Outsmart biotoxins and reduce inflammation

Guest: Louise Carder

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Kirsty Cullen - [00:00:16]

Welcome to the Fatigue Super Conference, I'm Kirsty Cullen, CEO at the Optimum Health Clinic. Today, I'm delighted to welcome Louise Carder.

Louise holds a BSC in nutritional medicine and is a certified functional medicine practitioner and also a member of both the Naturopathic Nutrition Association and the National Association of Phlebotomists. In 2018 Louise became the first European practitioner certified in the Shoemaker protocol and is also a Bredesen Practitioner. She's most recently founder and managing director of U.K. based Colab Services, which deliver specialist laboratory testing to European practitioners.

Louise, welcome.

Louise Carder

Thank you very much Kirsty for having me. It is great to be here.

Kirsty Cullen

So, we shall kick off with the fact that functional medicine talks a lot about the gut microbiome, quite rightly, but lesser discussed perhaps is the nasal microbiome. And I wonder if you could introduce us to the whole topic of the nasal microbiome.

Louise Carder

Yes, I can. I would love to do that. But first, I just want to track back to Dr. Shoemaker and Dr. Bredesen actually, because I have dedicated my clinical practice and research, ahead of an upcoming postgraduate study course that I'm really anxious to get started with, to the immune inflammatory responses and the nutritional factors associated with them. And that drew me to the work of Dr. Shoemaker and Dr. Bredesen who both recognized how much we're affected by the world around us, related to both our immune health, and then how we process nutrients, for energy and function.

So that's what really drew me to both of their work. And as a nutritionist, if you told me 10 years ago that I would have learned and just dived into the nasosinus area as much as I had, I would have laughed. Being a nutrition professional typically starts, apart from the sense of smell when it comes to food, with the mouth downwards. But actually our noses, our sinuses are an intimate part of our oral and throat environment and we need to start to think about that area as a whole.

Did you know, for example, that our oral microbiome can migrate to our sinus area as we age? And that's probably got a lot to do with how we age as well. So, as you just said, we've all heard, well most of us have probably heard of the gut microbiome, but we have these biomes all over us and they all have their own peculiarities, including not liking it when things get out of balance. So it's great that we're starting with the nasal microbiome because it is one of those key biomes in the body, but perhaps one that we don't know quite so much about.

And so, the key functions of the nasal area, which we could just recap them if that's okay, they're filtering, warming and recovering air as it comes in, then moistening it. It's where our smell sensors are located. We've got immune function there that can detect pathogens in the air and respond to those. And of course, we've got the snot, the mucus production and clear roots that we all know so well.

And the mucus is super important because it acts as a physical barrier to actually track pathogens and particulates. And the mucus sits on top of some tiny hairs themselves that lie beneath them to actually help clear the mucus down to the back of the sinuses and into the throat. So we've got a really good clearance mechanism.

So we have clear functions and processes in our sinuses. It's a really busy area. And the microbiome or the bacterial colonies in the sinus area are there from the birthing process. But as I've indicated already, they change with time as we grow and age and with whatever environment we're exposed to. So we think of bacteria more when we think about a microbiome. But we also have to consider that viruses and fungi have their own respective biomes too.

Kirsty Cullen - [00:04:14]

So when we're considering this topic, Louise, I'm right in thinking, and I know I've heard you say before, to consider a top down impact of the nasal microbiomes, so we're not just looking at this in the context of those cases that are typified by nasal symptoms, maybe cognitive symptoms, brain fog. We should also be considering this in the context of a bigger multisystem illness, shouldn't we?

Louise Carder

Exactly. And the work of Dr. Shoemaker and Bredesen, to just reference them again, speaks to that complexity. So when we're thinking particularly about inhaled pathogens or aerosols that might affect our immune system, this can set off an inflammatory cascade in certain parts of the brain. We know, to get technical for a moment, that the hypothalamus and the pituitary gland are two areas that can get really affected by that inflammatory process. And for those of us that are practitioners, we will also know that those areas really are the seat of our hormones.

So when we say a top down approach we're thinking about something that's coming into our body, typically in this upper area, and then the immune system is having a response to that, the inflammatory process is then affecting the seat of these hormones and then we'll get hormone cascade changes through our body. So that could relate then to the thyroid, the adrenals and many other of the protective hormones that really do look after us on a daily basis from our metabolism to actually also supporting our immune system.

Kirsty Cullen

And it's amazing because you wouldn't ordinarily necessarily make the link between those things would you?

