**Medical Insider COPD Podcast Season 4
The lung microbiome and ICS: Influence of airway inflammation on COPD treatment**

Richard (00:23):

This podcast is intended for healthcare professionals outside the United Kingdom and United States of America only. Welcome to the Medical Insider COPD by Boehringer Ingelheim. A podcast offering a breath of fresh air to clinicians treating COPD across the globe. My name is Dr Richard Russell and I'm a Consultant Chest Physician at Lymington New Forest Hospital in the United Kingdom. I'm a Senior Clinical Researcher at the University of Oxford and I'm the Editor-In-Chief of the International Journal of COPD. I'm delighted to be your moderating host for this season of Medical Insider COPD Podcast. I'm here to bring you news and insights in COPD right from the source directly to you. Thank you for joining us today. Be sure to subscribe to Medical Insider COPD Podcast to ensure you do not miss any of the exciting podcasts in this series.

Richard (01:13)

Today, we'll delve into a publication which I think is worth reading and certainly very thought provoking. It's an editorial in the European Respiratory Journal entitled, “Controlling Chronic Respiratory Diseases in Finland from 1996 to 2018.” Perhaps more interesting than its title. I'm also going to delve into an emerging and exciting topic from social media, looking at advance care planning.

But first, I'm delighted to introduce today's special guest, Professor James Chalmers from the University of Dundee. James is an old friend of mine and he's going to be here to discuss the issue of microbiome in COPD, a very much hot topic. So, welcome to the podcast, James.

James Chalmers ([01:50](https://www.rev.com/tc-editor/Edit?token=9eYbNJAMqXQ8dfTZYFSfOtyOzdVzmkpgGFObC7BXabE5qSLDkBAEX0brSmORf4nIC6paXRz9VED1nDe20k5XMBB9xmw&loadFrom=DocumentDeeplink&ts=106.74)):

Thank you very much, Richard. It's great to be back on the podcast. So, I'm a Consultant Respiratory Physician and a Professor of Respiratory Medicine at the University of Dundee and I do research into the microbiome in COPD and bronchiectasis. In my other role, I'm also the Chief Editor of the *European Respiratory Journal*. It's a pleasure to join you for today's podcast.

Richard ([02:10](https://www.rev.com/tc-editor/Edit?token=fvgR9KNFG8Oj04yz-_KWNivRItAj5sOHNXh8eX_5toV1hyJuogSgt6mUCeYA4aEnvE6F0LXwE_QSEG4Axv10WT-OZbs&loadFrom=DocumentDeeplink&ts=127.44)):

Well, brilliant. So just for the audience to start with, James. I'm hoping that we'll be able to understand what we mean by microbiome, look at the influences that it has on COPD, and also very much look at the treatment influences, and how we should treat people with COPD based perhaps upon the microbiome. Would you agree that would be some good goals?

James Chalmers ([02:26](https://www.rev.com/tc-editor/Edit?token=Dl413OGfLrRg03i9SDFT9PgP3dTqH8xjR9MnJA6InlCDLMCi4Hsy4yXQhdW_i_qXE7dP7Zn9cbvugs1AP57ljyOaG5w&loadFrom=DocumentDeeplink&ts=147.42)):

Yeah. I think that's right. So, the microbiome is something I think the listeners would have heard a lot about in relation to the gut, so we all hear about our gut health and how that can influence different aspects of our health. What we're going to talk about today is the lung microbiome and the fact that the lungs also have commensal organisms that are important for our health, but also important determinants of phenotype and endotype in COPD. And so, I think talking today about how we can take some of the really important basic science discoveries in recent years and put those into clinical practice is what we should do today.

Richard ([02:58](https://www.rev.com/tc-editor/Edit?token=OgVgpBplXjdCnTZAT0HVDI8w_BpsnTwGzP1ZoqwpsnDafd6PQ7xsdoViRH5lJuUz9R6t8W0LQ94qm1c6jXkeWosjr6w&loadFrom=DocumentDeeplink&ts=182.79)):

And if I was to challenge you right at the very beginning for a key message for our listeners, what would you say?

