The regulation of the rate of mitochondrial oxidative phosphorylation and arsenylation was studied at two external free Ca²⁺ concentrations. The rate of arsenate-stimulated respiration in absence of added ADP was not affected by external 10⁻⁸ and 10⁻⁶ M Ca²⁺ levels or carboxyatractyloside, while state 3 respiration was profoundly modified. In addition, the kinetic analysis showed that the rate of arsenylation in the presence of ADP was more efficient (Vₘ/Kₘ, ratio 3.5 times higher) in the catalytic process than phosphorylation. Therefore, this suggests that the activity of the ATP/ADP carrier is importantly controlled by Ca²⁺. The evaluation of the control in phosphorylation showed that the flux-control coefficients (Cᵢ) exerted by the ATP/ADP carrier (ranged between 0.23 and 0.48) and the ATP synthase (0.05–0.57) were modified in a reciprocal way by Ca²⁺ and Pᵢ concentrations. This suggests that these two enzymes are coupling sequentially through a common intermediate, the intramitochondrial ATP/ADP ratio. Other important steps controlling phosphorylation were the b-c₂ complex (Cᵢ = 0.30) and the cytochrome oxidase (Cᵢ = 0.23) but they were not modified by Ca²⁺. It was also found that the main step controlling arsenylation was the ATP synthase (Cᵢ = 0.74). The increment in the inorganic arsenate concentration induced a diminution in the control exerted by the ATP synthase (from 0.73 to 0.56). The results suggest that Ca²⁺ and Pᵢ (or inorganic arsenate) could be regulated by ATP synthesis through an activating effect on ATP/ADP carrier and/or ATP synthase.

The regulation of steady-state mitochondrial oxidative phosphorylation has been a problem of interest for several years. On the basis of studies on the effect of specific inhibitors and variations in the ADP/ATP and ATP/(ADP) × (Pᵢ) ratios on the rate of ATP synthesis, it has been proposed that the adenine nucleotide translocator is the rate-limiting step of oxidative phosphorylation (1-4). In contrast, through a thermodynamic approach, it was postulated that cytochrome oxidase is an important control point of oxidative phosphorylation, since it was observed that sites 1 and 2 of oxidative phosphorylation were in near-equilibrium with the extramitochondrial phosphorylation potential (5, 6). However, other studies showed that the translocator was approximately 2 kcal/mol out of equilibrium (7), and, more recently, the conclusions reached from the aforementioned thermodynamic studies were critically evaluated (8). In addition, using the theory of metabolic control developed by Kacser and Burns (9, 10) and Heinrich and Rapoport (11, 12), Tager and coworkers (13, 14) showed that control of oxidative phosphorylation is exerted at multiple points, the most important being the adenine nucleotide translocator, the dicarboxylate carrier, and cytochrome oxidase.

It has been described that the level of intramitochondrial Ca²⁺ affects the rate of oxidative phosphorylation (15-21). Apparently this effect of Ca²⁺ involves a change in the degree of kinetic control exerted by the various steps of oxidative phosphorylation (22); nevertheless, at two different rates of oxidative phosphorylation, it was found that the adenine nucleotide translocator is an important point of control (22). With respect to how intramitochondrial Ca²⁺ levels regulate the various steps involved in oxidative phosphorylation, it was considered of interest to evaluate the multiple control points in the absence of the contribution exerted by the adenine nucleotide translocator. Such a system could be the arsenate-stimulated respiration (23-26), the rationale being that arsenate that enters the mitochondria through the merosal-sensitized phosphate carrier (27) arsenylates internal ADP via the ATP synthase using the H⁺ gradient generated by the respiratory chain (27-30). As ADP-arsenate is rapidly hydrolyzed, an internal cycle of ADP-arsenate synthesis and hydrolysis would take place. Thus, in this system, the arsenate-stimulated respiration would not involve the function of the translocator. In this work, the control strength of all the steps involved in oxidative phosphorylation and arsenylation was quantitated at two different rates of oxygen uptake as controlled by the external Ca²⁺ level.

