ON THE VELOCITY OF ENZYMATIC REACTIONS
IN MICHAELIS–MENTEN-LIKE SCHEMES (ENSEMBLE
AND SINGLE-MOLECULE VERSIONS)

In searching non-standard ways of conformational regulation, various Michaelis–Menten-
lke schemes attract relentless attention, resulting in sometimes too sophisticated consider-
ations. With the example of monomeric enzymes possessing an only binding site, we define
the minimal schemes capable of bearing peculiar regulatory properties like “cooperativity” or
substrate inhibition. The simplest ways of calculating the enzymatic reaction velocity are ex-
emplified, either in the ensemble or single-molecule case.

Keywords: enzymatic reactions, Michaelis–Menten schemes, monomeric enzymes, confor-
mational regulation, reaction velocity.

1. Introduction

The Michaelis–Menten (MM) scheme [1] serves as
a basis for the enzymatic kinetics for more than
a century [2, 3]. Early attempts to search for in-
ternal mechanisms of regulating the enzyme activ-
ity, rooted in the conformational lability, and cor-
responding deviations from the classical MM kinet-
ics (see [4] and references therein) were practically
ignored for a prolonged period [5]. Since the begin-
ing of the 2000s, however, the situation has been
changing dramatically [3], and nowadays there is no
lack of theoretical papers devoted to various MM-like
schemes. This is mainly conditioned by implementation
of the single-molecule (SM) methods into enzy-
mology [6], and by similarities to heterogeneous catal-
ysis [7, 8]. Undoubtedly, this activity will be only
intensified. That is why it is expedient to try as-
sessing the intermediate achievements on this way
in order to outline its further direction more thor-
oughly, and to avoid unnecessary deflections and over-
complications.

With this purpose, the present work is restricted
by the most basic example – precisely, by reac-
tions of monomeric enzymes with an only binding
site. In this generic case, it is especially clear which
new possibilities arise due to conformational fluctu-
ations of the enzyme, how transparent their physi-
cal nature is, how complex their (theoretical) anal-
ysis should be, and eventually, how new the re-
results of the latter are. In Section II, the difference
in the methods of calculations of the reaction velocity
in the ensemble and SM versions – the point that
often lacks for proper attention – is elucidated. In
Section III, the minimal MM-like schemes which
bear the characteristic regulation phenomena (non-
monotonic dependence of the velocity on the rate
of substrate release, cooperativity, and substrate in-
hibition) caused by the presence of conformational
channels are defined, and the simplest, as distinct
from frequently used, algorithm of calculating this ve-
locity is pointed out. Section IV contains concluding remarks.

2. The Classical Scheme (Ensemble and SM Versions)

This starting point of enzymatic kinetics reads:

\[ E + S \overset{a}{\underset{b}{\rightleftharpoons}} ES \overset{R}{\rightarrow} E + P, \tag{1} \]

where \( a \) is the rate constant of binding substrate \( S \) to enzyme \( E \), forming the enzyme-substrate complex \( ES \); \( b \) is the rate constant of the backward “unproductive” substrate release, and \( R \) is that of the catalytic stage of converting substrate \( S \) into product \( P \).

The kinetic equations to scheme (1) are based on the mass action law. For example, for concentration \([ES]\) of the complex, one has:

\[ \frac{d[ES]}{dt} = -(b + R) [ES] + a[E], \tag{2} \]

where the rate constant \( a \) is in fact of the pseudo-first order, as the binding reaction is bimolecular, and \( a \equiv k[S] \), where \( k \) is the second-order constant. Non-linearity of those equations is avoided due to the assumption that usually \([S]\) is much greater than \([E]\) (at least at the starting stages of the reaction) or maintained constant during many turnovers. Then Eq. (2) added with the normalization condition \([E_i] = [E] + [ES] \), where \([E_i]\) is the total enzyme concentration, turns into

\[ \frac{d[ES]}{dt} = -(b + R + a) [ES] + a[E_i], \tag{3} \]

so that the stationary complex concentration is

\[ [ES]_{st} = \frac{a[E_i]}{b + R + a} = \frac{[E_i][S]}{[S] + K_M}, \tag{4} \]

where \( K_M = (b + R)/k \), the known Michaelis’ constant. Of prime interest is the stationary velocity \( v \) of product formation, \( d[P]/dt \), which, according to Eqs. (1, 4), reads

\[ v = R[ES]_{st} = \frac{Ra[E_i]}{a + b + R} = \frac{R[E_i][S]}{[S] + K_M}. \tag{5} \]