James Chalmers ([03:02](https://www.rev.com/tc-editor/Edit?token=yyqp2Frp_D8Uj5vGY7wqCTlHc8FJmGI7GZu-GV9LPyqQbhEhp_AFVOhd__j3fRosaQctOusmt671L1iE2G6_ar48fys&loadFrom=DocumentDeeplink&ts=188.07)):

So really important recent data shows that the microbiome has big effects on our patients' phenotype and endotype. Patients who have low blood eosinophil counts, which we recognise in clinical practice as an important biomarker, they have an abnormal microbiome with lots of gram-negative bacteria, what we call Proteobacteria. And recent studies have shown that inhaled steroid can stimulate overgrowth of them leading to infections and pneumonia. Whereas patients with high blood eosinophil counts tend to have a more normal healthy microbiome and are much less susceptible to harmful effects of inhaled steroid. So, my take home message will be we can use the blood eosinophil count not just to detect who's going to respond to an inhaled steroid, but also who's at risk of infectious complications, and that's an important new message, I think, that we need to get out there.

Richard ([03:52](https://www.rev.com/tc-editor/Edit?token=9khSxhcbw_MvGJmeZ5uhs246SVoRyYL5FFkhUoCDVZqxEvTvsysZ72xq6duwBq67oNlyGm4oV4RmU_wq0xBN1eX70jc&loadFrom=DocumentDeeplink&ts=241.14)):

Right. James, to start with, I think there are some issues here with terminologies somewhat and can you just describe, briefly, what we mean by microbiome, and also, briefly, how we have looked at it in the past and how we look at it now, scientifically?

James Chalmers ([04:04](https://www.rev.com/tc-editor/Edit?token=WfpkmSWkZiBetODVGbB1HyvztzZtPUo69q8f6fpNiwbJWHHNsf9FYCqzNLs_Q1Sz7ZP4gSUcf_7uO9tknPHdi2of8wQ&loadFrom=DocumentDeeplink&ts=265.08)):

Absolutely. So, the microbiome, that word means all of the organisms and their DNA that occupy a body space, so in this case we are talking about the airway. Now the microbiome includes bacteria, viruses, fungi other microorganisms. But typically, we're talking about bacteria because the vast majority of microorganisms in the lung and in the gut are bacteria. Where do those bugs come from? They come from the upper airway, mostly by microaspiration. So, when we're sleeping, little bits of our upper airways secretions get down into lungs and bacteria follow, and create a flora in the lungs that maintain health. So that's what we mean when we're talking about the microbiome in our terminology.

James Chalmers (04:44)

The technologies that we use, traditionally, the only thing we would use in clinical practice for COPD would be sputum culture, that's very insensitive, it would maybe pick up one organism, when in fact there's hundreds of different species of bacteria in the lung at any one time. And so, novel technologies like sequencing, allow us to read all of the DNA within a sample. And therefore, detect lots of bacteria, including some of that aren't easy to grow in the laboratory. So, you can build up a fingerprint, a profile of a patient's lung and show how different COPD patients differ at the molecular level.

Richard ([05:19](https://www.rev.com/tc-editor/Edit?token=bFC7N9P8riuvyXqbO9-jbLVeY9GRCgeXkh8WfVG1NkLeJzK_Vew69pkIJpW5yy6yK6DcYMy5NU4ohCEC4BHjw5xS8ho&loadFrom=DocumentDeeplink&ts=348.72)):

And you already mentioned the role of potentially blood eosinophils as something that we can measure as well. And this comes, therefore, down to the inflammatory endotype. Can you explain what's meant by that word inflammatory endotype?

