Heuristic modeling for prognosis of neuro-oncological cases and evaluation of treatment protocols

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Abstract

Heuristic models to generate sub optimal solutions in neyr-oncological case studies entail the incorporation of several elements of criticality drawn in from a myriad haze of parameters like pathology, hematology, cell morphology and the metabolic factors of muscle, glucose and blood besides the enzymatic factors related to the auto-immune disruptive forces.

SEQUENCING INFLUENCES - '	VITAL METABOLISM AND	IMMUNOLOGICAL	SEQUENCING
SIMULATED CONCLUSION -1			

SIMULATED CONCI				
VITAL PARAMETER	FUNDAMENTAL CAUSAL LINK	BLOOD METABOLISM INFLUENCE WEIGHT	GLUCOSE METABOLISM INFLUENCE WEIGHT	IMMUNOLOGICAL SEQUENCING WEIGHT
Diastolic	Serum quality	0.99	0.77	0.99
Systolic	Serum quality	0.99	0.77	0.99
HR / tachycardiac output/ functionality of arterial - venous blood volume ratio RR / Cp - oxygen/Cp - carbon monoxide/ Tidal and residual volume/ alveolar dead space / alveolar perfusion CBV Opacity ratio of	Plasma capacity for optimum coagulation, serum quality for rheological properties and fluid viscosity/ organ conductivity for electrical signaling and flux strength at plexus	0.99	0.98	0.99
cerebral cells - hypothalamus/ occipital/grey matter				

EEG - graphical analysis for field strength / NCV /				
CSF conductivity for electrical signaling - parietal / lumbar / cervical zone analysis	Field strength and dielectric field creation propensity	0.99	0.95	0.99

KEY DETERMINANTS OF ORGAN CONFIGURATIONS AS A FUNCTION OF PERFUSION PROPERTIES – SIMULATED CONCLUSION -2

OXYGENATION	PERFUSION STRENGTH (as measured by CBV)	FLUX STRENGTH (as measured by the phase angle of the field)	RESIDUAL DIELECTRIC FIELD (as measured by the phase angle)
65%	97%	0.73	0.21
73%	95%	0.73	0.2
71%	97%	0.75	0.19
68%	91%	0.74	0.17
74%	98%	0.77	0.13
71%	97%	0.73	0.22
58%	88%	0.62	0.28
54%	85%	0.59	0.34
52%	83%	0.57	0.35
49 %	80%	0.52	0.39
CORRELATION STRENGTHS	OXYGENATION - PERFUSION	PERFUSION - FLUX STRENGTH	FLUX STRENGTH - RESIDUAL DIELECTRIC
	0.944111703	0.959957187	-0.980789019

The key determinants in a heuristic model have the following characteristic features:

1. Underlying influence on cross-domain parameters

There are several attributes of the variables that do have underlying cluster influences on the key parameters of major impact on the outcome of a treatment protocol and the commensurate significance in definitive prognosis.

2. Attunement to clusters in an apparent binomial density with the possibilities of normalizing to continuum in the data scatter.

The variables of influence in the context of neuro-oncological sciences have the potential to influence multiple clusters of known impact based on empirical understanding. Heuristic models can unravel the underlying hidden clusters of influence as well as evolving the cross links that have escaped the notice of empirical understanding in the domain hitherto.

3. Poisson scatter evolution from the heuristic models

The critical influence paradigm in a heuristic model is one of Poisson repulsion from the various clusters and therefor having the seminal potential of disruptive impact in the prognosis. Such Poisson scatter of very rare variables and impacting influences can bring in major shifts in the treatment protocols and concomitant prognosis of neuro-oncological interventions.

Mathematical modeling with an exhaustive parametric definition can lead to major discoveries and drug inventions for achieving efficacious solutions in the treatment and consequent prognostic trends of a critical mass of suffering humanity.

The paper outlines the possibilities with greater clarity than ever and showcases the potential of heuristics in medical diagnostics and treatment pathways in the domain of oncology.

