TABLE I

Analysis, M.P. and conductance of the zinc (II) complexes with 25 acetyl pyridine

<table>
<thead>
<tr>
<th>Formula</th>
<th>M.P. °C</th>
<th>Conductance in mhos</th>
<th>% Metal Found</th>
<th>% Metal Calculated</th>
<th>% Halogen sulfur Found</th>
<th>% Halogen sulfur Calculated</th>
</tr>
</thead>
<tbody>
<tr>
<td><a href="ClO%E2%82%84">ZnL₂</a>₂</td>
<td>210</td>
<td>250</td>
<td>8.75</td>
<td>8.70</td>
<td></td>
<td>(S) 9.4</td>
</tr>
<tr>
<td><a href="SnCl%E2%82%83">ZnL₂</a>₂</td>
<td>158</td>
<td>166</td>
<td>9.70</td>
<td>9.80</td>
<td></td>
<td>(S) 9.4</td>
</tr>
<tr>
<td><a href="NCO">ZnL₂</a>₃</td>
<td>190</td>
<td>35</td>
<td>16.01</td>
<td>16.6</td>
<td></td>
<td>(S) 9.4</td>
</tr>
<tr>
<td>[ZnL₂]Br₂</td>
<td>185</td>
<td>6</td>
<td>13.65</td>
<td>13.99</td>
<td></td>
<td>(S) 9.4</td>
</tr>
</tbody>
</table>

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B. K. Patel.

September 28, 1976.

4. — and —, Inst. of Chemists (India), 1970, 5, 199.

STUDIES ON CHOLINESTERASE ACTIVITY IN THE PLASMA, RBC AND BRAIN OF STREPTOZOTOCIN TREATED RATS

Acetylcholinesterase (acetylcholine hydrase, EC 3.1.1.7) is present in many animal tissues and besides functioning as a neural transmitter it also aids in the mechanism of cell permeability and osmotic fragility, specially in the red blood cells (RBC). A decreased cholinesterase (ChE) activity was noted in the liver and blood of rats maintained on an atherogenic diet. A similar thing was reported in undernourished states. An increase of serum ChE activity was noted in albino rats and dogs in which diabetes was induced either by alloxan treatment or by pancreatectomy. The present communication reports observations made during studies conducted to determine the variations in ChE levels in rats in which diabetes was induced by the administration of streptozotocin.

The test rats (male albino rats of the Wistar strain) were injected intravenously streptozotocin in citrate buffer (pH 4.5), at a dose of 60 mg/kg body weight, while the control rats received only injections of the buffer. The test rats were given 2% glucose during the first 48 hours of injection. After this, both test rats and control rats were given feed and water ad libitum. The rats were sacrificed four weeks after the injection. Plasma and RBC were separated by centrifugation at 2000 RPM in a refrigerated centrifuge. The packed RBC were washed twice with 0.9% saline and finally resuspended in the same solution. The brain samples were homogenized in distilled water to a final concentration of 10%. AcetylChE and butyrylChE were assayed in plasma, RBC and brain by the method of Hestrin. Protein was estimated by the method of Lowry et al. and blood sugar by the method of Nelson and Somogyi.

Tables I and II summarize the results obtained. It was noted that (compared to the controls) the activities of both ChE were elevated in plasma as well as RBC of the test rats. No significant differences were noted between the test and control rats in any of the ChE in brain samples. The increase of blood ChE activity in pancreatectomized dogs was attributed by Fokin as due to impaired liver function and this was confirmed by Mc Daniel et al. who noted that alloxan diabetic rats who showed impaired conversion of L-tryptophan to niacin showed this conversion after treatment with insulin. The toxic effects of strepto-

Animal weights, tissue weights and blood sugar in normal and streptozotocin diabetic rats

(Values are averages of six rats and standard deviations)

<table>
<thead>
<tr>
<th></th>
<th>Normal rats</th>
<th>Streptozotocin diabetic rats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat (g)</td>
<td>247±12</td>
<td>190±13</td>
</tr>
<tr>
<td>Brain (g)</td>
<td>1.46±0.21</td>
<td>1.32±0.34</td>
</tr>
<tr>
<td>Liver (g)</td>
<td>6.32±0.73</td>
<td>4.98±0.70</td>
</tr>
<tr>
<td>Kidney (g)</td>
<td>1.53±0.28</td>
<td>1.21±0.35</td>
</tr>
<tr>
<td>Blood sugar (mg%)</td>
<td>72±8</td>
<td>298±30</td>
</tr>
</tbody>
</table>
**Table II**

