

University of Dundee

## Highlights of the ers lung science conference and sleep and breathing conference 2021 and the new ecmc members

Goodwin, Amanda T.; Karadoan, Dilek; De Santis, Martina M.; Alsafadi, Hani N.; Hawthorne, Ian; Bradicich, Matteo

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






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 Amanda T. Goodwin<sup>1,20</sup>,  Dilek Karadoğ an<sup>2,20</sup>,  Martina M. De Santis<sup>3,20</sup>,  Hani N. Alsafadi<sup>4,20</sup>,  Ian Hawthorne<sup>5,6,20</sup>,  Matteo Bradicich<sup>7,20</sup>,  Matteo Siciliano<sup>8,20</sup>,  Sezgi Şahin Duyar<sup>9,20</sup>,  Adriano Targa<sup>10,11,20</sup>,  Martina Meszaros<sup>7,20</sup>,  Michail Fanaridis<sup>12,20</sup>,  Thomas Gille<sup>13,14</sup>,  Holly R. Keir<sup>15</sup>,  Catharina C. Moor<sup>16</sup>,  Mona Lichtblau<sup>17</sup>,  Niki D. Ubags<sup>18</sup>,  Joana Cruz<sup>19</sup>

 joana.cruz@ipleiria.pt

 @EarlyCareerERS

Early career forum

# Highlights of the ERS Lung Science Conference and Sleep and Breathing Conference 2021 and the new ECMC members

The Lung Science Conference (LSC) and the Sleep and Breathing Conference (SBC) are two conferences organised by the European Respiratory Society (ERS), the latter held in association with the European Sleep Research Society. This year, the LSC and SBC were both held in a virtual format with the participation of researchers and clinicians from around the world. The participation of Early Career Members (ECMs) was notable in both events: 216 of 363 (60%) delegates attending the LSC were under 40 years old, and 315 of 920 (34%) delegates were  $\leq 40$  years of age at the SBC. Both conferences included outstanding talks on the most recent advances in respiratory medicine and science, oral/poster communication sessions on novel research, exciting opportunities to network with peers, and much more!

This paper provides a brief overview of some of the most remarkable sessions of the LSC and SBC, written by ECMs attending the sessions.

We also present the new members of the Early Career Member Committee (ECMC) of the ERS from Assemblies 1, 4, 10, 12 and 13, who were elected in the latest round of ERS elections. Welcome aboard!

## LSC Highlights 2021: Repair and regeneration in chronic lung disease and lung cancer

The LSC 2021 on “Repair and regeneration in chronic lung disease and lung cancer” was held

in a virtual format on 11–12 March 2021. In addition to an outstanding scientific programme, the LSC provides excellent opportunities for career development and inclusion of ECMs. Poster sessions were chaired by an ECM who was paired with a senior faculty member to allow ECMs to become acquainted with session chairing. In addition, the ECM authors of highly scored abstracts were able to take part in an online mentorship session. The ECMC organised a session specifically for ECMs on “Research output for career advancement”. Below, we describe the scientific highlights of LSC 2021 for those who could not attend and for those who wish to recall the sessions. The most important take-home messages of the LSC sessions are provided in table 1.

The ERS presents several awards at the LSC. The five highest ranked abstracts from ECMs were presented during the Young investigator session. Martina De Santis received the William MacNee Award for the best presentation in this session. In addition, the organisers presented eight Distinguished Poster Awards to Naoaki Wantanabe, Ian Hawthorne, Róisín Mongey, Georgia Giotopoulou, Manuela Platé, Carolin Koss, Cinta Iriondo and Emil Rehnberg for their excellent contributions during the poster sessions.

## Opening lecture

The opening lecture of the LSC was delivered by Naftali Kaminski (New Haven, USA). In this talk,

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 @ERSpublications

This article provides a brief description of some of the most remarkable sessions of the @EuroRespSoc Lung Science Conference and the Sleep and Breathing Conference 2021 and presents the new incoming members of the ECMC (@EarlyCareerERS) <https://bit.ly/2RSDP40>



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**Table 1** Take-home messages from the ERS Lung Science Conference 2021

Session	Type	Take-home messages
<b>Opening lecture</b>		Developmental programmes are reactivated in idiopathic pulmonary fibrosis and may represent an attempt at alveologenesis in the damaged lung.
<b>Cell plasticity in lung development and disease</b>	Invited lectures	Innovative methods such as single cell RNA sequencing (scRNASeq) and lineage tracing can be used to characterise distinct cellular populations in the lungs. Induced pluripotent stem cells (iPSCs) could be useful tools for the study of lung disease and regeneration.
<b>Stem cell niche interactions</b>	Symposium	External factors can impact the interaction between lipofibroblasts and alveolar type 2 (AT2) cells. The alveolar type 1 (AT1) cell is a signalling hub during alveologenesis.
<b>Epithelial stem cell in disease</b>	Symposium	Regarding squamous cell lung cancer (SqCC), step wise progression from metaplasia to carcinoma <i>in situ</i> and finally to invasive carcinoma is not irreversible and progressive samples had greater somatically acquired genetic damage than regressive samples. Lung cancers in smokers are clearly separated from lung cancers in nonsmokers based on the molecular and genetic analysis. Club cells are important in alveolar maintenance and carcinogenesis and may be a therapeutic target against premalignancy and chronic lung disease.
<b>Lessons from limb regeneration of the axolotl</b>	Keynote lecture	The understanding of molecular mechanisms of highly regenerative species may allow for uncovering processes for (re)activation of regenerative pathways in regeneration-incompetent species.
<b>Cell-matrix interactions in the lung and beyond</b>	Invited lectures	The extracellular matrix affects cell behaviour in several ways including mechanical cues, structure, composition, alignment and topology. Such effects have several implications on immune response, stem cell differentiation, cellular proliferation, and disease state.
<b>Therapies: organoids, drugs, cell therapy</b>	Invited lectures	Lung organoid culture techniques can be used to understand mechanisms of lung disease. Novel techniques can be used to better understand the clinical effect of mesenchymal stromal cell (MSC) therapy. Cellular reprogramming of cancer cells to antigen-presenting cells downregulates their tumourigenic capacity.
<b>Early-career delegates session</b>	Symposium	Pre-prints are important to accelerate science communication, facilitate networking. “Peer review” is a tool for scientific gatekeeping. Poor experimental design, unjustified conclusion, poor readability, poor introduction and poor review of literature, poorly described methods, confusing presentation are considered as reasons of getting rejections. Aim, Audience, Awareness, Articulation are the 4A principles of successful scientific writing. An ideal editorial should be an opinion maker, reconciliatory as well as providing its own intellectual contribution rather than just repeating or summarising findings of the publication it refers to.
<b>Young investigator session</b>	Oral presentations	Outstanding talks were given by early career scientists on their most recent advances in respiratory medicine and science.

Prof. Kaminski challenged the audience to consider whether pulmonary fibrosis, a disease usually associated with ageing, is driven by aberrant lung developmental processes.

Idiopathic pulmonary fibrosis (IPF) has been the subject of escalating research efforts in recent years. The current paradigm is that IPF occurs secondary to repetitive alveolar epithelial microinjuries in predisposed individuals, with the activation, migration, and proliferation of fibroblasts

and macrophages with matrix remodelling [1]. However, this model does not explain many characteristic features of IPF, such as myofibroblast foci, honeycomb cysts and traction bronchiectasis, in addition to the relentlessly progressive nature of this disease.

