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A Propose Boyo Universal Theory of Therapy (BUTT) in Focus, Posits that Globalized Control and Regulation of Signaling Mediums Such as RTK, MPK, MAPK, MPKK, ERT

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Abstract

The finding determines the dependence of body processes on STP and MCT, six processes which occur in the isolated rabbit ileum were used to prove the statement of Boy'o Universal Theory of therapy 1(BUTT 1), mainly effects of extract and Autonomic drugs on isolated rabbit ileum together with novel mathematical principles were used to show that seven normal processes (motility, secretion, metabolism, circulation, immunoreactivity, absorption, digestion) of the isolated rabbit ileum are directly proportional to activated STP and MCT and inversely proportional to inactivated STP and MCT given as R_s $R_s \alpha \frac{N_i}{N_y} and\ thus\ R_s = K\frac{N_i}{N_y}$ Acknowledge that from the above relation, STP magnitude responses R_{sm} was used base on the assumption that all body processes are responses of STP and MCT, in other words, body processes are manifestation of STP response, thus First and foremost, I posit that any number of response (R_{sn}) or magnitude of response (R_{sm}) is a function of or proportional to activated STP backbones (N_i) and inversely proportional to inactivated STP backbone (N_y).

i.e. $R_s \alpha \frac{N_i}{N_y} and\ thus\ R_s = K\frac{N_i}{N_y}$ (1)

K is a universal constant of proportionality of therapy.

To estimate (N_i) for each receptor in the isolated rabbit ileum, I determined number of molecules (N) for each drug and extract and number of molecules (N_x) towards the receptor direction, also using a biomarker (isolated rabbit ileum) the magnitude of effect (contraction and relaxation) of extract/drugs on the ileum was determined by combination interactions, novel mathematical equations and quantum physics concepts, which gave me insight about the autonomic nature of the extract. Again from the combination interaction of antagonists and agonists with the extract as well as novel mathematical equation and calculation, I deduced the receptor types which combined with the extract, the couple receptor types, and the linked cascades in unlimited sense of their complexity. Again, in spite that past finding have unraveled various receptor types, limitations of characterization and crystallography abounds and so warrant use of mathematic to gain useful insight, interestingly, observed height, STP components' flux time and mathematically determined magnitude of response (R_{sm}) shows strong correlation, of course as found in other findings, we assumed that alternating responses seen was due to bimodal on or off switches peculiar to STPs and MCT, these act in turn to activate many targets and therefore more than two responses might have occurred as predicted from various novel equation and calculation. The seven processes investigated are; directly; motility, indirectly; secretion, epithelial protein synthesis and transluminal absorption, digestion, circulation, and immunoreactive processes respectively, as a matter of veracity, it important to state that only the result of isolated rabbit ileum motility was obtained from which other processes were inferred, from perspective of STP and MCT vital cascade components, using platform of mathematics, in so far as this remain the case, the principle of hypothesis was investigated and lead to the emergence of Boy'o Universal Theory of Therapy (BUTT) 1, Theory 1 of BUTT called dependence theory, states that, any process; pathologic, physiologic, biochemical, genetic, metabolic and congenital occurring in the body, including drug efficacy and drawback, is directly proportional to activated signal transduction pathways (STPs) and inversely proportional to inactivated ones provided that MCT remains constant.

The result was subject to both statistical analysis and mathematical calculation, for the statistical result standard deviation was 1.05 and magnitude of response (R_{sm}) was 27.996 at number of active components (Nc1) of 0.7 to indicate inactivation of STPs, other values show STPs activation, a fact that is of clinical, pharmacological, and biomedical importance.

Keywords: BUTT theory; Drug adverse effects; Quantum concept

Introduction

The finding test the hypothetical statement of theory 1 of Boy'o universal theory of therapy (BUTT 1), it maintained that any process in body depends on STP and MCT interaction, signaling medium enumerated in most literature shed light on genesis of various processes in the body, be they binding or non-binding actions, STP encompass complex interrelated series of communicating cascades of proteins and enzymes, each cascade have particular attributes which they utilized in processes of transduction, they transmit signals by assigning roles to each cascade components and in this regard, adapter,

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recruiter, amplifier, deactivators and other forms exist, so that by 'on' and 'off' mode signal is amplify down the cascade stream, how these events are mediated by many interconnected pathway backbone remains to be demystified completely, which is why I incorporated mathematical equations and calculation in this study, importantly, the calculation and novel equation serve as effective tool which handles the complexity of STP and MCT, the organ bath/drug bioassay essentially ensured determination of the autonomic nature of the extract from contraction and relaxation response observed when extract and drugs interact with the receptors on the isolated rabbit ileum in organ bath, the autonomic nature of extract so determined, furnished the result from which the mechanism of action of extract as it relate to signal transduction pathways {STPs} of seven processes of the isolated rabbit ileum were inferred, in accordance to stipulation of previous literatures, for instance [1] shows that certain autonomic action are mediated via muscarinic and adrenergic receptors as well as their respective STPs such as cAM-IP3-PKA pathways. ras/raf/MEK/ERK signal transduction cascade is at the heart of signaling network that governs proliferation, differentiation and cell survival [2].

Additionally, the signal transduction of most receptors types and subtypes activated in seven GIT processes (Motility, secretion, metabolism, absorption, immunoreactivity, circulation and digestion) of the isolated rabbit ileum which have bearing to the bioassay had been described by past findings, therefore, it was possible to asses STPs cascade involves in the seven processes with aid of quantum physics and mathematics principles, and then go ahead to prove that their occurrence would not have been without the presence of STPs, actual experiment use motility processes, from which together with mathematical principles other STPs mediated processes were determined, [3] shows that MPKs pathways are positively regulated by several mechanisms which include scaffold proteins. The layout of this finding can be extrapolated to ones involving body abnormality and drug toxicity, given that STPs determine all processes of the body, body processes are manifestation of STP responses, instance gene transcription, contraction of heart muscle, cellular uptake of glucose etc are STP responses in physiologic manifestation. The benefits of application of the theory as detail in subsequent sections of this finding are priceless. Imagine a world free of body abnormality and drug adverse effects (side effect, toxicity etc). Additionally, effective determination of Rsm values will form linked between used and gauging of STPs activities in biomedical research, drug development, drug safety profiles determination, and use of target hit in relation to treatment.

Incidence

The incidence of body abnormalities and drug toxicity as they relate to STP and MCT certainly could be said to have assumed a universal status, in that STP and MCT are cosmopolitan in the body, again all diseases, abnormalities, normal processes and drug uses in the world have STPs components.

Statement of the problem

The proposed concept that all processes occurring in the body depends on STP and MCT remains to be proved, again the fact that all body processes are manifestation of STP and MCT responses remains to be validated, especially looking at it far reaching implication on theory 2 and 3 of BUTT which aim to control and regulate STP and MCT globally, treat abnormalities and eliminate drug adverse effects, for in spite that these incapacitating abnormalities affect everyone in all the nook and cranny of the universe irrespective of the geographical nature of the place across the globe, there is limitedness in application, owing to the inability of research to unravel limitless realistic benefits of STP as sort by these findings, investigators lacking in STP assessment equipments abound, they can use BUTT1 principles.

Aims and Objective

To know the sympathetic and parasympathetic nature of the extract from drug action on smooth muscles of the rabbit ileum

To determine from the result of combination interactions; quantum physics, chemistry principles and new mathematical equations, the types and subtypes of receptors, mechanism of action, receptor selectivity of extract and drug,

To from perspective of synergism, antagonism interactions get insight about dependence of seven processes of isolated rabbit ileum on STP and MC

To validate the propounded BUTT1 with aid of the statement of the hypothesis, mathematical and experimental results.

To experimentally (biomarker bioassay) and empirically, using mathematical equation and STP diagrams, show how the experimental result confirms the hypothetical statement as well as the propounded theory

Scope and Limitation

Scope

The scope of this finding encompass the fields of human physiology, clinical pharmacology, gastroenterology, neurology, proteomic, transcriptomic and genetic (genomics) and molecular biology, human anatomy and mathematics all of which are related fields of biomedical sciences, the array and the expanse of the scope becomes inevitable due to complex nature of STP in GIT motility and other processes.

