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mediated cytolysis. A combination of cytotoxic assays was used to study the interactions between PMN, platelets, lymphocytes, and tumor cells. Tumor cells were incubated with autologous lymphocytes in the presence of either autologous PMN or autologous platelets. Lymphocytes were recovered 3 days later. PMN mediated tumor cell lysis was assessed with a 51Cr release assay. Platelet-mediated cytotoxicity was assessed with a 51Cr release assay and a 72 hr platelet survival assay. Lymphocyte-mediated cytotoxicity was assessed with an 18 hr cytotoxicity assay using a chromium release assay. The ability of PMN to kill tumor cells was examined at early and late time intervals. The effect of PMN on platelet activity was examined after PMN removal. There was a correlation between PMN and lymphocyte-mediated cytotoxicity which was synergistic in the presence of indomethacin. There was a correlation between the degree of lymphocyte-mediated cytotoxicity and the platelet survival assay but a poor correlation between lymphocyte-mediated cytotoxicity and platelet-mediated cytotoxicity. PMN in an indomethacin-treated state were capable of phagocytosing platelets. However, the ability of PMN to phagocytose platelets was not enhanced by T cell-mediated cytotoxicity. The ability of PMN to interact with platelets is enhanced in the presence of indomethacin. However, once the indomethacin-induced cytotoxic state has been established, the interaction between PMN and platelets d0c515b9f4

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