Medllecta: Hematological preventive method (HPM)

Part 2.

[P-S Standard]

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Abstract.
A complete dynamic model of the protein and, in particular, the enzymatic process of synthesis and degradation could significantly improve the quality of diagnosis of diseases of various etiologies at the earliest stages of their development.
In this article, we describe our initial attempt to create the above model based on a radically new mathematical approach, Sense Logic [1] in terms of enzymatic kinetics.

1. Introduction

In a rough approximation, the human body can be represented as a topological space where any organ or internal system is open sets with the exception of the skin. The topology of this space, in this case, is the set of all internal systems of the human body. Accordingly, in order to diagnose the general state of the human body at a certain moment in time, we must clearly understand the principles of interaction and influence on each other of all internal systems of the body (systems of subsets of topology) of a person at a given moment in time. However,

the tools of traditional mathematics do not provide any physiological or pathogenetic interpretation in terms of, for example, the products of an enzymatic reaction. They also do not provide any tools for constructing models of protein metabolism leading, say, in autoimmune diseases.

For the purpose of the possibility of constructing the above models, as well as hematological patterns corresponding to a certain pathophysiological process of the human body, below in this article we use the Sense Logic of the theory of the Sense Theory.

2. Problem

Lack of a unified mechanism (instrumentation) for constructing a complete dynamic pathophysiological model of the human body with the ability to identify the primary source of protein transmutation.

3. Solution

One of the main mechanisms in determining the organ source of pathogenesis is the analysis of the biochemical processes of plasma proteins and amino acids.

Plasma proteins.

The vast majority of all pathophysiological processes in the human body occur with the direct participation of proteins.

In practical (clinical) medicine, plasma proteins are usually classified according to their functionality [3]:

Table 1. (see Appendix)

The total concentration of proteins in blood plasma depends on three main factors:

1. the volume of protein synthesis at the sites of their production
2. the proportions of their distribution in the human body
3. the rate of their elimination from the human body

For diagnostic purposes, it is advisable to take into account all possible factors that directly or indirectly affect the concentration of human blood plasma proteins. Among them [3]:

Physiological factors

- **Age**. the age of a person affects the numerical and qualitative indicators
- **Gender**. *α-fetoprotein, ferritin, IgM*
- **Drugs.** *oral contraceptives, testosterone, phenothiazines, estrogens*
- **Genetic characteristics.** *phenotype, individual proteins*
- **Nutrition.** *complement system, prealbumin, retinol-binding protein*
- **Pregnancy.** *transport proteins, α-fetoprotein*
- **Physical exercise.** *increase in protein concentration*
- **Environment.** *immunoglobulin level*

Pathological factors

- **Losses.** *damaged organ, nephrotic syndrome, glomerular proteinuria, tubular proteinuria*
- **Synthesis.** *liver disease, phenotypic deficiency*
- **Volume circulating blood.** *hypohydration, overhydration*
- **Catabolism.** *inflammation*
- **Disposal speed.** *kidney disease, inflammation*
- **Compensatory mechanisms.** *nephrotic syndrome*

Methodological factors

- **Storage.** *proteolytic enzyme activity*
- **Laboratory methods.** *different permissible error*
- **Sampling method blood.** *concentration change*

With damage of any etiology, the process of inflammation begins in the human body.

"Dysfunction of internal organs and systems of the human body is always an inflammatory process"

The process of inflammation itself can be divided into *acute* and *chronic*.

The concentration of acute inflammation proteins, the so-called acute phase proteins (APPs), changes during the first 6-48 hours. The acute phase can last for several days, depending on the duration of the action of the damaging factors. There is a *direct correlation* between the concentration of acute phase proteins and the phases of disease activity. In this regard, the indicator of the concentration of acute phase proteins can be used to monitor the course of the disease.

For example, it is possible to classify some proteins of the acute phase on the basis of a multiple increase in their concentration over time [3]:

Acute phase proteins (APPs)		
Group	**Protein**	**Serum concentration, normal (g/L)**
"Main" reactants, an increase of 20-1000 times in within 6-12 hours	C-reactive protein (CRP)	< 0,005
	Serum amyloid A (SAA)	0,001-0,03
Moderate increase of 2-5 times in within 24h	α1- antitrypsin,	1,4-3,2
	α1- antichymotrypsin	0,3-0,6
	α1- acidic glycoprotein	0,4-1,3
	haptoglobin	0,5-3,2
	fibrinogen	1,8-3,5
Minor increase of 20-60% in within 48 hours	C3 component of complement	0,5-0,9
	C4 component of complement	0,1-0,4
	Ceruloplasmin	0,2-0,5

Table 2.

For diagnostic purposes, several acute phase proteins should be analyzed at once, since different people may have their own disharmonious acute phase response due to the uniqueness of the internal state of the body.