The hyperbolic dependence \( v ([S]) \) (5) is the main result of scheme (1) and a primary test for investigations into any enzymatic reaction. With the progress in these investigations, the deviations from this dependence have been revealed even for some monomeric enzymes with the only binding site. This has eventually led to the development of various schemes of such kind of biochemical regulation, crucial for many physiological processes, see, e.g., [9]. All of them are related to the splitting of the pathway of scheme (1) by introducing a set of (at least two) conformational channels of the reaction, see Section III. So far, we concentrate on the analysis of scheme (1), when the reaction is traced on a single enzyme – i.e., in a way that nowadays becomes dominant. In this case, one and the same enzyme macromolecule works in a serial regime, converting a substrate into a product consecutively, one by one. Then the obtained arrays of the turnover times (up to tens of thousands of turnovers [10]) undergo statistical processing. The prime characteristic is the probability density \( f(t) \) of the first passage time, i.e. of that from the beginning to the end of a conversion act. Correspondingly, the kinetic equations related to scheme (1) are formulated not for concentrations but for probabilities \( P_i(t) \) of the residence in states \( i \in \{E, ES\} \). The quantity of the product released in the interval \((t, t + dt)\) is \( dP = f(t)dt \); therefore, \( f(t) = dP/dt = RP_{ES}(t) \), where \( P_{ES}(t) \) can be found from the set

\[ \begin{cases} \frac{dP_E}{dt} = -aP_E + bP_{ES}, \\ \frac{dP_{ES}}{dt} = aP_E - (b + R)P_{ES}, \end{cases} \tag{6} \]

with initial conditions \( P_E(0) = 1, \ P_{ES}(0) = 0 \).

At this point, it is necessary to make important remarks in order to avoid some misunderstandings occurring even in the best recent papers. Precisely, despite the similarities in appearance of, say, Eq. (2) and the second equation in (6), one has to bear in mind the obvious difference between the stationary problem formulation for the ensemble version and non-stationary(6) for the SM version. In the latter, in particular, there is no normalization conservation, since \( P_E(t) + P_{ES}(t) = 1 \) holds for \( t = 0 \) only, as it is obvious from Eq. (6). Moreover, \( P_{E,ES}(t \to \infty) \to 0 \) (in the first of Eqs. (6)), the term \( RP_{ES} \) of renewing \( P_E \) due to the return of the enzyme to its free state is absent, because this stage should not contribute to \( f(t) \) by definition, while it is mandatory in the stationary ensemble version! Nonetheless, in some works (e.g., [11–13]), wishing to avoid this rather natural difference, they introduce the stage \( E^0 \overset{\delta}{\rightarrow} E \) ad-
expression for \( f \) \[11\]. The mean first passage time is exponents which is given in many works, see, e.g., (sub)states and understand the content of new ef-

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Conformational Channels

3. Minimal Schemes of the Effects

Thus, solving set (6), one can obtain an explicit expression for \( f(t) \) as a difference of two time exponents which is given in many works, see, e.g., [11]. The mean first passage time is \( \langle t \rangle = \int_{0}^{\infty} t f(t) / dt \), and the inverse quantity \( 1 / \langle t \rangle \) serves as an analog of the reaction velocity \( v \). To calculate \( 1 / \langle t \rangle \), it is in fact sufficient to know the Laplace transform \( \tilde{f}(s) = \int_{0}^{\infty} f(t) e^{-st} / dt \), as \( \langle t^n \rangle = (-1)^n \int_{0}^{\infty} d f(s) / ds \) at \( s = 0 \). The Laplace transformation of set (6) gives \( \tilde{P}_{ES}(s) = a s^2 + s(a + b + R) + aR \) then \( \tilde{f}(s) = R \tilde{P}_{ES}(s) \), and \( \langle t \rangle = (a + b + R) / aR \), so that

\[
1 / \langle t \rangle = Ra / (a + b + R).
\] (7)

Comparing this with the ensemble result (5), we see their complete identity, if to adopt \( v / [E_i] = 1 / \langle t \rangle \). This identity has been checked experimentally [10], and Eq. (7) was named “single-molecule Michaelis–Menten equation” [11]. As it can be seen in the literature, its validity (which is considered as a version of the ergodic hypothesis) is not called in question even under complications of scheme (1) with confor-

