Carnicom Institute Research

2013

Acknowledgements

Mission Statement

Carnicom Institute is a non-profit organization working solely for the benefit of humanity. Our goal is to provide the public with beneficial and responsible information through scientific, educational, environmental, and health research for the public welfare. The Institute has devoted significant effort to the important issues of geoengineering and bioengineering.

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Morgellons : Infrared Spectroscopy – Culture Confirmation

carnicominstitute.org/morgellons-infrared-spectroscopy-culture-confirmation/

Morgellons :
Infrared Spectroscopy – Culture Confirmation
Clifford E Carnicom
Jan 01 2013

Note: I am not offering any medical advice or diagnosis with the presentation of this information. I am acting solely as an independent researcher providing the results of extended observation and analysis of unusual biological conditions that are evident. Each individual must work with their own health professional to establish any appropriate course of action and any health related comments in this paper are solely for informational purposes and they are from my own perspective.

An elderly, but wonderfully functional, Perkin Elmer 1320 infrared (IR) spectrophotometer has been acquired by the Carnicom Institute. This class of instrument has been sought after for many years by this researcher and organization. The value and purpose of an infrared spectrophotometer (along with other instruments as well) is that it can be used to gain insight into the molecular structure of organic compounds. This is a crucial need that has remained unfulfilled for many years in the biochemistry research that has taken place thus far. It is not an overstatement to realize that years of work can equivalently be accomplished with greater certainty and insight in relative moments of time with the proper instrumentation and resources. It is hoped that this equipment can be augmented or replaced with modern computer-based instrumentation at some point in the near future, however, the process of discovery at this important level can now begin.

And so it does begin…

With the use of infrared spectroscopy it can be stated, with a high level of certainty, that the *cultures* that have been developed over the past several years *from* oral samples *are essentially identical to the oral filament samples* themselves. This finding has numerous ramifications of importance. Before that point is clarified further, let us present the spectra that make this case:

The first IR spectrum that will be presented is that of the oral filament sample that has been discussed at length by this researcher in numerous reports on this site. This filament is produced with the so-called “red-wine test”, that was discovered and that is credited to Dr.

Gwen Scott several years ago. The process of this simple test as well as the general chemistry of the reaction (involving anthocyanins) has also been discussed elsewhere on this site.

The method of preparation of the sample in this case is as follows:

1. The oral filament sample is extracted from the gums of the individual using red wine.

2. The filament sample is rinsed thoroughly in numerous exchanges of distilled water.

3. The sample is dried thoroughly in a watch glass under low heat.

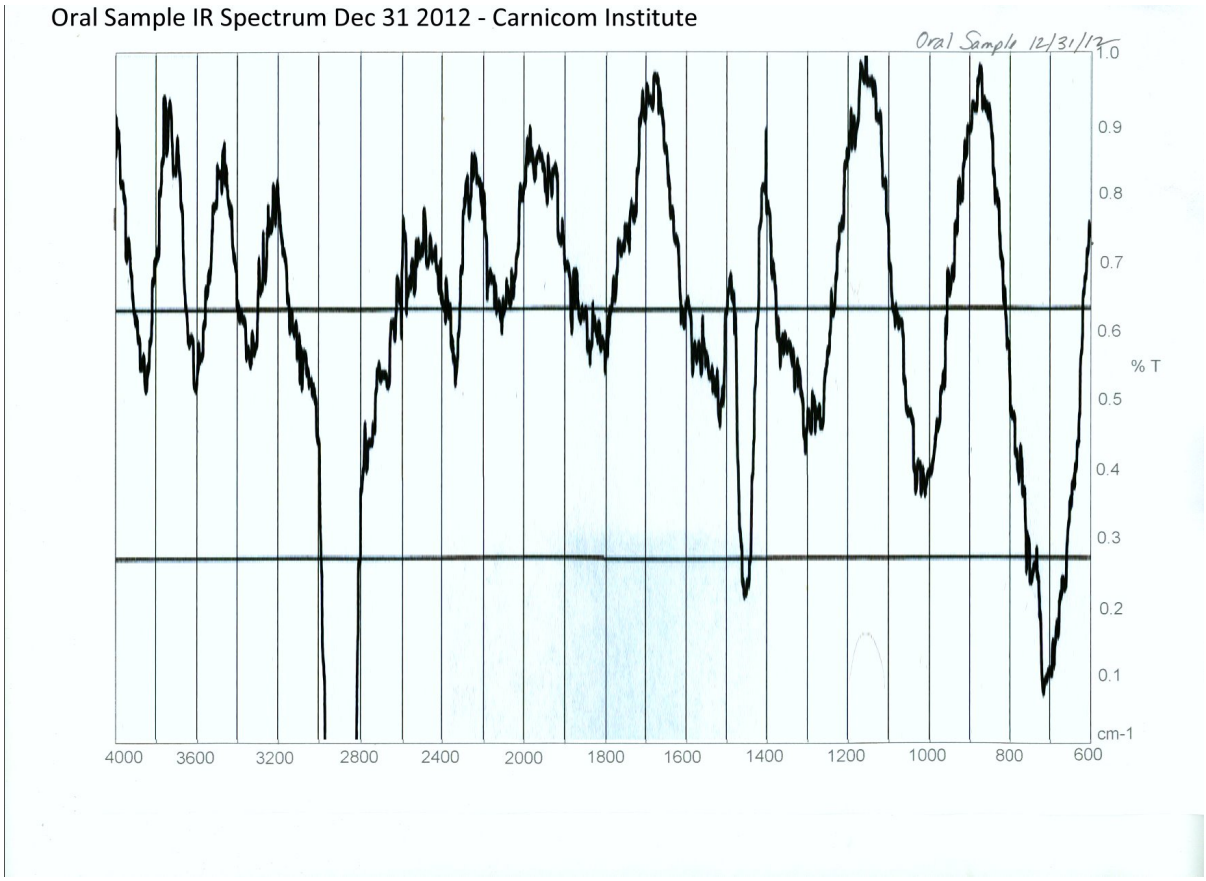
4. The sample is then scraped and pulverized to powder form.

5. A small amount of ethanol is added for purposes of further grinding with a pestle to small particle size.

6. The ethanol is then allowed to evaporate thoroughly.

7. The sample is then sandwiched into a polyethylene sample card and the spectrum compared to a reference card of polyethylene. Polyethylene has certain absorption peaks (alkanes) which are to be ignored in the analysis of the spectrum (especially ~2800-3000 cm-1 and the peak at approximately 1465 cm-1). Additional sampling equipment, hopefully to be acquired in the near future (e.g., salt crystals), will provide additional substrate references to eliminate this interference. Cost is a factor that prevents this from occurring at this time.

The resulting spectrum is shown below and it has many interesting features that will be discussed at length in future reports. The objective of this report is simply to make the case of identity between the oral sample and the subsequent cultures developed from those oral samples. Functional group analysis of the spectra will be an important topic of discussion, but it shall be reserved for the future as time and circumstances permit.

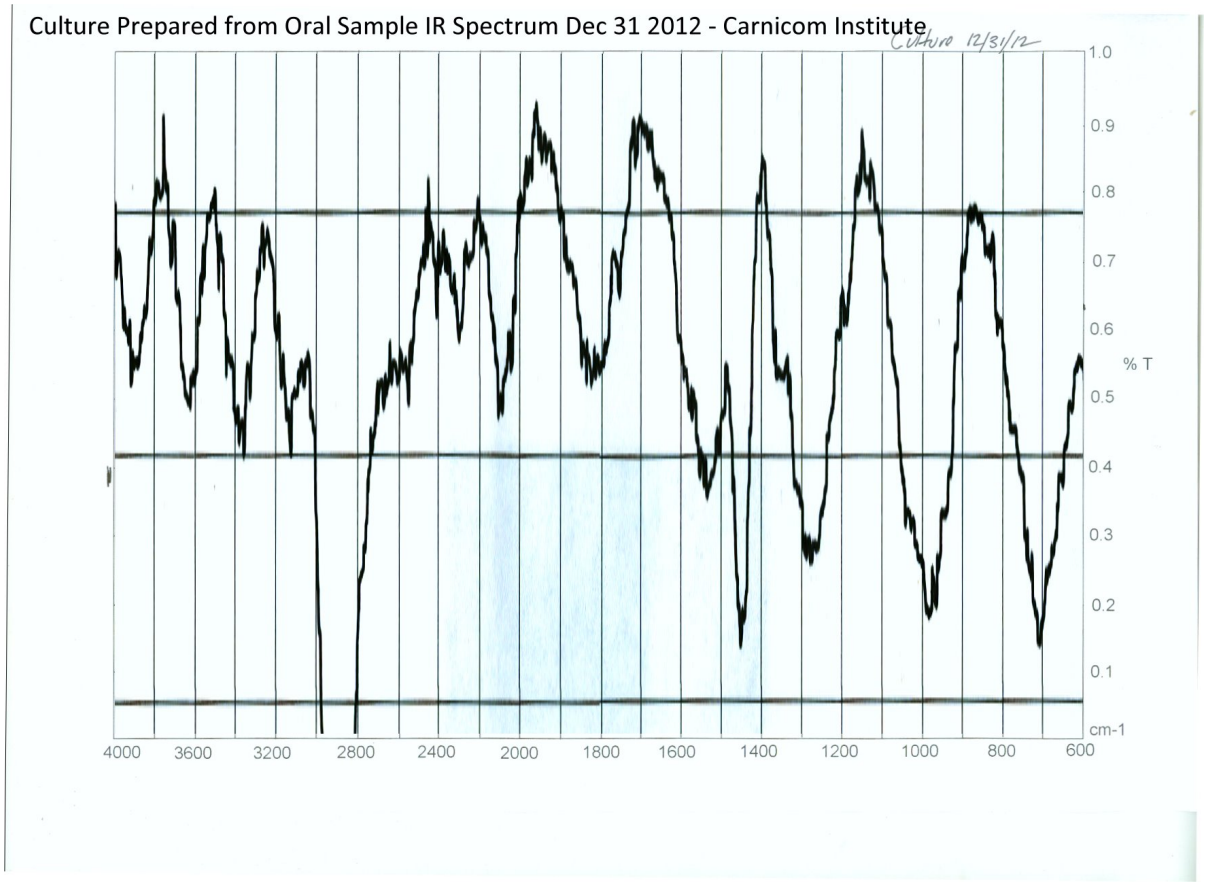


Oral Sample Infrared Spectrum. The horizontal axis corresponds to wavelength (cm-1) and the vertical axis is that of % transmittance (normalized). The spectrum is relatively complex in nature and the particulars of the functional groups inherent within will be discussed at length in the future. At this point, the locations of the troughs of the chart, e.g, ~2350 cm-1, 2100 cm-1, 1800-1900 cm-1, 1250-1300 cm-1, 1000-1030 cm-1 and 720 cm-1 can be noted as specific and distinct reference points. A spectrum is essentially a unique fingerprint of an organic substance or compound; the complexity of this particular spectrum even further emphasizes this point. The absorption peaks at ~2800-3000 and at ~1465 are to be dismissed in this analysis at this time (only) due to the spectral behavior of polyethylene used as a substrate; the clipping of the former peak range is intentional for the graphic presentation.

The spectrum of the culture growth, ***as developed from the oral filament samples***, is also shown below. The method of preparation is similar to that of the above, with a few exceptions. In the case of the culture growth, an extract is prepared prior to the steps outlined above. This extraction process has also been described elsewhere on this site, but it basically involves a process of substantial growth over weeks to months of time, several thorough rinsing stages, complete drying and pulverization and then subjecting the resulting powder to a sodium hydroxide and heating process. Extensive chemical testing, chromatography, electrolysis, ninhydrin analysis and visible light spectroscopy analysis has

been applied to this extract and this has been reported on in detail on this site. Please see these reports for additional background information on studies by the Carnicom Institute on the Morgellons condition.

The conclusion that can be reasonably made from the presentation of these spectra is that they are essentially of one and the same thing. This effectively means that all of the conclusions that have been drawn by this researcher over a period of several years that were applied previously to the culture forms may now be equally applied to the original oral filaments. This is an expression of the value of the culture process itself, and is one of the many reasons that so much effort and time was dedicated to that process over a period of many years. It also can be stated, in equivalent form, that the assessments and conclusions reached with the use of these cultures has a level of validity that is expected to apply to the human body. It is asserted once again that no medical diagnosis of any kind is implied or stated within this report; it *is* being stated that the culture forms represent a viable means of study of metabolism and biochemical structure that holds numerous advantages over attempting to study these same processes within the human body.



Infrared Spectrum of Culture Developed from an Oral Filament Sample (normalized). Note that the two spectra, the oral sample and the culture derived from that same oral sample, are essentially one and the same.

This report will therefore further dispute any claims that are made regarding the insignificance of the oral filament samples and the cultures that are derived from those same oral filaments. It will further dispute any inadequate research claims regarding unrelated precipitation reactions involving wine and saliva (also discussed elsewhere on this site); such claims do not apply to this situation and they remain ludicrous under the volume of study that has accumulated. This report will further dispute any claims of a delusional, psychological or metaphysical characterization of the physical samples that exist **and that can be reliably cultured and studied**. This report will dispute any claim of simple classification to a known mold or fungal species; such identification remains unestablished and all signs continue to point to uniqueness and complexity in the affair. The molecular structure of this organism exists quite aptly in this physical world and it is taking its toll upon the human population as long as it exists. This physical structure will continue to be elucidated by this researcher so that its biochemistry and its interaction with the body can be properly understood and eventually thwarted.

Clifford E Carnicom
Dec 31 2012

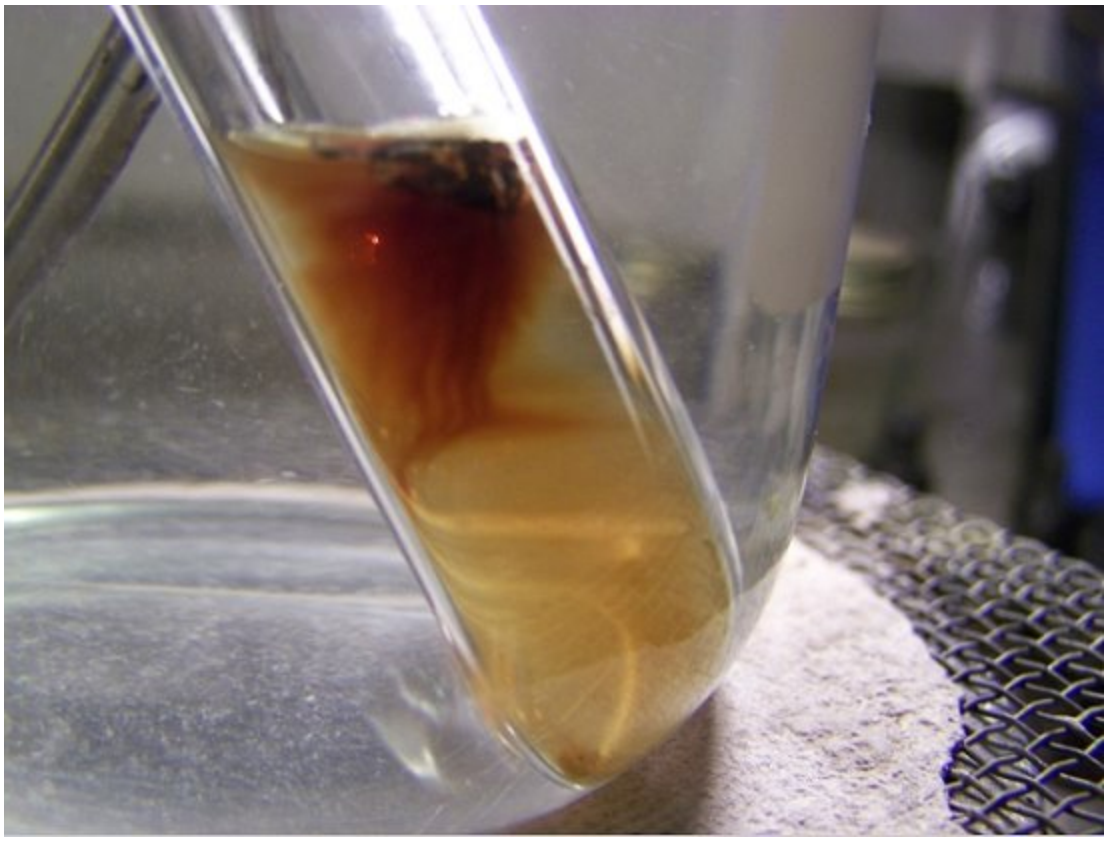
Environmental Filament Penetration

carnicominstitute.org/environmental-filament-penetration/

Environmental Filament Penetration
Clifford E Carnicom
Jan 06 2013
An improved method of penetration of the environmental (airborne) filament sample has been achieved. This accomplishment provides a pathway to an increased understanding of the structure and contents of the fibers. Numerous studies have been reported on the nature of this filament material over the years on this site. This material is the same type of material that was sent to the U.S. Environmental Protection Agency (EPA) over a decade ago. The EPA refused to identify this material on the behalf of the public interest.

The original sample in this case came from Serbia; much appreciation is extended for the effort that made this sample available for study. A photograph of this original sample is shown below:

Environmental Filament Sample that is the basis of this investigation. Additional photographs related to this sample can be found on the following outline page: Morgellons and Recent Findings.



Photograph of the chemical method established to break down the outer shell of the filament and to access the contents of the filament. The method uses a combination of concentrated sodium hydroxide, concentrated potassium hydroxide and heat in a boiling water bath. Note the separation of colors within the solution within the test tube, one yellowish and one a deep red color. These colors represent different chemical and structural components of the filament. Approximately 45 minutes are required at boiling temperature to complete the separation.

Considerable experimentation was required to achieve the method used in this report. Many trials have been taken in the past using sodium hydroxide and heat alone. The combination of the extreme caustic solutions applied to the filament along with a gentler heating process is a substantial improvement over previous methods. Initial conclusions about the nature of the filament using this method will be discussed in a following report. At this point it is of interest to note the following observations:

1. The original filament material is pure white, with no external or internal colors available.

2. The breakdown of the filament shows two colored effects, one a yellowish component and one a strong reddish component. The strong colors internal to the filament, especially the brilliant reddish hue, are of more than casual interest. Readers may wish to reexamine the numerous papers on this site involving blood and erythrocyte research as they relate to cultured (biological and environmental) filaments.

A great deal of information on the nature of these filaments is already available on this site; readers are encouraged to become familiar with this body of research that exists. The advantage of the current finding is that it will allow more direct and ready access to the chemical composition of the filament samples.

Environmental Filament : Keratin Encasement

carnicominstitute.org/environmental-filament-keratin-encasement/

Environmental Filament : Keratin Encasement
Clifford E Carnicom
Jan 07 2013

It can now be established with a high degree of certainty that the external casing of the environmental filament samples are composed of keratin or a keratin-like material. This supposition has been in place for a number of years by this researcher; it can now be demonstrated to be the case by direct chemical and spectroscopic means. Certain ramifications of this finding, in conjunction with earlier work, are as follows:

1. It is deduced that the environmental filament is not a naturally occurring material.

2. The filaments contains non-keratin based chemical and biological components within the internals of the filaments. Considerable information regarding the nature of the environmental filaments is available on this site; this information has been accrued over a period of several years of progressive research.

3. **The emphasis upon study of the filaments is to be directed to the sub-micron components (biological and chemical) that are *internal* to the filaments. The keratin aspect of structure is to be interpreted as an encasing mechanism only.**

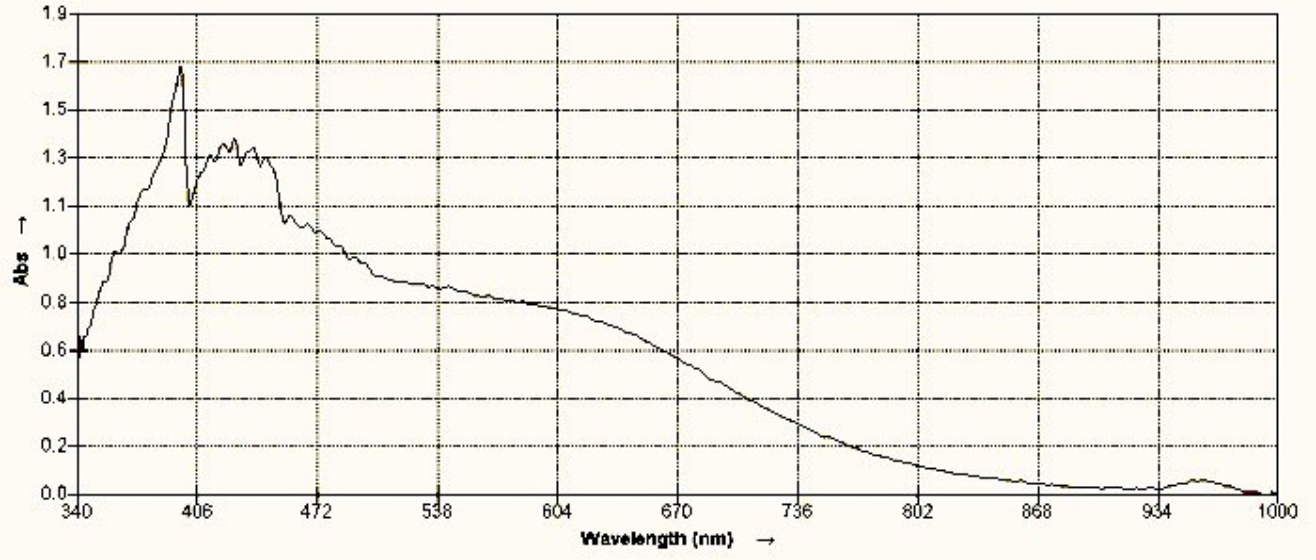
4. The filaments are not hair or spider webs.

5. A false laboratory report has been issued in the past regarding the identification of this filament material (to be discussed in a separate report).

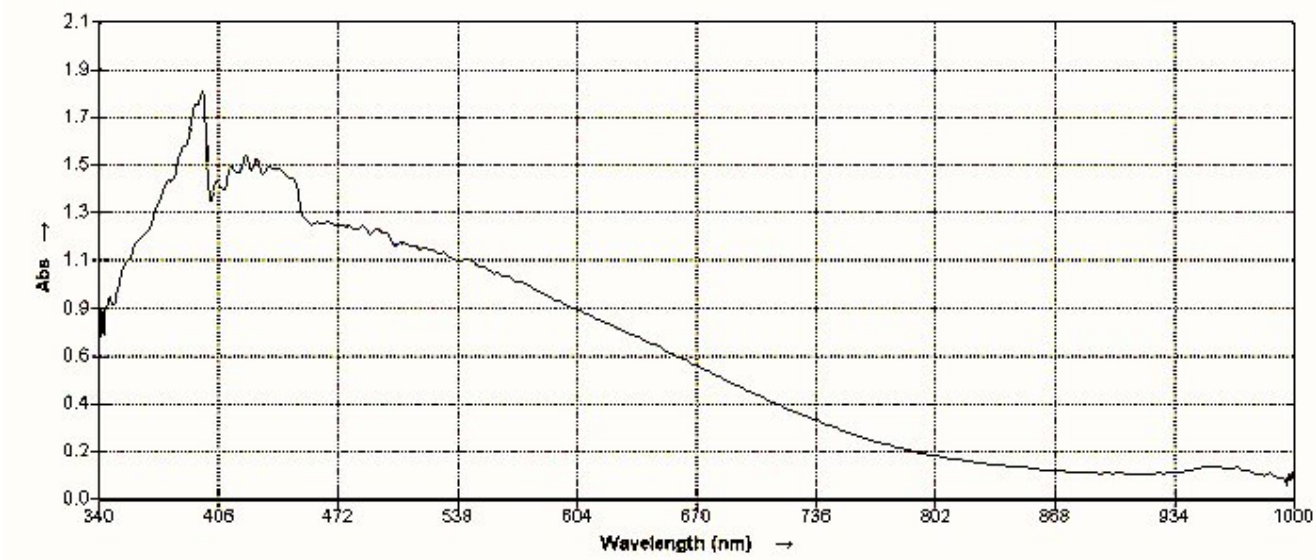
The primary method by which this conclusion has been reached is with chemical and spectroscopic comparison of a known source of keratin with the environmental filament by similiar means. This comparison has been made possible with the recent advance in methods of chemical decomposition of keratin based substances by this researcher. Please see the report entitled “Environmental Filament Penetrated” for this discussion and presentation.

Human Hair Environmental Filament Sample

Spectroscopic comparisons of keratin obtained from human hair and the same substance obtained from the environmental filament casing are shown immediately below; it will be seen that they are essentially identical. Additional notes and discussion will follow below the spectra.



A visual light spectroscopic analysis of keratin obtained from the decomposition of human hair in combination with ninhydrin and heating. Human hair is composed predominantly of keratin.



A visual light spectroscopic analysis of the environmental filament after chemical decomposition and in combination with ninhydrin and heat. It will be seen that the spectrum obtained is essentially identical to the keratin spectrum above. The keratin of the environmental filament is interpreted as an encasement structure and it ***does not account for*** the biological components that have been repeatedly identified within this protein housing.

A photograph of the colored solutions subjected to visual light spectroscopic analysis. The solutions result from chemical decomposition of keratin based structures, in combination with heat and ninyhdrin. The solution on the left is derived from the environmental filament sample; the solution on the right is derived from human hair. Both hues and spectra can vary to some degree by concentration levels within a solution; these examples and spectra indicate a coincidence of relative concentration in each case.

Additional Notes:

Keratin is an especially impervious protein structure. Observation and study of your own hair is a very good analogy for understanding the hardiness of this particular protein. During the recent trials of study in decomposition, chemical penetration of hair itself represents an excellent example of the challenge of examination of the environmental filaments and their internals. Numerous trials were conducted using strong solutions of sulfuric acid, sodium hydroxide, nitric acid, salicylic acid, sodium hypochlorite (bleach), ammonium thioglycolate and others. All essentially met with failure to the degree needed with the time available. Although some mild success was achieved with a hair sample, the environmental filament sample remained essentially impervious to almost all methods. The best success of decomposition has eventually come forth with the use of a commercial hair declogger used in plumbing systems. This solution is primarily a combination of concentrated sodium hydroxide and concentrated potassium hydroxide. This solutions is highly caustic. The greater success of this method also becomes dependent upon the use of applied heat over an extended time period. It was with the use of this method that valid comparisons, both chemically and spectroscopically, could be made. Considerable work remains before us to acquire the detailed biochemical knowledge of the internal nature of the environmental filaments; this work will continue as the proper resources and equipment avail themselves.

Environmental Filament : False Report

carnicominstitute.org/environmental-filament-false-report/

Environmental Filament : False Report
Clifford E Carnicom
Jan 08 2013
It is now appropriate to disclose the circumstances involving a laboratory report on an airborne filament sample that was paid for in the year of 1999. This report was issued jointly by three separate companies and they shall remain anonymous at this time. It is now appropriate to present this information as the conclusions of the report are undeniably false. Whether or not there was intent to misrepresent the facts of the case is not to be discussed in this paper; the purpose is to disclose information that is relevant to the public interest and welfare. The laboratory was hired and paid significant monies to analyze and identify the very same airborne environmental filament sample that was sent to the United States Environmental Protection Agency (EPA) during this same time period of 1999-2000. The failure of the EPA to identify that sample is adequately documented in this site. This report will chronicle the events that surround this affair.

The circumstances are generally as follows:

1. A laboratory in the southwestern United States was privately contracted in the fall of 1999 to identify an airborne environmental filament sample. The nature of this environmental filament has been discussed and researched extensively on this site over the subsequent years. A portion of this same sample was sent to the EPA for identification as noted above. The reason for contracting with the private company was because of the failure of the EPA to identify the material.

2. The laboratory report was issued in December of 1999 with joint responsibility of findings between three separate companies. The report claims to use the results of infra-red spectroscopic analysis and Polarized Light Microscope Analysis on the sample.

3. The final statement of analysis from the contracting laboratory is as follows (names of laboratories redacted). The conclusions of this report will be discussed in more detail below.

Attached please find enclosed two reports, one from [redacted] and one from [redacted] The [redacted] report is a bit subjective, indicating that the material could be a spider's web. In contrast, the [redacted] report is fairly definitive and is based on FTIR analysis. This analysis shows that the white material is a naturally occurring proteinaceous material such as wool or silk.

4. At the same time that the laboratory was conducting their tests, I also was conducting my own tests on this same sample material. The results of that testing process are extensively reported on within this web site. Certain primary conclusions were being reached on my side about the nature of the material such as size, chemical reactivity, microscopy results, conditions of collection and the like. Prior to the results being officially released, we were given the subjective information above relaying that the material "could be" a "spider's web". It was quite clear to me from my own analysis that the testing results were inadequate and inaccurate, as it was already evident that the material was not a "spider web". The final report claiming to use spectral analysis was then issued, and it was clear to me at this point that a contest of conclusions was in order. It was equally obvious through any reasoned analysis that the material was likewise not a wool fiber or any other obvious fabric or textile. Readers familiar with "counter arguments" of the period will also know that a commonly circulated theme by a relatively small group of vocal advocates was that the material was simply a "spider's web that had fallen from the sky."… There were also questions that had emerged from the spectral reports themselves.

5. At this point, it was obvious that a rather serious and important conflict of conclusions had developed. The first conflict arose from the failure of the EPA to identify the material on behalf of the public interest. The second conflict resulted from paid professional services that provided obvious and conflicting information to my own independent analysis of the material.

6. A personal visit and meeting with the president of the issuing company was then arranged. The meeting had three participants: the president of the company, Dave Peterson (a colleague of mine) and myself. The subject of the meeting was identified ahead of time to all parties as a discussion of the conclusions that had been issued by the laboratory. It is also a fact that the letter presented below was written by myself *prior* to the actual meeting and it was held in reserve until the outcome of the meeting was decided. It is fair to say that I had serious concerns and issues with the professionalism and honesty of the science that was on display by the laboratory.

7. Prior to the meeting, in addition to the letter written and held below, I had also prepared a list of nine line items that substantiated, from my own analyses, why the laboratory results issued were false. At the opening of the meeting, I expressed my concern that I had some reservations and conflicts with the validity of the report and that I would like to discuss them with him. It is also true that the atmosphere of the meeting was generally one of unspoken tension and alertness.

8. I began with my first item of nine on the list. This issue was simply the point and question of direct observation, especially under the microscope. I told the president of the company that the materials did not even look like spider webs under the scope. In my own analyses, I made extensive study of numerous filament, textiles, hairs and filaments in general, including those of spider webs. I actually had the serious issue as to whether or not

the sample had been properly *observed*, as it is the starting point of the scientific method. The president of the company did not contest or agree with or discuss my point of contention in any fashion, there was at most a tacit or implied acknowledgment of this first of nine points.

9. I then proceeded to the second item on my list of nine. This issue had to do with the size of the filaments. The size of the filaments is micron to sub-micron in nature, and it does not correspond in any physical or possible way to a hair or a spider web. My own measurements of spider webs were in the order of seven microns and hair is on the order of 60-100 microns. The conclusion on the laboratory report simply had no justifiable metric basis. I again wondered privately whether or not the laboratory had made the effort to even measure as well as look at the filament in any detail.

10. The next event in the meeting was entirely unexpected. At the end of the second of nine points to be raised, the president of the company immediately halted the discussion and my speech. The words that were uttered by this individual were the following:

"This meeting is now adjourned."

11. There was nothing more that was allowed to be said. The meeting was over as I had reached item two on my list of nine. At this point, I personally handed the letter that I had written apriori to the President of this company. Thirteen years later, it is now time to make this correspondence available to the public. The letter could not be presented until a certain confidence in laboratory results was achieved; this is now in place.

12. The letter written at the time of the meeting in the year 2000 is presented below for the record:

Clifford Carnicom
David Peterson
[redacted]

January 6, 2000

To: [redacted]

It is to be stated that the conclusions reached under laboratory analysis by joint participation of:

[redacted] Air Quality testing
[redacted] Analytical Services
[redacted] Analyze [redacted]
[redacted] Analyze [redacted]

are false. This statement is arrived at by independent analysis of the samples originally delivered to the above parties. Our analysis is the product of the following methods and techniques:

Visual
Metric
Behavioral characteristics
Chemical tests
Environmental conditions of sampling
Spectra analysis

The laboratory parties stated are hereby both requested and obligated to consider all available information and to further substantiate or re-examine the conclusions which have been professionally declared. If sufficient and complete information to substantiate by announced findings is not provided, we declare our intentions to:

1. Publicly refute the conclusions of the aforementioned parties and the associated laboratories [redacted].
2. Publicly proclaim our own independent analysis and findings for the samples under evaluation.

There is a need and obligation for further substantiation of your laboratory findings. In the absence of such a response, we will forward truthful and complete disclosure of our independent findings to the public and in addition, independent professional laboratory analysis will be sought.

Clifford Carnicom

David Peterson

13. There are additional details that can be discussed. In the short form, let me assert to you that these airborne environmental filaments, that have been repeatedly observed, reported and collected over the last decade and a half, at a minimum, are:

a) NOT naturally occurring.

b) NOT a spider's web or silk.

c) NOT wool (or any other common textile fiber or hair).

14. They are, however, at least in part, indeed a "proteinacous material", but that is another story….

Sincerely,

Clifford E Carnicom

Jan 07 2013

Additional Note from David Peterson provided on Jan 07 2013:

> The reason my signature does not appear on this statement is that I trusted that we were dealing with a legitimate laboratory at the time this document was presented to them. There were inconsistencies in their findings that were sent to us via USPS prior to this that were the reason the face to face meeting needed to take place. I attended this meeting with Clifford Carnicom to address our concerns with their findings, so I was indeed a witness to how the meeting transpired and in retrospect I would have absolutely signed this document when it was presented to them.
>
> David Peterson

(P.S. Dave, thank you, 13 years later…)

Then and Now

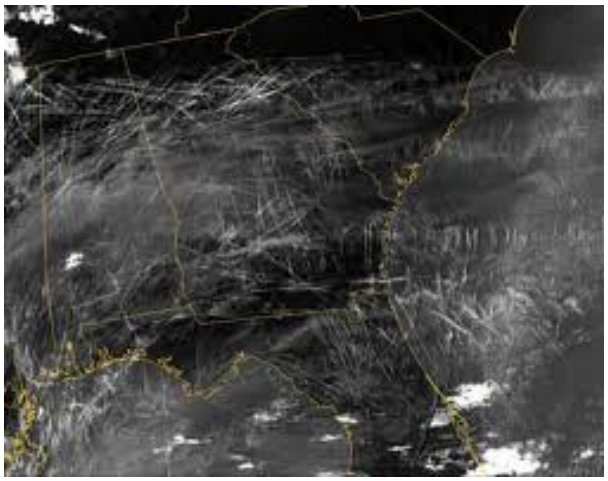
carnicominstitute.org/then-and-now/

Then and Now

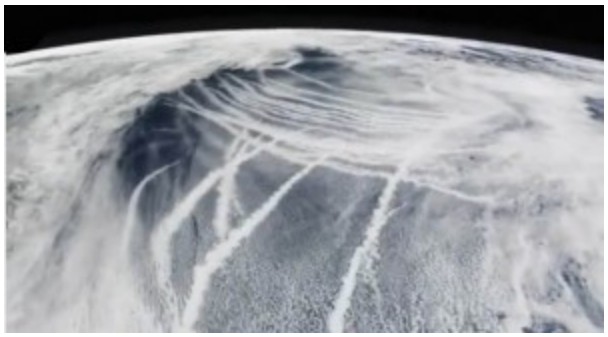
Clifford E Carnicom
June 28 2013

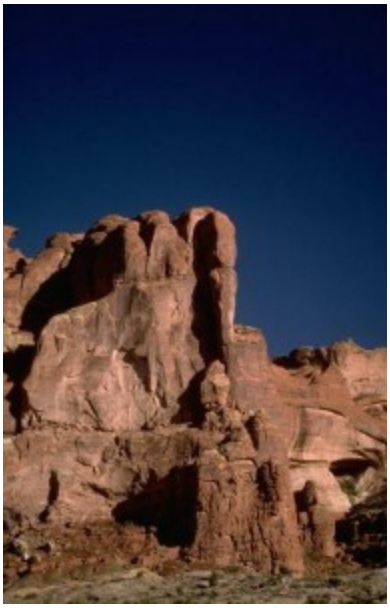
The following is a comparison between stock photography images that predate the year of 1999 and environmental photographs that have been published by the public on the internet after that same date. The reader can make his or her own determination, from both environmental and health perspectives, as to the source and impact of the significant changes that have taken place. Please show this page to your children so that they may understand what has been stolen from them.

Pre 1999 Stock Photography Images Post 1999 Public Internet Images



What can we do about it?





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Environmental Filament Project : An Introduction

carnicominstitute.org/environmental-filament-project-an-introduction/

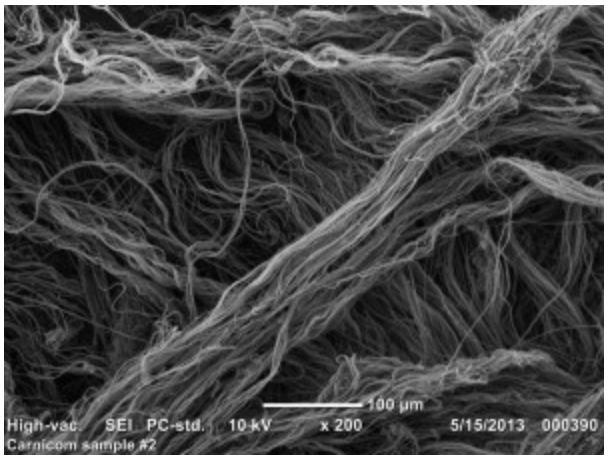
Environmental Filament Project :

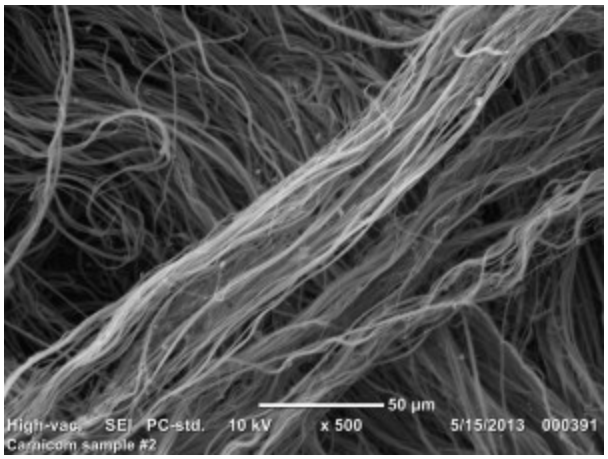
An Introduction

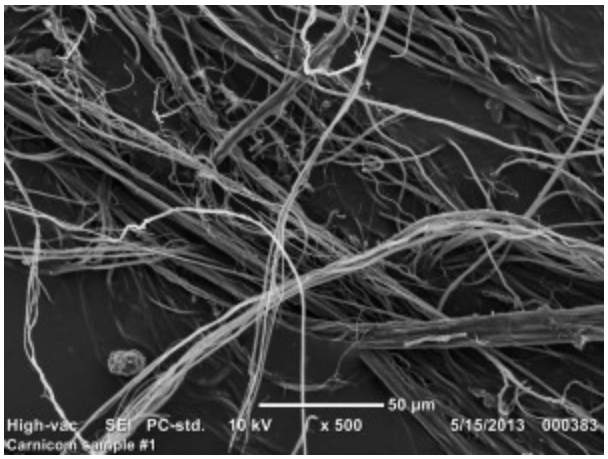
Clifford E Carnicom
Jul 09 2013

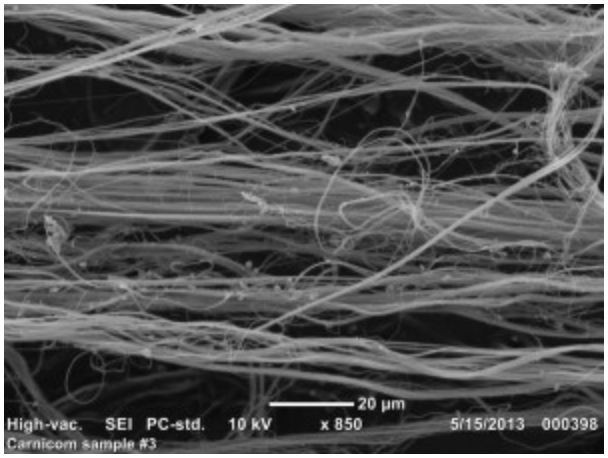
Under current projections, it wll be some months ahead before I will be able to engage fully into the Environmental Filament Project that has been outlined under this site. In the interim, however, an important introduction to what lies ahead can be presented. Carnicom Institute is now able to display a series of scanning electron microphotographs of a typical sample; they will not be discussed in any detail until I am able to begin the study project. Those familiar with my work may be aware of my reluctance to use the term nano-technology in association with any environmental or biological samples examined thus far; this has been due to the lack of any electron microscope images that are derived directly from these same samples. This is no longer the case, and ***the use of the nano-technology term in association with this material is now fully justified***. The samples shown below are identical to those that the United States Environmental Protection Agency has refused to identify or analyze. It has taken close to a decade and a half to acquire these images; appreciation is extended to all parties that have helped to make this information available to the public. Sufficient additional samples have been received, both national and internationally, to support the Institute project plans. This study will begin as the opportunity affords itself and as parallel work that is underway is completed. Light microscope images of the same material are also shown below.

Carnicom Institute : Electron Microphotographs of Environmental Filament Sample

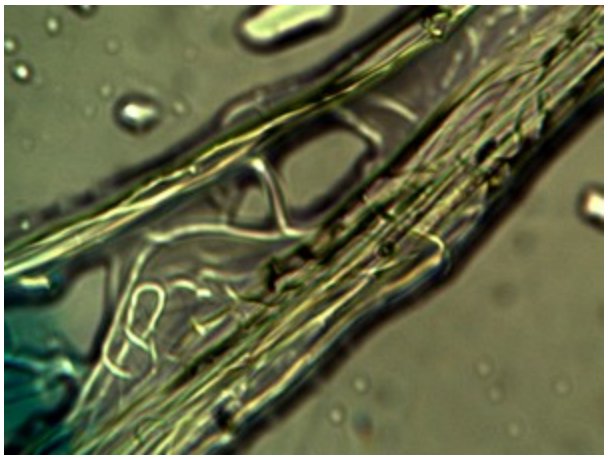


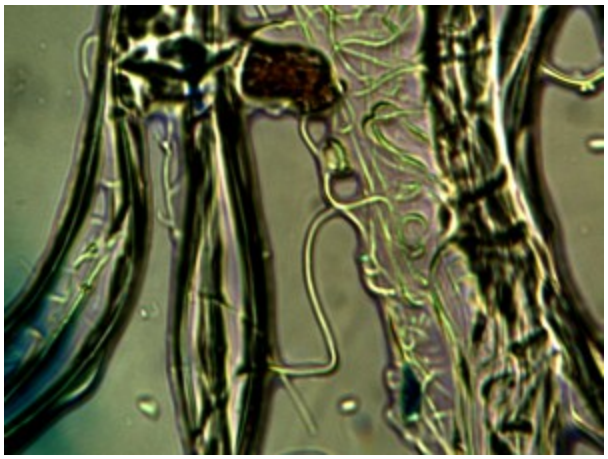


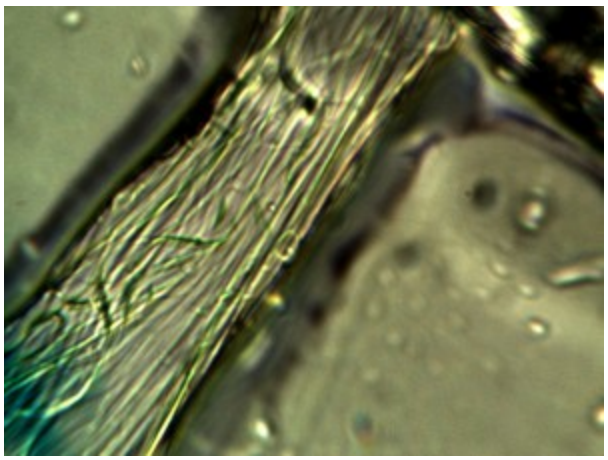




Carnicom Institute : Light Microscope (CMOS) Photographs of Environmental Filament Sample







Approximate magnification of original imagery : 6000x

Advances in Microscopy Blood & Skin Filament Examinations – A Slide Show

carnicominstitute.org/advances-in-microscopy-blood-skin-filament-examinations-a-slide-show/

Advances in Microscopy

Blood & Skin Filament Examinations – A Slide Show

(click on any image – controls available on each image)
Nov 19 2013
Clifford E Carnicom

A maximum magnification that combines optical and digital means has recently been achieved. The development allows, under suitable conditions and sampling, a magnification of images at a reasonable resolution up to a level of approximately 18,000 power. This method has been applied to the examination of human blood samples as they relate to the "Morgellon's" condition. A brief introduction to the results of this recent advance in microscopy that uses relatively limited means and equipment is presented below. Relevant topics of research that arise from the study include the more detailed appearance of the bacterial-like structure that has been studied extensively by this research. The degradation of the red blood cell exterior membrane is also clearly apparent. The rather striking appearance of white blood cells, their behavior with respect to the bacterial-like component, and the internal structures that are visible within the white blood cells are of high interest. The importance of an active immune system against the bacterial-like encroachment is immediately obvious. Introductory live-blood video analysis recently performed further emphasizes the importance of the relationship of the immune system to the Morgellon's condition. This level of awareness and visibility on the Morgellon's condition is a direct result of these recent advances in microscopy methods and techniques. The availability of more advanced equipment, should it become available, will accelerate this discovery process.

BLOOD MICROSCOPY

A brief discussion and history of the individual providing the blood control photographs above is in order. This particular individual, several years past, had blood conditions that are identical to those which are the primary subject of this paper. This individual has a history also of significant oral production of filaments (primarily in the upper oral cavity) accompanied by severe and protracted dental pain and damage to the upper teeth. Outward manifestation of skin-based filaments or skin lesions have never been significant issues with that individual.

It appears at this time that the change in the blood condition of the control individual shown above is due primarily to two main factors over a period of several years:

1. The application of the results of the extensive research results that are inherent within this site.
2. The removal of all upper teeth of the individual.

The control individual remains able to produce oral filaments, but the blood and the general health of the individual appears to have significantly improved over this same period. The chronic and severe dental pains have been eliminated at the expense of removal of the teeth. It remains of interest why the upper teeth were the primary source of injury and why they have been the primary source for oral filament production. The identification of all markers of the “Morgellons” condition and their relative importance remains a subject of much worthy discussion and research. It has long been a claim by this researcher that the state of the blood appears to be a primary factor in the evaluation of the condition along with the existence of filament forms internal to the body and their extent of distribution.

MORGELLONS SKIN FILAMENT MICROSCOPY

(Original magnification of all images 18,000x)

The images above represent another breakthrough in the analysis of the external skin filaments that are associated with the Morgellons conditions. The majority of the images are captured using oil immersion techniques in combination with a digital-optical modified microscope. This set of images are the most detailed to date that are known and they show a plethora of internal structural forms. A more reliable measurement of the “bacterial-like” (i.e., chlamydia-like) structure has now been acquired with the use of the advanced techniques. This measurement is on the order of 300 nanometers, and thus the world of nanotechnology is now within the domain of Institute research. It is now clear that both the internal sub-filament structure and the bacterial-like forms are both on the order of 300 nanometers in diameter or width. This measurement is within the range of the larger viruses and of the smallest bacteria. A fair amount of effort is required to acquire the imagery shown.

