A SIMPLE MODEL OF FLUID ABSORPTION IN THE GASTROINTESTINAL SYSTEM

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In this article, we present a simple mathematical model for water absorption in the gastrointestinal tract. We begin by developing models for nutrient transport across membranes, focusing on passive transport through diffusion and active transport via the Na+/K+ ATPase mechanism. Building on these models, we then define water absorption in greater detail. We analyse how variations in Na+ levels affect water absorption and examine the body's adaptive responses to dehydration.

ENOSTAVEN MODEL ABSORBCIJE TEKOČINE V PREBAVNEM TRAKU

V članku je predstavljen preprost matematični model absorpcije vode v prebavnem traktu. Na začetku razvijemo modele za prenos hranil čez membrane, pri čemer se osredotočimo na pasivni transport skozi difuzijo in aktivni transport prek mehanizma Na+/K+ ATPaze. Na teh modelih zgradimo podrobnejši opis absorpcije vode. Analiziramo, kako različne količine Na+ vplivajo na absorpcijo vode ter preučujemo prilagoditvene odzive telesa na dehidracijo.

1. Structure of gastrointestinal tract

The gastrointestinal (GI) tract is a complex system responsible for the digestion and absorption of nutrients. In its simplest form, the GI tract can be thought of as a long hollow tube with several key layers and parts, as illustrated in Figure 1. The innermost part, known as the lumen, is the hollow space within the GI tract through which food travels. Surrounding the lumen is the epithelium, a layer of cells that play a crucial role in absorption and secretion. Beyond the epithelium lies the mucosa, which includes the lamina propria - a connective tissue layer containing blood vessels, nerves, and lymphatics that support nutrient absorption and immune defence. This is followed by the muscularis mucosa, a thin layer of muscle that helps move the mucosa. Next is the submucosa, a thicker layer of connective tissue housing larger blood vessels, lymphatics, and nerves. The outer muscular layer, known as the muscularis externa, is responsible for the peristaltic movements that propel food along the GI tract. This structure is summarized from the detailed description presented in Chapter 18 of Ref. [1].

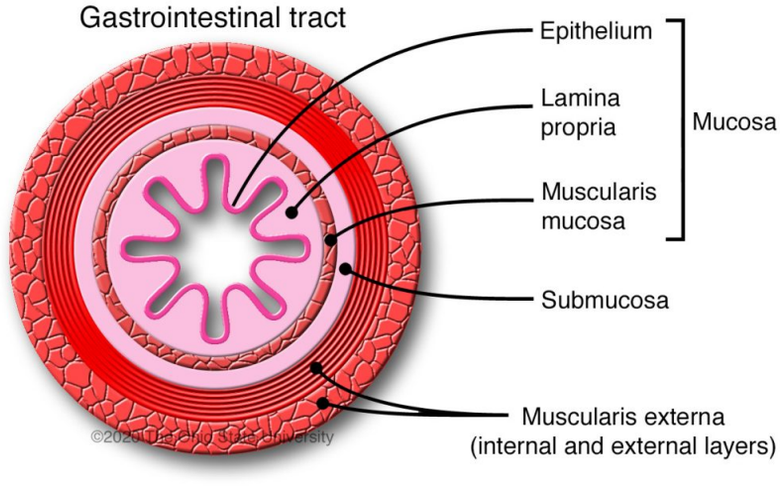

Figure 1. Structure of the GI tract, reproduced from Ref. [2]

In the context of the gastrointestinal tract, the interstitium is the space containing interstitial fluid, located between cells and blood vessels. In Figure 2, the interstitium is not explicitly labelled. However, it is located in the connective tissue components of the gastrointestinal wall.

The primary functions of the gastrointestinal system include fluid absorption, propelling food down the tract, and protecting the body from harmful substances. Fluid absorption is a particularly vital function, ensuring that the body retains necessary water and nutrients. This process is enhanced by numerous small folds called villi which increase the surface area for absorption.

In this article, we construct a simple mathematical model of water absorption form the ingested liquids into the body. We consider a very simplified structure of the GI tract consisting of only three components: the lumen, the epithelial cells, and the interstitium, as illustrated in the Figure 2. In general, the epithelial cells are not permeable to water on their lumenal side. However, there are 0.7–1.5 nm pores through the tight junctions between epithelial cells that permit water to diffuse readily between the lumen and the interstitium.