Fig. 1. Generic schemes for the effects caused by introducing different conformational channels into the classical MM scheme (see the text)

3.1. Direct calculations of the velocity in the ensemble and SM versions

In the ensemble version, the stationary velocity can be easily found from the corresponding set of linear algebraic equations, with one of them being the conservation condition of the total enzyme concentration. In the SM version, however, one has to be more careful with calculation of this velocity and manifestation of the validity of \( v / [E_i] = 1 / \langle t \rangle \). Let us illustrate this, starting from the example of simplified scheme (c). To avoid cumbersome formulae, set \( \beta = 0 \), and also \( A = a \), \( B = b \). Then the only distinction of the channels is the difference in the catalytic rates, \( R > r \):

\[
E_1 \xrightarrow{a} ES_1 \xrightarrow{R} E_2 \\
\alpha \xrightarrow{a} \beta \\
\alpha \xrightarrow{a} \beta \\
E_2 \xrightarrow{b} ES_2 \xrightarrow{r} E_2
\] (8)

Actually, there are three such effects, and each of them can be realized within its minimal scheme, see Fig. 1.

Scheme (a) was recently proposed in [15]. It illustrates the effect of non-monotonic dependence of the velocity on the rate constant \( b \) of “unproductive” sub-

strate release under conditions that the catalytic rate constants \( r \) and \( R \) are essentially different. The effect takes place even in the absence of conformational fluctuations, and the hyperbolic dependence \( v ([S]) \) still holds. The latter can be replaced with a sigmoid de-

pendence \( v ([S]) \) in schemes of the type (b) [16, 17], if conformation \( E_1 \) of the free enzyme is more stable, but conformation \( E_2 \) binds the substrate better \( (A > a) \). If the return to less active sub-state \( E_1 \) is sufficiently slow \( (a \) is small), then, with \( [S] \) growing, the enzyme stays in more active sub-state \( E_2 \) increasingly longer. This eventually results in imitation of cooperativity (in fact, this is a simplified version of Rabin’s scheme [4, 18]. Lastly, scheme (c), as a more general one, contains the effects listed above as its particular cases, added with the possibility of sub-

strate inhibition.

3. Minimal Schemes of the Effects Caused by the Presence of Different Conformational Channels

It is advisable to confine ourselves to two channels – at least until it is desirable to stay within discrete (sub)states and understand the content of new ef-

fets within not too cumbersome analytical expressions. There exist high-quality generalizations for an arbitrary number of channels (see, e.g., [11, 14]), but they are too overloaded with mathematics, while the characteristic effects are explained within two-

channel schemes all the same.

In the ensemble version, the corresponding set of kinetic equations reads:

\[
\begin{align*}
\frac{d[E_1]}{dt} &= -(a + \alpha) [E_1] + \alpha [E_2] + (b + R) [ES_1], \\
\frac{d[E_2]}{dt} &= \alpha [E_1] - (a + \alpha) [E_2] + (b + r) [ES_2], \\
\frac{d[ES_1]}{dt} &= a [E_1] - (b + R) [ES_1], \\
\frac{d[ES_2]}{dt} &= a [E_1] + [E_2] + [ES_1] + [ES_2] = [E_1],
\end{align*}
\]

with its stationary solution

\[
\begin{align*}
[E_1]_{st} &= [E_2]_{st} = \frac{(b + R)(b + r)}{a(2b + R + r) + 2(b + R)(b + r)}, \\
[ES_1]_{st} &= \frac{a(b + r)}{a(2b + R + r) + 2(b + R)(b + r)}, \\
[ES_2]_{st} &= \frac{a(b + r)}{a(2b + R + r) + 2(b + R)(b + r)},
\end{align*}
\]

so that

\[
v = R[ES_1]_{st} + r[ES_2]_{st} = \frac{a[R(b + r) + r(b + R)]}{a(2b + R + r) + 2(b + R)(b + r)}.
\]

One can see that Michaelis' hyperbola still holds here (since \(a = k[S]\)), but there appears the effect of non-monotonicity of \(v(b)\), provided that the inequality

\[
k[S] > \frac{2rR(R+r)}{(R-r)}
\]

is satisfied (a similar inequality was obtained in [15] for the scheme in Fig. 1, a).