Louise Carder

No and it blew my mind when I first realized it as well. It was a very exciting light bulb moment for me.

Kirsty Cullen

So let's discuss, what are some of the factors that can disrupt the nasal microbiome and that finely tuned balance?

Louise Carder

OK, so we know that when we're first born we have a particular microbiome that is, first of all, informed by the way that we are born. So they can be slightly different in people who've been birthed by a vaginal delivery or a C-section, and then, of course, the environment that we live in. But age is

really one of the main things that will affect the microbiome. As I've already said, as we age we tend to have less of a separate microbiome in the sinuses and it becomes more similar to the oral microbiome.

We know as well that if we don't have that clearance capacity working very well for mucus, that can also affect the nasosinus biome. We know that immune capacity and response will also impact on that. And then, of course, my favorite topic, nutrition. So right from breast feeding, that's going to impact it, to the sugars that we have, to the protective lining nutrients that we get from so many of our foods, that will also have a good benefit on our guts as well. That will also have an impact.

Unfortunately, we know that if we smoke, we're also more likely to have certain of the less helpful, less beneficial bacteria present in our sinuses as well. And that's the message that we don't often hear about with regards to smoking, there's usually some bigger highlights that are talked about, rather than some of these smaller but perhaps equally significant factors.

Chronic respiratory conditions. Obviously, to have them in the first place, you're going to have some kind of sinus biome changes. But these can be escalated the more serious and the more entrenched that they get. So things like COPD, asthma, infections are all part and parcel of that imbalance in the biome.

And then environment and exposure, so something that we're particularly focusing on today relating to the environment that we live in, the humidity, perhaps the temperature will all inform what kind of bacteria or fungi actually start to colonize in our sinuses, and also what biotoxins we might be exposed to.

And then we also know that chronic sinus cases in particular, can go hand in hand with high sugar diet because that can affect how the immune system, responds to the bitter signals that are actually released by some of the bacteria that we know that like to live in our sinuses.

Kirsty Cullen - [00:08:19]

So before we get into the broader topic, can you just tell us a little bit more about a picture of chronic sinusitis or rhinitis and what we actually mean by the term mucostasis?

Louise Carder

Sure. So when we're thinking about mucostasis, we are thinking about a change that's happened in the nasosinus biome biome that can lead to the production of excess mucus. It is, as I've already said, a protective mechanism, which is fine if we can clear it. But if there's changes to the clearance then that can contribute obviously, to an ongoing issue. So stasis means things aren't really changing and mucus, well, that's obviously the mucus. So you put the two words together and it becomes a little bit more obvious what we're talking about.

So, we can then see obstruction of the nasal passages, an increased immune activity. And then if we also start to see the presence of less helpful bacteria or perhaps tissue damage in the area and clearance issues, then we're much more likely to see a worsening of symptoms that persist over the longer term. Remember, mucus is a protective element. So when we see it, we know that our immune system is actually working really hard. It's really working to try to protect us, but we have to be able to clear it. And when we can't, we really do need to start to understand why.

So, just following on from that point, one of the changes that we can see then, can be that more of the less helpful bacteria become a little bit too happy in this kind of environment. So they can produce toxins that start to change the environment to suit themselves a little bit more. They can neutralize our body's natural defenses, they can also actually affect the hormones that do act as protection of the lining of the sinuses.

And then they do this really icky thing. They actually create like a sticky kind of clingfilm over their colony to enhance their own survival. I know we're going to touch on this a little bit more, but I will just

say that that is actually called a biofilm and it's related to antibiotic resistance as well, because the antibiotics can't get through it. So that's just an example of how that mucostasis can really then affect the nasal biome and make things that much more complex for somebody.

Kirsty Cullen - [00:10:35]

And on the subject of inflammation, immune activation, that brings us very nicely to this term CIRS and I wonder if you could explain what CIRS stands for and exactly what that is within the clinical picture.

Louise Carder

So my understanding of CIRS has really changed over the time that I've known it. First of all, I just learnt it by rote. It's a little bit of a wordy name, chronic inflammatory response syndrome. It's a bit of a mouthful but essentially means that the immune system response is going on longer than you would expect it to. So acute immune responses are actually very beneficial we're designed to have these. But a longer term or chronic response can happen for a couple of reasons.