The possibility that IPF may involve the abnormal reactivation of developmental signalling was first suggested over a decade ago [2]. Since then, transcriptomic analysis of human lung has revealed

that numerous developmental pathways, including wingless-integration (WNT), sonic hedgehog (SHH), transforming growth factor (TGF)- $\beta$  and bone morphogenetic protein (BMP) associated signalling are activated in the IPF lung [3]. Furthermore, the microRNA profile of the IPF lung is very similar to that seen in fetal lung, yet distinct from the healthy adult lung [4]. These data imply that a reversal of lung differentiation may occur in IPF, resulting in the expression of developmental markers.

Further data to support this hypothesis came from a large single cell RNA sequencing (scRNASeq) study of lungs from patients with and without IPF. The IPF Cell Atlas is an open access resource of scRNASeq data from over 300000 cells covering all human lung cell types ([www.ipfcellatlas.com](http://www.ipfcellatlas.com)). This work demonstrated dramatic alterations to epithelial cells within IPF lungs, with reduced numbers of type 1 and type 2 alveolar epithelial cells and increased numbers of airway epithelial cells. A novel population of epithelial cells, termed aberrant basaloid cells, was also identified in IPF lungs. These cells co-express basal epithelial, mesenchymal, senescence and developmental markers, and are located adjacent to myofibroblast foci [5]. Importantly, these aberrant basaloid cells express the development-associated transcription factors SOX-9 and SOX-11 [5], supporting the evidence for the involvement of developmental pathways in IPF.

In addition to the epithelial alterations, changes were seen in other cell populations. The RNA expression profiles of IPF lung endothelial cells shifted from that of specialised alveolar endothelial cells to a bronchial vascular endothelial cell pattern [5]. Furthermore, a subpopulation of myofibroblast-like alveolar fibroblasts accumulated in IPF lungs, which may be related to cells that drive alveolar septation during alveolarisation. Overall, these data suggest a reactivation of developmental programmes in IPF.

Prof. Kaminski concluded with the thought-provoking hypothesis that the altered cellular populations found in the IPF lung may result from a failed attempt at alveologenesis, with the aim of re-establishing normal alveolar respiratory units. Future work is required to confirm or refute this hypothesis, and the answer will be highly anticipated by those working in this field.

## Cell plasticity in lung development and disease

Killian Hurley (Dublin, Ireland) started this session by discussing how induced pluripotent stem cell (iPSC)-derived cells could be used as tools to understand lung regeneration and disease. Alveolar type 2 (AT2) epithelial cells are central to the pathogenesis of many diseases, but primary AT2s are difficult to obtain for *in vitro* studies. Induced type 2 epithelial cells (iAEC2s) can be derived from iPSCs generated from blood samples. In this 28-day protocol, iPSCs

are cultured with small molecules and growth factors which promote the differentiation of iPSCs through endoderm progenitors, eventually reaching NK2 homeobox 1-expressing (NKX2-1<sup>+</sup>) primordial lung progenitors which differentiate into iAEC2s in three-dimensional (3D) culture [6, 7]. However, the cells resulting from this protocol are heterogeneous and a significant proportion lose their lung (NKX2-1<sup>+</sup>) phenotype [8]. A scRNAseq study of NKX2-1<sup>+</sup> progenitor cells at various timepoints of this protocol revealed a bifurcation of cellular differentiation into lung and non-lung endodermal cell fates [9]. Importantly, this work found that a reduction in WNT signalling at day 17.5 of the protocol gave the optimal yield of the target NKX2-1<sup>+</sup> iAEC2s [9], which have numerous practical applications for disease models [10].

Next, Joo-Hyeon Lee (Cambridge, UK) described the journey of alveolar stem cell differentiation during alveolar regeneration. AT2 cells are essential for alveolar regeneration following injury, and differentiation of AT2 into alveolar type 1 (AT1) cells facilitates alveolar repair. Using data from scRNAseq, *in vivo* lineage tracing, and organoid models, a distinct AT2 cell population called damage-associated transient progenitors (DATPs) has been identified. During alveolar regeneration, interstitial macrophage-derived interleukin (IL)-1 $\beta$  primes IL-1 receptor-1-expressing AT2 cells to differentiate into DATPs *via* a hypoxia inducible factor (HIF)-1 $\alpha$ -mediated glycolysis pathway [11]. These DATPs can subsequently differentiate into mature AT1 cells. Notably, chronic inflammatory states mediated by IL-1 $\beta$ , such as IPF and lung adenocarcinoma, prevent AT1 differentiation, resulting in DATP accumulation and impaired alveolar regeneration [11]. Overall, these data suggest that DATPs are important for lung repair, and dysregulation of DATPs may contribute to lung disease.

Finally, Joachim Schultze (Bonn, Germany) discussed macrophage plasticity in COPD. scRNAseq data of immune cells obtained from the bronchoalveolar lavage fluid (BALF) of patients with COPD and healthy controls revealed that most BALF immune cells are derived from monocytes or macrophages. Furthermore, over 10 different macrophage subsets have been identified according to their gene expression profiles. While these macrophage subtypes share many aspects of their gene expression profiles, some expression patterns were specific to individual cell types. In COPD, alveolar macrophages expressed less HLA-related genes, increased proliferation markers, and increased markers of monocyte-like macrophages than cells from controls. The predicted pathways that lead to this reprogramming of alveolar macrophages in COPD include NOTCH, WNT, TGF- $\beta$ , tumour necrosis factor (TNF), and circadian-rhythm-related genes. This work has given great insight into the pathways that drive macrophage differentiation which are relevant to the pathogenesis of COPD [12].

## Stem cell niche interactions

The lung has an endogenous capacity for repair and regeneration after lung injury. To better understand how this repair occurs and why it is aberrant in some chronic lung diseases, there have been substantial efforts to identify the signals regulating lung stem cell self-renewal and differentiation. In this session, lung stem cell niche interactions were discussed.

Saverio Bellusci (Giessen, Germany) opened this session by describing the epithelial-mesenchymal interactions involving fibroblast growth factors (FGFs). He described the interaction of lipofibroblasts with AT2 cells that allow for AT2 self-renewal. External factors including diabetes, obesity, sex and ageing can impact on the interaction between lipofibroblasts and AT2 cells, with a progressive loss of function of lipofibroblasts and decline in AT2 cell renewal. When a viral infection occurs, AT2 cells are damaged and lose their renewal abilities as lipofibroblasts differentiate into myofibroblasts. Prof. Bellusci concluded his presentation by proposing that TGF- $\beta$  inhibitors could be potential candidates to reverse deranged repair in the alveolar epithelium.

The next talk focused on the role of the microvascular niche and AT1 cells in lung regeneration. Edward Morrissey (Pennsylvania, USA) discussed the endothelial heterogeneity in the alveolus, including CD34<sup>hi</sup>/Car4<sup>hi</sup> endothelial cells which express high levels of angiogenic factors and are probably important in guiding AT1 development and behaviour [13]. Prof. Morrissey further explained how the endothelial cell landscape changes after influenza infection, with an increase of proliferating endothelial cells. These proliferating endothelial cells form a signalling niche in the alveolus after injury, regulating AT1 and AT2 cell and mesenchymal cell population proliferation. Next, the role of AT1 cells was discussed in more detail. AT1 cells are usually described as simple structural cells required for gas diffusion. By showing scRNASeq data, Prof. Morrissey proposed a new role for the AT1 cell: a signalling niche during alveologenesis [14]. AT1 cells play an important role not only for lung development but also for lung regeneration and repair and this can have big implications for diseases such as IPF and COPD.