Now, consider the SM version of scheme (8). Taking the remarks after Eq. (6) into account, the correct equations to this non-stationary problem read:

\[
\begin{align*}
\frac{dP_{E_1}}{dt} &= -(a + \alpha) P_{E_1} + \alpha P_{E_2} + b P_{ES_1}, \\
\frac{dP_{E_2}}{dt} &= \alpha P_{E_1} - (a + \alpha) P_{E_2} + b P_{ES_2}, \\
\frac{dP_{ES_1}}{dt} &= a P_{E_1} - (b + R) P_{ES_1}, \\
\frac{dP_{ES_2}}{dt} &= a P_{E_2} - (b + r) P_{ES_2},
\end{align*}
\]

but the initial conditions should be distributed now between sub-states \(E_1\) and \(E_2\). So far, designate them as \(P_{E_1}(0) = n_1, P_{E_2}(0) = n_2,\) and \(P_{ES_1}(0) = P_{ES_2}(0) = 0\). Obviously, \(n_1 + n_2 = 1\).

Applying the Laplace transformation to set (13) and taking into account that now \(f(t) = R P_{ES_1}(t) + + r P_{ES_2}(t)\), after some straightforward algebra, one has:

\[
\frac{1}{\langle t \rangle} = \frac{\Delta^2}{R(\Delta_{ES_1}\Delta' - \Delta'_{ES_1}\Delta) + r(\Delta_{ES_2}\Delta' - \Delta'_{ES_2}\Delta)},
\]

where

\[
\begin{align*}
\Delta &= a \{ab (R + r) + (a + 2\alpha) r R\}, \\
\Delta' &= a \{[(a + b + 2\alpha) (R + r) + 2ab + 2r R] + + 2\alpha (b + r) (b + R)\}, \\
\Delta_{ES_1} &= a \{ar n_1 + (a + b + r)\}, \\
\Delta_{ES_2} &= a \{ar n_2 + (a + b + r)\},
\end{align*}
\]

To find the necessary initial values of \(n_1\) and \(n_2\), we note that, in the serial working regime of a single enzyme, the ratio \(n_1/n_2\) is equal to that of stationary flows of the returns to sub-states \(E_1\) and \(E_2\) after the catalytic stage. That is, \(n_1/n_2 = R[ES_1]_{st}/r[ES_2]_{st}\), where \([ES_{1,2}]_{st}\) are taken from Eqs. (10) of the stationary ensemble version (in fact, this was noted, for example, in [19], although sometimes \(n_1\) and \(n_2\) are erroneously called steady-state probabilities [11, 12]). According to (10), \(n_1/n_2 = R(b + r)/r(b + R)\), then

\[
n_1 = \frac{R(b + r)}{r(b + R)}, \\
n_1 = \frac{R(b + r)}{r(b + R)}. \\
\]

Substituting (16) into Eqs. (14), (15) and comparing the result with Eq. (11), one can eventually make sure that the identity \(v/\langle t \rangle = 1/\langle t \rangle\) really holds.

Adding conformational fluctuations of the complex and possible distinctions between the binding (unbinding) rates \((A \neq a\) and \(B \neq b\), see Fig. 1, c) into consideration does not change the calculation algorithm and eventually leads to the following expression:

\[
v/\langle E_1 \rangle = G/H,
\]

\[
G = a \{ar (B + R) + AR(b + r)\} + + a\beta(A + a)(r + R) + A a (r + R),
\]


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classical $M$-dependence on $S$. In the general case, Eq. (17) does not follow the
approximate [12], while being in fact rigorous (expression (17)
concentration $\langle x \rangle$). Henceforth,

$$
H = a [a (B + R) + A (b + r) + 2 (B + R) (b + r)] + \\
+ 2 \alpha \beta (A + a + B + b + R + r + \alpha) + \\
+ \beta [2 A a + a (B + R) + A (b + r)],
$$

(17)

which, again, coincides with $1/t$ obtained with the
help of the Laplace transform $\mathcal{L}(f)$, thereby confirming
this peculiar ergodic hypothesis (expression (17)
coincides, to the accuracy of designations, with that
obtained for $1/t$ in Ref. [11], albeit in a much more
complex way, via fictitious $E^0$ and $\delta$, so that it was
called approximate [12], while being in fact rigorous). In the general case, Eq. (17) does not follow the
classical MM-dependence on $S$ any longer. The latter,
however, still holds (at least, with rather good
precision) in many limiting cases [11, 14]. On the
whole, as mentioned above, the scheme in Fig. 1, c)
and Eq. (17) admit realization of practically all the
interesting deviations from the standard MM kinetics
which are related to regulatory abilities rooted in the
presence of (multiple) conformational channels: non-
monotonic $v(b)$, “cooperativity”, and substrate
inhibition, see Fig. 2.