In practice, C-reactive protein is considered to be one of the fastest acute-phase proteins that respond to the onset of the inflammatory process. The concentration of C-reactive protein increases significantly

in the first 6 hours of the inflammatory process and then gradually decreases after 7-10 days.

The absence of a decrease in concentration indicates a persisting inflammatory process in the body, for example, as a result of incorrectly selected treatment.

Thus, the level of C-reactive protein above 0.01 g/L but below 0.06 g/L may indicate:

1. rheumatoid arthritis
2. gout
3. ulcerative colitis
4. dermatomyositis
5. local bacterial infections
6. viral infections
7. Sarcoidosis
8. etc.

above 0.06 g/L:

1. systemic vasculitis
2. deep vein thrombosis
3. Crohn's disease
4. acute pancreatitis
5. metastatic necrotizing tumors
6. sepsis
7. pneumonia
8. etc.

The general effects of the acute inflammation process can lead to the following changes:

- increased concentration of IL-1, IL-6, IL-8, IL-11, IL-16, IL-17, G-CSF, TNF-α
- increased concentration of GM-CSF, G-CSF, H-CSF
- increased concentration of globulins
- decrease in the concentration of albumin
- increased erythrocyte sedimentation rate (ESR)
- increased catabolism
- increased SAS
- increased SCS

This is the so-called *systemic inflammatory response syndrome* (SIRS) - when the entire metabolic process in the body changes.

The chronic process of inflammation, in contrast to the acute one, is characterized by the presence of a long process of proliferation of monocytes, macrophages, fibroblasts, etc.

Along with this, the processes of angiogenesis proceed with a certain stable intensity (FGF, PDGF, TNFα, etc.). The main, for example, four differences between an acute process of inflammation from a chronic one are indicated in the following table:

№	Reactions	Chronic	Acute
1.	**Immune response**	specific	nonspecific
2.	**Vascular reactions**	angiogenesis	edema, vasodilation
3.	**Predominant cells**	lymphocytes, plasma cells, fibroblasts	macrophages, neutrophils
4.	**Local signs**	not expressed	expressed

Table 3.

The source of the process of inflammation, both acute and chronic, are the so-called *inflammatory mediators*, or rather enzymes that cause the synthesis of these mediators. There are **two types of mediators**: cellular and plasma.

Cellular mediators of inflammation are located in the focus of inflammation, in the cells of the human body.

Plasma mediators of inflammation are synthesized in the vascular epithelium of the human body.

№		Cellular mediators	Plasma mediators
1.	**Synthesis**	the focus of inflammation	the vascular epithelium of the human body
2.	**Molecules**	leukotrienes, prostaglandins, acetylcholine, norepinephrine, histamine, serotonin, leukokines, cytokines, enzymes	complement system, bradykinin, procoagulant, anticoagulant, fibrinolytics
3.	**Release**	active	inactive

Table 4.

In most practical cases in clinical practice with unclear organ pathology, *serum enzyme profiles* represent one of the qualitative ways to obtain information on the localization of the pathological process.

Enzyme profiles.

By the type of chemical reaction catalyzed, enzymes can be divided into several classes [3]:

№	Class	Type of reaction catalyzed	Enzymes
1.	**Oxidoreduct basins**	redox reactions	dehydrogenase, peroxidase
2.	**Transferases**	intermolecular transfer different groups of atoms from donor molecules per molecule acceptor	нехокinase, transaminases
3.	**Hydrolases**	cleavage of intramolecular bonds with the participation of a water molecule	alkaline phosphatase, trypsin
4.	**Lyases**	reversible cleavage reactions different groups from substrates not hydrolytically with double bond formation or double bond attachment	Carbonic anhydrase, dehydratase
5.	**Isomerase**	interconversion of various isomers	triose phosphate isomerase
6.	**Ligases**	synthesis reactions	heme synthetase

Table 5.

According to the localization of enzymes in cells and tissues, they are divided into *common* and *organ-specific*.

Common enzymes are found in almost all cells. They provide the basic processes of cell life: the metabolism of proteins, lipids, carbohydrates, the synthesis of adenosine triphosphate, the maintenance of osmotic pressure, hemataxis, and others.

Organ-specific enzymes are activated only in certain organs, tissues or groups of organs and tissues.