## Epithelial stem cell in disease

Sam Janes (London, UK) started the session with the topic "Tobacco smoking and somatic mutations in human bronchial epithelium". Detecting and treating lung cancer earlier was the rationale of the presentation. Regarding squamous cell lung cancer (SqCC), stepwise progression (from metaplasia to carcinoma *in situ* and finally to invasive carcinoma) was mentioned not to be irreversible. Prof. Janes gave examples of the analysis of pre-invasive lung adenocarcinoma *in situ* (CIS) lesions in two patients. One patient was diagnosed with CIS and, after stable

disease for 2 years, an index biopsy in the fourth year showed progression to invasive lung cancer. The second patient, with CIS detected at the beginning, showed regression to low grade dysplasia or normal epithelium by index biopsy after 4–6 years [15]. Comparison of progressive and regressive lesions regarding genetic analysis showed differences. Progressive samples had greater somatically acquired genetic damage than regressive samples. Progressive and regressive lesions have distinct transcriptomic and epigenetic profiles. Genomic, epigenetic and transcriptomic aberrations in genes involved in MHC class I antigen presentation are more prevalent in progressive than regressive lesions [16]. Prof. Janes also pointed out that, to understand cancer development, comparisons are required with normal tissues. Prof. Janes showed the differences between children, never-smokers, ex-smokers and current smokers. In the smokers' samples, the number of substitutions occurring with age and the effects of smoking status indicated that smokers had more damage and that also quitting smoking allowed the expansion of cells that do not carry smoking's mutational background [17].

Georgios Stathopoulos (Munich, Germany) talked about club cells in adenocarcinoma and alveolar repair. He started with the role of stem cells in COPD and lung cancer pathogenesis [18, 19]. Stem cells include not only basal cells, AT2 cells and undifferentiated progenitor cells, but also the club cells. Prof. Stathopoulos further discussed the role of replicative stress or environmental oncogenesis on stem cell mutations and abnormal cell proliferation. Regarding the underlying mechanisms, the type of cancer is important; whether it is smoking-induced lung cancer or not, because lung cancers in smokers are clearly separated from lung cancers in nonsmokers based on the molecular and genetic analysis [20]. Based on the molecular hallmarks of smoking, KRAS and TP53 mutations are strongly associated with smoking. However, the distinction they provide is not perfect. In addition, the C>A transversions signature was associated with smoking-caused cancers of the lung [21]. Prof. Stathopoulos described that in their study they triggered lung cancer and COPD models in mice using tobacco chemicals. The tumours developed in mice were highly similar to human smoke-induced lung adenocarcinomas [22]. The talk went on to discuss role of club cells in tobacco chemical-induced lung adenocarcinomas using the examples of their mouse model experimental studies. Club cells were shown to survive KRAS mutations and to form lung tumours after tobacco carcinogen exposure. Increasing numbers of club cells were found in the alveoli with ageing and after lung injury but go undetected since they express alveolar proteins. Ablation of club cells prevents chemical lung tumours and causes alveolar destruction in adult mice [23]. Therefore, club cells are important in alveolar maintenance and carcinogenesis and may be a therapeutic target against pre-malignancy and chronic lung disease.



## Lessons from limb regeneration of the axolotl

Regenerative capacity varies widely across species; some species are highly regenerative where they can recreate a whole body from any piece such as planarians, while others can regenerate an amputated limb such as lizards, and finally some species, such as humans, have a highly limited regenerative capacity. The keynote lecture was given by Nicholas Leigh (Lund, Sweden) on the cells and genes responsible for limb regeneration in the Mexican salamander (*i.e.* the axolotl). Dr. Leigh highlighted how scRNAseq has allowed us to uncover the cellular composition during axolotl limb regeneration [24].

The salamander is a neotenic aquatic species capable of fully regenerating its amputated limb while preserving all the anatomical structures [25]. Similar to a human limb, the salamander contains muscles, nervous system, vasculature, and a bone structure resembling that of a human. Once a limb is amputated, a tissue structure, called the blastema, will form and is responsible for the regeneration of the whole limb. The blastema is rich in a pool of restricted progenitor cells and was found to contain epithelial, mesenchymal and haematopoietic cell populations and a massive influx of immune cells across all stages of regeneration [24].

Several of the vertebrate species remain regeneration competent, such as amphibians, lungfish and Polypterids, while Amniota species are regeneration incompetent. These species share similar anatomical structures and cell types but different regenerative capacities. LEIGH *et al.* [26] screened for potential genes that are only present in the salamander blastema during regeneration and found von Willebrand Factor D and EGF domains (*wvde*) to be actively expressed through limb regeneration of several species. Inhibiting the translation of *wvde* using morpholinos in the salamander resulted in impaired limb regeneration [26]. *wvde* orthologs in higher species (mouse, chick, human) do exist, however with a lower number of EGF domains. It was found that 42.7% of human genomes tend to have a loss-of-function copy of *wvde*, which could indicate a drift towards their inactivation in humans [26, 27]. LEIGH *et al.* [26] speculate that the lack of a blastema in regeneration-incompetent species resulted in loss-of-functions in blastema-specific genes in these species.

There are several theories that attempt to explain why regeneration-incompetent species lack the ability to regenerate such as the development of a more intricate and complex adaptive immune system. However, the reasons behind this lack of regenerative capacity remain unclear. Learning the processes by which regenerative species function may allow for uncovering mechanisms of activating regeneration in injury and disease.

## Cell-matrix interactions in the lung and beyond

The extracellular matrix is the main structural support of the various cell types in the lung and various organs. Changes in extracellular matrix composition have been associated with several chronic disease phenotypes. As an example, COPD is characterised by emphysema which is represented by the destruction of parenchymal regions where gas exchange occurs. By contrast, diseases like IPF are characterised by the excessive deposition of extracellular matrix components, and the subsequent increase in the tissue stiffness. In both of these practically opposite cases, the extracellular matrix plays a pivotal role in these chronic diseases. In this session, Janette Burgess (Groningen, the Netherlands) shed light on the important aspects of extracellular matrix cell interactions in chronic lung disease, followed by Sara Wickström (Helsinki, Finland) whose work explores the role of mechanical cues on cell fate and integrity of the skin.

There are possibly two major ways in which the extracellular matrix can exert its interaction with the cells. First, changes in extracellular matrix composition and amount can lead to change in cellular behaviour. For example, extracellular matrix derived from asthmatic airway smooth muscle cells lead to increased proliferation of newly seeded cells in comparison to extracellular matrix obtained from healthy cells [28]. This indicates that matrix components deposited in the pathologic state contribute to such an effect. Among these factors is a small molecule, fibulin-1, which when knocked down, was shown to impair cellular migration in a wound closure assay [29]. Furthermore, a fibulin-1c knockout mouse was shown to decrease collagen deposition in the bleomycin-induced fibrosis model. This knockout was also associated with lower TGF- $\beta$  activation [30].

Second, changes in matrix organisation, topology and stiffness also influence cell behaviour. More organised collagen fibres are associated with chronic diseases such as IPF. Organisation of collagen fibres is driven by several processes including enzymatic crosslinkers, such as the lysyl oxidase (LOX) family enzymes. LOX not only cross-links collagens but was also found to influence extracellular matrix cues [31]. Inhibition of LOX activity using a small molecule inhibitor attenuated the effect TGF- $\beta$  driven remodelling of collagen I, which led to impaired cell adhesion and proliferation [31, 32]. Moreover, this inhibition of LOX activity has also resulted in a decrease of TGF- $\beta$  induced stiffness. The matrix topology also affects various cell behaviours such as macrophage infiltration.