3.2. Fractional polynomials

From the formal point of view, all these effects can
be analyzed within the so-called fractional polynomials,
i.e., expressions of the type $F_n(x)/G_n(x)$, where
$F_n$ and $G_n$ are polynomials of the $n$-th order, with
$n$ depending on the number of intermediates and dis-
crete conformational channels in an MM-like scheme
(see, e.g., [4, 20]). Henceforth, $x$ stands for substrate
concentration $[S]$. In particular, for the scheme in

$$\begin{align*}
&v(x) = v(\infty) \frac{x^2 + px}{x^2 + qx + c}, \\
&\text{where } p, q, \text{ and } c \text{ are positive combinations of the rate }
\text{constants involved, as Eq. (17) adopts this form after}
\text{replacements } A \rightarrow k_1 x, \ a \rightarrow k_2 x. \ \text{Generalizations of}
\text{Eq. (18) for more complex schemes can be found in [14], although they seemingly do not lead to prin-
cipally new regulation mechanisms. The general con-
clusions of such analyses are quite transparent, since}
\text{one can arrive at them by simply equating the deriva-
tive } v'(x) \text{ or } v''(x) \text{ to zero. The presence of a posi-
tive root of the equation } v'(x) = 0 \text{ testifies to the}
\text{possibility of substrate inhibition. In our case,}
\text{this condition is reduced to the quadratic equation}
(q - p) x^2 + 2cx + pc = 0 \text{ which has a positive root, if}
\text{only } q < p. \text{ In terms of parameters of the scheme in}
\text{Fig. 1, c, this condition reads}
\end{align*}$$

$$k_1 (b + r) - k_2 (B + R) > \frac{\beta (R + r)}{\alpha (R - r)},$$

(19)

and the greater the difference between the catalytic
rates, the better the validity of Eq. (19) (see an ex-
ample given in Fig. 2, c).

Possible cooperativity (i.e., flexion of $v(x)$) is re-
lated to the presence of positive roots of the equation
$v''(x) = 0$. Here, the latter is cubic,

$$(q - p) x^3 + 3cx^2 + 3pcx + c (pq - c) = 0.$$  (20)

According the known Descartes rule, Eq. (20) can
have positive roots, if only changes of signs of the

![Fig. 2. (a) Non-monotonic $v(b)$ in the scheme in Fig. 1, a. $a = 10$. Curve 1: $R = r = 3$; curve 2: $R = 10$, $r = 1$. (b) Weak cooperativity in the scheme in Fig. 1, b with the return after the catalytic stage to sub-state $E_2$. The flexion of $v(x)$ is negligible unless one looks at the concave in the inset for extremely small $x$ (see the text below Eq. (20)). Here, $\alpha = 1$, $k_1 = a/[S] = 0.1$, $k_2 = A/[S] = 1$, $b = 10$, $R = 100$. (c) Substrate inhibition in the scheme in Fig. 1, c, according to Eq. (17). $\alpha = 10$, $\beta = 1$ $k_1 = A/[S] = 10$, $k_2 = a/[S] = 1$, $b = 10$, $B = 1$, $r = 1$, $R = 10$.](image)
coefficients in its l.h.s. take place, i.e., if the following alternative is satisfied: \( p > q \), or \( p < q \), but \( c > pq \). It is hardly appropriate to write down these inequalities in terms of the parameters of Eq. (17); an illustrative example is given in Fig. 2, b). One can notice that cooperativity is barely seen (the corresponding Hill coefficient would only slightly exceed 1), as it is a commonplace in models with discrete conformational states [16, 17]. No wonder that, in many cases, the classic MM hyperbola holds practically true [11]. Examples of “phase diagrams” of regimes of cooperativity or substrate inhibition are given in [14].