So firstly, different parts of the immune system don't communicate so well and a response can go on for longer than is useful. Or secondly, we may continue to be exposed to something that keeps triggering a longer term response. So that's essentially both acute and chronic. And the term was actually first devised by Dr. Shoemaker, he coined the phrase in the mid 1990s to explain the human illness that he was investigating relating to fish kills in the Pocomoke river and an illness that the fishermen who were regularly exposed to those fish were experiencing.

And he recognized that there was a chronic immune response that was driving changes in hormones and some important brain messaging systems. So he later realized, putting 2 and 2 together, that he was actually seeing the same kind of response in patients with post Lyme, those who'd been exposed to an algal bloom that's quite common on the east coast of the states and those who had a kind of food poisoning relating to consuming warm water fish. He's best known though, of course, for associating this kind of chronic immune response to mold. So the kind of thing that we might see related to a water damaged building.

Kirsty Cullen

And I know when you talk about mold, we kind of need to extend that into this idea of a toxic soup because, of course, mold is not just mold, it's associated with other biotoxins isn't it?

Exactly. Yes. So, again, although CIRS itself has quite a wordy title, it also has a really wordy definition. And that definition includes exposure to water damaged building environments, that has all sorts of things, I'll read some of them out for you so that everyone's heard them, because this might be the first time you've heard them, but they're words you might need to rattle round and think about it.

So these are toxigenic organisms that include, but are not limited to fungi, bacteria, actin and mycetes, which are a kind of bacteria actually, mycobacteria, as well as something that we call inflammagen, such as endotoxins, which is basically the outer casing of some of these microorganisms that we're talking about. Things like beta-glucans, hemolysins, proteinasers, mannans and possibly something that's got the most fabulous name to say, spirocyclic drimanes.

As well as just the volatile organic compounds that these things release as well. So obviously, it's much simpler to say toxic soup, so we'll just use that one rather than that whole list. So it's not just the classic idea as you said, the molds or toxins that they produce. It is that whole soup, really.

And it's also about how we respond, isn't it? Because CIRS can be profound in those with a genetic predisposition. So can we talk, just very briefly about that genetic predisposition and how that impacts on people when they're exposed to those triggers.

Louise Carder - [00:14:10]

Sure. Well, because we're looking at something that is a multi system, multi symptom condition, it is going to be a profound illness that probably was always more likely to have its base in some kind of genetic changes and differences between us, because if we put 100 people in a moldy environment, not everyone's going to become ill, so there has to be some kind of genetic communication that's happening.

And in this case, it basically means that the HLA, or human leukocyte antigen presentation of the genes, which again is quite wordy, but I'll explain what that means. So the HLA presentation means that their immune system doesn't quite sense the outside world as well as it might. It may do fine with bacteria because bacteria have a membrane. So the innate part of the immune system, our first face response, I like to think of it as the left hand, has a response to the outside world. It can identify this really well, present it to the right hand side, what we call the adaptive immune system, so that it can make an antibody to it, and hey presto, you've got a normal immune response.

We've heard a lot about this lately with viruses of course due to COVID, but for some people at risk of recurrent infections and CIRS in particular, this left hand and right hand, the two sides of the immune system don't communicate quite so well. And so the presentation is there, this part of the immune system is going, hey, hey, there's a problem, but there's no backup coming from the right hand side. So the antibodies are not created.

And so, when you have fragments, particularly small organisms that are found in a water damaged environment, you might have this ongoing response because the right hand side of the immune system can't really understand what the left hand is trying to say. And this can be largely associated with how well someone's HLA genetics, basically the meet point between the two were actually primed. So that's what we're talking about in essence, when we talk about someone's underlying genetics, it's how well was their immune system designed and had the capacity for that meet point.

Kirsty Cullen

So obviously in the U.K. we are renowned for our weather. What percentage of people do you think are exposed to water damaged buildings and what kind of potential scale of an issue is this in the U.K?

Louise Carder

So, I think it's on a huge scale, more than we can probably even estimate. Dr. Shoemaker's early data actually shows that if 100 people, as I said, were in a water damaged environment, maybe 25 would show symptoms, but only between about 7 or 12 percent of people are actually at risk of becoming ill. But even so, when you're thinking about 100 people to think that possibly 10 of them could be coming ill, that's quite a large amount.

And the studies would also back this up in the sense that, yes, in the U.K. we do have a problem and in Europe, many parts of Europe also do. So a study that I like to cite from the British Medical Journal, which is a very well respected medical journal, noted that damp was found in just over 30 percent of buildings and actual mold growth in 45 percent of buildings. So a third to almost a half of the buildings in the U.K. have this kind of issue. And then associated with the health of those buildings, adults living in a damp or moldy environment were more likely to present symptoms overall, including nausea, vomiting, blocked nose, breathlessness, backache, fainting, bad nerves than respondents in drier dwellings.