It will be found that the shape, size, geometry, chemistry, and infra-red spectral response is identical for both the 300 nanometer structure within the blood and the 300 nanometer structure within the exterior skin filament. Ultimately it will be understood that the structure is also identical to that within the “environmental filament” so extensively studied by the Institute in the past. They are all of one and the same cloth, and at some point it will be equally understood and accepted that “Morgellons” does indeed have an environmental source for its existence.

The photos above show a great deal of detail with respect to the internal filament structure encased within the exterior filament housing, the sub-micron structures, the pleomorphism quality, aerosolization of the filaments at the filament boundary, and

numerous budding and generating structures that are at the heart of its growth process. Detailed examination will show that these same forms and processes occur within the blood of those affected by the so-called "Morgellons" condition and that this conclusion can be documented and replicated in a controlled environment.

There is a wealth of discussion that could take place with the photographs shown above. There is a strong and clear lineage of research over many years that leads us to these consolidated images of yet another examination of the blood and the impact of the Morgellon's condition upon the blood. The thesis of the blood condition as a primary indicator for the existence of the Morgellon's condition remains. The evidence supporting the broad display of these effects by much of the general population remains in place. The means and methods may improve slowly over time, but the general conclusions of harm have been reached some time ago. My time and opportunity constraints force me to leave this extended discussion for a later date, as another paper in progress for more than a year demands its conclusion. The need for a honest and thorough investigation, the call for full disclosure, and the dedication of resources to bring about an end to this suffering remains in place.

Sincerely,
Clifford E Carnicom
(Born Clifford Bruce Stewart Jan 19 1953)

Morgellons : A Working Hypothesis (Introduction)

carnicominstitute.org/morgellons-a-working-hypothesis-introduction/

Morgellons : A Working Hypothesis
Neural, Thyroid, Liver, Oxygen, Protein and Iron Disruption

Clifford E Carnicom
December 18 2013

Note: I am not offering any medical advice or diagnosis with the presentation of this information. I am acting solely as an independent researcher providing the results of extended observation and analysis of unusual biological conditions that are evident. Each individual must work with their own health professional to establish any appropriate course of action and any health related comments in this paper are solely for informational purposes and they are from my own perspective.

ABSTRACT

This paper seeks to identify a host of organic compounds that are likely to comprise the core physical structure of biologically produced filaments characteristic of the *Morgellons* condition. A biological oral filament sample will be analyzed for the presence of candidate organic functional groups using the methods of infrared spectrophotometry. Potential health impacts from these same core structures are examined and compared to the observed , reported and documented symptoms (in part) of this same condition. Potential mitigating strategies, from a research perspective only, are discussed.

A body of evidence, accumulated over a period of several years, reveals that the Morgellons condition is likely characterized by a host of serious physiological and metabolic imbalances. These imbalances are caused by the disruption of a variety of major body processes including, as a minimum, the regulation of metabolism by the thyroid, potential liver enlargement, a decrease of oxygen in the circulatory system, the utilization of amino acids important to the body, the oxidation of iron and a

potential impact to neural pathways. The impact of this degradation to human health can be concluded to be serious, debilitating and potentially lethal in the cumulative sense; the reports of those who suffer from the condition are in alignment with these conclusions. This paper will summarize the body of work and chronology which leads to this more comprehensive hypothesis.

The health, medical and governmental communities will again be invited to offer their expertise and contributions , as well as to assume their role of responsibility and the obligations of their professions to serve the public.

PART I
IDENTIFICATION

PART II
POTENTIAL HEALTH IMPACTS OF THE VARIOUS FUNCTIONAL GROUPS & COMPONENTS

PART III
POTENTIAL MITIGATING STRATEGIES (RESEARCH BASED)

Morgellons : A Working Hypothesis (Part I: Identification)

carnicominstitute.org/morgellons-a-working-hypothesis-part-i-identification/

Morgellons : A Working Hypothesis
Neural, Thyroid, Liver, Oxygen, Protein and Iron Disruption
(Link to Parts I, II, III – Click Here)

PART I
IDENTIFICATION

Clifford E Carnicom
Dec 18 2013

Art work courtesy of David Dees with permission.

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This paper seeks to identify a host of organic compounds that are likely to comprise the core physical structure of biologically produced filaments characteristic of the *Morgellons* condition. A biological oral filament sample will be analyzed for the presence of candidate organic functional groups using the methods of infrared spectrophotometry. Potential health impacts from these same core structures are examined and compared to the observed , reported and documented symptoms (in part) of this same condition. Potential mitigating strategies, from a research perspective only, are discussed.

A body of evidence, accumulated over a period of several years, reveals that the Morgellons condition is likely characterized by a host of serious physiological and metabolic imbalances. These imbalances are caused by the disruption of a variety of major body processes including, as a minimum, the regulation of metabolism by the thyroid, potential liver enlargement, a decrease of oxygen in the circulatory system, the utilization of amino acids important to the body, the oxidation of iron and a potential impact to neural pathways. The impact of this degradation to human health can be concluded to be serious, debilitating and potentially lethal in the cumulative sense; the reports of those who suffer from the condition are in alignment with these conclusions. This paper will summarize the body of work and chronology which leads to this more comprehensive hypothesis.

The health, medical and governmental communities will again be invited to offer their expertise and contributions , as well as to assume their role of responsibility and the obligations of their professions to serve the public.

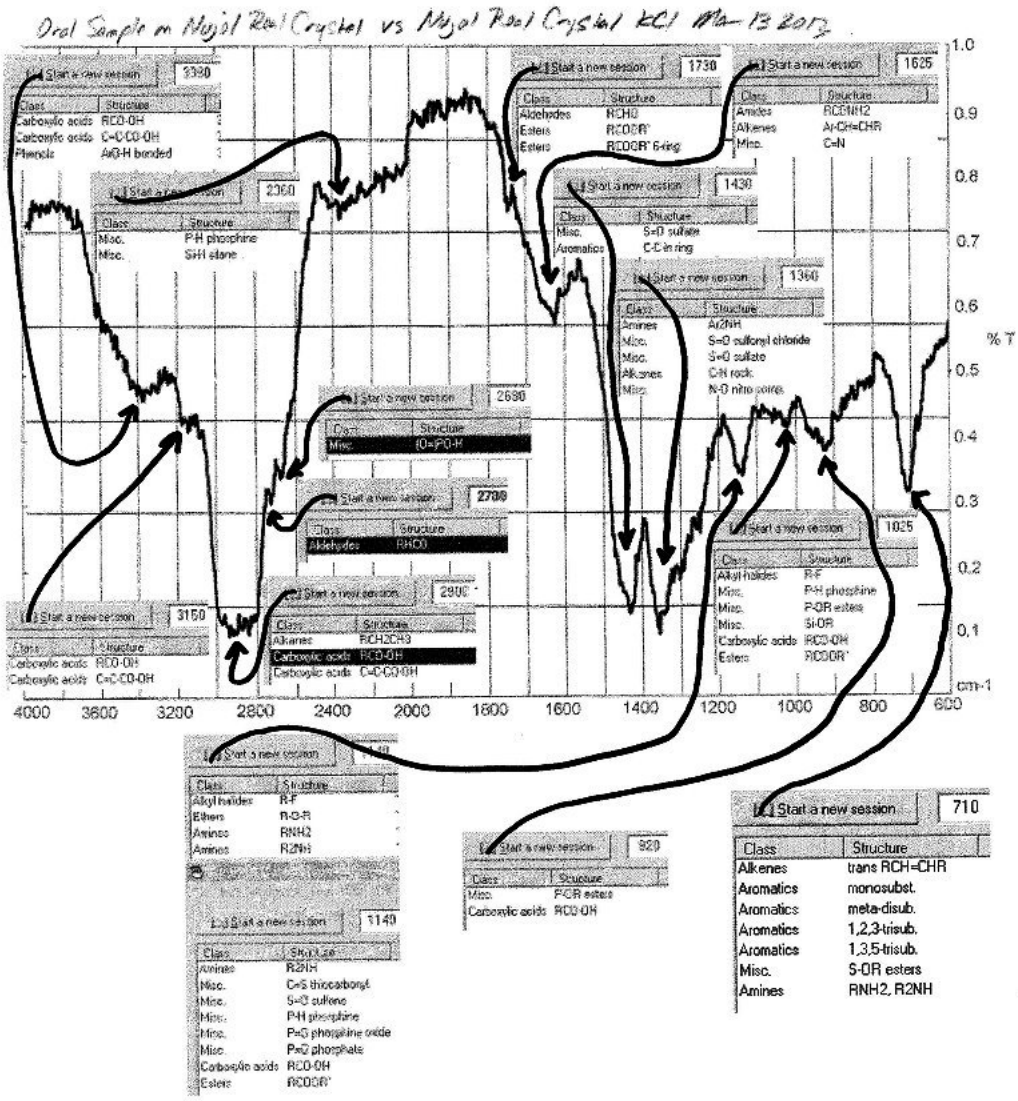
This paper will be divided into three parts:

I. Identification of the functional groups / components

II. Potential health impacts of the various functional groups identified.

III. Potential mitigating strategies (research-based)

PART I
IDENTIFICATION



The infra-red spectrum of the *Morgellons* oral filament sample in Nujol (mineral oil) on a KCl Real Crystal(TM) card and the interpretation/annotation (working notes) of its fundamental molecular and chemical composition.

It is now understood, to a relatively high level of confidence, the essential molecular and chemical composition of the *Morgellons* biological filamentous material. This knowledge is a prerequisite to understanding at least a portion of the impact to the body and human health. It now appears, from all available research, that this determined molecular composition can be summarized in the following complex phrase:

The structure of the filament form appears to be, based upon the best available information to date, primarily that of an "polycyclic organo-metallic halogenated aromatic amine". Substantial evidence also exists for the coupling of a iron-amino acid (cysteine and histidine dipeptide complex). The implications of such a compound and structure upon human health are profound.

The recently acquired spectrum that is shown above, along with all previous research to date, will be important in supporting the conclusions that are presented here. Before we begin with the detailed analysis of this infrared spectrum, let us recall briefly what has already been established with respect to the growth of the structure.

It has been established, through rather painstaking processes over a period of several years, that primary constituents of the growth form are comprised of both iron and amino acids. The methods to achieve this have been described in detail on previous reports. The essence of impact to health has also been discussed at length, namely, that if these elements are used by the organism for its own growth then those same nutrients are being denied to the human host that supports the invasive growth. Your iron is at the core of your respiration and hence of all energy transfer within your body; proteins are the structural framework that allows your body to exist and grow. The absconding of both iron

and amino acids (i.e., proteins) from the human body is by itself of sufficient damage to warrant a full and dedicated allocation of resources to this problem; this has not happened to date. This information has, however, been very useful to develop an entire host of strategies to mitigate this damage and these have been discussed on this site. There remains much to do.

Unfortunately, the information that is now gleaned from the use of infrared spectrometry only makes the situation more serious and compelling. There is, however, great value from two standpoints with our current discussion. First, a more comprehensive portrait of the actual structure of the growth form is now established. This is an absolute necessity to understand the expected impacts upon human health, and this problem remains unfinished until the full complement of investigative resources, equipment and personnel are aptly dedicated to this problem, i.e, the "Morgellons" problem. Second, and of even greater importance, is that the primary ***mechanisms*** of compromise and damage to human health are now identifiable to a greater extent. Armed with this knowledge, there is every reason to think that more effective strategies of alleviating suffering and improving health are at hand. This has been and remains a primary pursuit of this researcher. The health and medical communities are required to assume their role to evaluate the veracity of this information and to implement any potential benefits that might result from this work.

We now transition to the powers of infrared spectrophotometry, and what it can teach us about the current situation. To begin that process, let us devote a few words to the generalities of spectroscopy. One could easily devote a career to the study of this discipline alone; the history, the literature and the science itself is detailed and extensive. This speaks of the utility, value and importance of the methods. I will make no claim to being an expert in the field but I have applied myself in this, as well as dozens of other disciplines, to get certain questions answered in the face of urgency and need. An understanding of at least the basic science is in order.

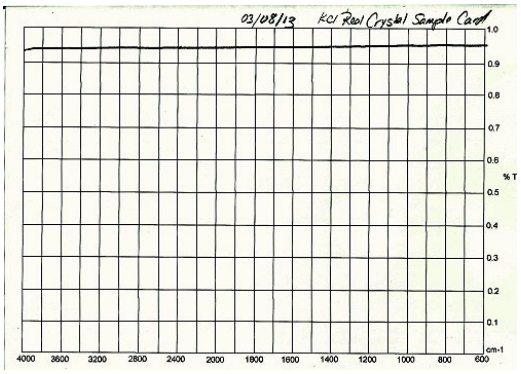
Spectrometry, in general, is the response of matter to electromagnetic energy. Spectrophotometry, in particular, is the reaction of matter to light waves, i.e., a specific and limited window within the electromagnetic spectrum. Furthermore, this light energy can be broken down into ultraviolet, visible and infrared light sections. When matter is subjected to the energy of the light source, it gets excited or vibrates. Depending upon the portion of the spectrum involved (i.e., visible, UV, IR) this excitement or vibration occurs in different forms. Carnicom Institute now owns both a modern visible light spectrophotometer and an infrared spectrophotometer (albeit aged but functional) and the public is to be commended for that accomplishment. Our focus within this paper is specifically infrared spectrophotometry (IR).

Infrared spectroscopy is an absolute core and stalwart of biochemical study for the following simple reason : it can be used to identify ***organic*** molecules, i.e., the stuff of life (natural or engineered, for that matter..). Visible light spectroscopy is useful if whatever you are looking at has color; in practice it will be found that this has serious limitations. The majority of organic molecules are transparent and have no color; you can not see them with your eye. This correspondingly makes the process of identification inherently difficult. What happens in the infrared spectrum is that molecules vibrate in characteristic ways that are known, understood and catalogued and this is helpful in identifying what are called "functional groups" in the discipline of biochemistry. Functional groups are combinations of molecules within biology that have identifiable characteristics and behavior. As has been mentioned, infrared spectrometry can be a lifelong pursuit of study in its own right; there is no magic single button that gives one a printout of what something is made of. The "building blocks" of a biological structure can be identified with the use of IR, but it is unrealistic to expect complete and total knowledge of detailed molecular composition. There are databases built into modern equipment that can radically accelerate the problems and details of IR interpretation, but even these will not address many of the problems at hand. This is especially the case if we are dealing with unknown, newly synthetic or engineered substances or complexes. There is both art and science in the practice of IR spectroscopy and those expert in the field are to be commended for their own dedication to the subject. The work that I offer here will, hopefully, point us in the right direction and allow us to anticipate what we are trying to see at the end of the tunnel. There are a host of other technologies (including additional spectroscopy methods) and instrumentation which *could* give us the level of knowledge and detail that is to our benefit and need; Carnicom Institute has no such access to what is truly needed at this time.

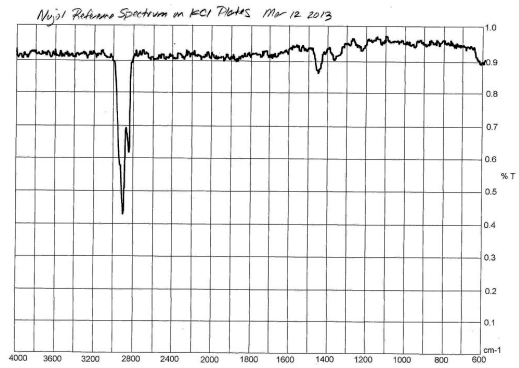
Now we must dig a little deeper, and into the thick of it. For those willing to expand your study of the discipline I encourage you to do so. For those that have the knowledge already, it is time to bring our best foot forward and make the analysis. It is time to study the spectrum presented above.

The instrument in use is a Perkin-Elmer 1320 Infrared Spectrophotometer, a dual beam dispersive IR instrument. The sample substrate being used is that potassium chloride (KCl) in an International Crystal Laboratories Real Crystal IR Sample Card (TM). Considerable time has been spent with study of numerous reference spectra and the oral sample spectrum using a polyethylene substrate; the advantages of uniform transparency to IR with KCl are immediately apparent as they have recently become available. KCl is highly preferred in many respects as, for example, it is free from the interference of Nujol and polyethylene absorbances and the transmittance in the IR spectrum is also extremely high and uniform across the range of 4000 to 600 cm^{-1}. This substrate form is also unique in that it will handle a certain level of water in the preparation of the sample on the salt crystal.

Reference spectra of KCL and Nujol on KCl plates to address any questions of interference in the spectral interpretation:



Example of the uniform transmittance of the reference KCl crystal as a sample substrate; an ideal IR material without interference absorption.



Reference IR spectrum of Nujol (mineral oil) on KCL plates. Nujol is a useful substrate for solid samples. Notice the only significant interference absorption peaks will be at approximately 2900 cm-1 and 2850 cm-1.

In the case here, the oral sample is collected and thoroughly rinsed and the moisture evaporated from the sample. The sample is then ground to a fine powder with mortar and pestle and mixed with Nujol (mineral oil) to a uniform consistency. A drop of the compound is then placed on a KCl Real Crystal (TM) along with a KCl cover slip. Any and all moisture must be completely driven from the sample before proceeding to avoid contamination of the spectrum with water; this is accomplished by evaporating the sample under mild heat to completion. The tools of analysis and cross-referencing applied to the interpretation of the absorption peaks will be as follows:

1. Infrared Absorption Spectroscopy – Practical, Koji Nakanishi.[1]

2. Reprint of Colthrup Chart of Characteristic Group Absorptions in Modern Methods of Chemical Analysis, Pecsok.[2]

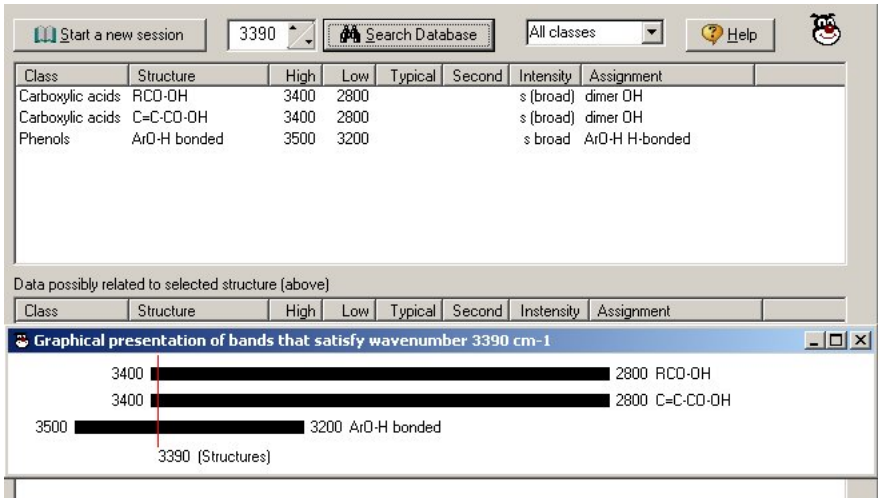
3. IR Pal Software 2.0, , A Table Driven Infrared Application, Dr. Wolf van Heeswjik.[3]

4. Spectral Database for Organic Compounds (SDBS), National Association of Advanced Industrial Science and Technology (AIST), Japan.[4]

The initial method of analysis will focus on the use of IR Pal and correlated Nakanishi. The work will progress through a series of iterations: the first stage will identify candidate functional groups, a second stage will examine the candidates from a geometric-graphical perspective, and a third stage will examine cross-correlations between the candidate functional groups. The final stage will form from a composite of all three approaches, as well as integration of knowledge gained from previous research. The end goal will be to create a more comprehensive assessment of the expected structural-chemical composition of the growth form. A leading discussion into potential health implications will be initiated along with the call for continued research under urgent conditions.

The Candidate Stage:

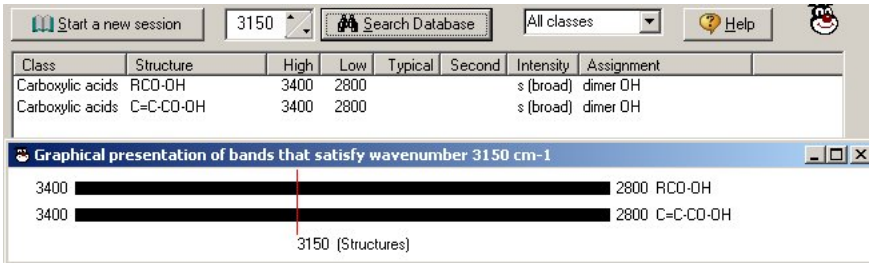
The first absorbance peak, occurring in the functional group region, is at approximately 3390 cm^{-1}. The 3390 cm^{-1} absorption peak (transmittance minimum) leads us to the following candidates:



IR Pal : 3390 cm^{-1} candidates

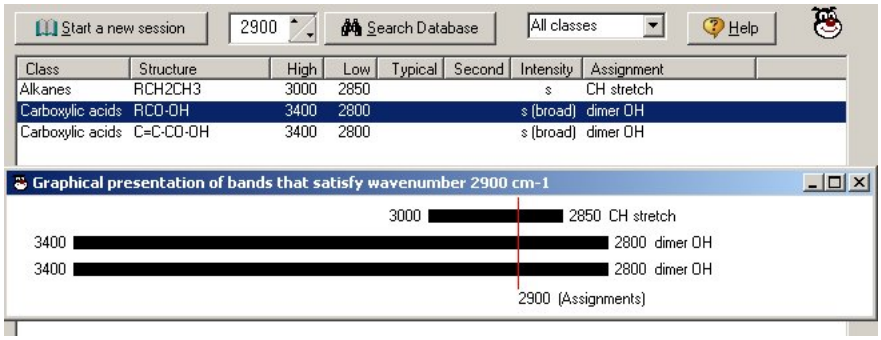
The functional group candidates here will therefore be carboxylic acids and aromatic phenols.

Our next absorption peak is at approx. 3150 cm^{-1}. The candidates are:



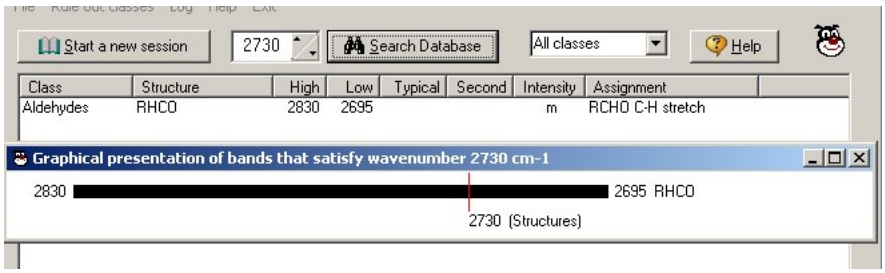
The candidates list is restricted to carboxylic acid functional groups in this case.

Next we have a major absorption peak in the range of 2850-2950 cm^{-1}. The candidate list is:



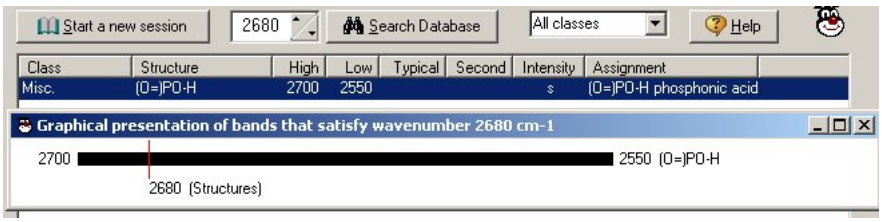
Our list of functional groups at the broad absorption peak includes alkanes and carboxylic acids.

Our next absorption peak is a minor peak at 2730 cm^{-1}:



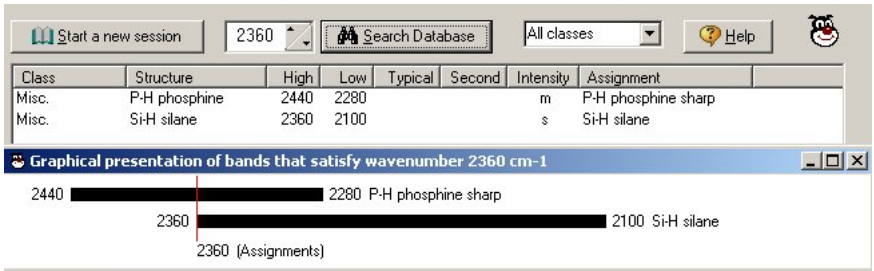
This introduces an aldehyde functional group for consideration.

Another minor peak absorption at approximately 2680 cm^{-1}:

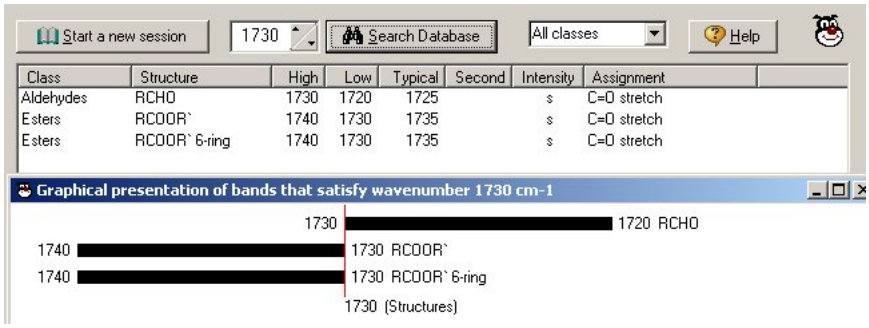


which leads to the consideration of a phosphoric acid group.

Our next absorption peak (i.e., transmittance minimum) is at approximately 2360 cm-1:

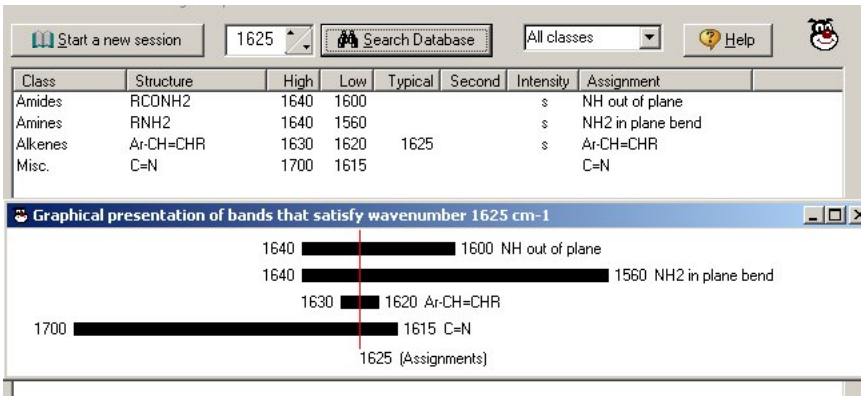


This now includes consideration of the miscellaneous categories of a phosphine and a silane. Next we see a minor absorption peak at approximately 1730 cm^{-1}:



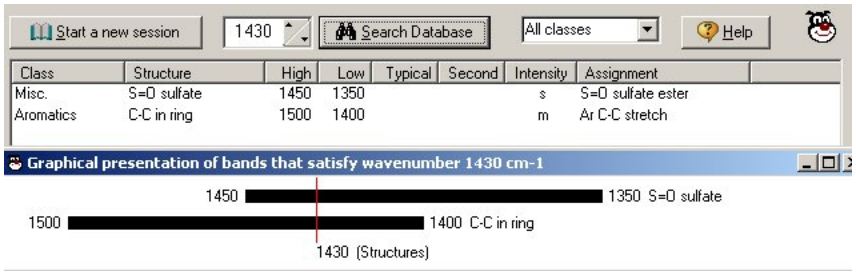
This minor peak introduces the consideration of both aldehydes and esters. We also note the reference to the 6-ring structure, i.e., the possibility of aromatic structure appearance.

Our next absorption peak is at approximately 1625 cm^{-1}:



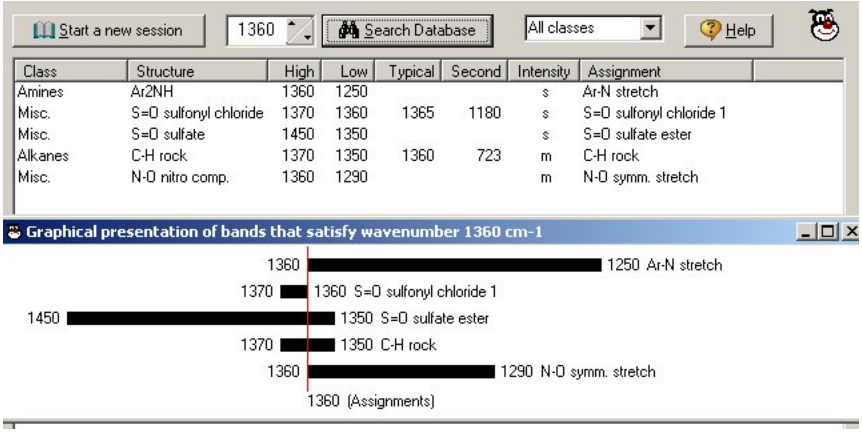
This search lists amides, amines, alkenes (notice aromatic reference again) and a C=N double bond structure.

The next absorption peak is at 1430 cm^{-1}:



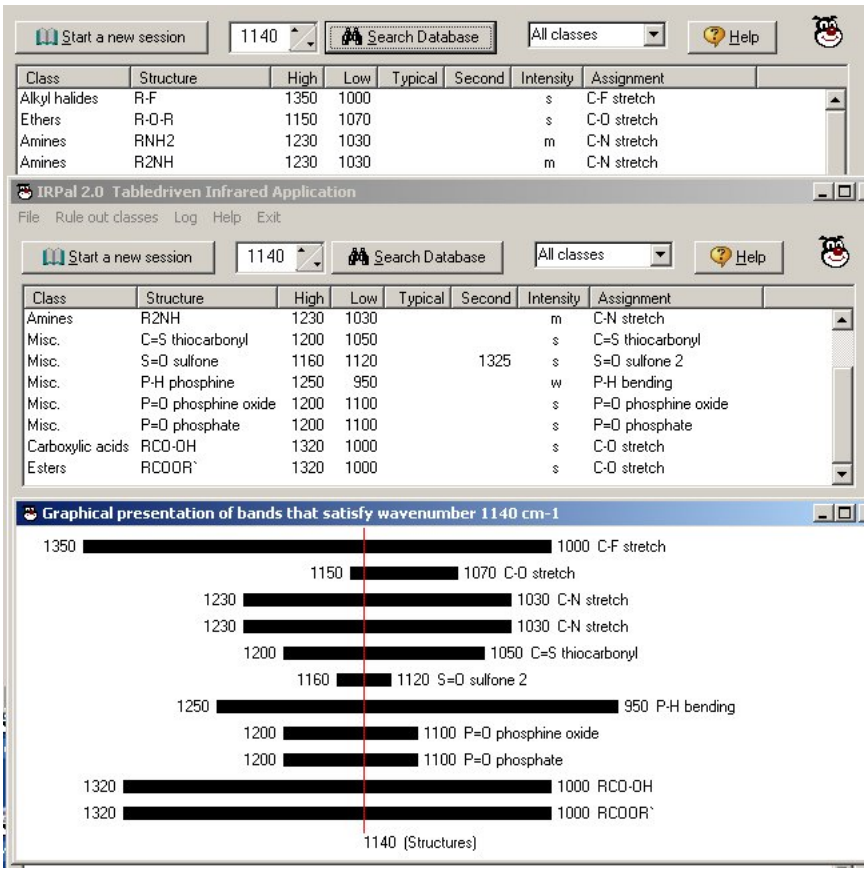
Our candidates here are a sulfate ester and, once again, an aromatic structure.

The next absorption peak is at approximately 1360 cm^{-1}:



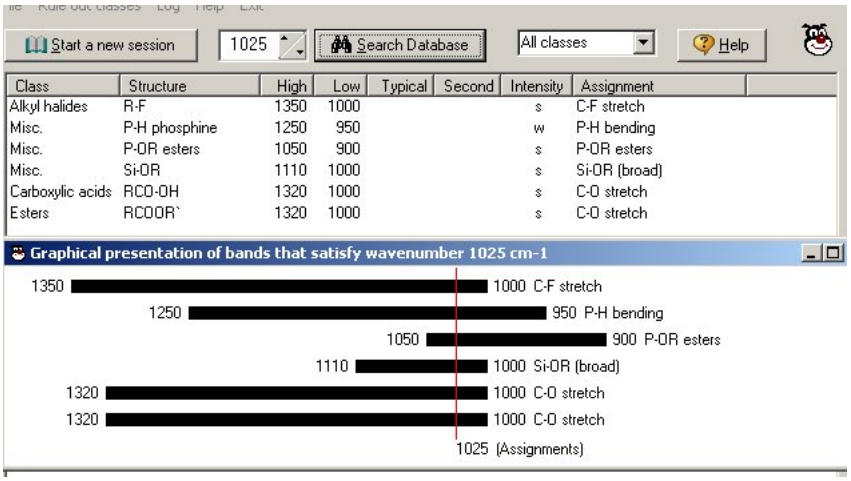
Here we have the following entries: an aromatic amine, sulfonyl chloride, sulfate ester, an alkane group and an N-O (nitrogen oxygen single bond).

The next absorption peak is at 1140 cm^{-1}:



Numerous candidate groups appear here: alkyl halides, ethers, amines, thiocarbonyl, sulfone, phospine, phosphine oxide, phosphate, carboxylic acids and esters.

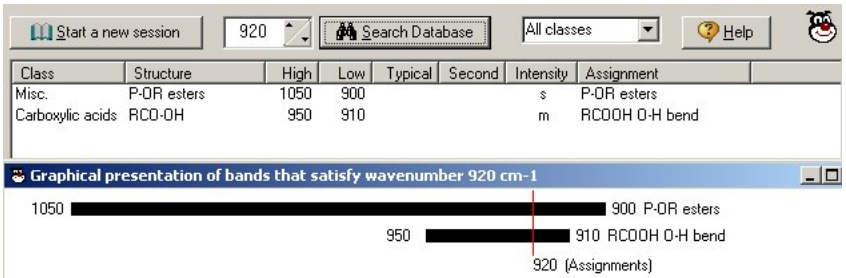
The next absorption peak (minor) is at approximately 1025 cm^{-1}:



Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P-OR esters	1050	900			s	P-OR esters
Misc.	Si-OR	1110	1000			s	Si-OR (broad)
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch
Esters	RCOOR`	1320	1000			s	C-O stretch

Here we have a listing of alkyl halides, phosphine, P-OR esters, Si-OR, carboxylic acids and esters.

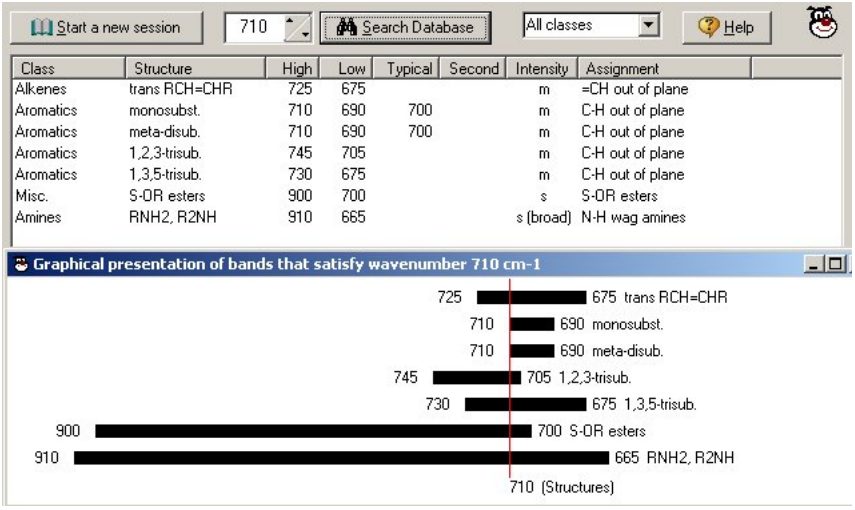
Our next absorption peak is at approximately 920 cm^{-1}:



Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Misc.	P-OR esters	1050	900			s	P-OR esters
Carboxylic acids	RCO-OH	950	910			m	RCOOH O-H bend

This set includes a P-OR ester and a carboxylic acid group.

Lastly, we have a strong absorption peak at approximately 710 cm-1:



Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters
Amines	RNH2, R2NH	910	665			s (broad)	N-H wag amines

This final candidate list shows an alkene, a strong presence of aromatics, a S-OR ester and an amine.

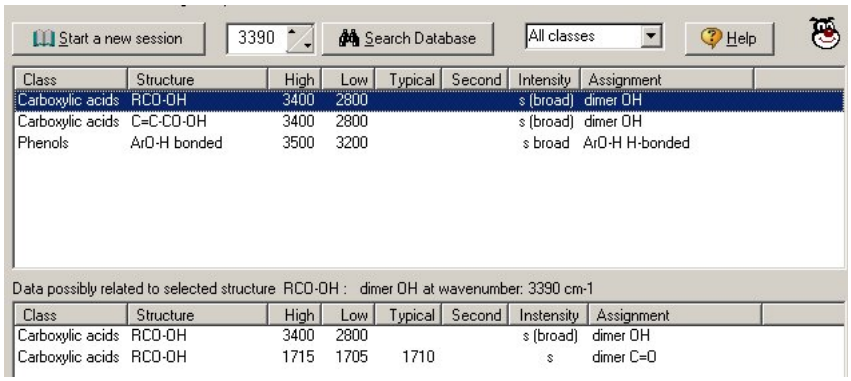
We are still at the early stage of analysis of the spectrum. It is of interest, however, even at this early stage to identify groups or combinations that are showing an increased relative frequency within the tabular listings. It will be found that such groups, terms or combinations such as:

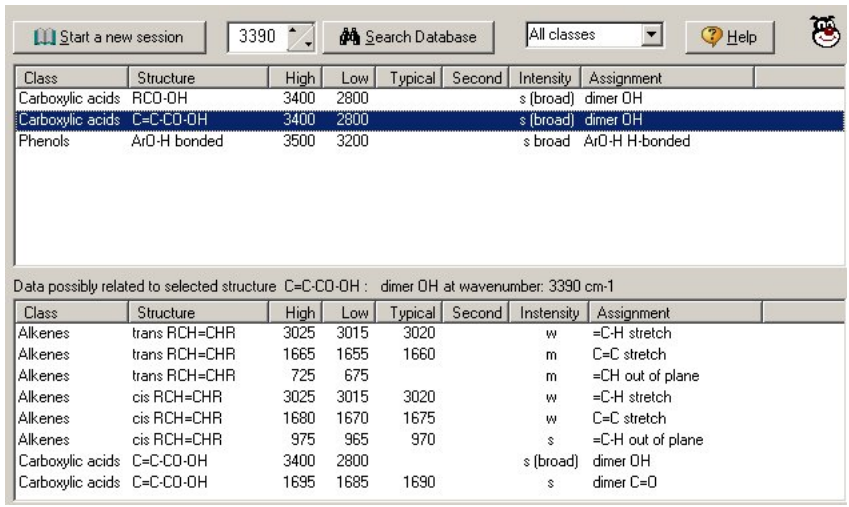
carboxylic acids
aromatics
amines
alkyl halides
esters
sulfur (or derivatives)
phosphorus (or derivatives)

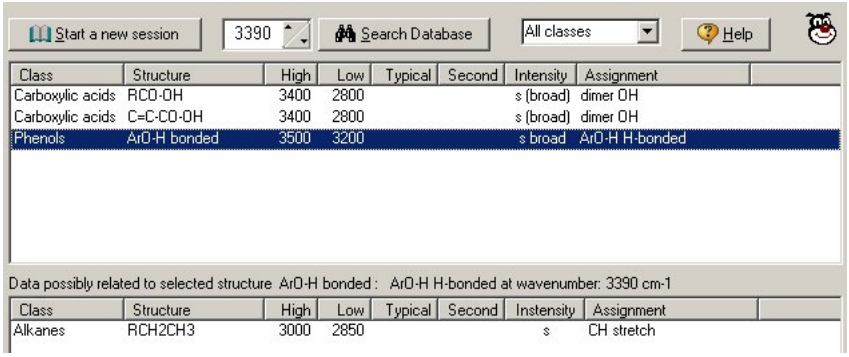
are present at a relatively increased level. We will keep these terms in mind, along with others, as we continue in the analysis below:

Our next phase of work is to begin screening, or filtering , the data that we have to work with as it can be a bit daunting at this stage of collection. We must be careful in this process, however, not to lose critical data along the way. The approach taken will be to look at cross-correlations in the data and to look for some of the patterns that may be stronger than others within the data set. Correlations identified will tend to strengthen the case for the existence of the group; they will be identified as weak, moderate or strong respectively. IR Pal infrared tabular software excels at this approach, and is to be commended as highly valuable software for assisting in infrared spectral analysis. Let us begin, and once again take each candidate absorption peak individually.

Begin with the absorption peak at 3390 cm^{-1}.







With the first carboxylic acid group, correlations are suggested at approximately 1710 cm^{-1}. Our closest peak here is at 1730 cm^{-1}. The 1730 cm^{-1} peak is also listed as strong. This correlation would appear reasonably weak at this point, and this rating will be assigned tentatively at this time.

The second carboxyl acid group shows a possible relation at 1690 cm^{-1}, also listed as a strong peak. This correlation is also determined to be weak at this time. This same carboxylic acid shows correlations with alkenes at 3020, 1660, 725-675, 1675, 970 cm^{-1} respectively. Of this set, the 725-675 cm^{-1} of medium strength is of strongest interest, and will be assigned a strong rating. The remaining peaks do not indicate correlation with the 3390 cm^{-1} absorption peak.

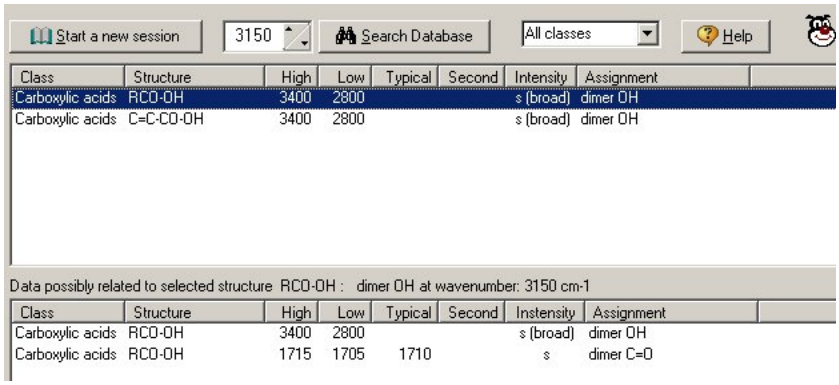
The phenol group indicates a correlation with an alkane expected at 3000-2850 cm-1. This will be rated as a strong correlation because of the observed broad and strong absorption peak at approximately 2950-2850 cm^{-1}.

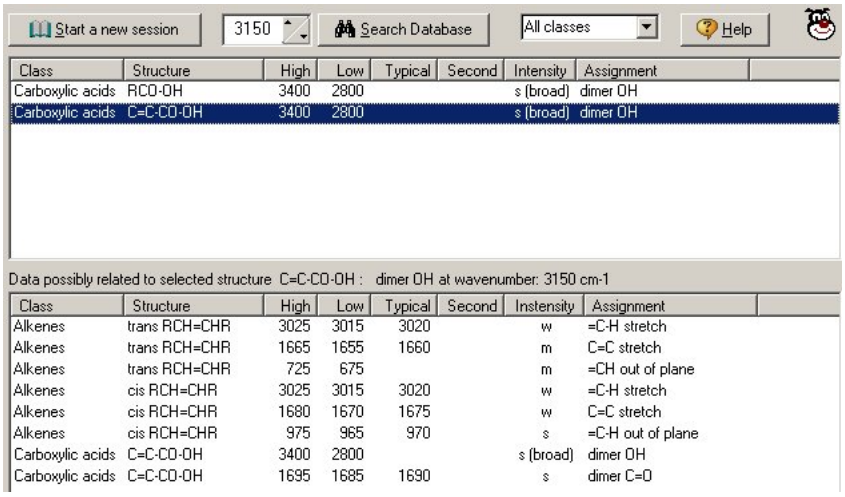
Correlation Summary : 3390 cm^{-1}:

Strong :
Carboxylic Acid > Alkene (725-675)
Phenol > Alkane (3000-2850)

Weak:
Carboxylic Acid > Carboxylic Acid (1730)
Carboxylic Acid > Carboxylic Acid (1690)

Our next correlation search is at 3150 cm^{-1}:





It will be seen that the discussion for the carboxylic acid group here is identical to that discussed for the case at 3390 cm-1 so the appropriate correlations are listed below:

Correlation Summary : 3150 cm^{-1}:

Strong :
Carboxylic Acid > Alkene (725-675)

Weak:
Carboxylic Acid > Carboxylic Acid (1730)
Carboxylic Acid > Carboxylic Acid (1690)

Our next correlation search is at 2850-2950 cm^{-1}:

Start a new session | 2900 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkanes	RCH2CH3	3000	2850			s	CH stretch
Carboxylic acids	RCO-OH	3400	2800			s (broad)	dimer OH
Carboxylic acids	C=C-CO-OH	3400	2800			s (broad)	dimer OH

Data possibly related to selected structure RCH2CH3 : CH stretch at wavenumber: 2900 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Alkanes	RCH2CH3	3000	2850			s	CH stretch
Alkanes	RCH2CH3	1470	1450	1460		s	CH2 and CH3
Alkanes	RCH2CH3	1380	1370	1375		s	CH2 and CH3
Alkanes	RCH2CH3	725	720	722		m	bending modes
Alkanes	C-H rock	1370	1350	1360	723	m	C-H rock
Alkanes	C-H rock	725	720	722	1360	m	C-H rock

The correlations of interest from the initial alkane group will be all subsidiary alkane groups shown, at moderate to strong levels. Continuing with the second carboxylic acid group:

Start a new session | 2900 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkanes	RCH2CH3	3000	2850			s	CH stretch
Carboxylic acids	RCO-OH	3400	2800			s (broad)	dimer OH
Carboxylic acids	C=C-CO-OH	3400	2800			s (broad)	dimer OH

Data possibly related to selected structure RCO-OH : dimer OH at wavenumber: 2900 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Carboxylic acids	RCO-OH	3400	2800			s (broad)	dimer OH
Carboxylic acids	RCO-OH	1715	1705	1710		s	dimer C=O

A weak relationship may or may not exist with the carboxylic group at 1710 cm^{-1}.

And lastly,

Start a new session | 2900 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkanes	RCH2CH3	3000	2850			s	CH stretch
Carboxylic acids	RCO-OH	3400	2800			s (broad)	dimer OH
Carboxylic acids	C=C-CO-OH	3400	2800			s (broad)	dimer OH

Data possibly related to selected structure C=C-CO-OH : dimer OH at wavenumber: 2900 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Alkenes	trans RCH=CHR	3025	3015	3020		w	=C-H stretch
Alkenes	trans RCH=CHR	1665	1655	1660		m	C=C stretch
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Alkenes	cis RCH=CHR	3025	3015	3020		w	=C-H stretch
Alkenes	cis RCH=CHR	1680	1670	1675		w	C=C stretch
Alkenes	cis RCH=CHR	975	965	970		s	=C-H out of plane
Carboxylic acids	C=C-CO-OH	3400	2800			s (broad)	dimer OH
Carboxylic acids	C=C-CO-OH	1695	1685	1690		s	dimer C=O

Correlations of the carboxylic acid group with the alkene at 1660 cm^{-1} is rated as weak. The correlation at 725-625 cm^{-1} is strong. The correlation at 1675 cm^{-1} is weak. The correlation in the range from 3400 – 2800 cm^{-1} is strong, and the correlation at 1690 cm^{-1} is weak to marginal.