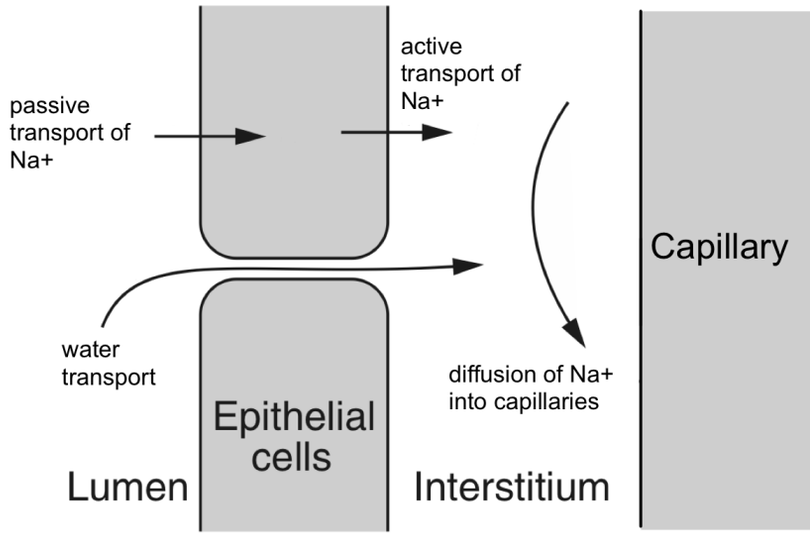

Figure 2. Simplified structure of GI tract, reproduced from Ref. [1]

Absorption of water through these pores is driven primarily by the Na+ gradient between the lumen and the interstitium. This gradient is created in several stages. In the first stage the concentration of Na+ ions is high in the lumen primarily due to the nutrients and electrolytes that are present in the ingested food and fluids. Meanwhile the concentration is low in the epithelial cells which allows Na+ to be transported passively with diffusion from the lumen to epithelium (move from a region of high concentration to a region of low concentration).

In the second stage Na+ is transported actively by a Na^+ - K^+ ATPase from the epithelium to the interstitium. The active transport needs energy, in our case the ATP, to transport Na+ against the gradient. Although the concentration of Na+ in the interstitium is not necessary higher than that in the cells, active transport is needed to maintain a low intracellular concentration of Na+ in epithelial cells. This then allows the first stage to happen again after the second stage is completed.

In the third stage, the concentration of Na+ is higher in the interstitium then in the lumen. The flow of water through tight junctions is then driven by the osmotic pressure difference between the lumen and the interstitium.

In the last stage the Na+ ions together with transported water in the interstitial fluid diffuse

into the capillaries due to the concentration gradient and are carried away by the bloodstream.

In the following we present a model of the passive and active transport. We will then use these models to describe the water transport in our system.

2. Passive transport

In this section we analyse the passive transport of chemicals with diffusion. We follow Chapter 2.2 in Ref. [1]. Assume that we have some chemical species U. The *conservation law* describes the change of U in a region Ω:

$$\text{rate of change of } U = \text{rate of production of } U + \text{accumulation of } U \text{ due to transport}$$

Mathematically, we can write this law as:

$$\frac{\mathrm{d}}{\mathrm{dt}} \int_\Omega u \, dV = \int_\Omega f \, dV - \int_{\partial\Omega} \vec{J} \cdot \vec{n} \, dA, \tag{1}$$

where u is the concentration of chemical species U, f the production density of U per unit volume, $\vec{J}$ the flux density vector of U and $\vec{n}$ the unit normal vector pointing outward of the boundary $\partial\Omega$. Note that if $\vec{J} \cdot \vec{n} < 0$ ($\vec{J} \cdot \vec{n} > 0$), then U is flowing into (out of) the area Ω respectively.