Cholinesterase activity in plasma and RBC of control and streptozotocin-treated rats

<table>
<thead>
<tr>
<th>Enzyme activity*</th>
<th>Control rats</th>
<th>Test rats</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Plasma</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AchE</td>
<td>4.4 ± 1.7</td>
<td>57.6 ± 6.5</td>
</tr>
<tr>
<td>BchE</td>
<td>2.0 ± 1.4</td>
<td>32.5 ± 7.0</td>
</tr>
<tr>
<td><strong>RBC</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AchE</td>
<td>4.5 ± 1.7</td>
<td>55.8 ± 28.7</td>
</tr>
<tr>
<td>BchE</td>
<td>2.2 ± 4.1</td>
<td>38.4 ± 9.8</td>
</tr>
</tbody>
</table>

* Expressed as micromoles of substrate utilized/mi-hour.

AchE, acetylcholinesterase; BchE, Butyrlycholinesterase; *p < 0.05 and *p < 0.01—Student’s *t* test.

zotocin were noted to last only for 48 to 72 hrs after the injection, which indicated that a different mechanism might be operating to account for elevated plasma and RBC ChE activities in the case of these rats unlike the alloxan diabetic rats. It is interesting to note that Keplý reported alteration in ChE activity of RBC of human subjects with protein caloric malnutrition. In order to check whether any such mechanism is operating in streptozotocin-treated rats, further studies are in progress.

The authors gratefully acknowledge the help and encouragement rendered by the late Prof. V. G. Harwalne. They are also thankful to Dr. C. H. Chakrabarti for his interest in the work and to the C.S.I.R. for financial assistance.


**KNUDSEN DIFFUSION AND NEGATIVE ACTIVATION ENERGY OF ADSORPTION**

Negative activation energy in chemisorption of gases on solids has been observed by many workers like Sastri and Ramanathan, Taylor and Lian, Kubokawa and Taylor, Low and Taylor and by Decrue and Susz. In most cases, surface heterogeneity effects have been proposed to account for this. In our case we observed also the occurrence of negative activation energy in the temperature range 31°C–253°C for oxygen adsorption on copper vanadate pretreated with hydrogen at 40 mm pressure and 130°C and we propose a Knudsen diffusion model to account for this behaviour.

In 1953, Sutherland and Winfield have developed a mathematical model for Knudsen diffusion for rapid gas adsorption on porous solids and in 1965, Peers has shown that such a model gives rise to an Elovich rate equation in a constant volume system. However no further work has been carried out in this aspect.

The conditions for the Knudsen diffusion being the rate-controlling step (according to these authors) are:

1. Pressure should be less than 2 atm.
2. Pore radius should be < 100 Å.
4. Rapid establishment of equilibrium between the pore wall and the gas molecules.
5. Adsorption taking place in a constant volume system.

For such conditions, Sutherland, and Winfield have deduced

$$\frac{dp}{dt} = \frac{p_0 Y}{\pi^{1/2}}$$

(1)

where,

$$p_0 = \text{initial pressure},$$

$$Y = \frac{D_k \pi^{1/2} b^{1/2}}{V D^{1/2}}$$

(2)

$b = \text{No. of cylindrical pores, each of length 1,}$

$V = \text{Volume of system,}$

$r = \text{Average pore radius,}$

$D_k = \text{Knudsen diffusion constant}$

$$\approx 1.33 r \left(\frac{2RT}{\pi M}\right)^{1/2}$$

(3)

$D_k/D^{1/2} = 1.94 k_{1/2} (RT)^{3/2} (\pi M)^{1/2}$

$k_e = \text{Intrinsic adsorption coeff.}$

Now,

$$\frac{d \ln K_p}{dT} = \frac{\Delta H}{RT^2}$$

Let $\Delta H = -Q$ where $Q$ is the heat of adsorption (it is negative since the adsorption is exothermic). So,

$$\frac{d \ln k_e}{dT} = -\frac{Q}{RT^2}$$