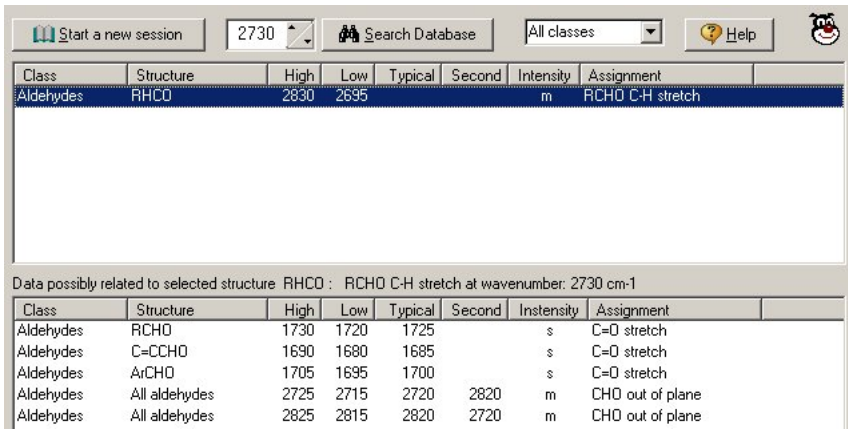
Correlation Summary : 2950-2850 cm^{-1}:

Strong :
Alkane > Alkane (722, 1360)
Carboxylic Acid > Alkene (725-675)
Carboxylic Acid > Carboxylic Acid (3400 – 2800)

Moderate :
Alkane > Alkane (1375)

Weak:
Alkane > Alkane (1460, 1375)
Carboxylic Acid > Carboxylic Acid (1710, 1690)
Carboxylic Acid > Alkene (1710, 1675)

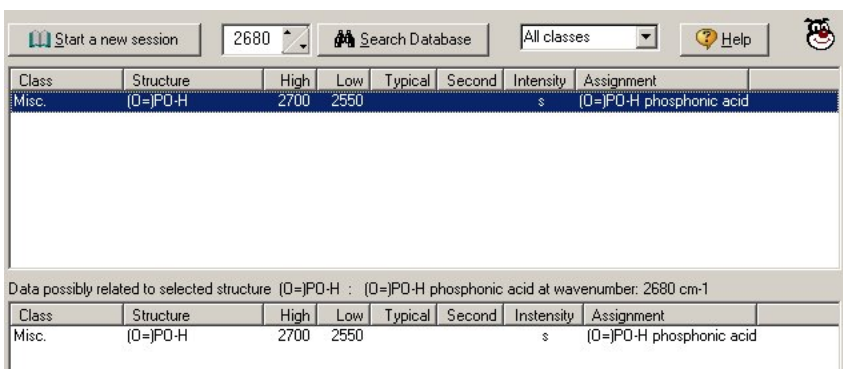
Our next correlation search is at 2730 cm^{-1}:



The correlation of the aldehyde candidate with the potentially associated aldehydes at 1725 cm^{-1} and 2820 cm^{-1} are rated as strong. In summary:

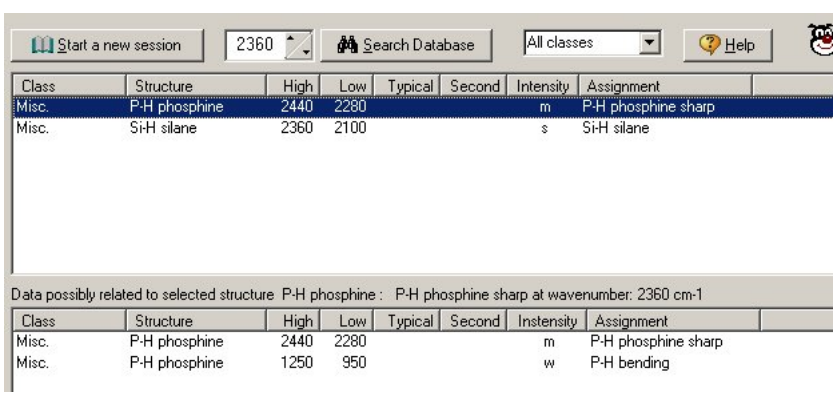
Strong :
Aldehyde > Aldehyde (1725, 2820)

Our next correlation search is at 2680 cm^{-1}:

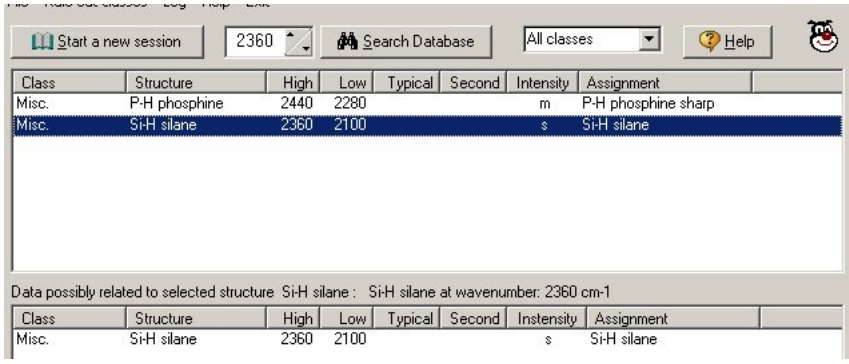


No correlations are identified with the candidate phosphoric acid group.

Our next correlation search is at 2360 cm^{-1}:

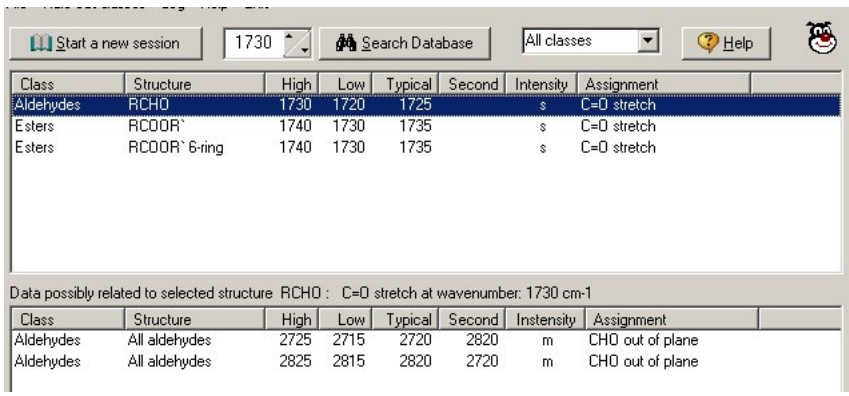


The phosphine candidate has a weak prospective correlation with the phospine group ranging from 1250 to 950 cm^{-1}, based upon the observed absorption peaks at 1140 cm^{-1} and 920 cm^{-1}. Notice the character of these latter peaks is listed as weak, however, and this is especially questionable with regard to the observed peak at 1140 cm^{-1}.

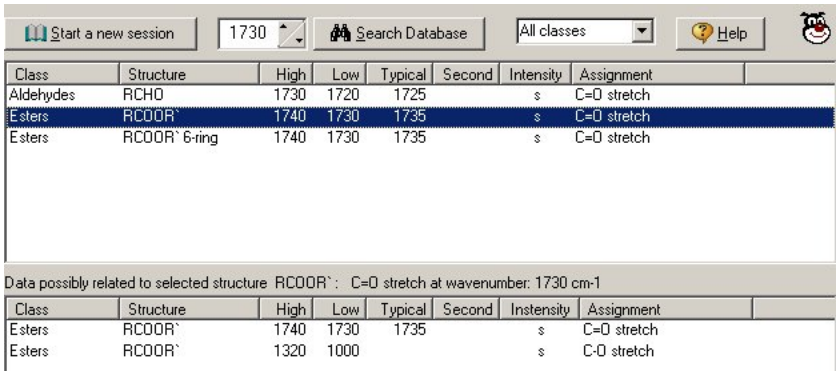


No additional correlation opportunities are listed for the silane candidate.

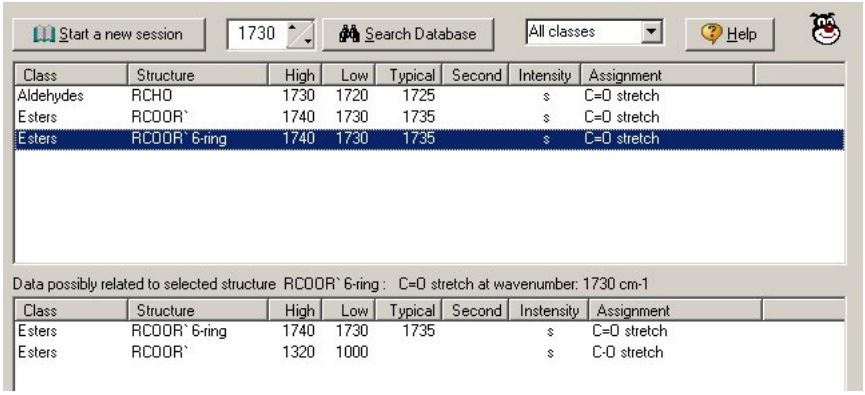
The next search is at 1730 cm^{-1}:



The first aldehyde group has potential correlation to the aldehydes at 2720 cm-1 and 2820 cm^{-1}, rated as strong.



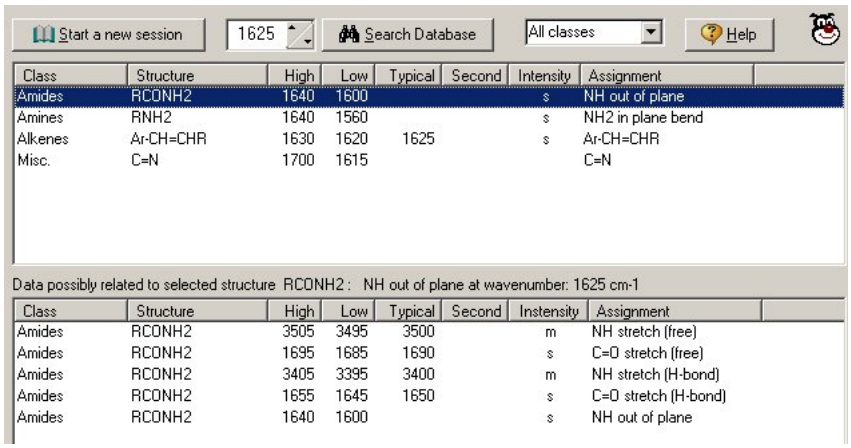
The ester group at 1735 cm^{-1} has potential correlation to the strong ester peak between 1320 cm^{-1} and 1000 cm^{-1}, rated as moderate at this point with consideration of the sharp observed peak at 1140 cm-1.



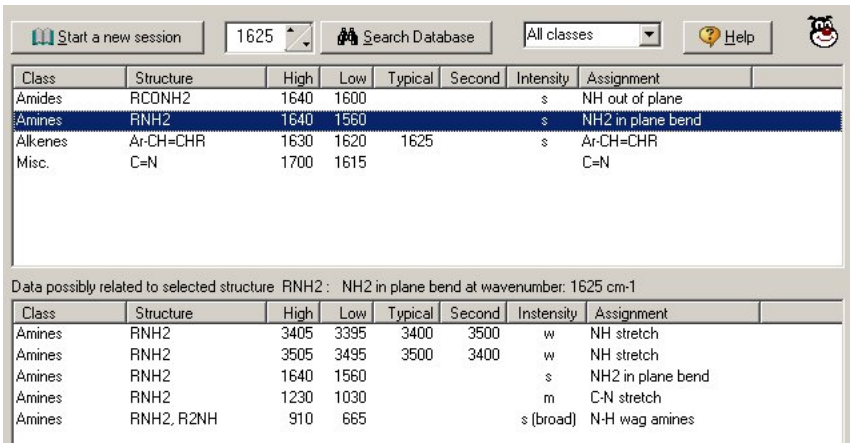
The second ester group at 1735 cm-1 has the same relationship as the prior case. We do note, however, that this ester is a 6-ring structure. This is to be kept in mind with respect to any aromatic structural discovery. In summary:

Moderate :
Ester > Ester, Ester (6-ring) (1320-1000)

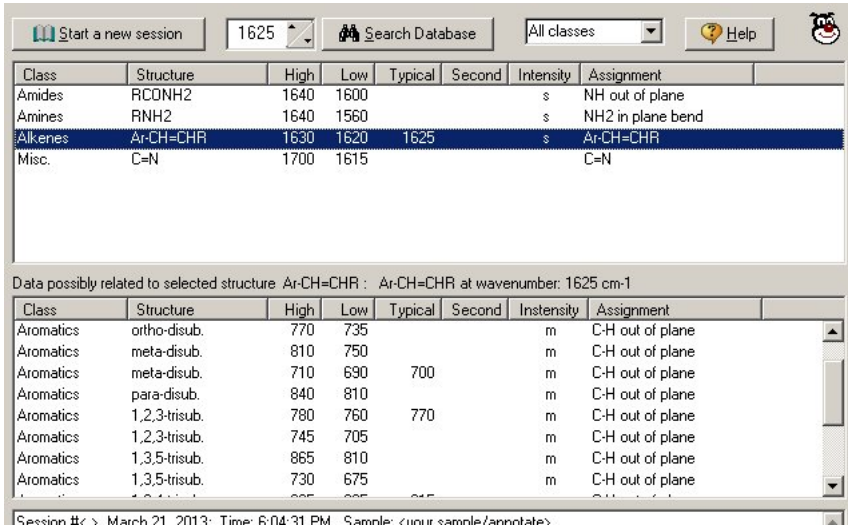
The next search is at 1625 cm^{-1}:



The amide group candidate shows no correlations of significance.



The amine group candidate shows amine correlations at 3400 cm^{-1}, 1640-1560 cm^{-1}, 1230-1030 cm^{-1} and 910-665 cm^{-1}. These correlations are rated as strong.



continuing…

Start a new session | 1625 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Amides	RCONH2	1640	1600			s	NH out of plane
Amines	RNH2	1640	1560			s	NH2 in plane bend
Alkenes	Ar-CH=CHR	1630	1620	1625		s	Ar-CH=CHR
Misc.	C=N	1700	1615				C=N

Data possibly related to selected structure Ar-CH=CHR : Ar-CH=CHR at wavenumber: 1625 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Aromatics	1,3,5-trisub.	865	810			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Aromatics	1,2,4-trisub.	825	805	815		m	C-H out of plane
Aromatics	1,2,4-trisub.	885	870	877		m	C-H out of plane
Aromatics	1,2,3,4-tetrasub.	810	800	805		m	C-H out of plane
Aromatics	1,2,4,5-tetrasub.	870	855	862		m	C-H out of plane
Aromatics	1,2,3,5-tetrasub.	850	840	845		m	C-H out of plane
Aromatics	1,2,3,4,5-pentasub.	875	865	870		w	C-H out of plane

Session #< > March 21, 2013; Time: 6:04:31 PM. Sample: <your sample/annotate>

The aromatic alkene candidate group shows correlations to the meta-disubstituted aromatic at 700 cm^{-1}, the 1,2,3 trisubstituted aromatic at 745-705 cm^{-1}, and the 1,3,5 trisubstituted aromatic at 730-675 cm^{-1}. These are rated as strong from the observed absorption peak at 710 cm^{-1}.

and lastly for this segment at 1625 cm^{-1}:

Start a new session | 1625 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Amides	RCONH2	1640	1600			s	NH out of plane
Amines	RNH2	1640	1560			s	NH2 in plane bend
Alkenes	Ar-CH=CHR	1630	1620	1625		s	Ar-CH=CHR
Misc.	C=N	1700	1615				C=N

Data possibly related to selected structure C=N : C=N at wavenumber: 1625 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	C=N	1700	1615				C=N

Here we have no correlations shown for the carbon-nitrogen double bond.

In summary:

Strong :
Amines > Amines (3400, 1640-1560, 1230-1030, and 910-665.)
Aromatic Alkene > meta distributed aromatic (700)
Aromatic Alkene > 1,2,3, trisubstituted aromatic (745-705)
Aromatic Alkene > 1, 3, 5 trisubstituted aromatic (730-675)

Next, we investigate any correlations at 1430 cm^{-1}:

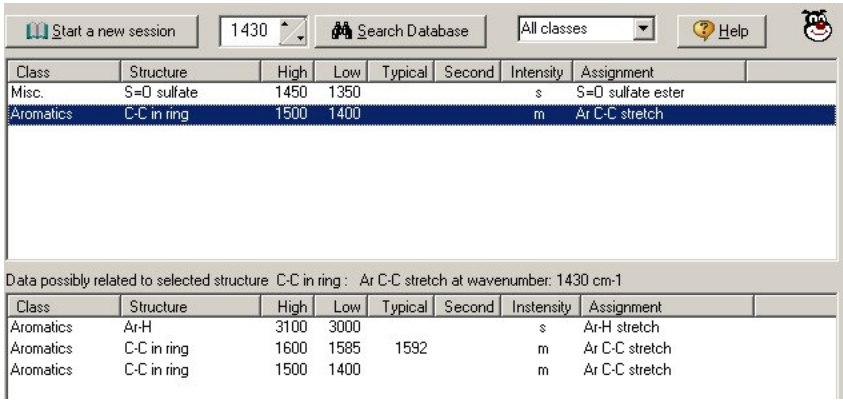
Start a new session | 1430 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester
Aromatics	C-C in ring	1500	1400			m	Ar C-C stretch

Data possibly related to selected structure S=O sulfate : S=O sulfate ester at wavenumber: 1430 cm-1

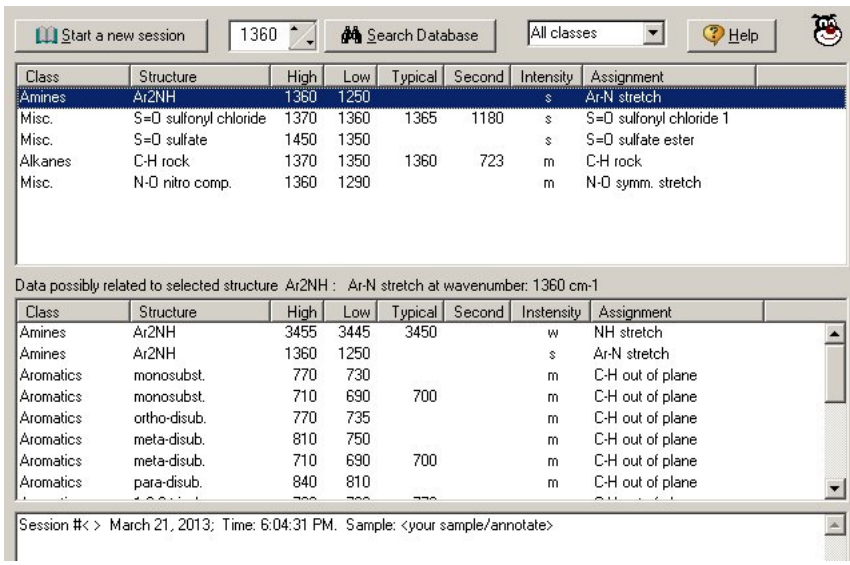
Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester

We see that no additional correlations exist for the sulfate.



The aromatic group also does not present any correlations of note.

We proceed now to correlation examination at 1360 cm^{-1}:



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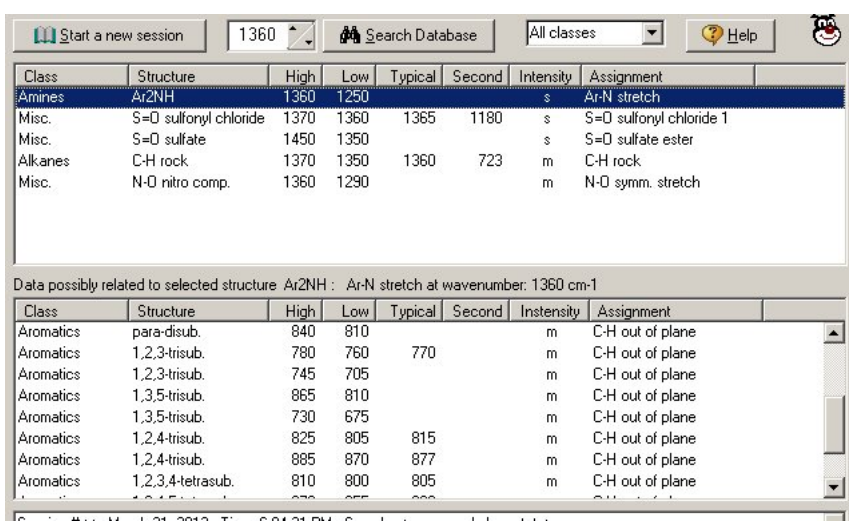

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Start a new session | 1360 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Amines	Ar2NH	1360	1250			s	Ar-N stretch
Misc.	S=O sulfonyl chloride	1370	1360	1365	1180	s	S=O sulfonyl chloride 1
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester
Alkanes	C-H rock	1370	1350	1360	723	m	C-H rock
Misc.	N-O nitro comp.	1360	1290			m	N-O symm. stretch

Data possibly related to selected structure Ar2NH : Ar-N stretch at wavenumber: 1360 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Aromatics	1,3,5-trisub.	865	810			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Aromatics	1,2,4-trisub.	825	805	815		m	C-H out of plane
Aromatics	1,2,4-trisub.	885	870	877		m	C-H out of plane
Aromatics	1,2,3,4-tetrasub.	810	800	805		m	C-H out of plane
Aromatics	1,2,4,5-tetrasub.	870	855	862		m	C-H out of plane
Aromatics	1,2,3,5-tetrasub.	850	840	845		m	C-H out of plane
Aromatics	1,2,3,4,5-pentasub.	875	865	870		w	C-H out of plane

The amine group (aromatic) shows strong correlation to the monsubstituted aromatic at 700 cm^{-1}, to the meta-disubstituted aromatic at 700 cm^{-1} and to the 1,3,5 trisubstituted aromatic at 730-675 cm^{-1}. Moderate correlation to the ortho-disubstituted aromatic exists at 770-735 cm^{-1}.

Start a new session | 1360 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Misc.	S=O sulfonyl chloride	1370	1360	1365	1180	s	S=O sulfonyl chloride 1
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester
Alkanes	C-H rock	1370	1350	1360	723	m	C-H rock
Misc.	N-O nitro comp.	1360	1290			m	N-O symm. stretch

Data possibly related to selected structure S=O sulfonyl chloride : S=O sulfonyl chloride 1 at wavenumber: 1360 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	S=O sulfonyl chloride	1370	1360	1365	1180	s	S=O sulfonyl chloride 1
Misc.	S=O sulfonyl chloride	1190	1170	1180	1365	s	S=O sulfonyl chloride 2

The sulfonyl chloride does not show any significant correlations; the observed peak at 1140 cm^{-1} is expected to be outside of the 1190-1170 cm^{-1} range and beyond the errors of observation.

Start a new session | 1360 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Misc.	S=O sulfonyl chloride	1370	1360	1365	1180	s	S=O sulfonyl chloride 1
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester
Alkanes	C-H rock	1370	1350	1360	723	m	C-H rock
Misc.	N-O nitro comp.	1360	1290			m	N-O symm. stretch

Data possibly related to selected structure S=O sulfate : S=O sulfate ester at wavenumber: 1360 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester

Similarly, the sulfate group shows no correlations present.

Start a new session | 1360 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Misc.	S=O sulfonyl chloride	1370	1360	1365	1180	s	S=O sulfonyl chloride 1
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester
Alkanes	C-H rock	1370	1350	1360	723	m	C-H rock
Misc.	N-O nitro comp.	1360	1290			m	N-O symm. stretch

Data possibly related to selected structure C-H rock : C-H rock at wavenumber: 1360 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Alkanes	RCH2CH3	3000	2850			s	CH stretch
Alkanes	RCH2CH3	1470	1450	1460		s	CH2 and CH3
Alkanes	RCH2CH3	1380	1370	1375		s	CH2 and CH3
Alkanes	RCH2CH3	725	720	722		m	bending modes
Alkanes	C-H rock	725	720	722	1360	m	C-H rock

The alkane group shows alkane correlation at 3000-2850 cm^{-1} at a strong level. The correlation at 1460 cm^{-1} is rated at the weak level. The correlations at 1375 cm^{-1} and 722 cm^{-1} are rated at the moderate level.

Start a new session | 1360 | Search Database | All classes | Help

Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Misc.	S=O sulfonyl chloride	1370	1360	1365	1180	s	S=O sulfonyl chloride 1
Misc.	S=O sulfate	1450	1350			s	S=O sulfate ester
Alkanes	C-H rock	1370	1350	1360	723	m	C-H rock
Misc.	N-O nitro comp.	1360	1290			m	N-O symm. stretch

Data possibly related to selected structure N-O nitro comp. : N-O symm. stretch at wavenumber: 1360 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	N-O nitro comp.	1545	1535	1540	1380	s	Alif. Nitro
Misc.	N-O nitro comp.	1385	1375	1380	1540	m	Alif. Nitro
Misc.	N-O nitro comp.	1525	1515	1520	1350	m,s	Arom. Nitro
Misc.	N-O nitro comp.	1355	1345	1350	1520	m,s	Arom. Nitro
Misc.	N-O nitro comp.	1550	1475			s	N-O asym. stretch
Misc.	N-O nitro comp.	1360	1290			m	N-O symm. stretch

The N-O single bond shows N-O moderate correlation at 1380 cm^{-1} and at 1350 cm^{-1}.

In summary:

Correlation Summary : 1360 cm^{-1}:

Strong :
Alkane > Alkane (3000-2850)
Amine (Aromatic) > mono-substituted Aromatic (700)
Amine (Aromatic) > meta-disubstituted Aromatic (700)
Amine (Aromatic) > 1,3,5 tri-substituted Aromatic (730-675)

Moderate :
Alkane > Alkane (1375, 722)
N-O > N-O (1380, 1350)
Amine (Aromatic) > ortho di-substituted Aromatic (730-675)

Weak:
Alkane > Alkane (1460)

The next examination is at 1140 cm-1:

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch

Data possibly related to selected structure R-F : C-F stretch at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Alkyl halides	CH2X	1300	1150			m	C-H wag (-CH2X)

The alky halide shows correlation rated as moderate at 1300-1150 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch

Data possibly related to selected structure R-O-R : C-O stretch at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Ethers	R-O-R	1150	1070			s	C-O stretch

The ether shows no correlation.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Amines	RNH2	1230	1030			m	C-N stretch
Amines	R2NH	1230	1030			m	C-N stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide

Data possibly related to selected structure RNH2 : C-N stretch at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Amines	RNH2	3405	3395	3400	3500	w	NH stretch
Amines	RNH2	3505	3495	3500	3400	w	NH stretch
Amines	RNH2	1640	1560			s	NH2 in plane bend
Amines	RNH2	1230	1030			m	C-N stretch
Amines	RNH2, R2NH	910	665			s (broad)	N-H wag amines

The first amine group shows strong correlation to amines at 3400 cm^{-1}, 1640-1560 cm^{-1}, 1230-1030 and at 910-635 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Amines	RNH2	1230	1030			m	C-N stretch
Amines	R2NH	1230	1030			m	C-N stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide

Data possibly related to selected structure R2NH : C-N stretch at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Amines	R2NH	3350	3310			w	NH stretch
Amines	R2NH	1230	1030			m	C-N stretch
Amines	RNH2, R2NH	910	665			s (broad)	N-H wag amines

The second amine group shows strong amine correlations at 1230-1030 cm^{-1} and at 910-665 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch

Data possibly related to selected structure C=S thiocarbonyl : C=S thiocarbonyl at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl

The thiocarbonyl shows no correlations.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch

Data possibly related to selected structure S=O sulfone : S=O sulfone 2 at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	S=O sulfone	1350	1300		1140	s	S=O sulfone 1
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2

The sulfone shows moderate correlation to the sulfone at 1350-1300 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkyl halides	R-F	1350	1000			s	C-F stretch
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch

Data possibly related to selected structure P-H phosphine : P-H bending at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	P-H phosphine	2440	2280			m	P-H phosphine sharp
Misc.	P-H phosphine	1250	950			w	P-H bending

The phospine shows strong correlation in the 2440-2280 cm^{-1} range.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch
Esters	RCOOR`	1320	1000			s	C-O stretch

Data possibly related to selected structure P=O phosphine oxide : P=O phosphine oxide at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide

The phosphine oxide shows no additional correlations.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch
Esters	RCOOR`	1320	1000			s	C-O stretch

Data possibly related to selected structure P=O phosphate : P=O phosphate at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	P=O phosphate	1200	1100			s	P=O phosphate

The phosphate shows no additional correlations.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch
Esters	RCOOR`	1320	1000			s	C-O stretch

Data possibly related to selected structure RCO-OH : C-O stretch at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Carboxylic acids	RCO-OH	3405	3395	3400		s	monomer OH
Carboxylic acids	RCO-OH	1765	1755	1760		s	monomer C=O
Carboxylic acids	RCO-OH	3400	2800			s (broad)	dimer OH
Carboxylic acids	RCO-OH	1715	1705	1710		s	dimer C=O

The carboxylic acid shows strong correlation at 3400 cm^{-1}. The carboxylic acid shows moderate correlation at 1760 cm^{-1}. The carboxylic acid shows strong correlation in the 3400-2800 cm^{-1} range. The carboxylic acid shows moderate correlation at 1710 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Ethers	R-O-R	1150	1070			s	C-O stretch
Misc.	C=S thiocarbonyl	1200	1050			s	C=S thiocarbonyl
Misc.	S=O sulfone	1160	1120		1325	s	S=O sulfone 2
Misc.	P-H phosphine	1250	950			w	P-H bending
Misc.	P=O phosphine oxide	1200	1100			s	P=O phosphine oxide
Misc.	P=O phosphate	1200	1100			s	P=O phosphate
Carboxylic acids	RCO-OH	1320	1000			s	C-O stretch
Esters	RCOOR`	1320	1000			s	C-O stretch

Data possibly related to selected structure RCOOR` : C-O stretch at wavenumber: 1140 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment

The ester shows no correlations.

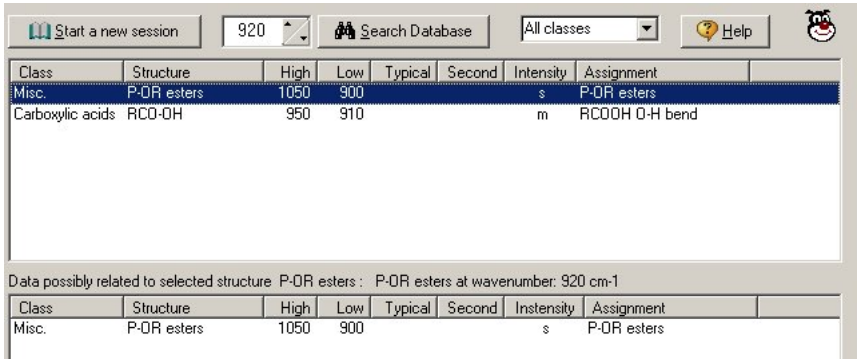
In summary:

Correlation Summary : 1140 cm^{-1}:

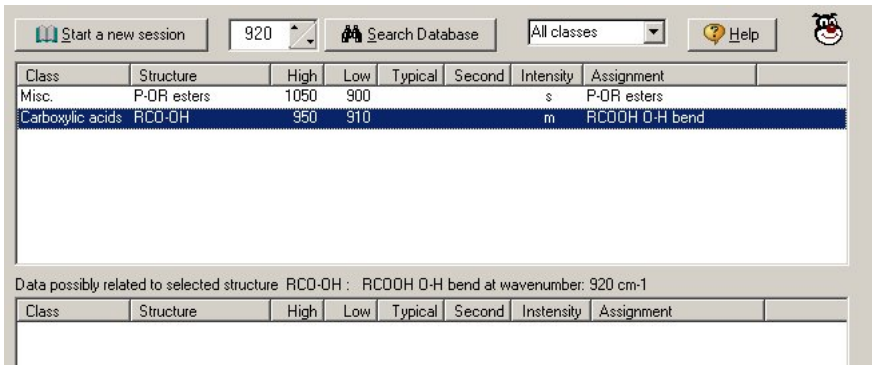
Strong :
Phospine > Phospine (2440-2280)
Carboxylic Acid > Carboxylic Acid (3400, 3400-2800)
Amine > Amine (3400)
Amine > Amine (1640-1560)
Amine > Amine (1230-1030)
Amine> Amine(910-635)

Moderate :
Alkyl Halide > Alkyl Halide (1300-1150)
Sulfone > Sulfone (1350-1300)
Carboxylic Acid > Carboxylic Acid (1760, 1710)

The next search is at 920 cm-1:



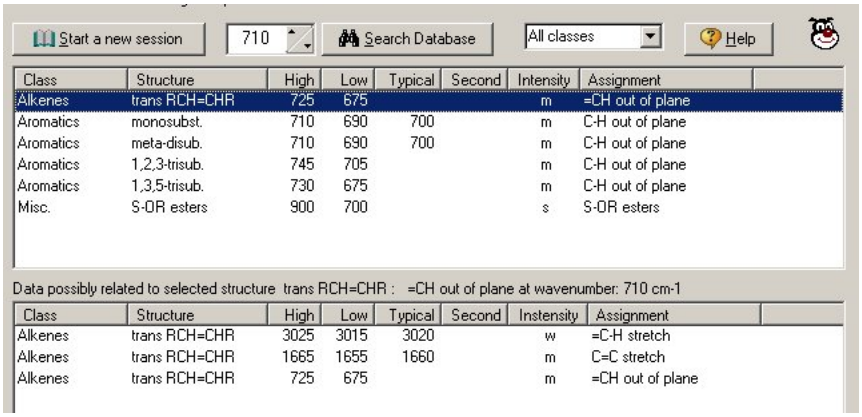
No additional correlations for the ester is found.



No additional correlations for the carboxylic acid are found.

Summary: No correlations found.

The last in the series of this correlation analysis will be at 710 cm^{-1}:



In the case of alkenes, we have a weak correlation at 1660 cm^{-1} with the alkenes.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters

Data possibly related to selected structure monosubst. : C-H out of plane at wavenumber: 710 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Aromatics	monosubst.	770	730			m	C-H out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	Ar-H	3100	3000			s	Ar-H stretch
Aromatics	C-C in ring	1600	1585	1592		m	Ar C-C stretch
Aromatics	C-C in ring	1500	1400			m	Ar C-C stretch

The mono-substituted aromatic has a moderate correlation with a mono-substituted aromatic at 770-730 cm^{-1}. We also have a moderate correlation with the aromatic at 1592 cm^{-1}. We also have a strong correlation with the aromatic at 1500-1400 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters

Data possibly related to selected structure meta-disub. : C-H out of plane at wavenumber: 710 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Aromatics	meta-disub.	810	750			m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	Ar-H	3100	3000			s	Ar-H stretch
Aromatics	C-C in ring	1600	1585	1592		m	Ar C-C stretch
Aromatics	C-C in ring	1500	1400			m	Ar C-C stretch

The meta-substituted aromatic shows moderate correlation to the aromatic at 1592 cm-1. It also shows strong correlation to the aromatic at 1500-1400 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters

Data possibly related to selected structure 1,2,3-trisub. : C-H out of plane at wavenumber: 710 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Aromatics	1,2,3-trisub.	780	760	770		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	Ar-H	3100	3000			s	Ar-H stretch
Aromatics	C-C in ring	1600	1585	1592		m	Ar C-C stretch
Aromatics	C-C in ring	1500	1400			m	Ar C-C stretch

The 1,2,3 tri-substituted aromatic shows moderate correlations to the aromatic at 1592 cm^{-1} and strong correlation to the aromatic at 1500-1400 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters

Data possibly related to selected structure 1,3,5-trisub. : C-H out of plane at wavenumber: 710 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Aromatics	1,3,5-trisub.	865	810			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Aromatics	Ar-H	3100	3000			s	Ar-H stretch
Aromatics	C-C in ring	1600	1585	1592		m	Ar C-C stretch
Aromatics	C-C in ring	1500	1400			m	Ar C-C stretch

The 1,3,5 tri-substituted aromatic shows moderate correlation to the aromatic at 1592 cm^{-1} and strong correlation to the aromatic at 1500-1400 cm^{-1}.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters

Data possibly related to selected structure S-OR esters : S-OR esters at wavenumber: 710 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Misc.	S-OR esters	900	700			s	S-OR esters

The S-OR ester shows no additional correlations.

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Class	Structure	High	Low	Typical	Second	Intensity	Assignment
Alkenes	trans RCH=CHR	725	675			m	=CH out of plane
Aromatics	monosubst.	710	690	700		m	C-H out of plane
Aromatics	meta-disub.	710	690	700		m	C-H out of plane
Aromatics	1,2,3-trisub.	745	705			m	C-H out of plane
Aromatics	1,3,5-trisub.	730	675			m	C-H out of plane
Misc.	S-OR esters	900	700			s	S-OR esters
Amines	RNH2, R2NH	910	665			s (broad)	N-H wag amines

Data possibly related to selected structure RNH2, R2NH : N-H wag amines at wavenumber: 710 cm-1

Class	Structure	High	Low	Typical	Second	Instensity	Assignment
Amines	RNH2	3405	3395	3400	3500	w	NH stretch
Amines	RNH2	3505	3495	3500	3400	w	NH stretch
Amines	RNH2	1640	1560			s	NH2 in plane bend
Amines	RNH2	1230	1030			m	C-N stretch
Amines	R2NH	3350	3310			w	NH stretch
Amines	R2NH	1230	1030			m	C-N stretch

The amine group shows strong correlation to the amine group at 3400 cm^{-1}, 1640-1560 cm-1, 1230-1030 cm^{-1} (RNH2) and 1230-1030 cm^{-1} (R2NH).

In summary:

Correlation Summary : 710 cm^{-1}:

Strong :
mono-substituted Aromatic > Aromatic (1500-1400)
meta-substituted Aromatic > Aromatic (1500-1400)
1,2,3 tri-substituted Aromatic > Arromatic (1500-1400)
1,3,5 tri-substituted Aromatic > Aromatic (1500-1400)
Amine > Amine (3400)
Amine > Amine (1640-1560)
Amine > Amine (1230-1030) (RNH2)
Amine > Amine (1230-1030) (R2NH)

Moderate :
mono-substituted Aromatic > mono-substituted Aromatic (770-730)
mono-substituted Aromatic > Aromatic (1592)
meta-substituted Aromatic > Aromatic (1592)
1,2,3 tri-substituted Aromatic > Aromatic (1592)
1,3,5 tri-substituted Aromatic > Aromatic (1592)

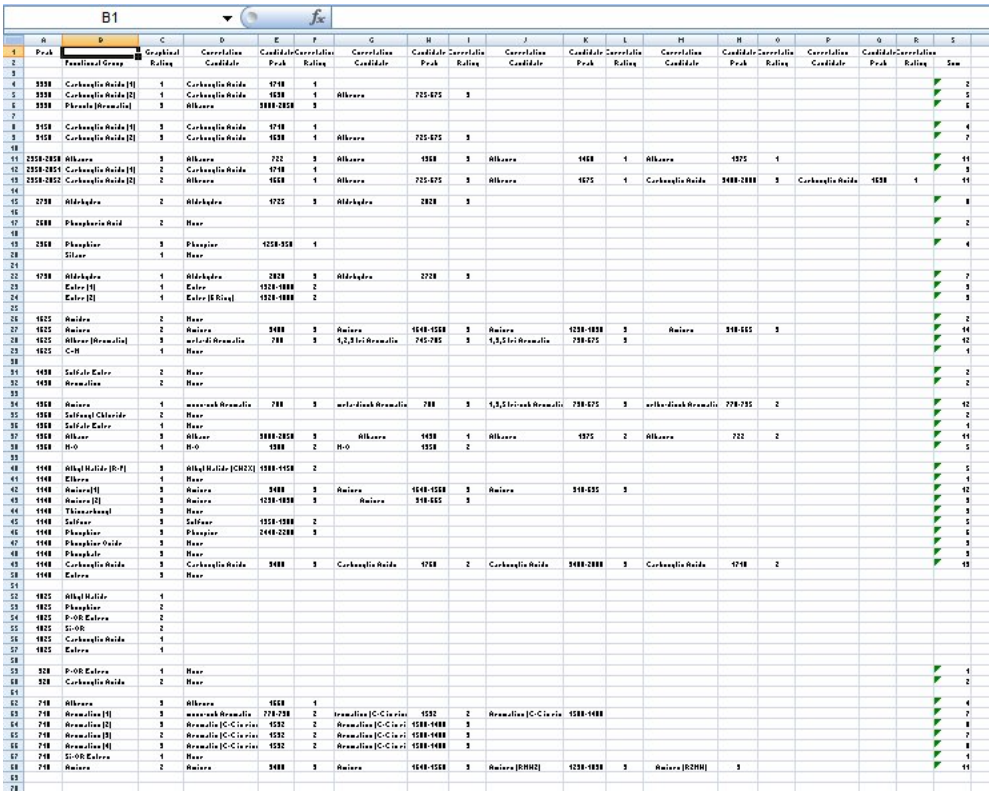
Weak:
Alkene > Alkene (1660)

We are now in a position to start collating the information that we have acquired. The goal is to identify the candidates that are most likely to be structural components of the oral sample under investigation. We now have three primary data points available to use in the approach that will be developed:

1. The functional group candidates themselves, as identified with the tabular data from IR Pal as well as additional tables or sources as needed (e.g., Nakanishi).

2. The position of the absorption peaks within the graphical ranges that have been shown above and that accompany these same tabular listings.

3. The extensive correlation analysis that is presented above. In addition to having these three sources of information available, a strategy to use them in a sensible fashion will need to be developed. In general, a linear combination of graphical and correlative rankings will be used to integrate and combine this data.



Peak	Functional Group	Graphical Rating	Correlation Candidate	Candidate Peak	Correlation Rating	Correlation Candidate	Candidate Peak	Correlation Rating	Correlation Candidate	Candidate Peak	Correlation Rating	Correlation Candidate	Candidate Peak	Correlation Rating	Correlation Candidate	Candidate Peak	Correlation Rating	Sum
3330	Carboxylic Acids [1]	1	Carboxylic Acids	1710	1													2
3330	Carboxylic Acids [2]	1	Carboxylic Acids	1630	1	Alkenes	725-675	3										5
3330	Phenols [Aromatic]	3	Alkanes	3000-2850	3													6
3150	Carboxylic Acids [1]	3	Carboxylic Acids	1710	1													4
3150	Carboxylic Acids [2]	3	Carboxylic Acids	1630	1	Alkenes	725-675	3										7
2950-2850	Alkanes	3	Alkanes	722	3	Alkanes	1360	3	Alkanes	1460	1	Alkanes	1375	1				11
2950-2851	Carboxylic Acids [1]	2	Carboxylic Acids	1710	1													3
2950-2852	Carboxylic Acids [2]	2	Alkenes	1650	1	Alkenes	725-675	3	Alkenes	1675	1	Carboxylic Acids	3400-2800	3	Carboxylic Acids	1630	1	11
2720	Aldehydes	2	Aldehydes	1725	3	Aldehydes	2820	3										8
2500	Phosphoric Acid	2	None															2
2360	Phosphine	3	Phosphine	1250-950	1													4
	Silane	1	None															
1730	Aldehydes	1	Aldehydes	2820	3	Aldehydes	2720	3										7
	Esters [1]	1	Esters	1320-1000	2													3
	Esters [2]	1	Esters [6 Ring]	1320-1000	2													3
1625	Amides	2	None															2
1625	Amines	2	Amines	3400	3	Amines	1640-1550	3	Amines	1250-1050	3	Amines	910-665	3				14
1625	Alkenes [Aromatics]	3	meta-di Aromatics	780	3	1,2,3 tri Aromatics	745-705	3	1,3,5 tri Aromatics	750-675	3							12
1625	C=N	1	None															1
1450	Sulfate Esters	2	None															2
1450	Aromatics	2	None															2
1360	Amines	1	mono-sub Aromatics	780	3	meta-disub Aromatics	780	3	1,3,5 tri-sub Aromatics	750-675	3	ortho-disub Aromatics	770-735	2				12
1360	Sulfonyl Chloride	2	None															2
1360	Sulfate Esters	1	None															1
1360	Alkanes	3	Alkanes	3000-2850	3	Alkenes	1450	1	Alkanes	1375	2	Alkanes	722	2				11
1360	N-O	1	N-O	1300	2	N-O	1350	2										5
1140	Alkyl Halide [R-F]	3	Alkyl Halide [CH2X]	1300-1150	2													5
1140	Ethers	1	None															1
1140	Amines [1]	3	Amines	3400	3	Amines	1640-1550	3	Amines	910-665	3							12
1140	Amines [2]	3	Amines	1250-1050	3	Amines	910-665	3										9
1140	Thioanhydrol	3	None															3
1140	Sulfoxe	3	Sulfoxe	1350-1300	2													5
1140	Phosphine	3	Phosphine	2400-2300	3													6
1140	Phosphine Oxide	3	None															3
1140	Phosphate	3	None															3
1140	Carboxylic Acids	3	Carboxylic Acids	3400	3	Carboxylic Acids	1750	2	Carboxylic Acids	3400-2800	3	Carboxylic Acids	1710	2				13
1140	Esters	3	None															
1025	Alkyl Halide	1																
1025	Phosphine	2																
1025	P-OR Esters	2																
1025	Si-OR	2																
1025	Carboxylic Acids	1																
1025	Esters	1																
920	P-OR Esters	1	None															1
920	Carboxylic Acids	2	None															2
710	Alkenes	3	Alkenes	1660	1													4
710	Aromatics [1]	3	mono-sub Aromatics	770-730	2	trans Alkenes [C-C in ring]	1592	2	Aromatics [C-C in ring]	1500-1400								7
710	Aromatics [2]	3	Aromatics [C-C in ring]	1592	2	Aromatics [C-C in ring]	1500-1400	3										8
710	Aromatics [3]	2	Aromatics [C-C in ring]	1592	2	Aromatics [C-C in ring]	1500-1400	3										7
710	Aromatics [4]	3	Aromatics [C-C in ring]	1592	2	Aromatics [C-C in ring]	1500-1400	3										8
710	Si-OR Esters	1	None															1
710	Amines	2	Amines	3400	3	Amines	1640-1550	3	Amines [RNH2]	1250-1050	3	Amines [R2NH]	3					11

Spreadsheet to evaluate the graphical and correlative weighted contributions to the expected structural composition of the oral filament sample.

