structure–activity relationships of antifungal phenylpropanoid derivatives and their synergy with n -dodecanol and fluconazole. *Letters in Applied Microbiology*, 74(3), 377–384.

<https://doi.org/10.1111/lam.13613>

118. Chen, F., Guo, Y., Kang, J., Yang, X., Zhao, Z., Liu, S., Ma, Y., Gao, W., & Luo, D. (2020). Insight into the essential oil isolation from *Foeniculum vulgare* Mill. fruits using double-condensed microwave-assisted hydrodistillation and evaluation of its antioxidant, antifungal and cytotoxic activity. *Industrial Crops and Products*, 144, 112052.

<https://doi.org/10.1016/j.indcrop.2019.112052>

119. Pessoa, M. L. de S., Silva, L. M. O., Araruna, M. E. C., Serafim, C. A. de L., Júnior, E. B. A., Silva, A. O., Pessoa, M. M. B., Neto, H. D., Lima, E. de O., & Batista, L. M. (2020). Antifungal activity and antidiarrheal activity via antimotility mechanisms of (-)-fenchone in experimental models. *World Journal of Gastroenterology*, 26(43), 6795–6809.

<https://doi.org/10.3748/wjg.v26.i43.6795>

120. Yu, H., Lin, Z.-X., Xiang, W.-L., Huang, M., Tang, J., Lu, Y., Zhao, Q.-H., Zhang, Q., Rao, Y., & Liu, L. (2022). Antifungal activity and mechanism of d-limonene against foodborne opportunistic pathogen *Candida tropicalis*. *LWT*, 159, 113144.

<https://doi.org/10.1016/j.lwt.2022.113144>

121. https://www.researchgate.net/publication/279551703_Antibacterial_and_antifungal_effects_of_Nigella_sativa_extracts_against_S_aureus_P_aeruginosa_and_C_albicans

122. dos Santos Barbosa, C. R., Scherf, J. R., de Freitas, T. S., de Menezes, I. R. A., Pereira, R. L. S., dos Santos, J. F. S., de Jesus, S. S. P., Lopes, T. P., de Sousa Silveira, Z., de Moraes Oliveira-Tintino, C. D., Júnior, J. P. S., Coutinho, H. D. M., Tintino, S. R., & da Cunha, F. A. B. (2021). Effect of Carvacrol and Thymol on NorA efflux pump inhibition in multidrug-resistant (MDR) *Staphylococcus aureus* strains. *Journal of Bioenergetics and Biomembranes*, 53(4),

489–498.

<https://doi.org/10.1007/s10863-021-09906-3>

123. İşcan, G., İşcan, A., & Demirci, F. (2016). Anticandidal Effects of Thymoquinone: Mode of Action Determined by Transmission Electron Microscopy (TEM). *Natural Product Communications*, 11(7), 977–978.

<https://pubmed.ncbi.nlm.nih.gov/30452175/>

124. Almshawit, H., & Macreadie, I. (2017). Fungicidal effect of thymoquinone involves generation of oxidative stress in *Candida glabrata*. *Microbiological Research*, 195, 81–88.

<https://doi.org/10.1016/j.micres.2016.11.008>

125. Li, X., Kim, J., Wu, J., Ahamed, A. I., Wang, Y., & Martins-Green, M. (2020). N-Acetyl-cysteine and Mechanisms Involved in Resolution of Chronic Wound Biofilm. *Journal of Diabetes Research*, 2020, 9589507.

<https://doi.org/10.1155/2020/9589507>

126. Penesyan, A., Paulsen, I. T., Kjelleberg, S., & Gillings, M. R. (2021). Three faces of biofilms: a microbial lifestyle, a nascent multicellular organism, and an incubator for diversity. *Npj Biofilms and Microbiomes*, 7(1).

<https://doi.org/10.1038/s41522-021-00251-2>

127. Dabeer, S., Rather, M. A., Rasool, S., Rehman, M. U., Alshahrani, S., Jahan, S., Rashid, H., Halawi, M., & Khan, A. (2022, January 1). Chapter 1 - History and traditional uses of black seeds (*Nigella sativa*) (A. Khan & M. Rehman, Eds.). ScienceDirect; Elsevier.

<https://www.sciencedirect.com/science/article/pii/B9780128244623000160>

128. Srinivasan, K. (2018). Cumin (*Cuminum cyminum*) and black cumin (*Nigella sativa*) seeds: traditional uses, chemical constituents, and nutraceutical effects. *Food Quality and Safety*, 2(1), 1–16.