As for a non-monotonic dependence of \( v(b) \) on the rate of “unproductive” substrate release – the effect that attracted attention quite recently [21], albeit in an unnecessarily sophisticated way – its simplest scheme is shown in Fig. 1, a [15], see also Fig. 2, a. It includes no conformational fluctuations at all. This effect is also possible within more general scheme in Fig. 1, c, even in its simplified version (8) \((A = a, B = b, \beta = 0)\). With \( b \) as an argument, Eq. (11) has even simpler form than (18):

\[
v(b) \sim \frac{b + [2rR/(R + r)]}{b^2 + (R + r + a)b + rR + a(R + r)/2},
\]

and the analysis of the equation \( d\frac{d}{db} = 0 \) shows that its positive root exists under the condition

\[
kx > \frac{2rR(R + r)}{(R - r)^2}
\]

(it is equivalent to Eq. (12), since \( a = kx \) here) which requires a sufficiently high substrate concentration; the greater the difference \( R - r \), the lower the concentration is required. Then, staying too long in the less-active conformational sub-state \( ES_2 \), the enzyme can return to its free state \( E \) with a chance to perform the catalytic act in a more effective way via sub-state \( ES_1 \) [15]. An analysis (again, somewhat over-complicated and involving unnecessary \( E^0 \) and \( \delta \)) of this effect in another version of the scheme in Fig. 1 a) was given in the recent paper [13].

4. Concluding Remarks

Our main goal is to distinguish the minimal schemes of enzymatic reactions that lead to deviations from the classic MM kinetics and are a signal for the relevant above-listed effects of conformation regulation. We point out the simplest and straightforward algorithm of calculation of the reaction velocity, avoiding unneeded complications that occur in the current literature. The reactions of monomeric enzymes with a single binding site fit these goals best.

We elucidate the important difference in the problem formulation in its ensemble and SM versions, despite the similarity of the corresponding kinetic equations. It is the difference that ensures the identity \( v/\langle t_E \rangle = 1/\langle t \rangle \) even in the presence of several conformational pathways of the reaction. Consequently, if interested in the velocity only, one can restrict her/himself to the stationary ensemble version, where calculations are quite trivial. Not much harder is to find \( f(s) \) in the non-stationary SM version in order to calculate, if needed, the higher moments \((\langle t^m \rangle \) and disorder parameters.

For the demonstrative purposes, it is sufficient to involve two conformational pathways. Considering multiple pathways makes only relative sense, since, apart from producing extremely bulky expressions, it can hardly be used in practice because of too many parameters involved.

In addition, one should bear in mind that the schemes described above, as well as the overwhelming majority of similar schemes in the literature, are those of the classical linear kinetics based on the mass action law \(^1\), with constant coefficients (rate constants). They were, are, and will be an important tool of the enzyme kinetics. Their simplicity is their undisputable merit, as well as the fact that they help to clarify the physical nature/mechanisms of the effects – but mainly qualitatively. Since, as it is well known from, for example, extensive investigations into ligand binding to myoglobin [22] or electron transfer in photosynthetic reaction centers [23], the rate “constants” of protein reactions often change in time and are distributed along “perpendicular” (i.e., structural) coordinates [16, 24]. This entails essentially non-exponential kinetics and nonlinear effects of structural memory/feedback based on an adequate treatment of the substrate-conformational interactions and inevitable, essentially non-equilibrium flux conditions of the enzyme functioning [25]. All these eventually lead to the molecular self-organization effects and changes of the functional regimes of the system.

\(^1\) Even if the kinetic equations are formulated for the probabilities in the SM version.
enzyme by means of non-equilibrium phase transitions. We have proposed and developed a proper concept quite long ago (for more details, see, e.g., [17] and references therein).

Apart from enzymes, the MM-like schemes concern many transport processes, absorption on soft substrates, and especially nanocatalysis [7, 8], so that nowadays they are in intensive use. That is why the above-presented analysis of their features seems important for the modern theory of conformational regulation.

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PROШІДНІСТЬ ФЕРМЕНТАТИВНИХ РЕАКЦІЙ У МІХАЕЛІС–МЕНТЕН–ПОДІБНИХ СХЕМАХ. АНСАМБЛІЕВА ТА ОДНОМОЛЕКУЛЯРНА ВЕРСІЇ

Р е з ы м е

У пошуках нестандартних шляхів конформаційної регуляції активно використовують різноманітні, іноді переукладені, версії схеми Міхельса–Ментена. На прикладі мономерних ензимів з одним місцем зв'язування визначено мінімальні схеми, що уможливлюють особливі регуляторні властивості типу "кооперативності", субстратного ініціювання тощо. Проілюстровано найпростіші шляхи розрахунку швидкості ензиматичної реакції в ансамблі© або на рівні по- одиночної молекули ензиму.