Using the *divergence theorem*, we can reformulate Equation (1) to

$$\frac{\mathrm{d}}{\mathrm{dt}} \int_\Omega u \, dV = \int_\Omega f \, dV - \int_\Omega \nabla \vec{J} \, dV \tag{2}$$

$$= \int_\Omega (f - \nabla \vec{J}) \, dV. \tag{3}$$

Assume that u is continuously differentiable. Then Equation (3) simplifies further:

$$\int_\Omega \frac{\mathrm{d}}{\mathrm{dt}} u \, dV = \int_\Omega (f - \nabla \vec{J}) \, dV \tag{4}$$

$$\Rightarrow \frac{\mathrm{d}}{\mathrm{dt}} u \overset{a.e.}{=} f - \nabla \vec{J} \, dV. \tag{5}$$

If we assume that both hand sides are continuous, we can drop the *a.e.* ("almost everywhere") and get the so-called *conservation equation*

$$\frac{\mathrm{d}}{\mathrm{dt}} u = f - \nabla \vec{J} \, dV. \tag{6}$$

Next we formulate *Fick's first law*. A steeper gradient indicates a larger difference in concentration over a small distance, therefore it is intuitively reasonable and also found experimentally, that it results in a greater flux. Moreover, the flow of substances is from high to low concentration regions. Taking all these observations into account Fick's first law reads the following.

Theorem 1 (Fick's first law). *Fick's first law of diffusion states that the flux $\vec{J}$ of solute through a membrane is proportional to the concentration gradient across the membrane*

$$\vec{J} = -D \nabla u, \tag{7}$$

where D is the diffusion coefficient and u is the concentration of the solute.

Combining the conservation equation and Fick's first law we get the *reaction-diffusion equation* that reads

$$\frac{\partial u}{\partial t} = D \nabla^2 u + f. \tag{8}$$

Next we would like to derive an analogue of this equation that describes diffusion through a membrane. Suppose that a membrane separates two large reservoirs of a dilute chemical, with concentration c_l on the left (at $x = 0$), and concentration c_r on the right (at $x = L$). Assume that there is no production of U ($f = 0$). The diffusion equation (in one dimension) then reads

$$\frac{\partial c}{\partial t} = D\frac{\partial^2 c}{\partial x^2}, \tag{9}$$

with boundary conditions $c(0, t) = c_l$ and $c(L, t) = c_r$.

For simplification we observe the steady state solution ($\frac{\partial c}{\partial t} = 0$), so that $-D\frac{\partial^2 c}{\partial x^2} = 0$. It follows that $c(x) = ax + b$, for some constants a and b. After applying the boundary conditions, we get

$$c(x) = c_l + (c_r - c_l)\frac{x}{L}. \tag{10}$$

From Fick's first law it follows that

$$J = \frac{D}{L}(c_l - c_r). \tag{11}$$

The ratio $\frac{D}{L}$ is called *permeability* and it is the effective resistance of the membrane.

3. Active transport

In this section we analyse the Na+ transport through a membrane of epithelial cells using the Na+ / K+ ATPase, also known as sodium-potassium pump. The Na+/K+-ATPase is an enzyme found in the plasma membrane that allows the Na+ ions to travel against the gradient. To analyse its behaviour, we first have to develop some basic theory about enzyme kinetics.

3.1 The law of mass action

We start with the fundamental law of reaction kinetics, also known as the *law of mass action*. We consider a very simple irreversible reaction, where two reactants A and B form a product C:

$$\mathrm{A} + \mathrm{B} \xrightarrow{k} \mathrm{C},$$

where k is the so called reaction rate that will be defined precisely below.

Let [A], [B] and [C] be the concentrations of A, B and C, respectively. We assume that the change of product in time corresponds to the number of collisions (encounters of molecules) between molecules A and B, multiplied by the probability that reaction happens in case of a collision.

The expected number of collisions per unit time is taken to be proportional to the product of the concentrations of A and B, with an appropriate factor r_1. Let r_2 be a fraction of these collisions that lead to a reaction. Therefore, the change of concentration of C over time is defined as:

$$\frac{d[\mathrm{C}]}{dt} = r_1[\mathrm{A}][\mathrm{B}] \cdot r_2 = k[\mathrm{A}][\mathrm{B}],$$

where $k = r_1 r_2$. This equation is the so-called *law of mass action*.

Similarly, we find the equations for concentrations of A and B:

$$\frac{d[\mathrm{A}]}{dt} = -k[\mathrm{A}][\mathrm{B}]$$
$$\frac{d[\mathrm{B}]}{dt} = -k[\mathrm{A}][\mathrm{B}].$$

For thermodynamic reasons all reactions proceed in both directions. If one direction is really slow we neglect it and proceed as above. Let us now define the law of mass action for the reversible reaction. We consider the reaction

$$A + B \underset{k_-}{\overset{k_+}{\rightleftharpoons}} C,$$

with k_+ and k_- denoting the forward and reverse rate constants of reaction, respectively.

