STUDIA UNIVERSITATIS BABEŞ-BOLYAI, CHEMIA, L, 1, 2005

LANGMUIR KINETICS AND ADSORPTION MECHANISM AT THE OIL/WATER INTERFACE

GHEORGHE TOMOAIA[a], MARIA TOMOAIA-COTISEL[b], CSABA RACZ[b] CRISTINA-RAMONA ISPAS[b] and CALIN FLOARE[c]

[a]Department of Orthopaedic Surgery, "Iuliu Hatieganu" University of Medicine, 400015 Cluj-Napoca, Romania
[b]Department of Physical Chemistry, "Babes-Bolyai" University of Cluj-Napoca, 400028 Cluj-Napoca, Romania
[c]National Institute for Research and Development of Isotopic and Molecular Technologies, P.O. Box 700, Cluj-Napoca, Romania

ABSTRACT. The adsorption mechanism of some biocompounds, e.g. two local anesthetics, like dibucaine and tetracaine, and of stearic acid, from bulk solutions to the oil/water interface was studied by using the pendant drop and ring methods. The biocompounds are approaching the oil/water interface from the opposite directions, namely anesthetics from water phase and stearic acid from oil phase. On the other hand, anesthetics are charged species and stearic acid is uncharged under working conditions. The kinetic analysis shows that Langmuir kinetic approach describes the dynamic interfacial pressures within the limits of the experimental errors over a wide range of time and for different surfactant concentrations in bulk solutions. It is also concluded that this approach allows the calculation of the ratio of the adsorption and desorption rate constants of these biocompounds at the oil/water interface. Obtained results are in substantial agreement with similar earlier reported data for the adsorption of different surfactants at various oil/water interfaces as well as with their molecular structure. The driving force for the adsorption of anesthetics (water soluble molecules) is the hydrophobic interactions among the penetrated hydrophobic chains and the oil phase, which are accompanied by the increase of the entropy of the system because of the destruction of the ordered structure of water molecules formed around the hydrophobic chains in aqueous phase. On the contrary, the driving force for the adsorption of stearic acid (practically water insoluble compound) is the change in the enthalpy of the system due to the hydration of hydrophilic polar head groups when they immerse in the water phase.

Key words: Dynamic interfacial pressures; dibucaine; tetracaine; stearic acid; oil/water interface; Langmuir adsorption kinetics; adsorption mechanism.

INTRODUCTION

The adsorption kinetics of various surfactants at the liquid interfaces has attracted a considerable attention in the last several decades [1-31] due to its industrial importance [6, 7, 28] and to its biological and medical significance [32-39]. These studies have involved various experimental techniques and different theoretical models describing the adsorption kinetics of surfactants at liquid interfaces.

Basically, the surfactant adsorption from a bulk solution to a liquid interface, like liquid/gas [6-18] or to a liquid/liquid interface [1-6, 19-27], occurs in two steps. Surfactant molecules are first transported from the bulk to the subsurface by diffusion; the subsurface is a liquid layer just below the interface, belonging still to the bulk. The second step consists of the transfer of surfactant molecules from the subsurface to the interface, implying sometimes a transfer through a potential barrier [25, 28-31]. The controlling rate may be either the diffusion or the transfer (i.e. the adsorption is barrier-controlled) and several theoretical models have been developed for both processes.

For instance, the adsorption kinetics of some aliphatic carboxylic acids and aliphatic alcohols [9-12], like 1, 9 nonane dicarboxylic acid and 1, 9 nonane diol [9], was studied at the air/water interface and described by Langmuir kinetics, considering the adsorption and desorption rate constants. Another example is related to the adsorption of some salts of fatty acids, like sodium laurate [13-16], sodium myristate [13-15], and sodium oleate [17], from aqueous solutions to the air/water interface, which was analyzed by means of diffusion controlled kinetics.

Recently, the very slow adsorption of two anesthetics from water phase to oil/water interface [4] and of stearic acid from oil phase to the oil/water interface [1] has received a considerable attention using the diffusion controlled kinetics. The calculated diffusion coefficient values are much lower values than the expected physical values. Usually, this decrease is interpreted as the effect of the energetic barrier to the adsorption according to the theoretical model proposed in [5, 29, 30].

The dynamic interfacial pressures of these biocompounds, namely two anesthetics and stearic acid, will be further studied and characterized at the oil/water interface by using the Langmuir kinetic approach. The strong adsorption of both types of compounds, anesthetics and fatty acids, is due to the considerable change in the free energy of the system due to the immersion of hydrophobic groups and of hydrophilic groups into the oil and into the water phase, respectively. Nevertheless, the mechanism of the change in adsorption free energy is different for anesthetics than that for a fatty acid.