James Chalmers ([05:30](https://www.rev.com/tc-editor/Edit?token=BWoV7t2KGwiVYL_gcxbh8b4owUNFr25QbKLPFaTS9Ycjjl9lKCmjFX4_lSHAVkDdTC__lD2yvzkF6LR3Gw-DceK31zU&loadFrom=DocumentDeeplink&ts=360.21)):

Absolutely. So, let's go back to first principles. You know, what happens when the bacteria in the lungs go wrong? What happens if you got too much of *haemophilus*, for example, in your lung? Your body reacts to that by releasing chemicals that recruit neutrophils to the lung. And we recognise that neutrophilic COPD is the most common inflammatory endotype. So, if you've got infection in the lungs, you will have a neutrophil response. On the other hand, if you have, for example, sensitisation to allergens or different stimuli, you get activation of T cells and you get recruitment of eosinophils. And so, you have a different inflammatory endotype which is eosinophilic COPD, which effects about 20% of the population. The reason why those two endotypes are important to recognise is the treatment is completely different. You don't want to give immunosuppressive drugs to somebody that's got lots of infection, and you don't want to give antibiotics to somebody that doesn't have infection. And so, that's where the clinical relevance of this kind of inflammatory endotyping comes in.

Richard ([06:27](https://www.rev.com/tc-editor/Edit?token=yxVigLtNU1gJ3Z_lyghGNfKMSu2OtwwRxX5F_49QyKnZ9DPPjxfXWMI-VCzgADA5OV2bLcW-22jxCc1WpS3TyIn5qS4&loadFrom=DocumentDeeplink&ts=425.25)):

Let's come back to the bugs for a second, and you mentioned that it's not just bacteria, but we do very much focus on bacteria because of our traditional focus on culture. But which bacteria are we really talking about? And actually, which ones would you pick out as being really clinically important to help us make decisions?

James Chalmers ([06:46](https://www.rev.com/tc-editor/Edit?token=pF8h1cFJjboWBwjDrFOvcfMHzfw2nco-YormrX6fWg8NC3vXTDti0TSoA1ngDsdFYB_y1SstROLf2r3NSIlwv7OQPrM&loadFrom=DocumentDeeplink&ts=442.86)):

Yeah. So, if you look at the, the recent GOLD statement which is the first time a clinical guideline or a clinical statement has put microbiome into clinical practice, they talk about the high-risk microbiome being the dominance of this thing called Proteobacteria. So, what does that mean? It's actually the gram-negative pathogens that you will all have heard about. So, in the lungs most commonly the nasty gram negatives are *Haemophilus influenzae* by far in COPD. Then organisms like *Moraxella catarrhalis* and very rarely *Pseudomonas aeruginosa*. There are other pathogens, of course, *Staph aureus*, *Strep pneumoniae*, but they're much less likely to cause chronic airway infection in people with COPD. So, those are the ones that we should be thinking about in clinical practice and they're associated with a low blood eosinophil count because they stimulate a neutrophil response.

Richard ([07:34](https://www.rev.com/tc-editor/Edit?token=5T_sDeUpOE7pCFN6GYPL_qDbycRgqMKVPllfrszMyr-1Osbd_j3Zs6xWNhvCJzqSnKSOwyIBoO0TpcRVUlcXizOJtzY&loadFrom=DocumentDeeplink&ts=495.75)):

So, you already mentioned eosinophils and microbiome and certainly inhaled corticosteroids and its use. Does the microbiome and eosinophils give us a clarity now how we should use these particular drugs in COPD?

James Chalmers ([07:49](https://www.rev.com/tc-editor/Edit?token=orBLLG2azaW138nk2Q29Jhka-KeuhrSoURV6jXUs7Ts1MKLc2QQvOV7EQneDG55YLREqdS98gVgbCpjkTpW_OonZ0kE&loadFrom=DocumentDeeplink&ts=511.38)):

So, we've known for a few years now that if you look at big randomized clinical trials, the patients, they get an exacerbation benefit, or patients with eosinophils 300 cells or above. Where the uncertainty has been is around what's the risk:benefit in patients with lower blood eosinophil counts? What the microbiome now adds is to say that those patients who've got eosinophils below, let's say 100 or 150, they're much more likely to have Proteobacteria gram-negative dominant negative microbiome. And randomised studies showed that if they're treated with inhaled steroid, those bacteria start to grow and increase in load. So, we have really stronger messages from the microbiome, that we should avoid inhaled steroid in patients that have gram-negative infection and who have low blood eosinophil counts, and I think that's really useful.