Since the concentration of A decreases with the forward reaction (A is consumed) and increases with the reverse reaction (A is produced), the rate of change of [A] reads:

$$\frac{d[\mathrm{A}]}{dt} = k_-[\mathrm{C}] - k_+[\mathrm{A}][\mathrm{B}].$$

Simmilarly,

$$\frac{d[\mathrm{B}]}{dt} = k_-[\mathrm{C}] - k_+[\mathrm{A}][\mathrm{B}]$$
$$\frac{d[\mathrm{C}]}{dt} = k_+[\mathrm{A}][\mathrm{B}] - k_-[\mathrm{C}].$$

3.2 Enzyme kinetics

In Section 3.1 we focus on simple reactions where all reactants are fully converted into products and thus consumed in the process. However, a different scenario emerges when enzymes are involved. Enzymes act as catalysts in biochemical reactions, meaning that they facilitate these reactions without being consumed or altered themselves. They ”take” a substrate molecule and convert it into a product. Unlike chemical reaction kinetics, enzyme kinetics exhibit the phenomenon of saturation. At very high substrate concentrations, the reaction rate reaches a maximum value, v_{max}, beyond which it cannot increase. In this section we follow Section 5.1.3 in Ref. [3].

Enzymes (E) function as biocatalysts by forming a complex (C) with their substrate (S), in which the product (P) is generated. This process can be written with the following chemical reaction

$$\mathrm{E} + \mathrm{S} \underset{k_{-1}}{\overset{k_1}{\rightleftharpoons}} \mathrm{C} \xrightarrow{k_2} \mathrm{E} + \mathrm{P}.$$

In many cases, it is practically impossible to retrieve the original substrate from the product, therefore we write the second reaction as irreversible. The rate constants k_1 and k_{-1} represent the association and dissociation rates of the enzyme-substrate complex, respectively.

In the following we define the following variables:

$$s = [\mathrm{S}], \qquad e = [\mathrm{E}], \qquad c = [\mathrm{C}], \qquad p = [\mathrm{P}].$$

Now, following the law of mass action we can write the following system of differential equations

$$\frac{ds}{dt} = k_{-1}c - k_1 se,$$
$$\frac{de}{dt} = k_{-1}c + k_2 c - k_1 se,$$
$$\frac{dc}{dt} = k_1 se - k_{-1}c - k_2 c,$$
$$\frac{dp}{dt} = k_2 c.$$

Since there is only substrate and the enzyme present at the beginning of the reaction, the initial conditions are

$$s(0) = s_0, \qquad e(0) = e_0, \qquad c(0) = 0, \qquad p(0) = 0.$$

To simplify the system, we neglect the last equation for a moment and observe that $\frac{\mathrm{d}}{\mathrm{dt}}(e+c) = 0$, therefore $e + c = e_0$. Using this observation the system reduces to:

$$\frac{ds}{dt} = -k_1 s e_0 + (k_1 s + k_{-1})c, \tag{12}$$

$$\frac{dc}{dt} = k_1 s e_0 - (k_1 s + k_{-1} + k_2)c. \tag{13}$$

Next, we nondimensionalise the system by introducing the following dimensionless variables:

$$\tau = (k_1 e_0)t, \qquad u(\tau) = s(t)/s_0, \qquad v(\tau) = c(t)/e_0,$$
$$\lambda = \frac{k_2}{k_1 s_0}, \qquad K = \frac{k_{-1} + k_2}{k_1 s_0} = \frac{K_m}{s_0} \qquad \epsilon = \frac{e_0}{s_0}.$$

Hence, Equation (12) rewrites as:

$$\frac{d}{d\tau}u(\tau) = \frac{d}{d\tau}\frac{s(t)}{s_0} = \frac{1}{s_0}\frac{ds(t)}{dt}\frac{dt}{d\tau} \tag{14}$$

$$= \frac{1}{s_0}\frac{1}{k_1 e_0}[-k_1 s e_0 + (k_1 s + k_{-1})c] \tag{15}$$

$$= -\frac{s}{s_0} + \frac{1}{e_0}\frac{sc}{s_0} + \frac{k_{-1}}{k_1}\frac{c}{s_0 e_0} \tag{16}$$

$$= -u(\tau) + u(\tau)v(\tau) + (K - \lambda)v(\tau) \tag{17}$$

$$= -u + (u + K - \lambda) \tag{18}$$

and Equation (13)

$$\frac{d}{d\tau}v(\tau) = \frac{1}{\epsilon}[u - (u + K)v], \tag{19}$$

with initial conditions $u(0) = \frac{s(0)}{s_0} = 1$ and $v(0) = \frac{c(0)}{e_0} = 0$.

