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The International Alzheimer Association has reported that by 2030, the number of dementia patients will double, reaching circa 70 million world-wide. It adds that global expenditure on treatment has now reached 600 billion dollars — 70% from Western Europe and North America alone.

In Israel, there are now approximately 120 thousand dementia patients, 1% of the population in their 60s. This percentage is doubled every five years—reaching 30% for ages 85-90, and 2/3 of the elderly over 90.

This data is even more alarming in view of solutions offered by medicine (nowadays appearing quite ordinary—such as nutrition, cholesterol, diabetes, blood pressure issues, hygiene, etc.) which have increased life expectancy and created a new demography.

In the last two decades, top brain scientists have rallied behind research into the causes of dementia, seeking methods of treating it in its various forms, and understanding how urgent and vital it is to find solutions that may save people and extend the life expectancy of mankind.

**What then is the catalyst causing the onset of degenerative brain disease? Is there any way to avoid being another statistic?**

All degenerative brain disease patients may be roughly divided into those with a genetic mutation causing one of these diseases and whose fate is clear in advance, and those without a mutation, but for whom the disease erupts spontaneously — labelled “degenerative brain disease beginning at an elderly age.” The difference between those with and without the genetic mutation is the age at which the disease erupts, and—moreover—the psychological aspect of life under the threat of the disease as a time-bomb amongst the carriers.

**What does this actually mean?**

Let us take, for example, the Creutzfeldt-Jakob hereditary disease. A young man or woman in their 30s, at the beginning of their life, have just finished their studies and started on a career, got married, and are raising kids. In the midst of this intensive endeavor — a mother or father, perhaps an uncle, becomes ill. The patient is not particularly old, in his 50s or 60s, and is still very much active—with future plans and unattained goals. Yet the disease is progressing at an alarming rate. At first, the patient doesn’t remember small details or his speech slurs. He gets lost in a familiar neighborhood, loses balance…something is happening, something is festering. He visits his family doctor and then a neurologist, undergoing CT or MRI; his condition continues to deteriorate relentlessly. And then our loved one is no longer really with us, even if he or she still lives a bit longer. This is of course shocking and very sad. But it is just the beginning of the story.

In the midst of all this mayhem, the specialist asks — Are you from Libya or Tunis? And then there are genetic examinations. Thus our man or woman, still in their 30s, and their entire family, find out that their loved one is afflicted with a hereditary disease. There is a mutation that causes an important protein in the brain (PRP) to change its form, so that instead of breaking down when it has completed its role, it oxidizes and is stored in brain cells in clusters called amyloids.

As a result of the accumulation of clusters of faulty protein, destructive “free radicals” are created, damaging the “quality” of brain cells. Ultimately, a process of accelerated destruction of brain cells is created by this combined co-dependent process (whereby protein does not break down, but rather accumulates in clusters causing free radicals that harm the quality of brain cells).

Processes similar to those described in the Creutzfeldt-Jakob hereditary disease appear in other degenerative brain diseases, such as Alzheimer’s (the A-BETA protein), Parkinson (aSYNUCLEIN), muscle atrophy protein, etc. In each of these diseases there is a group of hereditary patients, and many more people in whom the structure of the relevant protein is damaged without the aid of mutation, for reasons that are not all clear. These are the “sporadic” or “random” patients.

**Returning to the young couple beginning their life, we see that in addition to their terrible suffering at seeing a loved one deteriorate cognitively and functionally, the entire family has to cope with the possibility that a faulty gene, a “time-bomb,” will appear in their children, cousins and grandchildren, whoever the carrier may be. The gene is “dominant,” so that one carrier parent suffices to spread it further on, to half of the children.**

In the years 1985-1988, I was in the United States doing my post-doctorate studies, at the University of California, San Francisco, at the lab of Prof. Stanley Prusiner, brain researcher and 1977 Nobel Prize winner for medicine. We all focused on a fascinating topic in biology and medicine. We wanted to prove that the faulty PRP protein in the CJD (Creutzfeldt-Jakobdisease) and other similar ones may “infect” the carrier with the disease, without the presence of the disease mutation DNA in the patient.

We were busy examining faulty protein and its mechanism of creation in the cells—the theory of prions for which Prof. Prusiner received the Nobel Prize, gaining international recognition for his work on the protein particle that causes these diseases.

When I returned to Israel in 1988 and continued my lab research on prion-caused diseases in the lab I built at the Hadassah Hospital Neurology Dpt., I did not work on theoretical research detached from the physical disease; I was part of the clinical department, exposed to patients and their families.

This vital change increased my interest and empathy for the patients and their families, and yielded results ten years after my return. In 1998, the mad-cow disease erupted, and our lab focused on cure for the incoming patients (the mad-cow disease and CJD are “relatives”, since in both diseases the PRP protein is faulty).

The search for the cure/treatment for this disease - connected to the group of dementia diseases and also affected by the accumulation of faulty protein clusters in the brain and the increased destruction of brain cells - was based on the attempt to discover antidotes or other molecules that would break down the clusters and eliminate them. In several models of various researchers (------), particularly in the widespread Alzheimer’s disease, and with the aid of heavy financial investment, the amyloid clusters were indeed eliminated from patients’ brains - and yet the disease continued to operate. We felt that we were reaching a dead-end.

**Will an antidote be discovered in the future that will prevent or stop the destruction of brain cells? Only time will tell. Did we choose the wrong research strategy for treating the problem? God only knows. Meanwhile, how can we help incoming CJD patients cope with the disease? How can we help youngsters at risk?**

Since the disease accelerates rapidly, patients generally arrive at hospital at an advanced stage of diagnosis (nearly terminal); we cannot reconstruct brain cells that are already dead. We can, however, address the issue of the young carriers of mutation, and any person who has the potential to suffer a dementia-causing disease.