And the conclusion was that living in damp and moldy conditions has an adverse effect on symptomatic health. But the saddest thing of all about that study is that that is magnified in children. And we also have to consider that around 42 percent of asthma sufferers in the U.K. have an increase in symptoms when they're exposed to mold and fungi. And that piece of data actually just comes from the Asthma U.K. website. So you don't have to go very far or deep into the scientific journals to come up with a nugget of information about this. It's actually all around us. We just need to open our eyes to it a bit more.

Kirsty Cullen - [00:18:23]

And Louise you've mentioned, some of the symptoms there we might expect to see. Are there any other key symptoms that you would be looking out for that are maybe non-specific?

Louise Carder

Sure. So CIRS has been documented to be associated with a list of 37 fairly clear symptoms that are based on around 13 symptom clusters. So, again, this is something that was devised by Dr. Shoemaker. And if you have 8 clusters, out of the 13 clusters in that roster, with at least one symptom noted, then testing key immune and hormonal markers may be recommended. And in studies, this is 95 percent correlated with positive results.

So there is actually a really nice list, that I'm hoping that we can either show on the screen or share with people afterwards, that actually shows that list for people. But I will just read a few out if that's OK? So symptoms can include, and these are some of the top ones, fatigue, weakness, headaches and an ice pick or lightning style headache in particular, shortness of breath, excessive thirst or needing to pee a lot, static shocks, memory or concentration problems, difficulty finding words, hormone changes and many more.

But when we're thinking about the nasal side of things and how that associates to CIRS, we won't always have symptoms. So whilst we might have sinus or post-nasal drip symptoms, a crusty nasal passage with sores, these are all signals, yes that the delicate microbiome in the nose is being disturbed, but we might not always have those sinus symptoms.

Kirsty Cullen

And it's true to say isn't it, with CIRS we can frequently see this inability to sort of mount a good, a decent immune response, a robust immunoglobulin response. So just briefly, what are the type of markers that we might be looking at in testing to identify where we've got a compromised immune response in this situation?

Louise Carder

Sure. So I will touch on that but I think it's really important first to just note that there can be an acute delayed or chronic response to mold. And I think it's really important to note this because, an allergic response to mold is still actually really possible. So whilst we're talking about more of a longer term response, I think it is important to note here that an allergic response that could involve sneezing, post nasal drip, cough, respiratory distress, rash, hives, and in this case, regarding to testing, a high total IgE test or an IgE test that's high relating to mold and fungus, is something that really can sometimes need to be considered. Asthma also can be a guiding symptom for acute and chronic response.

So it is important to say at this point that I have had clients who didn't know they had a mold allergy, and it's really important to have that assessed by a doctor. So I will always encourage my own clients to seek medical advice if we suspect an allergy. This is hypersensitivity as a response, and it doesn't mean that somebody can't have a chronic picture, but it does need specific medical management and oversight.

And specifically, also, if there are respiratory symptoms and particularly lung related symptoms that have been going on for some time. Another medical diagnosis relating to mold is aspergillosis, which is a very serious lung condition. And again, I have also seen a few cases of this and have supported these clients to get the medical support that they need. I am not a doctor, so I work very collegiately with a few medically qualified practitioners who have oversight of my clients where required.

So we can also see a delayed response to mold sensitivity. So we might see IgG testing come back positive with that. But when we're actually talking about an immunoglobulin response, which isn't so common in CIRS, then we might actually see lower levels of the IgG, the IgM or the IgA markers. And we might see that threaded through other immunoglobulin results that we see.

So when we don't see an immunoglobulin response that's particularly robust, this goes back to what we were discussing earlier about HLA presentation, so some people don't produce that very well. And in CIRS we're more likely to see more of that first face response than an antibody response. And I see that over and over again with clients. And it means that someone is more likely to have a longer term response because the antibody, a memory B-cell response, helps us to not keep getting compromised by things over and over again.

So in CIRS we look at an innate immune marker called c4a. We can also look at another one called c3a, which is likely to go higher when we're actually exposed to a stealth infection because that will go high when we're exposed to a bacterial membrane. But c4a doesn't need an organism's membrane or outer shell like skin to trigger it, it can get triggered by fragments. So it's an ideal immune marker to consider when we're thinking about that toxic soup in a water damaged building.