**METHODS**

The preparation of rat liver mitochondria with a medium containing 250 mM sucrose, 5 mM HEPES,¹ 0.5 mM EGTA, pH 7.0, the determinations of oxygen uptake (by means of a Clark-type electrode) and protein, and the use of Ca²⁺-EGTA buffers were described in a previous work (22). Mitochondria (1 mg/ml) were incubated in a basic medium containing 120 mM KCl, 30 mM HEPES, 2 mM MgCl₂, 5 mM succinate, 2 mM EGTA, pH 7.20, at 30 °C. Other experimental conditions are detailed in the legends to the tables and figures.

¹The abbreviations used are: HEPES, 4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid; As₁, inorganic arsenate; Cᵢ, flux-control coefficient; EGTA, ethyleneglycol bis(β-aminoethyl ether)-N,N,N',N"-tetraacetic acid; FCCP, carbonyl cyanide 4-trifluoromethoxyphenylhydrazone; HQNO, 2-hexyl-4-hydroxyquinoline-N-oxide.
Control of Phosphorylation and Arsenylation

**RESULTS**

**Effect of Carboxyatractylloside on Oxidative Arsenylation**—The mechanism through which arsenate stimulates mitochondrial respiration is visualized as the formation of ADP-arsenate through the ATP synthase and its subsequent spontaneous internal hydrolysis (27–30). Nevertheless, the addition of ADP to the system increases the rate of the arsenate-stimulated respiration (24–26), which suggests that the metabolic cycle exhibits a certain dependence on the translocation of adenine nucleotide across the membrane. Therefore, the sensitivity of oxidative phosphorylation and arslenylation to carboxyatractylloside, a specific irreversible inhibitor of the translocator (31, 32), was assayed in mitochondria incubated for 5 min with 10⁻⁹ M external Ca²⁺ (Table I), which is assumed to be much lower than physiological concentrations of Ca²⁺ (33). As discussed in previous work in this condition (22), the amount of intramitochondrial Ca²⁺ is lowered from 20 to 11 nmol mg⁻¹, which is near to the possible physiological range of intramitochondrial Ca²⁺ of 1–5 nmol/mg of mitochondrial protein determined in entire cells (34). On the other hand, arslenylation in the absence of ADP was completely insensitive to carboxyatractylloside. In the presence of ADP, the rates of phosphorylation and arslenylation were diminished by carboxyatractylloside, but as expected the former was much more sensitive. These findings indicate that phosphorylation fully depends on translocator activity, while arslenylation is only partially dependent.

The increase in the rate of the arsenate-stimulated respiration as induced by ADP suggests that either intramitochondrial ADP concentration is limiting for respiration or alternatively, as phosphate is competitive with arsenate (23, 24, 26), the phosphorylation of ADP lowers the internal phosphate concentration, thus allowing full expression of the action of arsenate. These possibilities were explored by assaying the rate of arsenate-stimulated respiration of mitochondria preincubated for 5 min prior to the addition of arsenate in the usual conditions (Table I). The rate of respiration stimulated by 2 mM As₁ in the absence of added ADP and Pi was 129 nanomols min⁻¹ mg⁻¹. As₁ added to mitochondria after 15 s of incubation with 4 μM ADP induced a rate of 146. In mitochondria preincubated for 5 min with hexokinase, glucose, and 4 μM ADP, in which endogenous phosphate would have been converted to glucose 6-phosphate, As₁ induced respiration to a value of 143. Thus, a lowering of the internal phosphate concentration did not modify the effect of ADP on the As₁-stimulated respiration, which may suggest that the translocation of ADP per se may be involved in the increase by ADP of the As₁-stimulated respiration. The Km for ADP in the arsenate-stimulated respiration in the absence of hexokinase was 13.2 μM (data not shown). This was calculated by plotting the increase of As₁-stimulated respiration by various concentrations of added ADP.