Heuristic approaches on simulated mode

Baseline treatment of variables in a DFBL – depth first and breadth later mode entail the establishment of the efficacy of each of the variables in a binomial probability density function to discrete entities near independent of influences.

$P(x,n,p) = (n/x) p^{x} (1-p)^{(n-x)}$ -----(i) (Node determination -1)

(n) implies the number of trials and x is the scatter of outcomes in clinical trials for each of the competing variables in isolation treatment of the others.

(x) scatter is independent of each other in the function and has a probability density (p) for each trial.

Conceptualization of the Heuristic nodes of influence variables in the DFBL mode necessitate the independent probability density in a population scatter to track the poisson element. Cancerous growth is recognized as a poisson distribution and the probability density function is of seminal impact in predicting outcomes.

f(k) = $(e^{-\lambda} \lambda^x)/(x!)$ ------(ii) (Node determination -2)

 λ is the interval rate of the time series of events between occurrences. This is of fundamental importance in evaluating a sequence of events leading to the proverbial "black swan" event of a discovery of changing cellular morphology and mutations leading to oncological states. The time rate used in the poisson density function of likelihood events during the experimental stages should be narrow enough to accommodate detection of rare occurrences.

Euler number is used to denote the maturity of events around a data scatter and definitive clinical outcomes for each of the competing variables. It is important to arrive at **reproducibility of variables** by looking at a tapering constant wherein e>1 in the expression y=(1/x). Gravitating around euler gives referential clinical values that can be safely assumed to be benchmarks in a heuristic referential.

 $f(k) = e^{\lambda} \zeta_{1=0}^{[\kappa]} \lambda^{1/1!}$ (iii) - the cumulative density function for maximum likelihood of a poisson event in the data scatter of each of the clinical variables in isolation. [k] is the floor function in magnitude that helps achieve a saturation maturity in the stochastic process. (Node determination -3)

Cumulative density function equalizes the variances within the realm of reproducibility and enhances the data fidelity for feeding into the heuristic plane.

Decisive treatments on the DFBL grid to arrive at effective cluster strengths and deriving the functional crosslinking variables across the parametric breadth.

The decisive mechanism of the heuristic process in the DFBL mode is the reduction in entropy for arriving at cluster strengths. As the entropy reduces, the stochastic process reaches a stagnation and the cluster strength is derived for optimality.

The stochastic process in the heuristic DFBL mode described in the article is a Markove chain (fundamentally time invariant although the basic variables had been initially treated with a time rate differential) and consequently shall have the following expression:

$p(e_1, e_2, e_3...e_n) = p(e_1), p(e_2 | e_1), p(e_2 | e_3)....p(e_n) | (e_{n-1}) -----(iv)$ (Node determination -4)

where e is the clinical outcome or event and n trials are conducted to arrive at stochastic definitive and time invariant stationary.

$H(Y) = -\zeta_{ij} (\mu_{ij})P_{ij} \log(P_{ij}) - \cdots + (v) (Node determination -5)$

The rate of entropy in the stationary Markove chain is expressed above and builds the foundation of achieving the cluster strength of influences in the Heuristic grid.

The neural networks and the epochs are terminated at the stagnating rate of entropy and consequently define the nodes in the heuristic algorithms.

PROBLEM STATEMENT - FINDING THE ORIGINS OF MEDULLA BLASTOMA AND PREDICTING OUTCOMES ON PARAMETRIC ANALYSIS OF ANTENATAL CARE PROTOCOLS

DOMAIN	SPECIFIC ELEMENTS OF RESEARCH	MATHEMATICAL MODELS	OUTCOMES SOUGHT IN THE RESEARCH	CLINICAL ASSUMPTIONS - NARRATIVE	IMPLEMENTATION COHORT AND ECOSYSTEM DEVELOPMENT	
Antenatal studies of progression	Blood metabolism Glucose metabolism	Fourier analysis of data clusters Fourier analysis of data clusters	Precision limits for safe metabolic data and	ECMO analysis of the findings on metabolic quality could	Blackstone Synergy - Sai cluster of hospitals shall	

