Prolonged, aggressive stimulation with cytokines may also lead to the increased numbers of granulocytes as well as of activated lymphoid cells (Jung et al., 2001). In addition, normal granulocytes that are likely to be recruited to peritoneal cavity (Hau, 1990) have been shown to express CK 20 (Jung et al., 2007). Furthermore, peritoneal lavage samples of cancer-free healthy volunteers and of patients with chronic inflammatory diseases (Jung et al., 1998; Goeminne et al., 2006). CEA and CK 20 transcripts are markers of limited value for the detection of cancer cells for several reasons. Both markers have been shown to be expressed by the haematopoietic cells of healthy volunteers and of patients with chronic inflammatory diseases (Jung et al., 1998, 1999; Champelovier et al., 1999, Vlems et al., 2002). The qRT-PCR-based techniques of CEA and CK 20 detection present the same limitations as conventional RT-PCR, as researchers were unable to set the cut-off values to distinguish between cancer and haematopoietic cell expression (Bustin et al., 2004; Schuster et al., 2004). Especially in the experimental setting studied by Katsuragi et al. (2007), one should consider that CEA expression may be induced in peripheral blood mononuclear cells by cytokines (Jung et al., 1998; Goeminne et al., 1999), as increased concentrations of an array of cytokines characterise peritoneal fluids of cancer patients (Chechlinska et al., 2007). Furthermore, peritoneal lavage samples of cancer-free patients were found to contain CEA transcripts (Broll et al., 2001). In addition, normal granulocytes that are likely to be recruited to peritoneal cavity (Hau, 1990) have been shown to express CK 20 (Jung et al., 1999; Kruger et al., 2001). In fact, the increased numbers of granulocytes as well as of activated lymphocytes were demonstrated in the peritoneal fluids of gastric and colon cancer patients as compared with those of cancer-free controls (Olszewski et al., 2007).

Unfortunately, none of these limitations, although widely discussed in numerous papers, have been considered or even discussed by Katsuragi et al. (2007). In effect, the observed correlation between the positive CEA and CK 20 qRT-PCR signals and cancer recurrence seems reasonably well documented, whereas the statement that the authors detect ‘free cancer cells’ in their study, expressed already in the title and then subsequently used through the methods section to discussion, is totally unwarranted.

Considering the above, the measurements of the markers described by Katsuragi et al. (2007) may reflect the presence of inflammatory cells rather than micrometastatic cells; the more so because as many as 35% of cytology-negative/PCR-positive patients developed no peritoneal metastases. Nevertheless, prognosis for PCR-positive patients was shown to be significantly worse than for PCR-negative patients, and this was in accordance with the recent data of Crumley et al. (2006) and Deans et al. (2007), who linked inflammation symptoms with adverse prognosis in patients with gastric cancer. However, there are simpler and cheaper methods than qRT-PCR to assess inflammatory parameters. The study of Katsuragi et al. (2007) is also methodologically flawed. The GAPDH gene was used as an internal control and a reference gene. The GAPDH gene is known for the presence of its numerous pseudogenes, which makes it inadequate as a reference gene in non-DNased total RNA samples, such as those prepared by Katsuragi et al. (2007). In addition, the reader learns nothing about the standardisation of RNA quantities subjected to reverse transcription, necessary to make a reliable comparison of samples containing different cell counts.

Finally, Katsuragi et al. (2007) use the inexplicable and inappropriate term ‘tumour nucleotides’, and for reasons that are far from obvious the disseminated cancer cells are called ‘isolated’ cells, although no isolation or enrichment procedure was performed. The authors have not applied the idea of micrometastases detection in cancer cell-enriched populations, one of many detection methods focused to enhance specificity and the only one which produced an assay that received FDA clearance (Smerage and Hayes, 2006).
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