

From Digital to Quantum Epidemiology: The Quantum Data Lake concept for Big Data related to viral infectious diseases

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Abstract

The study is dedicated to the development of quantum epidemiology which is the expected next stage in epidemiology transformation as new quantum technologies have emerged. At the present time, epidemiology is entering the digital era and undergoes a paradigm shift from data-driven to value-driven strategy. The epidemiology data are characterized by uncertainty, multidimensionality, and disconnection, which drive to prefer the quantum approach for data exposition, creation of value, and modeling. The Quantum Data Lake concept is proposed. The data about DNA viruses with symptoms and diseases are shown as example of epidemiology data complexity. The Quantum Data Lake concept consists of several layers and quantum tools, including PT-symmetry and non-Hermiticity as intuitive modeling tools. PT-symmetry breaking is able to detect the hidden shift in the information which is permanently updated in the Data Lake. The duality of PT-symmetry breaking can be compared with the estimation of the best and worst scenarios simultaneously. In contrast to the widely discussed advantages of quantum computing such as high-speed performance and very large parallel scale, the proposed approach emphasizes the fundamental uniqueness of quantum theory for modeling. The study highlights the necessity to investigate the native processes of viruses' interaction with the human population by relying on quantum theory's natural properties. Implementation of quantum logic and reliance on a quantum theory is the fundamental difference between the current digital epidemiology and future quantum epidemiology.

Statement Of Significance

Problem or Issue The epidemiology data are characterized by uncertainty, multidimensionality, and disconnection, which make it difficult to model and prediction of situation development.

What is Already Known The problem under discussion drives to prefer the quantum approach for data exposition, creation of value, and modeling. The quantum solutions in analyzed previous works have allowed taking into account uncertainty, and multidimensionality, and overcoming data disconnection. But in our view quantum approach can open an entirely new door on the way to turning that kind of data into value, namely, time, energy, and symmetry together must be taken notice.

What this Paper Adds The Quantum Data Lake concept is proposed. It consists of several layers including a quantum database and quantum tools. In contrast to the widely discussed advantages of quantum computing such as high-speed performance and very large parallel scale, the proposed approach emphasizes the fundamental uniqueness of quantum theory for modeling.

Highlights

- The digital epidemiology undergoes acceleration and maturation, the essence of the modern paradigm shift in digital epidemiology is a change of point from data-driven to value-driven strategy.

- The epidemiology data are characterized by uncertainty, multidimensionality, and disconnection, which drive to prefer the quantum approach for data exposition, creation of value, and modeling.
- The quantum solutions in analyzed previous works have allowed taking into account uncertainty, and multidimensionality, and overcoming data disconnection. But in our view quantum approach can open an entirely new door on the way of turning data into value, namely, time, energy, and symmetry together must be taken notice.
- The Quantum Data Lake concept is proposed. It consists of several layers including a quantum database and quantum tools. The tools are the following: matrix product states with entanglement, generalization to the quantum tensor network projected entangled-pair states, tensor network on fractal Sierpinski gasket, and multi-scale entanglement renormalization ansatz, PT-symmetry, and non-Hermiticity, reshaping and teleportation.
- The special quantum capabilities help to estimate changes while computing the incoming Data Lake information and allow to predict the critical shift in the epidemic situation considering gain and loss changes with applying all possible influencing factors. Spontaneously broken PT-symmetry with the related bifurcation of gain/loss prevalence (or phase transition) at an exceptional point gives the two variants of epidemic modeling due to the appearance of the circumstances for output as a complex number.
- Despite the quantum dual complex nature of PT-symmetry breaking, this approach to modeling makes it possible to find the hidden phase transition regions in the data, i.e., to predict the shift in the epidemic situation with all its uncertainty, multidimensionality, and disconnection.
- The study highlights the necessity to investigate the native processes of viruses' interaction with human population relying on the natural properties of quantum theory. Implementation of quantum logic and reliance on a quantum theory is the fundamental difference between the current digital epidemiology and future quantum epidemiology.

Structure Of The Paper

The paper is organized as follows.

Section 1 (Introduction) includes a description of the paradigm shift in digital epidemiology and a discussion of the two main problems in epidemic data (how to describe the space in which data will be considered and modeled, and how to define the dynamics in space), providing actualization of quantum approach.

Section 2 (Related works) consists of the review of existing scientific papers on the application of quantum modeling approaches in the field of epidemiology and infectious diseases.

Section 3 (Study design, materials, and methods) provides the information respectively about two stages of the study: 1) data gathering about DNA viruses, and detailed ontology in relation to symptoms and diseases creation (with Supplementary Information files 1 and 2); 2) Quantum Data Lake architecture

proposal and application for data modeling the quantum theory with regard to time, energy and symmetry.

Section 4 (Results and Discussion) includes DNA viruses' detailed ontology and proposal of the Quantum Data Lake concept.

The paper ends with *Section 5* (Conclusion).

1. Introduction

Datafication is covering all spheres of social life including healthcare issues. Along with healthcare digital transformation, electronic health records development, and data-driven medical approach implementation, digital epidemiology undergoes acceleration and maturation. In the field of epidemiology, the task of data collection for an early epidemic or pandemic detection/forecast is growing to data strategy, embracing such issues as open, accessible data, up to the creation of knowledge-based decisions. Digital epidemiology gives an entirely new capacity for investigation of the invisible "micro world", as well as allows the fighting of communicable diseases on a new, most promising level (Tarkoma et al. 2020).

The paradigm shift in digital epidemiology has been boosted during the COVID-19 pandemic. Numerous data-driven approaches and modeling solutions have been elaborated quite successfully relying on raw data collection or synthetic data generation by artificial neural networks (Cao and Qing Liu 2021; Rahimi et al. 2021; Shakeel et al. 2021). Raw data are collected according to Big Data's main features: big volume of data, big velocity of data inflow, and big variety of data types. Raw data per se constitute the base for modeling but have minimum value in the modeling chain. The transformation from data to value has led to significant lessons learned after some years of Big Data models use. The essence of paradigm shift is a change of point from data-driven to value-driven.

Schmarzo described the analytical "line of sight" from data to value which is based on a framework as Data Lake with two types of data: raw data and curated data (data fabric) (2022; 2022). The aim of Data Lake's work is the creation of value that can constitute the decision factory. Behind this front side of created value should be the data management engine which is able to redirect data flows or processes in the needed way and at the right time. The Data Lake concept which was proposed in 2010 by Dixon (2010), is the common framework that allows building the analytical Big Data architecture from gathering data to machine learning and other computing processes (including high-performance computing or quantum computing). API platform for users is based on the Infrastructure as a Service (IaaS) approach that involves cloud orchestration technology with the opportunity for data discovery and predictive analytics. Data Lake framework manages to flexibly rebuild data presentation and to use unstructured and non-relational data for fundamentally different variants of analysis. No less important is an agile approach that requires the ability to quickly transform data structure. All mentioned turn raw data into curated data that is data fabric. Presentation of curated data may consist of clusters, principal component vectors, graphs, fractals, logical connections, etc. There may be different approaches to

understanding how the data reflect the real processes which undergo modeling. The difference itself in understanding how data should be presented determines the relevance of the obtained value and subsequent decisions made.

Epidemic data have a very complex structure and the maximum possible range in diversity. Raw data for epidemic modeling are characterized by multidimensionality and uncertainty. While processing epidemic data, the machine's rational logical output inevitably comes into direct conflict with reality because of the impossibility of accurately taking into account all the data that reflect all processes with all their interactions. Following Ashby's Law of Requisite Variety ("only variety can destroy variety") the number of possible states of obtained value should be greater than the number of states of the system which is being studied and modeled. The viral infectious diseases causative agents (viruses) circulate on a permanent basis in the global three-nexuses system: environmental, social, and immune. Each component of the three nexuses has an infinite number of options and their interactions. Furthermore, environmental, social, and immune systems as the parts of common space continuously evolve. The migration of viruses can be thought of as a random Brownian motion (random walk) within three nexuses. At the same time virus circulation obviously obeys the laws of nature, the essence of that isn't yet clear and has long been the subject of scientific study.

The science community faces two main problems in epidemic data modeling: how to describe the space in which data will be considered and modeled; and how to define the dynamics of objects. In essence, any space is the set of data, vectors, and rules for working with them. The space type is defined by the number of coordinates that are needed to find the position of a point. Very relevant to reality is the four-dimensional manifold that is the Minkowski space (three-dimensional Euclidean space and time) with elements that are called events. The three-nexuses system can be viewed as three coordinates (environmental, social, and immune) where the viral infectious diseases causative agent evolves for a time t (Fig. 1A). For Minkowski space the different events can be considered, for example, such as infection propagation or virus mutation. The space can be much more complex like the Riemannian manifold (smooth manifold M^n), for that tangent bundle gives the opportunity to use metric tensor (or tensor field) for determination of each point p on curvature (Lee 2003; Lee 2018). This allows considering infection propagation or virus mutation in a multiparametric manner as vectors spaces on a manifold M , for which smooth continuous function $F: M^n \rightarrow T(M^n)$ assigns to every point $p \in M^n$ a vector $v \in T_p(M^n)$, as well as this, allows considering the continuations of isometry φ regarding time t (Fig. 1B).

The more sophisticated space which corresponds to multidimensionality and uncertainty is the Hilbert space with conjugate symmetry (1):

$$\langle \psi | \phi \rangle = \langle \phi | \psi \rangle^\dagger. \quad (1)$$

Symmetries play a central role in field theories, as well as symmetries, are essential for understanding and describing the real world (Özdemir et al. 2019). The fundamental symmetry of physical laws in nature includes: C-symmetry which is the charge conjugation, changing the sign of all charges; P-

symmetry which is the parity transformation, simultaneous mirror flips in the sign of all three spatial coordinates, also chirality; and T-symmetry which is the theoretical time reversal or reversal of the entropy increasing as time flows towards the future time reversal. Charge, parity, and time reversal symmetries have all quantum systems with a Hermitian (operator equal to its own conjugate transpose) Hamiltonian (operator corresponding to the total energy of the quantum system). Hamiltonian H is assumed to be Hermitian, if (2):

$$\hat{H} = \hat{H}^\dagger, (2)$$

that denotes transposition plus complex conjugation (Özdemir et al. 2019). Hermitian product or inner product on Hilbert space is a conjugate-symmetric scalar multiplication (3):

$$\langle \phi | \hat{A} \psi \rangle = \langle \hat{A}^\dagger \phi | \psi \rangle. (3)$$

Hermitian matrix is equal to its own conjugate transpose. Hermitian symmetry is a fundamental principle in quantum mechanics, but in the big world, asymmetric matrix is observed or hypothesized. Interactions among objects in the medical, biological, social, and behavioral sciences are generally asymmetric in most cases (Chino 2020). Chino emphasized that the following question is important: what is an appropriate mathematical space in which objects are embedded with asymmetric relationships among them? (2020).

There are systems that cannot be described by a Hermitian Hamiltonian – that kind of non-Hermitian system (4)

$$\hat{H} \neq \hat{H}^\dagger, (4)$$

has been attracted by their nontrivial properties (Krasnok et al. 2021). Non-Hermitian Hamiltonian obeys a special form of symmetry which is known as parity-time (PT-) symmetry. The main feature of PT-symmetry is an exceptional point, which is also called singularity – the phase transition due to spontaneous breaking around an exceptional point. This phase transition is of great interest for epidemic modeling.

How to define the dynamics of objects in space is another main problem in epidemic data modeling. It refers to rules for working with data. Stochastic resonance is an example of how the chaotic complexity of virus circulation can be considered; strengthening, amplification of some “signal” is going on due to some “white noise”, i.e., influence factors. Stochastic resonance can help to answer the following questions: how does a virus transition from one host to another appear in the biota; what the circumstances, what the “white noise” does boost the specific mutations, and that way, how does mutated virus along a chain become pathogenic for humans? Epidemic is the subject of stochastic dynamics comparable to Markov chain (Markov process) for which the present state depends only on the previous step without relations with the past (“memorylessness”), thus there isn’t a certain relative connection between the past state and the future state. People who are standing close to the infected one

in a public place can become infected. This fact mainly depends on the last step in the events chain – these people are standing side by side together in the common space, but their past and future don't have a direct binding.

According to Virus-Host Database (<https://www.genome.jp>), there are 1384 known viruses for which *Homo sapiens* is the host (Mihara et al. 2016). Viruses and human immune system on population level mutually force the evolution of each other which can be compared with some symmetry. The immune system itself, as well as the virus with its mutations, both are dynamical multiscale systems that permanently interact. Altan-Bonnet et al. presented the broad set of quantitative immunology mathematical approaches which include modeling of antigen presentation, ligand-receptor equilibrium, signaling pathways, and gene regulation (2020). The authors mentioned Bayesian statistics and Lotka-Volterra model, both help to understand lymphocytes, cytokines, and antigens interaction through signaling pathways. Kullback-Leibler divergence or relative entropy, Moran model, and Wright-Fisher model allow studying a viral genetic drift and mutation-selection balance as an antigenic environment for human immune system response with antigen-presenting cell and major histocompatibility complex.