Limitations

The limitations lies in the fact that animal GIT and not human GIT was used in the bioassay which is also an in vitro exercise. Again lack of logistics was a major experimental constrained in that STP in pragmatic aspect use bioassay of extract and notwithstanding the validity of experimental exercise, advance approach with sophisticated equipment would have been preferred.

Another notable limitation is accurate protein characterization and data analysis in field of proteomics which need novel methods of investigation occasioned by complex nature of novel biomedical ideas like this one, requiring proteomics instrumental arsenal, Again, high avalanche of data output needs new mathematical and computation models [4].

Justification of the Study

It will lay foundation work for validation of theory 2 and 3, again these theories aim to treat completely and successfully various abnormalities, eliminate drug toxicity, and open diverse opportunity in biomedical research, especially use of STP and MCT in treatment and drug formulation.

More so, Recent and past findings indicated that small sample size, limited data, techniques and result of most studies makes proteomics and relevant findings underpowered to detect small to moderate differences, which necessitate the novel mathematical formula and use of some concepts of quantum physics, the equations I derived and how it in vivo application, probe concentration and number of molecule can be related to STP for vital deductions so that individual view of cascades

and cascade components can be linked together for accurate global view. All of these problems and solution proffered justify the laudable effort of these findings. The finding once again will be of important benefits to future research, the hallmark of STPs findings will be ability to combine probe+medcament+labbeled isotope at concentration or number of molecules deem save in addition to on screen in vivo real time visualization of their actions [5] also mathematical insight will aid target hit in research and clinical setting.

Statement of Hypothesis

(1) It states that any processes in the body is directly proportional to activated signal transduction pathways (STP) and inversely proportional to inactivated ones if MCT remains constant.

(2). Body processes are manifestation of STP and MCT responses, in order words body processes are STP and MCT responses.

Materials and Methodology

Animal: Rabbit, Organ bath apparatus, Tyrode solution, Petri dish, Scissors, Needle, syringe Thread, Oxygen source, Microdynamometer, Beaker, timer, kymograph, Dissecting kit, Transducers Thermometer, Conical flask, Weighing scale, Dissecting table, Distilled water, plants used for crude extraction of active ingredient are Senna Obtusifolia and Duranta repens

Addition of drug and extract assay

Plant collection and identification

The two plants used were obtained from Samaru in Zaria Kaduna state northern Nigeria, West Africa, *Senna obtusifolia* (V/No 1370) and *Durante repens* respectively belong to Leguminosae and Verbenacea family, they were identify by department of biological science herbarium faculty of science Ahmadu Bello University Zaria.

Plant sample preparation and phytochemical screening

Plant extract preparation: The leaves of the two plants obtained from their natural habitat were tried for two weeks at room temperature, subsequently, each of the tried leaves was macerated manually, then in the lab, the weight of the crushed extract was determined using electrical weighing apparatus, the determined weight of *Durante repens* and *Senna obtusifolia* are 132.09 g and 100.84 g respectively, after maceration of each of the plant leaves, the grit obtained was soaked in 900 ml (*Duranta repens*) and 600 ml (*Senna Obtusifolia*) of 700 ml methanol which was diluted with 300 ml of water and allowed to stand for 3 days, filtration was carried out with the aid of sieve covered with funnel in which liquid extract was separated from the Mac, the liquid extract was poured gently into the evaporating dish, set at 4°C to facilitate evaporation and solidification of extract isolated from the dried leaves of *D. repens* were exhaustively extracted with 85% methanol. NB in most methanolic extract Anthraquinone is absent and anticipated few number of contraction response as seen in the result of bioassay [6].

Experimental animal

The experimental animals used were matured New Zealand rabbits.

Animal preparation

The rabbits were housed in animal house for 3 days to acclimatize, they were given clean water and animal feed, also the animals were treated base on best international practice, eventually, they were fasted for 24 hours in order to allow for clearance of ileal luminal contents, each rabbits was then sacrificed humanly, and many pieces of the Ileum of about 3 cm were dissected out and carefully placed into Petri dish containing tyrod solution, after aeration with oxygen, a thread was tied through one of the lower wall of the 3 cm piece of the ileum, another thread was sewed in the upper part of the dissected 3 cm ileum which was attach to the organ bath, then speed and sensitivity of micro-dynamometer was adjusted and set at 24 mm/min and 2 mv respectively.

Addition/interaction of drug and extract

In the Bioassay therefore, the following interaction was done; 0.1 ml, 0.2 ml, 0.3 ml, 0.4 ml, 0.8 ml volumes respectively for each of 1 mg/ml, 10 mg/ml, 100 mg/ml doses of extracts alone or in combination with drugs, for drugs interactions doses and drugs used are ach 2 ug/ml, carbachol 0.5 ug/ml, physostigmine 80 ug/ml, adrenaline 10 ug/ml, atropine 10 ug/ml, nicotine 10 ug/ml, The drugs and was added in three mode, firstly in single form, secondly in double form and thirdly in triple form, similarly the extract in combination with the drug was added and their individual effect on the GIT was noted from reading on physiograph, also binding time between the drug or extract molecules and the various receptors on the GIT was noted, again we determined the terminal velocity, number of molecules from drug and extract concentration in order to know the number of cascade activated or inhibited and the time it was activated, other parameter are cascade flux velocity, response time of each cascade [7].

Measurement and calculations

Stock preparation of extract: 1 mg/ml, 10 mg/ml and 100 mg/ml of each of the extract was used in the combination interaction

Step (1) 1 gm of extract each was weighed and dissolved in 10 ml of water i.e.. 1/10=0.1 g/ml and 0.1 g/ml x 1000=100 mg/ml. Step (2) 1 ml of 100 mg/ml was taken from above to make up to 10 ml of the solution meaning that 100 mg/ml is contained in 10 m, X mg/ml is contained 1 ml And thus 100 x 1 /10= 10 mg/m. Step (3) 0.1 ml of 10 mg/ml was taken from above to make up to 1 ml of the solution thus 10 mg contains in 1 ml, X mg contain in 0.1 ml and 10 x 0.1/1=1 mg/ml, Therefore stock of extracts each are 1 mg/ml, 10 mg/ml and 100 mg/ml, Dose preparation of extract are 10 mg/ml is the stock concentration of the extract Thus 10 mg is contain in 1 ml, Xmg is contain in 0.1 ml, Therefore X mg= 0.1 x 10/1=1 mg, Meaning that,1 mg is contain in 1.038 kg (weight of rabbit), But X mg contain in 1 kg And thus 1 x 1/0.911=1.097 mg/kg, Therefore the dose of extract is 1.097 mg/kg.

Volume used: Volume=dose x weight of rabbit/stock, thus 1.097 x 0.911/10=0.1 ml Therefore 0.1 ml, then 0.2 ml, 0.4 ml and 0.8 ml were used Concentration of extract in organ bath also for *Duranta repens*

Stock concentration extract (C1)= 10 mg/ml, Volume of extract added (V1)= 0.2, Organ bath volume (V2)=10 ml, Organ bath concentration (C2)=?, C1V1=C2V2 and thus C2=C1V1/V2,=10 x 0.2 /10,=0.2 mg/ml, Thus, conc. of each extract in organ bath is 0.2 mg for 10 mg/ml (stock), 2 mg/ml for 100 mg/ml, (stock), 0.02 mg/ml for 1 mg/ml (stock), using above formula C2 at other volume (0.1,0.2,0.3,0.4,0.8) was be determined, it is crucial to note that in proving the BUTT we simply test hypothetical assumption using mass formulas, but putative probe could replace either drug or the extract since all are essentially molecules. Organ bath concentration for drugs include Ach C1=2 u, V1=0.2, C2=?, V2=10 ml, but C2=C1V1/V2 and 2 x 0.2/10=0.04 ug/m i.e. for Ach for others drugs the organ bath concentration (C2) are 0.04 ug/ml ach at 2 ug/ml (dose),0.01 ug/ml carbachol at 0.5 ug/ml (dose), 2.4 ug/ml physostigmine at 80 ug/ml (dose),0.4 ug/ml adrenaline at

10 ug/ml (dose),0.2 ug/ml atropine at 10 ug/ml (dose) and 0.6 ug/ml nicotine at 500 ug/ml (normal dose) NB volumes used are 0.1.0.2, 0.0.3, 0.4, 0.8 for each of above drug, extract [8].