The main goal of this study is to analyse the experimental data recorded in terms of the time dependent interfacial pressures for the adsorption of the two anesthetics, like dibucaine and tetracaine, and of stearic acid at the same benzene/ water interface and to discuss the adsorption mechanism in Langmuir kinetic approach.

LANGMUIR ADSORPTION KINETICS AT LIQUID/LIQUID INTERFACES

Adsorption kinetics of a surfactant from the bulk solution to a clean (pure) interface, without convection currents in the liquid [40], is described by the Ward and Tordai's diffusion [1, 4, 8] equation.

Previously, we found for these systems that experimental data cannot be understood by a simple diffusion mechanism [1, 4] and we replaced it by the Langmuir kinetic approach [9-11, 41] of the following form:

$$\frac{d\Gamma(t)}{dt} = k_1 c_s \left(1 - \frac{\Gamma(t)}{\Gamma_\infty}\right) - k_2 \frac{\Gamma(t)}{\Gamma_\infty} \quad (1)$$

where k_1 and k_2 are the rate constants for the adsorption and for the desorption processes, respectively, and Γ_∞ represents the maximum adsorption for the saturation of the liquid interface with biocompound molecules.

Eq. (1) is valid also at equilibrium, when $\frac{d\Gamma(t)}{dt} = 0$, $\Gamma(t) = \Gamma_e$ and $c_s = c_0$.

With these conditions Eq. (1) yields:

$$k_1 c_0 = \frac{k_1 c_0 + k_2}{\Gamma_\infty} \Gamma_e = k\Gamma_e \quad (2)$$

For some cases, with biocompound concentrations high enough, a diffusion equilibrium can be thought to be established, involving $c_s = c_0$. In this case [9], a combination of Eqs. (1) and (2) entails:

$$\frac{d\Gamma}{dt} = -k(\Gamma - \Gamma_e) \quad (3)$$

where k is the rate constant.

Integration of the Eq. (3) yields:

$$\Delta\Gamma = \Delta\Gamma_0 e^{-kt} \quad (4)$$

with $\Delta\Gamma = \Gamma - \Gamma_e$, $\Delta\Gamma_0 = \Gamma_0 - \Gamma_e$, where Γ_0 stands for the adsorption at t = 0, i.e. $\Gamma_0 = 0$.

If the increase of adsorption is proportional to the decrease of interfacial tension, Eq. (4) may be written as:

$$\Delta\sigma = \Delta\sigma_0 e^{-kt} \quad (5)$$

or in a logarithmic form:

$$\ln \frac{\Delta\sigma_0}{\Delta\sigma} = \ln \frac{\sigma_0 - \sigma_e}{\sigma - \sigma_e} = kt \quad (6)$$

where σ, σ_e and σ_0 stand for the actual dynamic interfacial tension, for the equilibrium interfacial tension, and for the interfacial tension in the absence of the surfactant, respectively.

These equations derived for the Langmuir kinetic mechanism are considered in more detail in the followings. For the beginning Eq. (6) has been tested in the case of the two anesthetics, viz. dibucaine and tetracaine, and of stearic acid.

MATERIAL AND METHODS

Biocompounds used were two local anesthetics: dibucaine (2-butoxy-N-[2-(diethylamino) ethyl] – 4 -quinoline carboxamide hydrochloride) and tetracaine (4-butyl amino benzoic acid 2-(dimethyl amino) ethyl ester hydrochloride) and a fatty acid, namely stearic acid (octadecanoic acid); all synthetic commercial products of high purity (minimum 99%) were purchased from Sigma. The purity of biocompounds was checked by thin layer chromatography and they were used without an additional purification.

As oil phase, benzene pro-analysis was used and it was purchased from Merck. We measured the interfacial tension as a function of time for the pure benzene/aqueous solutions of pH 2 interface and a time independent value was recorded. Thus, benzene did not contain surface active contaminants and, consequently, it was used without additional purification. Twice-distilled water of pH 2 was used, containing 0.01 mole/dm^3 of hydrochloric acid. The water had a resistivity of at least 18 Mohm cm and a surface tension at the interface with air of 72 mN/m at 25^{o}C.