The list of mathematical models and approaches for epidemics modeling can be said to be endless. In this study, DNA viruses were chosen for the creation of detailed ontology in relation to symptoms and diseases. The ontology itself indicates data uncertainty, multidimensionality, and disconnection in terms of dispersion across data sources, variety and often similarity of symptoms, the possibility of co-infection with different combinations of viruses, as well as an infinite number of influencing factors on infection outcome. Facing the complexity of obtained data, an approach to building the Data Lake for quantum computing is proposed. How the Quantum Data Lake can help in modeling a very complicated epidemic landscape with high relevant value is shown in the article.

2. Related Works

It is necessary to mention the article of Upham et al. (2021) in which the problem of disconnected data about viruses was raised. The authors emphasized that a lot of information is accumulating, in particular about host-virus interaction. But this taxonomic data remain 'dark', very scattered, and thus of no value. Data can be valuable and can manage to form the newly added knowledge, useful for epidemic forecasting and control, only if different data flows will be linked together on a massive scale.

Massad et al. (2003) asserted that in the field of biosciences the problem of uncertainty is the most critical in epidemiology and medicine. As the authors wrote, a single symptom may be indicative of different diseases. According to viral infections, a single symptom may appear in presence of a very wide range of viruses. Uncertainty can be shown in the example of health or disease states which should be opposites to each other. In the case of the asymptomatic presence of one virus or even several different viruses simultaneously the health state becomes not so clear considering the risk of disease progression. It is necessary to formulate a deeper problem associated with uncertainty – even if the symptoms of the viral disease are clear, the virus's presence doesn't guarantee until it can be proven by laboratory methods.

Fuzzy logic allows considering health and disease as some buffer equilibrium of different states and circumstances. Massad et al. divided uncertainty in epidemiology into two types: stochasticity and lack of knowledge.

Uncertainty finds its reflection in fuzzy logic, and fuzzy sets. For building the optimal fuzzy model (decision making, diagnosis, prediction, or classification) an algorithm should respond to the highest adaptability to data. That kind of adaptability can be reached due to fuzzy cognitive maps which are the combination of fuzzy logic properties with artificial neural networks (Amirkhani et al. 2017). Amirkhani et al. described three types of methods for training fuzzy cognitive maps to obtain a weight matrix: a) adaptive method which is based on the Hebbian law (Hebbian learning related to the neuroscientific concept of synaptic plasticity); b) population-based methods – particle swarm optimization, simulated annealing (also Metropolis-Hastings algorithm / Markov chain Monte Carlo), genetic algorithm, tabu search, ant colony optimization; and c) hybrid method combining adaptive and population-based approaches. Fuzzy logic implementation in epidemiology is a powerful tool for modeling complex processes. The example is the following. Mei et al. (2014) used the fuzzy cognitive map to study the influenza A/H1N1 virus spread in the condition of uncertainty associated with people's individual behavior.

Tchapet Njafa and Nana Engo (2018) presented the model of Quantum Associative Memory for four tropical diseases (malaria, typhoid fever, yellow fever, and dengue) to diagnose a single infection or combined polyinfection. The authors explain their choice in favor of quantum computing by the main quantum properties: 1) qubit can take two states $|0\rangle$ and $|1\rangle$ constituting Hilbert space; 2) n -qubits register can exist in all its possible 2^n states at the same time due to quantum superposition; 3) the power of quantum information is enhanced by quantum entanglement; all involved into computing qubits represent one unique physical system instead of separated subsystems in a conventional set of gates and parallel computing; 4) it is possible to perform multiple computations simultaneously that is quantum parallelism; 5) unlike classical conventional logic gates, the quantum logic gates are reversible, and therefore they can mitigate entropy. Tchapet Njafa and Nana Engo modified the associative Hopfield network (which consists of binary threshold units and is called the “spin glass system”) and created learning and retrieving algorithms like quantum. Qubits are neurons; network connections are substituted by quantum entanglement; learning means superposition of entangled states; search result is unitary transformation; and output is decoherence. The Quantum Associative Memory model includes 14 qubits and can distinguish a single infection from a co-infection without laboratory facilities. Each of the four flag qubits is associated with one disease. The authors emphasized that only two qubits are able to double the database of diseases.

Singh and Bose (2021) implemented the algorithm called “quantum” for conventional non-quantum computing. The purpose of the algorithm is the segmentation of chest computed tomography scan images so that pneumonia regions can be accurately detected in patients with the COVID-19. The authors described the algorithm as a novel heuristic search, fast-forward quantum optimization algorithm with K-means clustering (FFQOAK). Singh and Bose considered the computing task in terms of a quantum

system having quanta locations (own point of origin), quanta uncertain movements (state of location change), and displacements (completed change in location). Quantum can move to achieve the best displacement through an inertial time. The algorithm work is based on the simulation of quantum displacement activity following the Schrödinger equation. Each quantum Q is the search agent which moves in the multidimensional search space. Quanta uncertain movements lead to displacements that give both stability and optimal structure to the quantum system as a whole. The achievement of stability in the multidimensional search space denotes quantum successes. The displacement of a quantum is affected by the displacements of surrounding quanta. Each quantum maintains information about its own location, movement, displacement, and successes, as well as about displacement and successes of surrounding quanta. Enhancements in quanta displacements happen due to the exchange of information with successful quanta. The search for the best displacement continues until each quantum achieves its own best displacement. Thus, the FFQOAK algorithm fulfills the solving optimization problem generating segmented images. The authors compared the performance of the FFQOAK algorithm with other methods: genetic algorithm, dynamic particle swarm optimization, and ant colony optimization. The high efficiency of the FFQOAK algorithm over other methods was shown.

The algorithm elaborated by León and Pozo (2007) for quantum computing predicts HIV infection spread. Each individual is represented by a qubit (5):

$$|\psi\rangle = a|0\rangle + b|1\rangle. \quad (5)$$

The infected human represents by state $|1\rangle$, non-infected represents by state $|0\rangle$. In an uncertain situation, if a human doesn't know his particular infection condition, this individual is considered a superposition. The authors used the Toffoli quantum gate to study interactions between qubits, i.e., interactions among individuals. The evolution of epidemic is described by the evolution of the quantum system in time – unitary matrix U transformation due to the Schrödinger equation. The simulated dynamics of the HIV infection showed a correlation with actual retrospective data in the Chile population for 15 years.

Beneduci et al. (2021) simulated the spatio-temporal evolution of the COVID-19 epidemic applying quantum system dynamics determined by the Schrödinger equation for evolution over time of a wave function ψ . The authors implemented the analogy with quantum mechanics for SIR models. Functions $\psi_t^S(x)$, $\psi_t^I(x)$, and $\psi_t^R(x)$ represent the probability density in time for susceptible (S), infectious (I), and recovered / immune (R) individuals. The state of the epidemic is defined by three probability clouds (ψ^S , ψ^I , ψ^R) which are given by the square of the modulo of the wave function ψ . Σ is the area of infection spread, and the point on the area is defined as $(x,y) \in \Sigma$. Beneduci et al. denoted the probability that an infected person is placed in the region $\Delta \subset \Sigma$ at time t as $\int_{\Delta} \psi_t^I(x) d^2x$. According to quantum statistical mechanics, function ψ describes an infinite ensemble of copies of the particle in Hilbert space. A person can be infected with probability $\int_{\Sigma} \psi_t^I(x)$, as well as can become recovered $\int_{\Sigma} \psi_t^R(x)$ or might be susceptible $\int_{\Sigma} \psi_t^S(x)$. Three probability clouds give the quantum space distribution of the infected, susceptible, and recovered people among an ensemble of copies generated as many times as needed

which is reflected by the Hamiltonian operator as the value of the total energy of the quantum system. The Hamiltonian operator describes the interaction between the different particles of the quantum system. The authors encoded the infection transition from x_1 to x_2 at time t as kernel $W(\tau, x_2, x_1)$. Kernel W allows assuming the existence of the transition probability and serves as the analog of the Hamiltonian operator. The Fisher-Kolmogorov equation was implemented to elaborate the space-time non-linear continuous probabilistic model of the infected density evolution ψ_t^I among N individuals. It is worth adding that the Fisher-Kolmogorov equation (Fisher-Kolmogorov-Petrovsky-Piskunov equation) reflects the reaction-diffusion system, stochastic fluctuations, and wave propagation; the generalized version of the Fisher-Kolmogorov equation is related with non-homogeneous time-dependent diffusion (anomalous diffusion), i.e., non-linearity of the probabilistic model. Thus Beneduci et al. created the epidemic forecasting model which uses the kernel density estimation method to show the increase or decrease of the infection probability ψ_t^I in chosen locations.

Omar Alzeley (2019) in the paper “Epidemics with a Path to Quantum Epidemics” wrote that the compartmental model SIR still remains the most applied in epidemiology. The SIR model originated from Ronald Ross & Hilda Hudson (1916–1917), and Anderson McKendrick & William Kermack (1927–1933). The spread of infection in terms of the SIR model mostly has been investigated with regard to classical probability, considering epidemics as a deterministic or stochastic process, which happens in continuous or discrete time, by phase-type or exponential type. The author mentioned that the Bayesian Markov Chain Monte Carlo has been generally used for modeling random infection transition. Classical methods face multidimensionality and uncertainty such as non-homogeneous populations or different choices of compartmental models: SIR, SIS, SIRD (D – Deceased), MSIR (M – maternally derived immunity), SIC/R (C – Carrier state), SEIR (E – Exposed), SEIRD, SEIS, MSEIR, MSEIRS, as well as adding a state of V (vaccination), Q (quarantined), and IS (insusceptible). Considering multidimensionality and uncertainty, Omar Alzeley emphasized the advantage of the new way of epidemics modeling which is the quantum probability tools, one of the variants of which is a quantum random walk.

Pomorski (2020) investigated the quantum essence of epidemic uncertainty which comes from a superposition state: health or infectious disease. The author showed the example of a superposition state matrix based on a combination of quantum computing, quantum mechanics, and classical statistical equations (1). The co-dependence of two probabilities of states 1 (health) and 2 (disease) corresponds to S-matrix (scattering matrix). Pomorski wrote that the epidemic model can be reproduced by quantum entanglement. The author discussed the infection propagation for a time and probabilities of health or disease states, using such quantum notions as eigenstates ψ_{E1} and ψ_{E2} , vectors in a Hilbert space, quantum tight-binding model, quantum matrix Hamiltonian, quantum density matrix, quantum phases, quantum field theory, and Hamiltonians interaction between quantum systems for a time $\hat{H}_0(t) + \hat{H}_{A-B}(t)$.

Thus, in concluding this section, it must be emphasized that epidemic modeling or, in other words, modeling of infectious diseases spreading faces several significant problems such as disconnected data, uncertainty, and multidimensionality. In discussed works above researchers have made an impact in

solving these problems by addressing different approaches – Markov process, fuzzy logic, and quantum theory. In this article, the quantum theory application to databases and Data Lake creation for epidemic modeling are presented. Figure 2 shows the continuum of consecutive selection of approaches for modeling associated with infectious diseases in discussed works above. The starting point is raw data which has no value and includes the three main problems: disconnected data, uncertainty, and multidimensionality (Fig. 2–A). The path from data to value is beginning from fuzzy logic. Then, delving into the problem of uncertainty, the quantum approach becomes preferable. The proposed solutions are complicated by relying on basic concepts: superposition, entanglement, parallelism, unitary transformation, quantum random walk, quantum tight-binding, spaces, matrices, operators, logic gates, and neural networks (Fig. 2–A, B, C). These diverse concepts allow to take into account uncertainty, and multidimensionality, and overcome data disconnection. But the quantum approach can open an entirely new door on the way to turning data into value, namely, time, energy, and symmetry together must be taken into account in the presentation of epidemic data (Fig. 2–C). This is a step forward that can be made on the basis of the path already covered in the application of the quantum approach.

3. Study Design, Materials, And Methods

This study consists of two stages:

- 1) data about DNA viruses were collected and analyzed from databases of the International Committee on Taxonomy of Viruses (ICTV) and The National Center for Biotechnology Information taxonomy (NCBI); the detailed ontology in relation to symptoms and diseases was created by investigation of articles from NCBI, PubMed, and ScienceDirect, see the full list of sources in Supplementary Information file 1 and infographics in Supplementary Information file 2;
- 2) Quantum Data Lake concept is proposed; quantum theory with regard to time, energy and symmetry was applied to all considered data.

4. Results And Discussion

DNA viruses that infect humans include eight families: Poxviridae, Herpesviridae, Adenoviridae, Polyomaviridae, Papillomaviridae, Circoviridae, Parvoviridae, and Hepadnaviridae. Among these viruses, there is one high-consequence pathogen Variola virus (Variola major and Variola minor, family Poxviridae, genus Orthopoxvirus) which causes the particularly dangerous, deadly infection smallpox. The World Health Organization (WHO) confirmed the global eradication of smallpox in 1980 (<https://www.who.int/news-room/fact-sheets/detail/monkeypox>). In May 2022 WHO reported about the new outbreak of monkeypox expanded to 50 countries, which is less contagious than smallpox and causes less severe illness. More than 60 thousand people had confirmed cases of monkeypox due to WHO Health Emergency Dashboard (<https://extranet.who.int/publicemergency/#>).

Herpesviruses are characterized by long-term persistence in the human body, keeping a latent state inside the host's cell without clinical manifestation. Herpesviruses cause genital herpes, infectious mononucleosis, Hodgkin's lymphoma, and Kaposi's sarcoma in AIDS patients. Acute respiratory disease, gastroenteritis, and meningitis appear due to mastadenoviruses infection. Polyomaviruses can keep an asymptomatic presence, and cause a broad range of tumor types, including skin tumor – Merkel cell carcinoma, as well as central nervous system neoplasms.