Results and Discussion

Mathematical proof and results

Deductively and inductively, we determine binding time, and number of couple receptor, the flux time and other attribute which enable determination of various STPs involved, STPs proteins with low copies for detection can be known mathematically, since product of their action like wave or flux can be measured. Also Probe use is highly specific which justify number two reasons for conduct of the research, which is target hit at critical points; again safety lies in knowledge of globalised action of each STP components, this fit can never be attained without mathematics and quantum physics concepts (Figures 1-4).

I determined from the organ bath the effects of varying volume, concentration of drugs/extracts using certain parameter such as terminal speed of the molecules, stokes law, volumetric analysis parameters, firstly, to know the time either extract or drug or putative probes bind with receptor and the time the STPs backbone and the ones linked to it was activated, subsequently I determine the responses time taking into cognizance the time contraction or relaxation of the ileum starts, fortuitously, these novel approach indicated at what volume or concentration of molecules (drug or even probes) maximal or minimal response occurred,

From molecular kinetic Total number of molecules (N_x) towards x direction is given by $\frac{1 \text{ x } N \text{ x } Ad}{6}$ obtained from gas molecules kinetics.

Where Ad volume of the organ bath,

Thus similarly (N_x) values for other extract and drug was were determined, and so (N_x)

Alone (single combination) *S obtustifolia (S)*=1.56 X 10^6, *D repens (D)*=3.12 X 10^6 Acetylcholine (Ac)= 1.61 X 10^6, physostigmine (ph)=4.6 X 10^6 Nicotine (ni)=2.8 X 10^6 , carbachol (ca)=7.6 X 10^6, adrenaline (Ad)=3.8 X 10^6, atropine (at)=3.85 X 10^6, in two combination S+ph= 6.2 X 10^6, S +Ac=3.3 X 10^6 S+ Ca=9.2 X 10^6 D+Ad=7 X 10^6, S+at=5.4 X 10^6 S+Ad=5.4 X 10^6 S+D=4.7 X 10^6 S+ni=4.4 X 10^6 D+ni=6 X 10^6 S+D+Ad=9 X 10^6 S+D+Ph=9.3 X 10^6 S+D+Ac=6.3 X 10^6 S+D+At=8.6 X 10^6 S+D+ni=7.5 X 10^6 S+D+Ca=12.3 X 10^6

All values are approximated to 6 million (Boy'o 2014)

First and foremost, I posit that any number or magnitude of response (R_{sm}) is a function of or proportional to activated STP backbones (N_i) and inversely proportional to inactivated STP backbone (N_y)

i.e. $R_s \alpha \frac{N_i}{N_y} and\ thus\ R_s = K \frac{N_i}{N_y}$ (1)

K is a universal constant proportionality of therapy which reflex MCT at unity K=1

Determination of N_i

To determine N_i I used two methods, the novel one and another novel one which employ the law of mass occupancy theory as well as R*R, R*, or R*R* constitutive state theory (Figures 5-9).

Novel Method one without occupancy or constitutive state theory

$$N_x X \Sigma N_{LR1} - \{ Pr_{pb} + Pr_{sb} \} X N_{co} X nE = N_i \quad (2)$$

Where (N_{co}) denote number of receptors types coupled to transmembrane to receptors and effector. Generally, research show that Five known basic GIT receptors; GPCRs., guanylyl kinase (GK), Tyrosine kinase (TK), Cytokine kinase (CK) and Ionic channels (IC) are all couples or attached respective to 5 G protein, 1 kinase, 1 kinase,1 kinase, and 1 channels meaning that there are 9 (N_{co}) in contraction of the ileum in two or more combinations and 7 (N_{co}) for contraction (stimulatory response) and 6 (N_{co}) for relaxation (inhibitory response) and (N_{LR}) terms means sum of all the receptors in locus 1, 2, or 3(Boyo 2014) and thus (N_{LR1}) means sum of all receptors in locus 1 given by my novel formula Zo + η + V = N_{LR1}, (Zo= no of free ECM fluid cells receptors, V=no receptors free ECM cells, η= the values are determined mathematically or experimentally (see general derivation of vital parameters I advocated for below), Pr_{pb} + Pr_{sb} denote sum of respective

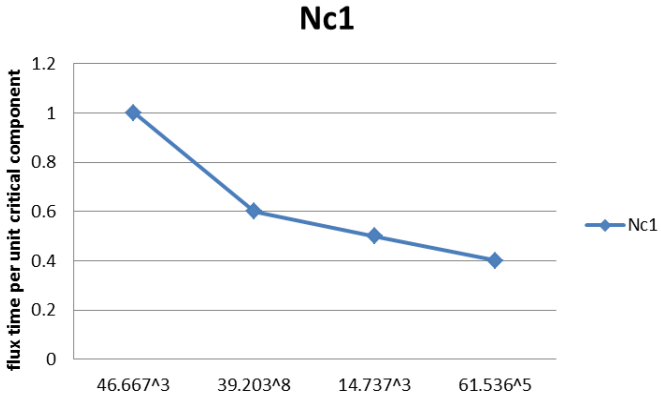

Figure 1: A graph of Nc1 vs. Rsm.

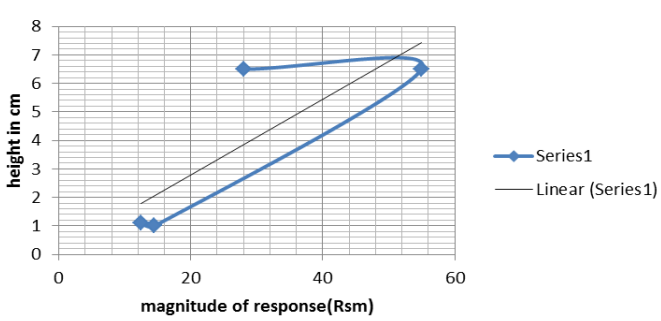

Figure 2: A graph of height vs. Rsm.

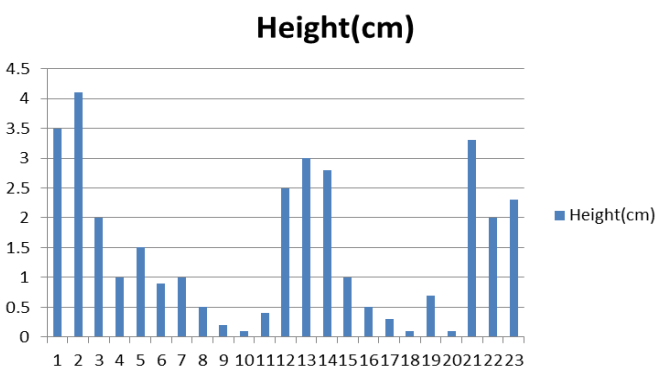

Figure 3: Graph of height of contraction of isolated rabbit ileum smooth muscle in centimeter.

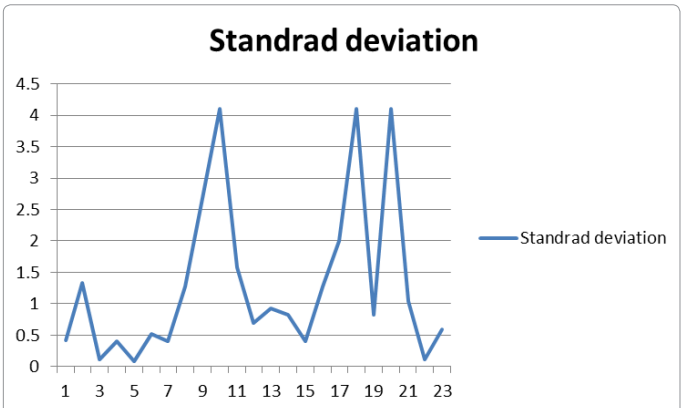

Figure 4: Graph of standard deviation of values of height of contraction of isolated rabbit ileum figures on x axis denote each case on Table 3 above, y axis denote height of contraction.

