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From the authors:

We appreciate the comments of H.E. Collard and T.E. King on our article [1]. The American Thoracic Society (ATS)/European Respiratory Society (ERS) consensus classification has been important in advancing clinical understanding of the idiopathic interstitial pneumonias [2]. In that document, the clinical entity of nonspecific interstitial pneumonia (NSIP) was proposed as a provisional term only. As we have argued, we believe that using histological appearances alone to define separate clinical entities is unhelpful [1]. Different histological patterns may occur in the same patient [3]. Furthermore, conditions with a defined aetiology (*e.g.* hypersensitivity pneumonitis, connective tissue disease or familial pulmonary fibrosis) may give rise to either usual interstitial pneumonia (UIP) or NSIP in different patients [4–6].

By associating the histological lesion of NSIP with a clinical diagnosis of NSIP, the ATS/ERS consensus classification has blurred the distinction between idiopathic and secondary NSIP in the minds of many clinicians. This has led some to consider NSIP as a single disorder. We agree strongly with H.E. Collard and T.E. King that many cases of NSIP are due to either connective tissue disease or hypersensitivity pneumonitis. However, when reviewing the clinical data in such cases there are often ancillary features that point to the underlying diagnosis [7]. Once secondary cases are excluded, there remains a large subgroup of NSIP patients who have a clinical phenotype that overlaps substantially with that of UIP/idiopathic pulmonary fibrosis (IPF). This group of patients have a sex distribution, smoking-exposure history, mode of clinical presentation, distribution of clinical signs and bronchoalveolar lavage cell differential that mirrors that of IPF/UIP [2, 8, 9]. The distribution of disease on high-resolution computed tomography is also strikingly similar [10].

We therefore propose that idiopathic UIP and idiopathic NSIP, sharing a common clinical phenotype, form a spectrum of disease with a common pathogenesis. The pathogenetic mechanisms involved in the development and progression of IPF are complex and are likely to involve abnormalities in a number of the multiple pathways of normal wound healing [1]. It seems likely that the balance of abnormalities in each of the key wound-healing pathways may vary between individuals. This variation is likely to be responsible for the range of clinical, radiological and pathological phenotypes observed in IPF.

Like H.E. Collard and T.E. King, we hope that future clinical and scientific research will further clarify these issues, as advances in our understanding of idiopathic pulmonary fibrosis can only be to the benefit of patients with this devastating and currently untreatable disease.

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STATEMENT OF INTEREST

None declared.

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Aqua jogging-induced pulmonary oedema

To the Editors:

The case study by WENGER and RUSSI [1], of pulmonary oedema occuring during aqua jogging, is interesting. Aqua jogging certainly lowers the burden to joints and tendons as compared with land running. Conversely, running or cycling at sustained intensity may lead to higher burden to lung tissue in water than on ground. In water, the exercising mechanical strain [2] is strengthened by congestion of pulmonary vessels (hence, decreased lung compliance) and also by the inspiratory loading due to hydrostatic pressure, which is likely to enlarge intra-airway pressure swings [3, 4]. In addition, the work of breathing increases progressively during endurance exercise at constant work [5]. Therefore, it seems difficult to believe that stress failure of alveolar or bronchial capillaries is improbable in the case reported by WENGER and RUSSI [1]. In an experiment designed to compare immersed *versus* ground 30-min cycling, thoracic electrical impedance was lower during recovery on land after exercising in water than on ground, which reflected a larger amount of thoracic fluid, while stroke volume was simultaneously lower, *i.e.* some degree of suboedema may have been present [4, 6]. In addition, 20°C water carries a cold stress, even to an exercising subject [4, 7, 8], and even mild cooling increases peripheral vascular resistance, left ventricular afterload and pulmonary congestion [7]. Finally, symptoms related to pulmonary oedema occur earlier during sustained exercising in water than on land. In the case reported by WENGER and RUSSI [1] symptoms occured after 20 min, which matches other reports (see references quoted by WENGER and RUSSI [1], and also recently gathered data [9]). All in all, the occurrence of pulmonary oedema during various conditions of immersed exercising is not rare, which encourages efforts for a better understanding of the underlying pathophysiological mechanisms. Cardiovascular strains linked to sustained exercise during immersion should not be overlooked. Detailed recording of each case's circumstances of occurrence should aid recognition of recurrent features and tracking possible underlying pathways [9].

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STATEMENT OF INTEREST

None declared.

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