More than 200 Human papillomaviruses (HPVs) were identified. Visualization of the classification by genera of HPVs (numbers of viruses' types) is presented in Fig. 7. HPVs are oncogenic and cause many different tumors, including cervix, ovarian, breast, skin, lung cancer, and also, papillomas, epidermodysplasia verruciformis, warts (condyloma acuminata, verruca vulgaris and planae, myrmecia), as well as HPVs are characterized by asymptomatic presence on normal skin.

Cycloviruses of the Circoviridae family were identified in patients with nonpolio acute flaccid paralysis and paraplegia patients, either both cyclovirus and circovirus have an asymptomatic presence in the stool. Parvoviruses cause very diverse diseases from glomerulonephritis and hepatitis to cutaneous malignant melanoma. Also, adeno-associated dependoparvoviruses are used as vectors for gene therapy. Among the Hepadnaviridae family there is one species contagious to humans – the Hepatitis B virus. WHO confirmed in 2019 that 296 million people have been infected by the Hepatitis B virus and are living with chronic hepatitis B infection. Over 1.5 million new hepatitis B cases appear per year (<https://www.who.int/news-room/fact-sheets/detail/hepatitis-b>). The detailed ontology in relation to symptoms and diseases is presented in Tables 1–9, and also it is shown as infographics in Supplementary Information file 2.

Obtained ontology of eight families of DNA viruses, which infect humans, with symptoms and diseases were considered in terms of quantum theory. All data apply to Hilbert space H which consists of all complex n -dimensional vectors. Information processing in a quantum system is implemented by qubits which have a continuum of possible values; n qubits are represented by a superposition state vector in 2^n dimensional Hilbert space (Knill et al. 2002; Rieffel and Polak 2014). Information about viruses' families, subfamilies, genera, and species can be computed using many quantum bits. Each single-qubit system can be represented as points on a sphere (Fig. 3).

Quantum approach could manage to consider the problem of assessing the spread of an epidemic as just a problem of sophisticated encryption. Quantum computing very depends much on quantum database architecture. Quantum databases should be based on qubits, can store exponentially more information, and get information instantaneously compared to conventional databases. In contradistinction to Structured Query Language (SQL) for relational databases and NoSQL for unstructured Big Data, Quantum Query Language (QQL) was proposed by Schmitt (2008) for quantum databases. Schmitt established by QQL the correspondences between quantum logic and querying where the unit vector is the database object, the state vector is the database tuple, projector/vector space is the query, the quantum measurement is the query processing, and probability values are the truth values. The

author emphasized that implemented in QQL mathematical formalism for quantum mechanics combines such concepts as Hilbert space, quantum logic, and probability theory (Schmitt 2008). A full-fledged result while working with databases can only be obtained within the special complicated framework as Data Lake. Two Data Lake concepts are shown in Fig. 4 – the traditional architecture with layers from raw data to value and the Quantum Data Lake architecture proposed in this article. The proposed Quantum Data Lake brings together all the benefits and capabilities of quantum data processing in one framework.

The Quantum Data Lake architecture consists of several layers. It works with raw Big Data, applying to data different quantum operators and gates. Plenty of information about quantum operators and gates for databases can be found in scientific articles (Cockshott 1997; Younes 2007; Younes et al. 2008; Gueddana et al. 2010; Hamouda et al. 2016; Figgatt et al. 2017; Luongo 2022) and sources: Microsoft (<https://docs.microsoft.com/en-us/azure/quantum/>); IBM (<https://quantum-computing.ibm.com/composer/docs/iqx/>); Qiskit (<https://qiskit.org/textbook/ch-states/introduction.html/>). The most important property of a quantum database is the capability to be in more than one state at the same time. Any transaction to data silos is in one of many quanta states which remain unknown until it will be fixed by measurement. Data processing can be implemented by single-qubit and multiple-qubit systems (a quantum system with more than one qubit is the quantum register). Vector operations include inner product expressed by Dirac \langle bra|ket \rangle notation, outer product (matrix operator), and tensor product \otimes of a set of vector spaces which combines smaller quantum systems in a single larger quantum system. The most common queries are SELECT, INSERT, UPDATE, DELETE, BACKUP, and RESTORE. QQL depends more on probability amplification for the amplitude. The quantum database records or superposed tuples (different columns for the same row) are represented by vector space in the quantum system. The measurement will cause the database to collapse on only one of its tuples. Quantum searching, as well as quantum teleportation, based on entangled state (two-qubit entangled states referred to as Bell state).

Grover's algorithm allows searching and implements SELECT statement (to retrieve any required information from a database according to the users' criteria) boosting the amplitude of the similar joined components, i.e., it takes into consideration the amplitude of the data in the database, amplifying the amplitude of the needed data while decreasing the amplitude for the unneeded data. Grover's algorithm searches through the database using parallelism, it acts on the register in a superposition of states of each element at one time. Grover's algorithm applies to the Hadamard gate (Hadamard transformation) which performs rotation of every qubit and is setting up an equal superposition of each of the database elements (Younes 2007; Gueddana et al. 2010; Hamouda et al. 2016). On the whole Grover's algorithm has four stages: initialization (Hadamard transformation creates an equal superposition of all states), quantum oracle (oracle O marks the solutions by flipping the sign of that state's amplitude), amplification (increasing the amplitude of the marked state), and measurement (Bender et al. 1999). Two alternative methods of encoding the marked state within the oracle exist: the Boolean Quantum Logic (CNOT gate and Toffoli-3 gate, Toffoli-4 gate) and the phase method (controlled-Z gate) (Figgatt et al. 2017). The

partial diffusion operator D_p is used for enhancement quantum searching by alternating inversion to resist the de-amplification behavior of a quantum system (Younes 2007; Younes et al. 2008).

UPDATE statement (updating a set of records) can be done using CNOT gate by permutation with unitary operator U or replacing the qubit states (Younes 2007; Hamouda et al. 2016). INSERT statement (inserting one or more records into the database) can be implemented using a controlled Hadamard gate (Younes 2007; Gueddana et al. 2010; Hamouda et al. 2016). DELETE statement (deleting records from the database) can be made by Grover operator G (Gueddana et al. 2010; Hamouda et al. 2016). BACKUP (backing up a required portion of a database) and RESTORE (restoring a backup) require key qubit, oracle, and partial diffusion operator D_p (Younes 2007).

Layer “Quantum tools” should contain special capability that allows building models for solving specific applied tasks. From our point of view, this layer should include the following: matrix product states with entanglement; generalization to the tensor networks as quantum tensor network projected entangled-pair states and multi-scale entanglement renormalization ansatz; ability to use fundamental symmetry properties (parity and time reversal symmetry, PT-symmetry); reshaping operations: matricization, vectorization and tensorization; and teleportation. Changes in the epidemic situation can be predicted through modeling with consideration of all incoming data. Using such unique quantum properties as Hermiticity and non-Hermiticity, predicted changes followed by continuously adding and updating data can be estimated as the shift in data in the fractal tensor network. Presented in Fig. 5 approach is explained in detail below.

The combined PT-symmetric operators constitute a simple subclass of pseudo-Hermitian operators. Operator P represents parity reflection and operator T represents time reversal. These two operators are defined by their combined action on the coordinates r and momentum p operators. Bender and Boettcher have shown that Hermiticity isn't a necessary condition for real eigenvalues of Hamiltonian under the combined PT-symmetry, thus non-Hermitian Hamiltonian can have real eigenvalues without Hermiticity (Bender and Boettcher 1998; Bender et al. 1999; Jones-Smith and Mathur 2010; Jones-Smith and Kalveks 2013). PT-symmetric Hamiltonian is (6, 7):

$$\hat{H}(p, r, t) = \hat{H}^\dagger(p, -r, -t), \quad (6)$$

$$[\hat{H}, PT] \equiv \hat{H}(PT) - (PT)\hat{H} = 0. \quad (7)$$

\hat{H} shares common eigenfunctions with the PT operator. A quantum system may be entangled and exchange energy with the environment, the evolution of that kind of system follows a non-Hermitian Hamiltonian (Zhang et al. 2021). The physical origins of non-Hermiticity include radioactive decay, nonreciprocity of state transitions in stochastic processes, dissipation in open quantum systems, population dynamics and biological networks, including neurons, and energy transfer in photosynthesis (Ashida et al. 2021). Eigenvalues of a PT-symmetric non-Hermitian Hamiltonian are real in PT-symmetry as a result of balanced energy gain and loss (Kanki et al. 2017).

PT-symmetry is unbroken, exact if every eigenstate of a PT-symmetric non-Hermitian operator satisfies PT-symmetry; then, the entire spectrum is purely real even though the operator of interest isn't Hermitian (Ashida et al. 2021). PT-symmetry is spontaneously broken or purely imaginary at an exceptional point if operators \hat{H} and PT have different eigenvectors. This is the breaking of the balance between energy gain and loss, and it should be distinguished from a genuine time-reversal symmetry breaking (Rüter et al. 2010; Kanki et al. 2017; Miri and Alù 2019). PT phase transition at an exceptional point is the real-to-complex spectral transition (spectral singularities in the parameter space of a system) which is accompanied by the real part and the corresponding imaginary part of non-Hermitian Hamiltonian eigenenergies with simultaneous coalescence of two or more eigenvalues, and their corresponding eigenvectors (Miri and Alù 2019). The system behaves as it loses its dimensionality near an exceptional point because the vector space becomes skewed (8) (Özdemir et al. 2019),

$$V_1, \lambda_1 = V_2, \lambda_2. \quad (8)$$

Such degeneracy as an exceptional point is a peculiar feature of nonconservative systems that exchange energy with their surrounding environment (Miri and Alù 2019).

Besides the special circuits created for non-Hermiticity, it is necessary to mention the distribution of non-Hermiticity among the usually applied gates. Most of the used quantum gates, such as Hadamard, Pauli, CNOT (Feynman), CCNOT (Toffoli), SWAP, and controlled SWAP (Fredkin), are self-inverse (implies Hermiticity for unitary operations) and reversible (gates with a property of the equal number of inputs and outputs, the outputs can be determined using the inputs, the inputs can be recovered from the outputs) (Pathak 2013; Samrin et al. 2022). But Pathak argued that there are many quantum gates that are non-self-inverse, i.e., are non-Hermitian in nature. The possibility of non-Hermiticity increases with a dimension of quantum gates from more than half cases for 2-qubits gates to a very high possibility for 4-qubits quantum gates (Pathak 2013).

Rasmussen and Zinner (2020) mentioned the often-used non-Hermitian gates: Phase shift R_φ ; Square root of NOT, \sqrt{NOT} ; Imaginary swap $iSWAP$; Square root of swap, \sqrt{SWAP} ; Ising XX coupling; Ising YY coupling; Ising ZZ coupling; Deutsch D_θ , as well as the authors presented the multi-qubit controlled- $iSWAP$ non-Hermitian gate, and also, they proposed the quantum circuit for probabilistic exponentiating of non-Hermitian quantum gates. Zhang et al. (2021) proposed the quantum circuit which can design the non-Hermiticity due to the fact that the non-Hermiticity of physical systems is generated from the entanglement with the environments. The "system" qubits are entangled with the ancilla qubits in the quantum circuit. Presented by Zhang et al. non-Hermitian unit includes unitary operator U, Pauli rotation operator $R_x(\theta)$, controlled rotation operator $CR_x(\theta)$, and ancilla qubits measurement.

Gao et al. (2021) showed that the dynamical evolution of qubit (quantum state) over time could be observed under a PT-symmetric Hamiltonian by using the conventional quantum gates. The authors designed the combined quantum circuit to simulate PT-symmetry which consists of a two-qubit system (ancillary and working qubits), unitary operators, and the Hadamard gate. The configuration of the

system includes three modules: preparation, evolution, and detection. The dynamical evolution of qubit under a PT-symmetric Hamiltonian is observed with high fidelity with respect to different time t . Dogra et al. (2021) realized a quantum simulation of PT-symmetry breaking at an exceptional point for a single-qubit (this phase transition is associated with a loss of state distinguishability) and for two- or many-qubit systems (that is associated with apparent violation of entanglement monotonicity). Entanglement can be increased with the local PT-symmetric operation (Chen et al. 2014). The authors used the unitary operator U , Pauli rotation operators $R_y(\theta)$ and $R_z(\theta)$, CNOT gate, and ancilla qubit.

Fang et al. (2021) described the phenomenon of scale-free formation of numerous exceptional points which have a relation to the scale-free distribution of critical gain/loss intensities and to the spectral fractal dimension of the corresponding Hermitian spectra. Chernodub and Ouvry showed strong evidence of the fact that the entire energy spectrum of a non-Hermitian Hamiltonian (real and imaginary parts) is a fractal, similar to the real energy spectrum (Chernodub and Ouvry 2015).