The rankings of the contributions of the various functional groups can now be made. We have the following relative contributions of the functional groups or structures, from the greatest likelihood to the less likely:

	A	B	D	G	J	M	P	S
1	Absorption		Correlation	Correlation	Correlation	Correlation	Correlation	Total
2	Peak	Functional Group	Candidate	Candidate	Candidate	Candidate	Candidate	Score
3								
4	1625	Amines	Amines	Amines	Amines	Amines		14
5	1140	Carboxylic Acids	Carboxylic Acids	Carboxylic Acids	Carboxylic Acids	Carboxylic Acids		13
6	1625	Alkene (Aromatic)	meta-disub Aromatic	1,2,3 tri Aromatic	1,3,5 tri Aromatic			12
7	1360	Amines	mono-sub Aromatic	meta-disub Aromatic	1,3,5 tri-sub Aromatic	ortho-disub Aromatic		12
8	1140	Amines(1)	Amines	Amines	Amines			12
9	2950-2850	Alkanes	Alkanes	Alkanes	Alkanes	Alkanes		11
10	2950-2852	Carboxylic Acids (2)	Alkenes	Alkenes	Alkenes	Carboxylic Acids	Carboxylic Acids	11
11	1360	Alkane	Alkane	Alkanes	Alkanes	Alkanes		11
12	710	Amines	Amines	Amines	Amines (RNH2)	Amines (R2NH)		11
13	1140	Amines (2)	Amines	Amines				9
14	2730	Aldehydes	Aldehydes	Aldehydes				8
15	710	Aromatics (2)	Aromatic (C-C in ring)	Aromatics (C-C in ring)				8
16	710	Aromatics (4)	Aromatic (C-C in ring)	Aromatics (C-C in ring)				8
17	3150	Carboxylic Acids (2)	Carboxylic Acids	Alkenes				7
18	1730	Aldehydes	Aldehydes	Aldehydes				7
19	710	Aromatics (1)	mono-sub Aromatic	Aromatics (C-C in ring)	Aromatics (C-C in ring)			7
20	710	Aromatics (3)	Aromatic (C-C in ring)	Aromatics (C-C in ring)				7
21	3390	Phenols (Aromatic)	Alkanes					6
22	1140	Phosphine	Phospine					6
23	3390	Carboxylic Acids (2)	Carboxylic Acids	Alkenes				5
24	1360	N-O	N-O	N-O				5
25	1140	Alkyl Halide (R-F)	Alkyl Halide (CH2X)					5
26	1140	Sulfone	Sulfone					5
27	3150	Carboxylic Acids (1)	Carboxylic Acids					4
28	2360	Phosphine	Phospine					4
29	710	Alkenes	Alkenes					4
30	2950-2851	Carboxylic Acids (1)	Carboxylic Acids					3
31	1730	Ester (1)	Ester					3
32	1730	Ester (2)	Ester (6 Ring)					3
33	1140	Thiocarbonyl	None					3
34	1140	Phosphine Oxide	None					3
35	1140	Phosphate	None					3
36	3390	Carboxylic Acids (1)	Carboxylic Acids					2
37	2680	Phosphoric Acid	None					2
38	1625	Amides	None					2

It is the position of this researcher that the above chart reveals, along with the amino acids and iron content previously disclosed, the most probable structural features of the "Morgellons" oral filament sample material. The job remaining before us is to form a more composite picture of this *structural whole* and the likely and expected health impacts from this same characteristic structure. The culminating discussion is then to bring into consideration various strategies that may be beneficial in mitigating these health impacts and to once again invite the health and medical communities to investigate the veracity of this accumulated research. These issues, to the degree appropriate and possible here, will be pursued.

The next step in our work is to investigate the general nature and characteristics of the functional groups that are indicated, at least to the level of probability appropriate to the means and equipment. This basic knowledge of functional group characteristics will be necessary in understanding the assemblage that is to come further down the road in this report. We will progress from the most prevalent to the least prevalent groups.

Let us start with the amines. The amines are a functional group that contains nitrogen, and they are derivatives of ammonia, whereby the hydrogens of ammonia (NH3) are replaced by various organic groups. A primary amine has one hydrogen replaced and has the formula NH2. Secondary amines replace two of the hydrogens and tertiary amines replace three of the hydrogens, respectively.[5] Amines can react with acids due to their basic nature; the basicity varies over a fairly wide range[6]. The chemistry of amines is dominated by the presence of a lone pair of electrons on nitrogen [7](this is in the ammonia form). They are produced by the decomposition of organic matter.[8] **Amines are a fundamental constituent of amino acids (i.e., proteins)**. Some of the important reactions that take place with amines includes interactions with alkyl halides, aldehydes, ketones, acid chlorides and nitrous acid.[9] Amines are the most important biological bases.[10] Amines often have a "fishy" odor and many drugs, such as quinine, codeine, caffeine and amphetamine, are amines.[11]

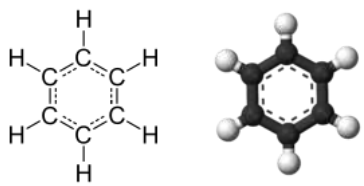
Our next group of significance is carboxylic acids. Carboxylic acids are one the most important biological acids. They react with bases (such as amines) to produce salts. These salts contain an ammonium ion from the amine and a carboxylate ion from the acid.[12] They are most acidic of the common functional groups[13]. The carboxyl group the formula COOH, i.e, a carbonyl group attached to a hydroxyl group. Many long-chain carboxylic acids occur as esters in fats and oils, and are known as "fatty acids".[14] Carboxylic acids are the largest group of organic acids. As more electronegative atoms in the acid increases, the strength of the acid increases. For example, if the hydrogen atoms in the acid (acetic acid, for example) are replaced with fluorine (a halogen) to produce trifluoroacetic acid, the increase in acidity is quite large. **Amino acids, by definition, contain combine both an amine group and a carboxyl group,** and a "R" group (i.e., variable group). Amino acids can act as both acids and bases, because of the combination of the amine (basic) and the carboxyl group (acidic), separated by the R group.[15] Some common examples of carboxylic acids are acetic acid, oxalic acid and formic acid. Carboxylic acids are amongst the most useful building blocks for synthesizing other molecules, both naturally and in the laboratory.[16]

The next category of interest is that of an aromatic-alkene complex. Let us begin with an introduction of the importance of the presence of aromatics in the structure identification:

Aromatics are an extremely important branch of organic chemistry, with many ramifications to follow. Organic chemistry can be divided into two main structural forms, that of aliphatic and aromatic organic chemistry. Aliphatic, in a very general sense refers to a chain-like structure and aromatics, in a general fashion, refer to ring based structures. This division is significant, especially with respect to stability and expected chemical reactions to take place. Examples of aliphatics are alkanes, alkenes and alkynes (basically carbon-hydrogen bonds in a chain-like structure)[17]. An example of an aromatic compound is benzene, a classic six carbon ring structure that many of us have some familiarity with. It is of interest that our category of an aromatic alkane is the next on our list. This alone informs us that we are likely dealing with a combination of both aliphatic and aromatic form, which alone would allow for infinite chemical flexibility from an organic chemistry perspective. For now, our focus will remain on the aromatic aspect of discovery that has taken place.

Let us discuss aromatic chemistry in a general fashion. Aromaticity, in general, is used to refer to benzene and its structural relatives. Although this may conjure up an image of a fixed six-ring carbon structure, this level of restriction is not at all appropriate in our understanding of aromatic chemistry. A more formal definition of aromatic is that of a "cyclic conjugated molecule containing 4n+2 pi electrons."[18] We will make some headway into that rather intimidating phrase as we go along, but for now let us work with the classical ring structure in mind and some of the general chemical characteristics of that same benzene structural form.

A couple of the more important characteristics of aromatics (or with benzene as a typical example) is that of its cyclic, or ring structure and its physical and chemical stability. These features go hand in hand because of the structural nature involved; a hexagon is one of the most stable structures of nature (e.g., the honeycomb). Here is an image of benzene to begin this visualization process:



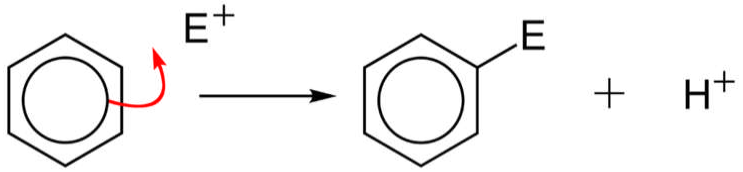
A typical aromatic structure – Benzene
source : www.wikipedia.org

Another feature characteristic of the aromatic is its "conjugated" nature. Conjugation refers to the alternation between single and double bonds in a chemical structure. Conjugation, in general, has the effect of lowering the energy of the molecule and of increasing its stability, an important complementing feature to this same stability mentioned earlier. Benzene is only one example of an aromatic structure; there are an infinite number of variations on this basic theme that will lead to the individual chemistry, i.e., biochemistry, of the form that is under investigation. Benzene by itself is toxic; it leads to bone marrow depression and lowered white cell counts. On the other hand, there are some amino acids in the body that contain aromatic structures (e.g., phenylalanine, tryptophan, tyrosine[19]). Other examples of aromatic compounds include natural fragrances, steroid hormones, and many drugs such as valium and morphine. The fact that a structure is aromatic is, therefore, not sufficient to characterize its general chemical influence upon the body. We must know more. The presence of an aromatic structure, nevertheless, is one of monumental significance in understanding the expected influences and impact upon human health. The rub will be in knowing how the aromatic structure is modified so that its impact can be more likely assessed with fairness. The identification of aliphatic compounds (that of alkenes in our case, to be discussed separately) in combination with an aromatic structure leaves us with plenty of room for further important discoveries.

To begin that process of discovery, we must now look at the *types of reactions* that are known to occur with aromatic compounds; this is our key to further progress In essence, the use of infrared spectroscopy has opened a very important puzzle for us to solve, and deeper we must now go into this unsolved mystery.

The most common chemical reaction with aromatics is that of *electrophilic aromatic substitution*. What this means, in the most basic sense, is that one of the carbon atoms on the ring gets replaced by *"something else*." The nature of the "something else" is crucial to an understanding of the expected chemical and biochemical impact of the filament structure upon human health and biology in general. In response to this need, let us introduce the definition of an electrophile and a nucleophile, respectively.

An electrophile is something (i.e., ion or molecule) that is deficient in electrons and that can accept electrons. Electrophiles are positively charged, and examples include the positive ion of NO_2^+ and the electron deficient SO_3 atom. Electrophiles are reducing agents and act as what is known as a *Lewis* acid. A nucleophile, in contrast, is an ion or molecule that has an excess of electrons and that can donate them. Nucleophiles are oxidizing agents and act as *Lewis* bases. Examples of nucleophiles are the Chlorine ion (Cl^-) and ammonia (NH_3)[20]. Here is a picture of the general process:



In this diagram, E^+ is the electrophile. The electrophile reacts with one of the hydrogens on the aromatic ring and substitutes itself on the ring.
The hydrogen ion is then left free. Source: commons.wikimedia.org

It is now sensible to introduce the types of aromatic electrophilic substitution reactions that occur. These are as follows[21]:

1. Halogenation :

The substitution of a halogen for one of the hydrogens.

2. Nitration :

The substitution of a nitro group (NO_2) for one of the hydrogens.

3.Sulfonation :

The substitution of a sulfonic acid group (SO_3H) for one of the hydrogens.

4.Alkylation :

The substitution of an alkyl group for one of the hydrogens. An alkyl group is formed when one of the hydrogens is removed from an alkane group. An example of an alkyl is a methyl group (CH_3-), which is formed from methane (CH_4). Alkanes are saturated hydrocarbons with the general formula C_nH_{2n+2}. Examples of alkanes are methane (CH_4), propane(C_3H_8) and butane(C_4H_{10}). Saturation refers to molecules that have only single bonds, i.e., no double or triple bonds. Alkanes contain only carbon and hydrogen, and all the bonds between atoms are single bonds[22]. A common term for alkanes is that of paraffins.

5.Acylation :

The substitution of an acyl group for one of the hydrogens. An acyl group has the form RCO-, where R is any organic group. An example of an acyl is the acetyl group, CH_3O-. Another variation of an acyl is the case of acyl halides, which has the form RCOX, where X is a halogen, such as acyl chloride (RCOCl)[23].

Each of these reactions requires certain reagents or catalysts to be present to take place. In human biochemistry, some of these reactions are more likely to be able to occur than others. Let us examine these groups and determine which reactions in the body are less likely to occur than others, thereby simplifying and restricting our scope of probable structural composition.

In the description of aromatic nitration[24], it will be found that this requires the presence of a mixture of concentrated nitric and sulfuric acids. Since this combination is not likely to be found within the human body, the process will be excluded further from our structural investigation. A similar situation will be found for that of sulfonation[25], which can occur in the presence of fuming sulfuric acid. Sulfonation will also be consequently diminished in our further consideration in this investigation, however, we must remain alert to alternative catalysts or pathways whereby a reaction might occur. Aklylation, acylaton and halogenation are expected to occur fairly readily within human biochemistry, and remain under full consideration in our structural analysis. Considerable discussion on the halogenation substitution reactions will take place.

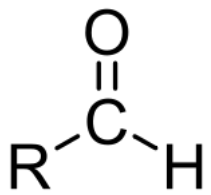
Since the group identified most recently within this discussion is that of an alkene aromatic, we must introduce this addition as well. The alkene is an unsaturated hydrocarbon that contains one or more double carbon bonds. The general formula of an alkene is C_nH_{2n} and examples include propene and butene. A common term used for alkenes is olefins or olefines.

Studying our list of probabilistically ranked functional groups further, the next item mentioned is again that of amines, with an important addition. The presence of the aromatics, this time in combination with the amines in addition to that previously noted for alkenes, must be recognized. This strengthens the case considerably for aromatic biochemistry within our structure. The importance of halogenation substitution within the aromatic group will also be further developed in our discussion as we proceed.

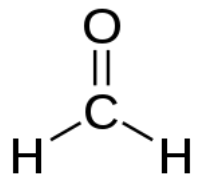
We next see the alkanes introduced, and they have already been discussed to some extent. They are a very common organic functional group to be found within organic compounds. They are saturated, single bond hydrocarbons with the general formula C_nH_{2n+2}. Alkanes are within the branch of aliphatic organic chemistry, which serves as a chain structure that links many different types of organic compounds together.

The carboxylic acids, the alkenes, the alkanes and the amines all repeat themselves subsequently on the list of functional group candidates. This further strengthens their consideration in our structural analysis that is in progress.

The next addition on our list is an aldehyde. The aldehyde group has the structure -CHO and can be visualized as follows: The simplest example of an aldehyde is also shown below, that of formaldehyde.

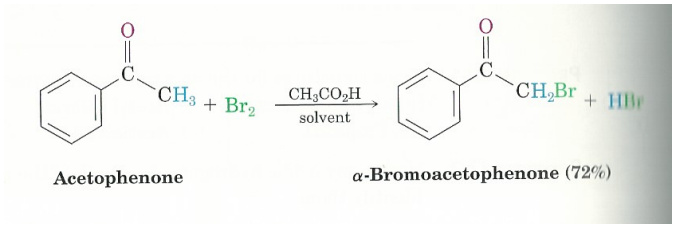


The aldehyde group. The R represents any generic organic structure, i.e., an organic variable.
Source:www.wikipedia.com



An example of an aldehyde, i.e., formaldehyde. We see here that the R group has been occupied by a hydrogen atom.
Source: www.wikipedia.com

Aldehydes are a reactive group and they readily polymerize[26]. Polymerization is the joining of molecules to form a series of repeating units. They are formed by the oxidation of alcohols, and further oxidation yields carboxylic acids (mentioned previously). Aldehydes can also be halogenated by reactions with chlorine, bromine or iodine in an acidic solution[27]. An example of halogenation (bromination) of an aldehyde, in this case with the use of acetic acid, is as follows:

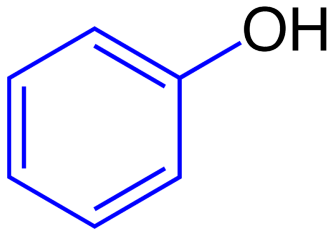


source : McMurray

Notice also the combination of an aromatic structure, and aldehyde and halogenation occurring in the presence of an organic acid in the above example of an aldehyde reaction.

Our next entries are those of aromatics and substituted aromatics, once again. This continues to reinforce the expected importance of aromatics and electrophilic substations in our future discussion of the composite structural portrait that continues to develop within this paper.

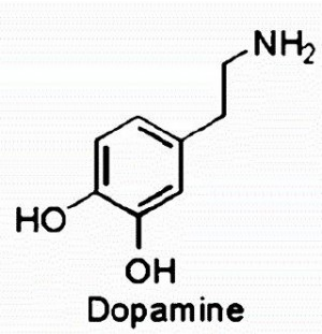
We next have the introduction of a phenol group, once again in combination with an aromatic form. A phenol, by definition, is the existence of a hydroxyl group (OH) that binds directly to a carbon atom on a benzene ring[28.] Hydroxyl groups normally indicate an alcohol, but in the case of the phenol, the structure is acidic because of the influence of the benzene ring. A diagram of the phenol structure is as follows:



The phenol group
Source : commons.wikipedia.org

One of the interesting structures involving the phenol group that has arisen within this investigation is that of dopamine. It will be noticed that dopamine is composed primarily of an aromatic ring, a couple of phenol groups attached, and an amine structure at the end of a carbon chain. Dopamine may well have to do primarily with the *motivation and drive* of an individual; see Eric Matzer's article : Dopamine is Not About Pleasure Anymore and How Science Evolves. What is of interest here is the importance of the role the relatively simple phenol group can play

in the behavior and neural functioning of an individual. The role of Parkinson's disease in relation to dopamine will also be worthy of our examination. Lastly, in the future we will examine how a slight tinkering of this molecule can lead to the development of neurotoxins that can easily be expected to seriously interfere with the neural functioning of an individual. More on this issue later.



The dopamine molecule
Source : www.matzner.com

We are approaching the end of the functional group ranking list, at least to the level that we can have a greater confidence in. The lack of repetition of functional groups that is developing is a signal that we should begin to exercise caution in extrapolating our results beyond an expected level of significance. Brief mention will be made of the finalizing set of groups to consider at this time, which includes phosphine, a repeat of carboxylic acids, a nitrogen-oxygen group, and an alkyl halide. Phosphine (PH_3) is a highly toxic gas formed by heating white phosphorus in concentrated sodium hydroxide. There is no particular reason to expect this particular compound in human biochemistry and notice no repetition of occurrence of the compound. Carboxylic acids have been mentioned previously and they remain as a primary candidate. Nitro compounds can also not be emphasized in this investigation due to the lack of repetition.

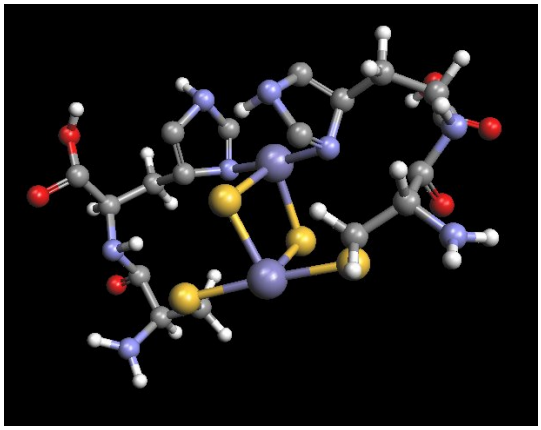
The alkyl halides do provoke a level of interest, due to the previous discussion of both alkyls and halogens. An alkyl halide (also known as a haloalkane) is a organic compound whereby one of the hydrogen atoms of an alkane has been substituted with a halogen. Alkyl halides can be formed by a combination of alkanes, halogens and ultraviolet light, in addition to reactions between alcohols and an halogenating agent. One example of an alkyl halide is dibromoethane, CH_2BrCH_2Br. Many alkyl halides are major pollutants or toxins. They are widely used in flame retardants, refrigerants, pesticides, propellants, solvents and pharmaceuticals. Most alkyl halides are synthetic, but natural sources do exist and they are produced by some bacteria, fungi and algae. We also note an additional minor absorption peak at 1025 cm-1 that corresponds to the alkyl halides and that increases our interest in this particular group.

This completes our list of functional groups that are to be considered in this analysis. The next stage in this project is to collect the information that now besets us, both from previous work and from this current work. Infrared spectrometry will not allow us to define a single finite structure, but it will serve to identify some of the building blocks. These building blocks along with some understanding of the expected biochemistry will end up serving us well for the effort that has been spent.

To begin with, let us recall what has been learned from previous work and from alternative methods. We know from previous papers entitled, *Morgellons : A Thesis* (Oct 2011) and *Amino Acids Verified* (Nov 2012), that iron and amino acids are core constituents of the biological filaments. These are crucial and important discoveries in their own right. Please be aware, however, that it has taken several years of work to arrive at a point that could have easily been understood and attained within a matter of months with the proper support and resources.

It is also of benefit, at this stage, to recall the beginnings of structural analysis that was taking place at the terminus of the papers mentioned immediately above. This work took place using primarily the methods of column chromatography, electrolysis, ninhydrin analysis and visible light spectrometry. The work was protracted, tedious and took well over a year to accomplish. An initial iron-amino acid complex molecular

model (shown below) was developed to open this door which we are now entering more deeply:



Proposed Model of Histidine-Cysteine Proteinaceous Dipeptide Complex
(Overlaps or Parallels Rieske Protein Structure)
Coordinated Iron Complex in Center of Structure

Please see *Amino Acids Verified*, CE Carnicom, (Nov 2012) for additional information on the work leading to the above model. This model depicts an amino acid-iron complex.

As with all nutrients that are redirected to support a parasitic or diseased relationship between living forms, this loss of nutrients and energy will be done at the expense of the host. Let us be clear that the human being is the host here, and there can be no expectation other than that of suffering to some degree. In many cases, the suffering is extreme and we all pay the price for this with each day that we allow this situation to pass unchallenged.

Next, let us collect the *probabilistic* list from the current work. Understand that nothing is definite here. All work here is evolutionary with highly limited resources and is subject to errors; I will, however, do my best to establish a path that others hopefully will assist in. This current paper based upon infrared spectrophotometry proposes the following additions to now incorporate into our structural analysis:

Amines
Carboxylic Acids
Aromatics
Aromatic substituted Alkenes
Aromatic substituted Amines
Alkanes
Aldehydes
Aromatic Phenols
Alkyl Halides

Time has been spent on discussing the general features and characteristics of each of these functional groups. We must use this information to attempt to create a greater composite picture of our structure involved, and its subsequent expected biochemistry and impact upon human health.

For the sake of consolidation and simplification, let us now repeat the candidate list in total and in combination, along with some relevant imagery:

hemoglobin

Iron, a primary constituent of blood (hemoglobin)
image source : healthtap.com

hemoglobin 2

Hemoglobin, the primary carrier of oxygen in the body
image source : natural-holistic-health.com

Iron
(Fe+3 in the more highly oxidized state)

amino acid

Amino Acids, the building blocks of protein
image source : whitetigernaturalmedicine.com

amino acid 2

source : compbio.cs.toronto.edu

source : pdsblog

Amino Acids – General

source : science.uvu.edu

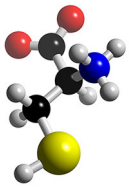
Vinegar, containing acetic acid, a representative carboxylic acid
image source : sites.google.com

Carboxylic Acids

Representative aromatic compounds
source : halo.wikia.com, source : meritnation.com

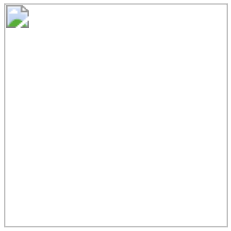
Aromatics

Cysteine
image source : wikimedia.org



Cysteine 3D Model
image source : 3dchem.com

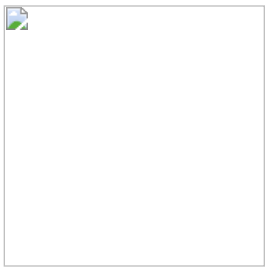
Specific Amino Acid: Cysteine



Histidine, Molecular & 3D Model
image source : wikimedia.org image source : 3dchem.com

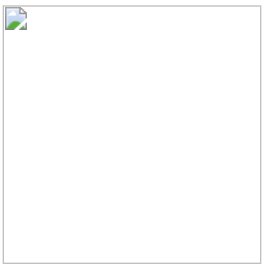
Specific Amino Acid : Histidine

Styrene, a representative aromatic alkene
image source :endlessplastics.com



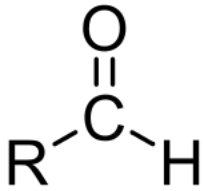
Styrene Molecular Model
image source :chemistry-reference.com

Aromatic substituted Alkenes



Aniline, a representative aromatic amine
image source : goiit.com, image source : chemmeddl.com

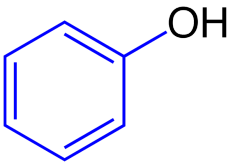
Aromatic substituted Amines



The molecular composition of an aldehyde. The “R” stands for any variable atom or organic group. image source : wikipedia.org

Formaldehyde, one of the most common and important aldehyde groups; known as a preservative.
image source : treehugger.com

Aldehydes



The phenol group; source:wikipedia.org

image source: buzzle.com –

3D Model o
oxydopamine
neurotoxin.
source:wikimedi

Aromatic Phenols

Iodine, one of the halogens, crucial to human metabolism.
image source : edtech2.boisestate.edu

R is any organic atom or functional group; X is any halogen. The halogens are the most reactive elements known.
image source : chemistry-drills.com mrstaylor.wikispaces.com.

Alkyl Halides

An amine group
image source : course1.winona.edu

amino acid group

An amino group within the structure of an amino acid..
image source :biology.about.com

image sourc
:docstoc.cor

Amines

The basic alkane structure (single carbon bonds, saturated)image source : intrestingthings4u.blogspot.com

Some common alkanes image source : daviddarling.info:

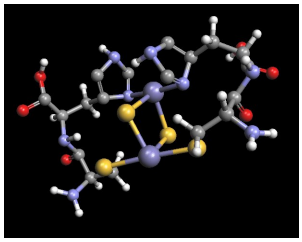


image source : carnicominstitute.org

Alkanes

Previously Proposed : Iron – Cysteine- Histidine – Amino Acid Complex M
(Nov 2012)
(shown earlier)

Table of Candidate Components – Functional Groups of *Morgellons* Oral Filament Sample :
Analyzed by Infrared Spectrophotometry, Chromatography, Electrolysis, Ninhydrin Tests and Visual Light Spectrophotometry

END OF PART I

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Morgellons : A Working Hypothesis – PART II : POTENTIAL HEALTH IMPACTS OF THE VARIOUS FUNCTIONAL GROUPS & COMPONENTS

carnicominstitute.org/morgellons-a-working-hypothesis-part-ii-potential-health-impacts-of-the-various-functional-groups-components/

Morgellons : A Working Hypothesis
Neural, Thyroid, Liver, Oxygen, Protein and Iron Disruption
(Link to Parts I, II, III – Click Here)

PART II
POTENTIAL HEALTH IMPACTS OF THE VARIOUS FUNCTIONAL GROUPS & COMPONENTS

Clifford E Carnicom
Dec 18 2013

Art work courtesy of David Dees with permission.

Note: I am not offering any medical advice or diagnosis with the presentation of this information. I am acting solely as an independent researcher providing the results of extended observation and analysis of unusual biological conditions that are evident. Each individual must work with their own health professional to establish any appropriate course of action and any health related comments in this paper are solely for informational purposes and they are from my own perspective.

This paper seeks to identify a host of organic compounds that are likely to comprise the core physical structure of biologically produced filaments characteristic of the *Morgellons* condition. A biological oral filament sample will be analyzed for the presence of candidate organic functional groups using the methods of infrared spectrophotometry. Potential health impacts from these same core structures are examined and compared to the observed , reported and documented symptoms (in part) of this same condition. Potential mitigating strategies, from a research perspective only, are discussed.

A body of evidence, accumulated over a period of several years, reveals that the Morgellons condition is likely characterized by a host of serious physiological and metabolic imbalances. These imbalances are caused by the disruption of a variety of major body processes including, as a minimum, the regulation of metabolism by the thyroid, potential liver enlargement, a decrease of oxygen in the circulatory system, the utilization of amino acids important to the body, the oxidation of iron and a potential impact to neural pathways. The impact of this degradation to human health can be concluded to be serious, debilitating and potentially lethal in the cumulative sense; the reports of those who suffer from the condition are in alignment with these conclusions. This paper will summarize the body of work and chronology which leads to this more comprehensive hypothesis.

The health, medical and governmental communities will again be invited to offer their expertise and contributions, as well as to assume their role of responsibility and the obligations of their professions to serve the public.

This paper will be divided into three phases:

I. Identification of the functional groups / components

II. Potential health impacts of the various functional groups identified.

III. Potential mitigating strategies (research-based)

PART II
POTENTIAL HEALTH IMPACTS OF THE VARIOUS FUNCTIONAL GROUPS & COMPONENTS

We now have a puzzle before *all of us*. We are likely to have some of the pieces that make up the whole, but we must all work on putting the pieces together. Infrared spectrometry alone cannot do this; additional resources, execution and smart thought will be required.

The earlier this puzzle is solved in detail, the better we will all be for it. I can only ask you to join in the crusade. Until that necessary level of understanding is achieved, I will continue to offer my own interpretations below. The discussion will progress through generalized structural interpretations, possible and projected health impacts, and the review of various strategies that may be worthwhile of consideration for mitigation of the anticipated and observed effects of the condition. It will again be emphasized and expressed that no medical advice or diagnosis is to be given here; each individual MUST pursue the counsel and advice of their own chosen health practitioner. The information provided here serves research purposes only.

The next sensible need is to tabulate the reported health impacts and symptoms alongside the functional groups so that we may begin the process of comparison, correlation and analysis between them. The lists are not necessarily exhaustive or complete or without error, but they should provide a useful beginning to the problems to be solved.

Table of Health Impacts or Symptoms vs. Functional Group Identification:

Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:	**Functional Groups Identified:** (no correlations established at this stage)	**Related Images:**
Skin lesions (non or slow-healing)		
	Iron (Fe+3 in the more highly oxidized state)	
Skin-borne filament production; skin manifestation at the more developed levels (the skin is an excretory organ).		
Chronic decreased body temperature.		

Oxygen deprivation; diminished oxygen carrying capacity of the blood.

Immune system breakdown

Metabolic disruption; indications of thyroid and adrenal complications

Significant oral filament production; the presence of filament structures (ferric iron – anthocyanin complexes) within oral samples. (red wine test)

Unusual or extreme dental issues; tooth decay or loss

Chronic itching, stinging, crawling, or biting sensations of the skin

Dark particles emerging from skin or scalp

Hair alterations, i.e., texture, thickness, loss of hair

Amino Acids – General

Amines

Carboxylic Acids

Aromatics

Neurological impairment, i.e., blurred vision or “floaters” in the eye, slurred speech, ringing of the ears (tinnitus), loss of coordination, loss of strength

Extended or Chronic Fatigue

Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog”

Gastro-intestinal imbalance

Joint pain

Specific blood abnormalities

An increased level of acidity in the body (may be most easily assessed by urine pH testing).

Aromatic substituted Alkenes

Aromatic substituted Amines

Alkanes

Aldehydes

The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.	Aromatic Phenols
Liver toxicity, gall bladder and bile duct complications.	Alkyl Halides
Respiratory problems, including proclivities toward a chronic cough or walking pneumonia-like symptoms.	
The presence of a bacterial-like component (chlamydia-like) within or surrounding the red blood cells.	
Recent research indicates that the urinary tract may be equally affected with the presence of the filament structures	
The smoking population may exhibit an increased incidence of the condition due to additional oxygen inhibition within the blood.	

The list of health impacts and symptoms involved is derived from two sources: analytical and research work from this site[29] and from the Morgellons Research Project[30], which is under the direction of Carnicom Institute.

Let us start to examine some of the health implications of these particular organic functional groups (and inorganic, as well) and how these may potentially relate to health impacts from the Morgellons condition that are frequently reported. This will never be as simple as a one-to-one correspondence – far from it. There are, however, some generalities to be made that may be very helpful in the interpretation of the plight that many find themselves within.

The importance of the iron component within the filament growth form has been extensively discussed; an entire paper has been devoted to that topic including its discovery. This discovery was based on chemical separation techniques, chromatography and visible light spectrophotometry, and it precedes the use of infra-red spectrophotometry described here for the detection of organics compounds. Iron is an inorganic substance and is not well suited to IR spectrophotometry. Please become familiar with the paper entitled *Morgellons : A Thesis* (Carnicom, October 2011) for the detailed information available there. For the sake of repeated introduction to this work, the abstract of that research will be repeated below:

Morgellons : A Thesis
(Abstract):

"A substantial body of research has accumulated to make the case that the underlying organism (i.e., pathogen) of the so-called "Morgellons" condition, as identified by this researcher, is using the iron from human blood for its own growth and existence. It will also be shown that the bio-chemical state of the blood is being altered in the process. The implications of this thesis are severe as this alteration affects, amongst other things, the ability and capacity of the blood to bind to oxygen. Respiration is the source of energy for the body.

This change is also anticipated to increase the number of free radicals and to increase acidity in the body. This process also requires and consumes energy from the body to take place; this energy supports the growth and proliferation of the organism. The changes in the blood are anticipated to increase its combination with respiratory inhibitors and toxins. The changes under evaluation may occur without any obvious outward symptoms. It is also anticipated that there are consequences upon metabolism and health that extend beyond the functions of the blood. This change represents essentially a systemic attack upon the body, and the difficulties of extinction of the organism are apparent. Physiological conditions that are in probable conjunction with the condition are identified. Strategies that may be beneficial in mitigating the severity of the condition are enumerated[29]."

The ramifications of the alteration of the chemical oxidation state of iron in the blood are enormous and they can only briefly be repeated here. Let us repeat some of the important aspects from that paper:

1. Iron in a highly oxidized state (Fe+3) is a core component of the biological filaments.

2. A primary source for iron within the human body is the blood (hemoglobin).

3. A primary nutrient source for the Morgellons growth form is this same iron from the human body.

4. Iron in this highly oxidized state can no longer bind to the oxygen in hemoglobin. For iron to bind to oxygen in the blood, it must be in the Fe+2 state.

5. The oxygen carrying capacity of blood is therefore reduced as this iron within the body has been converted to this more highly oxidized state. This same oxidation state of iron supports the growth of the organism.

6. Oxidizers cause oxidation. Some of the most important oxidizers in biology are the superoxide anion, peroxide and the hydroxyl radical. The cultures of the growth form have been demonstrated to flourish in the presence of oxidized iron (Fe+3), peroxide and the hydroxyl radical.

7. Oxidizers produce free radicals. Free radicals are highly reactive molecules that "wreak havoc within the living system"[31].

8. The presence of highly oxidized iron within the organism leads to the conclusion that a greater number of free radicals are likely to exist within affected individuals.

9. Iron in the oxidized state is likely to bind to several toxic respiratory inhibitors, such as cyanide ion and carbon monoxide.

10. Oxygen deficiency is a condition known as methoglobinemia and this exists as a continued topic of research in association with the iron problem.

11. Oxidation requires energy. Energy used to support the growth of the organism is provided by the human host.

12. Any bacterial forms that infect the blood requires iron if it is to grow and reproduce. Bacterial or bacterial -like organisms have been identified as a core component of the filament growth forms by direct microscopic observations.

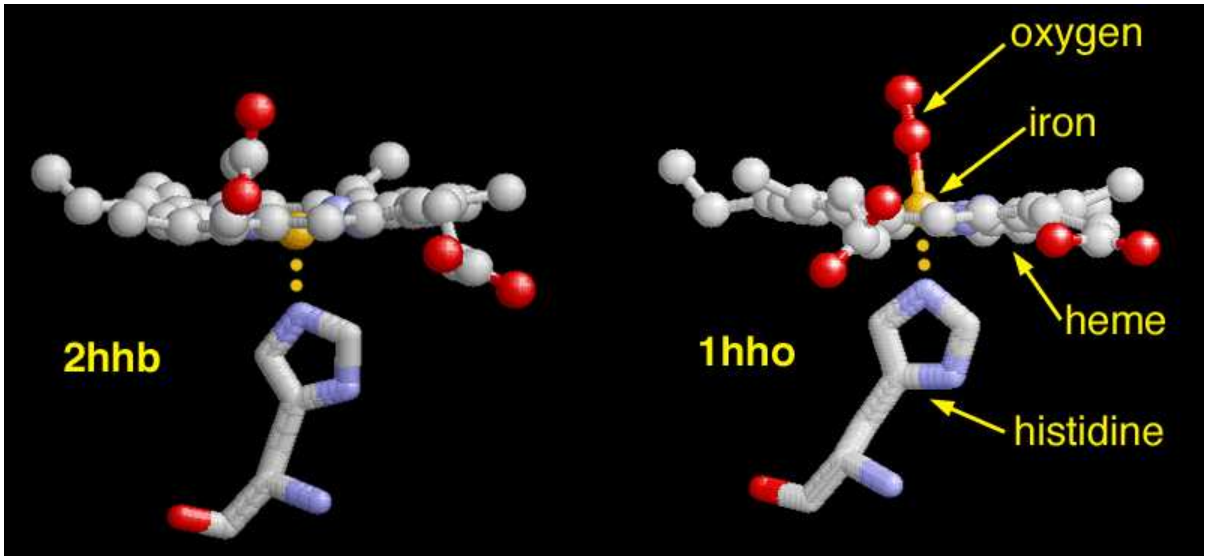
13. If the oxygen-carrying capacity of the blood is diminished, the capacity of the body to produce energy (ATP) is also diminished. Respiration is the process of imbibing energy into the body; diminishment of respiration affects all life processes.

14. A preliminary methemoglobinemia research project conducted on a series of individuals that claimed affliction with Morgellon's symptoms does indicate the presence of more highly oxidized blood from a statistical viewpoint.

15. A review of the research literature does indicate that excessive oxidation is detrimental to health. A layman's interpretation of oxidation is that of rust, or the visible wearing away of solid and metal compounds exposed to air.

16. Iron is a key element in the metabolism of almost all living organisms.

In the review of this paper, another most interesting observation has been made. Notice that the image from the Research Collaboratory for Structural Bioinformatics shows below that a significant structural component of the heme (i.e., hemoglobin) molecule is the presence of a bound ***histidine*** group. Histidine is an amino acid, and it is therefore one of the core building blocks of both human blood and human biology. At the time of writing of Morgellons : A Thesis, no special attention was called to this matter. ***It deserves this attention now***. Notice that one of the amino acids discovered in the more current research is exactly that same amino acid, histidine. In addition to the diversion of iron (an consequently the loss of oxygen) from the human blood to support the growth of the organism, it can reasonably be postulated that a similar diversion of this same amino acid chain, histidine in particular, is also taking place to support the growth of the organism. This hypothesis is further supported by the extreme damage to the red blood cells that has been directly observed and reported on in association with more severe cases of the condition.



The Heme Molecule
source:rcsb.org

Hopefully the benefits of previous research coupled with the current work can be understood, at least to some degree, at this point. We are now in position to begin the correlation of the functional groups or identified constituents with stated health impacts or symptoms. In the case of iron alone, the consequences of iron deficiency (and/or of iron diversion, in this case) are many[32]. Referring to the subject of iron deficiency (in addition to the relationships to bacterial or bacterial-like growth within the blood) from various references, the following table of likely or probable health impacts or associations can be made with relative ease at

this point. The charts below are not intended to be exhaustive of all possible associations; they are, however, intended to be representative of many of the relationships in existence resulting from the Morgellons condition.

Functional Groups or Constituent Identified within the Biological Filaments:	**Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:**
Iron (Fe+3 in the more highly oxidized state) & Bacterial or Bacterial-Like (Chlamydia P. or Chlamydia P.-like) Repeating Structure within both Blood and Filaments	Oxygen deprivation; diminished oxygen carrying capacity of the blood.
	Significant oral filament production; the presence of filament structures (ferric iron – anthocyanin complexes) within oral samples. (red wine test)
	Skin-borne filament production; skin manifestation at the more developed levels (the skin is an excretory organ).
	Extended or Chronic Fatigue
	Hair alterations, i.e., texture, thickness, loss of hair
	Gastro-intestinal imbalance
	Immune system breakdown
	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.

	Lower energy levels due to interference in the ATP production cycle; greater fatigue (iron is a transport medium for electrons within the cells)
	Any bacterial forms that infect the blood requires iron if it is to grow and reproduce.
	The smoking population may exhibit an increased incidence of the condition due to additional oxygen inhibition within the blood.
	Specific blood abnormalities
	Metabolic disruption (enzyme processes are interfered with)
	Liver toxicity, gall bladder and bile duct complications. (binding of oxidized iron to toxic molecules, e.g., cyanide and carbon monoxide)
	An increased level of acidity in the body. (Fe+3 is more acidic than Fe+2).

What we learn from the chart above is rather profound. This is that ***the existence of the iron problem alone, especially in combination with the bacterial or bacterial-like component that has also repeatedly been identified, goes a very long way in accounting for a large portion of the observed or reported health impacts from the Morgellons condition.*** This paper, therefore, reveals the importance of the investigative research that precedes this most current work. As we can see, there is much that can be learned from those earlier investigative studies.

We now introduce the subject of amino acids into the discussion. The previous paper of disclosure on this topic is entitled, Amino Acids Verified, written in November of 2012. Once again, the abstract of this work is presented:

Amino Acids Verified
(Abstract):

"The existence of certain amino acids, namely cysteine and histidine, as a dominant aspect of the "Morgellons" growth structure, appears to have been verified. This finding, along with that previously recorded on the important role that iron plays from a compositional standpoint, may be a highly important window into the structural framework of the Morgellons condition. It will also be found that deficiencies or disturbances of these particular amino acids correlate highly with symptoms that appear to frequently coexist with the condition, i.e., high oxidation levels and joint pains within the body[33]."

We shall continue the approach of correlating the constituents identified with their expected health impacts. Information related to the deficiency of amino acids is also readily available, such as in the Amino Acid Chart by Dr. Guy Wilson[34].

Candidate Functional Groups or Constituent Identified within the Biological Filaments:	**Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:**
Amino Acid Deficiency (in general)	Extended or Chronic Fatigue
	Gastro-intestinal imbalance
	Skin lesions
	Chronic decreased body temperature.
	Oxygen deprivation; diminished oxygen carrying capacity of the blood.
	Immune system breakdown
	Metabolic disruption; indications of thyroid and adrenal complications

	Hair alterations, i.e., texture, thickness, loss of hair
	Neurological impairment
	Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog”
	Joint pain
	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
	Liver toxicity
	Lower energy levels due to interference in the ATP production cycle; greater fatigue

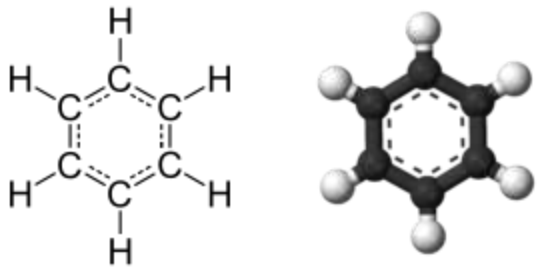
Functional Groups or Constituent Identified within the Biological Filaments:	**Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:**
Histidine Deficiency (specifically)	Joint pain
	Specific blood abnormalities
	Immune system breakdown
	Gastro-intestinal imbalance

Candidate Functional Groups or Constituent Identified within the Biological Filaments:	Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:
Cysteine Deficiency (specifically)	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
	Liver toxicity
	Skin lesions
	Hair alterations, i.e., texture, thickness, loss of hair
	Immune system breakdown

It becomes increasingly obvious, even at this early stage of presentation of identified constituents and functional groups, that the disturbances of the iron and amino acid balances within the body are at the very heart and essence of the symptoms and health impacts of the *Morgellons* condition.

It is now time to begin the examination of the functional groups from the same perspective, i.e., the comparison of the expected effects from a functional group with the actual reported health impacts of the Morgellons condition. An especially important functional group to begin this discussion with is the *aromatics*. The general properties of aromatics has been discussed earlier in this report; let us now apply ourselves more directly to the problems at hand.

We begin again with the classic example of an aromatic, the benzene ring:



A typical aromatic structure – Benzene
source : www.wikipedia.org

Benzene by itself is a toxic molecule; it leads to bone marrow depression and a lowered white blood cell count[35]. There is , however, no pretext here that the affected Morgellons sufferer is somehow directly being subjected to benzene. Organic chemistry is far more interesting and complex than this type of naiveté. One of the fascinating aspects of chemistry is that a simple and small change in a molecular structure can completely change its properties. It is this “change” that we are seeking to learn of here, and IR spectroscopy will give us some of the parts that we must work together with. As another example of aromatic variation, some of the very amino acids that we depend upon for our existence contain aromatic rings within them; surely we can understand that such a compound is not toxic to us. Our very livelihood depends upon them, so obviously whether something is aromatic or not is not the whole story. We will find, therefore, that it is the combinations of and the variations of the functional groups that are the main key to understanding the expected reactions, chemical and subsequent health impacts. Let us back up again, to the benzene ring structure (i.e., the foundation of aromatics) without any assumptions and then move forward.

We recall that one of the dominant characteristics of the benzene ring is its stability; this property is one of the defining reasons for the discovery and investigation of the aromatic class itself. Benzene did not chemically react in the ways that were originally expected. We can also recall from the earlier discussion that *electrophilic substitution* is one of the most common reactions that occurs within aromatics. It is time now to look at what are some of the substitutions that can take place.

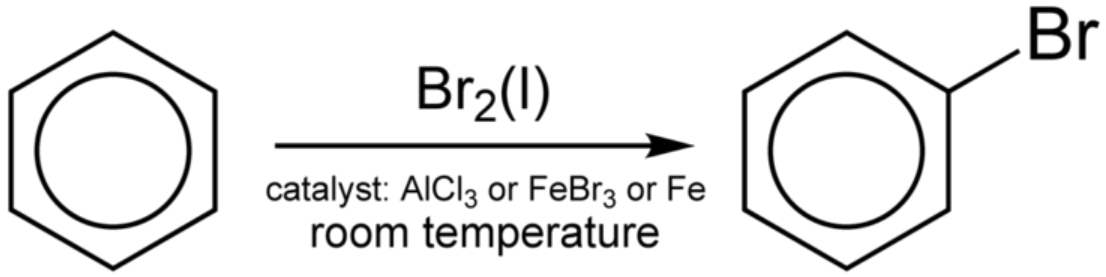
One of the most common forms of substitution that takes place is that of ***halogenation***. The halogens are the most reactive elements in existence and they are not prone to exist in their pure and free state in nature. *They are too reactive* for this to take place. Many of us are familiar with the halogens as we hear of them spoken, such as fluorine, chlorine, bromine and iodine. We also have a sense of major health concerns and toxicities that are known to exist, for example, such as the deadly hazards of a chlorine chemical spill. The controversies of fluoridation in water supplies are also know to many of us. Iodine is an

especially interesting case, as many of us also know that our bodies require small amounts of iodine and that it is somehow important to our metabolism with the thyroid. From an evolutionary perspective, it is of more than passing interest that many marine creatures and marine plant life regularly process iodine as a part of their existence. In essence, the human body has evolved to incorporate small but important levels of iodine into the body, but the remaining halogens are in general, very hazardous and harmful to our health. Within the halogens themselves, there is an order of reactivity, with fluorine being the most reactive and iodine being the least reactive.

Now, we have also emphasized that benzene, or the aromatic ring, is especially stable and is not particularly reactive in a stand alone fashion. And so, the interesting question is, how would we get the aromatic ring and the halogens to react with one another?

The answer is ***with a catalyst***. The actions of catalysts are a fascinating and wondrous aspect of chemistry in their own right, and they have also been discussed elsewhere on this site. Catalysts, by definition, lower the "threshold energy" that allows a chemical reaction to take place. The analogy that can be given is to be able to walk through a tunnel through a mountain versus having to climb over its top. It is an absolutely fascinating branch of chemistry.