In order to study the adsorption of the three biocompounds at the same liquid interface, the benzene/water of pH 2 systems were chosen. In the case of stearic acid, at pH 2, its adsorbed monolayer is an uncharged one, the molecules being completely unionized [32, 34], and insoluble in water phase [32]. On the other hand, dibucaine and tetracaine may exist in three forms [42] uncharged (free base) and charged ones, *i.e.,* monocation (mono-protonated) and dication (diprotonated) molecules. The calculations show at pH 2, that the dibucaine is almost in mono-protonated form, and the tetracaine is a mixture of monocation (45%, mono-protonated) and dication (55 %, di- protonated) molecular species [42]; all molecular species are completely insoluble in the bulk benzene phase.

Dynamic interfacial tensions in the time range from 1 minute up to 90 minutes were measured by pendant drop and by ring methods for the following systems: aqueous solutions (pH 2) of various anesthetic concentrations at the interface with pure benzene and benzene solutions of various stearic acid concentrations at the interface with water of pH 2. Therefore, due to the very low solubility of anesthetics in benzene and of stearic acid in water of pH 2, any transport across the benzene/ water interface can be neglected.

The pendant drop technique was described by us elsewhere [26]. By using a computer program the dynamic interfacial tensions were determined. These values were finally transformed into the dynamic interfacial pressures by subtracting the actual interfacial tensions from the interfacial tension of the pure interface in the absence of surfactants.

Experimental data obtained by the pendant drop technique were compared with the data obtained by ring method, which was described by us previously [27, 35, 43]. The agreement between the two methods is good and the deviations do not exceed the error of the individual method. The accuracy of the interfacial tension or the interfacial pressure measurements was ± 0.1 mN/m, in agreement with literature data [23, 41]. All measurements were performed at constant temperature of 20 ± 0.1 ^{o}C.

RESULTS AND DISCUSSION

Dynamic interfacial tension σ(t) values obtained experimentally allowed us to calculate the corresponding $\Pi(t) = \sigma_0 - \sigma(t)$ interfacial pressure values. The Π(t) *versus* time (t) curves, characterizing the adsorption process of the three biocompounds investigated from bulk phase to the benzene/ water (pH 2) interface are presented in Figs. 1-3. As can be seen, the interfacial pressures vary with surfactant bulk concentrations and with time over a wide range of time, from 0 to 15 for local anesthetics and from 0 to 90 min for stearic acid.

Generally, the equilibrium interfacial pressures (Π_e in mN/m) are recorded at 30 min for anesthetics and at 120 min for stearic acid when the adsorption equilibrium is completely attained and it is demonstrated by the constant value of the interfacial pressures.

The validity of the Langmuir kinetic approach, given by Eq. (6), has been tested by calculating the left hand side of this equation, by using the σ(t) and σ_e values (the latter ones being measured at 30 min for the two anesthetics and 120 min for stearic acid). The interfacial tension of the pure benzene/water interface in the absence of surfactant was taken σ_o = 34.7 mN/m under the chosen experimental conditions.

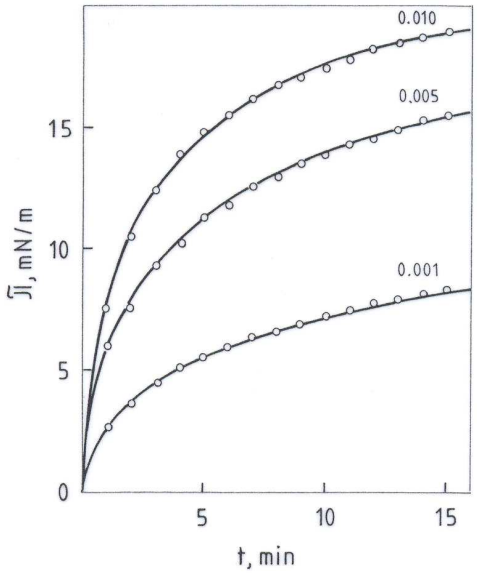

Fig. 1. Experimental dynamic interfacial pressure (Π in mN/m) of dibucaine aqueous solutions (pH 2) at the benzene/water interface, as a function of time (t, min). Figures indicate the dibucaine bulk concentration (c_0 in mole dm^{-3}).

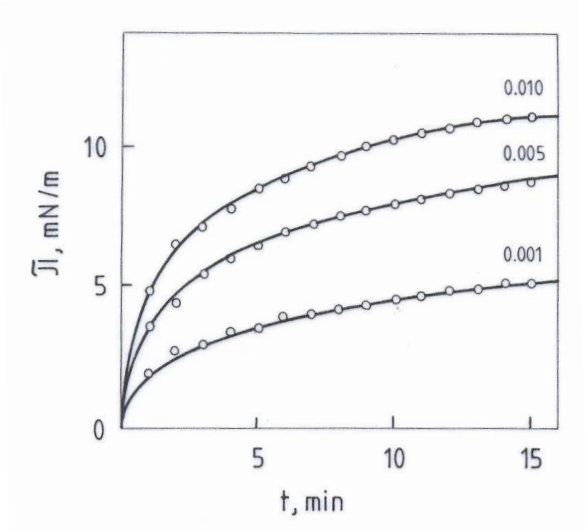

Fig. 2. Dynamic interfacial pressure (Π in mN/m) of tetracaine aqueous solutions (pH 2) at the benzene/water interface. Figures indicate the tetracaine bulk concentration c_0 in mole dm^{-3}.