Data-driven formation of a fractal is shown in Fig. 6. In the context of many-body quantum systems the tensor network methods found their application successfully (Cincio et al. 2008; Giovannetti et al. 2008; Evenbly and Vidal 2009; Evenbly and Vidal 2013; Montangelo 2018; Ran et al. 2020; Srinivasan et al. 2020; Araz and Spannowsky 2022). Tensor networks are used to simulate entangled quantum systems representing by themselves both quantum states and quantum circuits. In general, quantum tensor networks have been designed to implement real-space renormalization which allows for finding the ground state of a spin system and truncating the exponential increasing Hilbert space based on energy considerations. The many-body quantum system H_n is formed by the tensor product of all the local ones (9) and is constructed by adding the low-energy states of the Hamiltonian; the system size is increased at each iteration and the high-energy sectors of the different parts of the system are discarded (density matrix renormalization group). The wave function $|\psi\rangle$ gives the amplitude of probability of each possible system configuration. The many-body wave function is constrained to be the product of n independent single-body wave functions. The reduced density matrix of one block is obtained by taking the rest of the system as an environment (Montangelo 2018; Ran et al. 2020; Srinivasan et al. 2020).

$$H_n = H_1 \otimes H_2 \otimes H_3 \otimes H_4 \dots \quad (9)$$

The matrix product states (MPS) is the one-dimensional tensor network ansatz with entanglement in quantum many-body systems. MPS encodes the coefficients of the wave-function $|\psi\rangle$ in a product of matrices. The MPS structure represents the states whose entanglement only scales with the boundary of a region and gives finite correlations. MPS can be used to characterize state violating the area law of entanglement, such as ground states of critical systems (Ran et al. 2020). The projected entangled pair state (PEPS) is the generalization of the MPS; the tensors of PEPS are located in d -dimensional form instead of one-dimensional for MPS. These tensor networks have their own operators – matrix product operator (MPO) and projected entangled pair operator (PEPO) representing $\langle \text{bra} | \text{ket} \rangle$ notation. The tensor network on the fractal Sierpinski gasket is an example of some kind of PEPS for which the tensor is given by the probability distribution of the three spins in a triangle. The fractal tensor network can be exactly

contracted as a whole by iteratively contracting each three of the tensors located in the same triangle; the dimension of the tensors and the geometry of the network keep unchanged, but the number of the tensors decreases from N to $N/3$ (Ran et al. 2020).

The multi-scale entanglement renormalization ansatz (MERA) is a layered tensor network that allows for studying hierarchical scale-invariant systems [Cincio et al. 2008; Giovannetti et al. 2008; Evenbly and Vidal 2009; Evenbly and Vidal 2013; Montangero 2018; Ran et al. 2020; Srinivasan et al. 2020; Araz and Spannowsky 2022]. A quantum tensor network can be changed by means of local transformations by inserting an identity operator between two contracted tensors, without changing the global state, as well as a tensor network can undergo time-dependent variations due to the time evolution operator (Montangero 2018). Teleportation is a very promising quantum tool that allows transporting quantum information from one quantum state to another; it implements using entangled pairs or clusters of qubits (Bennett et al. 1993; Bouwmeester et al. 1997; Boschi et al. 1998; Cao and Song 2005; Huang et al. 2020; Rajiuddin et al. 2020; Ayoade et al. 2022). Also, for Data Lake operation is important to be able to implement tensor reshaping operations, i.e., converting a tensor into a matrix and vice versa (Cichocki 2018).

In this way, we have shown some unique properties related to quantum theory, which open a new opportunity in epidemic modeling due to changes in such basic categories as time, energy, and symmetry. In contrast to the widely discussed advantages of quantum computing such as high-speed performance and very large parallel scale, our approach emphasizes the fundamental uniqueness of quantum theory, providing new possibilities for modeling, exploiting the PT-symmetric Hamiltonian and spontaneous PT-symmetry breaking at an exceptional point, balance of energy gain and loss, and tensor network as many-body quantum systems formatted as a fractal continuum (Fig. 6). These special capabilities help to estimate changes while computing the incoming Data Lake data and to predict the critical shift in the epidemic situations considering gain and loss changes in a compartmental model SIR or in other data such as diseases and clinical symptoms or mutations of viruses, with applying all possible influencing environmental, social, and immune factors.

5. Conclusion

The study is dedicated to the development of quantum epidemiology which is the expected next stage in epidemiology transformation as new quantum technologies have emerged. The study highlights the necessity to investigate the native processes of viruses' interaction with the human population by relying on quantum theory's natural properties. Implementation of quantum logic and reliance on a quantum theory is the fundamental difference between the current digital epidemiology and future quantum epidemiology. The Quantum Data Lake concept is proposed. It consists of several layers including a quantum database and quantum tools. In contrast to the widely discussed advantages of quantum computing such as high-speed performance and very large parallel scale, the proposed approach emphasizes the fundamental uniqueness of quantum theory for modeling.

Understanding potential trajectories of situations development in healthcare is crucial for long-term policy, especially for viral infectious diseases the sophisticated modeling platform is needed on the world level. DNA viruses make a tremendous impact on morbidity and mortality, causing critical damage to various organs, including diverse types of cancer. For example, in a worse world health scenario twenty years ahead there will be a doubling of the death cases caused by cancer (Foreman et al. 2018). Undoubtedly, to overcome the increase in morbidity and mortality, the newest and most advanced approaches in modeling must be applied. But no less important is the development of modeling itself with the improvement of quantum databases and the development of the Quantum Data Lake architecture, which, of course, has a universal application and isn't limited only to the tasks of epidemiology and healthcare in general.

Faced with the challenges of sustainable development, on the one hand, and standing in front of the open door of quantum theory, on the other hand, it is necessary to realize that a qualitative leap is possible in modeling for healthcare tasks due to a fundamentally new understanding of the quantum world.

Declarations

CRedit authorship contribution statement

Olga Kolesnichenko: Conceptualization, Project administration, Methodology, Validation, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing.

Igor Nakonechniy: Methodology, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing.

Supplementary Information

Supplementary file 1

Supplementary file 2

Declaration of Competing Interest

The author has no competing interests to declare that are relevant to the content of this article.

Conflict of Interest

The author declares that they have no conflict of interest.

The author has no financial or proprietary interests in any material discussed in this article.

Non-financial interests

The author has no non-financial interests to disclose.

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Tables

Table 1
The Poxviridae family viruses which infect humans, and the diseases and symptoms they cause

Family: Poxviridae	
Subfamily: Chordopoxvirinae	
Genus: Molluscipoxvirus	
Species:	molluscum contagiosum (water warts)
Molluscum contagiosum virus	
Genus: Orthopoxvirus	
Species:	smallpox
Variola virus – (Variola major and Variola minor)	
Species:	cowpox
Cowpox virus	
Species:	monkeypox
Monkeypox virus	
Genus: Parapoxvirus	
Species:	contagious pustular dermatitis
Orf virus	
Species:	pseudocowpox
Paravaccinia virus	
Species:	skin lesion
Bovine papular stomatitis virus	
Genus: Yatapoxvirus	
Species:	tanapox
Tanapox virus	
Species:	skin lesion
Yaba monkey tumor virus	

Table 2

The Herpesviridae family viruses which infect humans, and the diseases and symptoms they cause

Family: Herpesviridae	
Subfamily: Alphaherpesvirinae	
Genus: Simplexvirus	
Species:	cold sores, skin lesion
Human alphaherpesvirus 1	
Species:	genital herpes
Human alphaherpesvirus 2	
Species:	encephalomyelitis
Macacine Herpesvirus 1 – (Herpes B Virus)	
Genus: Varicellovirus	
Species:	chickenpox (varicella)
Human alphaherpesvirus 3 – (Varicella-zoster virus)	
Subfamily: Betaherpesvirinae	
Genus: Cytomegalovirus	
Species:	from asymptomatic to multi-organ damage
Human betaherpesvirus 5 – (human cytomegalovirus)	
Genus: Roseolovirus	
Species:	infects CD4 + T cells
Human betaherpesvirus 7	
Species:	neuroinflammation, encephalitis, multiple sclerosis, roseola
Human betaherpesvirus 6A, 6B	
Subfamily: Gammaherpesvirinae	
Genus: Lymphocryptovirus	

Family: Herpesviridae	
Species: Human gammaherpesvirus 4 – (Epstein–Barr virus)	infectious mononucleosis, Hodgkin's lymphoma, Burkitt lymphoma
Genus: Rhadinovirus	
Species: Human gammaherpesvirus 8 – (Kaposi's sarcoma-associated herpesvirus)	Kaposi's sarcoma, primary effusion lymphoma, Castleman disease

Table 3

The Adenoviridae family viruses which infect humans, and the diseases and symptoms they cause

Family: Adenoviridae	
Genus: Mastadenovirus	
Species: Human mastadenovirus A	enteric infection
Species: Human mastadenovirus B1, B2	conjunctivitis, acute respiratory disease, hemorrhagic cystitis, meningitis, encephalomyelitis
Species: Human mastadenovirus C	acute respiratory disease
Species: Human mastadenovirus D	keratoconjunctivitis
Species: Human mastadenovirus E	conjunctivitis, acute respiratory disease
Species: Human mastadenovirus F	gastroenteritis, infantile diarrhea
Species: Human mastadenovirus G	gastroenteritis

Table 4

The Polyomaviridae family viruses which infect humans, and the diseases and symptoms they cause

Family: Polyomaviridae	
Genus: Alphapolyomavirus	
Species: Human polyomavirus 5 – (Merkel cell polyomavirus)	skin tumor (Merkel cell carcinoma)
Species: Human polyomavirus 8 – (Trichodysplasia spinulosa polyomavirus)	skin lesion (Trichodysplasia spinulosa)
Species: Human polyomavirus 9 – asymptomatic presence	asymptomatic presence
Species: Human polyomavirus 13 – (New Jersey polyomavirus)	vasculitis, myopathy, dermatosis
Species: Human polyomavirus 14 – (Lyon IARC polyomavirus) / asymptomatic presence	asymptomatic presence
Species: Sorex araneus polyomavirus 1 – (former Human polyomavirus 12)	asymptomatic presence in liver
Genus: Betapolyomavirus	
Species: Human polyomavirus 1 – (BK virus)	hemorrhagic cystitis, tubulointerstitial nephritis, hepatitis, pneumonitis, meningoencephalitis, CNS neoplasms
Species: Human polyomavirus 2 – (John Cunningham virus)	progressive multifocal leukoencephalopathy, gliomas

Family: Polyomaviridae	
Species: Human polyomavirus 3 – (Karolinska Institute virus)	respiratory infection
Species: Human polyomavirus 4 – (Washington University virus)	lower respiratory tract infection
Species: Macaca mulatta polyomavirus 1 – (Simian vacuolating virus 40)	broad range of tumor types
Genus: Deltapolyomavirus	
Species: Human polyomavirus 6	pruritic rash
Species: Human polyomavirus 7	pruritic rash
Species: Human polyomavirus 10 – (Malawi or Mexico polyomavirus)	asymptomatic presence
Species: Human polyomavirus 11 – (Saint Louis polyomavirus)	asymptomatic presence in intestines

Table 5

The Papillomaviridae family viruses (Human papillomavirus, HPV) which infect humans, and the diseases and symptoms they cause. The identifiers transcript is presented in Table 6; visualization of the Human papillomaviruses classification by genera is presented on Fig. 3

Family: Papillomaviridae			
Subfamily: Firstpapillomavirinae			
Genus: Alphapapillomavirus			
Species: HPV 2	Cr23, Cr25, Pa2, W1, W3, W4, W5, W9	Species: HPV 61	Cr23, R8
Species: HPV 3	Cr30, W1, W2, W9	Species: HPV 62	R6, R7
Species: HPV 6	Cr3, Cr9, Cr11, Cr16, Cr19, Cr20, Cr22, Cr23, Pa1, Pa2, Pa3, W5	Species: HPV 66	Cr1, Cr22, Cr23, R9, W5
Species: HPV 7	W6	Species: HPV 67	Cr1, Cr5, Cr22, Cr23, R3, W5
Species: HPV 10	EV, W2, W5	Species: HPV 68	Cr1, Cr5, Cr22, Cr23, R3, W5
Species: HPV 11	Cr3, Cr9, Cr11, Cr16, Cr19, Cr20, Cr22, Cr23, Pa1, Pa2, Pa3, W5	Species: HPV 69	Cr23, Cr25, Pa2, W1, W3, W4, W5
Species: HPV 13	H1	Species: HPV 70	R4, R5, W5
Species: HPV 16	Cr1a, Cr2, Cr3, Cr4, Cr5, Cr6, Cr7, Cr8, Cr9, Cr10, Cr11, Cr12, Cr13, Cr14, Cr15, Cr16, Cr17, Cr18, Cr21, Cr31, R3a, W7	Species: HPV 71	Cr23, R8
Species: HPV 18	Cr1a, Cr2, Cr3, Cr4, Cr5, Cr6, Cr7, Cr8, Cr9, Cr10, Cr11, Cr12, Cr13, Cr14, Cr15, Cr16, Cr17, Cr18, Cr21, R3a, W7	Species: HPV 72	Cr23, R8
Species: HPV 26	Cr17, Cr23, Cr25, Pa2, W1, W3, W4, W5	Species: HPV 73	Cr23, R8
Species: HPV 27	W1, W2, W9	Species: HPV 74	Cr22, Cr23, W5
Species: HPV 28	W2	Species: HPV 77	Cr17, Cr30
Species: HPV 29	Cr23, Cr25, Pa2, W1, W3, W4, W5	Species: HPV 78	W2

