solution of pyrethrins in the same solvent made up from concentrates of the same type but freshly prepared. In addition, appreciable absorption of light was observable (in alcoholic solution) at the absorption maximum characteristic of the pyrethrins[2]; thus (*A*) showed $\lambda_{max.}$ 2310 A., $E^{1\%}_{1cm.}$ 144, whereas (*B*) showed $\lambda_{max.}$ 2280 A., $E^{1\%}_{1cm.}$ 495.

It has been shown[3] that the light absorption of the pyrethrins is almost certainly due to a summation of the separate contributions of two isolated chromophores (*a*) a conjugated dienoid system in the pentadienyl side chain, and (*b*) an αβ-unsaturated ketonic grouping in the *cyclo*pentenolone ring. In view of these data and the fact that the chemical methods of assay mentioned above depend, respectively, upon the properties of the carbonyl group and of the chrysanthemum mono- and di-carboxylic acids, it seems most reasonable to assume that the αβ-unsaturated ketonic grouping and the acidic fragments of the pyrethrin molecules remain unaltered in these resinous products but that the change—probably polymerization—involves the pentadienyl side chain. The accompanying great reduction in toxicity of such products, with retention of some degree of biological activity, would appear to be in accord with the conclusions of Staudinger and Ruzicka[4] on the effect on toxicity of changes in the molecular structure of pyrethrins I and II and related synthetic compounds. Furthermore, it has been observed[1] that saturation of the side chain in the pyrethrin II molecule leads to a great decrease in both 'knock-down' and lethal effects.

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Relation of Tea 'Fermentation' to Normal Respiration

IT is correct, as Roberts states in his communication[1], that I did not "repeat the conditions under which the Tocklai experiments were carried out". I attempted[2] to simulate injuries to tea leaf comparable with those caused during normal tea manufacture. Roberts, however, used an attachment which will grind nuts to a fine paste and thereby caused more structural damage to the leaf than I did with a mincing machine and far more than ever results from tea-rolling during manufacture. He attaches great importance to the shearing forces produced by his attachment because he believes that "such shearing forces are to be considered as responsible for the disorganization of respiratory processes which results in tea-fermentation"—even in the absence of visible cell damage. His own published results[3], however, do not give support to that belief.

Roberts shows that two non-tanniferous tissues, *Tropæolum majus* and *Hibiscus rosasinensis*, have a carbon dioxide output of 1·98 µl. and 1·6 µl. respectively per mgm. dry weight of tissue per hour, which when compared with the value 1·29 µl. found by Boysen Jenson[4] in respiration experiments on *Tropæolum majus* shows them to be of the same order. There is no question of polyphenol oxidation in these tissues, and since the *R.Q.* is very near unity the carbon dioxide output is presumably all due to normal respiration.

As Roberts's experiment demonstrates that very severe mincing plus the shearing effects produced by the attachment do not completely disorganize the respiratory process in *T. majus* and *H. rosasinensis*, why must we *assume* that they will have a different effect on tea leaf? If, as I submit, respiratory activity persists in portions of crushed tissue (as caused by normal tea-rolling) independent of any fermentation proceeding at the same time, there appears no need to build up an elaborate hypothesis to account for the carbon dioxide produced during the two co-existing processes.

The higher carbon dioxide output from other tissues (including tea) of itself proves nothing when the wide variation of respiration intensities in different plant species is borne in mind.

Discussing my results with washed and unwashed tissue-mince, Roberts has stated that it is by no means unlikely that in the unwashed tissues the carbon dioxide produced (1) by carbohydrate oxidation in the damaged cells with *o*-quinones as H-acceptors and (2) normal respiration in the undamaged cells inhibited by free (oxidized ?) polyphenols are together equal to the respiration of the washed undamaged tissues now uninhibited by polyphenols. This would mean that the carbon dioxide produced by the *o*-quinone reaction always balanced the inhibition in normal respiration. Such a coincidence in quantitative results is, however, scarcely to be expected in a complex system such as minced tea leaf.

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Darwin and 'Water-Bloom'

SOME years ago, I described the formation of red 'water-bloom' in the seas round Cape Peninsular, by myriads of the ciliate *Mesodinium rubrum* Lohmann[1]. I had been unable to find references (in the literature available on the spot) to ciliates as a cause of this phenomenon, though 'water-bloom' caused by various other micro-organisms was well known in many localities. A reply from Prof. O. Paulsen[2] showed that formation of red 'water-bloom' due to Mesodinium had been seen by him at Iceland thirty years earlier. The Danish publication was out of my reach at the time, but I ought not to have missed the astoundingly accurate, detailed description of Mesodinium forming red water made by Darwin[3] some fifty years earlier still, before Mesodinium had in fact been named. No one who has examined swarming Mesodinium alive could have any doubt as to the identity of the organisms described by Darwin, and it would be very hard to improve his verbal description.

The red water was encountered by H.M.S. *Beagle*