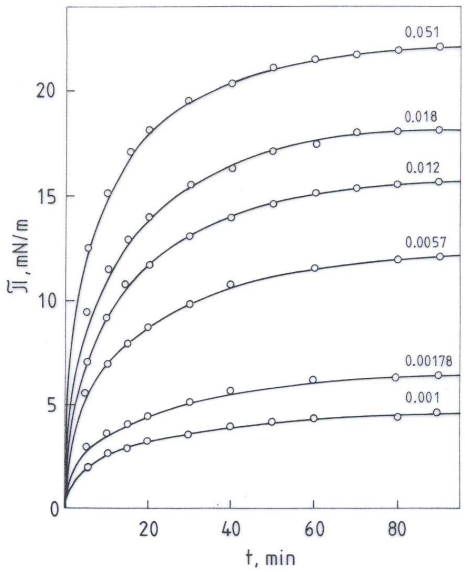

Fig. 3. Dynamic interfacial pressure (Π in mN/m) of stearic acid benzene solutions at the benzene/water (pH 2) interface. Figures indicate the stearic acid bulk concentration c_0 in mole dm $^{-3}$.

In order to explore Eq. (6), the logarithmic function *versus t* was investigated and it was found that it exhibits a quite good linearity as it is shown in a few examples given in Fig. 4 for the three biocompounds studied at the same bulk concentration of 10^{-3} mole dm $^{-3}$.

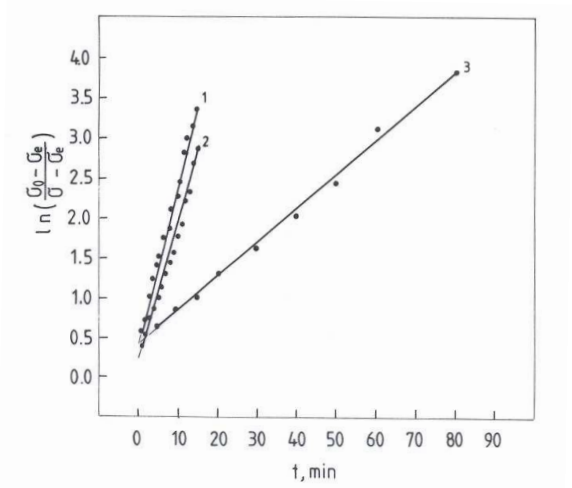

Fig. 4. The logarithmic function versus time, Eq. (6), for the adsorption of dibucaine (curve 1), tetracaine (2), and stearic acid (3) for the same biocompound bulk concentration c_o of 10^{-3} mole dm $^{-3}$.

As it can be seen in Fig. 4, the straight lines do not pass through the origin of the coordinate system, and in reality Eq. (6) is of the following form:

$$y = \ln\frac{\sigma_0 - \sigma_e}{\sigma - \sigma_e} = a + kt \qquad (7)$$

By performing a linear regression, the parameters a and k have been determined. Results are presented, together with the correlation coefficient (r), in Table 1. In the last column n indicates the number of experimental points used in the linear regression.

As shown in Table 1, the a values are different from zero, particularly for stearic acid. This means that the basic hypothesis used for deriving Eq. (6) is an approximation and it is not perfectly valid, presumably because the diffusion equilibrium is not completely established and the boundary condition $c_s = c_0$ is not yet fulfilled.

Table 1

The a and k parameters of Eq. (7).