Family: Papillomaviridae			
Species: HPV 30	Cr1, Cr22, Cr23, R9, Pa2, W5	Species: HPV 81	Cr23, R8
Species: HPV 31	Cr1, Cr5, Cr22, Cr23, R3, W5	Species: HPV 82	Cr23, Cr25, Pa2, W1, W3, W4, W5
Species: HPV 32	Cr5, H1	Species: HPV 83	Cr23, R8
Species: HPV 33	Cr1, Cr5, Cr22, Cr23, R3, W5	Species: HPV 84	Cr23, R8
Species: HPV 34	Cr23, R8	Species: HPV 85	Cr1, Cr5, Cr22, Cr23, R3, W5
Species: HPV 35	Cr1, Cr5, Cr22, Cr23, R3, W5	Species: HPV 86	Cr22, Cr23, W5
Species: HPV 39	R1, R2, W5, W7	Species: HPV 87	R6, R7
Species: HPV 40	Cr23, R10, Pa2, W5	Species: HPV 89	Cr26
Species: HPV 42	Cr22, Cr23, W5	Species: HPV 90	R6, R7
Species: HPV 43	Cr23, R10, Pa2, W5	Species: HPV 91	R6, R7
Species: HPV 44	Cr22, Cr23, W5	Species: HPV 94	W2
Species: HPV 45	Cr1, Cr5, Cr22, Cr23, R3, W5	Species: HPV 97	Cr22, Cr23, W5
Species: HPV 51	Cr23, Cr25, Pa2, W1, W3, W4, W5	Species: HPV 102	Cr24, In2
Species: HPV 52	Cr1, Cr5, Cr22, Cr23, R3, W5	Species: HPV 106	In1
Species: HPV 53	Cr1, Cr22, Cr23, R9, W5	Species: HPV 114	Cr 23, Cr23a, Cr24, Cr27b, In2
Species: HPV 54	Cr22, Cr23, W5	Species: HPV 117	Cr23, Cr25, Pa2, W1, W3, W4, W5

Family: Papillomaviridae			
Species: HPV 56	Cr1, Cr22, Cr23, R3a, R9, W5	Species: HPV 125	Cr23, Cr25, Pa2, W1, W3, W4, W5
Species: HPV 57	Cr20, Cr23, Pa2, Pa4, W1, W5, W9	Species: HPV 160	W2
Species: HPV 58	Cr1, Cr5, Cr22, Cr23, R3, W5	Species: HPV mEV06c12b	EV, W5
Species: HPV 59	Cr1, Cr5, Cr22, Cr23, R3, W5	–	–
Genus: Betapapillomavirus			
Species: HPV 5	Cr17a, Cr30, Cr31, EV	Species: HPV 113	Cr10, Cr15, Cr17a, Cr31
Species: HPV 8	Cr17a, Cr31, EV	Species: HPV 115	Cr17, W1
Species: HPV 9	Cr17a, Cr30, Cr31, EV	Species: HPV 118	W1
Species: HPV 12	Cr17a, Cr30, Cr31, EV	Species: HPV 120	Cr17a, Cr27, Cr30, Cr31, W5, Pa2, In1
Species: HPV 14	Cr17a, Cr30, Cr31, EV	Species: HPV 122	Cr17a, Cr30, Cr31, In1, In3
Species: HPV 15	Cr9, EV	Species: HPV 124	Cr9, Cr17a, Cr30, Cr31, In1
Species: HPV 17	Cr17a, Cr30, Cr31, EV	Species: HPV 143	In1, In3
Species: HPV 19	Cr17a, Cr30, Cr31, EV	Species: HPV 145	In1, In3
Species: HPV 20	Cr17a, Cr30, Cr31, EV	Species: HPV 150	Cr17, Cr31, W1
Species: HPV 21	Cr17a, Cr30, Cr31, EV	Species: HPV 151	Cr17, Cr31, W1
Species: HPV 22	EV	Species: HPV 152	Cr25
Species: HPV 23	Cr9, Cr17a, Cr30, Cr31, EV	Species: HPV 159	Cr27, W5, Pa2

Family: Papillomaviridae			
Species: HPV 24	Cr17a, Cr30, Cr31, EV	Species: HPV 174 (V001)	Cr17a
Species: HPV 25	Cr17a, Cr30, Cr31, EV	Species: HPV 182	No1
Species: HPV 36	Cr17a, Cr31, EV	Species: HPV 185	No1
Species: HPV 37	Cr17a, Cr30, Cr31, EV	Species: HPV 195	No1
Species: HPV 38	Cr17a, Cr30, Cr31, EV	Species: HPV 196	No1
Species: HPV 47	Cr17a, Cr31, EV	Species: HPV 206	No1
Species: HPV 49	Cr17a, Cr29, Cr30, Cr31, W1, W2	Species: HPV 209	No5
Species: HPV 75	Cr29, W1, W2	Species: HPV 217	No1
Species: HPV 76	Cr17a, Cr29, Cr30, Cr31, W1, W2	Species: HPV mEV03c09	EV
Species: HPV 80	Cr17a, Cr30, Cr31, EV	Species: HPV mHIVGc36	No3
Species: HPV 92	Cr17, Cr31	Species: HPV mm090c09	S2
Species: HPV 93	Cr17a, Cr31, EV	Species: HPV mm292c10	S2
Species: HPV 96	Cr17a, Cr31	Species: HPV mm292c14	S2
Species: HPV 98	Cr10, Cr15, Cr17a, Cr30, Cr31	Species: HPV mm292c88	S2
Species: HPV 99	No2	Species: HPV mm292c100	S2
Species: HPV 100	Cr10, Cr15, Cr30	Species: HPV mMTS1	EV, No2
Species: HPV 104	Cr10, Cr15, Cr17a, Cr31	Species: HPV mTVMBSFc09	No2
Species: HPV 105	Cr17a, Cr30, Cr31	Species: HPV mTVMBSGc2024	No4
Species: HPV 107	Cr17a, Cr30, Cr31	Species: HPV mw15c111	S1

Family: Papillomaviridae			
Species: HPV 110	Cr17a, Cr30, Cr31	Species: HPV RTRX7	Cr17a, Cr19, Cr21, Cr31, EV, Pa5
Species: HPV 111	Cr17a, Cr31	–	–
Genus: Gammapapillomavirus			
Species: HPV 4	W1, W3, W9, W10	Species: HPV 222	W5, S5
Species: HPV 48	Cr17a, R3a	Species: HPV 223	No1
Species: HPV 50	Cr17a, Cr31, R3a, EV, W5	Species: HPV 224	No1
Species: HPV 60	Cr32, W8, W10	Species: HPV 225	No1
Species: HPV 65	Cr17a, Cr31, Cr32, W1, W3, W10	Species: HPV mCG2	No2
Species: HPV 88	Cr17a	Species: HPV mCG3	No2
Species: HPV 95	W1, W3, W10	Species: HPV mCH2	No4
Species: HPV 101	Cr22, In1, In6, W5	Species: HPV mdo1c02	S5
Species: HPV 103	Cr27b, In1, In6	Species: HPV mdo1c232	S5
Species: HPV 108	Cr23	Species: HPV mga2c01	S6
Species: HPV 109	Cr17a, Cr27b, Cr34	Species: HPV mga2c70	S6
Species: HPV 112	W5	Species: HPV mDysk1	S4
Species: HPV 116	No3	Species: HPV mDysk2	S4
Species: HPV 119	Cr30, In1	Species: HPV mDysk3	S4
Species: HPV 121	In1	Species: HPV mDysk5	S4

Family: Papillomaviridae			
Species: HPV 123	Cr17a, Cr31, Cr33, In1	Species: HPV mDysk6	S4
Species: HPV 126	W1, W2, W10, EV	Species: HPV mEV03c05	EV
Species: HPV 127	No2	Species: HPV mEV03c40	EV
Species: HPV 128	W1	Species: HPV mEV03c45	EV
Species: HPV 129	Cr31, W1	Species: HPV mEV03c60	EV
Species: HPV 130	Cr33, W1, W5	Species: HPV mEV03c104	EV
Species: HPV 131	W1	Species: HPV mEV03c188	EV
Species: HPV 132	W1	Species: HPV mEV03c212	EV
Species: HPV 133	W1	Species: HPV mEV03c434	EV
Species: HPV 134	W1	Species: HPV mEV06c107	EV
Species: HPV 135	In1, In4	Species: HPV mEV06c118	EV
Species: HPV 136	In1	Species: HPV mEV07c367	EV
Species: HPV 137	W5, In1	Species: HPV mEV07c382	EV
Species: HPV 138	W5, In1	Species: HPV mEV07c390	EV
Species: HPV 139	In1	Species: HPV mFD1	No2
Species: HPV 140	W5, In1	Species: HPV mFD2	No2
Species: HPV 141	In1	Species: HPV mFi864	In7
Species: HPV 142	In1	Species: HPV mFS1	No2
Species: HPV 144	In1, In6	Species: HPV mHIVGc70	S5

Family: Papillomaviridae			
Species: HPV 146	In1, In4	Species: HPV mICB1	No2
Species: HPV 147	W5, In1	Species: HPV mKC5	No2
Species: HPV 148	Cr31, W5	Species: HPV mKN1	No2
Species: HPV 149	Cr31, Cr33	Species: HPV mKN2	No2
Species: HPV 153	W5	Species: HPV mKN3	No2
Species: HPV 154	W5	Species: HPV mL55	In5
Species: HPV 155	Cr17a, Cr31	Species: HPV mLCOS0c196	No2
Species: HPV 156	No5	Species: HPV mm090c10	S2
Species: HPV 157	No5	Species: HPV mm090c66	S2
Species: HPV 158	No5	Species: HPV mm090c145	S2
Species: HPV 161	No2	Species: HPV mMTS2	No2
Species: HPV 162	No2	Species: HPV mSD2	In7
Species: HPV 163	No2	Species: HPV mSE355	Cr17a, Cr30, Cr31
Species: HPV 164	No2	Species: HPV mSE379	W5
Species: HPV 165	W5	Species: HPV mSE383	W5
Species: HPV 166	No2	Species: HPV mTVMBSGc529	No4
Species: HPV 167	No2	Species: HPV mTVMBSGc2450	No4
Species: HPV 168	No2	Species: HPV mTVMBSHc13	No2
Species: HPV 169	No2	Species: HPV mTVMBSHc33	No2

Family: Papillomaviridae			
Species: HPV 170	No2	Species: HPV mTVMBSWc141	No2
Species: HPV 171	Cr10	Species: HPV mw02c24a	S1, No3
Species: HPV 172	No6	Species: HPV mw03c65	W5, S1
Species: HPV 173	In1	Species: HPV mw07c34d	S1, S3
Species: HPV 175	W5	Species: HPV mw07c68b	W3, S1
Species: HPV 176	No1	Species: HPV mw07c74b	W3, S1
Species: HPV 178	Cr17a, Cr31	Species: HPV mw11c13	S1
Species: HPV 179	W1, In4	Species: HPV mw11C24	S1
Species: HPV 180	Cr30, W5	Species: HPV mw11C39	S1
Species: HPV 181	No1	Species: HPV mw11C51	S1
Species: HPV 183	No1	Species: HPV mw18c07	W1, S1
Species: HPV 184	W1	Species: HPV mw18c11d	W1, S1
Species: HPV 186	No1	Species: HPV mw18c25	W1, S1
Species: HPV 187	No1	Species: HPV mw18c39	W1, S1
Species: HPV 188	No1	Species: HPV mw18c134	W1, S1
Species: HPV 189	No1	Species: HPV mw20c01a	S1
Species: HPV 190	No1	Species: HPV mw20c01b	S1
Species: HPV 191	No1	Species: HPV mw20c02c	S1
Species: HPV 192	No1	Species: HPV mw20c03a	S1

Family: Papillomaviridae			
Species: HPV 193	No1	Species: HPV mw20c04	S1
Species: HPV 194	No1	Species: HPV mw20c08a	S1
Species: HPV 197	Cr17	Species: HPV mw20c09	S1
Species: HPV 199	W1, W5, No6	Species: HPV mw20c10a	S1
Species: HPV 200	W5	Species: HPV mw21c693	W5, S1
Species: HPV 201	W5	Species: HPV mw22c09	W1, S1
Species: HPV 202	W5	Species: HPV mw23c08c	Cr19, W1, S1
Species: HPV 203	No1	Species: HPV mw23c77	Cr19, W1, S1
Species: HPV 205	No5	Species: HPV mw23c101c	W1, S1
Species: HPV 207	No1	Species: HPV mw27c04c	W1, S1
Species: HPV 208	No1	Species: HPV mw27c39c	W1, S1
Species: HPV 210	No1	Species: HPV mw27c52c	W1, S1
Species: HPV 211	No1	Species: HPV mw27c157c	W1, S1
Species: HPV 212	No1	Species: HPV mw34c04a	W1, W5, S1
Species: HPV 213	No1	Species: HPV mw34c11a	W1, W5, S1
Species: HPV 214	No1	Species: HPV mw34c14a	W1, W5, S1
Species: HPV 215	No1	Species: HPV mw34c28a	W1, W5, S1
Species: HPV 216	No1	Species: HPV mw34c34a	W1, W5, S1
Species: HPV 218	No1	Species: HPV mwg1c05	S1