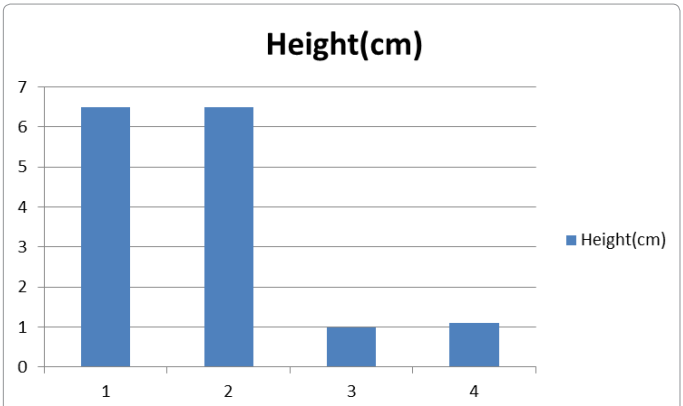

Figure 5: Bar chart of height of contraction against each case.

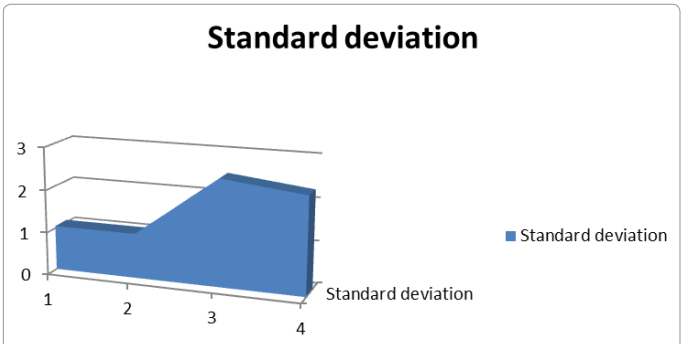

Figure 6: Graph of height of contraction of isolated rabbit ileum, X axis denote case wise list and Y axis denote height of contraction.

probability of molecules binding to protein (e.g. albumin) or any structure not involve in activation, they are determined experimentally and nE is the number of initial effectors, therefore number of STPs backbone or main pathways (N_i) for drug/extract added, We assumed that all molecules have equal chance of binding, molecules with affinity and efficacy were not considered because aside other binding which do not involve receptors {- Pr_{pb} + Pr_{sb}} definitely binding of all kinds ultimately activate the coupled receptor or ionic channels or transporters as the case may be, each of which in turn activate effectors linked to STP backbone or main pathways and that's why everything was multiply by (N_{co}) to get (N_i)in equation 2 above.

From extract

Assuming that (N_{LR1}) values are known experimentally (see relevant database) let just use 30000 X 10^{15} 40000 X 10^{16} and 30000 X 10^{10} respectively for Zo, η and V, their sum=4.300003 X 10^{20}

If from experimental values we know number of all non-activated binding proteins per unit cm of locus say locus 1 for instance, and area occupy by structures to which molecules are likely to binds, and number of molecules N_x then we can determine the probability (Pr) of binding for pb and sb i.e. for pb, Pr (pb1 or pb2……)=P(Pb1) + P(pb2)…+ Pr {pb1/pb2}…, let say that sum values each pb and sb are 5.001 and 8.033 respective

Value (N_{LR1}) 4.300003 X 10^{20}

Nco= 7

Nx for Extract=1.56 X 10^{37}

And so using $N_i = N_x X \Sigma N_{LR1} - Pr_{pb} + Pr_{sb} X N_{co} X nE$

N_i = 1.56 X 10^6 X 4.300003 X 10^{20} - 5.001 + 8.033 X 7 X 3 = 4.69 X 10^6 STPs per locus one,

Note that desired each values Zo, η and V of ΣN_{LR1} can be subtracted in evaluation to get respective STP i.e. per free cells, per ECM structure per fluid receptors respectively.

Therefore, using same methods activated STP back bone N_i for other drugs and extract includes

S=4.69 X 10^6, D= 9.38 X 10^6 Ach= 7.85 x 10^6, physostigmine 6.46 x 10^6, nicotine=8.65 x 10^6 carbachol=4.6 x 10^6 adrenaline=2.32 x 10^6, Atropine=1.16 X 10^6. S+Ach=12.6 x 10^6, S+ph=11.2 X 10^6, S+ni=13.3

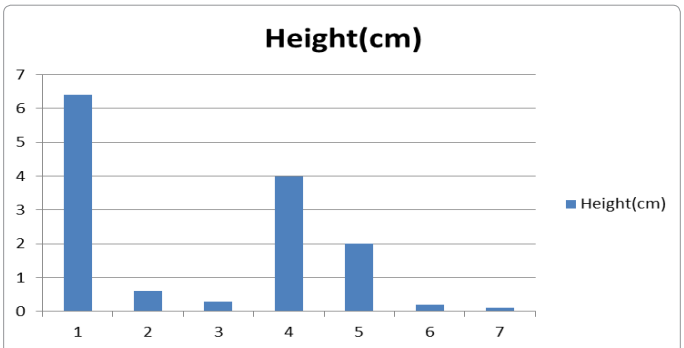

Figure 7: A graph of group B shows height of contraction of isolated rabbit ileum, figures on X axis denote case wise consideration , y axis is height of contraction as shown in the Table 2.

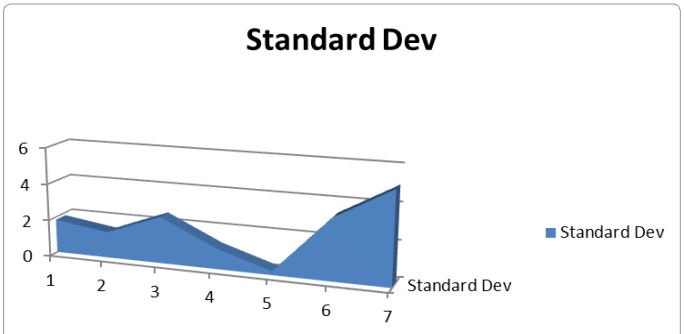

Figure 8: Graph of standard deviation of Table 3 results, figures on X axis denote case wise consideration, y axis is height of contraction as shown in the Table 2.