Biocompound	c_0 mole dm^{-3}	a	k min^{-1}	r	n
Dibucaine	0.001	0.152	0.169	0.9932	15
	0.005	0.202	0.190	0.9918	15
	0.01	0.284	0.234	0.9909	14
Tetracaine	0.001	0.342	0.154	0.9966	15
	0.005	0.340	0.176	0.9974	14
	0.01	0.330	0.199	0.9984	15
Stearic acid	0.001	0.430	0.0397	0.9991	9
	0.0018	0.374	0.0396	0.9977	7
	0.0057	0.395	0.0406	0.9990	8
	0.012	0.439	0.0415	0.9978	8
	0.018	0.568	0.0431	0.9994	9
	0.051	0.566	0.0501	0.9949	10

Nevertheless, from the k values reported in Table 1, some conclusions can be drawn. As seen, with increasing surfactant bulk concentrations c_0 the k values derived increase. This effect is expected on the basis of the Eq. (2) written in the following form:

$$k = \frac{k_1}{\Gamma_\infty} c_0 + \frac{k_2}{\Gamma_\infty} \qquad (8)$$

and the k values *versus* c_0 exhibit indeed an acceptable linearity. To illustrate this situation, the relative k_1/Γ_∞ and k_2/Γ_∞ rate constant values and the corresponding correlation coefficients are presented in Table 2.

Table 2

Relative adsorption (k_1/Γ_∞) and desorption (k_2/Γ_∞) rate constants derived by Eq. (8) from k values given in Table 1.

Biocompound	k_1/Γ_∞ $mole^{-1}\ dm^3\ min^{-1}$	k_2/Γ_∞ min^{-1}	r	k_1/k_2 $mole^{-1}\ dm^3$
Dibucaine	7.25	0.159	0.9914	45.6
Tetracaine	4.99	0.149	0.9988	33.5
Stearic acid	0.212	0.0392	0.9990	5.4

The analysis of data shown in Table 2 shows a quite good validity of Eq. (8). The relative rate constant values are rather reasonable and their ratios are in agreement with the published data for the adsorption of various surfactants at liquid interfaces [9-11].

ADSORPTION MECHANISM AT THE OIL/WATER INTERFACE

To give a better view on the adsorption mechanism, in Fig. 5, we have sketched the change of energy E versus distance Z to the interface and illustrated the energetic barriers for the adsorption and desorption process at the liquid-liquid interface.

The adsorption rate k_1 constant depends on the activation energy of adsorption, noted E_1, by the following relation $k_1 \sim \exp(-E_1/k_BT)$, where k_B and T have their known meaning, and $E_1 > 0$. Similarly, the desorption rate k_2 constant depends on the activation energy of desorption, noted E_2 ($E_2 > 0$), by the relation $k_2 \sim \exp(-E_2/k_BT)$. Evidently, $E_a = E_1 - E_2$, where E_a is the adsorption energy. For $E_2 > E_1$, the adsorption energy is negative ($E_a < 0$) and the surfactant molecules spontaneously adsorb at the oil/water interfaces. The k_1/k_2 ratio gives the equilibrium adsorption constant (K) given by $K \sim \exp(-E_a/k_BT)$, which does not depend directly on the adsorption barrier.

In the case of anesthetics, the molecules adsorb from water to the oil/water interface by penetrating their hydrophobic chains into the oil phase. Their adsorption at the oil/water interface is controlled by the hydrophobic effect. According to this effect, the hydrocarbon chains in aqueous phase are surrounded with ordered water molecules [10]. In the activated state for adsorption, the water soluble anesthetic molecule is assumed devoid of the structured water [10] and the transition of hydrated molecule to its activated state is entropic. In fact, a change in entropy appears and it is due to the destruction of the ordered layers of water molecules around the alkyl chains. Even more, the activation E_1 energy of adsorption for the water soluble ionic compounds, like the two anesthetics, depends on the surface potential and of electrostatic interactions between ionized polar groups. Then, the activated state molecule gives the adsorbed anesthetic molecule (i.e. adsorbed state) at the oil/water interface with its polar head group hydrated and hydrocarbon tail immersed in the oil phase.

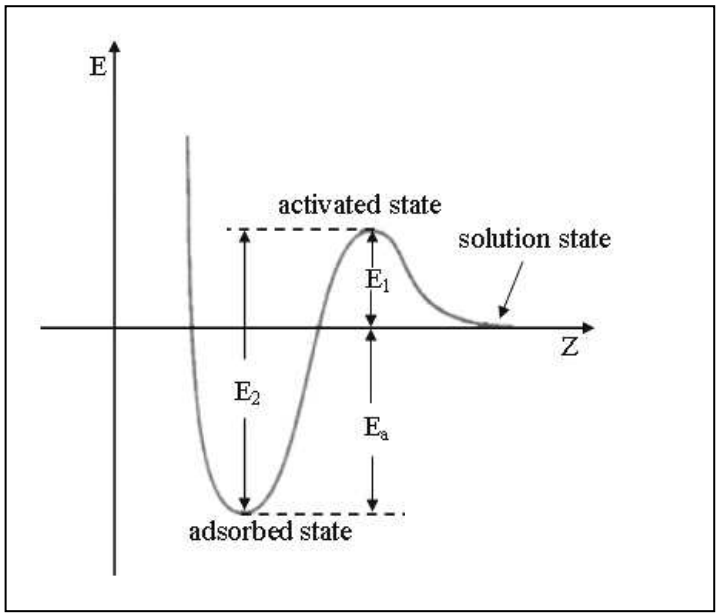

Fig. 5. Relation among the adsorption barrier E_1 (i.e. activation energy of adsorption), desorption barrier E_2 (i.e. activation energy of desorption) and the adsorption energy (E_a).