The extracellular matrix is also able to influence the function of progenitor cells through its composition, structure and mechanical cues. Sara Wickström talked about her work in the regulation of stem cell renewal and differentiation in the skin and how it is altered by mechanical

cues. Lineage tracing has revealed that there is an equilibrium between self-renewal and differentiation in epidermis stem cells during homeostasis and this equilibrium is disrupted during tissue growth in a non-isotropic manner [33]. The stem cells were proliferating following the arrangement of the collagen fibres on which they resided. Experimentally stretching the cells biaxially resulted in a major repressive transcriptomic profile change where the unifying aspect of the changed genes were the regulation of histone modification, specifically H3K27 trimethylation which was found at the promoter regions of lineage genes leading to an attenuation of differentiation [33]. Nuclei softening occurred in response to stretching as a protective mechanism and adaptation; cells where nuclear softening was inhibited were ruptured during stretch. The protective nuclear softening led to the increase in H3K27me3 and repression of lineage-related genes and, in turn, the attenuation of differentiation [34].

### Therapies: organoids, drugs, cell therapy

Carla Kim (Boston, USA) started the final session of the LSC 2021 by discussing how lung organoid culture techniques can be used to understand mechanisms of lung disease. The development of an organoid system revealed epithelial-mesenchymal mechanisms of bronchopulmonary dysplasia (BPD) and mimicked features of the disease. She found mesenchymal cells from hyperoxic BPD mice decreased alveolar organoid formation efficiency, providing evidence that mesenchymal cells may be critical in BPD disease. Prof. Kim also explained how AT2 organoid cultures derived from DsRed mice can be used to interrogate mechanisms of lung progenitor transplantation. Organoid cells engraft, return to a transcriptional state like their native counterparts and proliferate showing evidence that transplanted cells can retain their progenitor activity.

In the penultimate talk of LSC 2021, Pieter Hiemstra (Leiden, the Netherlands) provided an excellent overview of the current state-of-the-art of the mesenchymal stromal cell (MSC) therapy field, with particular insight on MSCs for severe emphysema. MSCs have shown promise *in vitro* and in animal models for many years now; however, there has been limited success in the translation to clinical medicine. Prof. Hiemstra discussed how the lack of clinical efficacy may be associated with several factors such as the origin of the MSCs, dosage and route of administration. Prof. Hiemstra also discussed clinical studies in emphysema where MSCs were infused into patients after the first stage of two-stage lung volume reduction surgery [35]. This unique approach provides a better opportunity to determine the effect of MSC therapy in a clinical setting by directly comparing patient lung tissue before and after MSC infusion.

The final talk of LSC 2021 was given by Filipe Pereira (Lund, Sweden) who discussed cellular reprogramming of cancer cells to antigen-presenting cells. Prof. Pereira initially showed that fibroblasts could be directly reprogrammed into antigen-presenting “induced” dendritic cells using the transcription factors PU.1, IRF8, and BATF3 [36]. Then, it was shown that the same could be performed with primary lung carcinoma cells and lung cancer-associated fibroblasts. This cellular reprogramming harnesses the ability to impose antigen presentation directly in tumour cells. Reprogrammed cancer cells downregulate cell cycle genes thus losing their tumourigenic capacity whilst also secreting inflammatory cytokines and cross-presenting endogenous antigens to T-cells. Prof. Pereira provided insight into the future prospects of directly targeting solid tumours with the combination of transcription factors and, in doing so, merging cellular reprogramming with cancer immunotherapy by turning cancer cells against themselves.

### Early-career delegates session

The early-career delegates session started with a brief introduction by current ERS ECMC Chair Niki Ubags (Epalinges, Switzerland). Dr. Ubags talked about ways of getting involved and taking active roles in ERS ECMC. In addition, Dr. Ubags stated the objectives of ECMC which are supporting ECMs to fully utilise the opportunities provided by ERS, and to support ECMs to flourish within ERS and apply for leadership roles. The next speaker, Darcy Wagner (Lund, Sweden), discussed the use of preprints in publishing science. A preprint is a scientific manuscript that is uploaded by the authors to a public server. Preprints allow scientists to directly control the dissemination of their work to the worldwide scientific community. The history of preprints started in 1961, and bioRxiv launched in 2013, covering life sciences and basic science; medRxiv launched in 2019 covering clinical sciences [37]. Preprints are important to accelerate science communication and facilitate networking. Dr. Wagner also mentioned “dos” and “don’ts” of preprints.

Pieter Hiemstra (Leiden, the Netherlands) talked about how to write a peer review, which is also considered as a tool for scientific gatekeeping. Prof. Hiemstra started describing the steps of the submission and review process. Poor experimental design, unjustified conclusion, poor readability, poor introduction and review of literature, poorly described methods, and confusing presentation of data are considered as reasons for manuscript rejection [38]. Prof. Hiemstra pointed out that there are important rules for reviewers to write their comments to the authors and the editor. The comments to the authors should not include any statements on whether the paper should be accepted or rejected. The talk continued with

the recommendations for feedback, suggestions that should be professional, positive and polite. Prof. Hiemstra pointed out that “every decline is a disappointment”; therefore, careful review and thoughtful consideration is needed.

Martin Kolb (Hamilton, Canada) presented a talk entitled “How to make me like your paper”. Prof. Kolb described the 4A principles of successful scientific writing: Aim, Audience, Awareness, Articulation. Afterwards, Prof. Kolb mentioned the joy of writing a paper and underlined that “Science is writing”. Your customers could be readers from different disciplines, but the key reader is the reviewer. Prof. Kolb pointed out that aligning the well-written manuscript to the scope of the target journal will reduce chances of manuscript rejection. A well-written manuscript is based on a clear understanding of the key message. Originality, importance, robustness of the data are also main points for overall assessment. Another important point is how to respond to reviewers. Writing in a polite way as well as sleeping 3–4 nights before responding to reviewers are recommended steps before covering every point raised by the reviewers.

The last speaker of this session, Susanne Herold (Giessen, Germany) talked about how to write an editorial. Prof. Herold described the features of an ideal editorial: it should be an opinion maker, reconciliatory, and balanced but crusading [39]. Additionally, an editorial should: 1) provide the own intellectual contribution of the authors, rather than just repeating or summarising the findings of the publication it refers to; 2) critically discuss the new findings against the background of current knowledge and evidence; 3) formulate viewpoints based on objective analyses; and 4) provide a refreshing view of the topic. It is important that an editorial is well-balanced in its statements, objective and fair, *e.g.* it should express an opinion without being opinionated. Moreover, an editorial can critically state limitations of the presented publication but should provide suggestions on how to overcome these in the future. In general, an editorial should leave a good aftertaste and stimulate the reader’s interest in the scientific article it refers to. Summary illustrations are a welcome addition to an editorial.