The regulation of enzyme activity is carried out:

1. limited (partial) proteolysis
2. allosteric regulation
3. covalent modification
4. protein-protein interaction
5. genes

1. *Partial proteolysis* can be expressed by the following scheme:

*proenzyme -> enzyme * peptide*

As an example,

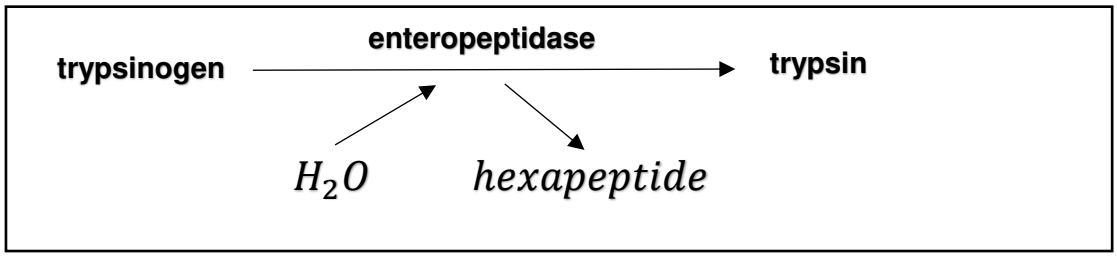


Fig 1.

Partial proteolysis is *irreversible*.

In the Sense Theory [6], partial proteolysis can be expressed through a sense derivative on union:

$$S_f^{diff}(\mathfrak{S}_K) = [S_f(\mathfrak{S}_K) \cup S_f(\mathfrak{S}_{O(L)})] = S_f(\mathfrak{S}_M) \quad (1)$$

where $\mathfrak{S}_K$ - No-Sense Set consisting of one element “trypsinogen”.
Trypsinogen, in turn, can be written approximately as an object:

$$O_T = \{lysine, agrinin\}, \quad (2)$$

$\mathfrak{S}_{O(K)} = \{O_T\}$ or $\oslash_T = \lim_S \mathfrak{S}_{O(K)}$, where $\oslash_T$ - zero-object (trypsinogen)

,

$\mathfrak{S}_{O(L)}$ - No-Sense Set consisting of two elements, “enteropeptidase” and H_2O

,

$\mathfrak{S}_M$ - No-Sense Set consisting of two elements, “trypsin” and “hexapeptide”

,

$S_f(\mathfrak{S}_K)$ - sense function [5] defined on $\mathfrak{S}_K$

,

$S_f(\mathfrak{S}_{O(L)})$ - sense function defined on $\mathfrak{S}_{O(L)}$

,

$S_f(\mathfrak{S}_M)$ - sense function defined on $\mathfrak{S}_M$

,

It's obvious that $\lim_S \mathfrak{S}_M = \oslash_{TR}$ (trypsin) or $S_f(\mathfrak{S}_M) = \oslash_{TR}$ on all $\mathfrak{S}_M$

We obtained this result in accordance with the definition of the sense sequence [6], which is the set $\mathfrak{S}_M$:

$$\text{◎}_{TR} \subset \mathfrak{S}_M = S, \text{ where } S - \text{Sense Set} \tag{3}$$

Moreover, it is worth noting that if at least one of the three elements (trypsinogen, enteropeptidase, H_2O) is removed from the set $\mathfrak{S}_K$ ($\mathfrak{S}_{O(L)}$), then we get the following inequality:

$$\lim_S \mathfrak{S}_M \neq \text{◎}_{TR} \tag{4}$$

or

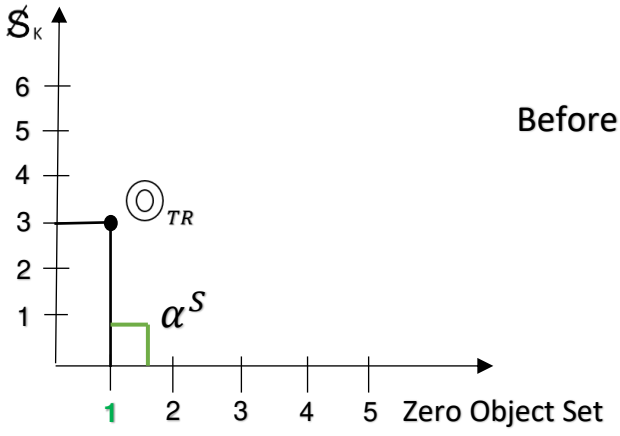
$$S_f(\mathfrak{S}_M) \neq \text{◎}_{TR} \tag{5}$$

This means that the operator "semantic union" - U_K^L in expression (1) takes into account the semantic interaction of its operands $\mathfrak{S}_K$ and $\mathfrak{S}_{O(L)}$.

Namely, if two or more elements make up a sense sequence [6], that is, they converge to some object by the sense limit, then they can be replaced in a finite set by this object.

Unlike the number set of traditional mathematics $X = \{x_1, x_2, x_3, \dots x_n\}$, where each of its elements is an independent variable, in the Sense Theory the addition or removal of at least one element **can drastically affect the final result**.