The adsorption k_1 rate constant is related to the transition energy (E_1) of a hydrated ionized anesthetic molecule in water (i.e. solution state) to its activated form (i.e. activated state). The driving force for adsorption, expressed by the adsorption rate constant, is at least in part entropic and might depend on the chain length and seems to be related to the hydrophobic effect. The activation E_1 energy of adsorption for the ionized anesthetics depends also on the surface potential and on the electrostatic interactions between ionized polar groups.

On the other hand, the desorption k_2 rate is related to the transition energy of the adsorbed anesthetic molecule at the oil/water interface to its activated complex described above, still in the water phase near the interface, devoid of its structured water particularly around its hydrocarbon chain. The desorption process apparently does not depend on the hydrocarbon chain length as we found in the case of anesthetics (see Table 2). This process is enthalpic and the activated molecule transforms into a surfactant molecule with the structured water around it in the aqueous phase. The desorption barrier E_2 is higher than E_1 and the adsorption E_a energy depends on the surface potential and on the adsorption energy of the hydrophobic tail.

In a first approximation, in the case of stearic acid, the adsorption energy and desorption barrier are assumed to be almost equal in magnitude mainly to the energy of transfer of the polar head group from the oil into the water phase. Because the hydrocarbon tail remains in the oil phase, the adsorption barrier is expected to be small and it might characterize the state of the adsorbed molecules due to the molecule reorientations in the oil/water interface [3]. Presumably, the

long saturated hydrocarbon chain must undergo a great number of conformational transitions to allow the polar headgroup of stearic acid to adopt a proper orientation and penetrate into the oil/water interface.

Stearic acid molecules adsorb from oil phase to the oil/water interface by immersing their unionized polar groups into the water phase. The driving force for the adsorption of stearic acid is the change in enthalpy due to the hydration of its adsorbed hydrophilic groups. Thus, stearic acid adsorbs strongly at the oil/water interface because the hydrophilic polar groups anchor and hydrate in water phase and may admit the possible conformational rearrangements in the adsorbed layer.

Returning to our data recorded in Table 2, one can observe that the relative adsorption k_1/Γ_∞ rate constant for dibucaine is higher than the k_1/Γ_∞ value for tetracaine, pleading for an adsorption barrier (i.e. an activation energy of adsorption) for dibucaine smaller than the corresponding one for tetracaine. This finding is also supported by the fact that the dibucaine is monocationic while tetracaine is a mixture of monocationic (45%) and dicationic (55%) forms, under the given working conditions. Therefore, it becomes clear that the electrostatic repulsion between the ionized polar groups represents at least a part of the barrier to the adsorption of the new arrival molecules.

Further, the relative desorption k_2/Γ_∞ rate constants are almost equal for both anesthetics and independent on the chain length, the chain being longer for dibucaine that for tetracaine. Thus, monolayers of anesthetics at the oil/water interface desorb at almost the same rate, also independent of the ionic molecular species existing within the monolayers.

It is interesting to compare the k_1/k_2 ratios, given also in Table 2. These ratios represent the equilibrium adsorption constants, given by $K \sim \exp(-E_a/k_B T)$, which are dependent on the adsorption energy and they are not dependent directly on the adsorption barriers. The equilibrium constants or the adsorption energies characterize the interfacial activity of these biocompounds at the oil/water interface. The value for dibucaine is higher than the corresponding value for tetracaine suggesting a higher interfacial activity of the dibucaine in excellent agreement with the interfacial pressure measurements.

It is to further observe an obvious similarity in the adsorption behaviour between these two types of biocompounds, i.e. anesthetics and stearic acid. They spontaneously adsorb at the oil/water interface leading to the decrease of the free energy of the system, but the mechanism of the adsorption is different as discussed above and consequently, the rate constants are determined by different factors.

