

# **Inherited Real Risk of Glioblastoma: pre-clinical diagnosis and primary prevention with Quantum Biophysical Semeiotics**

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## **Abstract**

Glioblastoma (GBM) is the most common primary malignant brain tumour and its histopathological features are necrosis, microvascular proliferation and remodeling, mitosis and cellular pleomorphism/nuclear atypia.

In addition, there is increasing evidence that mitochondrial dysfunctions appear able to induce not only genetic abnormalities but also alterations of the blood-brain-barrier's ability to interact with specific cells of the immune system (therefore impairing the immune system's response within the central nervous system).

Quantum Biophysical Semeiotics (an extension of medical semeiotics) by applying the clinical method of the Auscultatory Percussion of viscera and organs, allows the physician to detect specific signs useful for the diagnosis of Inherited Real Risk of GBM therefore offering a new diagnostic tool effective in the primary prevention of GBM (even decades before its clinical onset, or from birth).

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In this report, we discuss how Quantum Biophysical Semeiotics tests can be implemented not only for the diagnosis of Inherited Real Risk of GBM but also for monitoring how the abnormalities due to the underlying microcirculatory abnormalities can be modified following specific medical treatments aiming at completely removing the afore-mentioned Inherited Real Risk.

## **Patients and methods**

Quantum Biophysical Semeiotics tests are based on the technique of the Auscultate Percussion of the stomach and allow the physician to detect at the bedside (and even from birth) a number of signs relevant to both early diagnosis, evolution of risk, response to personalized treatment and overall prognosis.

Quantum Biophysical Semeiotics tests are performed as follows: the physician applies a digital pressure-stimulus on specific triggers points (which are related to the organ/system to be assessed) and contemporaneously detect (using the technique of the Auscultatory Percussion) the changes, over time (latency and duration of the reflex, in seconds) and space (intensity of the reflex, in cm) of the greater curvature of the stomach. Regarding the diagnosis of Inherited Real Risk of GBM the duration of the oculo-gastric reflex and the aspecific cerebrum-gastric reflex are particularly relevant and useful (normal values are between 3 and 4 seconds, showing a physiological microcirculatory reaction to tissue acidosis).

## **Results**

The senior Author (SS), between March, 2016 and July, 2017 has assessed the duration of the oculo-gastric reflex and the aspecific cerebrum-gastric reflex in 10 patients with a histopathological diagnosis of GBM (mean age: 52 years, min 40 years, max 63 years). In these patients, both tests are positive: also, during the follow-up, the numerical values (of both the oculo-gastric and the aspecific cerebrum-gastric reflex) correlate with the progression of the disease.

Interestingly, whenever it has been possible to repeat the afore-mentioned tests on the mothers of GBM patients, all of them (N= 10, mean age: 68 years, min 60 years, max 91 years) were positive to the Inherited Real Risk of GBM (duration times between 5 and 6 seconds).

The oculo-gastric reflex and the aspecific cerebrum-gastric reflex have been also assessed on a larger sample of 250 healthy volunteers (all negative for a family history of GBM – mean age: 32 years, min 8 years, max 58 years): out of this sample, 25 (10 females, 15 males, aged between 8 and 54 years-old) revealed a positive Inherited Real Risk of GBM (duration times between 5 and 6 seconds) while the remaining 225 were negative.

These 25 patient with a a positive Inherited Real Risk of GBM (in absence of clinical and family history of GBM) were followed up for about two years: in these subjects, during the follow-up, following changes in life-style (e.g., smoking cessation), diet (modified Mediterranean diet) and supplements (e.g. Carnitin, CoQ10, Bioflavonoids, Noni juice) components of Reconstructing Mitochondrial Quantum Therapy, the duration of

the oculo-gastric reflex and the aspecific cerebrum-gastric reflex progressively reduced until normal values (between 3 and 4 seconds) were reached.

### **Conclusions**

QBS tests are useful not only for monitoring the response to treatment(s) and prognosis for those patients with an already established diagnosis of GBM but also for identifying those individuals clinically healthy but with a positive Inherited Real Risk of GBM, thus allowing adequate prevention strategies to be implemented and to remove their Inherited Real Risk.

### **Introduction**

Glioblastoma is the most common and ominous of malignant primary brain cancers in the adult population (with Inherited Real Risk of Cancer) with an average age of onset between the fifth and sixth decades of life (and maximum incidence peak between 65 and 75 years).

In our paper, we discuss a case of Glioblastoma developed in a younger patient (40 years-old) with no Inherited Real Risk of Cancer: we speculate that this most unusual clinical case can be explained by a combination of factors such as heteroplasmic mitochondrial DNA mutations, oncogenic chromosomal abnormalities, some forms of genetic fusion (1), and an impaired action of the blood-brain barrier in regulating the cerebral motion of the cells involved in the response of the Immune system (2).