Graphically, partial proteolysis can be shown as follows:



Before U_K^L execution

Fig 2.

and correspondingly,

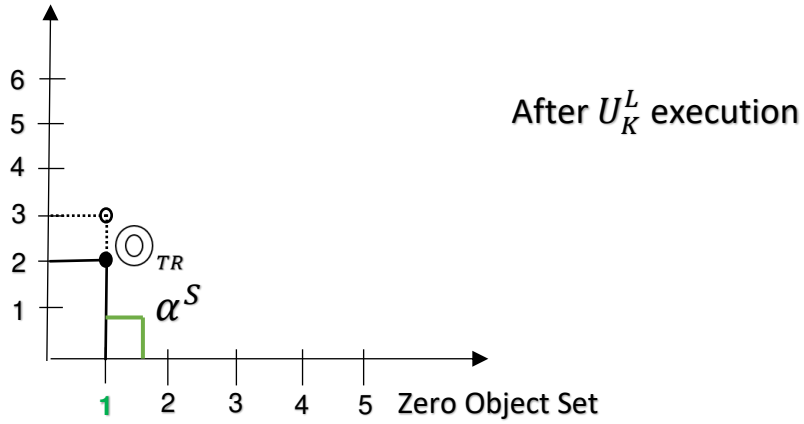
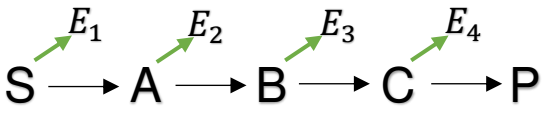


Fig 3.

2. *An allosteric enzyme* is called an enzyme whose activity is regulated by the reversible non-covalent attachment of an effector (activator or inhibitor) to the allosteric center of the enzyme.

Allosteric regulation can be expressed by the following scheme:



,

Fig 4.

where the substrate S was taken as the effector.

The enzymatic reaction is activated according to the principle of a direct positive connection until the reaction product P is obtained. Inhibition of the enzymatic reaction occurs according to the principle of negative feedback loop from the reaction product P to the original enzyme E_1.

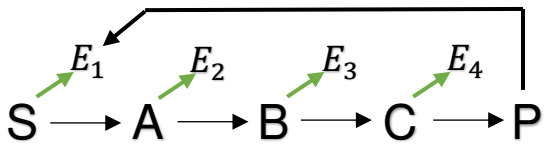


Fig 5.

In Sense Theory, we can write the entire chain of the above direct positive connection with one equation:

$$\{S, E_1\}[\underset{i=2,\dots4}{\cup} E_i] \overset{S}{=} \{S, E_1\}[\underset{i=2}{\cup} E_2 \underset{i=3}{\cup} E_3 \underset{i=4}{\cup} E_4] \overset{S}{=}$$

$$|\lim_{S} \{S, E_1\} = \mathrm{A}| \overset{S}{=} \{\mathrm{A}, E_2\}[\underset{i=3}{\cup} E_3 \underset{i=4}{\cup} E_4] \overset{S}{=} |\lim_{S} \{A, E_2\} = \mathrm{B}| \overset{S}{=}$$

$$\{B, E_3\}[\underset{i=4}{\cup} E_4] \overset{S}{=} |\lim_{S} \{B, E_3\} = \mathrm{C}| \overset{S}{=} \{\mathrm{C}, E_4\} \overset{S}{=} \mathrm{P} \tag{6}$$

or

$$A[\underset{i=2,\dots4}{\cup} E_i] \overset{S}{=} \mathrm{P} \tag{7}$$

For a negative feedback loop where $\{P, E_1\}$:

$$A'[\underset{i=2,\dots4}{\cup} E_i] \overset{S}{\neq} \mathrm{P} \quad , \tag{8}$$

where $A' = \lim_{S} \{\mathrm{P}, E_1\}$, that is, P binding to the allosteric center E_1 inhibits it and the entire metabolic pathway. This process is called *retroinhibition*.

Due to the fact that the regulation of allosteric enzymes E is reversible, we can assume the existence of a semantic antiderivative [7]:

$$\oint [S_f(P)] = \underset{\ominus}{diff}\ [S_f(P)]_{E_i} = S_f(\{P, E\}[\underset{i}{\cup} E_i]) \quad , \tag{9}$$

where $\{S, E_1\} - undefined$,

or

$$\oint\limits^{\bigcirc} [S_f(P)] = E' \quad , \tag{10}$$

where $E' - distorted\ active\ site\ enzyme$.

3. *Covalent modification* of an enzyme is the process of attaching a molecule to an amino acid residue of an enzyme.

There are many types of covalent modification, including acetylation, carboxylation and phosphorylation. For example, one of the most common types of post-translational protein modification, phosphorylation, is one of the most important biochemical reactions that regulate the activity of a given protein.