Therefore, the adsorption of the two anesthetics and of stearic acid has completely different adsorption energy and adsorption barriers and different mechanism of desorption at the oil/water interface. However, the relative desorption rate k_2/Γ_∞ constants from monolayers of ionized anesthetics molecules are much higher than the corresponding value for monolayers of non-ionized stearic acid molecules at the same interface benzene/water of pH 2. This finding is plausible because the stability of the uncharged monolayers [32], like stearic acid on pH 2, is generally higher than that of the charged monolayers, namely mono-protonated dibucaine monolayers and the mixture of monocation and dication for tetracaine monolayers. At high interfacial pressures at the saturation of monolayers, the charged

molecules desorb faster being expelled from monolayers by the high lateral pressure and being loosed from monolayers of ionized anesthetics molecules due to the repulsive interaction forces.

Furthermore, considering the equilibrium adsorption of stearic acid $\Gamma_\infty = 2.1\ 10^{-10}$ mole/cm^2 at the saturation [26] of monolayers at the benzene/water interface, the desorption rate constant k_2 in value of $8.26\ 10^{10}$ molecules cm^{-2} s^{-1} was calculated. This value of the desorption rate constant k_2 of stearic acid from the oil/water interface appears as a plausible one, still higher than the desorption rate constant k_2 for the spread monolayers; e.g. hexadecanol monolayers [40] desorb at a rate of about $0.5\ 10^{10}$ molecules cm^{-2} s^{-1} from the air/water interface.

Again, in spite of the different mechanism of desorption at the two different fluid interfaces, the difference between the two desorption rates might be explained due to the higher cohesive forces in the spread surfactant monolayers at the air/water interface than in the adsorbed monolayers at the oil/water interface. In adsorbed layers the oil molecules penetrate among the hydrocarbon chains of adsorbed stearic acid molecules diminishing the molecular interactions among the adsorbed biosurfactant molecules [27, 40, 43] and, consequently, the adsorbed molecules at the oil/water interface might desorb at a higher rate than the spread ones at the air/water interface.

CONCLUSION

The adsorption behavior of anesthetics (water soluble ionized dibucaine and tetracaine) and of stearic acid (oil soluble), which adsorb from the water phase and the oil phase, respectively, at the oil/water interface shows a certain type of similarity.

In the case of stearic acid, the molecules adsorb from oil phase to the oil/water interface by immersing their unionized polar groups into the water phase. Unlike the anesthetics, which are water ionisable soluble compounds, the driving force for the adsorption of stearic acid is the change in enthalpy due to the hydration of its adsorbed hydrophilic groups when they immerse in the water phase,

In the case of anesthetics, their adsorption at the oil/water interface is controlled by the hydrophobic effect, accompanied by the increase of the entropy due to the destruction of the ordered layers of water molecules structured around the alkyl chains, by the electrostatic interactions among the ionized polar groups oriented in the adsorbed layer and by the hydrophobic interactions among the hydrophobic chains and the oil phase.

The understanding of the adsorption kinetics and of adsorption mechanism of these biocompounds at fluid interfaces is important for the description of their dynamic surface properties. Nevertheless, the adsorption of these biocompounds at the oil/water interface might have an important role in vivo in various biological systems and in different pharmaceutical and biomedical processes.

REFERENCES

1. Tomoaia-Cotisel, M., Zsako, J., Tomoaia, Gh., Mocanu, A., Pop, V.-D., and Chifu, E., *Rev. Roumaine Chim.* **49**, 443 (2004).
2. Tomoaia-Cotisel, M., and Joos, P., *Rev. Roumaine Chim.,* **49**, 539 (2004).