As already demonstrated by the senior author (SS), Semeiotic-Biophysical Semeiotics tests allow physicians to identify, in a population of asymptomatic patients, those with an Inherited Real Risk of Cancer (or Inherited Real Risk of brain diseases) which can be eliminated by Restructuring Mitochondrial Quantum Therapy (6-8): we therefore speculate that there must also be an Inherited Real Risk of Cancer specific for Glioblastoma as well.

### **QBS tests for the diagnosis of Inherited Real Risk of Glioblastoma.**

The unilateral oculo-gastric aspecific reflex (13) plays a crucial role in the QBS diagnosis of Inherited Real Risk of Glioblastoma: when this reflex is absent, we can rule out the presence of an Inherited Real Risk of Glioblastoma.

The latency time of the gastric aspecific reflex (12, 13) still remains within the normal, physiological limits (NN=8 sec) however its duration may escalate up to 5-6 sec (4 sec < NN < 3 sec).

In regard to differential diagnosis, it is worthwhile noticing that in patients affected by migraine the oculo-gastric-aspecific reflex is unilateral, the latency time is normal but the duration of the reflex is increased up to 4-4.5 sec

Oculo-gastric-specific reflex	Latency time (Lt) in seconds	Duration (D), in seconds
Healthy	Lt = 8 s	4 s < D < 3 s
Subjects with migraine	Lt = 8 s	D ≈ 4 – 4.5 s
Inherited Real Risk of GBM	Lt = 8 s	D ≈ 5 – 6 s

Table 1

The parameters of the aspecific cerebro-gastric reflex can be evaluated by applying a medium to moderate finger-pressure (500-700 dyne/cm<sup>2</sup>) on the projections of the brain circonvolutions on the skin surface of the scalp (13) followed by a stimulation of the specific trigger-point of the Inherited Real Risk of Glioblastoma.

Cerebrum-gastric-specific reflex	Latency time (Lt) in seconds	Duration (D) in seconds	Tonic Gastric Contraction
Healthy	Lt = 8 s	4 s. < D < 3 s	NO (absent)
Inherited Real Risk of GBM	Lt = 8 s	D ≈ 5 – 6 s	YES (slight)

Table 2

The impairment of the glycocalyx when assessing the specific trigger-point of the Inherited Real Risk of Glioblastoma is particularly intense and is a sign of primary importance in both diagnosis and differential diagnosis (15-23): while in healthy subjects the endogen insulin test simultaneously activates the brain, in patients with Inherited Real Risk of Glioblastoma the latency time (which is related to the local histangi impairment) is raised to up 6 sec.

Interestingly from a diagnostic point of view, is also the fact that when an Inherited Real Risk of Glioblastoma is present, the secretion of the Taileverin in the pancreatic tail is also increased (even from birth) (25-27): for this reason, a normal secretion of Taileverin rules out the presence of Inherited Real Risk of Glioblastoma.

In the exact location of the Inherited Real Risk of Glioblastoma the latency time of the Somatosensory Evoked Potential Test is considerably increased (28).

The data of Clinical Microangiology demonstrate the characteristic microcirculatory activation type II (dissociated, of mild intensity) which progressively aggravates following the onset of glioblastoma (14).

### ***The Paolo's Sign\* in QBS diagnosis of Glioblastoma (from Inherited Real Risk of Glioblastoma).***

The auscultate percussion of the pancreas was discovered by the senior author (SS) around the end of the 1950's, it was illustrated for the first time in 1978 (1) and later in numerous other works (2-7): it allows to quickly identify the skin projection of the pancreas and in particular of the pancreatic tail.

The parametric values obtained through QBS tests allow physicians to observe and evaluate the dynamics of the body and tail of the pancreas (8): the Taileverin, secreted in the pancreatic tail, discovered by the senior author (SS) at the beginning of 2017, physiologically plays a trophic action in the liver and brain (9-11).

In healthy subjects the application of an intense digital pressure (1000 dyne / cm<sup>2</sup>) over the brain's trigger-points, after exactly 8 seconds causes a microcirculation type I activation associated with the pancreas tail, the lower margin of which decreases by 2 cm for the duration of 10 sec.

When the same experimental conditions are applied to subjects with Inherited Real Risk of Glioblastoma (12), the intense stimulation of the Glioblastoma-specific trigger point is sufficient to cause a lowering of the inferior margin of the pancreatic tail of 2cm (the duration of the reflex may last between 11 and 15 seconds): however, the lowering of the inferior margin of the pancreatic tail is observed to be at least 3 cm and for a duration of exactly 20 seconds in patients with an established diagnosis of Glioblastoma.

\* In memory of Paolo, son of Sergio Stagnaro, deceased on June 11, 2017 (New York, USA) because of Glioblastoma.

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