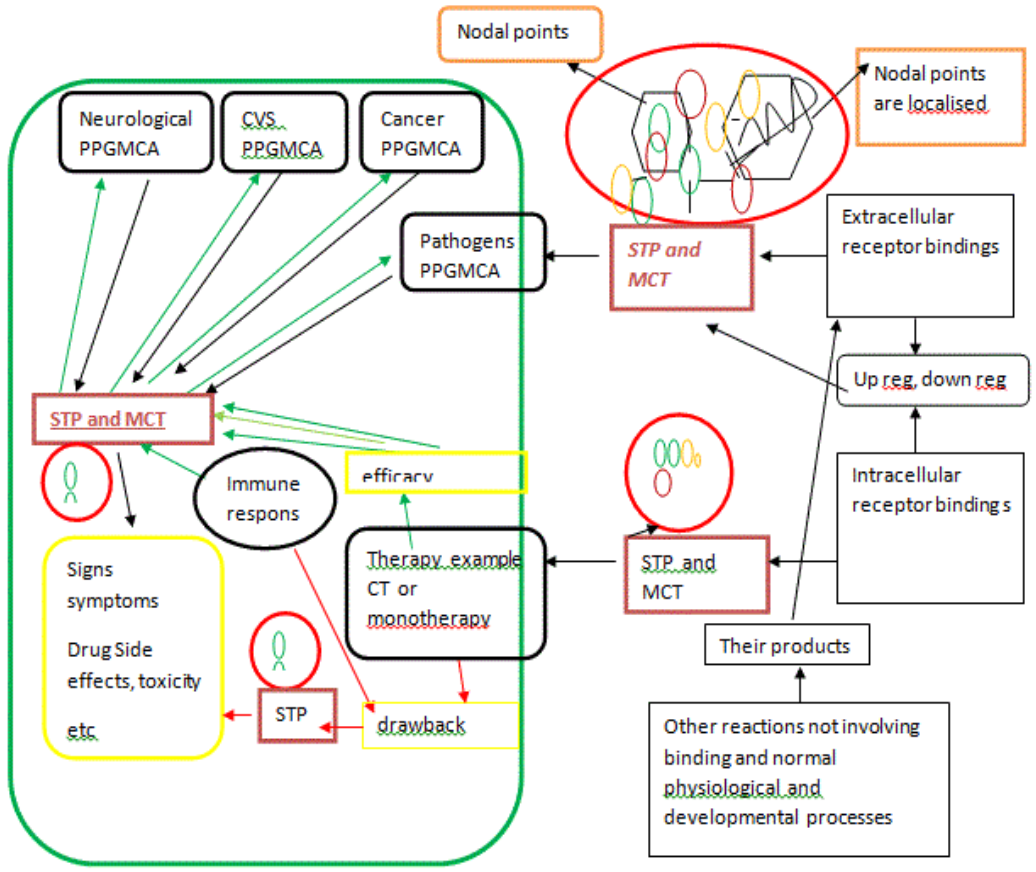

Figure 9: Diagrammatic shows dependent of body processes on STP and MCT.

X 10^6, D+ni=18 x 10^6 S+ca= 9.2 x 10^6, S+at=5.9 X 10^6 S+ad=7 x 10^6, D+ad=11.7 x 10^6 D+ni=18 x 10^6 S+D=14 x 10^6 S+D+ad=16.3 x 10^6 S+D+Ph=20 x 10^6 S+D+ac=21.9 x 10^6 S+D+at= 15.2 x 10^6 S+D+ni=22.7 x 10^6 S+D+ca= 18.6 X 10^6 STP s backbones or main pathways respectively

In the bioassay, beside motility about four other responses occurred, metabolism, absorption, secretion, proliferation of the epithelial cells, and motility, thus whether R,*, RR*, RR exist or not response magnitude occur by many actions of STP component, such as critical points actions, recruitment, phophorylation, acetylation, recruitment, and so on which the relation $(C_p + p_f + PD)^n / \Sigma_{n=1,2,3} nN_o$ in equation 6 attempt to capture for any given body process or magnitude of response emanating from backbone cascades., which was why I used the coupled receptor, and note that two methods were used for determination of activated back bone, beside equation 2, another novel method which incorporate law of mass actions and R*R, R*R, etc was used see equation 3 below

$$N_i = (N_x - Mef) + (N_x - M_{af}) N_x X L_{NR} - \Sigma N_{LR1} - Pr_{pb} + Pr_{sb} X N_{co} \quad (3)$$

Where M_{ef} is number of molecules with affinity and M_{af} is the number of molecules with efficacy as determined experimentally or from mass occupancy theory, with known values of other variable as determined in equation 2. N_i Can be known

From equation 1 above $R_s = K\frac{N_i}{N_y}$

We incorporate Ni, initially Ni from equation 2 and 3

From equation 2 $N_x X \Sigma N_{LR1} - \{Pr_{pb} + Pr_{sb}\} X N_{co} X nE = N_i$

Substitute equation 2 in 1 above we have

Thus $R_s = K\frac{(N_x x \Sigma N_{LR1} - \{Pr_{pb} + Pr_{sb}\} x N_{co} x nE)}{N_y}$

Determination of Ny

We employ concept of quantum physics, here we assumed each component to behave as wave and particles, before then we employ transduction time at critical point (CP) divide by magnitude or strength of the field due to electrical activities gives strength of each flux branching out from the critical points, thus CP/E, so that the range of values obtained to the power of flux velocity emanating from critical points to know changes CP/E caused flux velocity, thus $Vl^{CP/E}$, note that Vl is flux velocity per unit cascade, But for component that are not critical points, we determine how transduction time per unit component changes flux velocity per unit component VC^{nt} wher Vc is velocity per cascade component and nt transduction time per cascade component, thus Ny = $V_l^{CP/E} Vc^{nt}$ substitute in equation 1

Thus $R_s = K\frac{(N_x x \Sigma N_{LR1} - \{Pr_{pb} + Pr_{sb}\} x N_{co} x nE)}{V_l^{CP/E} Vc^{nt}}$ (4)

In the limit as signal tend to zero $V_l^{CP/E} Vc^{nt}$ cancels out

And therefore $R_s = K (N_x X \Sigma N_{LR1} - \{Pr_{pb} + Pr_{sb}\} X N_{co} X Ne$ (5)

But response depends on critical points, recruitment of several peripheral factors and wave nature of STPs, transduction per unit

time per unit component, flux per unit component, we introduced the following into equation 1; Cp, Pf, PD, tf, av, Nc1

And thus $R_s = K(N_x \text{ x } \Sigma N_{LR1} - \{Pr_{pb} + Pr_{sb}\} \text{ x } N_{co} \text{ x } nE)\frac{(C_p + \text{t}\,f + V1 + N_{c1})^{\Sigma n1-n2}}{\Sigma_{n=1,2,3...} nN_0 Nec}$ (6)

To assuage the yearning of proponents of mass occupancy theory or RR, R*, R*R* concepts We use equation 3 in equation 6 instead of equation 2 and thus from equation 3

$N_i = (N_x - Mef) + (N_x - M_{af})\, N_x \text{ X } L_{NR} - \Sigma N_{LR1} - Pr_{pb} + Pr_{sb} \text{ X } N_{co}$

$R_s = K(N_x - M_{af}) + (N_x - M_{af})N_x + L_{NR} - \Sigma N_{LR1} - Pr_{pb} + Pr_{sb} \text{ x } N_{co}\frac{(C_p + \text{t}\,f + V1 + N_{c1})^{\Sigma n1-n2}}{\Sigma_{n=1,2,3...} nN_0 Nec}$ (7)

Definition of term

R_s= STPs and MCT responses or body processes

t_f=flux time per unit cascade backbone

a_v=flux velocity of each STPs component

N_{c1}= flux velocity of each STP backbone activated

Vl= flux velocity per unit component

K= is a constant proportionality which is the Molecular cross talk (MCT)

$\frac{(C_p + \text{t}\,f + V1 + N_{c1})^{\Sigma n1-n2}}{\Sigma_{n=1,2,3...} nN_0 Nec}$= implies rate of monomial expansion (branching) of cascade for one STP components example (C_p), binomial expansion (branching) of cascade example for two components (C_p, tf) or polynomial expansion (branching) of cascade example for three STPs components (C_p, tf, Vl, N_{c1}.....) streams, components may be kinases or other protein types, raise to n, n could PF or PD, n1 is activating event at each component, whereas n2 is deactivating events, n1 or n2 can be express in particulate nature or wave nature (the quantum factor) example n1 particulate nature = phosphyrylation at binding site derivable from Pc and Pf in equation 10 and 11, n1 wave form is nature of wave flux generated by two or more interacting STPs components derivable from PD equation 9, is divided by sum of one or more number of activated STP backbone (nNo), note all the equation are subject to calculus derivation, and again from binomial expansion (vital for deduction of signal amplification)

$$(x + a)^n = \sum_{k=0}^{n} \binom{n}{k} x^k a^{n-k} \quad (7)$$

or $(a + b)^n = a^n + n_{C1}a^{n-1}b + \ldots + n_{Cr}a^{n-r}b^r + \ldots + b^n$ (8)

Where $n_{Cr} = n! / (n-r)!r!$

and again equation 7 and 8 though in binomial form must be change to polynomial or even monomial form, depending on cascade event under consideration, these equation and advance calculus can be used to derived vital relation for each component or many component of the expanding chains of cascades, for by their amplification, they expand the signals and the magnitude of the signal depends on their expansion. Aside these ones, other equations use to know vital STP parameters are shown below

$$Dp = \frac{v^2\alpha\left[\frac{\frac{s}{r}}{\frac{n1n2n3}{\delta t_n}}\right]_{\delta tN}\Sigma\left[q_{1,2}^2 + r_{1,2}^2\right]^2\left[\frac{\delta q + \delta p}{2\delta t}\right]\left[\frac{L_{oA^n}}{L_A}\right]\frac{\delta s}{\delta t}}{\left\{NcriP_f\right\}\frac{DaDw}{dt}} \quad (9)$$

$$Cp = \left\{\frac{\left(\left(\frac{\pi r^2}{12}\right) - \left\{\left|\frac{\frac{\theta\pi r^2}{360^0}X2}{4}\right|\right|N_i\right)}{5} - Nc_1 \text{ or } Nc_{2,} \text{ or } nv \text{ or } nt\right\}^2 \quad (10)$$

$$P_f = \left\lfloor \frac{\{\Sigma\{C_p\{s_1 - a_0)x\left\{\frac{2\pi}{\lambda}\right\}\{t_1 - a_0)\}\left\{\frac{\lambda\Delta 0_2 T}{\text{tf}}\right\}^2 X\{FE(vl\}}{Nc_1 a_v a_t} \right\rfloor \quad (11)$$

The detail of derivation of equation 9, 10 and 11 is given in boyo's work which borders findings on indirect role of STP on contractility of GIT.

