

Letter to the Editor

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Sir,

In a recent issue of this Journal, Merrett & Merrett (1978) described different methods of quantifying circulating IgE. They reported the discrepancy between RIST and CRIST (a modified PRIST) in the IgE region below 100 u/ml. The RIST showed falsely high IgE levels in this region, especially when IgE levels were around 10 u/ml. In this respect their results were in accordance with those of Johansson, Berglund & Kjellman (1976), who discussed a similar discrepancy between RIST and PRIST when IgE levels were measured in the sera of children. For the record, we wish to report that our own results are in complete agreement with these findings.

However, we have also reported rather unexpected discrepancies between IgE levels measured by RIST and PRIST in the sera of adult asthmatics in the region above 100 u/ml (Bruynzeel, Houben & Berrens, 1977), which have now been denied in the article of Merrett & Merrett. At the time, we did not fully understand the phenomenon and suggested that unidentified serum factors might be involved which, furthermore, might be age-dependent. We have now reinvestigated the matter and have, in a recent paper (Bruynzeel, Houben & Berrens, 1979), reported the phenomenon to be reproducible. We also reported that this phenomenon is not observed when a radiolabelled antibody with better homogeneity and affinity is used in PRIST. This result indeed eliminates the argument of interfering (and age-dependent) serum-factors.

Our more recent results induce further comment on this point. Brostoff, Johns & Stanworth (1977) have described the occurrence of IgE complexes in the sera of individuals with various atopic diseases, by means of ultracentrifugation experiments. These complexes were detected by means of RIST. Similar experiments performed in our laboratory have confirmed that IgE complexes are not only detected by RIST, but are even more pronounced when measured by RAST (with highly purified house dust coupled to paper discs) (Berrens, Guikers & Bruynzeel, 1979). From these and other findings the cautious conclusion was drawn that, in such complexes, IgE may be bound to an as yet unidentified serum factor. The nature of this factor so far remains unknown, as well as its possible clinical significance. We feel, however, that, with respect to actual IgE determinations by means of RIST or PRIST, the role of these complexes should not be neglected. In RIST and PRIST, immobilized antibodies of different qualities are used, with obviously different binding characteristics. It is quite possible that complexed IgE in these tests will therefore be bound with different affinity, thereby causing incompatible results. Evidence for false-positive results in the

measurement of allergen-specific IgE due to IgE-containing complexes has also been forwarded by Cromwell *et al.* (1979). The ease of formation of IgE-aggregates in sera with high RIST-values has even been proposed as a method for distinguishing between high and low total IgE sera (Baldo, Wells & Pepys, 1974). For the moment, we would therefore like to conclude that, when performing IgE assays, one should always be aware of the possible interference of unknown serum factors, irrespective of the method used.

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