And studies show that it can go super high within 24 hours of exposure of the sun. It can rise as much as 10 times higher than normal. And these are the people who can get really sick with exposure very, very quickly.

So it's the immune system markers like c4a, Tgf-beta 1, and then the hormone markers, we've got one called MSH, Melanocyte-Stimulating hormone and lots of the other hormone markers as well that we look at. So there's probably about 12 markers that we look at in total to understand the inflammatory process and then also the hormones.

Kirsty Cullen - [00:24:00]

So while we're talking technical terminology, I'm going to bring one last new concept before we can kind of rest. Can you explain to us what MARCoNS is and what's the difference between CIRS, as we've already just explained and described, and MARCoNS ?

Louise Carder

OK, so that's a great question. So Dr. Shoemaker noted that when his CIRS patients protocols were stalling, that they tended to have a form of staph bacteria, so staphylococcus bacteria in their sinuses. And it was a particular gram-negative one called coag neg staph. And this wasn't just present, usually in quite high amounts, it tended to be resistant to antibiotics. And so, multiple antibiotic coag neg staph became shortened to MARCoNS. And the association became established as part of his CIRS protocol.

So, MARCoNS is this nasal antibiotic resistant overgrowth that happens in CIRS, typically because it speaks to the lack of protection in the sinuses from the hormones which are low due to the inflammatory picture. And we see and studies show us, that when these hormone levels return to normal, because the inflammation has come down, there's less colonization of MARCoNS when those hormone levels return to normal. So, we see very clear associations with this ebb and flow of inflammation, hormones and presence of bacteria with biofilm.

Kirsty Cullen

A great segway into biofilm because obviously biofilm can be problematic can't it? We can identify certain pathogens, we can know what we're going to do from a protocol perspective, but biofilm is a sneaky barrier, potentially for our success around those protocols. Do you just want to say a little bit more about what biofilm is and how we circumvent it?

Louise Carder

Yeah, so the way that I explain biofilm to my clients, and it's not very technical at all, it's like some sticky clingfilm complex that they have just secreted over themselves to literally stick themselves to the lining of the tissue. It sounds quite delightful, doesn't it? And this allows them then to be using that, not only as a defense mechanism against the actual antibiotics they might come into contact with, but it's also an exquisite signaling mechanism. So they can share DNA material through that and

communicate with each other as well through this biofilm.

So, yes, when we do nasal swab testing, we can not only test for the bacteria to see what's present, we can also test to see whether there's any biofilm presence and also how strong it is.

Kirsty Cullen - [00:26:32]

Perfect. And so alongside the elements we spoke about, we might also find fungi in the mucus. Why is that important?

Louise Carder

So to some people it's not, actually, fungi are seen by many as just passengers in the mucus. And those people would say that it's the biofilm and the bacterial responses that are believed to be the most damaging. But I like to still consider the fungi, because in terms of building a body of evidence regarding exposure, the fungi side of the test can actually still be useful, especially if you're marrying it up with other tests to really provide that link between the outside and what's going on on the inside so you can really confirm that exposure.

And I think really, one of the main points I would want to get across from this talk today, is that we shouldn't just use one test for considering CIRS, mold or mycotoxins, really the most important thing is about building a body of evidence to say, yes, this is what it is, and we've got evidence from A, B and C related to that. So we've built a really good picture, and then tracking progress by repeating that evidence in line with symptom changes or to establish why the symptoms are not changing as expected. So to get that broad body of evidence relating to the environment, relating to a really good symptom roster and discussion, and then also actually doing some testing to confirm what you've seen. So I don't like to just use one test. We have to actually put that whole picture together for clients, I think.

Kirsty Cullen

And just to bring back around then Louise, why are both CIRS and MARCoNS potentially so relevant to fatigue syndromes? Why are we sort of considering them within this particular arena?

Louise Carder

So, what I see in my clients is a lot of people with chronic inflammatory responses occurring, and this has been exclusively my work for a few years now, and I have noted that this can be for a variety of reasons, but most of my clients have multi system, multi symptom issues for a long time, and they also feel like they've got an invisible illness.

And they may have been told previously that all their blood work looked normal or they have a diagnosis of something like CFS, ME or fibromyalgia, but perhaps a deeper dive, the more consideration of some of the chronic triggers might need to be considered, such as a stealth infection or mold exposure.

And MARCoNS in particular may be useful to consider if somebody's got so far with their practitioner, but then they've stalled because it really can have that stalling impact on cases.