Richard ([08:32](https://www.rev.com/tc-editor/Edit?token=-pQE-bL6bc_D4sbVrufbDvXDuosC__nWMGmsi0G08sCO67ZgJ-xWpClAHKpePXO1IbToJMwGWdx_kEiZ3b9jZbC3A6A&loadFrom=DocumentDeeplink&ts=561.09)):

Do you think it's justifiable or certainly arguable that we should be looking at this before we think about prescribing inhaled corticosteroid?

James Chalmers ([08:41](https://www.rev.com/tc-editor/Edit?token=i_2fb1Uim70HdPpedZXLVXOMpfq7gNzvBpZ7OJXoR4lNgkmoY7VYfthhpHXhl7hqR3xxVpP1B-POuX4WBlZip0oOM7M&loadFrom=DocumentDeeplink&ts=569.43)):

Yeah. And I do in my clinical practice. So, I use the blood eosinophil count as my first test to whether I should think about an inhaled steroid, and if it's low, I don't think about an inhaled steroid. But I also look at, "Does the patient have bronchiectasis? Are they likely to have gram-negative infection? What are the results of the sputum cultures?" And if the patient does have infection, even if the blood eosinophil count in that sort of intermediate range, it makes me think about other things before I think about an inhaled steroid, because we don't want to promote overgrowth of those infections. And that's where I think the microbiome data has helped to clarify our clinical practice.

Richard ([09:18](https://www.rev.com/tc-editor/Edit?token=jf_EEfRuUuyrcPA4iHXUWhROwYsai0ZnUVX7dcmRmnSmij7qcWnE6jGXuOaERUZqD496085dTy1mABjN88ftoj5QEgU&loadFrom=DocumentDeeplink&ts=606.96)):

And, I guess, very often when we're not aware of these data and actually don't think perhaps about them properly, we overprescribe inhaled steroids very often.

James Chalmers ([09:23](https://www.rev.com/tc-editor/Edit?token=0oTORAAaU-cVWMYbCdoKV2lO28Vaoc9aLD1Ta-YHa6WyEonzDYQn4RLZc_v-UAz8g5WZYTEuPvt4InLBHksNDG--jkM&loadFrom=DocumentDeeplink&ts=615.93)):

So, we know that there's a big problem that, probably around 20% to 25% of COPD patients benefit from inhaled steroid. But 60% to 70% in many countries are on inhaled steroid, and so we do have an over prescription problem. And that's why for a number of years, societies like ATS and ERS have been advocating consideration of ICS withdrawal in patients that have low blood eosinophil count. And that's something that I do in my clinical practice is consider taking them away, particularly if somebody's got bacteria in the airways and are suffering bacterial exacerbations.

Richard ([09:57](https://www.rev.com/tc-editor/Edit?token=AkUMyU6dIjiF6v5p1ig7shz4VBpOVPNKvljjkVfHs5eS6wSVzZdZKuyPWvgDlIzDhGwVcOKv1-CYi_t18muEBlMnCLk&loadFrom=DocumentDeeplink&ts=654.36)):

You mentioned exacerbations, and let's unpack that now. Is there a difference between the microbiome or the preponderant bacteria, particularly, that we find in exacerbation versus stable state?

