dependent increase of the single complement components levels of both pathways.

Whether this increase is due to fetal production or increasing transplacental passage remains to be established; Adinolfi's data (2, 3) suggest that fetal production plays a major role. Certainly, the defect in early complement component levels might explain the increased susceptibility to infections as a result of defective opsonization capacity. Comparison of the kinetics of AP activation in newborn and adult controls revealed no difference in the patterns, but a considerable delay in complement activation was evident in the low birth weight infants. Furthermore, in those AGA and SGA infants in whom the CH_{50} of the AP was undetectable, no lysis of RaRBC was observed even after 150 min. This phenomenon was present in eight AGA and three SGA infants suggesting that the defect of the AP may be severe especially in preterm infants and therefore of clinical relevance. An enhancing role of IgG $(Fab)_2$ on the AP activity has been reported in previous studies (5) and confirmed by our experience with agammoglobulinemic sera (data not shown). We, therefore, explored the possibility that the defect in AP activation in preterm infants, as compared with term infants, could be due to the lower serum IgG level; however, the *in vitro* addition of up to 1600 mg/dl of IgG had no effect. Low levels of complement factors are probably the main determinant of the defective activity of both CP and AP in low birth weight infants.

The activities of both pathways were preferentially correlated with gestational age rather than birth weight and also significantly correlated with each other, suggesting a similar developmental pattern. In conclusion, low birth weight infants, especially preterm infants, have an important defect of complement activity. Complement factors increase gradually during gestation and intrauterine growth retardation does not affect complement development.

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