And in terms of how impact for CIRS is on something like chronic fatigue, what we know over the last year is a whole set of newer testing has come out that shows actually that CIRS is a hypometabolic illness. And as I said, this definition came out just last year really, and essentially means that we now know that energy is not able to be produced in the mitochondria or the battery packs inside ourselves as optimally as it is in healthy people and nutrients can't be optimized either. So glucose fats, for example, from the food that we're eating, get diverted elsewhere, which means they're not burnt in a way that gives the body optimal energy.

[00:29:47]

And in addition to this, some of the more technical cellular channels can be affected by stealth infections and also by some of these toxicity factors that we talk about that can compromise energy still further. So they can be seen as ribotoxins that really get to the heart of the mitochondria, the ribosomes, and really cripple it, which means that energy is almost impossible to be created properly.

So if we're not able to metabolize the nutrients that we're consuming, we don't have enough energy to push those nutrients around the body and into the brain. Then we are much more likely to be fatigued, pure and simple. And also having neurological symptoms if we're not pushing those nutrients into the brain with oxygen as well. And that's before we even start to consider the immune and inflammatory effects of the kind of illness that we're talking about.

And in terms of fatigue, I think I need to just take an extra moment here, just because we also have to consider that the CIRS picture was defined by Alzheimer's expert Dr. Dale Bredesen, as being type three Alzheimer's. And this is very much down to the long term hypometabolic, and hypometabolic basically means not able to use the nutrients that we've got and so our metabolism changes. So, it's very much down to that long term hypometabolic picture of CIRS involving lack of energy, lack of delivery of nutrients and oxygen around the body and especially to the brain.

And this is not something that you want going on long term, especially as we can track these changes in the brain on a volumetric MRI scan, which is especially useful when tracking positive progress. So if the chronic immune response hasn't been considered in chronic fatigue case, then it would be really important to consider whether the immune system is involved and also how much of a hypometabolic picture there is.

And the other thing that we also see more of now is the histamine side of things, and how that is also affected by that left hand side, that innate immune response. Histamine is triggered when that is activated regularly. So I think it's important to recognize that a lot of people with a fatiguing picture may also have histamine issues.

And that's becoming a lot more understood now in terms of long-COVID, because we also very often see histamine elevations associated with the CIRS picture and this extends way beyond mast cell activation. So I think we're learning a lot more at the moment with what we've been going through with COVID to actually understand the innate immune response and the histamine response as well. So if that picture is very much something that you're starting to understand, then I think looking at some of the triggers that we talked about today could be useful.

Kirsty Cullen

And of course, it is intrinsically a complex picture, isn't it? And I think this underpins the reason why working with a practitioner can be so central, because it's not always important to understand all of these technical terms and everything that goes on and the complexities behind it. But it is really important to have a practitioner and a support team that understand it and can guide you through it in what is much more of a simplistic way and actually offer practical tools, practical protocol options, that at the end of the day, support and improvements in health.

Louise Carder

Absolutely. And that's why I was drawn to the Shoemaker protocol and Dr. Bredesen's work, because as a practitioner, I recognize this picture in a lot of my clients and I wanted to be able to understand the framework of not only identifying it, but starting to work with it. But as I've already alluded to, also knowing when to hand to a higher authority like a doctor. Also very important as well because of the different ways that this can manifest in people. So, yes, I think it is really important for people to work with practitioners, who not only understand what to do, but also understand when to refer on to people who might know a little bit more, or be able to do more for them.

Kirsty Cullen - [00:33:40]

Louise, it's been a really interesting introduction to these topics. And I know sometimes they're avoided because they can get quite wordy. But I just think it's so important to open that conversation.

If people want to read more about your work, where should we send them?

Louise Carder

So, I think generally, one of the first places I think people should look, if they haven't already, is the survivingmold.com, Dr. Shoemaker's website is a great repository of information.

And even if you don't want to use his protocol or agree with his methodology, the information there is fantastic.

And for anybody not in the U.S. who spells mold MOULD as we do in the U.K, the website is survivingmold.com.

And also, by the time this goes out to the public then there will be a sign post website www.cirs-eu.com that will guide people to many of the great resources that are already out there.

And there are a lot of resources out there. There are a lot of people doing work in this area and it's a really exciting time for sufferers of this kind of illness as well, because there are so many more resources out there now as well. So I'm really excited about that change.

Kirsty Cullen

Louise, thank you so much for your time today. It's much appreciated.

Louise Carder

No problem. Thank you very much for having me.