James Chalmers ([10:06](https://www.rev.com/tc-editor/Edit?token=AkUMyU6dIjiF6v5p1ig7shz4VBpOVPNKvljjkVfHs5eS6wSVzZdZKuyPWvgDlIzDhGwVcOKv1-CYi_t18muEBlMnCLk&loadFrom=DocumentDeeplink&ts=654.36)):

Surprisingly not. So, probably one of the most surprising results of all the microbiome studies that have been done in COPD has been that the microbiome is very similar between stable disease and exacerbation. Everybody thinks that exacerbations are caused by new bacteria jumping into the lungs. There's very little evidence from the microbiome that that's the case. Most exacerbations are probably viral, and they stir up the microbiome that's already there in the lungs. So why is that important to understand that? It means that you can take the inflammatory and infectious status of a patient when they're stable, when you see them in clinic, and predict from that, what their exacerbation biology is likely to be. So, if they have *Haemophilus* when you see them when they're well, they're going to have *Haemophilus* when they have an exacerbation probably. And inhaled steroid is not going to prevent their exacerbation. Similarly, if they've got a raised blood eosinophil count and no abnormal bacteria in the airways, they're probably not going to have any bacterial event when they exacerbate. And so, you can predict to an extent patient behavior based on their endotype, which is that word we keep coming back to, Richard.

Richard ([11:13](https://www.rev.com/tc-editor/Edit?token=_TW5VrRxJCwbD7QqkvYNZQQhXdRs2gAw_-7WTtlu0-VsBwVKWFxZ4ngdw9sQKu3YP-JH8llbfVsIuHg583pxPt8RpxM&loadFrom=DocumentDeeplink&ts=746.01)):

And do you think in the future or how far off are we, when we'll be able to use near patient testing to enable us to make real-time clinical decisions based upon measurements of eosinophil and the microbiome?

James Chalmers ([11:26](https://www.rev.com/tc-editor/Edit?token=_TW5VrRxJCwbD7QqkvYNZQQhXdRs2gAw_-7WTtlu0-VsBwVKWFxZ4ngdw9sQKu3YP-JH8llbfVsIuHg583pxPt8RpxM&loadFrom=DocumentDeeplink&ts=746.01)):

So, I think we're there now already with the blood eosinophil counts. I use it in my clinic every day. So, you have that full blood count, it gives you the eosinophil count and you can use that straight away. From an infection perspective, I think the pandemic has massively accelerated the development and uptake of molecular diagnostics. And I think we're not far away from every hospital having a molecular test that can rapidly detect *Haemophilus* and *Streptococcus* and other bacteria that could give us a usable point of care microbiome assessment. So, I think it's a really exciting time, Richard. And I think in the next 10 years, that'll be part of our assessment of people with COPD.

Richard ([12:0](https://www.rev.com/tc-editor/Edit?token=Lqt23JQrdWxXc1jT8ar5YjAzjTxP0NWPuBYDG6_Yqyv9Tos5MURlzmVnQ1t6Yf8UV31veYxcFDeOpl4CjHU7RgrO1DQ&loadFrom=DocumentDeeplink&ts=844.11)2):

You're absolutely right. And certainly, our ability to really very rapidly identify viruses has actually accelerated beyond all predictions, I think, which is true. Let's talk about some research that's going on, James, and really exciting to get into this, the understanding of the INCOGNITO study, tell us about the study.

James Chalmers ([12:18](https://www.rev.com/tc-editor/Edit?token=Lqt23JQrdWxXc1jT8ar5YjAzjTxP0NWPuBYDG6_Yqyv9Tos5MURlzmVnQ1t6Yf8UV31veYxcFDeOpl4CjHU7RgrO1DQ&loadFrom=DocumentDeeplink&ts=844.11)):

So, I've already spoken quite a bit about the influence of inhaled steroid on the microbiome. A question that we had was, if we take away inhaled steroid, and somebody has an abnormal microbiome, can we fix it? Can we change back to a more healthy microbiome if we take away the inhaled steroid? And so, the INCOGNITO trial was a randomized controlled trial of tiotropium and olodaterol, against fluticasone/vilanterol in patients that have been treated at least a year with inhaled steroid.