**Effect of Ca²⁺ on the Arsenate-stimulated Respiration**—The level of internal Ca²⁺ as regulated by the external Ca²⁺ concentration controls the rate of oxidative phosphorylation (22). Therefore, it was considered of interest to study whether Ca²⁺ levels affect the arsenate-stimulated oxygen uptake. In agreement with previous findings (22), it was found that, at a concentration of 10⁻⁸ M external Ca²⁺, the ADP-stimulated respiration decreases with time (Table II); with 10⁻⁹ M external Ca²⁺ in a 5-min period, the decrease in respiration is much lower. In the presence of ADP and arsenate, a similar pattern is observed, except that the decrease in respiration is much lower than in the presence of phosphate. In contrast, the arsenate-stimulated respiration in the absence of added ADP remains essentially unchanged regardless of the concentration of Ca²⁺ introduced into the mixture. Also, it is to be noted that, after a 5-min incubation period with 10⁻⁹ M external Ca²⁺, which lowers the level of internal Ca²⁺ (22), the rate of respiration in the presence of ADP and arsenate is higher than that obtained with ADP and phosphate. The data of Table II also indicate that the rate of oxidative phosphorylation is 20% faster than the rate of arslenylation after a 1-min preincubation, which is in accord with previous reports (28, 29, 35–37). However, when mitochondria were preincubated for 5 min at 10⁻⁸ M Ca²⁺, the rate of respiration in the presence of ADP and As₁ is 27% higher than that obtained with ADP and phosphate. Therefore, the data show that the rate of respiration is modified by the level of extramitochondrial Ca²⁺ only in conditions in which ADP determines the rate of respiration. These results suggest that Ca²⁺ levels modify mainly the functioning of the adenine nucleotide translocator.

**Catalytic Constants for Oxidative Phosphorylation and Arsenylation**—A double reciprocal plot of the rate of respiration at various concentrations of phosphate and ADP is shown in Fig. 1. In the Lineweaver-Burk plot, two clearly distinguishable slopes were detected; in a kinetic study of oxidative phosphorylation, Stoner and Sirak (38) noted a similar downward curve. These authors interpreted these findings as different affinities for phosphate during oxidative phosphorylation. Using the second slope of the data in Fig. 1, the kinetic parameters depicted in the figure were calculated. Apparently, oxidative phosphorylation has a 14% higher catalytic capacity than arslenylation in the absence of added ADP. In contrast, arslenylation plus ADP shows a higher Vmax and a km for arsenate 70% lower than that for phosphate in oxidative

---

**Table I**

Inhibition of oxidative phosphorylation and arslenylation by carboxyatractylloside

<table>
<thead>
<tr>
<th>Carboxyatractylloside</th>
<th>Oxidative phosphorylation</th>
<th>Oxidative arslenylation (+ ADP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pmol mg⁻¹</td>
<td>nanomols O mg⁻¹ min⁻¹</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>136</td>
<td>116</td>
</tr>
<tr>
<td>50</td>
<td>127</td>
<td>115</td>
</tr>
<tr>
<td>167</td>
<td>17</td>
<td>196</td>
</tr>
</tbody>
</table>

Mitochondria were preincubated in the conditions described in the legend to Table I, except that the concentration of Ca²⁺ and the time of preincubation were as indicated.

**Table II**

Rate of respiration stimulated by ADP and As₁ at two Ca²⁺ concentrations

<table>
<thead>
<tr>
<th>Addition</th>
<th>10⁻⁸ M Ca²⁺</th>
<th>10⁻⁹ M Ca²⁺</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 min</td>
<td>5 min</td>
<td>1 min</td>
</tr>
<tr>
<td>As₁</td>
<td>122</td>
<td>125</td>
</tr>
<tr>
<td>As₁ (+ ADP)</td>
<td>209</td>
<td>183</td>
</tr>
<tr>
<td>P₁ + ADP</td>
<td>252</td>
<td>144</td>
</tr>
</tbody>
</table>

*Preincubation time.
phosphorylation, and the catalytic constant \( V_m/K_m \) is 3.5 times higher in arsenylation (+ ADP) than in oxidative phosphorylation.

**Evaluation of Multiple Control Points of Oxidative Phosphorylation and Arsenylation**—The quantitative analysis of control of a metabolic pathway (9-12) introduced by Tager and co-workers (13, 14) for mitochondrial ATP synthesis was used here. The values of flux-control coefficient (see Ref. 39 for new terminology in the metabolic control theory) for the adenine nucleotide translocator and the ATP synthase of mitochondria incubated in \( 10^{-4} \) and \( 10^{-3} \) M external Ca**2⁺** were described before (22). These results were confirmed (Table III). It should be noted that the concentration of Ca**2⁺** modulates in a reciprocal form the flux-control coefficient exerted by the adenine nucleotide translocator and the ATP synthase. In addition, it is to be noted that cytochrome oxidase and b-c₁ complex also contribute in an important form to the control of the rate of ATP synthesis, but that the value is not modified by external Ca**2⁺** concentration.

