intracytoplasmic fat droplets and glycogen granules. Compared with the extravascular macrophages, the macrophages within the vitelline vessels have more numerous slender cytoplasmic processes. The intravascular macrophages are similar to presumptive Kupffer cells in embryonic hepatic sinusoids. The results indicate that the primitive macrophages in human yolk sacs participate in the avid phagocytosis and deggradation of primitive erythroblasts.

THE INDUCTION OF CYTOSTATIC MACROPHAGES AND ANTI-TUMOR EFFECTS BY INFLAMMATORY NEUTROPHILS. A. LICHTENSTEIN, J. KAHLE, M. ALI, J. BEREK, J. ZIGHELBOIM. V.A. Wadsworth-UCLA Medical Center, Los Angeles, Ca., 90073.

We studied the role of inflammatory neutrophils (PMNs) in the anti-tumor effects that follow intraperitoneal (IP) injection of Corynebacterium parvum (CP) into mice challenged with the murine ovarian teratocarcinoma (MOT). A Winn assay demonstrated that peritoneal cells (PCs) obtained 6 hrs after IP injection of CP were the most effective in preventing tumor growth. Percoll gradient centrifugation confirmed that the critical PCs exerting anti-tumor effects in the Winn assay are inflammatory PMNs. Non-viable CP-ellicited PMNs but not thioglycollate (thio)-ellicited PMNs were effective in the Winn assay. Whole body irradiation (WBI) or injections of silica of recipient mice abrogated the anti-tumor effects of 6hr-PMNs indicating these cells activated a second effector mechanism in recipient mice. Since WBI and silica prevent the activation of peritoneal macrophages (mØs), we investigated whether inflammatory PMNs activate anti-tumor mØs. MOT-cytostatic mØs were generated within the peritoneal cavity of mice following IP injection of PMNs obtained 6 hrs after CP treatment. In contrast, thio-ellicited PMNs did not activate mØs. The amount of CP present in the PMN inoculum could not account for the anti-tumor effect or generation of cytostatic mØs. The degree of mØ activation was related to the number of PMNs transferred. These data indicate that inflammatory PMNs play an integral role in the activation of anti-tumor effects following CP treatment and this parallels their activation of tumor-cytostatic mØs.

CHEMILUMINESCENCE RESPONSE OF FUNCTIONALLY DIFFERENT HUMAN PERIPHERAL BLOOD MONOCYTES AND ITS MODULATION BY PROSTAGLANDINS. C.G. FIGDOR, J. KLOMP, J. LEEMANS, W.S. BONT, C. THOMAS. Division of Biophysics, The Netherlands Cancer Institute, Plesmanlaan 121, 1066 CX Amsterdam, The Netherlands.

We previously demonstrated that subsets of human monocytes which differ both functionally and in their density can be isolated by centrifugal elutriation. In the present study we investigated the burst of oxidative metabolites after stimulation by serum treated Zymosan or TPA by means of chemiluminescence (CL). It was found that the monocytes with the highest density were 3-5 times more active than those with the lowest density. Monocyte subsets which were cultured for periods of 2, 4 and 16 hours, still showed these different CL responses. The addition of 10^{-6} M PGE_2, directly prior to the CL measurement decreased the response to 60% of normal in all monocyte fractions. However, if instead PGE_2 was added 4 or 16 hours before CL measurement, the response increased to 140% and 260%, respectively. To establish whether the different CL responses of the various monocyte fractions were related to differences in the production of prostaglandins, we measured the amount of the prostaglandins F_{2a}, PGE_2, 6-K-PGF and TBX_2 produced after 24 hours of incubation. No significant differences in the prostaglandin production of the monocyte subsets could be observed. TBX_2, which was found to be the major component produced by monocytes, did not affect the CL response, nor did indomethacin. These results indicate that human monocytes consist of various functionally different subpopulations. Furthermore PGE_2 but not TBX_2 can modulate the CL response both in a positive and negative manner. The different CL responses of the monocyte subsets cannot be explained by differences in the production of prostaglandins.