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COMMENTARY

What a long, strange trip it's been

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In 1996, I was recruited by George Martin to FibroGen to work on the role of "CTGF" in fibrosis. At the time, relatively little was known about either topic. Or, perhaps more accurately, the widely-held assumptions about both "CTGF" and fibrosis were overly simplistic, and could be summarized as follows: TGFbeta1 causes fibrosis through "CTGF". At that time, the concept of an autocrine, pro-adhesive signaling loop being necessary for fibrosis, the involvement of progenitor-derived myofibroblasts, and the central role of CCN2 in promoting the differentiation of progenitor-like cells into myofibroblasts was completely out of the mainstream. This concept of how fibrosis is initiated and perpetuated, and also contributes to cancer progression, is, I think, now established as is, I believe, the role of CCN2 in myofibroblast differentiation in fibrotic stroma (Hutchenreuther and Leask 2018; Hinz et al. 2019).

Since its inception in 2001, the International CCN Society (ICCNS) has been instrumental in providing a forum for evidence-based scientific communication, specifically the ICCNS-sponsored biannual workshops, which ultimately led to a transformation in how we think about CCN molecules. Finally, we have achieved worldwide acceptance of our concepts as seen by the official renaming, by HUGO, of the historical, non-heuristically-useful names Cyr61, CTGF, Nov and WISP1–3 as the conceptually satisfying CCN1–6 (Perbal et al. 2018). I should mention that our efforts had previously been recognized by organizers of American Society of Matrix Biology and the FASEB Matricellular Protein meetings. However, it is still extremely frustrating to attend larger conferences, including Keystone and Experimental Biology meetings, and be constantly told that "CTGF" is a growth factor, and acts downstream of TGFbeta as opposed to mechanotransduction.

In addition to the biannual CCN workshops, the ICCNS has sponsored, since 2007, the Journal of Cell Communication and Signaling (JCCS) and, prior to that, its predecessor journal, Cell Communication and Signaling, which still exists, albeit in a different form to JCCS. JCCS is now an established journal, with a 19% acceptance rate (2019), and an impact factor of 3.691. From its initial focus on CCN proteins, JCCS is now attracting a wide variety of excellent submissions from all over the world. Unfortunately, we have noticed, over the past year, an increase (~1 a week) of papers that are essentially identical in format, although the specific topic changes from paper to paper. From an editorial perspective, they all have the same methods used: there is a CKK8 proliferation assay, use of real-time polymerase chain reaction to measure RNA levels, and of miRNA mimics, siRNAs and reporter assays. The methods are written exactly in the same way, and lack the same experimental details, making it virtually impossible to repeat the work. In some cases, entire data sets are used multiple times in the paper, even though they are purported to represent different experimental conditions. These types of manuscripts have no place in JCCS; the aim of scientific publishing is to disseminate crucial experimental details that allow others to reproduce, validate and build on published data.

These issues have contributed to a rising rejection rate in JCCS, reaching 74% for 2019 (compared to 32%, 42% and 60% in 2016, 2017 and 2018, respectively).

This society was founded because there was a need to focus on evidence-based science in the CCN field so that the field could progress, and be founded on solid, reproducible evidence.

I can assure the reader that the same principle also instructs the editorial processes of JCCS.

"..and so it goes."—Kurt Vonnegut.

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