Cyclic phosphorylation of adenosine diphosphate (ADP):

$$\text{ADP} + H_3PO_4 + \text{E} \rightarrow \text{ATP} + H_2O$$

or in terms of the Sense Theory:

$$\underset{\bigcirc}{\text{diff}}\ [S_f(\not{\mathbb{S}}_{\text{ADP}})]_E = S_f(\not{\mathbb{S}}_{\text{ATP}}) \quad , \tag{11}$$

where E – energy,

$\not{\mathbb{S}}_{\text{ADP}}$ – No-Sense Set with elements {ADP,H_3PO_4},

$\not{\mathbb{S}}_{\text{ATP}}$ – No-Sense Set with elements {ATP,H_2O}.

The last expression can also be written as follows:

$$\oint\limits^{\ominus} [S_f(\not{\mathbb{S}}_{\text{ADP}})]_E = S_F^{\ominus} = S_f(\not{\mathbb{S}}_{\text{ATP}}) \tag{12}$$

or

$$S_f(\not\mathbb{S}_{ATP}) = S_F^{\ominus}, \tag{13}$$

that is, the result of the phosphorylation process of ADP is the sense antiderivative of the sense integral on disunion of the sense function of ADP.

Thus, by fixing the energy (E) and taking the sense derivative on union on property (E), we can obtain a number of chemicals resulting from catalytic (enzymatic) reactions:

$$\underset{\circledcirc}{\text{diff}}\,(\text{E})\,[S_f(\not\mathbb{S}_M)]_N = S_f(\not\mathbb{S}_M \circledcirc \not\mathbb{S}_N) = S_f(\not\mathbb{S}_{M+N}), \tag{14}$$

where

$\not\mathbb{S}_M$ – No-Sense Set with elements {E,e_1, e_2, e_3,… e_m},

$\not\mathbb{S}_N$ – No-Sense Set with elements {e_{m+1},e_{m+2},e_{m+3},… e_{m+n}},

$that\ is, P_S^N(\not\mathbb{S}_N(E)), where\ P_S^N\ -sense\ punctured\ neighborhood$

To obtain information about chemicals (proenzymes) at a certain point in time, you can use the sense antiderivative on union:

$$\oint^{\circ} S_f(\not\mathbb{S}_{M+N}) = S_F^{\circ} = S_f(\not\mathbb{S}_M) \tag{15}$$

If in practice, it is necessary to obtain a number of possible chemical compounds leading to one resultant product of chemical reactions of these compounds, then the sense derivative on object can be used:

$$S_f^{diff}(O_N)(\not\mathbb{S}_N) = \circledcirc = const, \tag{16}$$

where

$\circledcirc$ – chemical element (reaction product) that is obtained for any

choice of elements of the set $\mathfrak{S}_N$, $O_N = \lim_S \mathfrak{S}_N$.

4. During *protein-protein interactions*, certain proteins play the role of transmitting the received signal from the receptor domain of the cell to its internal molecules.

In general practice, it is customary to operate with such concepts as *primary messengers*, *primary effectors*, *secondary messengers*, *secondary effectors*, and so on.

So, the adenylate cyclase regulation system can be expressed by the following scheme:

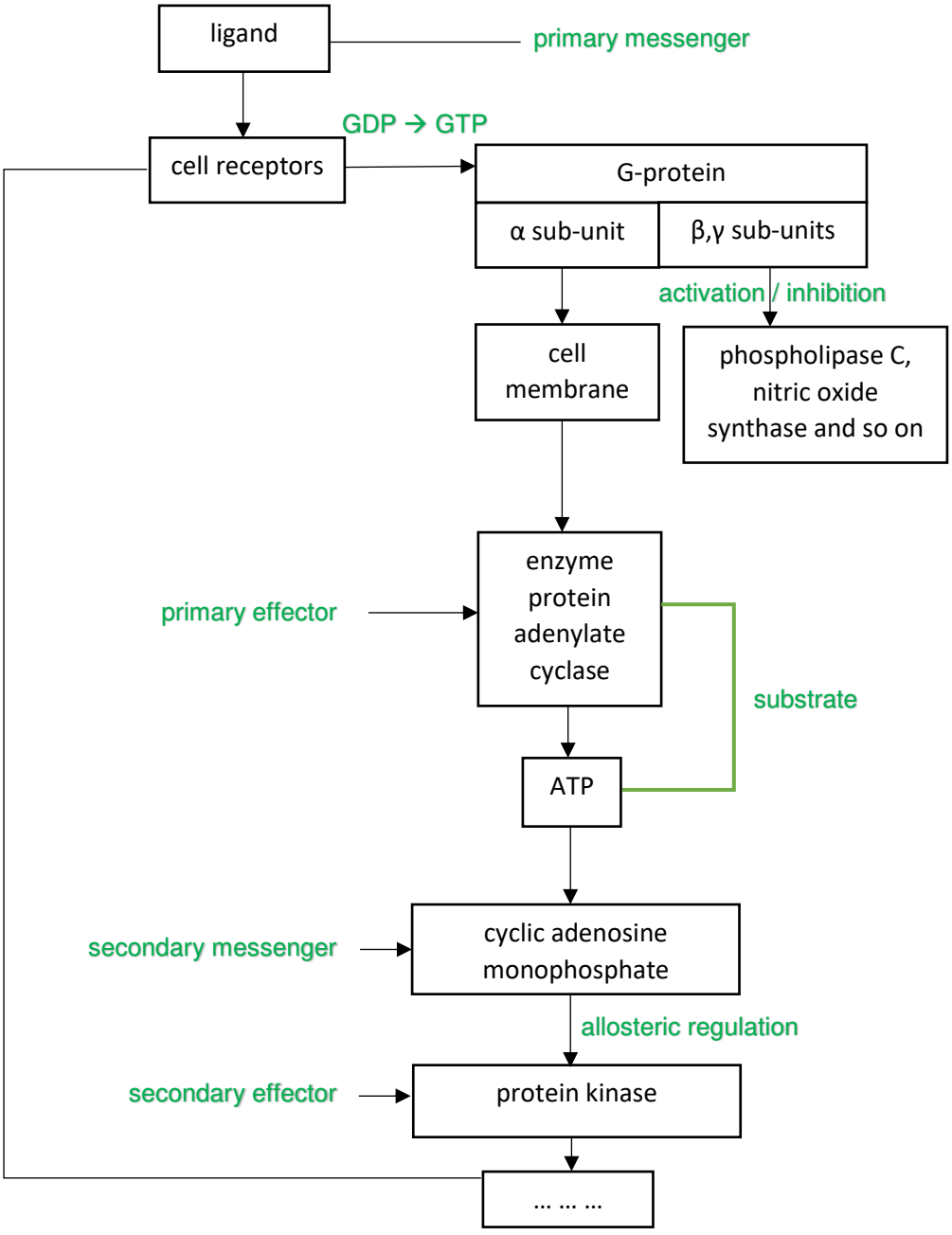


Fig 6.

As you can see from the Fig 6 above, each subsequent step directly depends on the existence of the previous step.

This sequential process is called the *signaling pathway*. In turn, the signaling pathway of the adenylyl cyclase regulation system can be expressed in terms of a sense series of the Sense Theory:

$$U_n^n = a_1 \circlearrowleft a_2 \quad , \tag{17}$$

where

$a_1 - ligand,$

$a_2 - cell\ receptor,$

$n = 2.$

$Futher, U_n^n = a_1 \circlearrowleft a_2 \circlearrowleft a_3, where\ a_3 - G - protein\ (not\ dissociated), n = 3$

And by virtue of *semantic synapse* [8] we get:

$$S_{SY}(a_2, a_3) = \circlearrowleft a_2, a_3 = \mathfrak{S}_2 \quad , \tag{18}$$

$where\ \mathfrak{S}_2 - sense\ sequence, due\ to\ the\ existence\ of \lim_S \mathfrak{S}_2 = \circledcirc_G = \{\circledcirc_\alpha, \circledcirc_\beta, \circledcirc_\gamma\}$,

where $\circledcirc_G$ – G – protein (dissociated)

And accordingly we get:

$$U_n^n = a_1 \circlearrowleft \circledcirc_G \tag{19}$$

When adding the enzymatic protein adenylate cyclase,

$$U_n^n = a_1 \circlearrowleft \circledcirc_G \circlearrowleft a_4 \quad , \tag{20}$$

or

$$U_n^n = a_1 \circlearrowleft \circledcirc_\alpha \circlearrowleft \circledcirc_\beta \circlearrowleft \circledcirc_\gamma \circlearrowleft a_4 \quad , \tag{21}$$

where a_4 – adenylate cyclase.

And due to the action of semantic synapse between two elements of the sense series $\circledcirc_{\alpha}$ and a_4, we have:

$$U_n^n = a_1 \cup a_5 \cup \circledcirc_{\beta} \cup \circledcirc_{\gamma}, \quad (22)$$

where a_5 – cyclic adenosine monophosphate, n = 4.