So now the question is, what is a specific catalyst that will allow a halogen, such as bromine for example, to combine with an aromatic ring? It, again, is of more than passing interest that a suitable compound is that of ***iron*** bromide. Aluminum is, as well, another catalyst that may be used in the presence of bromine. Metals frequently act as enzymatic catalysts (cofactors) in biochemical reactions[36]. This discussion will become even more interesting in our future when we begin to consider the other functional groups in the question of catalytic behavior. This particular reaction involving the aromatic ring and iron bromide, looks like this:



The Bromination of Benzene
source:commons.wikimedia.org

It may now become enlightening to begin asking the question :

What might be sources for halogens to be in the body?

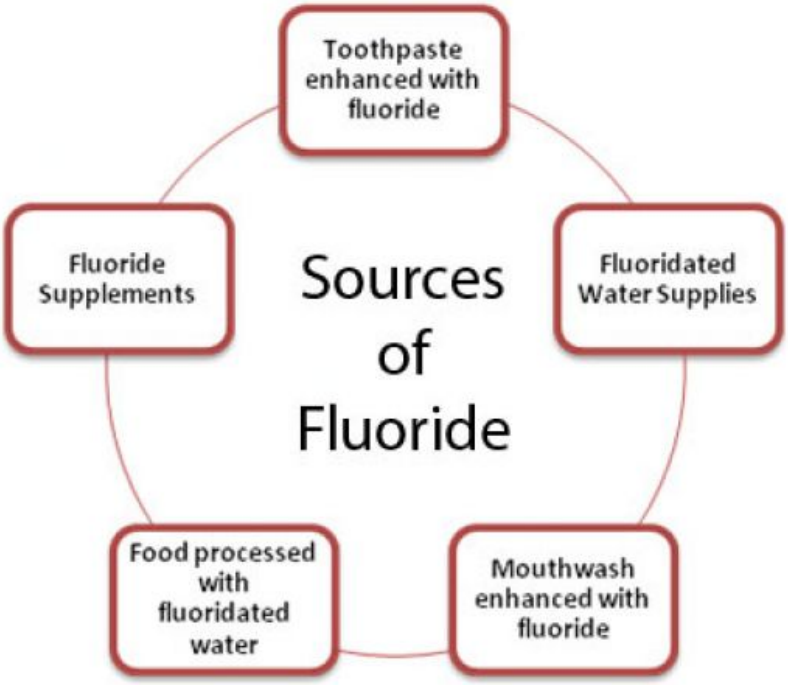
Let us start with a simple example on this:

"Purified Baby Water" with Fluoride (a halogen) added

Here are some other common examples:

Fluoridated Toothpaste, Fluoridated Tap Water

and therefore, more generally in the case of fluoride:



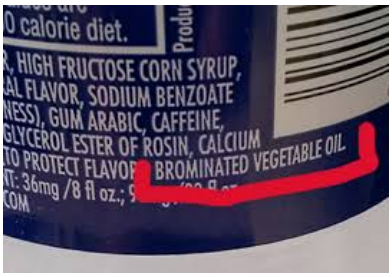
source : hubpages.com

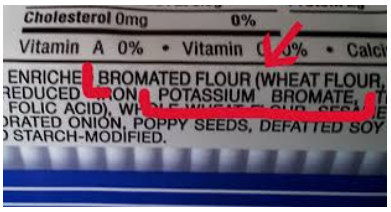
Next, let us look for some sources for bromine in our body, another very germane, common, and important case for "***aromatic halogenation***" to occur:

Bromine in breads

Bromine in sodas

Bromine in cereals





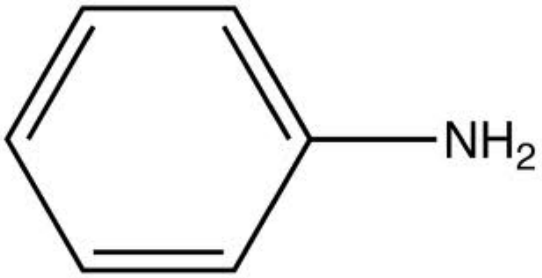
Bromine in vegetable oils	Bromine in flours	Bromine in pastas
Bromine as a disinfectant	Bromine in pesticides	Bromine as an insecticide
Bromine as a preservative	Bromine fire retardants	Bromine in pharmaceuticals

We can see now that there is no shortage of the halogens within our environment and within many diets. We must also allow for the possibility of more direct sources of halogens within the biological samples of study, let alone those introduced through environment or diet. In the presence of aromatic structures and a suitable catalyst, it is expected that aromatic halogen compounds will form.

The health impacts of aromatic halogens and additional catalysts are now to be discussed further. First, we continue with the issue of available catalysts for the halogenation of aromatics, and the situation is even more interesting than has already been presented. We have discussed that iron can act as a catalyst for halogenation, but there is another source

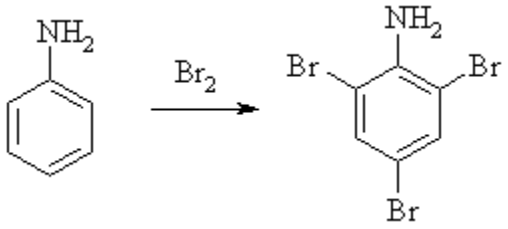
for us to consider. ***Notice the presence of the amine functional group (NH_2) in the spectroscopic analysis; this is likely be another especially important catalyst in the halogenation of an aromatic structure.***

We know that amines are a part of amino acids, but amines are a structure that can also attach to aromatics, and both are dominant functional groups that have been identified in this current work. It is entirely reasonable to consider the implication of an amine attached to an aromatic structure and this has important implications with respect to the effect upon biochemistry and health. An amine (one or more) attached to an aromatic ring is called an *aromatic amine* (or an arylamine) and it has the following example form:



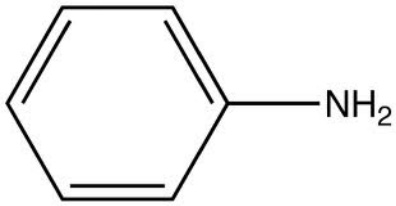
aniline, the simplest form of an aromatic armine(arylamine)
(Notice the amine group, NH2, attached to a benzene ring)
Source : chemwiki.uc.davis.edu

What is important here is that the amine group attached to an aromatic acts as a very powerful and ready catalyst for the halogenation, (i.e., the attachment of a halogen) to an aromatic ring. It is what is called an “activator”, and it increases the ease of this chemical reaction by several orders of magnitude. Recall that the function of a catalyst is to lower the threshold energy of a chemical reaction, and that is exactly what is taking place here. The following is an example of the type of reaction that is likely to occur under these conditions (in this case, even more extreme with tri-bromination taking place):



Polysubstition of aromatic with bromine in the presence of aniline (an aromatic amine)
Source : mhhe.com

This is an especially powerful and influential combination with respect to health in the human being; let us examine in part how this is so. We can begin with aniline alone, recalling its structure above:



We know that benzene is toxic, but we have already discussed that we must look at each case individually since certain amino acids themselves have an aromatic ring attached to them. This is why the functional groups are so important; it is the combination of functional groups that we must study in an effort to understand their likely influences upon health as well as the chemical reactions that are likely to take place. So, in the case here, what happens when we attach the amine group to a benzene ring (aniline) with respect to human health?

The following two effects are listed as primary effects upon human health by the Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Centers for Disease Control[37].

"Health Effects

> Aniline is irritating to the skin, eyes, and respiratory tract. Effects can result from all routes of exposure. ***Aniline induces methemoglobinemia, which impairs the delivery of oxygen to tissues***
>
> ***Aniline may also cause the destruction of red blood cells***, which manifests as acute or delayed hemolytic anemia. ***Heart, liver, and kidney effects may be secondary to hemolysis***."

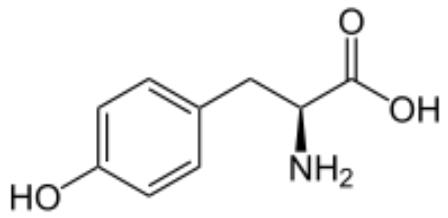
It is difficult at this point to avoid the significant correspondence between the health effects of aniline (i.e., a fundamental aromatic amine) as they are stated by the U.S. Department of Health and Human Services and the health impacts as they are now recognized by this researcher to be primary characteristics of the *Morgellons* condition.

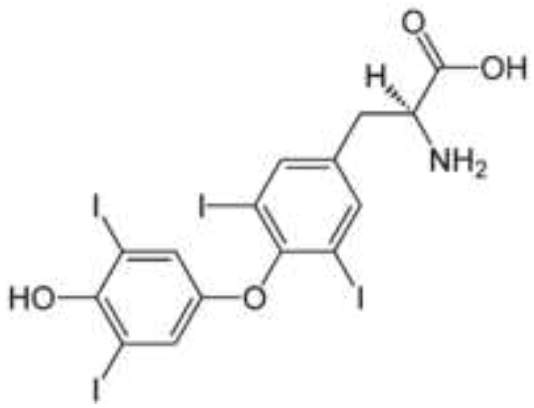
Now we turn to increasing our complexity of consideration by bringing the halogens into the picture. We do this because we now have a basis for the likely, if not very real, possibility of halogens joining into the aromatic amine structure. We know this because of the catalytic

nature of the amine group as it is attached to the ring. We also know the presence of halogens in our environment and the diets of many is now easy to justify. It is now time to begin talking about the thyroid and its importance to human health. We must also, by direct observation and research, relate this knowledge to that of the Morgellons condition.

To start, iodine is a halogen. The body needs iodine, and we have adapted and evolved to use this halogen in a very important way for our thyroid. The thyroid is the master regulator of metabolism for the human body; in many ways it can be considered to be at the crux of any health problems that we study. If the thyroid is off, the whole system is off to begin with. We go nowhere in solving our problems if the thyroid has been interrupted in any significant way. One of the places to look for a problem in the thyroid is the replacement of iodine in the thyroid by that of another halogen. Our bodies can use iodine (the only "normal" halogen in our body) but the other halogens in our body are destined to cause rather serious harm to havoc. Fluorine, chlorine and bromine are toxic to us.

Let us talk a little more about the thyroid, iodine, and a certain hormone called thyroxine as well as the amino acid, tyrosine.





Tyrosine, an amino acid	Thyroxine, a thyroid hormone

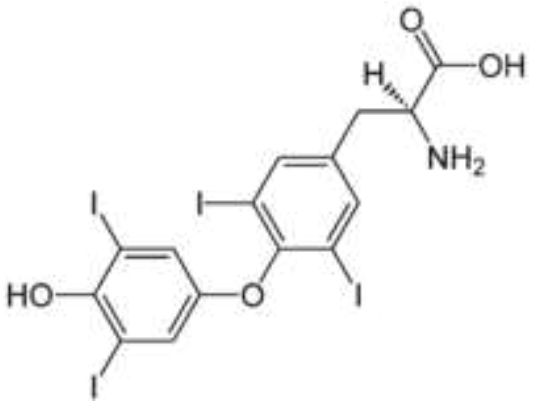
Source : wikiipedia.org

On the left side of the table above, an amino acid used by the human body, tyrosine, is shown. It is a constituent of many proteins. It can be seen that this amino acid has an aromatic side chain in its structure. Tyrosine is metabolized in the body directly to dopamine, a neurotransmitter, through an enzymatic pathway. Dopamine has been discussed earlier in this report, and it shall be discussed again at a later time. We may recall the structure of dopamine which has the functional hydroxyl group attached to the aromatic ring as well as the amine group. Dopamine is involved in motor functions, mood, attention and learning as well as other important psychological aspects.

Our interest at the current time, however, remains with the thyroid. Tyrosine is a precursor, i.e., an essential ingredient to, the formation of thyroxine, a thyroid hormone. Inspection will also show the strong similarity of chemical form between tyrosine and thyroxine, with the important addition of iodine (a halogen) that can be noticed in the previous table. Thyroxine forms by combining the amino acid tryosine with iodine.

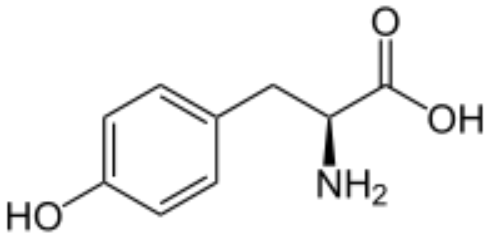
Thyroxine stimulates the production of oxygen in the body. Thyroxine is directly related to carbohydrate metabolism, protein synthesis and breakdown. Thyroxine stimulates the utilization of energy. Thyroxine directly affects the basal metabolic rate. Thyroxine stimulates the cells of the nervous system. Thyroxine is used to maintain the state of the cardiovascular system. Thyroxine stimulates the breakdown of fats. Thyroxine stimulates normal growth and development. Thyroxine stimulates the muscles to break down proteins. The thyroid is, therefore, a master regulator of metabolism for the body and any interference in that functioning is inevitably and seriously detrimental to human health.

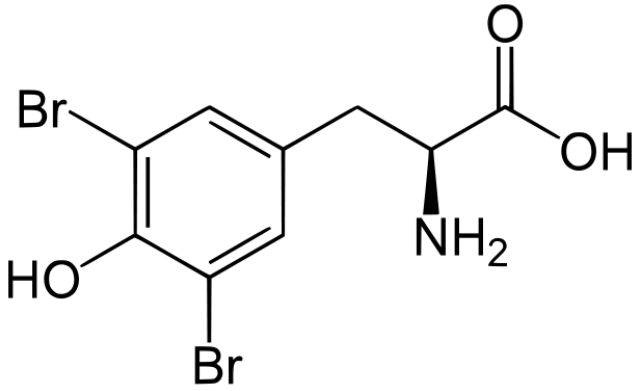
Such interference can be easily produced with the substitution of the toxic halogens on the aromatic ring in place of iodine. Given the chain of reactivity on the halogens (bromine, for example, is more reactive than iodine) and the strong evidence for the existence of an aromatic amine within the filament structure and the ready availability of the toxic halogens in both environment and diet (if not induced directly), the existence of this "interference" should come as no surprise to us.



Thyroxine, a thyroid hormone
Notice the addition of iodine (a halogen) to the molecular structure.

The structures shown below give us an example of what this *interference* can look like. On the left side of the table, we see tyrosine once again. Recall that tyrosine is a precursor to thyroxine, and that the functioning of the thyroid (i.e., metabolism in general) is dependent upon the proper existence and amounts of thyroxine within the thyroid gland. On the right side we see an example of the original amino acid (tyrosine) but now modified with the addition of a halogen (bromine) onto the aromatic ring. As long as the halogens are in existence with the catalytic amine present (let alone iron), this type of substitution reaction is one of the most common that can be expected to occur. It is difficult to not see it as being inevitable at this point. Bromine, in particular, is one of the strongest candidates for reaction, although fluorine and chlorine are not be eliminated as well, depending upon the ultimate combinations involved.





Tyrosine, an amino acid

Di-bromo tyrosine

This type of structure, a halogenated aromatic amine, will offer direct competition to the successful production of thyroxine for the thyroid. If this interference does take place, we can expect such interference in the functioning of the thyroid to develop

within the *"Morgellons"* condition. All of the research and observational evidence at this point indicates that this is exactly the case. *The simplest expression of this dilemma is with the chronic and widespread evidence and report of chronic low body temperature amongst the general population*. As such, we are now dealing with an additional issue that may truly be at the heart of the matter, in addition to that which is presented above with respect to iron, amino acids, and aromatic amines. An appalling and dreadful combination of factors is becoming overwhelmingly evident to us, and it is one that is inevitably to be confronted.

Specifically, in reference to this particular candidate structure of di-bromo tyrosine shown above, we find the following.

As predicted, this compound is a serious obstacle to normal thyroid functioning and it is used as a drug directly for that purpose. We find that:

> **"Dibromotyrosine** is an ***antithyroid*** preparation and a derivative of the natural amino acid tyrosine[38]."
>
> Furthermore, an "antithyroid" is defined as: A hormone ***antagonis*t** that acts upon thyroid hormones. This compound acts as a means to inhibit the metabolism of the individual and it is applied medically to cases of strokes, seizures and over-active thyroids (hyperthyroidism).

Before concluding this section, let us tabulate the correspondences from this most recent discussion:

Candidate Functional Groups or Constituent Identified within the Biological Filaments:	Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:
Aromatic Amines Halogenated Aromatic Amines Thyroid Inhibitors in General	Chronic decreased body temperature.
	Oxygen deprivation; diminished oxygen carrying capacity of the blood.
	Immune system breakdown
	Metabolic disruption; indications of thyroid and adrenal complications

	Extended or Chronic Fatigue
	Gastro-intestinal imbalance
	Joint pain
	Specific blood abnormalities
	An increased level of acidity in the body (may be most easily assessed by urine pH testing).
	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
	Liver toxicity, gall bladder and bile duct complications.
	Respiratory problems, including proclivities toward a chronic cough or walking pneumonia-like symptoms.
	The presence of a bacterial-like component (chlamydia-like) within or surrounding the red blood cells.

We see, therefore, that these types of compounds, (i.e. aromatic amines, halogenated aromatic amines) are at the high end of our candidate list of research observations that are correlated with expected health impacts. It should be clear to all that interference with the thyroid and basic metabolism of the human body will at the core of disease and ill health.

Unfortunately, we are not yet done with the discovery process.

We continue by addressing two other groups identified, that being the carboxylic acids and the phenols.

Carboxylic acids are, namely, acidic. We have introduced them briefly within this paper previously, and a few of those comments can be repeated here with potential relevance:

> *Carboxylic acids are one of the most important biological acids. They are* ***most acidic*** *of the common functional groups. Carboxylic acids are the largest group of organic acids. As more electronegative atoms in the acid increases, the strength of the acid increases. For example, if the hydrogen atoms in the acid (acetic acid, for example) are replaced with fluorine (****a halogen****) to produce trifluoroacetic acid, the increase in acidity is quite large. Some common examples of carboxylic acids are acetic acid, oxalic acid and formic acid.* ***Carboxylic acids are amongst the most useful building blocks for synthesizing other molecules****, both naturally and in the laboratory.*

One definition of an acid is that of an "electron acceptor" (Lewis acid). Another way of saying this is that an acid is more electron deficient than an alkaline, or basic, compound. *It seems quite fair to regard electron flow within the body as essentially the flow of energy within the body as well*. Organic chemistry itself is primarily the study of electron exchanges between molecules. An acid, therefore, seeks to draw electrons (i.e., a form of energy) towards it and, in a lay phrase, "steal" this energy. Another term for an electron acceptor ("*stealer?"*) is that of an oxidizer, or an oxidizing agent. Oxidizers cause oxidation, which is a significant topic within the current research (e.g., iron, blood). Common examples of oxidizing agents are oxygen gas (O2), the halogens, peroxides and the like.

The body itself has carboxylic acid groups within it, as it does essentially all functional groups, as they are the basis of organic chemistry. The issue is that our primary concern here is what we are studying *within* the biological filament sample and *what it is that supports and comprises the growth of that structure*. This is a significant difference, as it takes energy and molecules from the body to support its own existence. We know from previous research that amino acids comprise a part of this structure, and since amino acids are in part composed of carboxylic acids, this is an example of the displacement of nutrients from the body into the parasitic growth form of *Morgellons*. We also note that modifications of the carboxylic acid by other groups, such as other electron-seeking groups, will make the compound even more acidic. This is the example of the halogen case given above(e.g., trifluoroacetic acid), as halogens are the strongest electron-seeking groups available. The primary question from this section concerns the prospect of excessive acidity and its impact to the body – what would this be if it were to exist?

Here is a worthwhile introduction[39] by Dr. Michael Lam to some of the health issues involved; we can immediately see that the impact of excessive acidity in the body is serious business:

"The Effect of Body Acidity on Health

Excess body acidity is thought to be the first step towards premature aging, the interference with eyesight and memory, the beginning stages of wrinkling, age spots, dysfunctioning hormone systems, and a host of age related phenomena. Medical studies are confirming that body acidity is implicated in almost all diseases.

As we age we become more acidic. The body of most aged individuals is very acidic, loaded with toxic wastes in the blood stream, cells and lymphatic system. These acidic wastes come from many sources. If you were to keep your skin, muscles, organs and glands alkaline like they were when you were a baby, you would dramatically slow down the aging process.

Initial signs of body tissue acidity include:

- Feeling weak, tired and having low energy.
- Experiencing agitation, anxiety, panic attacks and depression.
- Having skin problems like eczema, psoriasis, acne and hives.
- Suffering generalized aches and pain.
- Experiencing diarrhea, constipation or bloating.
- Suffering from cramping before or during periods.
- Experiencing heartburn.
- Needing more sleep.
- Having increased dental decay.
- Feeling nauseous.
- Suffering from loss of libido.

Signs of long-term body acidity are far more serious and include:

- Osteoporosis.
- Weak immune system.
- Chronic digestive problems.
- Arthritis, joint and ligament problems.
- Kidney stones, kidney diseases and gout.
- Heart and circulation problems.
- Fungal and bacterial infections.
- Cancers.

Acidosis

Excess acidity is a condition that weakens all body systems. Excess acidity forces the body to borrow minerals including calcium, sodium, potassium and magnesium from vital organs, bones and teeth to buffer (neutralize) the acid and safely remove it from the body. As a result, the body can suffer severe and prolonged corrosion due to high acidity a condition that may go undetected for years. Acidosis leads to serious problems with major organs such as the liver, heart or kidneys. In this article, we will be looking into some of the reasons as to why we should avoid acidosis.

It can lead to weight gain and diabetes.

An acidic pH may result in weight problems such as diabetes and obesity. When our body is too acidic, we suffer from a condition known as Insulin Sensitivity. This forces excessive insulin to be produced. As a result, the body is flooded with so much insulin that it diligently converts every calorie into fat.

It is very likely that an acid pH, from an imbalanced diet, produces a condition, which stimulates the predetermined genetic response to starvation and famine. Thereafter, the body will have to increasingly hoard every calorie consumed and store it as fat.

Some people reckon that an acid pH immediately signals the powerful genetic response to an impending famine, directly interpreting with the all important and very sensitive Insulin-Glucagon Axis. When this happens, it makes the body produce more insulin than usual, and in turn, produce more fats and store it.

On the other hand, a healthy and slightly alkaline pH will yield normal fat burning metabolic activities, making no demands on the body to produce extra insulin and make fats. As such, this allows fat to be burned and naturally lost. A healthy pH diet is also less likely to have any yo-yo effects, or rebounding from a diet with additional weight gain.

We should try to maintain a healthy slightly alkaline pH so as to allow fats to be burnt normally for energy, rather than hoarded and stored under the mistaken biochemical belief of an impending famine.

Acidosis also disrupts the insulin producing pancreatic beta cells.

These beta cells are especially sensitive to pH and cannot survive if the body is too acidic. When this occurs, beta cells will lose phase with one another. Their cellular communication will be thwarted and the body's immune system will start to over-respond. Stress within the cells will increase, making them more difficult to perform adequately and survive.

It accelerates free-radical damage and premature aging.

Acidosis leads to partial lipid breakdown and destructive oxidative cascades accelerating free radical damage of cell walls and intracellular membrane structures. In this process, many healthy cells are destroyed.

Acidosis is the first step towards premature aging and accelerated oxidative cascades of cell wall destruction. Signs of acidosis may include wrinkling, age spots, failing hormonal systems, interfering with eyesight, memory, and a host of other age-related phenomena. Unwanted wastes not properly eliminated from the body actually poison the cells.

It disrupts lipid and fatty acid metabolism.

Acidosis generally disrupts lipid and fatty acid, which are involved in nerve and brain function. This disruption causes neurological problems such as MS, MD as well as problems with hormonal balance within the endocrine system.

An acidic environment also causes LDL-cholesterol to be laid down at an accelerated rate in the heart, inappropriately lining and clogging up the vascular network. In other words, an acid pH initiates electrostatic potential, damaging arterial walls, which in turn initiates a PDGF-dependent immune response, causing cholesterol oxidation and the formation of plaque with heavy metals.

It corrodes arteries, veins, and heart tissues.

Like acid eating into marble; acidosis erodes and eats into cell wall membranes of the heart, arteries and veins. During this process of erosion, our heart structures and inter connective tissues are weakened.

All living tissues are sensitive to their chemical environment. The muscle cells of the heart are no different. The entire cardiovascular system is directly affected by blood plasma pH and works as one large working “system of tubular muscles” to carry blood and nutrients to all living tissue in the body. The pumping of the heart drives blood through the arteries, veins and capillary beds and helps to regulate blood pressure and the flow of blood circulation.

The heart is normal when the pH of blood plasma is slightly alkaline, having a pH of 7.35 to 7.41. When the heart plasma rises to an acidic pH of more than 7.35, it gradually erodes away the smooth muscle tissues of the inner walls of the arteries and veins, as well as the heart itself. This process will start to weaken the structural composition of the heart, arterial and venous walls, causing lesions and microscopic tearing throughout its framework.

At the same time, an acid pH destabilizes free ionic balances within circulation, increasing the populations of positively charges particles (cations, an ion with a positive charge of electricity: H, Ca) which directly interfere with the muscle contractility (contraction and relaxation) of the heart and arteries.

Acid pH changes of blood are now thought to result in the following:

- Development of arteriosclerosis (hardening of the arteries).
- Aneurysm (widening and ballooning of artery walls).
- Arrhythmias (abnormal rhythms of the heart including tachycardia).
- Myocardial infarction (heart attacks).
- Strokes (a cardiovascular accident).

The structural weakening of the cardiovascularity also creates irregularities of blood pressure, which further exacerbates the above problems.

It alters the energy metabolism and reserves.

When your body has an acidic pH, it will prevent efficient cellular and body metabolism. Acidosis results in chemical ionic disturbances, interfering with cellular communications and functions. Acidosis reduces plus calcium binding of plasma proteins, therefore reducing the effectiveness of this intracellular signal. It also results in a disease of calcium cations (positive calcium) entry through positive calcium channels. This leads to a reduction of cardiac contractibility, or the ability of the heart to pump efficiently and rhythmically.

Positive calcium and hydrogen regulate the activities of intracellular proteins and are driven out of the cells by the “Sodium-Potassium pump” (Na-K pump). This pump provides a strong incentive for sodium to be driven into cells. It also regulates the amount of both sodium and potassium in the body stores, and uses as much as 25 percent of our caloric input daily.

Positive calcium exchanges the plus sodium, being forced out of cells, but naturally, the electrochemical gradient for positive calcium favors both positive hydrogen and positive calcium entry into cells, as there is less calcium and positive hydrogen in cells than in the

extra-cellular fluids. In extra-cellular fluids, there is 10 times more the amount of positive sodium.

In acidic solutions, less plus sodium is available, therefore slowing down the processing and induction of nutritional items going into the cells. This increases positive hydrogen and calcium buildup within the plasma, making it more available to electro-statically bind with LDL-Cholesterol.

As a result, with free positive calcium populations and channels being disrupted, calcium may become inordinately leached from the bone masses. This causes osteoporosis. In a nutshell, an acidic pH drains us of energy and disallows stored energy reserves to be used.

It slows the delivery of oxygen into the cell.

Acidosis reduces oxygen in the blood. As all living tissues, especially the heart and brain need oxygen to function; a lack of it will lead to eventual death. Having an acidic pH will reduce the amount of oxygen that is delivered to the cells. They will eventually die.

Diseases associated with acidosis.

It is important to note that the body's biochemistry is an important but just one of many tools to help the physician understand the whole body. A pH result on its own is not a diagnostic tool and is not a medical diagnosis of any disease. What then happens when the body is too acidic?

An acidic balance will:

- Decrease the body's ability to absorb minerals and other nutrients.
- Decrease energy production in the cells.
- Decrease the body's ability to repair damaged cells.
- Decrease the body's ability to detoxify heavy metals.
- Enables tumor cells to thrive.
- Make the body more susceptible to fatigue and illness.

Some people who have high acidity levels tend to exhibit these symptoms such as: anxiety, diarrhea, dilated pupils, extroverted behavior, fatigue in early morning, headaches, hyperactivity, hyper sexuality, insomnia, nervousness, rapid heartbeat, restless legs, shortness of breath, strong appetite, high blood pressure, warm dry hands and feet.

Most of the time, the body becomes acidic due to a diet rich in acids, emotional stress, toxic overload, and/or immune reactions or any process that deprives the cells of oxygen and other nutrients. When this happens, the body will try to compensate for acidic pH by using alkaline minerals such as calcium. As a result, calcium is removed from the bones, causing osteoporosis. Acidosis, which is an extended time in the acid pH state, can result in rheumatoid arthritis, diabetes, lupus, tuberculosis, osteoporosis, high blood pressure and most cancers.

Two main factors leading to cancer are an acidic pH and a lack of oxygen. As such, are we able to manipulate these two factors so as to prevent and control cancer? Everyone knows that cancer needs an acidic and low oxygen environment to survive and flourish. Research

has proven that terminal cancer patients have an acidity level of 1,000 times more than normal healthy people. The vast majority of terminal cancer patients have a very acidic pH.

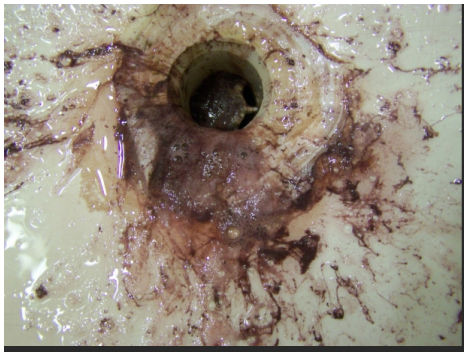
Why is this so?

The reason is simple. Without oxygen, glucose undergoing fermentation becomes lactic acid. This causes the pH of the cell to drop to 7.0. In more advance cancer cases, the pH level falls further to 6.5. Sometimes, the level can even fall to 6.0 and 5.7 or lower. The basic truth is that our bodies simply cannot fight diseases if our pH is not properly balanced."

(About The Author Michael Lam, M.D., M.P.H., A.B.A.A.M. is a specialist in Preventive and Anti-Aging Medicine. He is currently the Director of Medical Education at the Academy of Anti-Aging Research, U.S.A. He received his Bachelor of Science degree from Oregon State University, and his Doctor of Medicine degree from Loma Linda University School of Medicine, California. He also holds a Masters of Public Health degree and is Board Certification in Anti-aging Medicine by the American Board of Anti-Aging Medicine. Dr. Lam pioneered the formulation of the three clinical phases of aging as well as the concept of diagnosis and treatment of sub-clinical age related degenerative diseases to deter the aging process. Dr. Lam has been published extensively in this field.

He is the author of The Five Proven Secrets to Longevity (available on-line). He also serves as editor of the Journal of Anti-Aging Research.)

It presently becomes difficult to rank the impact of a ill-functioning thyroid against excessive acidity in the body (or the other described potential impacts, for that matter), it is impossible to choose the lesser of two when there are two heinous influences to begin with. But we must begin somewhere, and so we have by confronting the extent of the problem.



Teeth decay in direct association with chronic oral filament production characteristic of the Morgellons condition.	Extracted teeth that show serious decay in direct association with chronic oral filament production characteristic of the *Morgellons* condition.	Significant oral filament characteristic of the *Morgellons* condition (red wine test in porcelain sink). The same individual that produced this sample suffered the dental condition shown to the left.

It is not difficult to identify the health impacts of excessive acids in the body; numerous sources of information abound on this topic. From another medical source of information[40], we have the effects of over-acidity including the following:

1. Accelerated aging

2. Demineralization, or loss of the body's mineral stores

3. Fatigue

4. Impaired enzyme activity

5. Inflammation

6. Proliferation of bacteria, fungi, molds, yeast and viruses.

7. Diminishment of oxygen supply to the body

8. Reduced energy production

9. Damage to cell walls

10. Loss of protein production, including collagen and elastin.

11. Inhibition of brain and neuron functioning

12. Bone and/or teeth damage or loss

13. Osteoporosis, rheumatism, and joint pain

14. Loss of calcium

15. Inflammation and organ damage (body-wide)

16. Impairment of immune function; production of white blood cells is diminished.

17. Greater likelihood of cancer due to anaerobic metabolism within the body

There is another similar listing of the impacts of chronic acidosis in the following reference by Dr. Susan Brown[40b], and the level of correspondence is overwhelming at this point. An abbreviated listing of the effects is as follows:

1. Loss of calcium and the dissolution of bone.

2. Reduced bone formation, brittle bones, and susceptibility to fracture.

3. The loss of additional critical mineral stores, including magnesium and potassium.

4. Depressed protein metabolism, with corresponding decreases in muscle mass and cellular repair.

5. Irritation of the urinary tract and bladder.

6. Suppression of growth hormones.

7. Accelerated aging.

8. Increased production of free radicals and the lowering of immune capacity.

9. Greater oxidation of free radicals and the impairment of antioxidants.

10. Connective tissue weakening

11. Greater risk of kidney stone formation.

12. Decreased efficiency of energy (ATP) production and eventual impaired organ function.

13. Increased fluid retention in the body.

14. Intestinal bacterial disruption and digestive problems.

15. Increased yeast and fungal growth.

16. Greater proliferation of many viruses, including HIV.

17. Weakened mental capacity.

18. Decreased capacity to perform exercise.

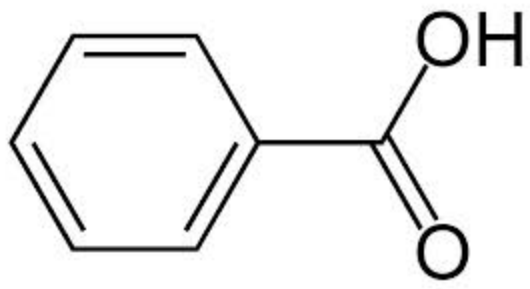
19. Increased acidity of the mouth, leading to oral bacterial imbalances, dental decay, and periodontal (gum) disease.

20. Lowered thyroid function.

21. Lowered ability of the liver to detoxify the body.

How much repetition from creditable sources is required before we must accept the obvious and the evident? Does the refrain sound familiar at this point? It is not difficult to conclude that chronic acidosis is quite likely also at the heart of the Morgellons condition.

Now let us examine a specific example which is highly relevant to the functional groups that have been specifically identified. Let us ask the question of what happens when an aromatic ring (i.e., the benzene structure) is combined with a carboxylic acid? This would appear to be a quite realistic scenario under the current knowledge. The structure looks as follows:



Benzoic Acid
source : wikipedia.org

Here we see our familiar benzene aromatic structure on the left combined with the carboxyl functional group (COOH) on the right side. This particular example therefore leads to benzoic acid. Benzoic acid is also one of the most common preservatives used in food, and according to Dr. Prior, this can lead to serious dental and demineralization issues within the body[41].

“I’m very alarmed by how much acid erosion and the resulting tooth sensitivity I’m seeing. And most people have absolutely NO IDEA that it’s happening to them. This is a Real Threat… In the past few years, I have seen more and more patients who are presenting with this problem…

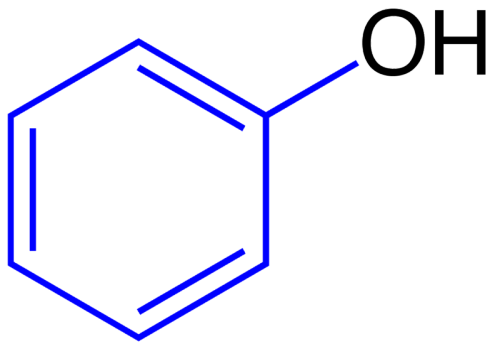
…Benzoic acid and its salt forms (sodium benzoate, potassium benzoate, etc.) are amongst the most widely used food preservative in the world. It’s cheap and very effective. Prolonged shelf life translates into higher profits. In the food industry, it is used in wide range of items from jams, juices and salad dressings to ice cream, soft drinks and candies. It’s also used in toothpaste, mouthwash, and as a rust inhibitor in anti-freeze. Being weakly acidic, benzoic acid won’t harm your enamel directly. This chemical’s preservative effect is pH dependent –it works best in a low pH (acidic) environment. Other strong acids are being added to food and beverage products to establish a low enough pH for this preservative to work. Many food substances, such as soft drinks, ice cream, and candies, are being acidified (juiced up) this way. That’s the big, hidden acid spike many of us are being hit with! On a further note, benzoic acid can combine with ascorbic acid (vitamin C) to form benzene ??” a known carcinogen. Vitamin C is often added to food or beverage products as an anti-oxidant. These two ingredients are still being used together in a wide range of beverages throughout the World (fortunately banned in North America). Another good reason to read those ingredient labels. Watch out for the Double Dose! A high sugar and strong acid combo make some of these food and beverages particularly devastating for your teeth.”

We are in a fair enough position to list the expected health effects of excessive acidity in the body; a primary culprit for this condition is an excess of carboxylic acids .

Candidate Functional Groups or Constituent Identified within the Biological Filaments:	**Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:**
Carboxylic Acids Over-Acidity (Acidosis)	Skin lesions (non or slow-healing)
	Oxygen deprivation; diminished oxygen carrying capacity of the blood.
	Immune system breakdown
	Unusual or extreme dental issues; tooth decay or loss

	Chronic itching, stinging, crawling, or biting sensations of the skin
	Neurological impairment, i.e., blurred vision or “floaters” in the eye, slurred speech, ringing of the ears (tinnitus), loss of coordination, loss of strength
	Extended or Chronic Fatigue
	Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog”
	Gastro-intestinal imbalance
	Joint pain
	An increased level of acidity in the body (may be most easily assessed by urine pH testing)
	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
	Liver toxicity, gall bladder and bile duct complications.
	The presence of a bacterial-like component (chlamydia-like) within or surrounding the red blood cells.
	Associations between oxygen deprivation, glycolysis, anaerobic respiration, cancer, energy production(ATP) and intracellular acidity[42,43].

We continue now with the phenol group; a phenol group by definition is an aromatic that has a hydroxyl group attached to it. It appears again as follows:



The phenol group
Source : commons.wikipedia.org

One of the primary characteristics of the phenol group is that it also is acidic, although not usually so much as the carboxylic acids are[44]. It is also known as carbolic acid. Some phenols, although rarely, are more acidic that carboxylic acids. The term phenol is used as the name for the specific functional group as well as being applied to a class of compounds. We have already discussed the role of acidity so that will not be repeated here; it does suggest, however, that the problem is expected to be compounded further with the existence of phenols and the health risks may be justifiably repeated. The structures of phenols are diverse, so it is difficult to generalize on their health effects as they are likely to very widely. Phenols occur naturally and they are synthesized. Phenol in its pure form is a strong neurotoxin and can lead to instant death by shutting down the neural transmission system. Phenols also occur in natural substances, such as flavonoids, coal tar, creosote, anthocyanins, salicyclic acid (precursor to aspirin) and tryosine (an amino acid, presented earlier). They are also synthesized into such products as adhesives, antiseptics and food additives. They also occur in many neurotransmitters, such as seratonin, dopamine and adrenaline. We see, therefore, that we have a wide variety of considerations and possibilities with respect to potential heath effects; of these, acidity has been duly noted.

Attention was brought forth some time ago to the interesting reaction of the biological filaments with red wines, and to the expected role of anthocyanins (i.e., pigments in the red wine)[45]. This interaction is now even more probable based upon this functional group analysis. Here was the report at that time:

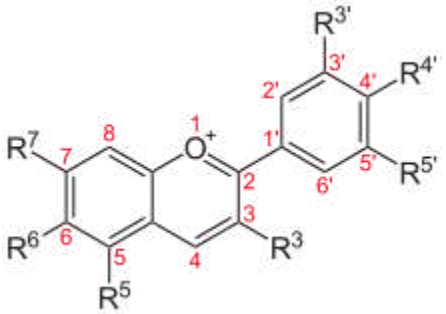
"It has long been a mystery as to why there is such a definite and visible reaction, especially of color, between the oral filament samples and red wine or related solutions. This mystery has now been resolved with a combination of investigative chemical research and the knowledge of iron changes in the body. The reason for the strong reaction is the formation of a metal complex of Fe(3+) in combination with the pigments found in red wine. Once again, at least some knowledge of *coordination chemistry* in combination with transition metal characteristics proves fruitful. Grapes, red wine and many related fruits or vegetables contain a group of pigments called anthocyanins. A search of the literature will reveal that iron, especially in the ferric state (Fe3+), will form metal complexes with these pigments. The color of many of these metal complexes is often a deep purple, exactly that which is known to occur in the combination of the oral filaments with the red wine.
It is also of interest to learn that the molecular structure of the complex, i.e, the combination of Fe(3+) with anthocyanins, has a chemical structure with some similarity to that of ferrichromes. Ferrichromes are a product of bacterial consumption of iron, and they involve the formation of strong chemical bonds that tie up the iron within a ferric metal complex.

It is the understanding of the chemistry of iron in its various states along with the important but more complex branch of coordination chemistry that has allowed us to understand the nature of the ferric iron – red wine reaction. This understanding provides one further level of verification and confirmation of the change of iron that occurs within the body as a direct result of the pathogenic metabolism."

The current work suggests strongly that we are dealing with a polycyclic (i.e., multiple rings) aromatic structure with many possibilities for both aliphatic (chain-like) and aromatic ring combinations.

Let's now look at the structure of anthocyanins in some greater detail. Recall that anthocyanins are dark pigments found in such foods as grapes, red cabbage, red wine, blackberries, cherries and other fruits and vegetables. It was predicted at the onset that the chemistry of the red wine reaction would become important in the understanding at least a portion of the nature of the biological filaments that they so strongly react with. This has shown itself to be true, but it has also taken far too long to achieve this position. With the current knowledge from this report, it is increasingly clear how and why this particular chemistry is so reactive. We appear to have the proper combination of aromatic compounds, phenols, iron and pigments to produce exactly the type of reaction referred to in the inset immediately above.

Characteristics of Anthocyanins

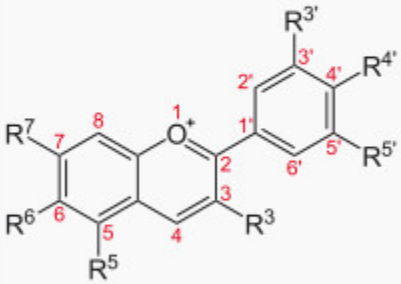


The general structure of an anthocyanin. Notice the polycyclic aromatic nature. The “R”‘s in this structure symbolize variable molecular structures or functional groups, as exemplified in the table below. A common example of an R group is the hydroxyl group (OH), which leads us to the phenol structures we are currently discussing.

An example of anthocyanin pigments in nature

An example of red cabbage pigment variation with respect to acid and alkaline conditions. Red cabbage pigment can therefore be used as an effective pH indicator.

Selected anthocyanidins and their substitutions

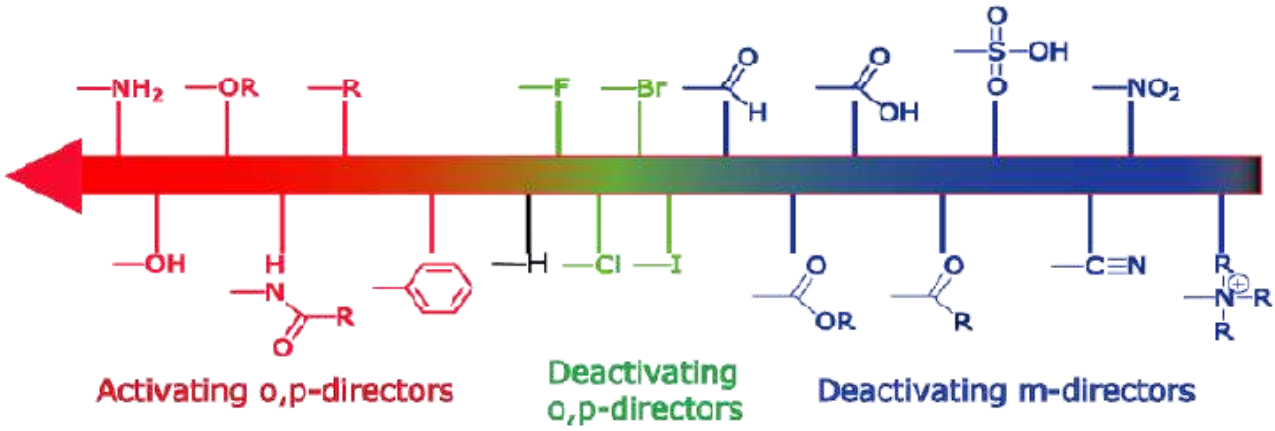
Basic structure	Anthocyanidin	R_3'	R_4'	R_5'	R_3	R_5	R_6	R_7
	Aurantinidin	–H	–OH	–H	–OH	–OH	–OH	–OH
	Cyanidin	–OH	–OH	–H	–OH	–OH	–H	–OH
	Delphinidin	–OH	–OH	–OH	–OH	–OH	–H	–OH
	Europinidin	$-OCH_3$	–OH	–OH	–OH	$-OCH_3$	–H	–OH
	Luteolinidin	–OH	–OH	–H	–H	–OH	–H	–OH
	Pelargonidin	–H	–OH	–H	–OH	–OH	–H	–OH
	Malvidin	$-OCH_3$	–OH	$-OCH_3$	–OH	–OH	–H	–OH
	Peonidin	$-OCH_3$	–OH	–H	–OH	–OH	–H	–OH
	Petunidin	–OH	–OH	$-OCH_3$	–OH	–OH	–H	–OH
	Rosinidin	$-OCH_3$	–OH	–H	–OH	–OH	–H	$-OCH_3$

Examples of variable ("R") groups that can attach to the polycyclic aromatic structure of an anthocyanin are listed in the table above. Notice the repeated presence of the OH group, leading to the phenol structure currently under discussion. **The likely similarities in structure (in part) between the biological filaments and the anthocyanin structure are offered as a partial explanation for the high chemical reactivity between the biological filaments and red wines (i.e., the so-called "red wine test")**. Incidentally, the OCH_3 group (methoxy, not identified in this report) leads to methoxy benzene (or "anisol") when attached to an aromatic ring, and it is relatively non-toxic. No special further interest in that particular group is inferred at this time. Lastly, also recall the comments previously with respect to the combination of ferric iron (Fe3+) with anthocyanins that lead to the ferrichrome (i.e., bacteria associated) compounds mentioned in the *Morgellons : A Thesis Paper*.

image sources: wikipedia.org

In summary here, we now see very interesting patterns of combinations that likely involve the presence of polycyclic aromatics, amines, iron, phenols, and halogen groupings. These combinations serve to account for many of the projected health impacts of these combinations and well as to explain (in part) the unusual and pronounced reactions of the filaments with the anthocyanin pigments of red wine.

Certain functional groups are called "activators" with respect to their chemistry with aromatic rings. An activator will be something that is electron deficient, i.e., an *electrophile*, as discussed previously. These activators lower the threshold energy for a reaction to take place. A prime example of this reaction type is the halogenation of the aromatic ring, which we have seen can potentially lead to the competition for iodine within thyroid hormones. There are tables that rank the various functional groups with respect to this *activation level*. An example of such a chart follows below:



A graphic of the activating and deactivating functional groups, from the most activating on the left to the most deactivating on the right. Activators significantly affect the threshold energy required for substitution to take place on the aromatic ring, e.g, by a halogen for example.

image source : www.erowid.org

It is of special and paramount interest that the amine group and the hydroxyl group (two of the strongest candidates of identification within this report) are at the top of this list with respect to activation energy. This means that these same functional groups are extremely likely to be involved in *electrophilic aromatic substitution* reactions herein. **Halogenation of the aromatic ring structure is one of the most likely such reactions to take place in this situation and this fact easily justifies the heightened interest by this researcher in the subjects of thyroid influences and metabolic interference.**

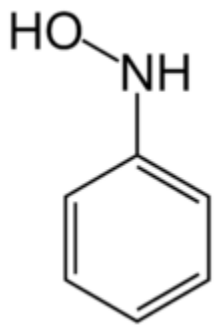
The situation becomes even more intriguing when one considers the prospect of *joint* existence of the amine group and the hydroxyl group attached to the aromatic ring. One example of a structure with this characteristic is phenylhydroxylamine. The following statement from Michigan State University, although not quite favorable bedtime reading material, is actually quite revealing in its importance[46]:

> "The strongest activating and ortho/para-directing substituents are the amino ($-NH_2$) and hydroxyl (-OH) groups…. *Bromination of both phenol and aniline is difficult to control, with di- and tri-bromo products forming readily*".