In biochemical reactions there is usually much less enzyme than substrate present, therefore $\epsilon = \frac{e_0}{s_0} \ll 1$. This means that there are two processes on two different time scales: the process of v is very fast in comparison to the one of u. If we rewrite Equation (19) as $\epsilon\frac{dv}{d\tau} = (u - (u + K)v)$ and send $\epsilon \to \infty$, we can solve the right hand side for u:

$$v = \frac{u}{u + K}.$$

This means that when ϵ is very small, v lies on the manifold $\frac{u}{u+K}$, which we name *slow manifold* and is plotted in Figure 3. To see how the solution travels on this manifold, we insert $v = \frac{u}{u+K}$ in Equation (18):

$$\frac{du}{d\tau} = -u + (u + K - \lambda)\frac{u}{u + K} = -\frac{\lambda u}{u + K},$$

which is negative for $u > 0$. Therefore, the solution travels down the manifold, as seen in Figure 3.

To analyse the system even further, we introduce a new variable $\hat{\tau} = \frac{1}{\epsilon}\tau$, which transforms the system to

$$\frac{du}{dt} = \epsilon(-u + (u + K - \lambda)v),$$
$$\frac{dv}{dt} = u - (u + K)v.$$

Taking $\epsilon \to 0$ results u being constant, $u(\hat{\tau}) = u_0$, and $\frac{dv}{dt} = u_0 - (u_0 + K)v$, which is solvable. The solution reads:

$$v(\hat{\tau}) = \frac{u_0}{u_0 + K} + v_0 e^{-(u_0+K)\hat{\tau}}.$$

For large times

$$\lim_{\hat{\tau}\to\infty} v(\hat{\tau}) = \frac{u_0}{u_0 + K}.$$

This means that for a small ϵ the variable u is constant and v eventually settles on the slow manifold, as seen in Figure 3.

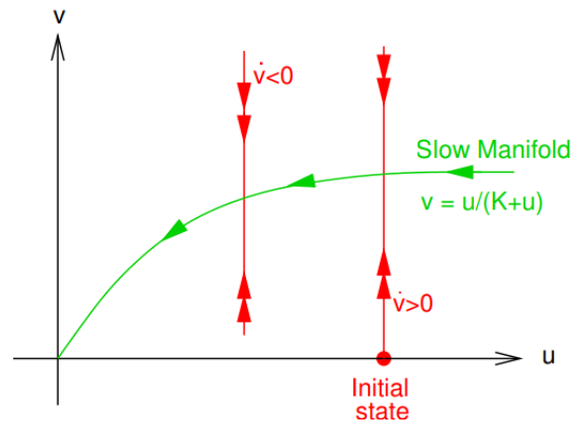

Figure 3. Slow manifold, reproduced from Ref. [3]

From this we can conclude that the formation of the complexes C (here represented with the variable v) tends quickly to the equilibrium, while the substrate S (represented with u) almost does not change. Then the substrate is converted along the slow manifold into the product until it is used up.

This behaviour allows us to use $v = \frac{u}{u+K}$ as a good app roximation. Since our main interest is to express the rate of product formation $V := \frac{dp}{dt}$, we use this aproximation and substitute back to the original variables

$$V = \frac{dp}{dt} = k_2 c = e_0 k_2 v = e_0 k_2 \frac{u}{u+K} = e_0 k_2 \frac{\frac{s}{s_0}}{\frac{s}{s_0} + K\frac{s}{s_0}} = v_{\max}\frac{s}{K_m + s}.$$

This equation is called the *Michaelis–Menten law.*

3.3 Hill equation and application to the Na+/K+ ATPase

For enzymes with cooperative binding sites, the Michaelis–Menten equation can be generalized using the Hill equation. The Hill equation is particularly useful for describing enzymes with multiple binding sites that exhibit cooperative interactions. The formulation of the Hill equation is explained in more detail in Section 1.4.4 in Ref. [1]. In the Hill equation, the rate of product formation V is given by

$$V = \frac{v_{\max}[\mathrm{S}]^n}{K_m^n + [\mathrm{S}]^n}$$

where n is the Hill coefficient, indicating the degree of cooperativity among the binding sites. For the Na^+/K^+ ATPase, which typically binds three Na^+ ions cooperatively, we can set $n = 3$.