### Young investigator session

The five highest ranked abstracts submitted to the LSC by early-career delegates were presented during the Young Investigator session. Simon Coyle Rowan (Dublin, Ireland) kicked off the session by discussing pulmonary mesenchymal populations present in the healthy and IPF lung. Dr. Rowan showed that pulmonary mesenchymal cells are a conglomeration of molecularly distinct subtypes and that the transcriptomic signatures of each subtype are conserved between species. Importantly, Dr. Rowan showed that all mesenchymal subtypes can contribute to scarring in IPF. Martina M. De

Santis (Lund, Sweden) described the development of a novel bioink for 3D bioprinting lung tissue [40]. This bioink, known as an extracellular-matrix-reinforced (rECM) bioink, can be used to bioprint human small airways. rECM bioinks were shown to be biocompatible and immunocompetent mice and allowed for vascular infiltration from the surrounding tissue. In the next talk, Georgia A. Giotopoulou (Munich, Germany) described a novel candidate lung tumour promoter: the cAMP response element-binding (CREB) protein. CREB is a regulator of key cellular processes such as survival, proliferation, differentiation and inflammation, and is involved in the development of normal respiratory epithelium. Her talk showed that CREB also mediates the immune-evasion of KRAS mutant lung adenocarcinoma by preventing the recruitment of tumouricidal neutrophils. Next, Pien Goldsteen (Groningen, the Netherlands) described the development of a lung on a chip model with the final aim to study neuronal innervation of airway smooth muscle cells in asthma and as a drug screening platform. For this, Goldsteen developed a novel protocol to derive airway cholinergic neurons from human pluripotent stem cells and combined the pluripotent derived airway cholinergic neurons with organ on a chip technology. In the final talk, Ying Wang (Leiden, the Netherlands) proposed the use of differentiated primary human airway epithelial models to study severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection biology and pathogenesis. Interestingly, Wang identified bronchial epithelial cell maturation and differentiation as a contributing factor to airway susceptibility to SARS-CoV-2.

In conclusion, the virtual LSC 2021 was an inspiring meeting, where great science was presented along with a variety of career development opportunities for ERS ECMs. We can only highly recommend this meeting and encourage all ECMs to be part of this conference again next year!

## Highlights from the ERS/European Sleep Research Society Sleep and Breathing 2021

The SBC 2021 took place online on 16–17 April 2021, connecting respiratory medicine professionals on an international level to discuss the latest updates in sleep medicine from a clinical, translational and basic science perspective.

Here, ECMs of ERS Assembly 4 have aimed to summarise some of the newest insights presented during the symposia and oral sessions of the SBC 2021. Each section has been written by ECMs specialised in the different fields of this interdisciplinary assembly. Take-home messages from the conference sessions are provided in table 2.



**Table 2** Take-home messages from the Sleep and Breathing Conference 2021

Session	Type	Take-home messages
<b>Sleep disordered breathing and cardiovascular disease: still an open problem</b>	Symposium	Cardiovascular risk factors need to be better quantified in obstructive sleep apnoea (OSA) patients. Sleep disordered breathing other than OSA and its association with cardiovascular risk should be considered for further studies.
<b>Sleep apnoea and cancer: how strong is the link?</b>	Pro/con debate	Potential OSA-related tumourigenic mechanisms can be listed as intermittent hypoxaemia, sleep fragmentation and circadian cycle disruption. The results of current epidemiological studies have not supported the evidence obtained by translational research. Further research is needed especially on single organ cancer type, the effect of OSA treatment, and OSA-related specific cancer biomarkers.
<b>Circadian disruption in sleep and health</b>	Symposium	Circadian disruption may increase the susceptibility to mental disorders. The daylight-saving time should be carefully revised, given its debatable importance and potential adverse consequences.
<b>Treatment and management of sleep respiratory diseases</b>	Oral presentation	Not only continuous positive airway pressure (CPAP) therapy, but also other therapy options provide benefit on the management of OSA and OSA-related symptoms. Focusing on pharmacological treatment of OSA: pitolisant and solriamfetol seemed to improve OSA-associated excessive daytime sleepiness.
<b>Sleep in the new era of COVID</b>	Symposium	The coronavirus disease 2019 (COVID-19) pandemic has a great impact on sleep quality and sleep disorders across all age groups as well as a reduction of sleep medicine services. Sleep medicine faces new challenges due to the pandemic that can be answered by e-health services and implementation of new technologies.

### Sleep disordered breathing and cardiovascular disease: still an open problem

Obstructive sleep apnoea (OSA) among patients with coronary artery disease (CAD) has a prevalence of 16% [41]. Observational studies showed the beneficial effects of continuous positive airway pressure (CPAP) on cardiovascular outcomes in sleep clinic cohorts. Nevertheless, evidence by means of randomised controlled trials with cardiovascular cohorts is currently lacking [42].

The RICCADSA Study included non-sleepy OSA patients with newly revascularised CAD and an apnoea-hypopnoea index (AHI) >15 events per h, randomised to auto-titrating CPAP or no CPAP. A significant beneficial effect of CPAP on repeated revascularisation, new myocardial infarction, stroke and death attributed to cardiovascular causes was seen only after adjusting for baseline comorbidities and CPAP adherence [43].

In the SAVE trial, participants with coronary or cerebrovascular disease and moderate-to-severe OSA were randomised to CPAP treatment or usual care with no OSA therapy. CPAP therapy showed no significant effect on the prevention of recurrent serious cardiovascular events, though significantly reducing sleepiness among other OSA symptoms and improving quality of life [44].

The ISAACC trial included non-sleepy OSA patients who performed apnoea sleep testing within 3 days after acute coronary syndrome, randomised

to CPAP therapy and usual care or usual care alone. The presence of OSA was not associated with an increased prevalence of cardiovascular events. Interestingly, CPAP treatment, although with an average use of 2.8 h per night, did not significantly reduce this prevalence [45].

Renata Riha (Edinburgh, UK) focused on the link between OSA and stroke. In cerebrovascular patients, severe OSA is a rather common condition [46], with a particularly high stroke risk if left untreated [47]. However, many factors influence CPAP efficacy, *e.g.* where the stroke occurred and the level of residual impairment [48]. Similarly, insomnia slightly increases the cardiovascular risk [47]. The role of other forms of sleep disordered breathing in increasing cardiovascular risk needs to be further investigated.

Carolina Lombardi (Milan, Italy) highlighted the importance of early diagnosis and treatment of OSA in patients with atrial fibrillation (AF). The estimated prevalence of OSA in patients with AF ranges up to 80% [49]. An elevated AHI represents a risk factor for incidental, paroxysmal and recurrent AF [50]. The hypoxic burden is considered a key factor in elevated cardiovascular risk among OSA patients. A cross-sectional analysis of the ESADA [51] cohort showed a link between AHI and arrhythmias prevalence in relation to OSA severity and CHA<sub>2</sub>DS<sub>2</sub>-VASc score. Moreover, central hypopnoeas observed in the AHI computation suggested that these could be a consequence of rather than a cause of AF. It is still unclear if central apnoeas and hypopnoeas

could be a specific target for treatment in patients with AF and OSA [52]. Furthermore, an elevated night-to-night variability of AHI has been observed in this patient population and associated with haemodynamic and ventilatory instability, thus suggesting the need for long-term monitoring and treatment optimisation [53]. In addition, autonomic imbalance was associated with a higher risk of cardiovascular events and arrhythmogenesis in OSA [54]. In conclusion, further research is needed for better phenotyping of OSA patients with cardiovascular diseases to quantify their cardiovascular risk.

