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RESPONSE

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We thank Dr Velu and coauthors for their supportive comments. We acknowledge the preeminence of this group in understanding the predictive and prognostic impact of an inflammatory milieu in determining cancer outcomes in many settings. The work of Jamieson et al. (1) in operable patients was novel, and we are pleased to extend their findings to the metastatic setting.

We agree that the gold standard for such work involves the use of C-reactive protein and albumin. This unfortunately is not yet a routine test in clinical trials or indeed cancer therapy in clinical practice. Hence the neutrophil-lymphocyte ratio (NLR) and derived NLR, which have been shown to be a reasonable alternative measure (2–3), were chosen by us, as they were accessible in the largest number of patients. We are indeed pleased that their group has shown this to be an acceptable surrogate measure of an activated inflammatory milieu (2).

We also are intrigued by the prospect that this finding, regarding prognosis and the ability to identify a group with particularly poor outcomes, might also identify a group appropriate for novel interventions. This includes the phase II ruxolitinib data quoted, which is now the subject of a randomized phase III study. However, it may also uncover the potential for alternative targeted agents, including anti-angiogenesis agents, which could be revisited for this subgroup (4).

We agree that the recent understanding of the role of the host in determining outcomes in pancreatic cancer is highly significant, not only the inflammatory response but also those of critical stromal cells (5–7).

We look forward to novel treatment directions being tested, with both tumor and host factors influencing the choice of how to personalize therapy and improve results in this difficult-to-treat disease.

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