The titration curves from which the values of Table III were calculated are shown in Fig. 2. For reasons unknown, the values obtained for the b-c₁ complex and the dicarboxylate carrier are significantly different from those obtained by Tager and co-workers (13, 14). In different experiments, the \( C_i \) values for \( 10^{-6} \) M Ca**2⁺** exerted by the b-c₁ complex using HQNO were 0.40, 0.33, and 0.46. At \( 10^{-6} \) M Ca**2⁺**, values were 0.34, 0.27, and 0.26. Titration with antimycin, another inhibitor of the b-c₁ complex (48), also gave a high value of the flux-control coefficient. Malate was considered a specific inhibitor of the dicarboxylate carrier on the rationale that mitochondria in the presence of rotenone would be unable to oxidize malate, and that malate could compete with the influx of succinate (43, 44). In different experiments on the effect of malate, the values obtained for the flux-control coefficient exerted by the dicarboxylate carrier at \( 10^{-9} \) M Ca**2⁺** were 0.04 and 0.03, and at \( 10^{-8} \) M Ca**2⁺**, 0.11 and 0.04. The values determined for this carrier using mersalyl, phenylsuccinate, and n-butyralonate confirmed that the dicarboxylate carrier exerted a low control of the rate of oxidative phosphorylation.

From titrations with oligomycin, it has been reported (49, 50) that the ATP synthase does not exert significant control of state 3 respiration (however, see Ref. 51). In our experimental conditions and using also oligomycin, this seems to be the case at \( 10^{-9} \) M Ca**2⁺**, but at \( 10^{-9} \) M Ca**2⁺**, the contribution of the ATP synthase is high (Table III). In various experiments, the values obtained at \( 10^{-9} \) M Ca**2⁺** were 0.32 and 0.31, while at \( 10^{-8} \) M Ca**2⁺**, the values obtained ranged between 0.01 and 0.14.

The flux-control coefficients of several of the reactions involved in oxidative arsenylation were also calculated from titrations with various inhibitors (the titration curves are shown in Fig. 3 and the results of calculations of the flux-control coefficient in Table IV). Noteworthy is that the most important point of control of this process is the ATP synthase, notwithstanding the presence of ADP or external Ca**2⁺**. In addition, it is of interest to note that the contribution of the ATP synthase to the arsenate-stimulated respiration is about 3 times and approximately 12 times higher at \( 10^{-4} \) and \( 10^{-6} \) M Ca**2⁺**, respectively, than its contribution in oxidative phosphorylation (compare data of Tables III and IV). The contribution of the other metabolic steps to the control of the arsenate-stimulated respiration was similar to that detected in oxidative phosphorylation, except for the case of b-c₁ complex, which was lower in the case of the arsenate-stimulated oxygen uptake.

Up to this point, the results suggest that there is a reciprocal relation in the control of oxidative phosphorylation between the adenine nucleotide translocator and the ATP synthase. Indeed, by poising the rate of oxidative phosphorylation at two different steady-state levels, it was observed that the contribution of the translocator and the ATP synthase varied in opposite directions. Moreover, without a functional adenine nucleotide translocator, as in the case of the arsenate-stimulated respiration, the main point of control is the ATP synthase. Therefore, it was of interest to estimate the control
exerted by the two enzymes at different steady-state rates of oxidative phosphorylation. The rates of arsenylation and phosphorylation were adjusted to different levels by varying the concentration of phosphate or arsenate introduced into the mixture; at each of these concentrations, titrations with carbonylactylxoside and oligomycin were carried out. From the slope of the inhibition curve the flux-control coefficient was calculated. The results (Table V) showed that, as the rate of phosphorylation increased, the contribution of the translocator increased, while that of the synthase diminished. In the arsenylation reaction, it was observed that, as the rate of respiration increased, the contribution of the synthase to the regulation of the process also diminished. It should be noted that, at all levels of respiration, the control exerted by the synthase is significantly higher for the arsenylation than for the phosphorylation reaction.