Family: Papillomaviridae			
Species: HPV 219	W5, S5	Species: HPV mwg1c09	S1
Species: HPV 220	W5, S5	Species: HPV mZJ01	No3
Species: HPV 221	W5, S5	–	–
Genus: Mupapillomavirus			
Species: HPV 1	W1, W2, W9	Species: HPV 204	W5
Species: HPV 63	W3, S7	–	–
Genus: Nupapillomavirus			
Species: HPV 41	Cr19, W1, W5	–	–
unclassified Papillomaviridae			
Species: HPV 198	No1	Species: HPV RTRX5	Cr19, EV, W1
Species: HPV AZ1_1	No1	Species: HPV RTRX6	EV
Species: HPV HANO464	Cr23	Species: HPV RTRX8	Cr19, EV
Species: HPV ICPX1	Cr17b	Species: HPV RTRX9	Cr19, Cr21, EV, W1
Species: HPV JEB2	Cr1a	Species: HPV RTRX10	Cr19, EV
Species: HPV me180	Cr1	Species: HPV Xc	Cr1
Species: HPV RTRX1	Cr19, Cr21, EV, W1	Species: HPV Xd	Cr1
Species: HPV RTRX2	Cr19, EV, W1	Species: HPV Xf	Cr27b
Species: HPV RTRX3	EV, S8	Species: HPV Xg	Cr27b

Family: Papillomaviridae

Species: Cr34, EV
HPV
RTRX4

Species: HPV Xh Cr27b

Table 6

The identifiers transcript for diseases and symptoms which are caused by Human papillomaviruses

Cr1	cervix carcinoma
Cr2	ovarian cancer
Cr1a	cervical and uterine cancer
Cr3	vulva carcinoma
Cr4	vaginal cancer
Cr5	anus carcinoma
Cr6	penile cancer
Cr7	urinary bladder and urethra cancer
Cr8	prostate cancer
Cr9	breast cancer
Cr10	oropharyngeal cancer
Cr11	laryngeal carcinoma
Cr12	head and neck squamous cell carcinoma
Cr13	eye cancer
Cr14	nose cancer
Cr15	oesophageal carcinoma
Cr16	lung squamous cell carcinoma
Cr17	skin cancer
Cr17a	squamous cell skin carcinoma
Cr17b	basal cell skin carcinoma
Cr18	ungual squamous cell carcinoma
Cr19	verrucous carcinoma (squamous cell carcinoma, SCC)
Cr20	giant condyloma acuminatum (Buschke–Löwenstein tumor)
Cr21	Bowen's disease
Cr22	high-grade squamous intraepithelial lesion (HSIL)
Cr23	low-grade squamous intraepithelial lesion (LSIL)
Cr23a	Atypical Squamous Cells of Undetermined Significance (ASC-US)
Cr24	genital mucosal lesion

Cr1	cervix carcinoma
Cr25	mucosal benign lesion
Cr26	apocrine acrosyringal keratosis associated with syringocystoadenoma papilliferum
Cr27a	vulvar and vaginal intraepithelial neoplasia (VIN)
Cr27b	cervical intraepithelial neoplasia (CIN)
Cr28	pre-malignant skin lesion
Cr29	cutaneous benign lesion
Cr30	keratoacanthoma
Cr31	actinic keratosis (solar, senile keratosis), precursor of cutaneous SCC
Cr32	cutaneous horns (cornu cutaneum)
Cr33	verrucous acanthoma
Cr34	seborrhoeic keratosis
R1	high-risk of high-grade cervix squamous intraepithelial lesion
R2	high-risk of low-grade cervix squamous intraepithelial lesion
R3	high-risk mucosal malignant lesion
R3a	high-risk malignant lesions
R4	possibly high-risk of high-grade cervix squamous intraepithelial lesion
R5	possibly high-risk of low-grade cervix squamous intraepithelial lesion
R6	low-risk of high-grade cervix squamous intraepithelial lesion
R7	low-risk of low-grade cervix squamous intraepithelial lesion
R8	low malignant risk mucosal lesion
R9	high-risk mucosal benign lesion
R10	low-risk mucosal and cutaneous lesions
EV	epidermodysplasia verruciformis (Lewandowsky–Lutz dysplasia)
H1	Heck's disease (oral focal epithelial hyperplasia)
Pa1	respiratory papillomatosis
Pa2	laryngeal papilloma
Pa3	conjunctival papilloma
Pa4	maxillary sinus papilloma

Cr1	cervix carcinoma
Pa5	virus papilloma
In1	oral infection
In2	oropharyngeal infection associated with head and neck squamous cell carcinoma
In3	oral cavity infections associated with oropharyngeal cancer
In4	sporadic infections in mucocutaneous tissue
In5	children with diarrhea
In6	cervical infection
In7	febrile respiratory syndrome
W1	common warts (verruca vulgaris)
W2	flat warts (verruca planae)
W3	plantar warts
W4	mosaic warts
W5	anogenital warts (condyloma acuminata)
W6	butcher's warts
W7	bowenoid papulosis
W8	cystic nongenital warts (plantar epidermoid cysts)
W9	deep palmoplantar warts (myrmecia)
W10	palmoplantar pigmented warts with homogeneous intracytoplasmic inclusion bodies
No1	no indication
No2	detection on normal skin
No3	detection on normal genital area
No4	asymptomatic presence in healthy human
No5	detection on sun-exposed skin
No6	detection in oropharynx of healthy human
S1	WHIM syndrome (Warts, Hypogammaglobulinemia, Immunodeficiency, and Myelokathexis)
S2	asymptomatic presence in Merkel Cell carcinoma patient (skin tumor, Human polyomavirus 5)
S3	psoriatic lesions

Cr1	cervix carcinoma
S4	pruritic and dyskeratotic dermatoses
S5	asymptomatic presence in HIV/AIDS patients
S6	asymptomatic presence in GATA2 deficiency patient
S7	punctate palmoplantar keratoderma
S8	glans lichen sclerosus

Table 7

The Circoviridae family viruses which infect humans, and the diseases and symptoms they cause

Family: Circoviridae	
Genus: Circovirus	
Species:	identified in stool samples, gastroenteritis, diarrhea
Human associated circovirus 1	
Genus: Cyclovirus	
Species:	identified in stool samples
Human associated cyclovirus 1	
Species:	identified in stool samples
Human associated cyclovirus 2	
Species:	identified in stool samples
Human associated cyclovirus 3	
Species:	identified in patients with nonpolio acute flaccid paralysis
Human associated cyclovirus 4	
Species:	identified in stool samples
Human associated cyclovirus 5	
Species:	identified in stool samples
Human associated cyclovirus 6	
Species:	identified in stool samples
Human associated cyclovirus 7	
Species:	encephalitis (CyCV-VN)
Human associated cyclovirus 8	
Species:	identified in patients with nonpolio acute flaccid paralysis and paraplegia patients
Human associated cyclovirus 9	

Family: Circoviridae	
Species: Human associated cyclovirus 10	respiratory infection
Species: Human associated cyclovirus 11	encephalitis
Species: Human associated cyclovirus 12	encephalitis

Table 8

The Parvoviridae family viruses which infect humans, and the diseases and symptoms they cause

Family: Parvoviridae	
Subfamily: Parvovirinae	
Genus: Bocaparvovirus	
Species:	respiratory diseases (HBoV1);
Primate bocaparvovirus 1 –	enteritis (HBoV3)
human bocavirus 1 (HBoV1);	
human bocavirus 3 (HBoV3)	
Species:	enteritis
Primate bocaparvovirus 2 –	
human bocavirus 2c (HBoV2c);	
human bocavirus 4 (HBoV4)	
Genus: Dependoparvovirus	
Species:	possible tumor protective effect;
Adeno-associated dependoparvovirus A –	AAV vectors for gene therapy: cystic fibrosis; hereditary emphysema: alpha-1-antitrypsin deficiency; hemophilia; muscular dystrophy; Parkinson's disease; Alzheimer's disease; Canavan disease; spinal muscular atrophy; Batten disease; rheumatoid arthritis;
(AAV1, AAV2, AAV3, AAV4, AAV6, AAV7, AAV8, AAV9, AAV10, AAV11, AAV12, AAV13, AAVS17)	potential therapeutic vaccines for cancer (malignant melanoma) and prophylactic vaccine against HIV;
Adeno-associated dependoparvovirus B – (AAV5)	AAV2 – hepatocellular carcinoma
Genus: Erythroparvovirus	

Family: Parvoviridae	
Species:	erythema infectiosum (fifth disease); systemic lupus erythematosus; Henoch-Schönein purpura (IgA vasculitis); Wegener's granulomatosis;
Primate erythroparvovirus 1 –	papular–purpuric gloves and socks syndrome; Gianotti–Crosti syndrome; livedo reticularis; erythema; rheumatological arthritides; systemic sclerosis; myositis; pure red cell aplasia (erythroblastopenia); transient aplastic crisis; thrombocytopenia; neutropenia; idiopathic thrombocytopenic purpura; virus-associated hemophagocytic syndrome; acute leukemia and myelodysplasia; Kikuchi disease; myocarditis, pericarditis, vasculitis, acute heart failure; giant cell arteritis; polyarteritis nodosa; meningitis, encephalitis; Guillain–Barré syndrome; cerebellar ataxia; transverse myelitis; peripheral neuropathy; carpal tunnel syndrome; congenital neurological disease; hepatitis, acute liver failure; glomerulonephritis; foetal hydrops; fetal loss
human parvovirus B19 (B19V)	
Species:	infects human bone marrow mononuclear cells
Primate erythroparvovirus 2 –	
simian parvovirus B20 (SPV)	
Genus: Protoparvovirus	
Species:	gastroenteritis, diarrhea
Primate protoparvovirus 1 –	
bufavirus 1a (human), BF86 (BuV1a)	
Species:	cutaneous malignant melanoma,
Primate protoparvovirus 3 –	cutaneous T cell lymphoma (mycosis fungoides)
cutavirus (human), BR-337 (CutaV)	
Genus: Tetraparvovirus	
Species:	rash; arthralgia; anaemia; respiratory tract symptoms; encephalitis; glomerulonephritis; hepatitis; gastrointestinal symptoms; foetal hydrops
Primate tetraparvovirus 1 –	
human parvovirus 4 (PARV4)	

Table 9. The Hepadnaviridae family, Hepatitis B virus

Family: Hepadnaviridae
Genus: <i>Orthohepadnavirus</i>
Species:
Hepatitis B virus Hepatitis B

Figures

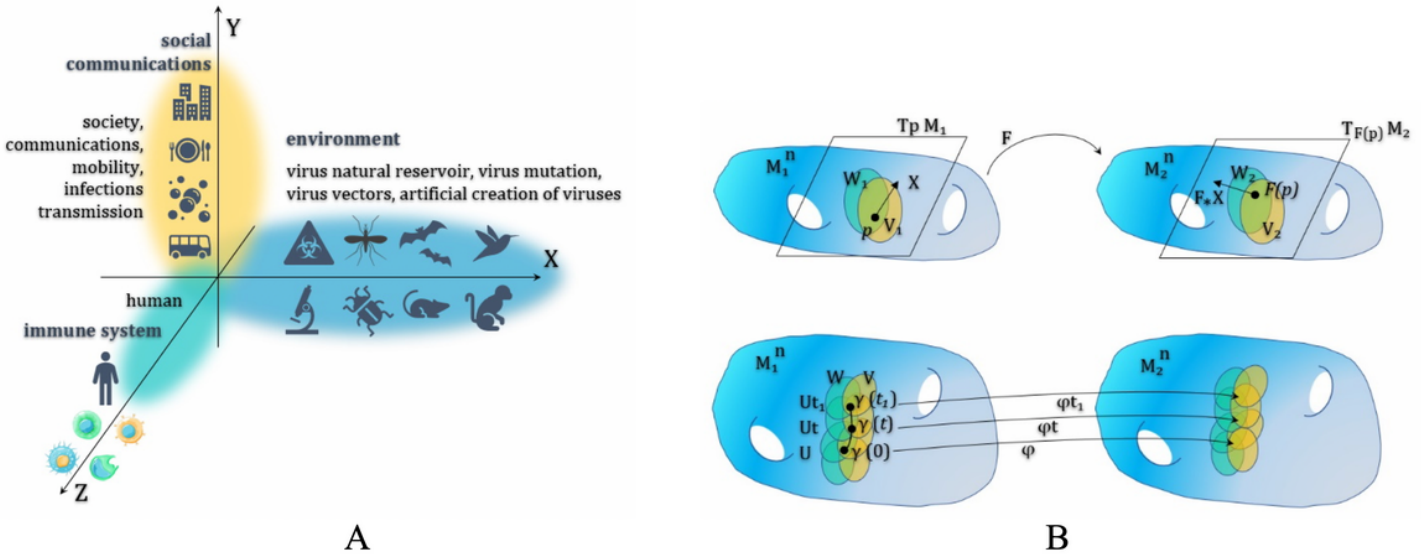


Figure 1

A – Global three-nexuses system of the whole life cycle of viral infectious diseases causative agents (viruses) which are viewed as Minkowski space (three-dimensional Euclidean space and time); x – environmental nexus; y – social nexus; z – immune nexus.

B – presentation of the three-nexuses system as smooth Riemannian manifold M with embedded submanifolds $W \oplus V \in M$, (M – environmental space; W – immune space; V – social space), homeomorphic to \mathbb{R}^3 ; p – a point on curvature; F – continuous function; X – linear map (derivation at p); F^*X – diffeomorphic operator; T_p – tangent vector bundle; pushforward associated with F – $F_*: T_p M_1 \rightarrow T_{F(p)} M_2$. A smooth manifold of constant curvature, where U – open subset, $U \cap W \oplus V$; φ – isometry defined on some connected open subset $U \subset M_1$, $\varphi: U \rightarrow M_2$; γ – curve segment.

