which the queens develop. The reasons suspected for such a high protein content in queens are as following: 1. the queens are sufficiently fed with proteinaceous food by workers: 2. they are much bloated and physogastric, weighing from 20 to 200 times more than the female alates: 3. the ovaries being filled with an enormous number of proteinaceous eggs comprise the bulk of the body weight of the physogastric queen.

The kings, which are developed from male alates, show the same 47% protein as male alates. The reason may be presumed to be that the kings do not undergo any marked change, as do the queens. Among neuter castes, the soldiers have almost an equal amount of protein as potential sexual castes (Table). This is suspected to be for their proteinaceous salivary fluid which fills the salivary receptacles which in turn occupy the major portion of the abdomen in soldiers. In support of this opinion regarding the content of salivary fluid, BOUILLON[7], while studying the termites of Ethiopian region, mentions that the saliva doubtless contains proteinaceous material. In conclusion, the differential occurrence of protein in various castes and undifferentiated instars of the termite *O. assmuthi* is solely dependent upon the different roles played by each caste and as due to the regulated feeding.

Résumé. L'évaluation de la somme de protéine contenue dans les représentants des diverses castes du termite *Odontotermes assmuthi* donne les résultats suivants: 1. Les formes reproductrices potentielles, mâles et femelles, ont des quantités significatives (47,9 et 37%) que requirèrent leur activité reproductrice. 2. Les rois issus de mâles ailés n'atteignant leur pourcent maximum de protéine qu'à l'âge adulte. 3. Les reines en augmentent leur quantité (à 65,7%) en proportion de leur intense production d'œufs. 4. Les ouvriers et les soldats soumis à des tâches ardues contiennent beaucoup de protéine (33,5 et 44,5%).

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The Membrane Expansion Theory of Anesthesia: Direct Evidence Using Ethanol and a High-Precision Density Meter

The membrane expansion theory of anesthesia[1] states that anesthetics and other nerve-blocking drugs adsorb to hydrophobic regions of excitable membranes[2–5], expanding the hydrophobic regions of membrane proteins[6–12] and thus blocking the ionic conductance channels underlying nerve cell action potentials.

Surgical concentrations of general anesthetics do expand the membrane area of erythrocytes by about 0.4%, while the volume of the anesthetic which occupies the membrane under these conditions is only 0.02% or less[1,7]. Since the membrane expansion is roughly 20 times the occupying volume of drug in the membrane phase, it has been suggested that extensive conformation changes in membrane proteins may be involved[1]. Although it is known that anesthetics expand the area of erythrocyte membranes, this report provides the first direct evidence that anesthetics also expand the specific volume of such membranes.

Erythrocyte membranes and guinea-pig brain synaptosomes[13] were suspended in 10 mM sodium phosphate buffer ($[H^+]$ = 40 nM). The final dry weight of the membranes ranged from 0.2 to 1 g per 100 ml of suspension. Using a precision density meter[14] at 25 ± 0.01 °C, the density of the dry membrane (d_m) was obtained by eq. 1 (see appendix):

$$d_m = \frac{W \times d_{ms}}{f(d_b - d_{ms}) + h(1 - d_{ms}) + W} \quad \ldots\ldots\ldots \quad (1),$$

where W is the dry weight of the membranes (in g dry membrane per ml of suspension), where d_{ms} and d_b are the measured densities of the membrane suspension and the buffer solution, respectively, in the presence of varying concentrations of drug, where h is the fraction of membrane-associated water (i.e. non-solvent water) in the suspension (having dimensions of ml water per ml of suspension), and where f is the fraction of buffer medium in the suspension (in units of ml buffer medium per ml suspension). The amount of membrane-associated water is generally 0.3 ml per g of dry membrane[15]; that is, h = 0.3 W. The value for f was then taken as 1-W-h.

Ethanol lowered the density of the membrane suspension disproportionally more than that of the buffer solution, qualitatively indicating that the density of the biological membranes decreased in the presence of the drug. The values for d_b and d_{ms}, respectively, (in units of g/cm^3, with an error of $\pm 1.5 \times 10^{-6}$ g/cm^3) in a typical experiment were: 0.997950 and 0.998649 for 0 M ethanol