### **Sleep apnoea and cancer: how strong is the link?**

All conflicting aspects of the relationship between OSA and cancer were presented as a pro/con debate. Isaac Almendros (Barcelona, Spain) presented the pro side by showing the evidence on the effects of intermittent hypoxaemia and sleep fragmentation on tumourigenesis [55, 56]. First, Prof. Almendros mentioned the results of translational research proving that both intermittent hypoxaemia and sleep fragmentation accelerate tumour growth [56, 57]. In studies in which OSA challenge was applied to animals, the tumour growth, carcinogenesis, invasion and angiogenesis were investigated in mice with different types of tumours, including melanoma, myeloma, lung, breast, colon and kidney cancers. Inflammation, oxidative stress, sympathetic activation and immune deregulation were shown to be potential underlying mechanisms. However, the clinical data regarding melanoma, breast and lung cancer showed no statistically significant result. Despite the limitations and the conflicting results of previous studies, melanoma emerges as the type of tumour most likely to be OSA-related in terms of incidence and aggressiveness. Except for the studies focusing on cutaneous melanoma, the large-scale epidemiological studies including different types of cancers have not shown any result supporting laboratory evidence at the moment. Some potential confounding factors include genetic heterogeneity, multiple mutations, obesity and other comorbidities, OSA severity and age of the patients [57]. An *in vitro* study proved that the effect of intermittent hypoxaemia on tumourigenesis varies according to the magnitude of hypoxia and the cell line [58]. It is also known that ageing and obesity reduce the effect of intermittent hypoxaemia on tumour growth in mice [56, 59]. In conclusion, further research is needed for highlighting the association between OSA and cancer and assessing the role of these confounders.

On the opposite side, Maria Bonsignore (Palermo, Italy) showed how OSA and cancer have some common risk and protective factors. Evidence showing an effect of CPAP on lung cancer is currently lacking. Moreover, the epidemiological studies do not support the link between OSA and cancer in

terms of incidence, metastasis risk and mortality [60, 61]. The uncertain effect of age, the lack of data on the preventive role of OSA treatment and the difficulties in establishing the temporal relationship between OSA and cancer represent currently crucial issues. It has been shown that OSA increases the incidence of melanoma, kidney and pancreas cancer. Conversely, a decrease in the incidence of breast, prostate, colon and rectum cancer was observed [62]. Future prospective studies with large patient cohorts might focus on a single organ cancer type and OSA-related specific cancer biomarkers. Furthermore, as the circadian cycle alters cancer cell dissemination [63], the influence of OSA on circadian cycle disruption might be also addressed in future research. In conclusion, more evidence is needed to conclude that OSA plays a role in human cancer.

### **Circadian disruption in sleep and health**

The symposium presented by Tiina Paunio (Helsinki, Finland) and Erna Sif Arnardóttir (Reykjavik, Iceland) highlighted the importance of circadian rhythms for mental health. The daylight-saving time adopted in several territories around the world was critically appraised and a brief overview of the current state of European countries in this regard was presented.

Alterations in circadian rhythms such as phase delay, decreased amplitude and high instability are often experienced by individuals with psychiatric disorders [64–68]. However, the association between circadian rhythms and mental health is not completely understood and further studies investigating the causality between these factors are needed. Tiina Paunio presented aetiological models possibly explaining this relationship [69]. On one hand, circadian disruption may contribute to an increased risk of mental disorders. A second model suggests that mental disorders and their treatments lead to alterations in circadian rhythms, while the third model considers both possibilities, suggesting a bi-directional relationship between these factors.

Several circadian-related characteristics and behaviours may account for different susceptibilities to mental disorders. Accordingly, the individual timing preference (chronotype) is associated with a higher rate of various mental disorders including seasonal and bipolar depression [70–72]. In addition, inappropriate exposure to light due to social schedules, such as shift work and jetlag, demonstrably affects the circadian rhythms and consequently increases the susceptibility to mental disorders [73–75]. Considering this, interventions controlling the light exposure are interesting options to prevent alterations in circadian rhythms and their undesirable consequences.

Under this scenario, the daylight-saving time adopted in several countries around the world needs to be critically evaluated for its relevance and associated outcomes. Erna Sif Arnardóttir presented

an overview of the scientific community's attempts in promoting awareness and changes in relation to this aspect. Considering the debatable rationale underlying the use of daylight-saving time and potential adverse outcomes associated with it [76, 77], on 12 September 2018, the European Commission presented a proposal to dispense with the biannual clock changes. This was later supported by the European Parliament on 26 March 2019, letting each member state decide whether they would keep the daylight-saving time or the standard time [78]. However, this was not effectively implemented up to the present date, arguably due to the coronavirus disease 2019 (COVID-19) pandemic.

The European Sleep Research Society, in association with the European Biological Rhythms Society and the Society for Research on Biological Rhythms, state that the current scientific knowledge supports the use of standard time over daylight-saving time during summer or year-round for better sleep and general health [79, 80]. This position is seconded by the American Academy of Sleep Medicine, which highlights the acute effects of daylight-saving time such as the increased risk of cardiovascular events, traffic accidents, and mood disorders [81].

In summary, the symposium addressed the importance of circadian rhythms for mental health, demonstrating factors with the potential to disrupt these rhythms and ultimately increase the susceptibility to mental disorders. Considering this, the maintenance of the daylight-saving time should be carefully revised, given its debatable importance and potential adverse consequences.

## Treatment and management of sleep respiratory diseases

This oral communication session provided new insights on OSA treatment options.

Martina Meszaros (Budapest, Hungary) presented the work carried out by her team on the assessment of night-to-night variability of the oxygen desaturation index based on two consecutive nocturnal pulse oximetry measurements. They found that night-to-night variability was high in OSA, and more than 30% of patients changed severity class within two nights. The mean change in the oxygen desaturation index between two nights was almost 7 per h and increased with OSA severity.

Kallirroi Lamprou (Athens, Greece) presented a study conducted by her team on the discrepancy between patient- and partner-reported daytime sleepiness measured with the Epworth Sleepiness Scale (ESS). This study did not find significant differences in ESS scores at baseline or after 3 months on CPAP.

Izolda Bouloukaki (Heraklion, Greece) investigated the effect of positive airway pressure

(PAP) on mortality and hospitalisation caused by COPD exacerbations. Improved survival and hospitalisation rates were associated with  $\geq 6$  h of PAP. Moreover, ESS values and Beck Depression Scale values were also improved by PAP, suggesting that it also has an impact on several aspects of health-related quality of life.

Focusing on pharmacological treatment of OSA, Yves Dauvilliers (Montpellier, France) showed the findings of a study on the effects of pitolisant on excessive daytime sleepiness [82]. Pitolisant reduced the ESS score and improved the sleep latency test and Pichot fatigue score. Frederick Vickenbosch (Maastricht, the Netherlands) showed a positive effect of solriamfetol on driving performance. The results suggested both drugs could be potential options for treating excessive daytime sleepiness in OSA.

Laetitia S. Gaspar (Coimbra, Portugal) provided information about the impact of short- and long-term CPAP on biological clock disruption in OSA. Long-term CPAP therapy seemed to improve OSA-associated clock disruption as shown by melatonin levels, cortisol levels and body temperature.

Francisco V. Machado (Porto, Portugal) showed that 82% of the patients were diagnosed with OSA in the primary care. The unexpectedly high number of patients with OSA suggested that sleep laboratories should have more focus on large screening programmes and standardised referral protocols in collaboration with primary care. However, CPAP is the only universally funded OSA treatment option. Nevertheless, Sean Treanor (Manchester, UK) concluded that up to 56.8% of patients on CPAP are uncompliant with treatment. Mandibular advancement devices (MAD) were presented as a cost-effective treatment option for these patients.