In the bioassay, beside motility other responses occurred, metabolism, absorption, secretion, proliferation of the epithelial cells which were not recorded, however they can be ascertain with aid of graph of Ni vs Rs, and Rs vs Height of contraction (H), we use H from group 2 and 3 result.

So we determine magnitude of response Rsm

From binomial equation, we look at two cases of expansion of cascade component, Cp and av, we solved for $(a + b)^n$ i.e. $(cp + av)^{pf}$ from group B result *senna obtusifolia* and cabachol (ca) *senna obtustifolia* and acetylcholine (ach), *Duranta repens* and Adrenaline (adr), *senna obtustifolia* and adrenalin (adr)

But from Binomial series $(a + b)^n = a^n + n_{C1}a^{n-1}b + \ldots + n_{Cr}a^{n-r}b^r + \ldots + b^n$

For *senna obtusifolia* and ca, av=4.9, cp=11.1X10^6

Thus

$(cp + av)^{pf} = cp^{pf} + pf_{C1}cp^{pf-r}\,av + \ldots + pf_r cp^{pf-r}b^r + \ldots + b^{pf}$

Note that = $pf_{c_1} = \frac{pf!}{(pf - r)!r!}$ but pf=7.4 X10^{55}, taking r=6 STP components

thus

$pf! = \frac{7.4 \text{ x } 6.4 \text{ x } 5.4 \text{ x } 4.4 \text{ x } 3.4 \text{ x } 2.4 \text{ x } 1.4}{12.9 - 6!6!} = \frac{12,855.1}{4286.6 \text{x} 720} = \frac{12855.1}{3086352} = 0.04165 \text{x} 10^{55}$

$(cp + av)^{pf} = 11.1 \text{ x } 10^{6(7.4\text{x}10^{55})} + (0.04165 \text{ x } 10^{55})\,(7.4 \text{ x } 10^{55} - 6) \text{ x } 4.9 + (0.04165 \text{ x } 10^{55})\,11.1 \text{ x } 10^{6(7.4\text{x}1055 - 6)} \text{ x } 4.9^6 + 4.9^{7.4 \text{ x}}\,10^6$

$(cp + av)^{pf} = 152192.5$

From equation 6 or 7 rate of expansion and combination is

$\frac{(C_p + \text{t}\,f + V1 + N_{c1})^{\Sigma n1-n2}}{\Sigma_{n=1,2,3...} nN_0 Nec}$

Let sum of number of components per cascades (No) STP components=10 and Nec =5

Thus 152192.5/ 10x 5 = 3043.9, note for group 3 No=15, Nec=8

But N_i for for *senna obtusifolia* and ca= 9.2 X 10^6

From equation 2

$N_x + L_{NR} - \Sigma N_{LR1} - Pr_{pb} + Pr_{sb} \text{ x } N_{co} \text{ x } nE = N_i$

And $R_s = \frac{(C_p + \text{t}\,f + V1 + N_{c1})^{\Sigma n1-n2}}{\Sigma_{n=1,2,3...} nN_0 Nec}$ N see equation 6 and K=1

Therefore magnitude of response

R_{sm} for *Senna obtussifolia* and cabachol = 9.2 X 10^6 X 3043.9 =27.99652 X 10^3

Increase in height correspond perfectly with increase in Rsm until at certain values for a certain group, meaning that STPs responses or body processes depends on activated STP and inactivated one, from the graph many STP were deactivated at point were black line cross the curve (blue line), such drug molecules are potential target use in enterprise meant to deactivate STP cascade, as in cancer, thus *senna obtusifolia* and cabachol molecules are potential candidates for STP deactivation. We again adduced that some extract molecules might have entered the cell membrane to bind with any of the STP, for research show that isoprenylated acetophenone and rosenonolactone are part of active constituent of the extract [9-14].

Discussion

Theory one was proved from two perspectives

(1) Experimental proof; which include: (a) Bioassay of biomarker (rabbit ileum) and (2) Mathematical proof; which include (a) algebraic equations and (b) calculus derivative arrived at from experimental deductions.

Proof of theory 1 of butt

Experimental proof: as shown in the result obtained above, I used combination interactions (synergism, potentiation and inhibition) of antagonist and agonist drugs with extract to deduce the following: (1) The autonomic nature of the extract and hence the receptor types which combined with the extract deduced from drug action of 2 ug/ml of acetylcholine and 10 ug/ml of adrenaline height recorded are 4.1 cm and 1 cm, standard deviation are 1.33 and 0.40 respectively) (Tables 1 and 2) (*Senna obtusifolia* and *Duranta repens* maximum height was 3.3 cm and 1.5 respectively at 1.40 and 0.08 standard deviation, which validate their autonomic nature, meaning that the extracts acted via G protein and possibly via other receptors as the atypical nature of contractions show, but recall that we want to prove that the seven processes which occur in the isolated rabbit ileum were dependent on STPs, firstly we show that as far as autonomic action of the drugs are concern, they must have acted through adrenergic or muscarinic receptors, we suspected other dose dependent non autonomic actions of extract via other receptors which may or may not be G protein coupled, these receptors includes but not limited to dopamine, 5HT, adenosine, NK, VGF, EGF, NK1, NK2, T1R, T2R, and transducin in addition to their subtypes which are GPCRs, RTKs, integrin etc, *Senna obtusifolia* generally, at low dose caused contraction (1 mg/ml of 0.8 ml height was 2.5, however at 10 mg/ml of 0.8 relaxation was seen, standard deviation was 0.69 and 0.40 respectively), the mode of action of drug and extract shows that many STPs and MCT were activated, as shown in mathematical proof, for instance as far as the seven processes are concern, Binding between drug or extract molecules activated ECM receptors and plasma membrane receptor on the GIT, from result of interaction it is the STPs and MCT components which mainly regulate response, binding effect was additive, it is well established that ECM cascade have direct bearing on cellular and nuclear cascades, of course the contribution of binding at cell membrane as describe by mass occupancy theory, RR, R*R* state theory to seven response is acknowledged, which is why number of activated STP (Ni) is derived as

$$N_i = (N_x - Mef) + (N_x - M_{af})\, N_x \, X \, L_{NR} - \Sigma \, N_{LR1} - Pr_{pb} + Pr_{sb} \, X \, N_{co}$$