However, instead of focusing on the formation of a product, we are interested in the rate at which Na^+ ions are transported through the membrane by the Na^+/K^+ ATPase. The Na^+/K^+ ATPase transports Na^+ ions from the cytoplasm to the extracellular space which is a crucial process for maintaining cellular ion gradients. The rate of Na^+ ion transport, J_{Na}, can be defined analogously to the rate of product formation in enzyme kinetics. Hence, we adapt our Hill equation to describe

this transport rate:

$$J_{\mathrm{Na}} = \frac{J_{\max}[\mathrm{Na}^+]^3}{K_m^3 + [\mathrm{Na}^+]^3},$$

where J_{Na} is the rate of Na^+ ion transport, $J_{\max}$ is the maximum rate of Na^+ ion transport when the Na^+/K^+ ATPase is fully saturated with Na^+, $[Na^+]$ is the concentration of Na^+ ions in the cytoplasm, and K_m is the apparent Michaelis constant for Na^+ binding.

This equation highlights the cooperative nature of Na^+ binding to the Na^+/K^+ ATPase, where the binding of one Na^+ ion increases the affinity of the enzyme for additional Na^+ ions, facilitating efficient transport. The Hill coefficient $n = 3$ indicates a strong cooperative interaction among the three Na^+ binding sites on the enzyme.

4. A simple model of water absorption

With the models of passive and active transport developed in the previous sections, we now develop a model of water transport.

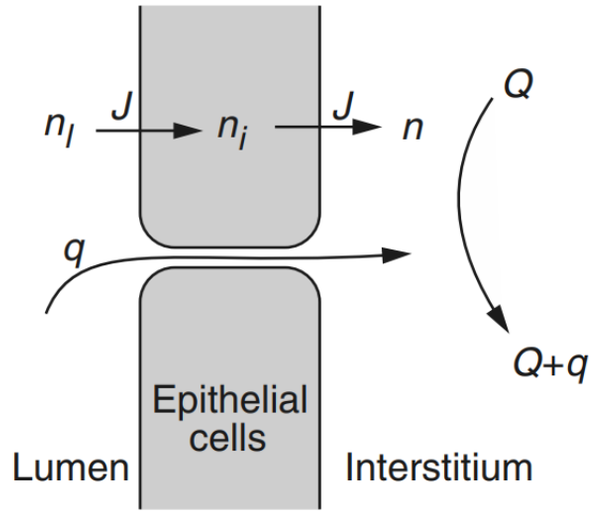

Figure 4. Flows of Na+ and water and concentrations in different parts of the GI structure, reproduced from Ref. [1]

We suppose that the Na+ concentration is n_l in the lumen, n_i in the interior of the cells, and that the concentration of all osmolites in the interstitium is n. Let J denote the flow of Na+ from the lumen to the interior of the cells. The transport is passive, therefore, we follow the model defined in Section 2:

$$J = g(n_i - n_l), \tag{20}$$

where g is permeability of the membrane. In steady state, the conservation of Na+ implies that the flow from the lumen into the cells is equal to the flow from the cells to the interstitium. Therefore

$$J = f(n_i), \tag{21}$$

where $f(n) = \frac{J_{\max} n^3}{N^3 + n^3}$ is the function defined in section 3.3, where $N = K_m$.

With q we define the flow of water through the tight junctions which is driven by the osmotic pressure difference between the lumen and the interstitium. Therefore

$$Rq = n - n_l, \tag{22}$$

where R is the resistance of the tight junctions.