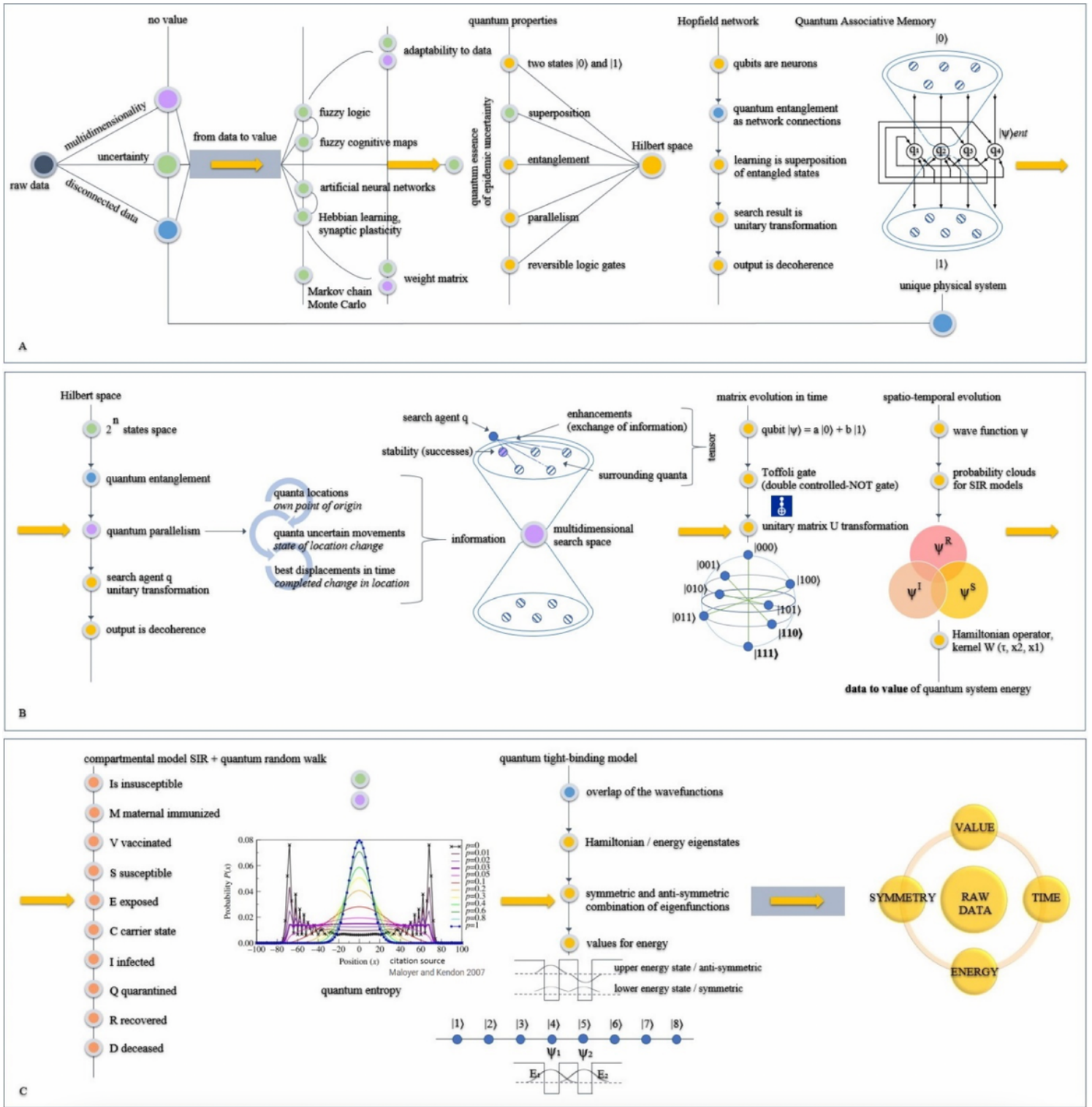


Figure 2

The continuum of consecutive selection of approaches for modeling associated with infectious diseases. See the detailed description in the text.

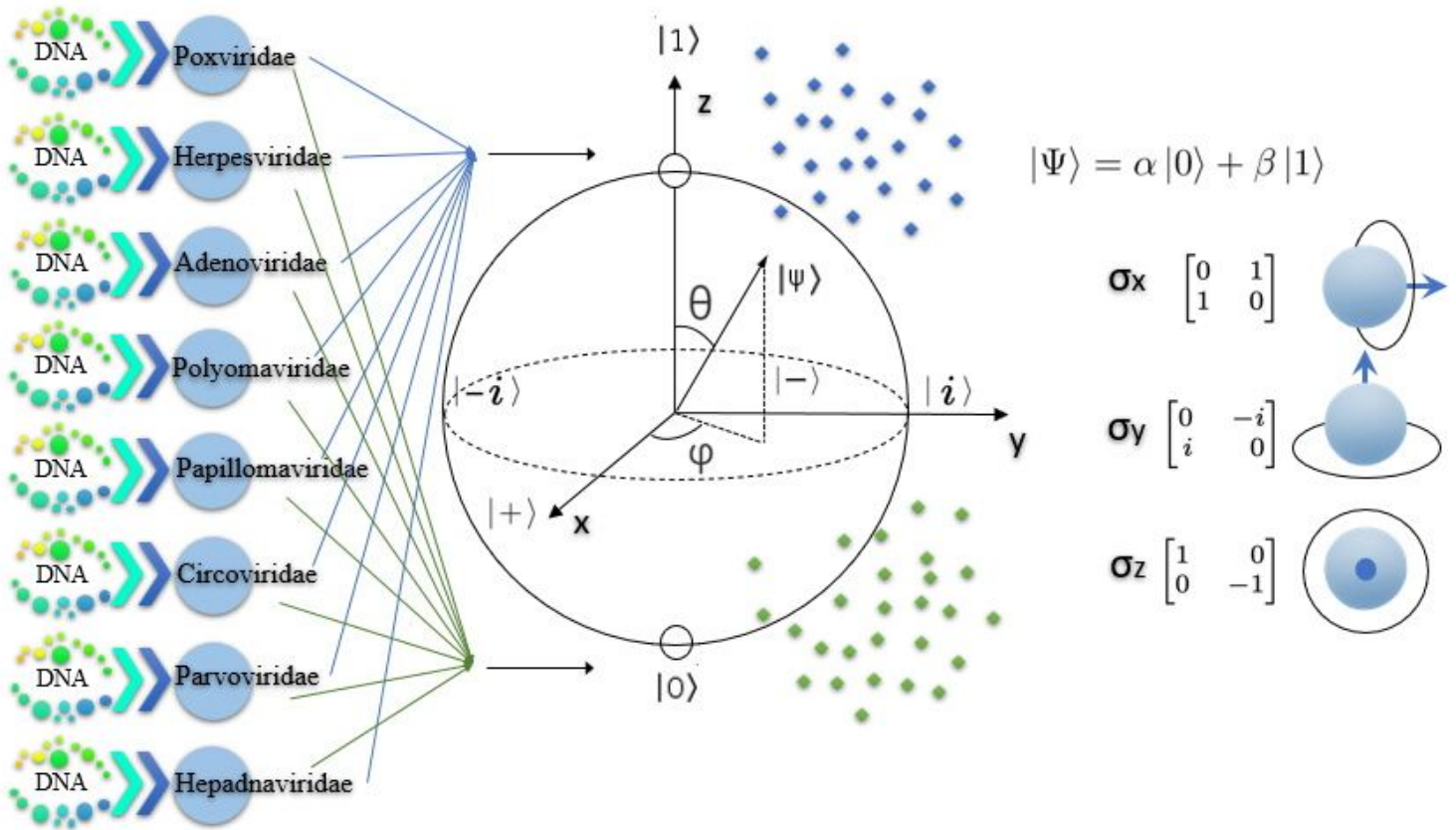


Figure 3

Presentation of Hilbert space which includes the set of all possible states of a physical quantum system $|\psi\rangle$ (state space) on the Bloch sphere; Pauli matrices (reversible one-qubit gates), rotation around the x, y, and z axes of the Bloch sphere.

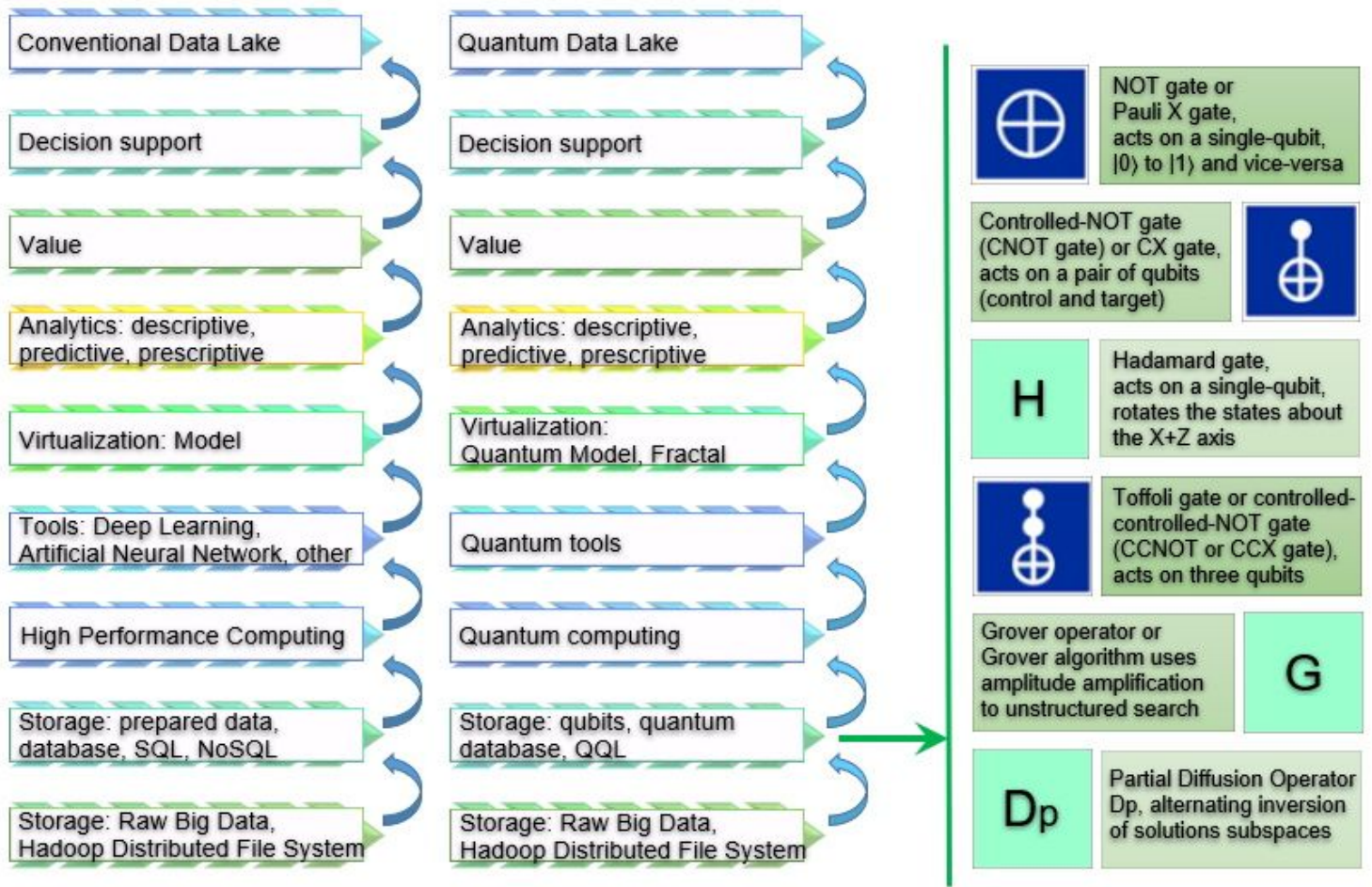


Figure 4

The Data Lake concept (left) and the proposed Quantum Data Lake concept (right) with the layer “Storage: qubits, quantum database, QQL” details.