		defining the potential benchmark measures for predictive analytics on medulla blastoma or related metastizing tumor regression in the CNS	be an essential derivative for confirmig the findings	evaluate the possibilities of technical tie-ups with EU / US leaders in cognitive research of the fetus, clinical analyses of fMRI and ECMO and finally the parametric hosts of big pharmaceutical
Blood perfusion in the embryo and through the various stages of development of the fetus	Heuristics modeling of key clinical parameters to deduce the equalty of perfusion	Predicting regressive changes in perfusion that can form the basis for further	fMRI findings on perfusion, opacity of the cell and mapping the v/q parameters in the	research models
Cognitive movements of the fetus Psycho- somatic mapping of the expecting mother	Heuristics modeling of key clinical parameters to deduce the equality of perfusion	aggregation of research with fMRI functionality on advanced clinical set- up	contextual comprehension of the perfusion quality in the CNS shall be the bedrock of predictive derivatives	

Stochastic conditions for the Heuristic mode:

1. Epochs = 100,000

- 2. Data smoothing = exponential with power 2 for the pathological elements and logarithmic compression for the thoracic elements
- 3. $R^2 = 0.44$ for pathological elements and 0,38 for thoracic elements

PATHOLOGICAL - HEMATOLOGICAL GRID						
Lipid profile	Uric acid	TPC	WBC count / esonoph ils and family	Hb%	HCT%	Creatini ne and renal profile
Food combust ion	Muscle metabol ism	Muscle metabolis m	CNS signal fidelity	CNS signal fidelity	CNS signal fidelity	CNS enzyme s with food combus tion triggers
Muscle metabol ism	Fast twitch / slow twitch muscle spread	Fast twitch / slow twitch muscle spread	Peripher al nerve signal strength	Peripher al nerve signal strength	Peripher al nerve signal strength	Pancre atic enzyme s
Fast twitch / slow twitch muscle spread	Pancrea tic enzymes	Blood metabolis m	Diastolic - systolic gap percent age	Diastolic - systolic gap percent age	Diastolic - systolic gap percent age	Muscle metabo lism
Pancrea tic enzymes	CNS enzymes with food combust ion triggers	Pulmo - Cardiac output signified by V/Q	Blood metaboli sm	Blood metaboli sm	Blood metabo lism	Fast twitch / slow twitch muscle spread
enzymes with food combust ion triggers	Glucose metabol ism in the CNS	Glucose metabolis m in the CNS	Glucose metaboli sm in the CNS	Glucose metaboli sm in the CNS	Glucose metabo lism in the CNS	Food combus tion
Cluster str	ength > 0.9	1				
Cluster str	ength > 0.7	3 = 0.79</th <th></th> <th></th> <th></th> <th></th>				
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PATHOLOGICAL - HEMATOLOGICAL GRID

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		Muscle metabol ism	CNS signal fidelity	Pulmo - Cardiac output signified	Muscle metabol ism	CNS signal fidelity	Muscle metabolis m	Muscle metabo lism
Depth (nodes	dı	Fast twitch / slow twitch muscle spread	Peripher al nerve signal strength	CNS signal fidelity	Fast twitch / slow twitch muscle spread	Peripher al nerve signal strength	Fast twitch / slow twitch muscle spread	Fast twitch / slow twitch muscle spread
) for the influen cing	d.	Blood metabol ism	Muscle metabol ism	Peripheral nerve signal strength	CNS signal fidelity	Diastolic - systolic gap percent age	CNS signal fidelity	Glucose metabo lism in the CNS
es	U2	Pumo - Cardiac output signified by V/Q	Blood metabol ism	Muscle metabolis m	Peripher al nerve signal strength	Blood metaboli sm	Peripheral nerve signal strength	Blood metabo lism
	d₃	Glucose metabol ism in the CNS	Glucose metabol ism in the CNS	Fast twitch / slow twitch muscle spread	Glucose metabol ism in the CNS	Glucose metaboli sm in the CNS	Glucose metabolis m in the CNS	Pumo - Cardiac output signified by V/Q
cluster st	reng	th of influer	nce > 0.83	1				

cluster strength of influence > 0.55</= 0.61

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