What this indicates is that the combination of both of these groups at the proper locations on the aromatic ring can easily lead to even the double and triple presence of the halogen on the ring. This would reasonably expect to present an even stronger source of interference in human metabolism, especially involving thyroid issues. This type of combination must realistically be considered in our current scenario as the three groups required (aromatic, amine and hydroxyl) appear to have been readily identified.

There is also a strong implication of cancer when certain combinations of these functional groups occur. At this time, let us bring into view a paper published by the International Agency for Research on Cancer (IARC) under the auspices of the World Health Organization (WHO). The paper is entitled "General Discussion of Common Mechanisms for Aromatic Amines"[47]. The primary function of the paper is to address the question of whether and how aromatic amines (please recall the previous discussion of aniline) cause cancer. The

answers to be found are without any serious doubt in the affirmative. As a suitable example for discussion, let us choose the following representative structure, as it is entirely within our subjects of discussion here:



Phenylhydroxylamine
image source : wikipedia

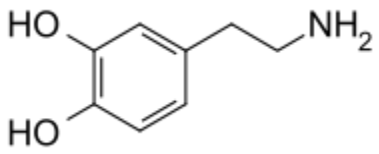
This type of structure ends up being a very important one within cancer research. A *phenyl* group occurs when you remove one of the hydrogens from a benzene ring (also called an *aryl* group). When the hydrogen is removed, various substitutions can occur. In this case we have a secondary amine attached. Another name for this aryl-N-hydroxylamine. Now let us return to the IARC paper, and extract several relevant statements from this paper:

1. “Ever since certain aromatic amines have been shown to be carcinogenic in humans the question has been raised how the chemical structure determines the biological effects, because a better understanding of this relationship could help assess the hazard and the risk associated with exposure to these chemicals. *The common denominator is an amino-group bound to an aromatic system*.”
2. “Metabolic activation was the leading concept to find out how aromatic amines cause biological effects. Both acute and chronic toxicity are held to depend on the metabolic activation of the amino group. *The key reaction responsible for all the biological activities is the N-oxidation to aryl-N-hydroxylamines.”*

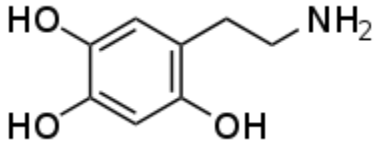
3. “The process starts with the N-oxidation of aniline to N-phenylhydroxylamine in the liver. In the erythrocytes, phenylhydroxylamine is then co-oxidized to nitrosobenzene, and Fe2+-haemoglobin is oxidized to *Fe3+-methaemoglobin*. Methaemoglobin has a reduced capacity to bind oxygen and causes a hypoxic situation.”
4. “It was, therefore, concluded that any exposure to *aniline* contributes to a background of methaemoglobin formation. …In addition to methaemoglobin formation, *erythrocytes* are damaged by reactive metabolites that react with proteins and membranes.

5. “The study of *carcinogenic N-substituted aryl compounds*, a large group of chemicals not only present at many workplaces but also in the general environment, teaches us an important lesson. If suitable conditions are chosen it is possible to demonstrate, with practically all of them, the formation of ultimate metabolites, *their reaction with DNA, RNA and proteins, mutagenic activity, the formation of methaemoglobin and other acute toxic effects*.”

Hopefully, after reading the above section, it will not be lost upon the reader that the topics of oxidation, iron detection, oxidation states of iron, amines, toxicity and liver effects, methaemoglobin, proteins, genetic engineering and damaged erythrocytes have been at the core of the research that has now been presented over a period of several years. The advantage of the current work is that a more direct mechanism to explain many of the research results may now be before us, along with others that have been detailed within this paper.



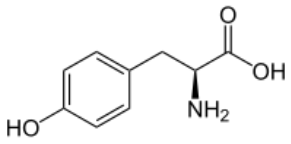
Dopamine
source : www.matzner.com

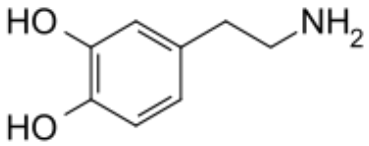


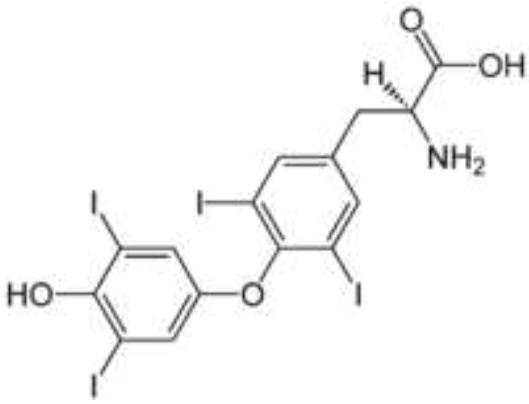
Oxidopamine
source : www.wikipedia.org

Let us now consider another relatively minor modification to the hydroxyl amine aromatic structure that can have profound ramifications. On the left side of the image group above, we have dopamine. Dopamine has been discussed earlier in this paper. As a follow through

and as an understanding of the relationships between these important chemical structures, dopamine is a metabolic product of tryrosine. Tyrosine is an amino acid, and it is also responsible for the formation of thyroxine, the primary hormone of the thyroid. For our review, here are the images for each:





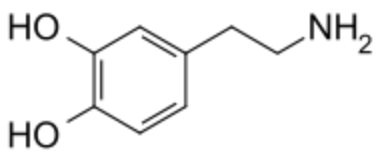


Tyrosine is an amino acid	Dopamine, a metabolic product of tyrosine	Thyroxine, a thyroid hormone that is a product of tyrosine

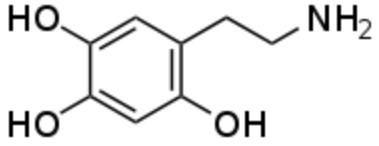
image sources : wikipedia.org

One of the reasons for presenting the comparison above is to show the importance of understanding the structure of these crucial biological compounds; we can see that relatively minor changes in a structure can completely change its function within the body. Hopefully, these structures are becoming somewhat familiar to us by now and can start to interpret them with a degree of meaning versus trepidation. We see here that dopamine (a fundamental neurotransmitter of the brain), essentially results by shifting an amine group and removing a carboxyl group from tyrosine (an amino acid). Small changes, big changes in function… We can also see that thyroxine results primarily from introducing a polycyclic (more than one aromatic ring) component into the structure of tyrosine and also brings in the all important element of iodine. We have relatively minor changes but entirely different crucial functions that develop from each.

Now, here is an interesting question for us : what if we were to introduce a minor “twist” into the dopamine structure – what might result from this change? Here is our picture again to introduce such a prospect:



Dopamine
source : www.matzner.com



Oxidopamine
source : www.wikipedia.org

We have already been introduced to dopamine on the left and hopefully we have some appreciation of its importance; ***dopamine, amongst other functions, plays a role in motor control, motivation, cognition, arousal and reward***. Research in the areas of Parkinson's Disease, schizophrenia, obsessive compulsive disorder, attention deficit hyperactivity disorder, sleep cycles, and drug addiction are also very active, to name just a few. If we can "manage", once again, the relatively small change of introducing another hydroxyl into the structure, we create a new organic compound called *oxidopamine*. Under normal circumstances, this will be a synthetic event; "normalcy" is difficult to predict at this stage of circumstance.

What, then, are the characteristics of oxidopamine compared to what we have seen for dopamine? This small change we are speaking of, the addition of a hydroxyl group (OH) to the original dopamine structure, creates oxidopamine which is a dopamine inhibitor. While dopamine is known to inhibit the development of Parkinson's Disease, in contrast, **oxidopamine encourages or induces Parkinson's Disease**. One of the main use for oxidopamine in scientific research is to induce Parkinsonism in laboratory animals such as mice, rats and monkeys[48]. Dopamine is a neurotransmitter necessary for the proper functioning of the brain; **oxidopamine is a neurotoxin**. Small change, big change in function and consequence. As cognitive abilities are substantially known to be significantly affected under serious cases of the Morgellon's condition, this type of "variation" in the neurotransmitters of the brain can not seriously dismissed. To the contrary, the research should proceed full force to quickly assess the likely numerous interactions that are presented herein, including those of neurotransmitter modifications.

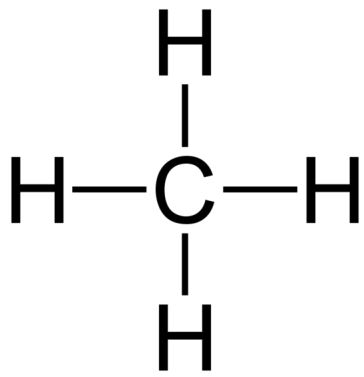
We continue by presenting the table of potential health effects from the functional group(s); in this case we will present the combination of the hydroxyl and the amine groups (due to their high ranking together on the activation energy table above):

Candidate Functional Groups or Constituent Identified within the Biological Filaments:	Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:
Phenols (in combination with aromatic amines (*note acidity and thyroid inhibition associations*)	An increased level of acidity in the body (may be most easily assessed by urine pH testing)
	Significant oral filament production; the presence of filament structures (ferric iron – anthocyanin complexes) within oral samples. (red wine test).
	Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog”
	Oxygen deprivation; diminished oxygen carrying capacity of the blood.
	Immune system breakdown
	Metabolic disruption; indications of thyroid and adrenal complications
	Unusual or extreme dental issues; tooth decay or loss
	Neurological impairment, i.e., blurred vision or “floaters” in the eye, slurred speech, ringing of the ears (tinnitus), loss of coordination, loss of strength
	An increased level of acidity in the body (may be most easily assessed by urine pH testing).
	Extended or Chronic Fatigue

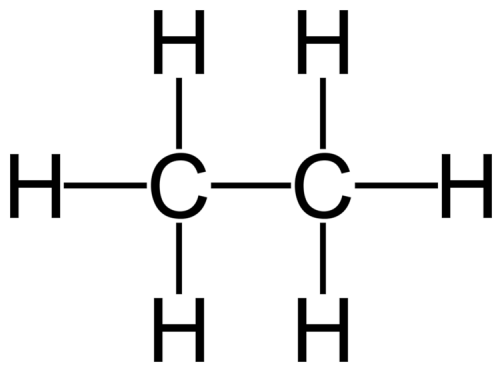
	Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog”
	Gastro-intestinal imbalance
	Joint pain
	Specific blood abnormalities
	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
	Liver toxicity, gall bladder and bile duct complications.
	Respiratory problems, including proclivities toward a chronic cough or walking pneumonia-like symptoms.
	The presence of a bacterial-like component (chlamydia-like) within or surrounding the red blood cells.
	Skin lesions (non or slow-healing)
	Chronic itching, stinging, crawling, or biting sensations of the skin

We now examine the akyl halide (also known as halogenoalkanes or as haloakanes) functional group in more detail; this group has been briefly and previously introduced briefly within this report. We recall that an akyl halide is created by substitution of

one of the hydrogens of an alkane with a halogen. An alkane, as we recall, is composed of only single carbon bonds within the structure; the simplest examples of alkanes are ethane and methane as are shown below:

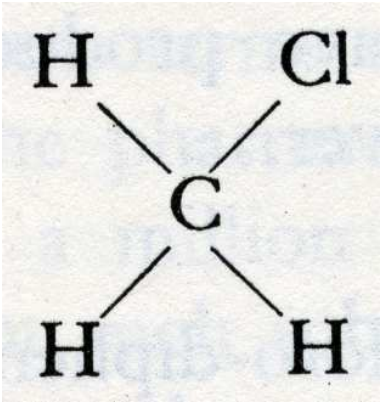


Ethane
image source : commons.wikimedia.org

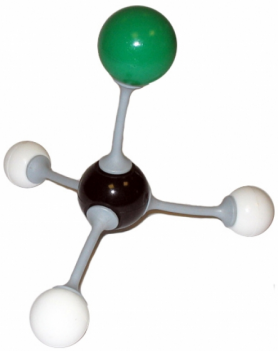


Methane
image source : commons.wikimedia.org

The simplest example of an akyl halide is, therefore, methyl chloride, which appears as follows:



Methyl Chloride, a simple example of an alkyl where chlorine(a halogen) replaces a hydrogen atom)
image source : american-buddha.com



3D view of Methy Chloride
image source : inventec.dehon.com

Akyl halides occur as a free radical reaction, and it is classified as a *radical chain reaction* process. Alkyl halides are commonly used as flame retardants, fire extinguishants, refrigerants, propellants, solvents, and pharmaceuticals. Many of the halocarbons have also

been shown to be serious pollutants and toxins, such as chlorofluorocarbon (freon) which leads to ozone depletion. Methyl bromide (where bromine, a halogen, substitutes for the hydrogen instead of chlorine, for example) is an example of a fumigant that has become controversial in its use. The use of haloalkanes has largely been curtailed because of their environmental effects and toxicity. Many alkyl halides occur naturally, but many of them are also produced synthetically. The synthetic production usually involves the use of enzymes, bacteria, fungi or microalgae. The most common form of haloalkanes is that of the bromoalkanes, involving the combination of bromine (a halogen) and alkanes. The above factors as well as the identification of this functional group should heighten our interest and concern for the existence of halogenated organics in the body, especially as they apparently occur in combination with the Morgellons condition.

Let us examine some of the expected health impacts from this functional group. In the Hazardous Material Chemistry for Emergency Responders handbook[49], the alkyl halides are listed as a **primary family of toxins** (along with amines, incidentally). The halogens as a group are toxic, with toxicity increasing as the element becomes lighter (e.g., bromine, chlorine, fluorine); iodine is unique as it has an important role in the human biochemistry but can still be toxic in sufficient amounts. We have also examined in some detail the impact of the halogens with respect to the thyroid and the seriousness of that issue. Alkyl halides are also an important precursor in the synthesis of organo-metallic compounds, another of the important classes of compounds that are presented within this report.

Haloalkenes are especially toxic to the liver[50], and can lead to fatty tissue degeneration, interference in the metabolism of fats, lipid peroxidation, fibrosis and cancer. Inhibition of protein synthesis, necrosis (cellular death due to lack of enzymatic activity), oxygen interference and free radical formation are also stated as damaging factors.

Haloalkenes can be toxic by inducing oxidative stress and the creation of intermediate free radicals[51]. Alkyl halides also react readily with the alkali metals and to a lesser extent the alkali earth metals (lithium, sodium, potassium, magnesium, calcium, etc.) to form organometallic compounds[52].

From the ILO Encyclopedia of Occupational Health and Safety, we can easily see that the internal effects of halogens upon health include detrimental impact upon the respiratory system, liver and kidneys[53].

Our table of potential health impacts from the alkyl halides and the halogens follows:

Candidate Functional Groups or Constituent Identified within the Biological Filaments:	Associated & Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:

Akyl halides Halogens	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
	Liver toxicity, gall bladder and bile duct complications.
	Immune system breakdown
	Metabolic disruption; indications of thyroid and adrenal complications
	Gastro-intestinal imbalance
	Respiratory problems, including proclivities toward a chronic cough or walking pneumonia-like symptoms.
	Recent research indicates that the urinary tract may be equally affected with the presence of the filament structures

It is now time to summarize and combine much of what has been learned thus far. A composite table will be formed, with the functional groups on the left side and the potential health impacts on the right side. Two changes will separate this table from those above : first, all functional groups and identified components will be combined into a single listing on the left side. Second, on the right side, redundancies in the potential health impacts will be eliminated. The objective here will be to present all potential health impacts in combination with all functional groups examined. In an ideal situation, the majority of the potential health impacts will be, at least in part, accountable to the presence of the specific functional groups that have been identified. This will provide some simplification, order and structure to the vast array of information that has been presented within this research report thus far.

Combined Correlation Table:
Functional Groups/Identified Components vs. Likely, Expected or Potential Health Impacts

Candidate Functional Groups or Constituent Identified within the Biological Filaments: (potential correlations are established at this stage)	**Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:** (potential correlations are established at this stage)
Iron (Fe+3 in the more highly oxidized state) Bacterial or Bacterial-Like (Chlamydia P. or Chlamydia P.-like) Repeating Structure within both Blood and Filaments Amino Acid Deficiency – in general Specific Amino Acid: Cysteine Specific Amino Acid : Histidine Amines Carboxylic Acids Aromatics Aromatic substituted Alkenes Aromatic substituted Amines Alkanes Aldehydes Phenols	
	Oxygen deprivation;diminished oxygen carrying capacity of the blood [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Significant oral filament production; the presence of filament structures (ferric iron – anthocyanin complexes) within oral samples. (red wine test) [Iron & Bacterial or Bacterial-Like Structure, Phenols – Aromatic Amines]
	Skin-borne filament production; skin manifestation at the more developed levels (the skin is an excretory organ). [Iron & Bacterial or Bacterial-Like Structure]
	Extended or Chronic Fatigue [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]

Alkyl Halides	**Hair alterations, i.e., texture, thickness, loss of hair** [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Cysteine Deficiency]
	Gastro-intestinal imbalance [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Histidine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
	Immune system breakdown [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Histidine Deficiency, Cysteine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
	The impact of increased oxidation, greater free radical presence and their damaging effects upon the body. [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Cysteine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
	Lower energy levels due to interference in the ATP production cycle; greater fatigue (iron is a transport medium for electrons within the cells) [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency]
	Any bacterial forms that infect the blood requires iron if it is to grow and reproduce. [Iron & Bacterial or Bacterial-Like Structure]

The smoking population may exhibit an increased incidence of the condition due to additional oxygen inhibition within the blood. [Iron & Bacterial or Bacterial-Like Structure]
Specific blood abnormalities [Iron & Bacterial or Bacterial-Like Structure, Histidine Deficiency,Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Phenols – Aromatic Amines]
Metabolic disruption [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
Liver toxicity, gall bladder and bile duct complications. (binding of oxidized iron to toxic molecules, e.g., cyanide and carbon monoxide) [Iron & Bacterial or Bacterial-Like Structure, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Alkyl Halides – Halogens]
An increased level of acidity in the body. [Iron & Bacterial or Bacterial-Like Structure, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Phenols – Aromatic Amines]

Skin lesions [Amino Acid Deficiency, Cysteine Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
Chronic Decreased Body Temperature [Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General]
Neurological Impairment (e.g., blurred vision, slurred speech, ringing of ears (tinnitus), loss of coordination, loss of strength) [Amino Acid Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog” [Amino Acid Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Phenols – Aromatic Amines]
Joint Pain [Amino Acid Deficiency, Histidine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis]
Liver Toxicity [Amino Acid Deficiency, Cysteine Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]

	Respiratory problems, including proclivities toward a chronic cough or walking pneumonia-like symptoms [Iron & Bacterial or Bacterial-Like Structure, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
	The presence of a bacterial-like component (chlamydia-like) within or surrounding the red blood cells [Iron & Bacterial or Bacterial-Like Structure, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Unusual or extreme dental issues; tooth decay or loss [Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Chronic itching of the skin [Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Associations between oxygen deprivation, glycolysis, anaerobic respiration, cancer, energy production (ATP), and intracellular acidity [Carboxylic Acids – Over Acidity – Acidosis]
	Research indicates the urinary tract may be equally affected with the presence of the filament structures [Alkyl Halides – Halogens]

It is apparent at this point that the majority of the reported symptoms of the Morgellons condition appear, in part, to be accounted for by the presence of the dominant functional groups that have been described in this report. This confirms the hypothesis that this specific combination of functional groups and identified components remains worthy of additional investigation as to their presence in correlation with expected health impacts. The results also appear to validate the

strategies of investigation that have been presented here, i.e., infrared spectrophotometry, functional group identification and analysis, and a study of the expected and potential health impacts from these specific functional groups.

There is another important line of research that is appropriate at this time, and this is the comparison of the spectrum attained here with any known reference sources. One of the main difficulties before us is that there is no known absolute reference spectrum which exists to compare to in this case. Any attempts or requests for such identifications and comparisons over a period of many years have yielded no such returns that I am aware of. Representative of this problem is the failure of the U.S. Environmental Protection Agency to examine a similar filament structure (as has been extensively reported upon within this site) on behalf of the public interest. Had such work been completed with accuracy and full disclosure we would likely be much further along with the environmental and health problems that remain with us. This lack is the very reason this work has been embarked upon with no presumptions of structure being made; it is a tedious process to systematically examine an compound for specific functional groups as I think is now apparent to all. Modern infrared spectrophotometers have reasonably comprehensive databases built into the instruments to afford some comparison, but this still may be of limited value with the research of an original and unknown compound. The current instrument under use has no such modern features, and all such work must be done essentially in a manual fashion – this is both tedious, difficult and time consuming. The availability of a modern IR spectrophotometer would undoubtedly ease and accelerate this process.

We are compelled to ask, therefore, what reference databases, if any, exist for the public to access and compare to. The problem has not been an easy one to solve. Nevertheless, significant progress with the need has been achieved. One of the more remarkable infrared spectral analysis databases that exist with public access exists in Japan, entitled the *Spectral Database for Organic Compounds* (SDBS), published by the National Institute of Advanced Industrial Science and Technology (AIST), Japan54. Roughly 30,000 organic compounds (with IR spectra) appear to exist within this database and this represents, without question, a sizeable library of benefit. No such comparable databases (i.e., without cost) have been identified within the U.S. public sources, or elsewhere within the world, for that matter. The database is a marvelous asset for public research and much gratitude is extended for its availability.

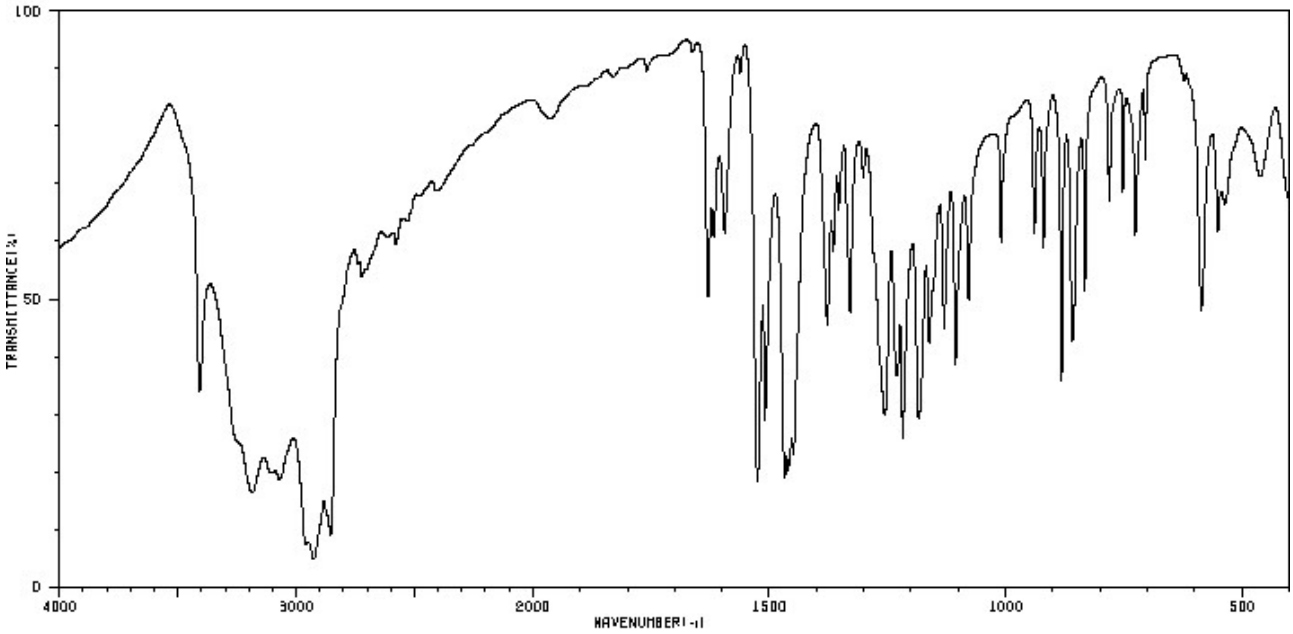
The results of a comparison search are highly intriguing. We do not expect to have a single and absolute match to any known or specific compound for a myriad of reasons. Sensitivity (or lack thereof) of the current instrument is one reason alone, let alone that of any inaccuracies or error in the work that might exist. What is expected to be of value is *any similarity of structure* that might exist (at a functional group level) between that which is proposed within this paper and that which resides within the database. The number of candidates that match closely within the search is also a point of interest. In the search that

has been performed ***a most interesting result has***, nevertheless, ***occurred*** and it is shown below as a basis for comparison. The compound below appears to represent the best match in this effort thus far, and the method used involves a comparison of all major spectral absorption peaks found in the current spectrum with those of the database. There may well be other compounds that will prove to be of interest in the future, but this one does represent a very suitable starting point for examination.

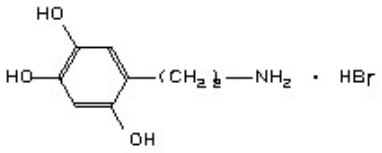
HIT-NO=5637	SCORE= ( )	SDBS-NO=13294	IR-NIDA-22223 : NUJOL MULL

5-(2-AMINOETHYL)-1.2.4-BENZENETRIOL HYDROBROMIDE

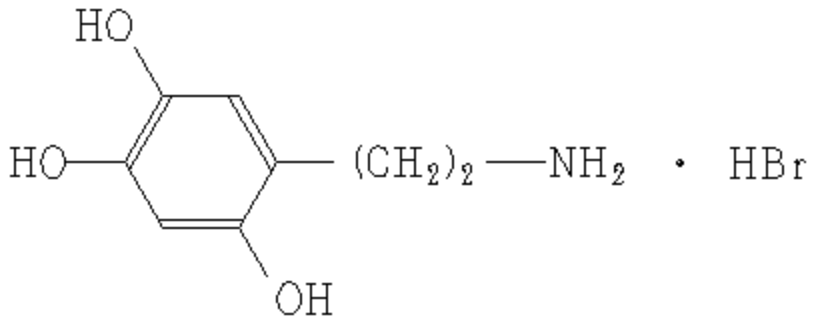
$C_8H_{11}NO_3 \cdot HBR$



3408	33	2866	8	1609	27	1267	28	1078	47
3184	16	2722	52	1467	18	1230	35	1010	58
3103	19	2711	53	1460	20	1218	25	920	57
3092	19	2678	57	1450	22	1184	28	882	34
3071	18	1630	49	1378	43	1162	41	859	41
2957	7	1594	58	1365	57	1130	45	833	49
2925	4	1526	17	1329	46	1106	37	586	46



SDBS-NO= 13294
5-(2-AMINOETHYL)-1,2,4-BENZENETRIOL HYDROBROMIDE



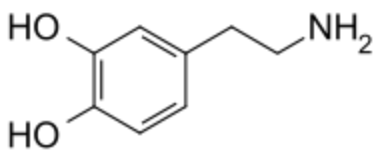
An organic compound of interest from the SDBS Database:

(2-aminoethyl)-1,2,4-benzenetriol hydrobromide
6-hydroxydopamine hydrobromide (alternate name)
2,4,5-rich hydrobromide (alternate name)

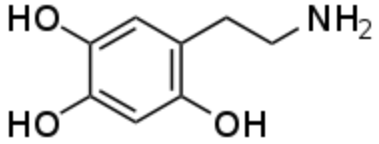
Our goal here is to look for any similarity in functional groups and structure in comparison to those identified within the biological filament sample. We are looking for broad and general overlaps at this stage and not necessarily any specifics of finite structure. We will also examine this same database compound for any anticipated or likely health impacts that may be similar to those that have been examined above.

There are several interesting observations that can be made from this compound that has been searched upon based upon the spectrum that has been observed directly and reported on here. First, we can see that *several of the very same functional groups that have been identified in the current research exist in this same compound*. Specifically, we see that we have an aromatic group, an aliphatic group, the phenol group, the amine group and a halogen showing up in this compound; these are some of the exact groups that are predicted from the analysis. These functional groups are at the very heart of the identification process that has been used in this report. They are functional groups of great significance and they have been discussed at length in this paper, along with the potential health impacts that are likely to occur with their existence.

We also note another interesting occurrence here, both visually as well as in terminology. Notice the appearance of the three hydroxyl groups that are attached to the aromatic ring. We also should now be alert to the presence of the term “oxydopamine” within the nomenclature above. Hopefully the reader will now recall the relevance and importance of our previous discussion on the oxidopamine compound, a neurotoxin. For memory sake, here is the compound shown earlier as well as a portion of the previous discussion:



Dopamine
source : www.matzner.com



Oxidopamine
source : www.wikipedia.org

> “We have already been introduced to dopamine on the left and hopefully we have some appreciation of its importance; ***dopamine, amongst other functions, plays a role in motor control, motivation, cognition, arousal and reward***. Research in the areas of Parkinson’s Disease, schizophrenia, obsessive compulsive disorder, attention deficit hyperactivity disorder, sleep cycles, and drug addiction are also very active, to name just a few. If we can “manage”, once again, the relatively small change of introducing another hydroxyl into the structure, we create a new organic compound called *oxidopamine*. Under normal circumstances, this will be a synthetic event; “normalcy” is difficult to predict at this stage of circumstance.”

We have also already established important ties and relationships between tyrosine (an amino acid) and dopamine, a neurotransmitter. We have also discussed the damaging impacts expected from oxidopamine or related compounds, especially in relation to neural functioning. We have also discussed the importance of the existence of halogens, both in relation to thyroid interference as well as their general toxicity. It is fair to say that the research that precedes this particular search in the SDBS database appears to be in good order and should serve as a strong pathway toward future investigation and knowledge to be acquired.

Lastly, before finishing up this important examination of potential health impacts, we can also look at the specific health impacts expected from this specific compound that has been queried from the SDBS database. We find, as is expected, that this particular compound depletes dopamine and brain amine levels and that it can cause significant neurological damage[55]. The previous discussion on Parkinson’s Disease does remain relevant here. We also find strong reference to memory loss and cognitive dysfunction in the presence of

depleted dopamine levels[56]. It would appear that this compound class and related compounds are worthy of further investigation by the medical and health communities in relation to the reported symptoms of the Morgellons condition.

END OF PART II

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Morgellons : A Working Hypothesis – PART III POTENTIAL MITIGATING STRATEGIES (RESEARCH BASED)

carnicominstitute.org/morgellons-a-working-hypothesis-part-iii-potential-mitigating-strategies-research-based/

Morgellons : A Working Hypothesis
Neural, Thyroid, Liver, Oxygen, Protein and Iron Disruption
(Link to Parts I, II, III – Click Here)

PART III
POTENTIAL MITIGATING STRATEGIES (RESEARCH BASED)

Clifford E Carnicom
Dec 18 2013

Art work courtesy of David Dees with permission.

Note: I am not offering any medical advice or diagnosis with the presentation of this information. I am acting solely as an independent researcher providing the results of extended observation and analysis of unusual biological conditions that are evident. Each individual must work with their own health professional to establish any appropriate course of action and any health related comments in this paper are solely for informational purposes and they are from my own perspective.

This paper seeks to identify a host of organic compounds that are likely to comprise the core physical structure of biologically produced filaments characteristic of the *Morgellons* condition. A biological oral filament sample will be analyzed for the presence of candidate organic functional groups using the methods of infrared spectrophotometry. Potential health impacts from these same core structures are examined and compared to the observed , reported and documented symptoms (in part) of this same condition. Potential mitigating strategies, from a research perspective only, are discussed.

A body of evidence, accumulated over a period of several years, reveals that the Morgellons condition is likely characterized by a host of serious physiological and metabolic imbalances. These imbalances are caused by the disruption of a variety of major body processes including, as a minimum, the regulation of metabolism by the thyroid, potential liver enlargement, a decrease of oxygen in the circulatory system, the utilization of amino acids important to the body, the oxidation of iron and a potential impact to neural pathways. The impact of this degradation to human health can be concluded to be serious, debilitating and potentially lethal in the cumulative sense; the reports of those who suffer from the condition are in alignment with these conclusions. This paper will summarize the body of work and chronology which leads to this more comprehensive hypothesis.

The health, medical and governmental communities will again be invited to offer their expertise and contributions , as well as to assume their role of responsibility and the obligations of their professions to serve the public.

This paper will be divided into three phases:

I. Identification of the functional groups / components

II. Potential health impacts of the various functional groups identified.

III. Potential mitigating strategies (research-based)

PART III
POTENTIAL MITIGATING STRATEGIES (RESEARCH BASED)

We now begin the final phase of this paper, and this is to introduce, recall and compile a host of strategies and considerations that may be helpful to mitigate some of the impacts upon health by the Morgellons condition. Some of the work that has been done previously will also be incorporated into and repeated within this section; much of this work remains especially valuable and relevant here as well. **It is important to understand that this**

information is derived from an individual research standpoint only, and that it does not represent any medical advice or diagnosis whatsoever. The usual disclaimer and caveat will be repeated before we begin the conversation:

> Note: I am not offering any medical advice or diagnosis with the presentation of this information. I am acting solely as an independent researcher providing the results of extended observation and analysis of unusual biological conditions that are evident. Each individual must work with their own health professional to establish any appropriate course of action and any health related comments in this paper are solely for informational purposes and they are from my own perspective.

Before we begin in earnest, it is worthwhile to examine the basic mechanisms of disease. In my own journey of study and discovery in the field of health and disease, it astounds me that these principles are actually so well established and yet they are often not understood and applied. There may be a fairly broad gap between what has been understood for some time and what the public is generally aware of with respect to disease; it is hopeful that this situation continues to improve. What especially interests me is that **these principles exist irrespective of the particular condition or disease examined**; we often think of each situation as being so unique and complex that we think that we can only make headway with advanced and specialized knowledge. This is not necessarily the case if we take the list that follows to heart. We find these "mechanisms" listed in the standard textbooks of pathology and, in particular, within the very first chapter of the well established tome entitled, *Robbins Pathological Basis of Disease*[57], 4th Edition. It is succinctly stated that:

> *"Although it is not always possible to determine the precise biochemical site of action of an injurious agent,* ***four*** *intracellular* ***systems are particularly vulnerable****:*
> *(a)* ***maintenance of the integrity of cell membranes***
>
> *(b)* ***aerobic respiration***
>
> *(c)* ***synthesis of enzymic and structural proteins*** *and*
>
> *(d)* ***preservation of the integrity of the genetic apparatus of the cell****."*

What we have been given here close to 25 years ago, if we care to address it, is the basis of disease in the body. **The statement is not qualified with respect to what type of disease is taking place**, *it is the basis of disease itself*. From my studies, I find no important exceptions to this as it applies to the so-called "Morgellons" condition.

We can all interpret this in our own light, but my rudimentary interpretation is that to remain healthy:

1. We must remain intact and structurally sound at a cellular level.

2. We must use oxygen efficiently and effectively in our bodies, as respiration is the source of all energy to the body.

3. We must continue to repair the actual structures of the body to compensate for decay and age.

4. We must be able to reproduce in a healthy fashion to flourish and prosper as a species.

This is the challenge that we must assume to combat disease or ill health, regardless of what the particular situation or circumstances are. It is no different here, at the most basic level of understanding, from the case of harm in general. Of course we seek to be specific as to how this is done under the specific plight of the Morgellons condition, but it is rather astounding how evident that course is when armed with the most rudimentary knowledge of pathology. Dr. Stanley Robbins will also get us off to a good start on this topic[58], with his equally succinct listing of causative agents, such as physical agents, chemical agents and drugs, infectious agents, immune responses, genetic damage and nutritional imbalances. We should never miss this grand view before becoming engrossed in the detail.

I have spoken earlier to those that seek a simple pill in life to take care of the complexities that are before us; I am not your person to listen to for a myriad of reasons. **My course of research is one that seeks the fundamental understanding of the situation and that seeks to make this information accessible to all**. Remedies to problems as needed must, therefore, also be accessible to all – at least to the highest degree possible. Specialized drugs and technologies are under the purview of others with resources, means and motive. You must seek them elsewhere. **The work of this Institute is to research and educate on behalf of the general welfare and public with the resources that the public makes available to us**.

Keeping the above foundation in mind at all times, let us go to work on the specifics. It is helpful to have the master list that has evolved before us again. This list basically identifies potential causative agents or mechanisms in conjunction with potential heath impacts (either reported or research-based). This master list, as formulated, will have numerous overlaps and redundancies occurring between the two sets, and it is not to be viewed in a style of one-to-one correspondence. Let us see if we can make some headway after the table is reviewed again:

Candidate Functional Groups or Constituent Identified within the Biological Filaments: (potential correlations are established at this stage)	**Reported, Observed or Research-Based Candidate Health Impacts or Symptoms of the Morgellons Condition:** (potential correlations are established at this stage)
Iron (Fe+3 in the more highly oxidized state) Bacterial or Bacterial-Like (Chlamydia P. or Chlamydia P.-like) Repeating Structure within both Blood and Filaments Amino Acid Deficiency – in general Specific Amino Acid: Cysteine Specific Amino Acid : Histidine Amines Carboxylic Acids Aromatics Aromatic substituted Alkenes Aromatic substituted Amines Alkanes Aldehydes Phenols Alkyl Halides	
	Oxygen deprivation;diminished oxygen carrying capacity of the blood [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Significant oral filament production; the presence of filament structures (ferric iron – anthocyanin complexes) within oral samples. (red wine test) [Iron & Bacterial or Bacterial-Like Structure, Phenols – Aromatic Amines]
	Skin-borne filament production; skin manifestation at the more developed levels (the skin is an excretory organ). [Iron & Bacterial or Bacterial-Like Structure]
	Extended or Chronic Fatigue [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Hair alterations, i.e., texture, thickness, loss of hair [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Cysteine Deficiency]

Gastro-intestinal imbalance
[Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Histidine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Alkyl Halides – Halogens]

Immune system breakdown
[Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Histidine Deficiency, Cysteine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Alkyl Halides – Halogens]

The impact of increased oxidation, greater free radical presence and their damaging effects upon the body.
[Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Cysteine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Alkyl Halides – Halogens]

Lower energy levels due to interference in the ATP production cycle; greater fatigue
(iron is a transport medium for electrons within the cells)
[Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency]

Any bacterial forms that infect the blood requires iron if it is to grow and reproduce.
[Iron & Bacterial or Bacterial-Like Structure]

The smoking population may exhibit an increased incidence of the condition due to additional oxygen inhibition within the blood. [Iron & Bacterial or Bacterial-Like Structure]
Specific blood abnormalities [Iron & Bacterial or Bacterial-Like Structure, Histidine Deficiency,Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Phenols – Aromatic Amines]
Metabolic disruption [Iron & Bacterial or Bacterial-Like Structure, Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
Liver toxicity, gall bladder and bile duct complications. (binding of oxidized iron to toxic molecules, e.g., cyanide and carbon monoxide) [Iron & Bacterial or Bacterial-Like Structure, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Alkyl Halides – Halogens]
An increased level of acidity in the body. [Iron & Bacterial or Bacterial-Like Structure, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Phenols – Aromatic Amines]
Skin lesions [Amino Acid Deficiency, Cysteine Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]

Chronic Decreased Body Temperature [Amino Acid Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General]
Neurological Impairment (e.g., blurred vision, slurred speech, ringing of ears (tinnitus), loss of coordination, loss of strength) [Amino Acid Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
Cognitive impairment, i.e., mental confusion, inability to concentrate, short term memory loss, “brain fog” [Amino Acid Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines, Phenols – Aromatic Amines]
Joint Pain [Amino Acid Deficiency, Histidine Deficiency, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Carboxylic Acids – Over Acidity – Acidosis]
Liver Toxicity [Amino Acid Deficiency, Cysteine Deficiency, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]

	Respiratory problems, including proclivities toward a chronic cough or walking pneumonia-like symptoms [Iron & Bacterial or Bacterial-Like Structure, Aromatic Amines – Halogenated Aromatic Amines – Thyroid Inhibitors in General, Phenols – Aromatic Amines, Alkyl Halides – Halogens]
	The presence of a bacterial-like component (chlamydia-like) within or surrounding the red blood cells [Iron & Bacterial or Bacterial-Like Structure, Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Unusual or extreme dental issues; tooth decay or loss [Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Chronic itching of the skin [Carboxylic Acids – Over Acidity – Acidosis, Phenols – Aromatic Amines]
	Associations between oxygen deprivation, glycolysis, anaerobic respiration, cancer, energy production (ATP), and intracellular acidity [Carboxylic Acids – Over Acidity – Acidosis]
	Research indicates the urinary tract may be equally affected with the presence of the filament structures [Alkyl Halides – Halogens]

On a macro scale, we can see that some of the more obvious issues to be addressed concern iron disruption, amino acid presence and protein rebuilding, acidity, oxidative stress, availability of oxygen, thyroid and metabolism issues, halogen toxicity and substitution concerns, joint and skeletal integrity and elasticity, blood and cellular integrity, and potential neural disruption. Unfortunately, the list is not exhaustive but it is representative of some of the health concerns that have been brought to the forefront and reported on.

One of the interesting prospects of mitigation is that improving a limited set of disrupting influences may have benefits that extend to the wider system of health. It might seem overwhelming to address such an array of problems, but the more that is understood between the relationships of mitigation, the greater are the chances of improvement to health on the whole. We must all start somewhere when we begin to assume greater responsibility for our awareness and state of health; this beginning can basically become a way of life rather than a fix to a singular problem.

Let's begin with the iron problem, as it has been discussed extensively[59,60,61] and it remains as a paramount issue. If it is accepted that iron interference is taking place, what course(s) of action might exist? Studying these previous papers, it can be seen that a fair deincreasing thegree of effort has already been extended to this question. It is appropriate to recall some of this information as follows:

1. ” Increasing the utilization and absorption of existing iron within the body. Iron is certainly one of the most important elements of the body. Referring to the Linus Pauling Institute,

> “Iron has the longest and best described history among all the micronutrients. It is a key element in the metabolism of almost all living organisms. In humans, iron is an essential component of hundreds of proteins and enzymes.”

One of the findings from the study of coordination chemistry described above is that iron has the ability to bond with numerous other molecules. For example, iron (in the Fe2+ state) preferentially bonds to oxygen. If the iron is altered to the Fe(3+) state. it will no longer bond to oxygen. In this modified state, the iron will then form additional bonds to other molecules, many of which are harmful as has also been described above. The idea of a chelator is to keep the oxygen bound in a protected state where it can not bind so easily with other, often harmful, molecules. Heme itself, within hemoglobin, is a classic example of a chelator. If our iron has been altered to where it becomes free or bound to other molecules (potentially harmful ligands), the solution to that problem would not seem to be to take more iron, any more than increasing the oxygen intake is expected to resolve a problem of oxidation.

The more effective solution would appear to be to keep the iron in a chelated state, where it is bound and protected by the expected molecules and proteins such as heme in the body. This therefore suggests that increased attention would be devoted to the study and role of chelators in human health. It does not seem reasonable that we would automatically pursue a path of increasing iron intake; indeed this process can be quite harmful and dangerous to human health. Again, the importance of consultation with the medical professionals of choice is unequivocally stated; the stakes of the issues we are speaking of are of the highest importance.

2. The inhibition of the growth of iron-consuming bacteria (and bacteria-archea like) forms.

We know now that the organism uses iron for its existence and growth. It appears that iron in the further oxidized state (i.e, Fe3+) is of primary benefit to the organism. We also know, in retrospect, that iron is a critical metabolic element within many of the bacteria (or bacteria-archaea like forms). One strategy that develops with such organism is that of inhibiting the ability of the organism to access or metabolize the iron. This once again brings up the idea of a chelator. This topic has also been discussed in an earlier paper, and introduced the role of human breast milk and its resistance to bacterial forms in infant growth[96]. Lactoferrin (found in whey) was identified as a potential strong chelating protein within that research. Transferrin is another protein chelator within the human digestive tract that serves a similar purpose, i.e., binding of the iron and consequently it becomes less accessible to iron-consuming bacteria (or bacteria-archea like forms).”

We also recall from the earlier papers mentioned an important discussion about the potential benefits of Vitamin C, NAC (N-Acetyl Cysteine) and glutathione. These three compounds are powerful anti-oxidants and they also relate directly to the issue of oxidative stress in addition to that of iron disruption:

"Three methods that appear to interfere with the molecular bonding of the iron-dipeptide complex that is now understood to be characteristic of the "Morgellons" growth structure have been established and identified. The iron-protein complex is believed to be of, or similar to, the "Rieske Protein" (iron-sulfur) form. These three methods also appear to be variably successful in reducing the oxidation state of the encapsulated iron from the Fe(III) state to the Fe(II) state. The discovered methods involve the use of ascorbic acid (Vitamin C), N-acetyl cysteine (NAC) and glutathione. The results of applying glutathione appear to be especially promising at this time, as it appears that a major disruption in the bond structure has taken place after approximately 72 hours. The methods have been established and verified through visual, chemical and spectroscopic methods and each has an effect independent of the others. **The hypothesis to be made here is that the growth of the organism itself may be interfered with as a result of this work.**"

The reader is advised to consult the Institute referenced papers for the detailed information that underlies the excerpts given:

Morgellons : In the Laboratory

Morgellons : A Thesis

Morgellons, The Breaking of Bonds and the Reduction of Iron

A Carnicom Institute research discussion on this and related issues has also been made available to the public previously. In addition, a series of videos that discuss the importance of glutathione (and its precursors) has also been included within the earlier papers.

Listen to a Research Discussion on This Topic

View A Series of Informational YouTube Videos on Glutathione
(Note the references to N-Acetyl Cysteine (NAC) and whey(lactoferrin) in the second video of the series(Dr. Mark Hyman))
(No endorsements of products to be implied or stated herein)

The question of whether to take a supplement of iron or not will not be discussed here; this harkens to the pill philosophy discussed earlier. This question will not only apply to iron supplements, it will apply to any and all questions that will be addressed concerning supplements of any kind. I will report on the research facts available to us all; we must then assume our individual responsibilities of action or discussion with the health professionals and advisors of our choice. It is not my role or position to be involved in any individual concerns or requests. I am not acting in any medical capacity whatsoever; I am acting fully and completely as an independent researcher.

The importance of the honest and dedicated involvement of the health and medical communities should be obvious to all of us; I encourage you to force this issue as it deserves.

In the particular case of iron supplements, there are risk involved as with most to all things that human beings can ingest. Specifically, unwarranted iron consumption can lead to:

> *"Iron supplements can cause indigestion, stomach pain, constipation, diarrhea, nausea, vomiting, back pain, muscle pain, chest pain, chills, lightheadedness and fainting, rapid heartbeat, fever, sweating, flushing, headache, metal taste, numbness or tingling in the hands and feet, rash and breathing problems[62]."*

In the case of high dosages, it can:

> *"High doses of iron can cause stomach and intestinal problems, liver failure, dangerously low blood pressure and death. Iron poisoning is the most common cause of poisoning deaths in children, according to Medline Plus. Symptoms of iron poisoning include bloody diarrhea, fever, nausea, sharp stomach pain and severe vomiting — possibly of blood — a blue tint to the lips, nails and palms, seizures, pale or clammy skin, shallow or rapid breathing, extreme fatigue and a weakened or fast heartbeat[63]."*

Obviously, it would seem to be of greater interest to efficiently utilize existing iron within the body rather than to assume the addition of iron automatically addresses the problem. It has also been discussed in previous papers that Vitamin C (ascorbic acid), in addition to being a powerful antioxidant, helps to increase the absorption of iron into the body. From a current reference, we can see that there are two methods by which this occurs[64]:

1. Vitamin C (ascorbic acid) helps to prevent the formation of non-soluble iron forms.

2. Vitamin C reduces iron from the ferric (Fe^{3+}) to the ferrous (Fe^{2+}) state.

The importance of this latter statement must be emphasized again, and it is the very basis of the paper entitled "The Breaking of Bonds and the Reduction of Iron" presented in November of 2012[65].