So, we took away the inhaled steroid in half the patients, we kept it going in the other half and we did sputum cultures on a monthly basis to see whether the microbiome changed. What we saw is that the bacterial load, the amount of bacterial didn't change, but the composition of the airway did change. So, the gram-negative bacteria seem to go down in patients that were randomised to withdraw ICS and the beneficial commensal bacteria like S*treptococcus* went up. So, the message of the study seems to be that you can to an extent return to a more normal microbiome if you take away inhaled steroid in patients that have low blood eosinophil count who shouldn't really have been on them in the first place.

Richard ([13:27](https://www.rev.com/tc-editor/Edit?token=EtdFTyjMmu5w1PhARVEI5byCXKpAoKK8dwG15bL7XXICOfVD7lyrzszAXU7vOk-2MXE7MNFggluidKNl6o-PpaTARfc&loadFrom=DocumentDeeplink&ts=918.96)):

Do you think that has implications therefore for prognosis patient to disease behavior?

James Chalmers ([13:32](https://www.rev.com/tc-editor/Edit?token=S33xiztZ_UmFkfFQM_6LKAPsi09j0SoUtFXrG5vmeGUA84brNevmg4oC_1gHCyCivacecNNElX4GR5OA_ntn8EcIn5c&loadFrom=DocumentDeeplink&ts=924.48)):

Yeah. I mean I think we now recognize that the microbiome is an important part of health, and so we want to maintain healthy microbiome in our patients’ lungs and in our patients’ gut, and that's part of returning patients to a normal homeostasis. And so, I think this has implications for saying there are potential long-term benefits in the appropriate patients of avoiding inhaled corticosteroid to maintain a normal lung microbiome.

Richard ([13:58](https://www.rev.com/tc-editor/Edit?token=EtdFTyjMmu5w1PhARVEI5byCXKpAoKK8dwG15bL7XXICOfVD7lyrzszAXU7vOk-2MXE7MNFggluidKNl6o-PpaTARfc&loadFrom=DocumentDeeplink&ts=918.96)):

Well, I always conclude by asking my guests to tell us a brief summary and the key take aways from this discussion. This isn't easy. So, James, you've got a couple of sentences to really pull out some pithy points for our listeners.

James Chalmers ([14:12](https://www.rev.com/tc-editor/Edit?token=lhAbzBrbsRdtuJas_NxKQNYPediq0E92KDuJLLoN8Oq3DbzfsWsafQlsN1aRS_AV_mjnz3VqYlAFc-Et6svT14o1dXQ&loadFrom=DocumentDeeplink&ts=979.14)):

So, GOLD recently published an opinion piece in the Blue Journal, where for the first time they said, "The microbiome should influence what we do in clinical practice." They said, "People who got low blood eosinophil counts have high levels of Proteobacteria and abnormal microbiome, and for that reason we should avoid ICS in that group. And in patients that have high blood eosinophil counts, they typically have a more normal microbiome. And it's appropriate to consider inhaled steroid in that group." So, the pithy summary is, understanding and interpreting the microbiome can help us to better target therapy to prevent exacerbations.

Richard ([14:48](https://www.rev.com/tc-editor/Edit?token=sqA33HuDrG_Cpg7hvfO7npXM9QVu5LWcSy9Q6hpxY0l6tVEvDMp8H4Y5roNuu7vNqY1NtBeFPPPO_kEtil1u1YkBUsI&loadFrom=DocumentDeeplink&ts=1022.37)):

Professor James Chalmers, it's been fantastic talking to you as ever. Thank you for joining us again on the Medical Insider COPD Podcast. I look forward to working with you again very soon. Thank you, James.