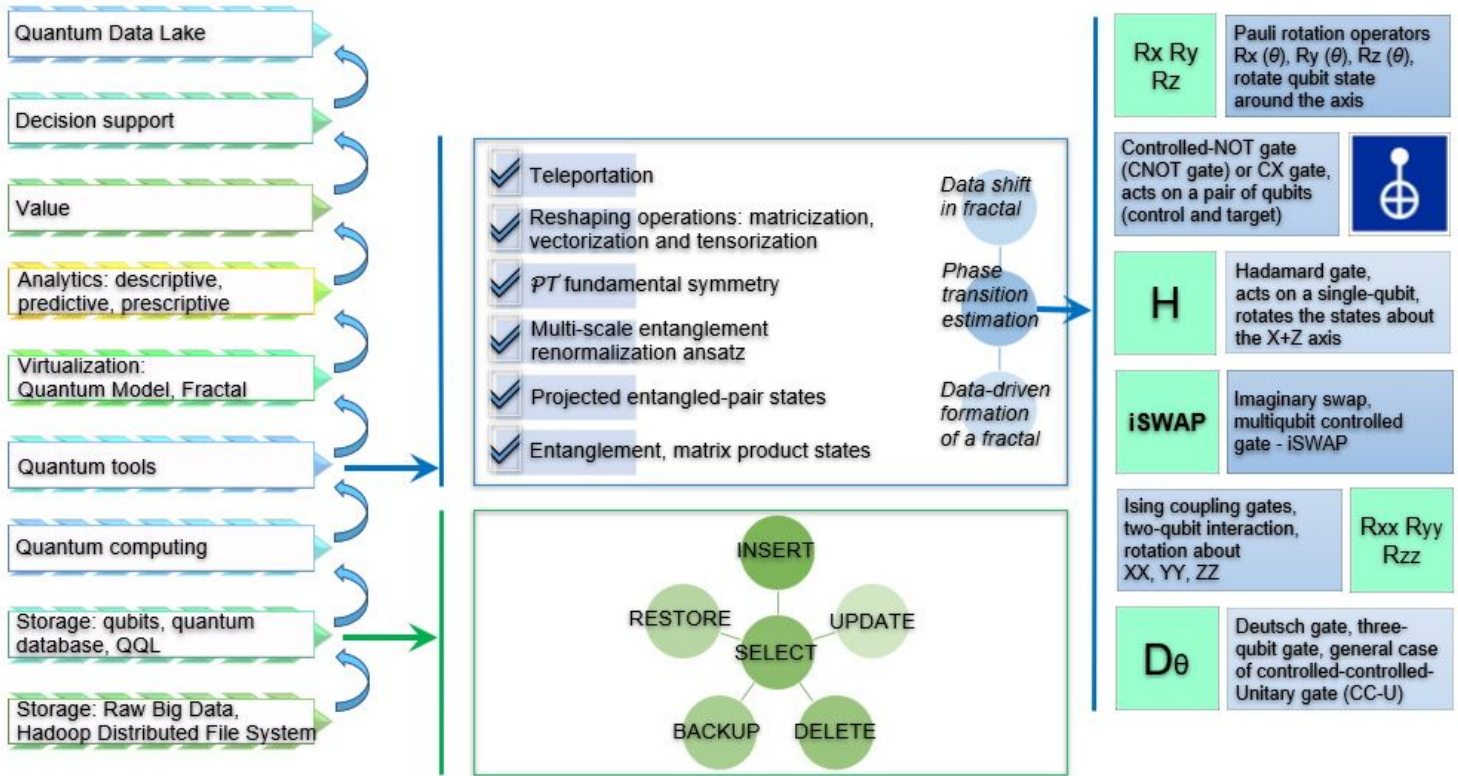


Figure 5

The proposed Quantum Data Lake concept with layer “Quantum tools” details and common queries for layer “Storage: qubits, quantum database, QQL”.

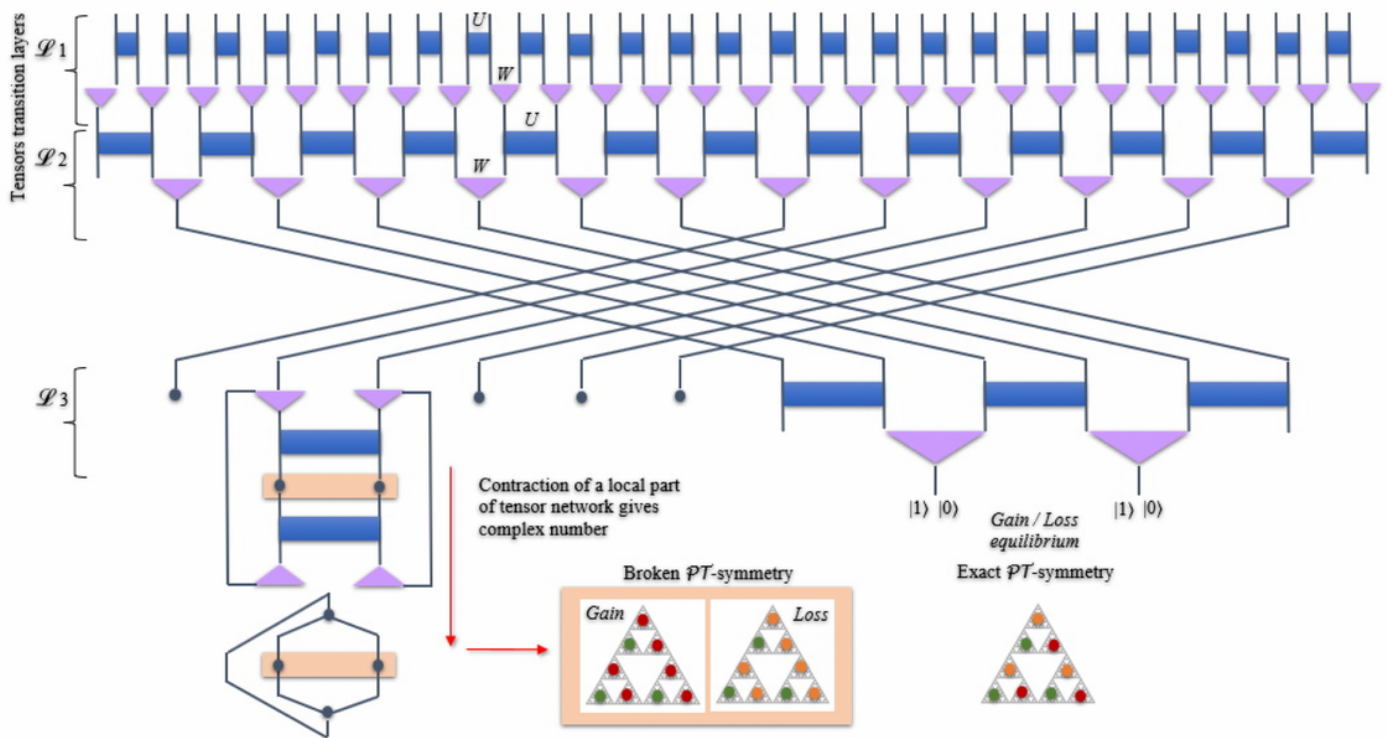
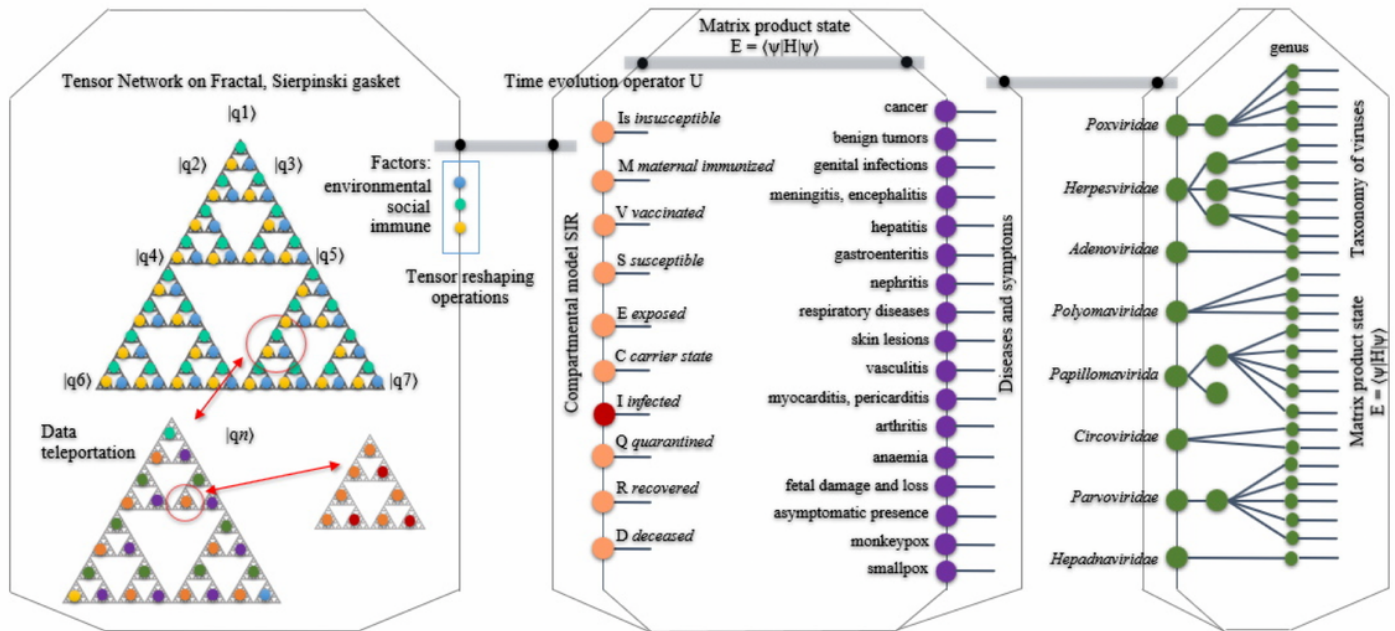


Figure 6

The proposed Quantum Data Lake concept with layer “Quantum tools” details. Data embeddings into the tensor network on fractal Sierpinski gasket (triangle); different data sets – environmental, social, and immune factors; compartmental model SIR; data about eight families of DNA viruses that infect humans; diseases and clinical symptoms caused by these viruses; data organized as matrix product states and layered \mathcal{L} tensor network MERA (unitary gates U , isometry gates W). Example of computation output as gain/loss equilibrium with unbroken, exact \mathcal{PT} -symmetry and example of contraction of a local part of the tensor network which gives a complex number and spontaneously broken \mathcal{PT} -symmetry with the related bifurcation of gain/loss prevalence or phase transition at an exceptional point. Broken \mathcal{PT} -symmetry gives

the two variants of epidemic modeling due to the appearance of the circumstances for output as a complex number while computing the incoming Data Lake information. It can point out, for example, an increase in the number of infected people or other important predictions such as an increase in cancer cases, etc.

1	2	3	4	5	6	7	8	9	10
11	12	13	14	15	16	17	18	19	20
21	22	23	24	25	26	27	28	29	30
31	32	33	34	35	36	37	38	39	40
41	42	43	44	45	46	47	48	49	50
51	52	53	54	55	56	57	58	59	60
61	62	63	64	65	66	67	68	69	70
71	72	73	74	75	76	77	78	79	80
81	82	83	84	85	86	87	88	89	90
91	92	93	94	95	96	97	98	99	100
101	102	103	104	105	106	107	108	109	110
111	112	113	114	115	116	117	118	119	120
121	122	123	124	125	126	127	128	129	130
131	132	133	134	135	136	137	138	139	140
141	142	143	144	145	146	147	148	149	150
151	152	153	154	155	156	157	158	159	160
161	162	163	164	165	166	167	168	169	170
171	172	173	174	175	176	177	178	179	180
181	182	183	184	185	186	187	188	189	190
191	192	193	194	195	196	197	198	199	200
201	202	203	204	205	206	207	208	209	210
211	212	213	214	215	216	217	218	219	220
221	222	223	224	225	226	227	AZ1_1	HANOA464	ICPX1
JEB2	mCG2	mCG3	mCH2	mDO1c02	mDO1c232	mGA2c01	mGA2c70	mDysk1	mDysk2
mDysk3	mDysk5	mDysk6	me180	mEV03c05	mEV03c09	mEV03c40	mEV03c45	mEV03c60	mEV03c104
mEV03c188	mEV03c212	mEV03c434	mEV06c12b	mEV06c107	mEV06c118	mEV07c367	mEV07c382	mEV07c390	mFD1
mFD2	mFi864	mFS1	mHIVGc36	mHIVGc70	mICB1	mKCS	mKN1	mKN2	mKN3
mL55	mLCOSOc196	mm090c09	mm090c10	mm090c66	mm090c145	mm292c10	mm292c14	mm292c88	mm292c100
mMTS1	mMTS2	mSD2	mSE355	mSE379	mSE383	mTVMBSFc09	mTVMBSGc529	mTVMBSGc2024	mTVMBSGc2450
mTVMBSHc13	mTVMBSHc33	mTVMBSWc141	mw02c24a	mw03c65	mw07c34d	mw07c68b	mw07c74b	mw11c13	mw11c24
mw11c39	mw11c51	mw15c111	mw18c07	mw18c11d	mw18c25	mw18c39	mw18c134	HPV-mw20c01a	mw20c01b
mw20c02c	mw20c03a	mw20c04	mw20c08a	mw20c09	mw20c10a	mw21c693	mw22c09	mw23c08c	mw23c77
mw23c101c	mw27c04c	mw27c39c	mw27c52c	mw27c157c	mw34c04a	mw34c11a	mw34c14a	mw34c28a	mw34c34a
mwg1c05	mwg1c09	mZJ01	RTRX1	RTRX2	RTRX3	RTRX4	RTRX5	RTRX6	RTRX7
RTRX8	RTRX9	RTRX10	Xc	Xd	Xf	Xg	Xh		

Figure 7

Visualization of the classification by genera of HPVs (numbers of viruses' types). The identification by color is following: pink – genus Alphapapillomavirus; blue – genus Betapapillomavirus; green – genus Gammapapillomavirus;

yellow – genus Mupapillomavirus; violet – genus Nupapillomavirus; dark grey – unclassified Papillomaviridae; light gray – no information; white – numbers which were withdrawn.

Supplementary Files

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