> We can see, therefore, that iron in the ferrous (Fe^{2+}) state is generally going to be more bio-available in the ferric state vs. in the ferric state, both from the standpoint of iron-oxygen binding in the blood as well as in the direct absorption of iron by mucosal cells. **It has been shown in the laboratory through Institute research that vitamin C, NAC (N-acetyl cysteine) and glutathione have each been effective in this reduction process from the ferric to the ferrous state.** It would be worthwhile to review the details of the Institute reference papers that have been cited in this report; the discussions related to glutathione and its precursors (as opposed to direct supplementation) are especially important (i.e., the use of NAC).

The roles of chelation as well as anti-oxidants, as they have been discussed, should also be given full consideration for their potential benefits prior to assuming supplementation is a logical strategy.

Understanding the co-existence between iron and bacteria should also help in the process of setting priorities for healing. Also, from the earlier paper[66]:

"A bacterium that infects the blood requires a source of iron if it is to grow and reproduce."
"Like their human hosts, bacteria need iron to survive and they must obtain that iron from the environment. While humans obtain iron primarily through the food they eat, bacteria have evolved complex and diverse mechanisms to allow them access to iron… Iron is the single most important micronutrient bacteria need to survive… understanding how these bacteria survived within us is a critical element of learning how to defeat them"We may, therefore conclude that:

The elimination of bacterial infections in the body would, therefore, obviously be beneficial in increasing the utilization of existing iron; additional iron via a supplement might simply act as a facilitating nutrient to detrimental bacterial forms.

It is also of much interest to present within in this research that NAC (N-acetyl cysteine) has two additional benefits in addition to its effectiveness as an anti-oxidant. In the following paper by the pathologist David Wheldon it is clearly stated that **NAC** also[68]:

1. has the ability to destroy chlamydial *elementary bodies*.

2. replenishes intracellular glutathione.

Those familiar with the research of this site will be aware of the extensive investigation and study that has been placed upon the "chlamydia-like" or "bacterial-like" form that has been repeatedly identified within the filament structures. The difficulties of eliminating that particular bacterial form have also been made apparent; hence the chronic respiratory symptoms that accompany its presence. The imperviousness of these "elementary bodies" (i.e, a spore-like form that remain dormant for extended periods) is at the heart of that difficulty. The use of NAC as an important precursor to the formation of glutathione (one of the most powerful anti-oxidants that exists) has also been previously discussed on this site. **The many benefits of NAC (and also its reported anecdotal success with its use with Morgellons) can now be better understood with respect to its chemistry, its specific actions of reduction (anti-oxidation) and its precursive role in the formation of glutathione. It is also of clear and immediate interest that this same paper states that the mechanism of destruction of these *elementary bodies* is by the breaking of *disulphide bonds* within the chlamydia organism** (see discussion immediately below)**.**

We now migrate to the amino acid – protein issue, and it becomes increasingly apparent that any separation of our topics is largely artificial. In addition to the withdrawal and diversion of iron from the body to support a parasitic life form, the redirection of amino acids and proteins to support such a life form is an equally serious matter. Proteins make up more than fifty-percent of our bodily constitution and they are made from amino acids; if these are interfered with in any fashion it is inevitably to our detriment. The research evidence does indicate that such interference is taking place. There are a minimum of three amino acids that exist at the top of the interest list, and it should not be surprising if there are others. The three of immediate interest include cysteine, histidine and tryrosine; again, there may well be others.

The original interest in cysteine emerged from the original observations of strength of the bonds of the filament materials, both environmental and biological. The materials, from the beginning, have shown tremendous resistance to chemical and physical agents, such as acids, alkalies and temperature. This indicates, from the onset, the likely existence of disulphide bonds, which are characteristic of both cysteine and cystine forms. Cysteine is an amino acid that is characterized by the presence of sulfur, which can then further form disulphide bonds. For example, hair is largely composed of keratin (a protein), and this protein is largely composed of such disulphide bonds. This is one reason that hair is similarly so resistant to chemical breakdown. The presence of cysteine with the filament forms (environmental and biological) has been further supported by direct observation via visible light spectrophotometry in combination with ninhydrin testing. The presence of amines (building blocks of amino acids) has been further confirmed with infra-red spectrophotometry described in this report.

The interest in histidine has also come about with the use of visible light spectrophotometry in combination with ninhydrin; please refer to the earlier paper entitled “Amino Acids Verified” for additional details of this earlier project[67]. This interest has also been extended with the knowledge of the existence of the histidine side chain in the heme (hemoglobin) molecule coupled with the observation of the extensive breakdown in the integrity of the red blood cells (erythrocytes). There is ample reason to focus on the likely existence of histidine (an amino acid) as a part of the biological filament growth form.

The interest in tryosine comes solely from the current work with IR spectrometry and the subsequent relationships that have been identified with both dopamine (a neuro-transmitter) and thyroxine (a primary metabolic hormone of the thyroid). Interest in this particular amino acid is also increased due to knowledge of some of the mechanisms of aromatic chemistry, particularly the substitution reactions involving the halogens and the hydroxyl groups. These have been discussed at length in this report and they both suggest the very real possibility of structural disturbances to both thyroxine and dopamine within the body. In addition, the observed symptoms of the Morgellons condition are primary data points in our study and must not be denied. The strong presence of metabolic, neural and cognitive interference in conjunction with the Morgellons condition gives, by itself, just cause to investigate any tyrosine disruptions that may be in place. The combination of all factors

above, IR observations, aromatic chemistry and reported symptoms all lend themselves to a deep investigation of the tyrosine, thyroxine, dopamine and oxidopamine issues and relationships.

An additional interest regarding collagen, a protein, has also developed prominently over the this last year especially in relation to the issue of joint pains. Joint pains are another of the primary symptoms that are on record in association with the Morgellons condition. These issues were introduced in a Carnicom Institute webinar presented last year and the access to it is repeated here for your listening and review:

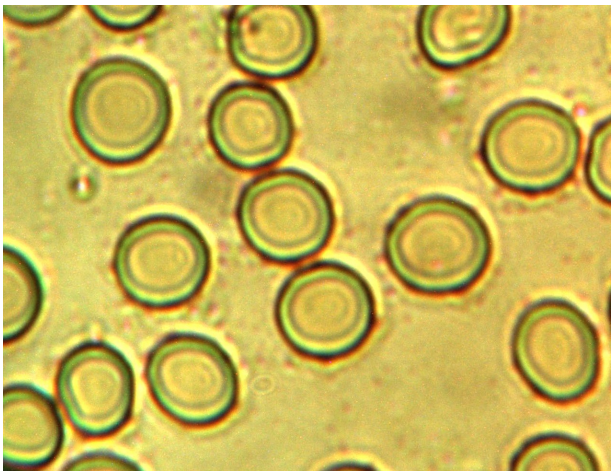
Listen to an Institute Research Discussion on Amino-Acids – Collagen

The research interest, as a result of all of the above, now includes amino acids and proteins in general. If there is strong evidence to show that a host of amino acids are either diverted or disrupted to support the growth of the filament structure then there is an equally strong case to consider supporting the body with those same amino acids. Amino acids and proteins are structural features of the body that give it both form and function; these proteins are to be rebuilt if they are lacking in the body. There should be no hesitation in promoting the use of foodstuffs in the body and the proteins are once such main group. There are numerous methods by which one might accomplish such an increase, such as in the use of supplements, protein powders, diet and the like. No specific recommendations on that approach will be given here, but such means are readily available for all to consider. We have already considered whey (a particular protein form that has value with iron chelation). Gelatin is another form of protein that is strongly associated with collagen production. Protein rich foods and/or protein powders may be additional forms of nourishment that can be considered in light of the findings. Again, the reader has the responsibility to develop any health related strategies with the counsel of their own health practitioner and the information here is provided from a research standpoint only.

Let us turn now to the issues of oxidation, oxidative stress and free radical damage. Our first clue that a serious issue with oxidation exists is with the repeated and definitive detection of highly oxidized iron within the biological filaments and the cultures that have been developed from them. Iron in the blood is required to be in the Fe+2 (ferrous) state to bind to oxygen; if the iron is changed to the Fe+3 state (ferric) it will no longer bind to oxygen and the primary function of the blood to transport oxygen transport throughout the body is no longer properly fulfilled. The iron in the biological filament is in the Fe+3 state; this means that an electron has been stolen from the iron in the blood in the Fe+2 state, and this represents a transfer in energy, in essence, from the blood of the human to support the growth of the organism.

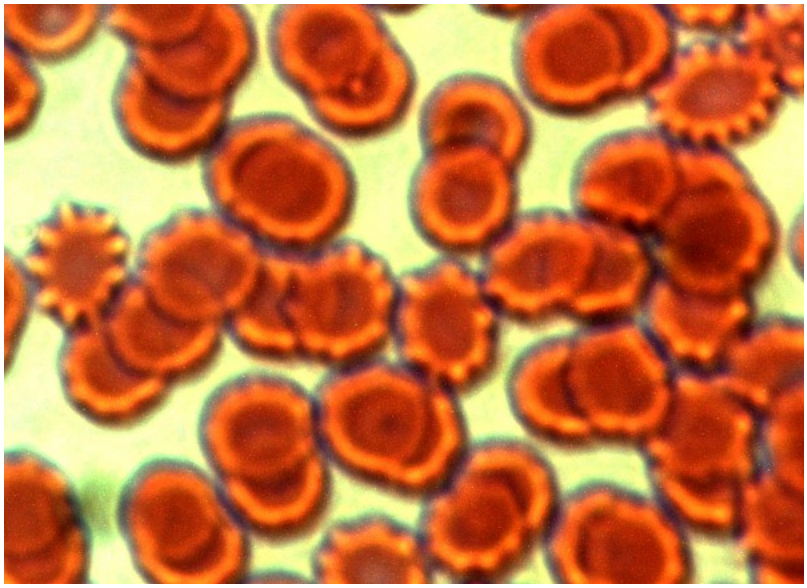
Another more direct method to investigate the state of oxygen carrying capacity in the body is to look at the blood under sufficient magnification. It has long been reported on this site that the integrity of the blood cells and the presence of the chlamydia-like bacterial structures

within the blood are direct windows into the health impacts from the Morgellons condition. Please refer to the earlier paper (amongst others) entitled “A Mechanism of Blood Damage”, authored in December of 2009[69]. In the table below are two blood slides of the same individual over a period of several years. The image to the left is during the earlier investigations of the blood as they relate to the Morgellons condition and as they were extensively reported upon within this site. The image to the right is of this same individual in a more contemporary state after considerations of the research within this site have been applied. Knowledge of the benefits of anti-oxidation strategies, along with the strategies to eliminate free radicals within the body, can potentially be demonstrated with these images as examples.



Examples of variability in general red blood cell integrity and the penetration of the cell membrane by the chlamydia-like organism within the blood. The oxygen carrying capacity of the blood is severely impacted by this breakdown in cellular integrity. The role of anti-oxidants and free-radical scavengers may be worthy of consideration in the improvements that are demonstrated in the image to the right. These images are of the same individual over a period of several years of research. It is of interest that the chlamydia-like structures appear to remain in the serum external to the cells in the image to the right; they do not appear, however, to be successful in breaching the cell membrane as they do in the image to the left. It is presumed that the state of the immune system is a primary factor in the defensive effectiveness.

It is also of passing interest that a recently acquired commercially prepared human blood slide also shows this same detrimental blood condition upon sufficient magnification:



A commercially prepared human blood slide presumably representative of the general population.

Some may consider this particular human blood slide condition as a coincidence or as irrelevant; others may be aware of strong claims by this researcher over the years that the general population appears to be subject (by varying degrees) to the health impacts of the "Morgellons" condition. Any recent statements by the National Institutes of Health (NIH) that classify "Morgellons" as a "rare condition" are in conflict with the assessment that has evolved from the research here.

Another indication of excessive oxidative stress in association with the Morgellons condition derives from a study briefly mentioned within the earlier research report, "Morgellons : A Thesis"[70]. The particular section of the paper being referred to is entitled "A Proposed Spectral Project". In this study, albeit with a limited sample, the results strongly indicate a deficiency in oxygen carrying capacity of the blood of a set of individuals claimed to be severely impacted by the Morgellons condition.

In addition, there is a body of anonymous functional medicine survey data made available to me that indicates severe oxidative stress conditions that are highly statistically significant within a separate set of individuals that claim to be severely affected by the Morgellons condition

Further, the functional group analysis from this paper reveals a host of structural features (identified, implied and plausible) that form a reasonable basis for the development of significant oxidative stress. These include (in addition to the oxidized iron – chlamydia-like presence situation), amino acid deficiencies, any cysteine deficiency, the presence of

aromatic amines (with special attention to aniline or aniline-like structures, halogenated aromatic-amines and thyroid inhibitors in general, carboxylic acids and phenols (acidosis) and alkyl halides.

If we assess that oxidative stress is likely a reality rather than a suspicion or conjecture, we then seek to address the problem with various strategies. Let us review what these strategies might include.
The first and most obvious consideration is the liberal use of anti-oxidants to combat an oxidative stress situation. First, we review what has already been presented once again in an earlier paper[71]:

" The research indicates that excessive oxidation is detrimental to health. This topic has also been discussed previously in an earlier paper[72]. Common oxidizers include the bleaches, peroxides and ozone. The research indicates, from the vantage point of this researcher, that internal use of these substances is likely to be harmful to human health. We do not solve the problem of oxidation within the body by necessarily increasing the intake of oxygen. Indeed, one of primary arguments of this paper is that the blood of the affected individual has been oxidized in a fashion that has the net effect of decreasing the oxygen carrying capacity of the blood. Excessive and misplaced oxidation also creates free radicals, which as been noted, "wreak havoc in the living system."We do not solve that problem by taking more oxygen; we work on the problem by hindering the oxidative process. The manner in which this process is conducted in the chemical world is known as reduction. In common terms, the appropriate term is that of an anti-oxidant, and many of us are familiar with that parlance.
I take stock in the following statement, again from Cotran[73]:

"Once free radicals are formed, how does the body get rid of them? There are several systems that contribute to termination or inactivation of free radical reactions:

1. **Antioxidants** (.e.g, vitamins, glutathion, transferrin..)

2. Enzymes."

The statements here are direct and understandable and come from a standard textbook in pathology. It is relatively straightforward that if a problem of excessive oxidation exists within the body, one should strongly consider the role that anti-oxidants play in reversing those effects. It is equally inadvisable, from this researcher's point of view, to compound the issue with the addition of known strong oxidizers internal to the body

Vitamins, across the board (A, B, C, D, E) are powerful antioxidants. An additional powerful antioxidant identified in the research is that of glutathion. The role of Vitamin C (ascorbic acid) in the inhibition of the culture growth has already been described.
There remain many additional anti-oxidants of importance in human health[74].

I will further discuss and present resources on the topics of oxidative stress (from a health perspective) as well as "free radicals" in more detail to further emphasize their importance. As mentioned, oxidation is defined as the loss of electrons and reduction is defined as a gain in electrons. The process of exchanging, transferring or sharing of electrons has already been described as being fundamental to essentially all biochemical reactions; electron transfer is at the core of biochemistry. It is essentially the flow of energy within living organisms. An introduction to the importance of the oxidation stress and free radical issues as they relate to health is given as follows:

> *"Mounting scientific evidence may support the important role of free radicals in the development of some diseases. Free radicals are molecules or atoms that have at least one unpaired electron which usually increases the chemical reactivity of the molecule. Environmental radiation and physiological processes in the body cause free radicals to form. Free radicals can react with other molecules to cause cell damage or DNA mutation. Molecules called antioxidants protect against free radical damage. When antioxidants are ineffective, enzymes produced by the body work to repair free radical damage. Higher levels of free radicals tend to cause increased cellular damage. This effect is called oxidative stress. Oxidative stress may contribute to cardiovascular disease and cancer. Chemical compounds found in some foods may decrease the accumulated effects of oxidative stress, thus helping to prevent disease.[75]"*

As an alternative, the pathological approach of description to the relevance of oxidative stress and free radicals to health is as follows:

> *"One important mechanism of membrane damage…is injury introduced by free radicals, particularly by activated oxygen species. It is emerging as a final common pathway of cell injury in such varied processes as chemical and radiation energy, oxygen and other gaseous toxicity, cellular aging, microbial killing by phagocytic cells, inflammatory damage, …and others… Free radicals are chemical species that have a single unpaired electron in an outer orbital… the radical is extremely reactive and unstable and enters into reactions with ..proteins, lipids, carbohydrates.. and nucleic acids…Free radicals may initiated by oxidative reactions that occur during normal metabolic processes… Iron is particularly important in toxic oxygen injury… The main effects of these reactive species are on membrane, lipid bonds… of proteins and nucleotides of DNA[76]"*

Another serious consequence of oxidative stress, and one that is increasingly important within the context of this paper, is that of neural degeneration. From the following paper on the subject of oxidative stress and neurodegenerative diseases[77], we find that:

"Though, oxygen is imperative for life, imbalanced metabolism and excess reactive oxygen species (ROS) generation end into a range of disorders such as Alzheimer's disease, Parkinson's disease, aging and many other neural disorders….Antioxidants have a wide scope to sequester metal ions involved in neuronal plaque formation to prevent oxidative stress. In addition, antioxidant therapy is vital in scavenging free radicals and ROS preventing neuronal degeneration in post-oxidative stress scenario."

It is clear that the combination of any neurotoxin with that of oxidative stress, both of which are serious contenders in the research course underway, represents a serious threat to neurological health and functioning. The list of reported, observed and research-based health impacts of the Morgellons conditions must always be at the forefront in the setting of priorities for research. The evidence of neurological dysfunction in association with the condition deserves this spotlight in combination with the findings of this report. It is of no small interest that the commonly (i.e., formerly so) attached "diagnosis" of "delusional parasitosis" (even by supposed medical professionals) appeared at the ***onset*** of public knowledge of the condition and that this ***occurred prior to any proper investigation or research***. It is fair to ask what motives and what knowledge base were in place to support such an ***a priori*** analysis and conclusion.

Continuing to present a series of references that further illustrates the extent of discussion with respect to oxidative stress, the following more comprehensive paper from Enrique Cadenas is also available[78]. In this paper, we find further clarification on what the term oxidative stress actually means. Metabolism, oxidation, and free radicals are an intrinsic part of the living process, and by themselves are not inherently "bad" or "good". Cadenas explains quite simply that **an *imbalance* between oxidants and anti-oxidants is what defines oxidative stress**. This imbalance and "consequent damage to cell molecules constitutes the basic tenet of several pathophysiological states, including neurodegeneration, cancer, mutagenesis, cardiovascular diseases, and aging. A summary of free-radical formation, reactions, impacts upon health and various defenses against these effects is further detailed within this report.

A parting comment from referenced sources regarding oxidative stress is in order, especially as it relates to the iron situation. From the Alcohol, Research and Health Journal[79], Wu Defeng, PhD discusses the role of metals in oxidative stress. He states that:

"Because of iron's critical contribution to hydroxyl radical formation, ***anything that increases the levels of free iron in the cells*** *promotes ROS [Reactive Oxygen Species] and oxidative stress"*

Recalling that free iron in the body is usually in the Fe+3 state (ferric) and that this form of iron is being definitively identified within the biological filament samples, it would seem as though the conditions for setting up an imbalance between oxidation

and reduction (i.e., oxidative stress) *have been satisfied*.

It is also recommended that an earlier paper presented be reviewed at this time, entitled "Morgellons : A Discovery and a Proposal"[80]. In this paper a series of direct observations and trials that show interactions between biological filament cultures, iron in different oxidation states, oxidants, antioxidants and culture growth is presented. The results of those trials and observations are in complete accord with the expected biochemical reactions of oxidative stress that are being discussed here.

Now that information about the mechanisms of oxidative stress are amply available, It is time to start recalling the defensive part of the equation and to reiterate some of the many notable antioxidants that exist. These anti-oxidants form the basis for one of the primary mitigating strategies under consideration to reduce oxidative stress. Once we know the source of a problem and its likely impacts, we are in a much better position to make headway in solving it. This will be the case in terms of oxidative stress, and for other problems as well.

Returning to Robbins[81] where the question was posed most directly, "Once free radicals are formed, how does the body get rid of them?"

and he answers equally succinctly, with one very important addition in this round of the research:

"There are several systems that contribute to termination of inactivation of free radical reactions. These include:

1. ..Antioxidants *[e.g., vitamins, glutathione, cysteine, transferrin]*

2. ..Enzymes *[superoxide dismutase, catalase]*

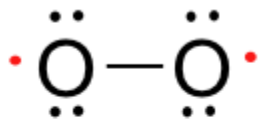
3. ..Glutathione peroxidase" *[which catalyzes reduced glutathione]*

A definite pathway for research and consultation with health professionals has been charted here for anyone that cares to regard this information. I would encourage you to consider it and evaluate it accordingly.

Let's provide a specific example of what the oxidation process entails, along with important definition of what an oxidant is. An oxidant is known by several different names, all of which are the same thing, including, oxidizing agent, oxidant, free radical, or oxidizer. An oxidant, or free radical, by definition, is "any species that contains ***one or more*** **unpaired electrons** occupying an atomic or molecular orbital by itself.[82]" An oxidizer will essentially aggressively seek an electron from another species to form a bond with another atom or structure.

It is insightful to start the study by looking at oxygen itself, a major source of oxidation in its own right. The bonding between oxygen atoms is a fascinating case that defies conventional interpretations, and further examination will show that oxygen itself is a radical with two unpaired electrons, and it is therefore known as a *diradical*. This explains some of the

reactivity characteristics of oxygen as we observe it, along with the interesting properties of paramagnetism (as can be shown with liquid oxygen experiments). Oxygen in the free state that we breath (O_2) has the following Lewis structure[83, 84, 85, 86]:



triplet oxygen
(ground state)

The diradical nature of oxygen, showing the two lone electrons of oxygen. The special reactivity of oxygen can be understood more readily with this interpretation and understanding of oxygen bonding.

We are now in a better position to understand the sequence of free radical formation from oxygen as it forms within the body. In healthy cellular metabolism, roughly 98% of oxygen is converted to water with the remaining 2% involving free radical production[87], and Cadenas shows us a sequential pathway that can take place, with the appearance of intermediate free radicals along the way (superoxide anion and they hydroxyl radical)[88]:

1.3. Formation of Oxidants by Electron Transfer Reactions

The following scheme illustrates the sequential univalent reduction of oxygen to water with formation of different intermediates: superoxide anion radical ($O_2^{\cdot -}$), hydrogen peroxide (H_2O_2), and hydroxyl radical ($HO^{\cdot}$):

$$O_2 \longrightarrow O_2^{\cdot -} \longrightarrow H_2O_2 \longrightarrow HO^{\cdot} \longrightarrow H_2O$$

Molecular oxygen	superoxide anion	hydrogen peroxide	hydroxyl radical	water

Source : Enrique Cadenas, PhD

In more common language, we have the following description of the situation[89]:

"..oxidants or free radicals are the major cause of over a hundred human diseases. The process of ageing is also hastened by the onslaught of oxidants in the body. Oxidants are normally produced during healthy cellular metabolism, wherein 98 per cent of the oxygen consumed by a cell is converted to water. The remaining one to two per cent of the unutilised oxygen is free to escape as free radicals. Free radicals or oxidants are molecules containing single unpaired electrons, and are on the lookout for electrons to pair up. Examples of oxidants are superoxide anion, hydroxy one radical, reactive oxygen species like peroxides, hydroxides and singlet oxygen."

The emphasis upon oxidative stress being an imbalance in the equation above is described again here, again in more generalized terms[90]:

"When the body's antioxidative defences are inadequate, or when the supply of nutritional antioxidants is unreliable, or when the oxidant attacks are consistently alarming, the state of balance is tilted from a state of health to a state of slow degeneration."

With this increased understanding of the route of oxidation within the body, let us return to the topic of countermeasures to oxidative stress. Fortunately, understanding the nature of the problem does place in a stronger position to pursue countermeasures. The recurring theme of the role of antioxidants is quite evident in the literature and health related papers available to us all; we simply must avail ourselves to them. Again, methods of mitigation are clearly portrayed in the Pharmaceutical Field article:[91]

"Over the years of evolution the human body has developed a whole arsenal of antioxidative enzyme systems and vitamins for its protection. Antioxidant systems of the body are critically dependent upon external dietary sources. What are these exogenous antioxidants and where are they found?

Natural Antioxidants To The Rescue

The vitamins particularly vitamin A in the form of beta-carotene and other carotenoids, vitamin C or ascorbic acid, and vitamin E as tocopherols and tocotrienols function as independently active natural dietary antioxidants. Minerals are the other dietary antioxidants that are critical to the activation of vital antioxidant enzyme systems in the body. Selenium is required for the antioxidant activity of the enzyme glutathione peroxidase. Zinc is essential for the activity of at least 90 enzymes including the antioxidant enzymes catalase and superoxide dismutase. Copper and manganese are also needed for superoxide dismutase activity.

Vitamin A and its polymers are available from brightly coloured vegetables and fruits such as carrots, apricots, dark green leafy vegetables like spinach, red, yellow and green peppers, sweet potatoes, and blue-green algae. Vitamin C is obtained from lemons, limes and other citrus and sour fruits. Vitamin E is found in nuts, whole grains, vegetable oils and to some extent in fruits and vegetables. In general, minerals are available naturally from fruits, nuts and lentils, whole cooked and germinating grains, shell-fish, vegetables and many others. Many other naturally occurring antioxidants that have been studied are pycnogenol from pine bark, grape seeds and red wine, lycopenes from tomatoes and beets, and coenzyme Q10 from red lean meat and blue-green algae. Garlic, tea and blueberries are also rich in natural antioxidants.

Conclusion

Supplementation of these dietary antioxidants in the right concentrations is important for protection against disease and premature ageing. Nutrition, like all sciences is constantly changing. Vitamins, minerals and other nutrients are no more 'boring' or old fashioned in the public consciousness. They have now been proven to act as antioxidants and protect against illnesses, repair tissues, and safeguard against the daily stresses of pollution and lifestyle. All of these substances are useful since they act as antioxidants at different levels and with different modes of actions. Consumption of these natural antioxidants through natural foods or commercially available nutraceutical or nutritional supplements will help in retarding the ageing process and increasing life spans, preventing and / or reducing the intensities of diseases like diabetes, artherosclerotic heart disease, cancer, arthritis, skin diseases, eye disorders and many other ailments."

We continue to consolidate and extend our arsenal against oxidative stress, this time with a short presentation from the University of Colorado. In the paper entitled "Free Radicals and Reactive Oxygen", we find a helpful section at the tail of the article. Two different classes of antioxidants are presented in this approach, those that are enzymatic in nature and those that are non-enzymatic. More explicitly, the section of interest is as follows:

"Mechanisms for Protection Against Radicals

Life on Earth evolved in the presence of oxygen, and necessarily adapted by evolution of a large battery of antioxidant systems. Some of these antioxidant molecules are present in all life forms examined, from bacteria to mammals, indicating their appearance early in the history of life.

Many antioxidants work by transiently becoming radicals themselves. These molecules are usually part of a larger network of cooperating antioxidants that end up regenerating the original antioxidant. For example, vitamin E becomes a radical, but is regenerated through the activity of the antioxidants vitamin C and glutathione.

Enzymatic Antioxidants

Three groups of enzymes play significant roles in protecting cells from oxidant stress:

> *Superoxide dismutases (SOD) are enzymes that catalyze the conversion of two superoxides into hydrogen peroxide and oxygen. The benefit here is that hydrogen peroxide is substantially less toxic that superoxide. SOD accelerates this detoxifying reaction roughly 10,000-fold over the non-catalyzed reaction.*
>
> $$O_2^{\bullet -} + O_2^{\bullet -} \xrightarrow{SOD} O_2 + H_2O_2$$
>
> *SODs are metal-containing enzymes that depend on a bound manganese, copper or zinc for their antioxidant activity. In mammals, the manganese-containing enzyme is most abundant in mitochondria, while the zinc or copper forms predominant in cytoplasm. Interestingly, SODs are inducible enzymes – exposure of bacteria or vertebrate cells to higher concentrations of oxygen results in rapid increases in the concentration of SOD.*
> *Catalase is found in peroxisomes in eucaryotic cells. It degrades hydrogen peroxide to water and oxygen, and hence finishes the detoxification reaction started by SOD.*
>
> *Glutathione peroxidase is a group of enzymes, the most abundant of which contain selenium. These enzymes, like catalase, degrade hydrogen peroxide. They also reduce organic peroxides to alcohols, providing another route for eliminating toxic oxidants.*

In addition to these enzymes, glutathione transferase, ceruloplasmin, hemoxygenase and possibly several other enzymes may participate in enzymatic control of oxygen radicals and their products.

Non-enzymatic Antioxidants

Three non-enzymatic antioxidants of particular importance are:

> *Vitamin E is the major lipid-soluble antioxidant, and plays a vital role in protecting membranes from oxidative damage. Its primary activity is to trap peroxy radicals in cellular membranes.*
>
> *Vitamin C or ascorbic acid is a water-soluble antioxidant that can reduce radicals from a variety of sources. It also appears to participate in recycling vitamin E radicals. Interestingly, vitamin C also functions as a pro-oxidant under certain circumstances.*
>
> *Glutathione may well be the most important intracellular defense against damage by reactive oxygen species. It is a tripeptide (glutamyl-cysteinyl-glycine). The cysteine provides an exposed free sulphydryl group (SH) that is very reactive, providing an abundant target for radical attack. Reaction with radicals oxidizes glutathione, but the reduced form is regenerated in a redox cycle involving glutathione reductase and the electron acceptor NADPH.*

In addition to these "big three", there are numerous small molecules that function as antioxidants. Examples include bilrubin, uric acid, flavonoids and carotenoids.

Readers may notice the level of overlap and correspondence that is now becoming evident in the specific compounds and substances that are regarded as highly effective antioxidants. One may refer to the previous section from the pathology textbook that emphasized the role of antioxidants, superoxide dismutase (SOD), catalase and glutathione peroxidase to become aware of certain standards that have evolved in the oxidative stress research. The special emphasis upon glutathione should also be noted, to the effect that[93]:

> "***Glutathione may well be the most important cellular defense against damage by reactive oxygen species*** [free radicals]***".***

Continuing the discussion on the importance of glutathione as an antioxidant, it is highly relevant to again recall the previous research paper entitled, "Morgellons : The Breaking of Bonds and the Reduction of Iron" from November of 2012[94]. This paper chronicles in depth research that describes the important role that glutathione is anticipated to assume in the mitigation of the Morgellons condition. There are strong conclusions arrived at within this report, particularly those that concern the ability of glutathione to break down bonds in the identified proteinaceous structures, as well as the ability of glutathione to reduce the oxidation state of iron. It is thought that it may be highly beneficial to review the research presented in that earlier paper, as the proposals mentioned are now only further corroborated with the current research. The link to this paper is presented immediately below:

Morgellons : The Breaking of Bonds and the Reduction of Iron

It may also be worthwhile to become familiar with an independent physician's evaluations of the Morgellons issue and to take note of the acknowledgement of the prospects for glutathione benefits within that same paper[95]:

Morgellon's : The Role of Atmospheric Aerosolized Biological Nano-Particulates

A few other points related to the glutathione issue bear repetition within this current collection. It has been described in earlier papers that glutathione is another one of the cases where direct supplementation may be of little value. If we suppose that a body is lacking in a particular compound, substance, or enzyme, for example, a common instinctive reaction by many is that somehow it should simply be taken as a "supplement" to fulfill that deficiency. This approach can be both unwise and foolhardy as our previous discussion on iron substantiates. When information becomes available, it is of little value unless it has been interpreted properly and comprehensively. It is another of the many reasons that professional health and medical counsel is to be sought and why education must be a lifelong pursuit. There are risks in assuming that we know more than we do. In that discussion, it was communicated that direct ingestion of glutathione appears to be of marginal value in human health. The emphasis in glutathione production within the body appears to revolve heavily around the precursor biochemistry of glutathione, more than with glutathione directly. The role of N-acetyl cysteine (NAC) has already been discussed in that regard in the previous papers mentioned. It may be wise to become familiar with that the role of "precursors", especially as they relate to the glutathione issue. Over simplification of a problem and the seeking of immediate rewards without proper understanding and comprehension can have their own price in our lives. The case for immediate and intensive participation by the health and medical communities to solve the health problems before us is patent.

Another topic of developing interest, especially in light of the current research findings, is the role that glutathione may assume in combating neural disorders, such as Parkinson's Disease. We are forced to consider the prospect of neural toxins (e.g., oxydopamine related compounds or structures) as being a potential component of the biological filament growth form. This discussion has already taken place within this paper to some extent. What is of interest here is to reacquaint ourselves with an introductory library of media on the glutathione issue, as also available in the earlier paper referenced[96].

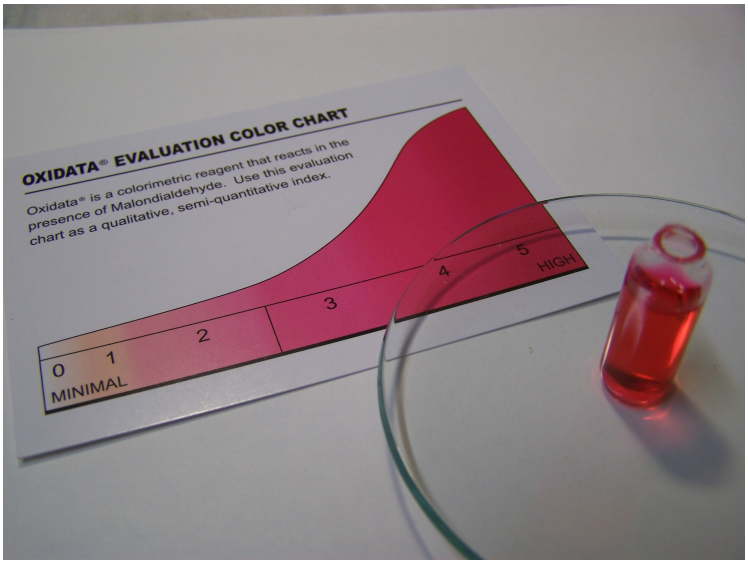
Dr. Oz Glutathione – Master Antioxidant (3 min)	Dr. Mark Hyman Glutathione – The Mother of all Antioxidants (10 min)	Dr. Don Colbert Glutathion – The Master Antioxidant Webinar Presentation (1 hr)

(No endorsements of products or services to be implied or stated herein)

Dr. David Perlmutter Glutathion Therapy – Part I (2 min)	Dr. David Perlmutter Glutathion Therapy – Part II (5 min)

(It may be of benefit to research additional presentations by these and other speakers on the issues of
glutathione, oxidative stress, chronic diseases and neuro-degenerative conditions)

An accessible and relatively inexpensive test (~$20) is available to test for oxidative stress in the body; this appears to be a highly valuable piece of information to assess with respect to the impact of the Morgellons condition and health in general. A body of information at an anonymous level is available to this researcher and it does indicate that oxidative stress may be an especially important factor, as the research also shows in numerous and substantial ways. The details of this colorimetric test are available at the manufacturer's site[96b] and it may be found through numerous sources available to the public[96c]. An example of a urine test result for one individual is shown below:



An example of the Oxidata(TM) test for oxidative stress and free radical production. This test result indicates a high level of oxidative stress for this individual.

The following topic is deserving of more consideration in the future, but the relationships between antioxidants, minerals and enzymes has been made more than once along this journey of discovery. As one pharmacist relates (now from a perspective which emphasizes nutrition), speaking of reactions that involve antioxidants[96d]:

*"**Most of these reactions need something called an enzyme to make them work**. And many of these enzymes are actually antioxidants themselves- your body even makes them- that's how important antioxidants are! **Many minerals are vital parts of these reactions too**, even though they're not antioxidants themselves (so they're equally important to have). These include selenium, manganese, copper and zinc."*

Clearly, there is more research and work to do, but the point has been made, and I suspect that it is an important one.

The next impact upon health that we transition to is that of excessive acidity, or acidosis. There is significant evidence from the research of record to implicate a serious acidic component to the Morgellons condition. The effects of excessive acidity in the body have been previously discussed, including demineralization, low energy, dental decay, weak immune system, chronic digestive problems, joint pains, bacterial and fungal infections, and many others. Please review that section of this paper to recall the numerous and significant health effects that can accrue from over-acidity within the body. Our desire here is to suggest what means might exist to counter the many problems are know the result from acidosis, and to suggest means by which extent of the problems might be monitored.

Researching the available literature, it is apparent that there is a fair amount of controversy regarding the strategies to counter the effects of excessive acidity. There are individuals that claim that eating certain food groups are effective at changing the acid state. There are individuals that claim that drinking water that is alkalized, often by various devices or with additives to the water, will result in beneficial effects. There are individuals that claim that testing the pH of either urine or saliva is representative of the body chemistry. There are individuals that will attempt to refute all of the above claims. This section of the paper is neither to advocate or to dismiss potential methods that be beneficial; it is to increase awareness of the importance of the issue and to provide a modicum of education to point the reader to various possibilities for further research, advice or action. The process of becoming aware of an idea or method does not imply or state endorsement, agreement or disagreement for that matter; it is to inform us of choices and research ideas. What is clear is that a fair amount of controversy exists on this particular subject; usually in such cases there is an abundance of misinformation or disinformation (intentional or otherwise) that must be sorted through. The profit motive of advocating certain and particular strategies, means, products and devices must also be considered in this regard.

One method to approach this problem is to focus, at an introductory level, on the medical condition of acidosis and to learn what are the identifying characteristics of that problem. The term itself is usually used in a strict medical sense applying to reduced pH of the blood, however, it is also sometimes used to express generalized excess acidity at the cellular and tissue level. Acidosis in the strictest sense of the term may well be a medical emergency, but we can use a study of that condition to our advantage to understand what systems of the

body are being most seriously impacted. It may then be considered from that point on as a matter of degree as to how much the body may be impacted by excess acidity and to what extent.

We can start with the definition of acidosis itself. Acidosis is an "increased acidity in the blood ***and other body tissue***. If not further qualified, it usually refers to acidity of the blood plasma"[97]. Note here that there is no requirement from the onset to restrict our discussion to the issue of blood only, as it is not required by definition. The measurement of blood acidity (pH) is not a common affair for the majority of us, and we prefer to not restrict our methods of measurement to that method alone. Another very important statement within this same article to recognize is that "**the rate of cellular metabolic activity affects and, at the same time, is affected by pH of the *body fluids"***[98]. We will keep this statement close at hand, as we shall see that the issue of cellular metabolism will be at the heart of excess acidity within the body.

We can once again see that we are in no way restricted to the consideration of blood alone when we are dealing with the determination of acidity within the body. Measurement of additional body fluids, such as saliva and urine, already appear to be reasonable to consider in our scope of acidity assessment, especially in a relative sense. It is also a fact that the pH of urine is regularly used as a diagnostic aid in the medical professions. Low pH values (i.e., high acid) of urine are indeed indicative of acidic conditions within the body, especially for those at risk of producing urinary stones[99]. The measurement of the pH of urine does indeed appear to be a viable point of measurement for acidic conditions within the body. One might also presume that such measurements could also be useful in a relative sense, i.e., to indicate changes of acidity within the body over a period of time.

Before seeking out the root causes of acidosis, it is worthwhile to mention that acidosis comes in two primary forms, metabolic acidosis and respiratory acidosis. Metabolic acidosis can result from the increased production of metabolic acids (please recall the discussion of organic acids earlier in this paper and the relationship to the carboxylic acid functional group) and kidney disturbances that excrete excess acids. Lactic acidosis is a form of metabolic acidosis and it is characterized by low pH in the body tissues and blood. Respiratory acidosis results from a buildup of carbon dioxide in the blood.

An investigation into the research literature reveals two strong recurring themes as the basis and cause for acidosis. The first of these will center on the issue of incomplete metabolism under conditions of reduced oxygen and the second will involve the depletion of minerals. We will now begin to document these important threads which immediately tie in with the leading statement that we called attention to:

> "**the rate of *cellular metabolic activity affects and*, at the same time, *is affected by pH* of the body fluids*"***

We refer to Dr. Michael Lam, once again, for a more lay interpretation of the importance of pH to body chemistry and for important sources of acid increase within the body. Dr. Lam will also reveal to us the primary mechanisms by which the body compensates for this change. Furthermore, Dr. Lam will make the case in his article[100] that diet, in addition to other measures, is indeed a significant factor in affecting a change in acidity within the body. This will come as no surprise as we investigate further the root causes of acidosis.

"One of the key determinants of the speed of aging and onset of degenerative diseases is the internal biochemistry and terrain of the body. **Internal biochemistry is best measured and discussed in terms of the pH**."

"The term acidosis is relative and only meant to convey a shift in total body chemistry towards the acidic direction."

The principal sources of acid buildup are:

1.) **The metabolism and/or incomplete breakdown (oxidation) of foodstuffs or metabolic "waste" produced as a by-product of cellular activity**. During normal cellular respiration and energy production , acids as produced as part or "waste" products. These acid must be "balanced", neutralized, or removed by the body's buffering and detoxification systems through the kidneys, lungs, liver, and blood.

2.) **The consumption of acid present in the food, air, and water supply**. Nitrogen emissions from automobiles and industrial plants, food dyes, sprays, waxes, preservatives, additives, artificial sweeteners, fertilizers, water pollutants, and even chloride and fluoride in tap water are just some of the highly acidic chemicals are ingested by millions everyday.

How does the body overcome the acidity?

The body undergoes an natural and ongoing balancing act constantly. Underlying regulatory forces work continually to balance an acidic body chemistry to remove excess acid and return the body to a more neutral state.

These internal buffering mechanisms include:

a. **The production of bicarbonate from the organs and cells of the body**.

b. **The removal of minerals such as calcium from bones to be used as buffering agent to neutralize the acid**. This is one of the leading causes of osteoporosis.

c. **The blowing off CO2 or carbon dioxide from the lungs**. Carbon dioxide is an acid. It leads to symptoms of shallow breathing and hyperventilation.

d. **The release of alkaline bile from the liver and alkaline digestive secretions from the pancreas** and the retention of sodium from the kidneys in response to the secretion of the hormone Aldosterone. Aldosterone is produced from adrenal gland, and stimulation of this gland leads to the feeling of internal "stress".

For those seeking a somewhat more detailed explanation of how the body compensates for an imbalance in pH, it is instructive to examine the medical model approach. In the paper entitled "Acid Base Balance in Critical Care Medicine[101]", we find a modeling process that is applied to this problem that further confirms the statements in lay language by Dr. Lam above. This model introduces the balance that occurs in the body extracellular fluid between

positive and negative ions, primarily that of sodium, potassium, calcium and magnesium on the positive side and the chloride ion on the negative side (i.e., *Strong Ion Difference*). Furthermore, it will be stated that a decrease in these positive ions will increase the hydrogen ion concentration (the very definition of an acid) through the buffering system in the body, resulting in acidosis.

This modeling process is essentially equivalent to what has been stated by Dr. Lam, i.e., demineralization will accompany acidity within the body.

Acid-base chemistry in the body can become a complex affair, and a detailed examination of the situation, factors and chemistry can be found at Dr. Grogono's site entitled "Acid-Base Tutorial[102]". A good introduction to acid-base chemistry is provided at the onset, where the two essential factors are described as follows:

> "The Bird's Eye-View, Two Components:
> **Respiratory:** When breathing is inadequate carbon dioxide (respiratory acid) accumulates. The extra CO_2 molecules combine with water to form carbonic acid which contributes to an acid pH. The treatment, if all else fails, is to lower the PCO_2 by breathing for the patient using a ventilator.
>
> **Metabolic:** When normal metabolism is impaired – acid forms, e.g., poor blood supply stops oxidative metabolism and lactic acid forms. This acid is not respiratory so, by definition, it is "metabolic acid." If severe, the patient may be in shock and require treatment, possibly by neutralizing this excess acid with bicarbonate, possibly by allowing time for excretion/metabolism."

> As we continue to strike toward the heart of acidosis, at least from the more critical medical emergency perspective, one cannot help but notice that efficient aerobic respiration, complete metabolism and the lack of oxidative stress are at the absolute core of the issue. These issues have emerged time and time again within this current research, and it would be foolhardy to ignore this deep-seated theme at this point. In addition, we have learned that demineralization of the body (e.g., degradation of bone and teeth as examples) are expected to occur as a result of an acidic condition because of the body's natural buffering systems that attempt to maintain ionic balance within the blood and body fluids.

The topic of excess acidity was first posited several years ago, in the paper (2010) entitled, Morgellons : A Discovery and a Proposal[103], where attention was called to the following:

> *"In the culture environment, it has been established that the organism(s) flourish within an acidic environment. In addition, it has also been stated in earlier reports that many biochemical reactions only take place within a narrow pH [acid or alkaline] range. Therefore, one of the first strategies to consider is to change the acidity or alkalinity of the growth environment and see if progress results. What has been observed in the cultures thus far is that an increase to the alkaline side does indeed appear to inhibit the growth of the culture."*

In a succeeding paper (2010) we find the following conclusions that were presented[104]:

"The growth of the bacterial-like organisms that appear to be at the foundation of the so-called Morgellons condition has been positively inhibited…The basic strategy that has been adopted is a transformation of the growth environment to a more alkaline condition along with adding specific antioxidants that are directed toward the scavenging of the hydroxyl radical.

We turn now to an additional important means to alkalize the body, that of bile production by the liver.

Attention was called in 2011 to the role of bile as one of several mitigation strategies listed in the paper, Morgellons : A Thesis[105](2011):

…"Improving the flow of bile in the system to further alkalize the body and aid the digestive system. The liver, the gall bladder and the bile duct play an extremely important role in alkalizing the digestive tract. For those that demonstrate a persistent acidic condition within the body it may be beneficial to learn of the importance of bile production and its alkalizing function.
An acidic condition can easily be created with a blockage of the bile duct, as the bile is the alkalizing agent within the intestine. Gall bladder removal and gall stones appear to be a frequent occurrence; this would suggest that overloads of toxicity to the liver could well be at the root of this problem. Non-invasive methods of breaking down gall stones (conglomeration of bile) are available to consider, such as Chanca Piedra (breakstone). If the bile flow is restricted, an acidic condition within the body is expected to exist.
Knowledge of the physiology of the liver, gall bladder, bile duct and its relationship to digestion may be beneficial in mitigating the consequences of acidity within the body and digestive system."