Next, we assume that capillary flow provides a movement of fluid into and out of the interstitium. The incoming fluid has a flow rate of Q and a concentration of osmolites n_0, whereas the outgoing fluid carries osmolites at a flow rate of $Q+q$ with a concentration of n. The conservation of Na+ again implies that the flow of incoming fluid from the cell to the interstitium (active transport) is equal to the difference between the outflow of osmolites and influx of fluid with the capillaries. Therefore

$$(Q+q)n - Qn_0 = f(n_i). \tag{23}$$

If we express n from Equation (22) and plug it into Equation (23), we get

$$Rq^2 + (RQ + n_l)q + Q(n_l - n_0) - f(n_i) = 0 \tag{24}$$

Then we nondimensionalize this problem by scaling all concentrations by N, setting $u_j = \frac{n_j}{N}$ for $i \in \{l, i, 0\}$ and $y = \frac{q}{Q}$, so Equation (24) reads

$$RQ^2y^2 + (RQ + Nu_l)Qy + Q(Nu_l - Nu_0) - \frac{J_{\max}N^3u_i^3}{N^3 + N^3u_i^3} = 0, \tag{25}$$

where the last term is evaluated function f at Nu_i. In the next step we divide Equation (25) with NQ,

$$\frac{RQ}{N}y^2 + (\frac{RQ}{N} + u_l)y + (u_l - u_0) - \frac{J_{\max}}{NQ}\frac{u_i^3}{1+u_i^3} = 0. \tag{26}$$

We now define the following variables: $\rho = \frac{RQ}{N}$, $\beta = \frac{J_{\max}}{gN}$, $\gamma = \frac{g}{Q}$ and $F(u) = \frac{u^3}{1+u^3}$. Equation (26) now reads

$$\rho y^2 + (\rho + u_l)y + (u_l - u_0) - \gamma\beta F(u_i) = 0.$$

Since f is a positive, monotone increasing function, we can express n_l with n_i uniquely, from Equation (20) and (21),

$$n_l = n_i + \frac{1}{g}f(n_i).$$

With the new rescaled variables this reads

$$u_l = u_i + \beta F(u_i) \tag{27}$$

Finally, we define $\kappa = u_i - u_0 + (1-\gamma)\beta F(u_i)$. Together with Equation (27) the final form of Equation (24) reads:

$$\rho y^2 + (\rho + u_l)y + \kappa = 0. \tag{28}$$

The main goal of this article is to analyse the water absorption q, now rescaled and denoted by y. Therefore, we try to solve Equation (28) for y as a function of u_l. The easiest way to do so is to view the solution as the curve $y = y(u_i)$, $u_l = u_l(u_i)$, parameterized by u_i, since u_l is determined by u_i from Equation (27).

We observe that Equation (28) is a quadratic polynomial in y. Its roots are $y_1 = \frac{-\rho - u_l - \sqrt{D}}{2\rho}$ and $y_2 = \frac{-\rho - u_l + \sqrt{D}}{2\rho}$, with $D = (\rho + u_l)^2 - 4\rho\kappa$. We observe that y_1 is always negative, therefore it is not biologically relevant, whereas y_2 is positive if and only if $\kappa < 0$.

To analyse the root y_2, we first delve into the behaviour of $\kappa(u_i) = u_i - u_0 - (\gamma - 1)\beta F(u_i)$ as a function of u_i. The behaviour of greatest interest occurs when $\beta(\gamma - 1) \gg 1$. Under this condition κ is negative at the origin $u_i = 0$. The derivative reads

$$\kappa'(u_i) = 1 - \beta(\gamma - 1)\frac{u_i^2}{(1+u_i^3)^2}.$$

Note that for small u_i the derivative is positive and therefore κ increases. After that it becomes negative, so κ decreases, however for large u_i the fraction in the derivative becomes small,

$$\lim_{u_i \to \infty} \frac{u_i^2}{(1+u_i^3)^2} = \lim_{u_i \to \infty} \frac{2u_i}{6(1+u_i^3)u_i^2} = 0,$$

resulting the increase of κ again. Therefore, assuming that $\beta(\gamma - 1) \gg 1$, κ becomes an N-shaped function of u_i, as seen in Figure 5.