Indeed, it is not contestable that autonomic drugs or the extract bind to three types of receptors,(1) those that penetrate membrane with or without intrinsic enzymatic activities (2) those couple intercellular such as gtpase binding receptor or G-protein receptors (3) those found intracellular and in the nucleus or on each STP cascade component binding site, adrenaline, acetylcholine and extract acted through adrenergic receptors to activated first part of STPs via Gα (Gs, Gαq, Gα12, Gα13, Gα15, Gα16, Gαi, Gαo, Gαz, Gαt, Gαgust) to activate PLC/cAMP/IP3/DAG/calcium/CMLK pathways which open up calcium store and mediate local and globalised calcium actions leading to motility. Again, ca^{2+} during bioassay also act on phosphorylase kinase which activate phosphorylase or calmodulin kinase to mediate synaptic junction secretion of neurotransmitters or through calmodulin kinase III to cause protein synthesis or through calcinurin which is probably implicated in relaxation of the biomarker, since calcinurin a phosphate has been implicated in inactivating Ca^{2+} channels by dephosphorylation, also efflux and influx of calcium is determined by Ca^{2+}-H+ exchanger, intracellular release from endoplasmic reticulum through rynadone receptor is mediated by cyclic adenosine diphosphate ribose (cADPR) also calcium act on ras super family (any one Rho, Ran, Rheb, Rad, Rit) and various kinases through signals and activated Raf/ERK/PKA/MPAK/ other pathways are MEKK 2,3 Tpl2/MEK5/ERK5/BMK,1 cascades, and MAPKKK/MAPKK/MAPK/ pathways, followed by biological response which is the contraction of the isolated rabbit ileum, again mathematically determined result of Rsm, Nc1, and Tf are consistence with above observations and as shown by other findings, it crucial to state that STPs usually act in interconnected fashion and not in isolation, so that other localized processes (circulation, growth of epithelial cells, drug metabolism, immunoreactivity, secretion etc) on the isolated rabbit ileum were up regulated or down regulated, the extract interaction is the hallmark of the finding in that not only are STPs and MCT implicated in changes in height of contraction but also by acytylation, phosphyrylation, receptor internalization, protein modulation of various cascades component lead to the following process circulation, growth of epithelial cells, drug metabolism, immunoreactivity, secretion, we deduced that motility observed caused secretion of GIT peptide hormones, importantly beside these signaling molecules, the extract molecules no doubt bind to other non autonomic receptor thereby, besides motility, mediate processes of circulation, growth of epithelial cells, drug metabolism, immunoreactivity, and secretion, few among the cascade actually activated these processes by membrane activation, translocation, activation of transformational changes, agonist activation, and then by phosphorylation, as values of transduction per unit time showed, generally in double and triple interactions, and at moderate concentration, extract did not affect the action of drug, however at high concentration parasympathetic effect of acetylcholine was inhibited by *Duranta repens*. and sympathetic effect of adrenaline was overcome by *Senna obtusifolia* at standard deviation

dr and ext	Rsm	Height	conc(vol)	Nc1
S O +ca	27.996	6.5	10 mg(8 ml)	0.7s
S O +ach	54.949	6.5	10 mg(8 ml)	0.6s
S O+Adr	12.509	1.1	10 mg(0.2 ml)	0.4s

Table 1: The comparative results of magnitude of STP response (Rsm) and flux time per unit critical component of STPs cascade.

dr and ext	Rsm	height	conc(vol)	Nc1
2ext+ach	46.667^3	6.4	10 mg(0.8 ml)	1
2ext+ca	39.203^8	4	10 mg(0.8 ml)	0.6
2ext+adr	14.737^3	0.4	10 mg(0.8 ml)	0.5
2ext+phys	61.536^5	2	10 mg(0.8 ml)	0.4
D R+adr	14.499	1	10 mg(8 ml)	0.3s

Table 2: The comparative results of magnitude of STP response (Rsm) and flux time per unit critical component of STPs cascade.

2.28, height was 1.1 cm as against 0.9 cm when adrenaline was added alone, on the whole *Duranta repens* in group A caused relaxation of ileum (height 0.1 and standard deviation 4.11), in group 3 result, mainly decrease in contraction was seen at high concentration, these events can be accounted for looking at the following; transduction time, cascade recruitments, localized cascade components, waves amplitude of docking sites of adaptar proteins, 'on' and 'off' mode of the STP switches as shown below, for theory like mass occupancy which consider event at binding sites are not sufficient to explained theses variations, the fundamental determinants of changes or STPs responses per say are interactions of cascade components, note that Rsm for group C show increase in activities per unit transduction time, first at individual level and then at global level of their interconnected network, thus any of the processes (magnitude of response Rsm) is function of STPs and MCT to a larger degree, and to a limited degree binding at plasma membrane, for in GIT secretion process, motility was the source of stimulus which by STPs activate secretory cell, latter release signaling molecules in the isolated rabbit ileum, the signaling molecules (motilin, substance P, VIP, secretin etc) in turns act on various receptors or ion channels to activate many STP, example ca2+/PKC/Ras/cRaf/MEk1/2/ERK pathways was activated by calcium channels, cytokines and even GPCR serve as stimulus activating MLK3/TAK/DLK/MKK3/6/P38/MAPKα/MAPKβ/MAPKγ, OR through A Raf/B-Raf/c-Raf/Mos/Tpl2/MEK1/3/ERK to cause growth and differentiation of epithelial cells, the extract molecules or the GIT signaling molecules from luminal secretions response, presumably, activate GIT epithelial cells growth by protein synthesis via RTK, integrin and cytokines receptors, the underlining signaling processes are fak/p3/akt/mtor/ mtor the last cascade component activated transcriptional factors and protein synthesis, so that all the GPRC, RTK and intergrin signals converge on akt, pok1 and pip2 kinase, in both kinase pten, PP2A phosphtase regulate progression of the signals, and AKT/raf1/ERK/p70S1k cascades activate luminal epithelial cell growth, if it were not so absence of receptor would have made impossible subsequent interaction of drug while the expirement last, mathematical prove below is consistence with these result of past findings, group C Rsm are 46.667^3, 39.203^8, 39.203^8, 14.737^3, 61.536^5, all show increase activity per active components per cascade (perhaps the critical points), another processes which depend on STPs and MCT is circulatory processes, in spite that it was isolated, atypical contraction which decreases progressively aided circulation of blood through RTK/AKT//eNOS/NO pathways, see graph on deactivation of components (point of crossing between black line and blue line), findings show that the secreted GIT signaling molecules have cardiovascular effects for they diffuse from luminal cell to nearby blood vessels, again antigenic reactions at luminal border cannot be rule out, sources of antigen could be drug, extract, or content of organ bath, thus owing to immunoreactivity, I anticipated activation of Toll pathway, IMD pathway and JAK/STAT pathway and so on, this is true looking at Rsm values from group C and their corresponding Nc1 values in (Tables 3 and 4). At moderate concentration (10 mg/ml) and high volume (0.8 ml) it is clear that if by occupancy theory drug with high affinity binds and occupy available autonomic receptors, then extract molecules bind to other non autonomic receptors, besides hydroxylation affect or other extracts molecular changes caused by methanolic or ethanolic molecules may have affected rate of extract molecule permeability through the membrane, such that direct binding between extract molecules and cascade components occurred leading to increase in Rsm values. Note that values of height for group C was not consistence with Rsm values or Nc1, which means that some STPs were inactivated, in graph 2 below (Table 5), at some point the blue curve turns left and descend a bit, which show that Rsm had remained constant and the decreases slightly at 10 mg of 0.8 ml of extract and cabachol [15-18].