<https://doi.org/10.1093/fqsafe/fyx031>

129. Noorbakhsh, M.-F., Hayati, F., Samarghandian, S., Shaterzadeh-Yazdi, H., & Farkhondeh, T. (2018). An Overview of Hepatoprotective Effects of Thymoquinone. *Recent Patents on Food, Nutrition & Agriculture*, 9(1), 14–22.

<https://doi.org/10.2174/2212798410666180221105503>

130. Tabassum, H., Ahmad, A., & Ahmad, I. Z. (2018). *Nigella sativa* L. and Its Bioactive Constituents as Hepatoprotectant: A Review. *Current Pharmaceutical Biotechnology*, 19(1), 43–67.

<https://doi.org/10.2174/1389201019666180427110007>

131. Cui, B.-W., Bai, T., Yang, Y., Zhang, Y., Jiang, M., Yang, H.-X., Wu, M., Liu, J., Qiao, C.-Y., Zhan, Z.-Y., Wu, Y.-L., Kang, D.-Z., Lian, L.-H., & Nan, J.-X. (2019). Thymoquinone Attenuates Acetaminophen Overdose-Induced Acute Liver Injury and Inflammation Via Regulation of JNK and AMPK Signaling Pathway. *The American Journal of Chinese Medicine*, 47(03), 577–594.

<https://doi.org/10.1142/s0192415x19500307>

132. Rashidmayvan, M., Mohammadshahi, M., Seyedian, S. S., & Haghizadeh, M. H. (2019). The effect of *Nigella sativa* oil on serum levels of inflammatory markers, liver enzymes, lipid profile, insulin and fasting blood sugar in patients with non-alcoholic fatty liver. *Journal of Diabetes & Metabolic Disorders*, 18(2), 453–459.

<https://doi.org/10.1007/s40200-019-00439-6>

133. Darand, M., Darabi, Z., Yari, Z., Saadati, S., Hedayati, M., Khoncheh, A., Hosseini-Ahangar, B., Alavian, S. M., & Hekmatdoost, A. (2019). *Nigella sativa* and inflammatory biomarkers in patients with non-alcoholic fatty liver disease: Results from a randomized, double-blind, placebo-controlled, clinical trial. *Complementary Therapies in Medicine*, 44, 204–209.

<https://doi.org/10.1016/j.ctim.2019.04.014>

134. Mohamed, S., & Wyson, J. (2017). IN VITRO IMMUNOSTIMULATION ACTIVITY OF NIGELLA SATIVA LINN. AND PSORALEA CORYLIFOLIA LINN. SEEDS USING A MURINE MACROPHAGE CELL LINE. www.semanticscholar.org.

<https://www.semanticscholar.org/paper/IN-VITRO-IMMUNOSTIMULATION-ACTIVITY-OF-NIGELLA-AND-Mohamed-Wyson/114f14cc437d06e59b6dd8677aee6c179451afff>

135. [RESTRICTED]

136. Alshatwi, A. A. (2014). Bioactivity-guided identification to delineate the

immunomodulatory effects of methanolic extract of *Nigella sativa* seed on human peripheral blood mononuclear cells. *Chinese Journal of Integrative Medicine*.
<https://doi.org/10.1007/s11655-013-1534-3>

137. Niu, Y., Wang, B., Zhou, L., Ma, C., Waterhouse, G. I. N., Liu, Z., Ahmed, A. F., Sun-Waterhouse, D., & Kang, W. (2021). *Nigella sativa*: A Dietary Supplement as an Immune-Modulator on the Basis of Bioactive Components. *Frontiers in Nutrition*, 8.
<https://doi.org/10.3389/fnut.2021.722813>

138. Salem, M. L., Alenzi, F. Q., & Attia, W. Y. (2011). Thymoquinone, the active ingredient of *Nigella sativa* seeds, enhances survival and activity of antigen-specific CD8-positive T cells in vitro. *British Journal of Biomedical Science*, 68(3), 131–137.
<https://doi.org/10.1080/0>

139. Kheirouri, S., Hadi, V., & Alizadeh, M. (2016). Immunomodulatory Effect of *Nigella sativa* Oil on T Lymphocytes in Patients with Rheumatoid Arthritis. *Immunological Investigations*, 45(4), 271–283.
<https://doi.org/10.3109/08820139.2016.1153649>