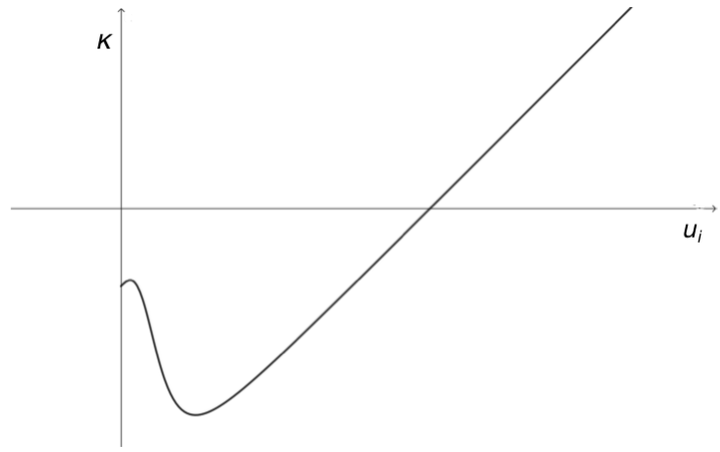

Figure 5. Sketch of κ as a N-shaped function

This N-shaped behaviour of κ translates to a N-shaped behaviour for the positive root $y_2(u_i) = y_2(\kappa(u_i))$ of Equation (28). Since y_2 is a monotonically decreasing function of κ, the N-shape will invert, since when κ increases y_2 decreases, as seen in Figure 6. Specifically, with $u_l = u_l(u_i) = 0$, the root y_2 is positive. As u_l changes, this root initially decreases to a minimum value, then increases to a maximum value, and ultimately decreases again, becoming negative. This behaviour is illustrated in figure 6, using concrete parameter values $\rho = 1$, $u_0 = 1$, $\beta = 1$, and $\gamma = 10$.

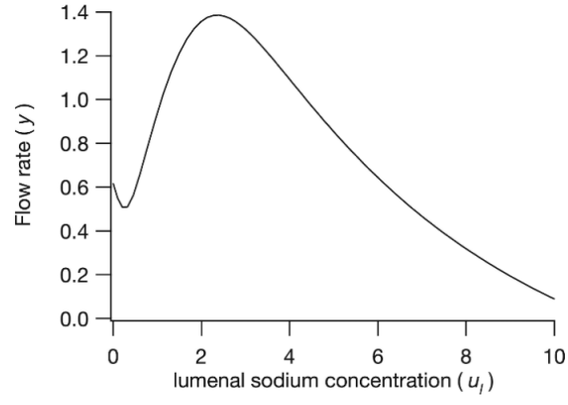

Figure 6. Flux of water through the epithelial membrane plotted as a function of luminal Na+ concentration, reproduced from Ref. [3]

This finding has a very interesting application. It suggests that the absorption of water can be optimized by adjusting the sodium (Na^+) level in the luminal water. Consequently, hydration is more efficient with electrolyte-containing fluids compared to pure water. However, an excessive

amount of Na^+ can have the adverse effect of dehydrating the interstitium. This is a localized effect since water is reabsorbed further along the tract.

When a person becomes dehydrated the adrenal glands release a lot of aldosterone. This hormone helps epithelial cells transport more Na+ by increasing the production of channel and pump proteins. This boosts both the passive and active transport of Na+. Over time, a person can adapt to heavy exercise in hot weather. This adaptation happens because, over several weeks, the increased aldosterone secretion from the adrenal cortex reduces excessive Na+ loss in sweat, so the person does not need extra dietary Na+. However, losing K+ can still be a problem.

In this model, aldosterone's effect can be shown by increasing g (the conductivity of Na+ transport) and/or J_{max} (the maximum rate of active Na+ pumping). It's clear that the total Na+ flux $J = f(n_i)$ and the water flux q both increase if either g or J_{max} go up. But this increase has limits. When g becomes very large, n_i gets close to n_l, making

$$\lim_{g\to\infty} J = f(n_l)$$

and

$$\lim_{g\to\infty} q = Q(\frac{n_0}{n_l} - 1) + \frac{f(n_l)}{n_l}$$

when $R = 0$. So, when a person is dehydrated, aldosterone helps increase Na+ absorption and reduce water loss.

5. Conclusion

This paper presented a mathematical model to describe water absorption in the gastrointestinal (GI) tract, emphasizing the role of passive and active transport mechanisms, particularly the sodium (Na+) gradients. The findings highlight how varying Na+ levels can either enhance or inhibit water absorption, suggesting that electrolyte-containing fluids are more effective for hydration. For those interested in a deeper understanding of the biological background behind these processes, the chapter on intestinal water and electrolyte transport by Chang and Leung [4] provides valuable additional context and detail.

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