50% of the cells were positive, and in 1 case, only 20% of the cells reacted with J5. PLL is a distinct variant of CLL, and the B-cell type is defined by a high density of SmIg, weak expression of mouse rosettes, and a clear reaction with the monoclonal antibody FMC7, resulting in a distinct membrane phenotype of the B prolymphocyte.

Our results, added to those of the authors quoted above, demonstrate the need to further study PLL cases in order to give a definite conclusion on the value of the antibody J5 in this disease.

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PLATELET AGGREGATION

To the Editor:

We read with great interest the paper by DiMinno et al. on the effect of single 650-mg dose of aspirin on platelet thromboxane-A2 generation. Use of two aggregating agents in combination to stimulate platelets is an attractive concept in studying actions of pharmacologic agents on platelet aggregation and thromboxane-A2 generation. Likewise, two or more aggregating agents, each in subthreshold concentration, used successively can result in complete and irreversible platelet activation. This may have physiologic relevance, since it is unlikely that platelet stimuli, such as epinephrine, collagen, or ADP, are present in circulation in aggregating concentrations. We have recently demonstrated that very small amounts (50–100 ng/ml) of calcium blocker, diltiazem, inhibit platelet aggregation and thromboxane-A2 generation induced by subthreshold concentrations of ionophore A23187 plus ADP or epinephrine. In contrast, much higher amounts (>500 ng/ml) are necessary to inhibit platelet activation induced by aggregating concentrations of a single platelet stimulus. In their study, DiMinno et al. used luciferin-luciferase reagent to measure platelet aggregation and ATP release simultaneously. We have observed that this reagent (Chronolog Corporation, Haventown, PA) potentiates the effect of subthreshold or threshold concentrations of ADP, epinephrine, and ionophore A23187, although luciferin-luciferase reagent per se may not induce platelet aggrega-

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To the Editor:

Mehta et al. suggest that the aggregation of platelets observed by us in response to subthreshold amounts of 2 aggregating agents in platelet-rich plasma, obtained from normal humans as early as 4 hr after ingestion of aspirin, really occurred in response to 3 agents, since Chronolume was also present in the aggregation tests. In Fig. 1 of that study (Blood, 61:1081, 1983), it was clearly shown that little or no aggregation occurred in response to AA, epinephrine, ADP, or collagen in the presence of Chronolume. Combinations of these aggregating agents at similar concentrations were shown to induce full aggregation in the presence of Chronolume. In other experiments (unpublished), we showed that similar aggregation occurred in the absence of Chronolume. In addition, Rao et al. (Prostaglandins and Medicine, 5:45, 1980) showed that pairs of aggregating agents, when used together at subthreshold concentrations, could cause full aggregation of platelets in platelet-rich plasma obtained from normal humans after the ingestion of aspirin. It seems clear that platelets from an individual who has ingested aspirin can aggregate fully in response to the combined effects of pairs of aggregating agents and in the absence of Chronolume. It is possible that Chronolume may potentiate or inhibit under other circumstances, and proper controls should always be run to clarify this point.

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Platelet aggregation [letter]
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