Lishun Yue (Beijing, China) and colleagues investigated different expiratory positive airway pressure (EPAP) levels of noninvasive ventilation (NIV) in elderly patients with OSA. There were significant differences in leakage between fixed-scale EPAP and wide-scale EPAP. Intra-abdominal hypertension had a lower prevalence in the wide-scale EPAP group. Moreover, the occurrence of patient-ventilator desynchronisation was higher in the fixed EPAP group. Varying EPAP levels seemed to be more comfortable for older patients with OSA.

Abdelkebir Sabil (Paris, France) and his team investigated the prevalence of predominant positional OSA (p-POSA) and exclusive positional OSA (e-POSA) and the outcomes of CPAP in these patients. p-POSA and e-POSA were highly prevalent (53.5% and 20.1%, respectively). Patients with p-POSA and e-POSA showed a lower CPAP compliance after 6 months compared with patients without positional OSA. These data suggest that CPAP may not be the optimal choice in positional OSA.

In summary, therapy options other than CPAP were shown to provide benefit in OSA and its symptoms.

## Sleep in the new era of COVID

The symposium focused on the role of COVID-19 in OSA.

Winfried Randerath (Solingen, Germany) presented the relationship between obesity, sleep apnoea and COVID-19. Obesity, a frequent medical condition, is a risk factor for severe course of the disease in patients with COVID-19, especially the younger ones, including admission in the intensive care unit and potential need for invasive mechanical ventilation [83, 84]. High inflammatory status, disturbed metabolism and impaired cardiac and lung function are considered factors that may increase the severity of COVID-19 in these patients [85, 86]. Patients with COVID-19 and OSA seemed to have higher mortality rates; however, more research is needed to identify possible mechanisms linking the severity of COVID-19 with OSA, such as the involvement of the renin-angiotensin-aldosterone system, the cardiovascular consequences in OSA and cardiac complications in COVID-19 and the accentuation of COVID-19 coagulopathy due to OSA [87].

The COVID-19 pandemic, lockdown and home confinement resulted in a great impact on sleep, as discussed by Sophia E. Schiza (Heraklion, Greece). Healthy individuals, as well as healthcare workers, reported impairments in the sleep quality, nightmares and features of insomnia compared with the pre-pandemic period [88–90]. Anxiety, depressive symptoms and stress may have led to sleep disturbances and exposure to screens near bedtime. Female sex, younger age and financial difficulties due to the pandemic were factors associated with worse sleep quality and increased use of sleeping medications [88, 91]. Additionally, patients with COVID-19 reported sleep disturbances, fatigue and muscle weakness after hospitalisation, potentially representing features of post COVID-19 syndrome [92]. Managing sleep disturbances during lockdown and home confinement is of great importance as they may have long-lasting effects on the overall health status. More research is needed for assessing possible mechanisms and treatment of sleep disturbances after COVID-19.

Refika Hamutcu Ersu (Ottawa, Canada) showed that the COVID-19 pandemic caused sleep disturbances in young children, although the total duration of sleep was stable over 24 h and nocturnal sleep duration was increased due to online school [93, 94]. Children had difficulties in initiating and maintaining sleep and showed an increased frequency of parasomnias. Furthermore, lockdown led to changes in children's lifestyle, *e.g.* decreasing sport activities [95]. The closure of sleep laboratories and the delay in ear-nose-throat

surgical procedures during the COVID-19 pandemic changed routine paediatric sleep practice [96]. Alternatives like telemedicine, home sleep studies and nonsurgical treatment options may be considered for management [97]. Moreover, there are limited data suggesting that OSA in children may be a risk factor for developing severe COVID-19 as it is in the adult population.

Ludger Grote (Göteborg, Sweden) presented the changes in sleep medicine and practice due to COVID-19. During the pandemic, there was a substantial reduction in both diagnostic procedures and initiation of PAP treatment of sleep disordered breathing [16, 17]. Moreover, the staff in sleep laboratories was reduced up to 25% due to their re-allocation to COVID-19 units [98]. Although there are guidelines from scientific societies on mitigation strategies and reopening of sleep laboratories, until now sleep medicine services are not fully recovered. E-health services like telemedicine may facilitate sleep disordered breathing management during the COVID-19 pandemic with home sleep studies and remote monitoring [97]. Prioritisation of patients in large waiting lists after the COVID-19 pandemic with the use of e-health services is a point to be assessed by future research.

## Meet the new members of the ECMC

The ECMC is composed of 14 members, one ECM representative per assembly of the ERS. The ECMC members work together to ensure that ECMs are well-represented in ERS committees, councils and assemblies, to provide them with opportunities for professional development and education, and to encourage them to flourish within ERS and to apply for leadership roles. An overview of the activities carried out by the ECMC is described elsewhere [99].

Every 3 years, elections are held for the position of ECM representative for each assembly. This year, five new elected ECM representatives joined the ECMC. Below we introduce each of the new ECM representatives of Assemblies 1, 4, 10, 12 and 13.

### Assembly 1: Thomas Gille

Thomas Gille is the new ECM representative of Assembly 1 (Respiratory clinical care and physiology). He is a former respiratory physician, presently Associate Professor of Physiology in Université Sorbonne Paris Nord/Hôpitaux Universitaires de Paris Seine-St-Denis and a member of Inserm laboratory "Hypoxia and the Lung" (Director: Prof. C. Planès), Bobigny, France. He is currently involved in several official societies of respiratory medicine and physiology. His translational research is mainly about interstitial



lung diseases (especially pathogenesis, functional explorations at rest and exercise, associated sleep disorders, dyspnoea, post COVID-19 sequelae) and alveolar epithelial cells (biological and functional properties in normal and pathological conditions, particularly sustained or intermittent hypoxia). He also has a special interest in spirometry in primary care and simulation-based medical education.

#### Assembly 4: Matteo Bradicich

Matteo Bradicich is the new ECM representative of ERS Assembly 4 (Sleep disordered breathing). He is currently working as a resident physician at Felix Platter University Center for Geriatric Medicine in Basel (Switzerland), in the framework of the specialisation programme in internal medicine and pulmonology. He graduated at the University of Pisa (Italy) in 2017 with a joint final dissertation at the University of Pisa (Italy) and University of Oxford (UK), under the supervision of Prof. Pierluigi Paggiaro and Prof. Ian Pavord. He worked as research clinician in the Respiratory Pathophysiology ward of Cisanello University Hospital of Pisa (Italy), mainly working with patients with asthma, COPD and eosinophilic granulomatosis with polyangiitis. Moreover, he worked during this period as data manager for the Severe Asthma Network Italy (SANI). Since the end of 2018, he is affiliated to the Department of Pulmonology and Sleep Disorders Centre of the University Hospital Zurich (Switzerland) and focuses on OSA. In 2021, he was awarded the Dr. med. title at the University of Zurich (Switzerland) with a doctoral thesis on heart rate variability in OSA. His main research topics in the field of sleep medicine are OSA-related cardiovascular pathophysiology, domotics applied to sleep disordered breathing and OSA diagnostics.

#### Assembly 10: Holly R. Keir

Holly Keir is the new ECM representative of Assembly 10 (Respiratory infections). She is a postdoctoral researcher working at the University of Dundee Ninewells Hospital, UK. She completed her undergraduate degree in Biological Sciences at the University of Dundee (UK) in 2016 before joining the Chalmers laboratory. Her research interests are in the role of neutrophilic function and the lung microbiome in patients with bronchiectasis and COPD. She is in the final stages of completing her PhD investigating the role of neutrophil extracellular trap formation in chronic lung disease.