The sense series U_n^n can increase in terms of the total number of its elements if at least one of its elements has another "external" element associated with it by means of semantic synapse. *Moreover, with the introduction of an external element into the total set of elements of the sense series, the total number of elements of this series may decrease significantly depending on the number of existing "semantic synapse" links between them.* In other words, when a certain molecule (secondary messenger) appears inside the cell, the latter can trigger a series of chemical reactions causing the appearance of new molecules. However, in the case of inhibition, the number of elements of the sense series upon the introduction (by semantic union) of the "external" element (molecule) will remain unchanged due to the following expression:

$$S_{SY} \not\subseteq_{S} \mathcal{S}_N \quad (23)$$

Semantic synapse is the primary (secondary, Nth) effector.

Semantic union is the primary (secondary, Nth) messenger.

Unlike semantic synapse between two elements of a sense series, which determines the natural chemical process of interaction, we can "artificially" stimulate a chemical reaction through the use of *a sense partial derivative* for a certain element of the series under consideration:

$$U_n^n = a_1 \cup \circledcirc_G \cup a_4, \quad (24)$$

$$S_f^{diff}(U_n^n)_{a_4}^s = a_1 \cup \circledcirc_G \cup (a_4)_s^{diff} \quad (25)$$

or

$$S_f^{diff}(U_n^n)_{a_4}^s = a_1 \cup \circledcirc_G \cup S_f(\mathcal{S}_2), \quad (26)$$

$where\ \mathbb{S}_2 = \{adenylate\ cyclase, ATP\},$
$S_f(\mathbb{S}_2) = a_5$ (cyclic adenosine monophosphate), $due\ to\ the\ existence\ of \lim_{S} \mathbb{S}_2$
S - substrate ATP.

Therefore, we can investigate how a change in the enzymatic activity of a single protein in the "signaling pathway" chain affects the overall metabolic state of the cell.

It is worth noting that in the last equation the dissociated G-protein will be considered in the part of the semantic union with cyclic adenosine monophosphate (not with adenylate cyclase), which will undoubtedly affect the general chemical state of the system described by U_{diff}^{n}.

Definition: *Partial semantic derivative* $S_f(A_N)_{a_k}^{x}$ on union (disunion),

$$where\ k \in N, x \notin A_N, is\ \ S_f(a_k) \Cup S_f(x) = S_f(\mathbb{S}_N)$$

,

$$where\ S_f(\mathbb{S}_N) = \begin{cases} S_f(\mathbb{S}_{O(N)}), if\ \Cup a_k, x = C, & where\ C\ -semanntic\ synapse \\ \mathbb{S}_2\ , if\ S_f(\mathbb{S}_N) \neq \circledcirc \end{cases}$$

$$(S_f(a_k) \Cup S_f(x) = S_f(\mathbb{S}_N), if\ and\ only\ if\ x \in a_k)$$

Due to the fact that the semantic function $S_f(A_N)$ is defined on the set A_N [5], after calculating the partial semantic derivative, three cases are possible:

A. $S_f(A_N)$ becomes undefined.
B. $S_f(A_N)$ becomes already defined on another (new) set A_N and, accordingly, has another zero object to which it converges.
C. $S_f(A_N)$ becomes defined on primary set A_N.

In the second case, we will have a situation where the "signaling pathway" will lead to an "effector" different from the original final "effector".

If we denote by M the set of all chemical elements of the cell C, then the static semantic equation for this cell will look like this:

$$S_f(\$_M) = \circledcirc_c \overset{\ominus}{\circledcirc} \emptyset_S \quad , \tag{27}$$

$where\ \circledcirc_c - zero\ object\ (cell\ at\ a\ certain\ point\ in\ time),$
$\$_M - No - Sense\ Set\ with\ chemical\ elements\ of\ C - cell$

If we want to check the reaction of the C cell to an external influence, for example, *ligands*, then the semantic equation will take the following form:

$$S_f(\$_M) \circledcirc L = \circledcirc_c \overset{\ominus}{\circledcirc} \emptyset_S \quad , where\ L\ - ligand \quad , \tag{28}$$

or

$$\underset{\circledcirc}{diff}[S_f(\$_M)]_L = \circledcirc_c \overset{\ominus}{\circledcirc} \emptyset_S \quad , \tag{29}$$

or

$$\oint_L^{\ominus}[S_f(\$_M)] = S_F^{\ominus} = \circledcirc_c \overset{\ominus}{\circledcirc} \emptyset_S \tag{30}$$

5. The process of genetic regulation of enzyme activity is based on the induction or repression of enzyme synthesis with the participation of inducers or repressors.

The substrates of the enzymatic reaction are often the inducers, and the end product of this reaction is the repressor.

