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The Reply

We would like to thank Singh et al[1] for their interest in our review of "Black Pleural Effusion," which proposed a classification based on the pathophysiological mechanism. They stated that active crack cocaine use might be a cause of a black pleural effusion via dense deposition of carbonaceous material in macrophage cytoplasm. This may certainly be the case. However, not all patients with crack cocaine use have a black pleural fluid, and the cytoplasmic pigmentation caused by the carbonaceous materials seemed to vary in individual cases (cases 1 and 2), as did the cytoplasmic melanin within the tumor cells in patients with malignant melanoma.[2] Furthermore, many erythrocytes were noted in the crack smokers' pleural fluid sediment smears in their report,[1] which suggests that complicated thoracic hemorrhage or hemolysis might have a potential role in generating black pleural effusions. Examination of a greater number of such cases would be required to develop the precise classification for black pleural effusion.

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