3. Joos, P., Tomoaia-Cotisel, A., Sellers, A. J., and Tomoaia- Cotisel, M., *Colloids Surfaces. B. Biointerfaces* **37**, 83 (2004).
4. Tomoaia-Cotisel, M., Zsako, J., Mocanu, A., Salajan, M., Racz, Cs., Bran, S., and Chifu, E., *Stud. Univ. Babes-Bolyai, Chem.* **48***,* 201 (2003).
5. Babak, V. G., and Boury, F., *Colloids Surfaces A: Physicochem. Eng. Aspects* **243**, 33 (2004).
6. Ravera, F., Ferrari, M., and Liggieri, L., *Adv. Colloid Interface Sci.* **88**, 129 (2000).
7. Eastoe, J., and Dalton, J. S., *Adv. Colloid Interface Sci.* **85**, 103 (2000).
8. Ward, A.F.H., and Tordai, L., *J. Chem. Phys.* **14**, 453 (1946).
9. Joos, P., Bleys, G., and Petre, G., *J. Chim. Phys*. **79**, 387 (1982).
10. Bleys, G., and Joos, P., *J. Phys. Chem.* **89**, 1027 (1985).
11. Joos, P., and Serrien, G., *J. Colloid Interface Sci.* **127**, 97 (1989).
12. Fainerman, V. B., Zholob, S. A., Miller, R., and Joos, P., *Colloids Surf. A* **143**, 243 (1998).
13. Van den Bogaert, R., and Joos, P., *J. Phys. Chem*. **83**, 2244 (1979).
14. Van den Bogaert, R., and Joos, P., *J. Phys. Chem.* **84,** 190 (1980).
15. Rillaerts, E., and Joos, P., *J. Colloid Interface Sci.* **88**, 1 (1982).
16. Coltharp, K. A., and Franses, E. I., *Colloids Surf. A* **108**, 225 (1996).
17. Theander, K., and Pugh, R. J., *J. Colloid Interface Sci.* **239**, 209 (2001).
18. Czichocki, G., Makievski, A.V., Fainerman, V. B., and Miller, R., *Colloids Surf. A* **122**, 189 (1997).
19. Vermeulen, M., and Joos, P., *Colloids Surf.* **33**, 337 (1988).
20. Liggieri, L., Ravera, F., and Passerone, A., *J. Colloid Interface Sci*. **169,** 226 (1995).
21. Liggieri, L., Ravera, F., and Passerone, A., *J. Colloid Interface Sci.* **169,** 238 (1995).
22. Li, J., Miller, R., and Mohwald, H., *Colloids Surf. A* **114**, 113 (1996).
23. Li, J., Fainerman, V. B., and Miller, R., *Langmuir* **12**, 5138 (1996).
24. Beverung, C. J.**,** Radke, C. J., and Blanch, H. W., *Biophys. Chem.* **81**, 59 (1999).
25. Baret, J. F., *J. Chim. Phys*. **65**, 895 (1968).
26. Chifu, E., Salajan, M., Demeter-Vodnár, J., and Tomoaia-Cotisel, M., *Rev. Roum. Chim.* **32**, 683 (1987).
27. Chifu, E., Tomoaia-Cotisel, M., Andrei, Z., and Bonciu, E., *Gazz. Chim. Ital.,* **109**, 365 (1979).
28. Chatterjee, J., and Wasan, D. T., *Chem. Eng. Sci*. **53**, 2711 (1998).
29. Liggieri, L., Ravera, F., and Passerone, A., *Colloids Surf. A* **114**, 351 (1996).
30. Ravera F., Liggieri, L., and Steinchen, A., *J. Colloid Interface Sci.* **156,** 109 (1993).
31. Yousef, A., and McCoy, B. J., *J. Colloid Interface Sci.* **94,** 497 (1983).
32. Tomoaia-Cotisel, M., Zsako, J., Mocanu, A., Lupea, M., and Chifu, E., *J. Colloid Interface Sci.* **117**, 464 (1987).
33. Zsako, J., Tomoaia-Cotisel, M., Chifu, E., Mocanu, A., and Frangopol, P. T., *Biochim. Biophys. Acta* **1024**, 227 (1990).
34. Tomoaia-Cotisel, M., *Progr. Colloid Polym. Sci.* **83**, 155 (1990).
35. Tomoaia-Cotisel, M., and Cadenhead, D. A., *Langmuir* **7,** 964 (1991).
36. Asgharian, B., Cadenhead, A. D., and Tomoaia-Cotisel, M., *Langmuir* **9**, 228 (1993).
37. Zsako, J., Tomoaia-Cotisel, M., Chifu, E., Mocanu, A., and Frangopol, P. T., *Gazz. Chim. Ital.,* **124**, 5 (1994).
38. Hata, T., Matsuki, H. and Kaneshina S., *Biophys. Chem.,* **87**, 25 (2000).
39. Frangopol, P.T. and Mihailescu, D., *Colloids Surfaces. B. Biointerfaces*, **22,** 3 (2001).
40. Davies, J. T. and Rideal, E. K., Interfacial Phenomena, Second Edition, Academic Press, New York, 1963, Chapter 4, pp. 154-216.
41. Miller, R., Joos, P., and Fainerman, V. B., *Adv. Colloid Interface Sci.* **49**, 249 (1994).
42. Zsako, J., Tomoaia-Cotisel, M., Albu, I., Mocanu, A., Chifu, E. and Frangopol, P. T., *Rev. Roum. Biochim.,* **28**, 33 (1991).
43. Chifu, E., Tomoaia, M., and Ioanette, A., *Gazz. Chim. Ital.* **105**, 1225 (1975).