#### Assembly 12: Catharina C. Moor

Catharina Moor is the new ECM representative of Assembly 12 (Interstitial lung disease). She is a pulmonary resident and postdoctoral researcher at

the Erasmus University Medical Center (Rotterdam, the Netherlands). In 2020, she obtained her PhD, which focused on innovative approaches to care and research in interstitial lung disease, such as eHealth solutions, home spirometry, and electronic nose technology. As postdoctoral researcher, she is involved in many research projects dedicated to new innovations, facilitating personalised and comprehensive care for patients with interstitial lung disease.

#### Assembly 13: Mona Lichtblau

Mona Lichtblau is the new ECM representative of Assembly 13 (Pulmonary hypertension). She is a pulmonary resident and postdoctoral researcher at the University Hospital of Zurich, Switzerland. Albeit being interested in the whole spectrum of pneumology, her clinical and research focus is the pulmonary vasculature in healthy people and in patients with lung diseases, at low and high altitude. Furthermore, she is interested in several treatment options for patients with pulmonary hypertension such as rehabilitation, exercise and oxygen therapy.

### Final remarks

This article has provided an overview of the LSC and SBC highlights. For those who could not attend the conferences or who want to re-watch the sessions, learning resources are available: 1) recordings of the LSC sessions are available to ERS members on the e-learning website ([www.ers-education.org/events/conferences/?idP=217272](http://www.ers-education.org/events/conferences/?idP=217272)); 2) the SBC replay is available upon registration (<https://live.sleepandbreathing.org/user/login>). In addition, the abstracts from the LSC and SBC 2021 are already available online as a supplement in *ERJ Open Research* ([https://openres.ersjournals.com/content/7/suppl\\_6](https://openres.ersjournals.com/content/7/suppl_6) and [https://openres.ersjournals.com/content/7/suppl\\_7](https://openres.ersjournals.com/content/7/suppl_7), respectively).

We also provided a brief introduction to the new members of the ECMC. Please feel free to contact the ECM representative of your assembly if you have any questions and ideas and/or if you want to be more involved in ERS activities. Their contact details can be found in the ERS members' directory. You can also sign up to the competence list *via* myERS (<https://my.ersnet.org/>) and indicate your areas of expertise and interests. You can also follow the ECMC social network pages:

- Facebook: [www.facebook.com/groups/585753791780360](http://www.facebook.com/groups/585753791780360)
- Twitter: <https://twitter.com/EarlyCareerERS>
- ResearchGate: [www.researchgate.net/project/European-Respiratory-Society-Early-Career-Members](http://www.researchgate.net/project/European-Respiratory-Society-Early-Career-Members)



## Affiliations

**Amanda T. Goodwin**<sup>1,20</sup>, **Dilek Karadoğan**<sup>2,20</sup>, **Martina M. De Santis**<sup>3,20</sup>, **Hani N. Alsafadi**<sup>4,20</sup>, **Ian Hawthorne**<sup>5,6,20</sup>, **Matteo Bradicich**<sup>7,20</sup>, **Matteo Siciliano**<sup>8,20</sup>, **Sezgi Şahin Duyar**<sup>9,20</sup>, **Adriano Targa**<sup>10,11,20</sup>, **Martina Meszaros**<sup>7,20</sup>, **Michail Fanaridis**<sup>12,20</sup>, **Thomas Gille**<sup>13,14</sup>, **Holly R. Keir**<sup>15</sup>, **Catharina C. Moor**<sup>16</sup>, **Mona Lichtblau**<sup>17</sup>, **Niki D. Ubags**<sup>18</sup>, **Joana Cruz**<sup>19</sup>

<sup>1</sup>Nottingham NIHR Biomedical Research Centre, University of Nottingham, Nottingham, UK. <sup>2</sup>Dept of Chest Diseases, School of Medicine, Recep Tayyip Erdoğan University, Rize, Turkey. <sup>3</sup>Institute of Molecular Biotechnology of the Austrian Academy of Sciences (IMBA), Vienna BioCenter (VBC), Vienna, Austria. <sup>4</sup>Lung Bioengineering and Regeneration, Dept of Experimental Medical Sciences, Stem Cell Centre, Wallenberg Center for Molecular Medicine, Lund University, Lund, Sweden. <sup>5</sup>Cellular Immunology Laboratory, Dept of Biology, Maynooth University, Maynooth, Ireland. <sup>6</sup>Kathleen Lonsdale Institute for Human Health Research, Maynooth University, Maynooth, Ireland. <sup>7</sup>Dept of Pulmonology and Sleep Disorders Centre, University Hospital Zurich, Zurich, Switzerland. <sup>8</sup>IRCCS Fondazione Policlinico Universitario A Gemelli – Università Cattolica del Sacro Cuore, UOC Pneumologia, Rome, Italy. <sup>9</sup>Pulmonology, University of Health Sciences Atatürk Chest Diseases and Thoracic Surgery Education and Research Hospital, Ankara, Turkey. <sup>10</sup>Translational Research in Respiratory Medicine, Hospital Universitari Arnau de Vilanova-Santa Maria, IRBLleida, Lleida, Spain. <sup>11</sup>Diseases Network Research Centre on Respiratory Diseases (CIBERES), Madrid, Spain. <sup>12</sup>Sleep Disorders Unit, Dept of Respiratory Medicine, Medical School, University of Crete, Heraklion, Greece. <sup>13</sup>Inserm UMR 1272 “Hypoxia & the Lung”, UFR SMBH Léonard de Vinci, Université Sorbonne Paris Nord (USPN), Bobigny, France. <sup>14</sup>Physiologie et Explorations Fonctionnelles, Hôpitaux Universitaires de Seine-Saint-Denis (HUPSSD) Avicenne/Jean Verdier/René Muret, Assistance Publique – Hôpitaux de Paris (AP-HP), Bobigny, France. <sup>15</sup>Scottish Centre for Respiratory Research, University of Dundee, Ninewells Hospital, Dundee, UK. <sup>16</sup>Dept of Respiratory Medicine, Erasmus University Medical Center, Rotterdam, The Netherlands. <sup>17</sup>University Hospital Zurich, Zurich, Switzerland. <sup>18</sup>Faculty of Biology and Medicine, University of Lausanne, Service de Pneumologie, CHUV, Epalinges, Switzerland. <sup>19</sup>Center for Innovative Care and Health Technology (ciTechCare), School of Health Sciences (ESSLei), Polytechnic of Leiria, Leiria, Portugal. <sup>20</sup>These authors contributed equally.

## Conflict of interest

A.T. Goodwin has nothing to disclose. D. Karadoğan has nothing to disclose. M.M. De Santis has nothing to disclose. H.N. Alsafadi has nothing to disclose. I. Hawthorne has nothing to disclose. M. Bradicich has nothing to disclose. M. Siciliano has nothing to disclose. S. Şahin Duyar has nothing to disclose. A. Targa has nothing to disclose. M. Meszaros has nothing to disclose. M. Fanaridis has nothing to disclose. T. Gille reports personal fees from Boehringer Ingelheim, personal fees from Roche, other from Oxyvie (oxygen provider), other from LVL Medical (oxygen provider), other from Vitalaire (oxygen provider), outside the submitted work. H.R. Keir has nothing to disclose. C.C. Moor has nothing to disclose. M. Lichtblau has nothing to disclose. N.D. Ubags has nothing to disclose. J. Cruz has nothing to disclose.

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