140. Shahid, F., Farooqui, Z., Khan, A. A., & Khan, F. (2018). Oral *Nigella sativa* oil and thymoquinone administration ameliorates the effect of long-term cisplatin treatment on the enzymes of carbohydrate metabolism, brush border membrane, and antioxidant defense in rat intestine. *Naunyn-Schmiedeberg's Archives of Pharmacology*, 391(2), 145–157. <https://doi.org/10.1007/s00210-017-1444-6>

141. Samarghandian, S., Farkhondeh, T., & Samini, F. (2018). A Review on Possible Therapeutic Effect of *Nigella sativa* and Thymoquinone in Neurodegenerative Diseases. *CNS & Neurological Disorders - Drug Targets*, 17(6), 412–420.
<https://doi.org/10.2174/1871527317666180702101455>

142. Mahmoud, Y. K., & Abdelrazek, H. M. A. (2019). Cancer: Thymoquinone antioxidant/pro-oxidant effect as potential anticancer remedy. *Biomedicine & Pharmacotherapy*, 115, 108783.
<https://doi.org/10.1016/j.biopha.2019.108783>

143. Abbasnezhad, A., Niazmand, S., Mahmoudabady, M., Soukhtanloo, M., Abdolrahim Rezaee, S., & Mojtaba Mousavi, S. (2016). Nigella Sativa Improve Redox Homeostasis in Heart and Aorta of Diabetic Rat. *Current Nutrition & Food Science*, 12(1), 35–41.

<https://www.ingentaconnect.com/content/ben/cnf/2016/00000012/00000001/art00009>

144. Hassan, Md. Q., Akhtar, Mohd., Ahmed, S., Ahmad, A., & Najmi, A. K. (2017). Nigella sativa protects against isoproterenol-induced myocardial infarction by alleviating oxidative stress, biochemical alterations and histological damage. *Asian Pacific Journal of Tropical Biomedicine*, 7(4), 294–299.

<https://doi.org/10.1016/j.apjtb.2016.12.020>

145. Ezirganli, S., Kazancioglu, H. O., Ozdemir, H., Inan, D. S., & Tek, M. (2016). The Effects of Nigella Sativa Seed Extract on Bone Healing in an Experimental Model. *Journal of Craniofacial Surgery*, 27(7), 1905–1909.

<https://doi.org/10.1097/scs.0000000000002986>

146. Hassan, Md. Q., Akhtar, Mohd., Ahmed, S., Ahmad, A., & Najmi, A. K. (2017). Nigella sativa protects against isoproterenol-induced myocardial infarction by alleviating oxidative stress, biochemical alterations and histological damage. *Asian Pacific Journal of Tropical Biomedicine*, 7(4), 294–299.

<https://doi.org/10.1016/j.apjtb.2016.12.020>

147. Badary, O. A., Al-Shabanah, O. A., Nagi, M. N., Al-Bekairi, A. M., & Elmazar, M. M. A. (1998). Acute and subchronic toxicity of thymoquinone in mice. *Drug Development Research*, 44(2-3), 56–61. [https://doi.org/10.1002/\(sici\)1098-2299\(199806/07\)44:2/3%3C56::aid-ddr2%3E3.0.co;2-9](https://doi.org/10.1002/(sici)1098-2299(199806/07)44:2/3%3C56::aid-ddr2%3E3.0.co;2-9)

148. Wong, P., Lou, Assaw, S., Lokman, M., Suhaimin, N., & Yusof, H. (2018). SUB-ACUTE TOXICITY OF BLACK SEED (*Nigella sativa*) AND HONEY MIXTURE. *Malays. Appl. Biol*, 6, 11–18.

<https://core.ac.uk/download/pdf/195387730.pdf>

149. Ali M. Al-Amri, M. D., & Abdullah O. Bamosa MBBS, P. H. D. (2009). Phase I Safety and Clinical Activity Study of Thymoquinone in Patients with Advanced Refractory Malignant Disease. *Shiraz E-Medical Journal*, 10(3), 107–111.

<https://brieflands.com/articles/semj-76453.html>

150. Thomas, J. V., Mohan, M. E., Prabhakaran, P., Das S, S., Maliakel, B., & I.M., K. (2022). A phase I clinical trial to evaluate the safety of thymoquinone-rich black cumin oil (BlaQmax®) on healthy subjects: Randomized, double-blinded, placebo-controlled prospective study. *Toxicology Reports*, 9, 999–1007.
<https://doi.org/10.1016/j.toxrep.2022.04.020>

|

How to join the daily conversation, follow the latest news and discoveries and read direct reports from those on the protocol:

Join our new Reddit Community

<http://www.reddit.com/r/cosmicdeathfungus>