DISCUSSION

It has been repeatedly observed (26, 28, 29, 35-37) that the rate of respiration during ATP synthesis is higher than that
induced by As in the presence or absence of ADP. This observation was confirmed, but it was observed that, in mitochondria incubated for 5 min with 10^{-9} M external Ca^{2+}; the rate of oxidative phosphorylation declined (Table II and Ref. 22) to a point in which the arsenate-stimulated respiration became higher. The diminution of the rate of oxidative phosphorylation correlates with a drop in the level of internal Ca^{2+} (22). In agreement with previous conclusions (17, 21, 22, 52, 53), it would appear that a main effect of Ca^{2+} levels on mitochondrial phosphorylation is on the activity of the adenine nucleotide translocator, since the arsenate-stimulated respiration was not affected by variations in Ca^{2+} levels.

The quantitation of the flux-control coefficient exerted by the various metabolic steps involved in oxidative phosphorylation at two different steady-state rates as determined by the concentration of external Ca^{2+} showed that only the control exerted by the adenine nucleotide translocator and the ATP synthase underwent significant variations; more-

**Table IV**

Flux-control coefficient of various steps of oxidative arsenylation

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>10^{-4} M Ca^{2+}</th>
<th>10^{-6} M Ca^{2+}</th>
<th>Inhibitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP synthase</td>
<td>0.74</td>
<td>0.60</td>
<td>Oligomycin</td>
</tr>
<tr>
<td>Cytochrome</td>
<td>1.20</td>
<td>1.00</td>
<td>Azide</td>
</tr>
<tr>
<td>b-cy complex</td>
<td>0.00</td>
<td>0.02</td>
<td>HQNO</td>
</tr>
<tr>
<td>As carrier</td>
<td>0.12</td>
<td>0.10</td>
<td>P, (C)</td>
</tr>
<tr>
<td>Carboxyatractyloside</td>
<td>0.05</td>
<td>0.10</td>
<td>N-ethylmaleimide</td>
</tr>
</tbody>
</table>

\[ \sum C_i = 0.94 - 1.15 \]

**Table V**

Contribution of the translocator of adenine nucleotides and the ATP synthase to the control of oxidative phosphorylation and arsenylation at different steady-state rates

Mitochondria were incubated at 10^{-9} M Ca^{2+} in the basic medium detailed in the legend to Table I. Arsenate-induced phosphorylation was started by the addition of 2 mM As. Where indicated, 200 M ADP was added 20 s before As.

over, opposite variations in the control exerted by the synthase and the translocase were also observed when the rate of phosphorylation was modified by variations in the concentration of phosphate. In agreement with previous considerations (58, 54), the observed reciprocal changes that occur with the adenine nucleotide translocator and the ATP synthase as induced by Ca^{2+} or phosphate suggest that there is a strong functional coupling between the two enzymes, i.e. an activation of the synthase would lead to an inverse shift in the kinetic control exerted by the translocase and the synthase.

The theory of metabolic control (9–12) describes a quantitative relation between the flux-control coefficients and the elasticity coefficients (39) of adjacent enzymes through common metabolites; the elasticity coefficient of an enzyme being defined as the "local" response of the rate to a change in the concentration of one of the intermediates (10, 11). Such relation was named the connectivity property (10):
The values obtained of the C/C ratios at two different Ca²⁺ and Pi concentrations are illustrated in Table VI. When the concentrations of Ca²⁺ or phosphate were increased, the rate of oxidative phosphorylation also augmented, and this is characterized by a higher value of the C/C ratio. The increment in the C/C ratio with the rate of oxidative phosphorylation indicates that the elasticity coefficient of the synthase (εₜ) increases and/or the elasticity coefficient of the translocator (εₜ) decreases. As it has been shown that in respiring mitochondria the intramitochondrial ATP/ADP ratio increases with the concentration of phosphate (3), it would appear improbable that the higher εₜ/εₜ ratio is due to a diminution in εₜ this is because the increase in the ratio occurs in a condition in which the existing higher ATP/ADP ratio would favor the activity of the translocator (however, see Ref. 55). Therefore, at this stage it would appear that the high C/C ratio at high rates of oxidative phosphorylation is due to or correlates with an increase in the elasticity coefficient of the synthase. In other words, it would seem that ATP synthase has a greater capacity of response with respect to variations in the intramitochondrial pool of adenine nucleotides.