Furthermore, the reader was introduced at that same time to an educational video on the relationships between the liver, bile production, acidity, alkalinity and immunity was made available at the following site:

Video Series: Liver, Gall Bladder and Bile Duct Physiology
(www.balancedhealthtoday.com/glytamins.html)

(No endorsements of products or services to be implied or stated herein)

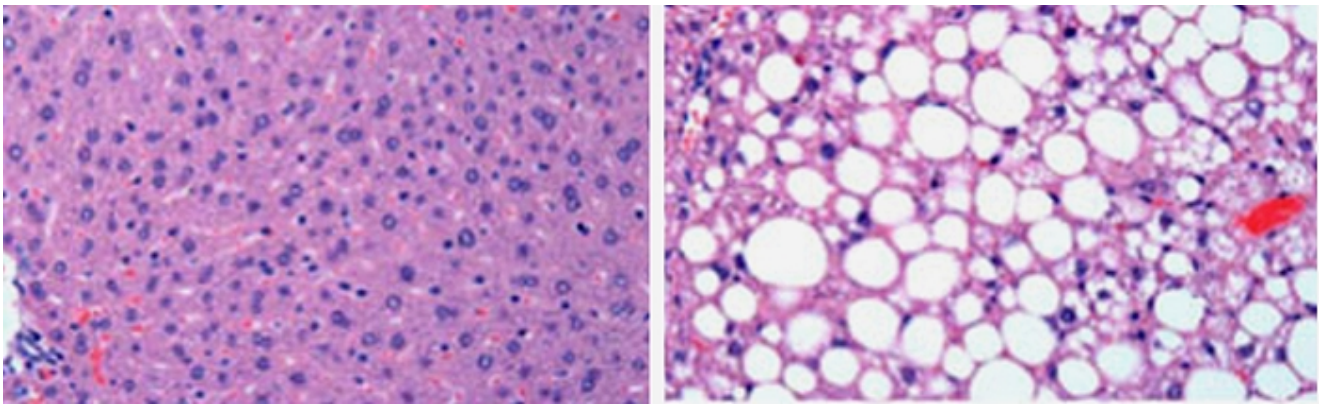
There are recent observations of liver tissue that are important to be briefly introduced at this time; the subject will be discussed in more detail at a later date. If the body is unable to process the toxic load placed upon the digestive system, there will be an accumulation of these toxins within the body. The function of the liver is manifold, and detoxification and

waste removal is foremost on that list. The liver is also responsible for protein synthesis, the breakdown of fats with the production of bile, glycogen storage, decomposition of red blood cells, iron regulation, and many others. We only have one liver and we cannot afford to have a serious problem with it.

One of the major problems with the liver (of increasing incidence) is the accumulation of fatty tissue within the liver. It is estimated that more than 1/3 of the population now suffers from fatty liver disease that is unrelated to excess alcohol use. The net impact from the accumulation of these toxins is an enlarged and fatty liver. If the liver is unable to process the toxic overload, fat cells with the toxins will accumulate and be stored within the liver. It is potentially a serious situation and one that is difficult to reverse quickly; weight gain is often associated with the condition. What follows is a photographic comparison of a healthy and a fatty liver:

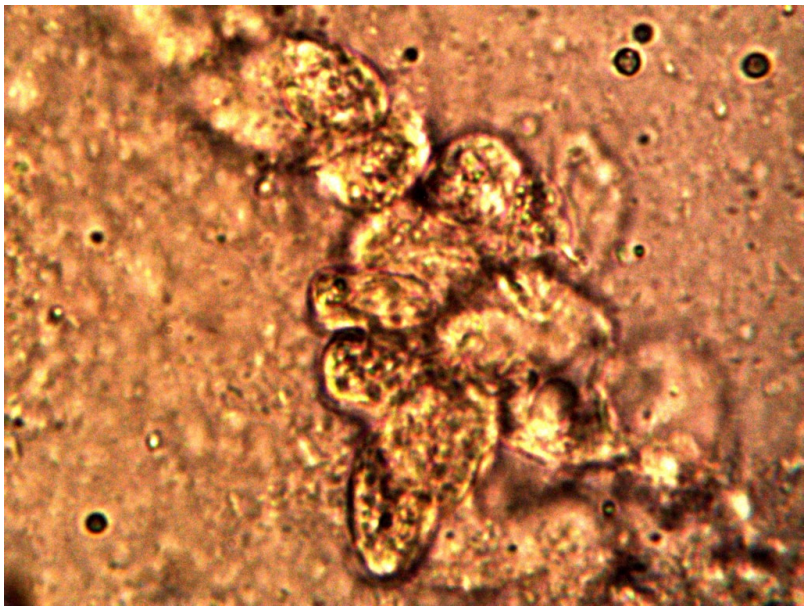
source : www.healthadvice4life.com

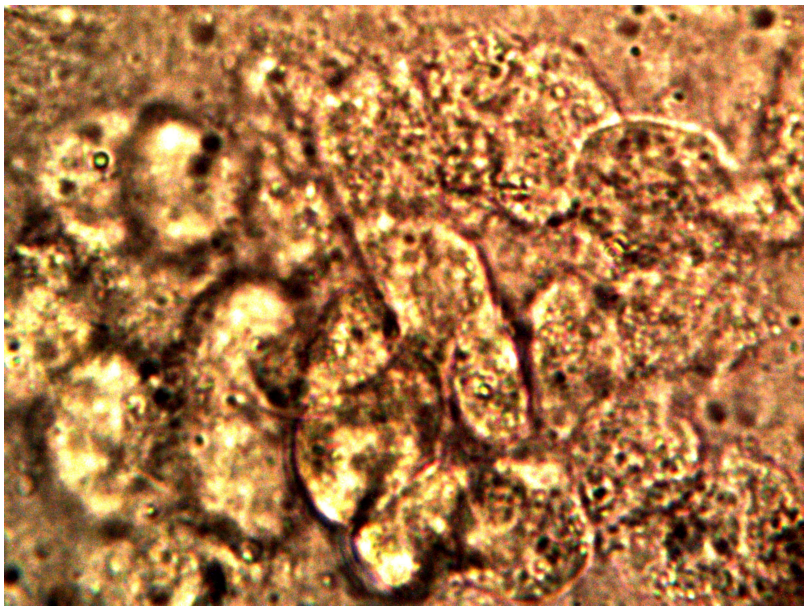
From visual impressions alone, it is clear that this condition is not a healthy one. It is reasonable to conclude that the functioning of the liver is seriously impaired with this condition. We can also find examples of what the fatty liver looks like under the microscope, also in comparison to healthy liver tissue:



Comparison of normal liver tissue(left) and fatty liver tissue (right)
source : advance.uconn.edu

Recent observations of calf liver under the microscope show this condition of fatty issue existing. There are two concerns present from this initial observation:





Appearance of significant fatty liver tissue within a calf liver recently analyzed.
Sub-micron bacterial-like structures (identical in size and shape to those studied extensively on this site)
are abundant within the fatty cells. Magnification approx. 8000x.

1. The animal is young, and therefore excess fatty tissue would not be anticipated at this stage of growth.

2. **The fat cells are enclosing large numbers of what appear to match (identical in size and geometry) the sub-micron, bacterial-like structures that are the subject of much scrutiny within the research of this site and this current paper.**

It is reasonable to surmise that the abundant presence of the encapsulating fat cells represents a toxic-overload response by the liver in the young animal. Other issues of equal and concern arise from this recent observation; this research is to be presented at a later date.

For numerous reasons, there is a legitimate case for concern about the impact of the Morgellons condition upon the functioning of the liver; this includes potential toxic overload, fatty tissue development and the prospect for an enlarged liver that results. If such proves to be the case, there is an obvious need for consideration of liver detoxification strategies to be incorporated within this report.

Although he have now forged through some of the controversies regarding acid-base imbalances, it is certain that some shall remain after this paper is complete. The issue of drinking water that has been “alkalized” must be given mention, however unappealing the circumstances may be to certain parties along with their particular knowledge base. There

are those that advocate that certain alterations of water, either chemically or with certain devices and technologies, will be sufficient to address the acid-base imbalances under study here. I do not find such arguments, thus far, generally sufficient to justify such conclusions at this point. It is evident from the work at hand that sufficient oxygen available at the cellular level, thorough aerobic metabolism, reduction of excess carbon dioxide, mineral balance, diet (especially as it relates to mineral intake) and the alkalizing processes of the body (e.g., bile production and flow) are at helm of acid-base balances within body and health. The argument for a modification of the "water molecule" (under question in its own right) to account for and compensate for the complex systems mentioned above appears that it may be lacking in the necessary substance of this problem. Whether one "agrees" or not with the following information from Dr. Lawrence Wilson on the topic of "alkaline" water, it behooves us to become familiar with the arguments put forth[106]:

"The pH balance of the body is very important, and most people's bodies are too acidic at the cellular level. It does not matter if the saliva, urine or other fluids test alkaline. In almost all cases, the body cells, which is the site of metabolism, are too acidic. This predisposes one to many metabolic imbalances and diseases including cancer. The rationale for drinking alkaline water is that it will correct this important physiological imbalance.
Problems with this rationale for alkaline water. The main problems with this theory are

1. ***The real cause of excess acidity at the cellular level is a deficiency of what are called the alkalinizing or alkaline reserve minerals.*** *These come from what one was born with, and from the diet. If one lives a stressful life, one also depletes these quickly. They include calcium, magnesium, zinc, selenium, and a few others.*

Unfortunately, drinking artificially alkalinized water does little or nothing to replace these vital minerals. In fact, it may deplete them for unusual reasons. It may make the body think it is alkaline, so the body does not need to hold on to its alkaline reserve minerals as much, and it eliminates some of them, making the person even more deficient.

2. Water from alkaline water machines replaces the vital minerals with a little cadmium, lead, arsenic and other toxic metals found in tap water and not filtered by any carbon filters that I am aware of. Some filtering systems claim to filter out toxic metals, but I have not observed this in practice. Those filters that I have seen that are said to remove a lot of toxic metals tend to damage the water even worse. Reverse osmosis is an example of this type, along with KDF and other types of "advanced" filtration media.

3. In addition, the alkaline water machines also replace the good minerals with a little platinum and titanium found in the plates that the water passes over to make it alkaline. These are both supremely toxic metals, especially platinum. In addition, I have observed slightly higher levels of nickel in those who use alkaline water machines for several years.

The nickel is probably leached from the stainless steel in the machine, or perhaps from a nickel-plated machine part. Nickel is a deadly toxic metal. The alkalinity of the water may cause a little to be leached out of the machine parts.

4. Carbon filtration also does not remove enough of the toxic chemicals in the water, so one is also getting a dose of chlorine, fluorides, aluminum, copper, residues of medical drugs in many areas, pesticides and more.

5. As a result, alkaline water machines do not really balance the body's pH, although they will change it a little, giving some people the impression they are getting well when, in fact, they are becoming more ill. ***The only way to truly balance the body is to replenish the alkaline reserve minerals.*** *To do this, one must eat a lot of cooked vegetables. The cooked vegetables, and perhaps some mineral supplements, when carefully chosen such as kelp, can and do supply the alkaline reserve minerals. Good quality, natural spring water also supplies some alkaline minerals, as does good quality sea salt. Using these on a daily basis, the body can be slowly remineralized. This is the way to do it, not drinking artificially alkalinized water."*

We can once again see the emphasis upon the alkaline reserve minerals as a major pathway toward the restoration of the acid-base imbalance, with an emphasis upon diet to accomplish this. The verdict on exotic or expensive technologies to alter the "state" of water is left to the reader to investigate further; I would only encourage that the study be rooted in chemistry, biochemistry and physics as opposed to promotional claims. At this point of study the causes of and factors affecting increased acidity, from numerous and varied sources, parallel the summary given by Dr. Lam to us at the onset (please review) remarkably well. These include incomplete metabolic breakdown of foods and nutrients, the lack of availability of sufficient alkaline reserves, the ingestion of acidic toxins or foodstuffs into the body, carbon dioxide imbalances and the failure of the bile system to adequately alkalize the intestinal tract. The road to recovery from the impact of such damage is to reverse the courses above, i.e., increase the efficiency of the oxidation of fuel, assure adequate oxygen in a form that the body can actually use, intake sufficient alkalizing minerals (such as calcium, magnesium, etc.) and improve the flow of bile and improve the digestive processes in general. It is obviously a tall order, but courses of action are readily available to all of us. Consultations with health practitioners about the sensibility or validity of the information being relayed here is a good start in the process. Supporting this process with your own studies and research on the matter can only be of further benefit.

For those that continue to profess that what you consume does not materially affect your body chemistry, or for that matter, the acid-alkaline imbalance in general, let us cite a more traditional example from the American Journal of Clinical Nutrition on the subject of diet, pH and oral health. It states clearly that what we eat is not a neutral affair[107]:

> *"..**Diet affects the integrity of the teeth**; quantity, **pH**, and composition of the saliva, and plaque **pH**. Sugars and other fermentable carbohydrates..provide substrate for the actions of oral **bacteria**, which in turn **lower plaque and salivary pH**. The resultant action is the beginning of tooth **demineralization**."*

Readers may once again be struck by the association between acidity and demineralization; the relationships between diet, body chemistry, acidity and alkalinity are all too apparent in the literature.

The preceding section serves as a welcome segue into the realm of what may be rather unsung heroes; those that have devoted themselves to health, nutrition and "functional medicine". Many of us look to a particular type of "doctor" to "heal" an ailment or disease, but those that study the role of **nutrition** in promoting health and those that study **health** in a more holistic sense are likely our true and best allies. There are many individuals that have studied extensively the relationships between oxidative stress and acidity, for example, with nutrition and the body systems in an integrative sense. It is wise for us to avail ourselves of their talent and knowledge, as opposed to only seeking a particular "cure" to a "particular ailment", including that of "Morgellons".

If we now open our discussion to include the counsel of those that study nutrition as a lifelong passion and its role in our health, the association between health and the acid-alkaline balance is prominent within the literature. For those that continue to advocate that saliva and urine pH have no real value in the assessment process, it may be prudent to become familiar with a portion of the following discussion. For those that seek out the credentialing process, Dr. Biamonte is no lightweight in the profession of clinical nutrition. One article worth studying in detail is entitled, "Urine and Saliva pH Testing"[108] from the Biamonte Center for Clinical Nutrition. A few excerpts are in order here, but it is advised to study the article in detail. The acid-base balance discussion, the role of minerals (once again) in the process, and various simple testing procedures for both saliva and urine are worthy of your consideration. Please conduct your own research with the critics as well and reach your own conclusions as to motives and intentions of various parties. You may also wish to examine the documented effects of demineralization and destruction of teeth and bone documented earlier in this paper, as well as to study those that have suffered from these effects. The mineral loss in these cases is clear and evident, and it is difficult to deny that the acid-base balance is an important part of the process.

In the meantime, let us proceed with some representative sections from the article:

"Simple tests of your saliva and urine that you can perform yourself can give you a good idea of the pH levels of your body…"
"Testing urine and saliva after sleeping at least five hours gives you an idea of how your body is operating. Urine pH tells of how your body is responding to the food your ate the day before. Saliva pH tells your how your body has accepted the past few weeks and months. If you have not been eating foods that contain alkalizing minerals, your body has adapted its function to keep pH of your blood and other vital fluids as correct as possible. It is often these long term adaptations that are necessary for survival- that eventually lead to symptoms of chronic degenerative diseases such as arthritis, osteoporosis, emphysema, or even cancer."

"Alkalizing minerals are stored in many organs and tissues of the body. The liver is the greatest storehouse of sodium; the bones are the greatest storehouse of calcium. Yet these storehouses can be emptied if the minerals that are used can't be replaced. The food you eat determines how well your reserves are replenished. Fresh fruits and vegetables contribute the usable alkalizing minerals you need to restock your alkaline reserve. When there are enough reserves to buffer the acid produced naturally by cellular activity saliva pH will register around 7.0. Readings of considerably lower or higher than 7.0 usually indicate that your buffering reserves have been depleted and your body is being forced to accommodate by other means."

"The urine represents what we are eliminating, the saliva represents what we are keeping. The urine does not accurately represent the state of the body, but does represent what it is eliminating – ideally acidic wastes. The first morning saliva pH is the indicator of the state of the body: tissue, lymph, interstitial fluids and blood."

This urine/saliva tests shows basically how many minerals are left in our bodies, i.e. what the MINERAL RESERVES of the body are and what we must do to remineralize it. This lays the foundation for any and all healing therapies.
** The test is simple:*

All of the following pH tests should be done on same day.

1. Saliva test upon waking. First thing in the morning right when you get out of bed, lick and wet the MIDDLE BOX OF THE PH TEST STRIP. Note the color change and write down that pH number. Do this before brushing your teeth, drinking, smoking, or even thinking of eating any food. This pH should be 6.8.

2. Then test your second urine of the morning. The urine stored in your bladder during the night, that is ready to be eliminated when you get up, should be acid so you don't want to test that. Drain your bladder in the morning, the last time you get up if you get up during the night and then see what that urine pH is. Again, record this number. This number should be the pH of your urine after you got rid of your acid load from the day before. The acids should be gone the second time you go to the bathroom so your urine pH should be around 6.8 also.

3. Eat breakfast, an apple will do, anything, and five minutes after breakfast check your saliva again. Write this number down also. This number should go up from what it was before you ate, the more the better.

4. Then check your urine pH between breakfast and lunch. . The pH should always be 7.0 to 8.5, a couple of hours after meals.

5. Then check your urine pH between lunch and dinner. The pH should always be 7.0 to 8.5, a couple of hours after meals.

These five tests show the following:

1. How well your digestive system dealt with what you ate the night before, i.e. the AM urine pH. These numbers may change from day to day depending on what you did eat the night before.

2. How well we treat ourselves in general, i.e. how "strong" the liver is. This is the AM saliva pH. This number shows the overall state of our health, the condition of the alkaline reserve of our bodies which reflects the diet we have eaten over the last months to years. This number stays rather constant and will only change after some work has been done in re-mineralizing the body. Pleomorphism and its changes can be viewed under the darkfield microscope, but the saliva pH shows what you will see.

Since the saliva pH is an indicator of intracellular pH, saliva pH readings should never be below the pH of the phosphate buffer system, 6.8. (see below). The most accurate reading of saliva pH is recorded immediately upon awakening–after sleeping at least five hours and before brushing the teeth. It is during sleep that the body removes waste and is in an anabolic state restoring and replenishing the body. If the patient has a saliva pH of 5.5 at this time and only 5.6 after eating, you know that this person has no alkaline reserve and that his body is devoid of the minerals necessary to process food properly–his body cannot adequately respond to the physiological crisis of handling food.

3. The pH of your saliva after you eat gives an indication of what the mineral reserves of your body are (the pH number should increase after you eat). The ideal saliva pH pattern is 6.8 on awakening, 7.0 before eating and 8.5 following breakfast.

4. The pH's of the urine between meals should be kept in the basic range, pH 7.0 to 8.5. After one eats, the stomach generates the necessary acid to digest the food. While doing this, it also performs the opposite action, i.e. it makes an equivalent amount of base or baking soda, sodium bicarbonate, that is picked up by the blood stream and delivered to the alkaline glands of the body, the saliva, the pancreas and the liver. The maximum amount of base in the blood and therefore in the urine occurs one to two hours after you eat. The body fluids and therefore the urine is most acid at 2:00 A.M. (pH 5.0 to 6.8) in the morning (the base tide) and most alkaline at 2:00 P.M. (pH 7.0 to 8.5) in the afternoon (base flood).

Along the course of the article, Dr. Biamonte also introduces us to the "lemon-test", a relatively simple test that can be used to give an indication of the available mineral reserves in the body. The history of the test and its use extends well beyond any single practitioner, and it is also described in some detail by Dr. Dicken Weatherby in his book on functional medicine, "In Office Lab Testing : Functional Terrain Analysis[109]". Various examples of test results are described within this same book. The origin of the test appears to reside with a Dr. Henry G. Bieler, MD.[110], the well known author of "Food is Your Best Medicine".

DR. BIELER'S SALIVARY PH ACID CHALLENGE

Background

Dr. Bieler's test is a dynamic measurement of the body's alkaline mineral reserves- one of the secondary buffering systems of the body. We are looking to see whether the body has the reserves necessary to respond to an acid challenge. During this test we challenge the body with acid in the form of lemon juice. The initial acidity of the lemon juice will cause the saliva to buffer this acidity over the course of a few minutes by becoming more alkaline. We expect the saliva to get more alkaline to show that the body can respond to an acid challenge by marshalling up the necessary alkaline mineral reserves. If there are enough alkaline minerals in the body, the body will use them as a buffer. If there is an alkaline mineral insufficiency, the body may start to use ammonia as a buffer, which is an indication of low mineral reserves.

The introduction to the pH Saliva test, from
"In Office Lab Testing : Functional Terrain Analysis" by Dr. Dicken Weatherby

I have witnessed the administration of the test locally, and I find the results to be of much interest and expressive of variation between individuals. The test is simple enough in principle and practice to monitor individually if desired, as in the following example with two separate individuals.

The Acid-Lemon Test conducted by two separate individuals. The individual on the right demonstrates a stronger decrease in pH over the time interval measured. The reasoning behind this test concludes that the individual on the right is likely to have reduced mineral reserves available, and consequently a higher acid level may be anticipated within the body tissues. The sharp rise in pH on the individual's test to the left may apparently also reflect ammonia imbalances and is also worthy of further study. Important nuances in the test do exist and they are worthy of further research; Dr. Weatherby's book may be helpful in this regard.

Additional simple tests for an acid-alkaline imbalance (i.e., breath holding test and respiratory rate test) are described in one of several of Dr. Dicken Weatherby's books[110b] on the subject of functional medicine.

Any readers with a further interest in these topics may wish to consult those that practice in the field of functional medicine, or as stated repeatedly, the health practitioners of choice.

A general introduction to the fundamental principles and philosophy of functional medicine is available courtesy of Dr. Mark Hyman:

Mark Hyman, M.D., Introduces Functional Medicine

Listen to a more extended discussion by Dr. Hyman on the state of health and Functional Medicine.

We can speak of such issues of oxygen, energy, oxidation, acidity and pH at length, but me must move on to make further progress. For those that continue to profess that there are no relationships of consequence between these factors, or little that can be done about them, let us make a more blunt parting observation as to what happens in the body *when we die*. On the topic of acidosis (i.e., excessive acidity within the tissues), under the subtopic of *associations*, we learn starkly that[111]:

> *"Lactic **acidosis** is an underlying process of **rigor mortis**. Tissue in the muscles of the deceased carry out anaerobic metabolism in **the absence of oxygen**, using muscle glycogen as the energy source, and significant amounts of lactic **acid** are **released into the muscle tissue**. With depletion of muscle glycogen, the loss of **ATP*** [i.e., energy production]*causes the muscles to grow stiff, as the actin-myosin bonds cannot be released. (Rigor is later resolved by enzymatic breakdown of the myofibers.) In meat-producing animals, **the** post-mortem **pH drop in muscle tissue** contributes to meat quality (by influencing water retention, cutting color and texture of meat) and also contributes to food safety by inhibiting several acid-intolerant spoilage organisms that otherwise might proliferate, even at refrigerator temperature."*

It would seem as though there are obvious relationships that exist between acidity, oxygen, and energy production when we are dead. There is every reason to think that such relationhips exist while we live as well.

One famous line from mutated history and the cinema is that, "Today is a good day to die…"

Our alternative line for today (not quite so famous) is that, "***It is a good time to talk about the thyroid***." And so on we go…

We can now recall the importance of the thyroxine, the primary hormone of the thyroid:

> **"Thyroxine stimulates the production of oxygen in the body. Thyroxine is directly related to carbohydrate metabolism, protein synthesis and breakdown. Thyroxine stimulates the utilization of energy. Thyroxine directly affects the basal metabolic rate. Thyroxine stimulates the cells of the nervous system. Thyroxine is used to maintain the state of the cardiovascular system. Thyroxine stimulates the breakdown of fats. Thyroxine stimulates normal growth and development. Thyroxine stimulates the muscles to break down proteins. The thyroid is, therefore, a master regulator of metabolism for the body and any interference in that functioning is inevitably and seriously detrimental to human health."**

We can see that the thyroid is the metabolic master of our system, and it is harder to get much closer to home than that. We also have good reason to suspect that thryroid processes are being interfered with in conjunction with the Morgellons condition. Our most fundamental indicator of this disturbance is that of body temperature. There is good reason as well as evidence to show that the body temperature of the general population is operating frequently at a temperature less than normal. The mantra of "98.6" that many of us grew up with may not exactly be quite so vocal these days, and the mystery of that silence is deserving of intensive study.

We also have reason to consider interference from the standpoint of aromatic chemistry; this has been discussed at length earlier in this paper. Essentially, the existence of aromatics along with amines is a perfect setup to initiate the halogenation of the aromatic structure. Halogenation of an aromatic structure by the halogens foreign to the body, e.g., fluorine, chlorine and bromine, is also the perfect setup to interfere with thyroxine, or the thyroid itself.

Third, we have a relationship of interference to consider between tyrosine (an amino acid), the thyroid (with thryoxine production) and dopamine (a neurotransmitter), as it has been discussed previously. We will also revisit this topic when the subject of neural disruption is discussed later. No matter which way go about it, we obviously have important issues at hand here, and metabolism and body temperature indicators are the heart of it. We will focus on this issue of body temperature, as it is direct and apparent, and it is easy to measure, and monitor for change. It is a macro indicator that affects the entire body system. **Our Biochemistry is essentially non-functional without the proper conditions of temperature and pH in place (**for *ALL* reactions**), and we must never lose sight of this fundamental fact** as we wade through this maze of complexity and interaction.

What does low body temperature mean, at the most fundamental level? It means the body is not working up to speed; the engine is not running at the proper temperature. If the engine does not run at the proper temperature we have incomplete combustion and less energy is produced. In essence, the body is not working as it should, and it is definitely not firing on all cylinders. In more conventional terms, an underactive thyroid is called *hypo*thyroidism and the overactive thyroid is called *hyper*thyroidism. The signs of research in place point quite strongly to the former in association with the Morgellons condition.

Let's look at the connection between temperature, metabolism and the thryroid from several sources and in more detail: First, a direct statement of the relationship from a detailed source on medical testing[112]:

> *"There is a direct link between low body temperature and low thyroid function. In fact, one of the symptoms of hypothyroidism is the reduction in body temperature."*

In equal plainspeak, Dr. Rind introduces us to the importance of the relationship, as well as the role the adrenals have with respect to temperature variations[113]:

> *"If you're not feeling quite up to par, take your temperature. Not to determine if you've got a fever – rather, temperatures reflect an individual's metabolic energy state. The average daytime temperature of a healthy individual is 98.6 thus making 98.6 the optimal (as opposed to normal*) temperature. Lower than optimal temperatures reflect a lower than optimal metabolic state which is usually controlled by the thyroid mechanism.* ***Wide variability of temperature reflects an unstable or fatigued adrenal system*** [please note this addition to our considerations -CEC]. *Thus, on the road to health, one wants to go from low and/or unstable temperatures to 98.6 and stable if possible."*

> Detailed information about a method to monitor your temperature and its variability is available on Dr. Rind's site as well. The awareness of the state of temperature in the body, its importance to efficient metabolism, and a system to track and monitor temperature on a regular basis may be a beneficial first step in becoming aware of the importance of thyroid functioning to good health.

Lastly, to cement the importance of the relationship of body temperature to thyroid functioning, Dr. Weatherby also describes a Body Basal Temperature Test[114] and reiterates the primary point made:

> ***"A reduced core body temperature is one of the hallmarks of thyroid hormone deficiency and hypothyroidism."***

The widespread reporting and observation of reduced body temperature amongst the general population , in addition those those more visibly suffering from the Morgellons's condition, should make it apparent that hypothyroidism is a central topic of research here. This paper can only hope to introduce the importance of this issue in this and future studies..

If we postulate that a state of hypothyroidism exists, i.e, a state of lowered metabolic rate associated with the lower body temperature cited above, what then are some of strategies offered by the health community to alter this situation? It would certainly seem, then, that the cause of such a problem would need to be identified first. We have certainly called attention in this paper to the possible role that the halogens might assume in such a case, and the interference that the toxic halogens can create. An obvious first approach might be to reduce or eliminate the presence of toxic halogens within the body, and to avoid contact or ingestion of them. Let us seek out how the health communities might react this to this potential problem.

If we begin by asking the question of how one would detoxify from an excess of halogens, especially that of fluoride and bromide, we are immediately led to an abundance of discussion related to iodine therapy. The reasons for this have already been discussed, and these relate to the relative reactivity of the halogens and their competition of iodine within the thyroid. We can repeat that relationship with the words, to start, from Dr. Mark Sircus :[115]

> "It is well known that the toxic halides, fluoride and bromide, having structure similar to iodine, can competitively inhibit iodine absorption and binding in the body."

We are also immediately led, therefore, to a strategy of great importance and interest, i.e., iodine therapy, or the intake of additional iodine into the body. Expressed as follows by Dr. Sircus in conjunction with Dr. Gyula:

> "Iodine intake immediately increases the excretion of bromide, fluoride, and some heavy metals including mercury and lead. Bromide and fluoride are not removed by any other chelator or detoxifying technique. Dr. Kenezy Gyula Korhaz states that iodine chelates heavy metals such as mercury, lead, cadmium and aluminum and halogens such as fluoride and bromide, thus decreasing their iodine inhibiting effects especially of the halogens."I

It is more than explicit at this point that no medical advice is ever given or implied within these papers, however, information and education **IS** to be freely available to all. It is clear from the literature and research that iodine supplementation in connection with thyroid performance is extensively discussed and employed. The chemical and molecular rationale for that strategy has already been made clear within this paper; the details of consideration and application will be left to the reader. The responsibility for education and professional consultation in any such matters is also equally obvious.

Examples of the need for education and consultation on the matter is apparent from the following two complicating factors:

1. Another strategy, commonly employed, is that of prescribing an increase in the thyroid hormone (T4) itself to remedy hypothyroid (decreased thyroid function) imbalances. In the book, Thyroid Balance, by Dr. Glenn Rothfeld, we read that:[116]

> "Doctors typically prescribe a thyroid supplement -a drug that boosts the thyroid hormones in your system -to treat most thyroid imbalance. This is a tried-and-true therapy that has been the standard for more than a century. The earliest documented use of this therapy dates to 1891, when doctors started using ground thyroid gland tissue from sheep to treat severe hypothyroidism"

2. Allergic reactions to iodine supplementation are known to exist. The extent and reaction of an allergic reaction is certainly outside the scope of this article, but attention will at least be drawn to the matter.

3. Another question that can be asked, similar to those issues that were raised earlier with respect to deficiencies in iron, is whether or not we take care of a deficiency by simply adding more of the same thing back into the system? *If we do not understand what is causing the deficiency of a particular substance to begin with, supplementing it with the same substance may be a completely futile exercise*. Hence the interest of "Thyroid Inhibition" *increases*, and consequently the interplay with iodine remains a focal point of the strategies discussed here.

4. The relationship between adrenal performance (and cortisol levels) and thyroid function is also important to be aware of. There is some information from the research of this Institute that the undue stresses on the adrenal glands may well be another point of serious research as it relates to the Morgellons condition. There is an additional caution provided to us for the simplistic response of simply increasing the thyroid hormones with supplements:[117]

> "Thus, it can be important for you and your doctor to rule out insufficient adrenal function before raising too high on natural dessicated thyroid or T3…"

Self-tests for adrenal function are subsequently described in this article, but the point is again made that thyroid hormone supplementation may be a diversionary exercise.

Education and research are the goals here, not therapy. Education and research are obviously on the path toward therapy, and these are our pursuits.

While we are on the subject of self-tests, let us include another test, this time related to iodine deficiency. There is an additional test entitled the Iodine Patch Test within Dr. Weatheryby's book mentioned earlier[118]. It is a simple test that monitors the fading of an

iodine patch (2%) painted onto the skin over a 24 hour period. As Dr. Weatherby describes,

> "The Iodine Patch Test is an excellent test for assessing for iodine deficiency… Unfortunately, iodine deficiency is widespread because of the prevalence of chemicals such as chlorine, bromine and fluoride [note halogen emphasis – CEC] in our environment and water supply. *These chemicals will quickly deplete iodine from the body and interfere with iodine metabolism leading to a number of problems including hypothyroidism, lowered vitality, cognitive dysfunction, lowered immunity, and obesity*. The iodine patch test is an easy method of assessing your iodine levels."

Once again, we may ask, does this sound familiar, relevant and germane to the findings of this report?

Readers are referred to Dr. Weatherby and other sources for more particular details on the interpretation of the test results. There are those who think that the iodine patch test is not reliable and therefore not useful; as such it exists in controversy amongst some practitioners.[119] It would appear that the lower body temperature test is less so and it is simple in principle to comprehend. Recall from earlier discussions that ***all biochemical reactions take place at a specific temperature and pH***; alteration of either of these parameters will inevitably lead to impairment of some sort or fashion.

We start this by mentioning forms that are not advisable and that have varying levels of toxicity – conventional antiseptic iodines. The first clue that such forms are not of benefit is the warning label, which will clearly state that this form is not to be used for internal purposes. There are two forms described that are in common use:[120, 121]

1. Tincture of iodine – a mixture of elemental iodine and either potassium or sodium iodide, dissolved in ethanol and water Denaturing of the alcohol is also know to be used in commercial tinctures. A 2% free iodine solution contains about 1 mg of free iodine per drop. Ethanol is poisonous in sufficient amounts and denatured ethanol is deliberately poisoned to prevent consumption. Tincture solutions can vary between 2% – 7% in strength.

2. Povidone iodine is a mixture of PVP (polyvinylpyrrolidone) and elemental iodine. It is soluble in both water and alcohols, and is more stable chemically than tincture of iodine. The deposition of PVP in human tissues reported in toxicology tests warrants abstention from use internally.[122]

We now transition to forms that are more suitable internally to the body (notwithstanding the prior caveats of potential allergic reactions, etc.). A statement of additional risk factors associated with the ingestion of iodine are included from the following medical bulletin from the National Institutes of Health; all readers are advised to be aware of all information that is contained within this report.[123]

Medline Plus : Iodine :
(http://www.nlm.nih.gov/medlineplus/druginfo/natural/35.html)

Now that we have given due notice to the federal standards for recommended levels of iodine in the body, let us open up the discussion to various professionals that have devoted significant study to iodine as it relates to health. It will be clear that the federal recommended levels are dramatically at odds with many serious research studies on the subject. For an extended discussion and debate between those at the forefront of iodine therapies (Abraham, Brownstein) and those advocating more restrictive conventional approaches (Gaby), please see the following paper:[124]

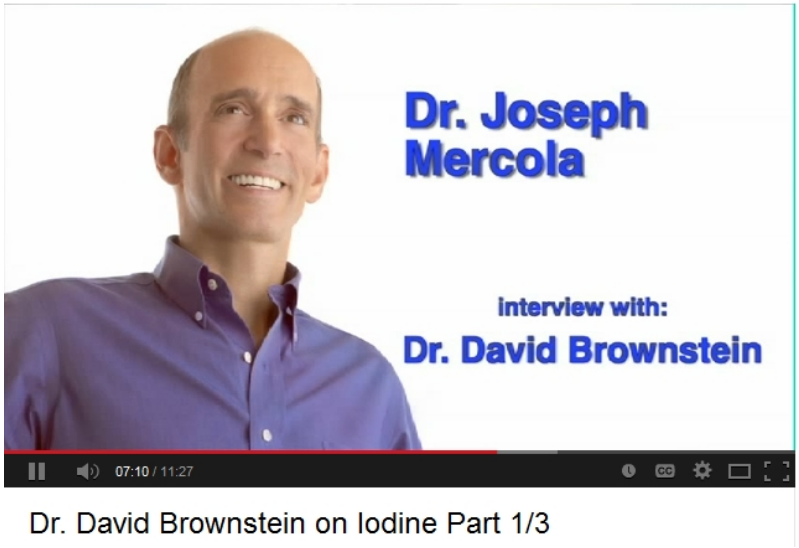
The Great Iodine Debate

The following media presentations are recommended as an introduction to the important role that sufficient iodine and iodide levels plays with in our health. **More extended discussions of the necessary levels of iodine in the body, the manufacture and storage of iodine, differences between iodine and iodine forms, body capacity and removal of excess iodine, skin issues related to iodine deficiency, the damage and competition for iodine by the halogens, the dangers of the halogens with respect to modern diets, and improved brain functioning with appropriate iodine levels are all important topics that are covered in these presentations. The reader is advised to become familiar with the material that follows**.

(Here is a test for you: find the place in one of the videos where “RDA” is stated to stand for a “really dumb idea (sic?)”.)

Jorge Flechas, MD, on the topic of Iodine Sufficiency

Dr. Brownstein, MD, Iodine, the most misunderstood nutrient



Drs. Mercola & Brownstein, MD, on the topic of Iodine Deficiency
(note the attention given to the bromine issue)

Dr. Tenpenny, on the topic of Iodine Deficiency
(Advanced Discussion)

We can see from the presentations by numerous doctors and extensive research that there is a strong case for the existence of increased levels of toxic halogens in the body (i.e., fluorine, chlorine and bromine) and for the competition that they exert upon iodine and the thyroid. This case in in alignment with the spectral and biochemical analyses that are a core result of this paper.The symptoms of impaired thyroid function such as reduced body metabolism and energy production, lowered body temperature, skin complications, brain dysfunction, cancers and many other serious health issues are intimately related to iodine deficiency. Iodine therapies are also offered as a significant prospect for improvement by these same doctors. The case for the existence of the aromatic halogens in association with the Morgellons condition has also been made by this researcher through the use of infrared spectral analysis, with a particular interest in bromine substitutions. It is also important to emphasize the major differences in the amounts of iodine that are necessary and utilized in the body compared to those identified in the federal standards; this difference ignores the prospect of increased competing halogen sources that may now have been introduced into the body. This difference, even based upon conventional medical research of recent decades as outlined in the presentations above, is on the order of 100 times. *Impairment of thyroid functioning and iodine supplementation therapies exist, therefore, as compelling and major topics of further research in the investigation of the Morgellons condition.*

We now begin to close this chapter of research in the history of Carnicom Institute, and we depart (temporarily, of course) with a brief revisit to, and a discussion of, the neural disruption issue. It is difficult to 'rank' the relative importance of the numerous issues that have evolved within this current research and their combination is devastating and ostracizing to far too many. This level of harm and suffering is much greater than that which is currently acknowledged, and many individuals deserve recognition for the battles they are fighting.

These battles are often fought in solitude and they can be literally a fight for life itself. We must offer our compassion, our care and our help in haste, as the frog pot continues to warm for most of us.

It is clear that cognitive functioning, concentration ability and mental acuity in general are companions of study here, and that they are closer to home than many of us would like to admit in our pursuit of improved health.

The technical and evidentiary argument for this situation has already been made in this report, and our question here is what can be offered as a prospect for improvement? Certainly the first fact to recognize is that the broad health impacts that have been discussed here are usually related to one another, and it is simplistic to separate them as islands of trouble. How can we possibly suspect a simple 'cure' to any demise of neural and cognitive functioning; in the majority of cases modern medicine is still in its infancy here. Nonetheless, there are ALWAYS paths to pursue to improve the lot of us, and there are no exceptions here.

We may start with the glaring theme of oxidative stress, which is pervasive and illustrative of the connections between the topics of this report and those that are at the foundation of pathology. This foundation (e.g., Robbins[125]) has already been discussed some time ago in a context that is much broader than the Morgellons issue by itself. Is it any surprise when we learn, therefore, that:[126]

> "Oxidative stress plays a pivotal role in the pathogenesis of neurological disorders."

and, in the discussion of a professional textbook on the subject, that[127]:

> "The role of free radicals and oxidative stress in neurological disorders has only recently been recognized… Oxidative Stress and Free Radical Damage in Neurology sets the record straight, focusing on clinical and research issues regarding the interplay of free radicals and the human nervous system. Crucially, the chapters cover numerous antioxidants and their possible therapeutic role in neurological disorders. Key illnesses such as epilepsy, multiple sclerosis and Parkinson's are analyzed, and chapters also examine more general issues such as the link between free radicals and inflammation of the central nervous system.."

And again, to eliminate any doubts on relevance[128]:

> "It has been demonstrated that oxidative stress has a ubiquitous role in neurodegenerative diseases."

We see the consequences of oxidative stress over and over, and at this point we are not entitled to remain ignorant of what we can and must do to improve the situation. The details and important role that anti-oxidants play in combating oxidative stress have been repeatedly emphasized in this report. We must take advantage of that same information here as it relates to neurological functioning.

We have also introduced leading research on the importance of glutathione as it relates to neurological diseases and Parkinson's disease. It should be recognized that glutathione is one of the most powerful antioxidants known, and from the above, it should come as no surprise to us that its effectiveness against oxidative stress is important to neural functioning. The importance of understanding the precursors of glutathione (e.g., N-acetyl cysteine (NAC) and alpha lipoic acid) vs. dietary supplemention or ingestion has also emphasized in this paper.

Through the introduction of iodine therapies that are practiced to improve the functioning of the thyroid, we have also learned that adequate levels of iodine are also strongly related to mental functioning, acuity and intelligence. This topic is especially prominent in the presentation by Jorge Flechas, MD, above. We have also learned that the difference between the federal guidelines of minimum daily iodine levels and the levels deemed beneficial by certain medical practitioners is dramatic, to say the least. This dosage issue is entirely independent from any need to compensate for the potential ***reduction or removal of iodine stores within the body by competing aromatic halogen compounds*** (as they have been identified and postulated within this report).

We have also called strong attention to the intriguing and serious implications of oxydopamine and its related compounds within this growth form. There is a strong case in the data of this report for this type of existence, and the damage that these compounds have upon neurological function is unambiguous. Recall that such compounds are used in the laboratory to deliberately ***induce*** Parkinson's Disease. Such compounds reduce dopamine and brain amine levels and this, as a minimum, is known to affect memory loss and cognitive functioning.

With respect to the potential mitigation from this effect, oxidative stress is responsible for dopamine loss[129], so this is now a familiar refrain to us. The role of antioxidants has been discussed at length in this report, and this presents the roles and use of vitamins (e.g, A, B, C, D, E), enzymes, and the precursors to glutathione for example.

Readers and health practitioners will also want to investigate the role of tyrosine and L-tyrosine as they relate to dopamine levels in the brain. Tyrosine is an amino acid, and it is the building block for dopamine as it has been discussed. The prospect of structural interference in the synthesis of dopamine has also been raised within this report, especially with prospect of halogen substitutions on the aromatic ring of tyrosine.

The role of diet and nutrition is also important to dopamine levels.[130] I am hoping that it is now understood, from the journey that has been shared, that this researcher advocates nutrition as one of the primary pathways towards better health, and that those who are knowledgeable in such ways deserve our greater recognition and attention. They have been driven to the heart of the matter, and that is that all life will eventually be a product of the nourishment that it consumes. The business of "supplementation" is essentially a *band-aid* to attempt to compensate for a deficiency that never should have existed if we were wiser and more complete in our ways, especially from youth onwards.

While we are on the subject of nutrition, a specific nutritional drink recipe is available on this site. This recipe is a culmination of health research by Carol Carnicom over a period of several years and it now also combines many of the important research findings from the study of the Morgellons condition. It is a nutritional approach to some of the needs that have been established, therefore, from a variety of perspectives. Protein sources, joint issues, iron utilization and the need for iodine are each examples of the ties that have evolved over the years between research and nutrition. The reader may find this information to be of value in some unexpected ways, and I encourage you to become familiar with both its pleasures and its constitution. The link to "Carol's Smoothie Recipe" follows below:

"Carol's Smoothie Recipe"
(.pdf download)

Continuing on the subjects of neural, mental and cognitive functioning, some of the food sources that are known to benefit tyrosine and dopamine levels include, therefore[131],

"Foods highest in L-tyrosine include:

Fava beans

Duck

Chicken

Ricotta cheese

Oatmeal,

Mustard greens

Edamame

Dark chocolate [Now, there's an excuse…-CEC]

Seaweed

Wheat germ "

The role of diet, nutrition and enzmyes in improved neural functioning is also discussed at length in the following report from the U.S. Department of Agriculture, entitled "Nutrition and Brain Function". We learn here again the important role that antioxidants play with the summary statement that:[132]

> "Perhaps there is no better place in which to gauge the power of antioxidants than between the minute connections of the nerve cells."

Two additional points of interest are also mentioned in this report. The first is the recognized benefits of enzymes (specifically, 'kinase" enzymes) to brain functioning. Notice also that even though the size of the brain is quite small relative to the body, it ends up using significant amounts of oxygen during mental activity. The availability of sufficient oxygen and body's ability to use this oxygen effectively are obviously of importance here. We see once again that it is a hopeless exercise to seek out singular causes, effects and 'cures' to the complex health problems before us, and the joint appearance of antioxidants and enzymes in the crusade against oxidative stress has again made its mark here.

Another word of importance within this paper concerns the regeneration of brain neuron cells, termed "neurogenesis". This work shows that adages die slowly, and that it is only recently accepted in the mainstream scientific community that the brain is not a fixed organ which can only deteriorate with age. The research shows that brain neurons can be regenerated, albeit at a slower rate, at more advanced ages with proper nutrition. This means "new" brain functioning and development can continue in the aging process and that "disease" is not a fixed sentence. One must be careful of old adages, lest we become fixed ourselves in our ways, thinking methods and perceptions.

Additional well known natural approaches to improve mental clarity and function include those of Gingko Biloba and CoQ10. Whether or not these particular supplements will be of known benefit with the Morgellons situation remains to be seen; they have, however, established reputations with respect to improving mental clarity and memory loss. The following paper[133] discusses a series of natural remedies to enhance memory and mental function, and it is anticipated that they may be of some benefit.

Enhancing Memory and Mental Functioning – NYU Langone Medical Center

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It is a primary argument of this researcher that the solution to a problem is not necessarily found by introducing additional complexity into the situation. The case for "supplementation" of diet to compensate for health problems is a primary example of this dilemma. If one has

known sources for health problems, it is usually wiser to eliminate the source of the problem rather than try to compensate for it with an infinite combination of variables, such as pills, supplements, or drugs, for that matter. The proper approach for the “Morgellons” condition, as with any health impairment or “disease” is to strike to the source of the matter. If the cause or source of such a condition can be identified, it is to be removed or stopped in its tracks, if at all possible. It is not be be accepted as intrinsic to the environment and then compensated for with a myriad of protocols, drugs, treatments, and supplements in a state of perpetual uncertainty and ignorance. A body of information is available to those who wish to seek it out, and this information (along with your participation) can be a pathway towards striking at that source of health and disease. The general population, the health communities, the professional communites, and the governmental structures are obligated, as has been stated repeatedly, to combine, use and express their knowledge and talents to improve the state of the environment and the health of the people. We, our children, and our future children deserve no less than this, and the entire world deserves the more of it.

This episode of research now comes to a close; this paper has taken more than a year to complete and additional needs remain before us. Additional work will be done in the future to summarize and consolidate the essentials of this research. Work of this nature is a journey in itself, and I do not know where it will lead and end when I start. The process of identification, correlation and analysis has now taken place, and it is hopeful that it provides a beneficial foundation from which we may accomplish greater things in the future. I thank you for your patience and endurance to reach these closing comments and I hope that the work has been of value to the general readership. The future remains to be influenced by the decisions and actions that we now take together.

Sincerely,

Clifford E Carnicom
(born Clifford Bruce Stewart, Jan 19 1953).

Additional Note:

Appreciation is extended to Lucretia Smith and Dr. Jimmie McClure for their sustained interest, research and communication to CI over a period of several years about the importance and relevance of thyroid issues to the work presented here. These individuals, along with others, deserve credit for their prescient assessments of the role that thyroid dysfunction is likely to play in the “Morgellons” condition. Many thanks to both of you.

Sincerely,

Clifford E Carnicom

END OF PART III

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