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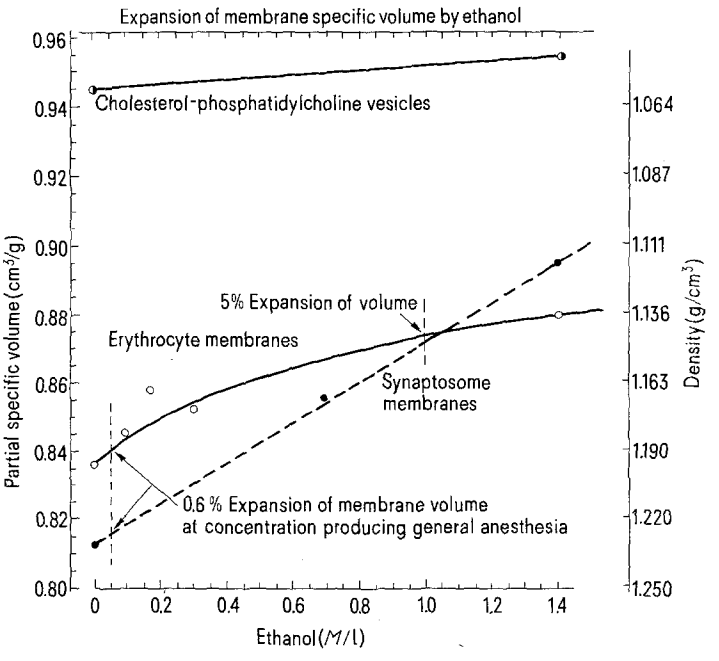

Ethanol expands the specific volume of erythrocyte and brain synaptosome membranes by 0.6% at general anesthetic concentrations (0.05 *M*), and by 5% at local anesthetic concentrations (1 *M*). Liposomes were expanded by only 0.01% and 0.3% at these ethanol concentrations, respect ively. 25 °C.

(control); 0.992487 and 0.993137 for 0.7 *M* ethanol; and 0.987146 and 0.987699 for 1.4 *M* ethanol.

The results for d_m are in the Figure. At concentrations of ethanol which produce general anesthesia[16] (0.05 *M*) the specific volumes (= reciprocal of the specific density) of the membranes increased by 0.5–0.6%. At ethanol concentrations which produce local anesthesia[1] (1 *M*) the specific volume expansions were between 3 and 6%.

The Figure also shows that 0.05 *M* ethanol expanded the specific volume of liposome membranes[17] (lecithin: cholesterol: 1:2 molar ratio) by only 0.01%, in agreement with the membrane-occupying volume of 0.02% by the drug in the membrane phase[1].

Since the specific volume expansion of the biological membranes (0.5%) is many times more than that for lipid membranes (0.01%), this strongly suggests that conformation changes in membrane proteins underly the membrane expansion. Pressure reversal of anesthesia would then involve a return of the protein conformation to its native state, rather than modify the lipids. Although it has been claimed that pressure reverses or antagonizes the anesthetic-induced leakiness or disorder in liposomes[18–20], this is not true antagonism, since pressure by itself changes the leakiness and ordering of lipids[1,18–20] without shifting the dose-response curve[21].

Résumé. Utilisant une nouvelle mesure de densité, de haute précision, on étudia l'effet de l'éthanol sur le poids spécifique des membranes des érythrocytes et des synaptosomes. La concentration de l'éthanol qui produit l'anesthésie générale (0.05 *M*) étendit le volume spécifique des membranes à 0.5–0.6%. Aux concentrations de l'éthanol qui produisent l'anesthésie locale (1 *M*) l'extension du volume spécifique fut de 3 et 6%. Ces résultats supportent la théorie de l'extension des membranes par les anesthétiques et suggèrent que l'extension des constituants protéinés des membranes est plus importante que celle des constituants lipides.

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[21] Appendix: Equation 1 is derived as follows. $d_{ms} = (m_b + m_{mw} + m_m)/(V_b + V_{mw} + V_m)$, where m_b, m_{mw} and m_m are the masses (in g per test-tube) of buffer medium, membrane water, and dry membrane, respectively, and where V_b, V_{mw} and V_m are the corresponding volumes (in ml per tube). Dividing all 6 terms by V_s (= volume of suspension in tube), and substituting W for m_m/V_s, m_m/d_m for V_m, and $d_b V$ for m_b, one obtains $d_{ms} = [(d_b V_b/V_s) + (m_{mw}/V_s) + W]/[(V_b/V_s) + (V_{mw}/V_s) + W/d_m]$. Further substituting f for V_b/V_s, and h for m_{mw}/V_s, and rearranging, one arrives at equation 1.

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Time Dependence of a Neurochemical Correlate of a Learning Task: a Non-Disruptive Approach to Memory Consolidation

Learning and memory constitute the acquisition and retention of information in time. Most techniques, such as electro-convulsive shock[1], anaesthetics[2] and inhibitors of protein and RNA synthesis[3,4] used to establish the time course of memory consolidation are 'disruptive' in that they depend on the extinction of memory; furthermore, the results based on these techniques are conflicting. It is felt that the time course of a neurochemical correlate