There are reports that indicate that Ca²⁺ (20, 56) and phosphate (57) control the hydrolytic activity of the ATP synthase by modifying the interaction of the natural ATPase inhibitor protein with the ATP synthase in heart mitochondria and that these interactions affect the rate of ATP synthesis (58, 59). Moreover, phosphate and arsenate (60) modify per se the catalytic activity of the ATP synthase. Therefore, it is possible that an important point of control of oxidative phosphorylation and arsenylation is the regulation of the activity of the ATP synthase or the number of active enzymes as determined by the interaction of the enzyme with the inhibitor protein. Although the molecular events that occur at each of the steps of the metabolic pathways and which account for the overall rate cannot be ascertained at this moment, the present results are consistent with the idea (38) that there is a strong functional relation between the ATP synthase and the translocator, and that variations in the catalytic activity of the synthase influence the kinetic control exerted by the translocase. The role of the inhibitor protein in the modulation of the rates of oxidative phosphorylation and its kinetic control in whole mitochondria is a problem that merits detailed studies.

Finally, two points deserve some comment. The first is the present inability to explain the different flux-control coefficient exerted by the b-citrate complex on oxidative phosphorylation and arsenylation. The second is that, in the theory of control proposed by Kacser and Burns (9, 10) and Heinrich and Rapoport (11, 12), the sum of the contributions of the individual steps to the regulation of a metabolic pathway should be a unit; this has been referred to as the summation theorem (9, 10). However, in the present studies, the sum of the control exerted by the individual steps of oxidative phosphorylation was found to be higher than one. The possibilities for obtaining higher values have been discussed (61), and it was proposed that substrate inhibition, expansion of the system, and branched pathways could account with negative contributions to the control of the flow, i.e. reactions that affect the pathway by a lateral draining of the main flux.

In the present experimental conditions, product inhibition would be difficult to postulate, while the expanding system applies to a growing system where masses and volumes increase exponentially. On the other hand, the existence of a branched pathway may well exist in mitochondria, and this could be the passive permeability of the inner membrane to protons. It should be noted that, if respiration is evaluated, H⁺ permeability would exert a positive control, but as it has been assumed that rates of state 3 respiration correspond to phosphorylation rates, passive H⁺ permeability would exert a negative contribution to the regulation of oxidative phosphorylation. The passive H⁺ permeability of mitochondria was evaluated according to Groen et al. (13). This method estimates membrane H⁺ leakage through titration of respiration with FCCP and extrapolation to zero respiration. The intrinsic proton diffusion was calculated to be 11-19 nM FCCP in state 4 conditions. With the latter value and results of titrations with FCCP on state 3 and arsenylation rates, the degree of control of H⁺ permeability during the operation of oxidative phosphorylation and arsenylation was calculated and ranged between 0.04 and 0.12 and 0.11 and 0.23, respectively. Thus, it would appear that an important point with a negative contribution to the overall control of ATP synthesis is the intrinsic permeability of the mitochondrial membrane to H⁺.

**Acknowledgments**—I gratefully acknowledge helpful discussions with Dr. A. Gómez-Puyou from Departamento Bioenergetica, Centro de Investigaciones en Fisiología Celular, Universidad Nacional Autónoma de México. The collaboration of Guadalupe Ramírez in typing the manuscript is acknowledged.

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**Table VI**

The C/C ratios of the rate of oxidative phosphorylation

<table>
<thead>
<tr>
<th></th>
<th>2 mM P&lt;sub&gt;i&lt;/sub&gt;</th>
<th>10°C M Ca&lt;sup&gt;2+&lt;/sup&gt;</th>
<th>0.5 mM P&lt;sub&gt;i&lt;/sub&gt;</th>
<th>5.0 mM P&lt;sub&gt;i&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>C&lt;sub&gt;T&lt;/sub&gt;</td>
<td>0.32</td>
<td>0.48</td>
<td>0.23</td>
<td>0.52</td>
</tr>
<tr>
<td>C&lt;sub&gt;T&lt;/sub&gt;</td>
<td>1.6</td>
<td>0.45</td>
<td>0.57</td>
<td>0.23</td>
</tr>
</tbody>
</table>