Enzymatic reaction kinetics

The initial activity of many enzymes is manifested only in the presence of non-protein substances. These non-protein substances are called *cofactors* ($Mg^{2+}, Zn^{2+}, Mn^{2+}, Fe^{2+}, Ca^{2+}, etc.$). For example, the polypeptidase enzyme is activated by Co and Zn ions, the carboxylase enzyme by Fe, Cu, Co, Ca, Zn and Mn ions. Accordingly, the ligand (L) can be used as the first element of the semantic formula of chemical

kinetics. Each enzyme is highly specific for the substance with which it interacts. This substance is the substrate. The enzyme (E) and the substrate (S), in turn, form the so-called *enzyme-substrate complex*.

Further, the degradation of the enzyme-substrate complex leads to the production of the enzyme and the final product (P).

In terms of the Sense Theory, ***the formula for enzymatic kinetics*** can be written as follows:

$$L_0 \circledcirc \ [\underset{i=1,\dots n}{\overset{\ominus}{\circledcirc}} E_i, S_i] \overset{S}{=} P_i \quad , \tag{31}$$

$where \ \overset{\ominus}{\circledcirc}$ – abbriviation of two sense operation, $\ominus$ and $\circledcirc$ by analogy as "$\pm$" in traditional mathematics,
S_i – substrate, protein molecule depending on the type of regulation of enzymatic activity.

Obviously, as the value of i increases, the set S will also increase due to an increase in the amount of proteins and other substances involved in the transmission of the initial signal.

The semantic operation $\ominus$ is added to the above formula due to the "reversibility" of the enzyme-substrate complex, and also, in the case of an inhibition reaction.

The speed of formation of the product P, or rather *the speed of the enzymatic reaction* V_E, depends on several factors. The most significant factors such as T, pH and S concentration significantly affect the value of V_E. Further, the path D_p which the enzymatic reaction must go through to obtain, say, product P_1 is equal to:

$$L_0 \circledcirc [\ \circledcirc E_1, S_1] \tag{32}$$

The time T_p spent on obtaining the product P_1 will depend, first of all, on the values of the factors T, pH and S:

$$L_0 \circledcirc [\ \circledcirc E_1, S_1]^{T}_{pH}_{S} \quad , \tag{33}$$

that is, the problem of finding the minimum value of $T_p(T, pH, S)$ is reduced to finding the optimal values of the factors T, pH and S. In other words, the problem of finding the minimum of the function T_p.

By analogy with physics, we can calculate the average speed of obtaining a product P:

$$\frac{D_p}{\underset{\circledcirc}{diff}[T_p, pH, S]_t} = V_p \tag{34}$$

However, in the Sense Theory there is no division operator in the sense in which it is used in traditional mathematics. Therefore, one of the methods for calculating V_p is “to measure" the time T_p at each change in the sample D_p values. Moreover, the "measurement" is carried out if the following condition is met:

$$\lim_{S} D_{p(i)} = \circledcirc_i \, , where \circledcirc_i = P_i \tag{35}$$

Graphically, the signaling path (signal transduction) can be represented by the following graph:

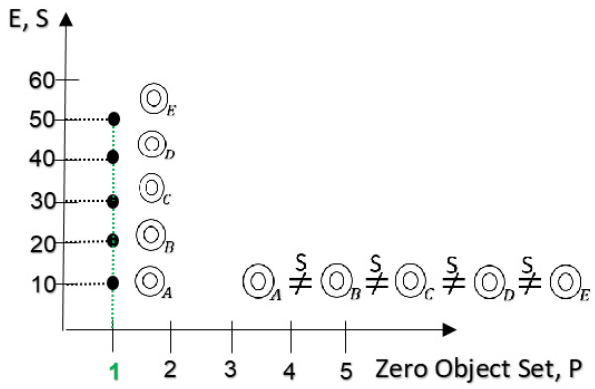


Fig 7.

4. Conclusion

In this article, we have presented an initial description of the enzyme kinetics by the methods of the Sense Theory.

We believe that a radically new approach with new tools for analyzing biochemical data will help us get closer to diagnosing many diseases at its early stages. We hope that our decent work will help other AI researchers in their life endeavors.

To be continued.

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Appendix

 - abbreviation of two semantic operations:  and 

APPs	Complement proteins	Transport proteins	Enzymes	Protein hormones	Immunoglobulins	Clotting factors	Proteinase inhibitors	Oncotic pressure	buffer capacities of plasma
C-reactive protein	C3	Transferrin	Aminotransferase	Insulin	IgA	Prothrombin	α1 - Antitrypsin	Albumen, all proteins	All proteins
Serum amyloid A	C4	Thyroxine-binding protein	Lipase	Glucagon	IgM	Factor VIII	α2 - Macroglobulin		
Haptoglobin		Albumen	Amylase	Vasopressin	IgG	Fibrinogen			
Fibrinogen		Retinol-binding protein			IgE (general)				
		Ceroloplasmin							
		Sex hormone-binding globulin							
		D-binding protein							

Table 1.