Findings reported that these pathways are active in antigenic attack, additionally, local immune response may have been activated by MLK3/TAK/DLK/MKK3/6/p38/MAPK or MEKK/MLK3/ASK1/MKK/SAPK/JNK cascades, the immune response protected the GIT luminal lining from organ bath pathogenic onslaught if it were not so result of group 3 would have beens same for the drug used were not anti-pathogens, thus extract lowers contractile effects of the drugs, from 6.4 cm to 0.1 cm with each increase in dose for the group, standard deviation minimum is 0.21 and maximum is 5.05, again were it not for immunoreactive response the isolated ileal cells would have been killed by pathogenic poison making it useless while before the experiments begins, but that was not the case, again immunoreactivity interaction between the following STP components p21 activated kinase (PAK) which mingle with exchange factor and G protein couple receptor

Group A single interaction	Height(cm)	Standard deviation
Ach 2 ug/ml of 0.1 ml	3.5	0.42
Ach 2 ug/ml of 0.2 ml	4.1	1.33
ADR 10 ug/ml of 0.2 ml	2	0.12
ADR 10 ug/ml of 0.4 ml	1	0.4
DR 1 mg/ml of 0.8 ml	1.5	0.08
DR 10 mg/ml of 0. 2 ml	0.9	0.52
DR 10 mg/ml of 0.4 ml	1	0.4
DR 10 mg/ml of 0.8 ml	0.5	1.27
DR 100 mg/ml of 0.2 ml	0.2	2.68
DR 100 mg/ml of 0.4 ml	0.1	4.11
DR 100 mg/ml of 0.8 ml	0.4	1.58
SO 1 mg/ml of 0.8 ml	2.5	0.69
SO 10 mg/ml of 0.2 ml	3	0.92
SO 10 mg/ml of 0.4 ml	2.8	0.83
SO 10 mg/ml of 0.8 ml	1	0.4
SO 100 mg/ml of 0.2 ml	0.5	1.27
SO 100 mg/ml of 0.4 ml	0.3	2
SO 100 mg/ml of 0.8 ml	0.1	4.11
DR 100 mg/ml of 0.4 ml	0.7	0.83
DR 100 mg/ml of 0.8 ml	0.1	4.11
SO 0.1 mg/ml of 0.8 ml	3.3	1.04
SO 0.1 mg/ml of 0.4 ml	2	0.12
SO 10 mg/ml of 0.2 ml	2.3	0.59

Table 3: Result of single interaction of drug and extract.

Group B double interaction	Height(cm)	Standard deviation
SO 10 mg/mlof 0.8 ml & 0.5 ug of 0.1 ml ca	6.5	1.05
SO 10 mg/mlof 0.8 ml & 2 ug of 0.1 ml ach	6.5	1.05
DR 10 mg/ml 0.8 ml &ADR 10 ug/ml of 0.8 ml	1	2.5
SO 10 mg/ml 0.2 ml &ADR 10 ug/ml 0.2 ml	1.1	2.28

Table 4: Result of double interaction between drugs and extract.

Group C tripple combination interaction	Height(cm)	Standard Dev
2 ext 10 mg/ml of 0.8 ml & ach 2 ug of 0.4 ml	6.4	1.85
2 ext 100 mg/ml of 0.4 ml & ach 2 ug of 0.4 ml	0.6	1.42
2 ext 100 mg/ml of 0.4 ml & ca 0.5 ug of 0.2 ml	0.3	2.55
2 ext 10 mg/mlof 0.8 ml & ca 0.5 ug of 0.2 ml	4	1.15
2 ext 10 mg/ml of 0.8 ml & Phys 2 ug of 0.25 ml	2	0.21
2 ext 100 mg/ml of 0.4 ml & phys 2 ug of 0.25 ml	0.2	3.35
2 ext 10 mg/ml of 0.8 ml & ADR 10 ug/ml of 0.2 ml	0.1	5.05

Table 5: Result of triple interaction between drug and extract.

kinase factor, the activated Rac and CdC42 GTPase by manner of PAK and G protein recruit activator PIX, another recruited protein is a scaffolding protein called GIT, findings SHOWS that integrin and PAK/PIX/GIT/ model can act non redundantly in regulating other pathways, perfect correspondence between values of Nc1 and Rsm support these result of action of these characterized proteins [19-20].

Proximal GIT columnar epithelium contain high oxidative conjugates and hydrolytic drug metabolizing cyp450s enzymes, mdri-cyp3a4, mpr2, ugt have synergic roles work on them is ongoing, also in GIT metabolism, transcription facto nf-e2 related factor 2(nrf2) is activated in presence of reactive species like superoxide or trioxygen, it sense cysteine residue which is modified by toxic chemical of xenobiotic metabolism, result of other findings show that it has protective effect on GIT metabnolism, with jun, fos and maf, they interact to mediate transcription, also they interact with glutanthion synthase to synthesize GSH which have protective ability against biotransfromed radical in GIT, the findings are consistence with our observation that the isolated rabbit ileum died some hours after the experiment starts, looking at the graph of Rsm and height of contraction, decrease in response correspond to height, not until deactivated STPs of set it, drug used were certainly metabolized, we could not show which STPs was involved, nevertheless result from calculation supported this assertion, we suspected that drug metabolism play part in slowing down responsiveness and initiating death of isolated rabbit ileum owing to exhausted load of nrf2, thus metabolism is factor of STP and MCT given their roles as shown by Rsm values, for mathematical determination shows that beside motility other processes might have occurred, findings shows that motility may mediate secretion and products of secretion could serve as signal to other GIT events, example motilins and sceretin are implicated in GIT processes of contraction, the fact of occurrence of above processes and their dependence on STPs was reinforce from Ni values, and magnitude of response (Rsm), which was high enough to show that other responses occurred, and that, they are the sole making of STP component mainly, events at binding site play only contributory role as mentioned.

Note as far as above result and equations are concerns, a fundamental truth is that drug could be replaced with a probe (both are molecules nonetheless) but probe use need be extremely specific to allow for one to one binding (example probe to rab binding). And with equation 6 and 7, we have shown that any processes or STP response is function of activated STPs backbone, it is increase with Ni and decrease with Ny provided K (MCT) remain constant. the values were derived experimentally and inserted into the equation, by so doing, the magnitude or number of response R_{sm} was determined, and insight from this result may be manipulated for use in biomedical research, drug development, clinical application and in control and regulation of STPs which is the aim of BUTT theory, note that further derivative of the equation may be required, STP effects on the waves generated by interstitial cells of cajal lead to increase in intra-cellular concentration of Ca^{2+}. Ca^{2+} either through ligand gated or voltage (T or L) gated enter the cells and bind to calcium binding protein calmodulin which activate myosine dependent kinase such as myosine kinase latter phosphorylates myosine and cause contraction, also different forms of agonist/partial agonist (synergism, potentiating) or antagonism (competitive or non competitive equilibrium) interactions activated many receptors which transduce signal and amplify them by STP mechanisms, and generate waves of spatio-temporal dynamic actions, these in turn amplify the motility response, as deduced from equations below.

Normal body embryological, physiological and biochemical systemic processes and products of none bindings interaction involves STPs (brown boxes), binding relate to body milieu interactions (green boxes) via STP and responses (block and yellow boxes inside the green box) are mediated through STP and in rabbit ileum assay motility, secretion. Immunoreactivity, metabolism etc are STPs responses,

It crucial to not that determination of some parameter ensures derivation of equations used above, few among them are; organ bath concentrations (C2) for each extract and drug used was determined from C1V1=C2V2, binding time of the ligand and the flux time which denote the time binding activate STP to the time response was observed, flux time per unit STP components (FTPUSC) i.e. net time it takes for signal to move from one components to another, thus I showed that there are four types, (1) flux per unit critical points, (2) flux per unit peculiar inter STP components, (3) flux per unit peculiar peripheral factor and, (4) flux per unit components of the backbones. Other variable determined are Number molecules, velocity of flux (Vl), number of activated STP backbone (Nc1), also vital parameters includes; total number of receptors express on intracellular (membrane) and extracellular domain, and their respective number of binding site, number and magnitude of charge of sites, number conformational changes after binding and other such parameter relevant to the equation 1, their determination are detailed in a work which investigate indirect effect of STP mathematically [20-22].

Conclusion

It can be concluded, from experimental and mathematical results that height of contraction correspond perfectly with magnitude or response (RSm), and that STPs and MCTs control and regulate all processes, they are the basic unit of control and regulation of all processes inclusive of neural and hormonal ones, and thus magnitude of response (Rsm) remains an important attribute of STPs in biomedical research, in clinical application and drug formulation, since it relate response to specific and global effects of STPs activities, which prove BUTT 1. in fact the BUTT concepts wrap in mathematics and quantum physics principles make possible handling of several complex pathways in several cells, organ or systems, for globalised view of STP is fundamental to it control and exploitation in research.

Future Outlook

Discovered cascade and their characterize proteins or enzymes show that biological processes can't do without STP, this constitute a riddle of some sort in the field of biomedical research, the aim of BUTT 1 is to unravel this mystery so that everything about biomedical research is conducted from STPs line of reasoning.

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