James Chalmers ([14:59](https://www.rev.com/tc-editor/Edit?token=o4Q-yfyP9uu6TWMukAyNCjPPfMCcD892BdKOuNkoliKWRSalOH2Qgafkx7TZyqLD2kuWiXZpYZv3ZWjJx0VABQwp_7Q&loadFrom=DocumentDeeplink&ts=1033.74)):

Pleasure, Richard.

Richard (15:05)

So, before we look at an important hot topic in social media, advance care planning in COPD, let's look at this interesting and different type of paper. This is an editorial. It's entitled “Controlling Chronic Respiratory Diseases in Finland from 1996–2018”. It's published by Tiina Mattila and colleagues from Finland and it's published in the July 21st edition of the European Respiratory Journal, Volume 60. This is an editorial and it describes the actions and the outcomes of what's gone on in Finland, summarising five programs in respiratory medicine from the early 1990's. The population of Finland is around four million people and they put in place significant public health primary and secondary care programs to improve the respiratory health of this nation.

So, what did they do in COPD? Well, they looked at investing in secondary care to improve diagnostics and improve guideline related care. And in primary care, they put in place a network of nurses and doctors to look at smoking cessation, early diagnosis with accurate spirometry, and also implementing guidelines. So, what did they find? They looked at care, time off work, costs, and healthcare interventions. They found that over the period of the intervention and following, there was no rise in prevalence of COPD, indeed it reduced a little bit. There was a significant reduction in inpatient workload from COPD. No change in outpatients though, people were still being seen. And there was no real change in overall cost of this disease. They hope, over time, there'll be a reduction prevalence because of the smoking cessation. Changes in asthma, for example, were even more dramatic than COPD.

And I think it's important to contextualise this finding and this work that's been done in every other country, particularly in the West, COPD is increasing because of smoking and because of age. So, what does this mean for us? Well, I think this should inspire us to challenge our governments and challenge our respiratory environments to say, actually, we can make big changes. We can see those changes within a 10-year time period. And actually, just because it's difficult doesn't mean we can't do it by sensible investment in things which really work. And I hope that we'll listen to the lessons from Finland and actually maybe take some of them forward in our own countries.

Richard ([17:31](https://www.rev.com/tc-editor/Edit?token=EtdFTyjMmu5w1PhARVEI5byCXKpAoKK8dwG15bL7XXICOfVD7lyrzszAXU7vOk-2MXE7MNFggluidKNl6o-PpaTARfc&loadFrom=DocumentDeeplink&ts=918.96)):

And I'm now going to talk about what's exciting and new in the COPD world online. This is talking about advance directives. This is advance, not advanced. It's talking about what a patient would like. What are their wishes? What are their goals? What would they like to do and have done to them? And also, what they'd not like to have done to them as their disease progresses and they approach the end of life.

There are many patient quotes about this and "palliative care is not just about hospice," is one particular one. And there are many positive experiences of having these discussions with patients, much of it driven through Twitter but also on the COPD Foundation website. And on this website, you'll find downloads for patients about how to discuss their condition, downloads for you to discuss with your patients how to discuss these conditions, and also for family members to understand how a patient can plan for the future. So, this is useful for us in healthcare, as healthcare practitioners, care organisations, and also hospices. So, please have a look at it. Remember, this is about advance care planning. Planning for their future and for your interactions with them.

Richard ([18:38](https://www.rev.com/tc-editor/Edit?token=EtdFTyjMmu5w1PhARVEI5byCXKpAoKK8dwG15bL7XXICOfVD7lyrzszAXU7vOk-2MXE7MNFggluidKNl6o-PpaTARfc&loadFrom=DocumentDeeplink&ts=918.96)):

So, I hope you've enjoyed today's Medical Insider COPD podcast. Be sure you subscribe so you don't miss any of the exciting podcasts going forward. And I hope you've enjoyed what you've heard today on medical microbiome, on the new paper looking at medical practice and how we change policy and improve the life of our patients, and indeed, how we talk to our patients to improve their end-of-life experiences. Thank you very much for joining me and I look forward to speaking to you again very soon.