In view of the dead-end reached by all researchers in treating the disease, we decided at the lab to tackle the problem from a totally different direction. If we cannot “clean” the brain cells of destructive faulty protein clusters, thereby curing the patients, perhaps we can strengthen their durability, extend the life span of brain cells, and improve their functioning even under dire conditions such as these, with all of the “biological garbage” and the destructive oxidizing free radicals.

To this end, we decided to research the influence of anti-oxidization on the brain cells, through lab mice in which we planted the Libyan mutation of the PRP protein. The research hypothesis was that if we treat them with sufficiently strong anti-oxidization that can reach the brain, this may protect the cells and compensate for the damage already incurred.

We further stipulated that the treatment should be completely safe of side effects, in order to offer it to young carriers before the disease erupts—as a preventive measure used over many years with no risk involved.

Thus, the extract of pomegranate seed oil was chosen—containing a unique component of punicic acid, one of nature’s strongest anti-oxidants. In cooperation with Prof Magdassi’s lab at the Nanotechnology Center of the Hebrew University, we managed to produce a natural supplement that can be ingested, with a unique formula. When the oil reaches the stomach, aided by additional ingredients of the formula, it dissolves into micro-drops. These tiny drops of oil can avoid breaking down in the liver—which is what happens to all oils we digest—and through the circulatory system they can then reach the central nervous system and the brain.

It was apparent in the first experiment conducted that the treatment could keep lab animals alive longer than a placebo.

Additional experiments showed that the supplement, named GRANAGARD, does indeed prevent anti-oxidization and the death of brain cells. It releases the cells of various amyloid components and its action seems to connect to an energy creation system in the cells. Biochemical testing has shown that the microscopic drops of oil do indeed reach the brain and the central nerve system, at a proven dosage and effect. There, the punicic acid of the pomegranate seed oil extract converts into CLA — a component known to protect the brain cells. Additional tests showed that the treatment is also efficient in the mouse model of multiple sclerosis, a disease in which the oxidization of lipids is also an important factor.

Given these encouraging results, we began—my research partners and myself—to use the supplement ourselves, so we could better understand its impact. It has, in the meanwhile, been approved by the Ministry of Health for use by the wider public. We were soon joined by mutation carriers among our patients’ families, and we learned from them additional points regarding the treatment—namely, that it gently activates both mentally and physically, and helps cognitively. Each report of experimenters from the “field” sends us back to the lab for further experiments, with the understanding that the strengthening of the cell and extension of its life span is possible, and may provide an intermediate solution for carriers as well as improving the lot of patients of dementia diseases, until a full cure is found for the disease.

These days we are planning several clinical experiments in various diseases, first and foremost, of course, for the carriers and patients of the cruel CJD. We hope that we may be able to save the lives of the next generation of patients.

Sincerely,

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**The various degenerative brain diseases harm 50% of the population over 80, and cause significant mental and physical suffering. A cure has yet to be found for the disease, but a new nutritional supplement may prevent it.**

**By Prof. Ruth Gabizon**

The most common dementia-causing diseases in Israel (and world-wide) is Alzheimer’s, in its various forms. This disease is associated with memory problems, cognition loss and ultimately other neurological issues. A dementia-causing disease might appear, in certain cases, in people as young as 60, but it usually occurs at an older age, with 50% of the over-80 population afflicted. There may also be an intermediate stage of light cognitive loss, from which Alzheimer’s might later—but not necessarily—develop.

**What causes dementia?**

The main reason for vascular dementia seems to be extensive areas of brain blockage, which cause cells to die from lack of oxygen, inducing symptoms of dementia.

Moreover, dementia may be caused by a host of degenerative brain diseases, in which it is not necessarily the primary neurological symptom, but only one of many. Diseases with these symptoms include Parkinson’s disease, CJD (Creutzfeldt-Jakob disease) and others, as well as advanced stages of multiple sclerosis.

**What are the known causes of these diseases?**

Not all is known about the pathological mechanisms of these diseases. However, two known factors are at fault in the spatial structure and the breaking down process of key proteins, specific to each disease. Thus, A-BETA in Alzheimer’s, aSYNUCLEIN in Parkinson’s, and PRP in the prion diseases such as CJD. Furthermore, in all of these diseases there is increased oxidization of proteins (particularly the faulty ones) and lipids, which comprise the cell membranes. These two pathological processes together cause accelerated death of nerve cells.

**How are dementia-causing diseases diagnosed primarily?**

Primary diagnosis is usually symptomatic. The patient reaches the family doctor with a neurological complaint and is referred to a neurological physician. He is subsequently referred to the appropriate diagnosis, including simulation, lab tests and neuropsychological ones, evaluating the level of dementia.

**What are the conventional treatments common nowadays for these diseases?**

While diseases such as Parkinson’s and multiple sclerosis have specific treatments, the other dementia diseases mainly employ symptomatic treatment, as well as attempting to reduce risk factors that might worsen the situation. This is done by maintaining proper blood pressure and levels of cholesterol and sugar.

Healthy nutrition together with physical and cognitive exercise contribute to maintaining of brain cells, as well as medication for proper synapses of the brain, such as Exelon and Aricept.

**Why is the development of new medicine necessary for treatment of Alzheimer’s disease?**

Over the last few years, much research has been directed toward finding treatment that may prevent the creation of amyloid clusters containing A-BETA protein, or that may break them down after they’ve been created. However, this treatment concept has not been successful. It seems that although special antidotes can break down the faulty protein clusters (as shown successfully with lab animals), there is no change for human beings—in patients’ tests—and not even a slowing down of the disease’s acceleration.

These are the circumstances faced by “real” patients, and for people with light cognitive loss. It does not mean that the entire concept has failed, but it has definitely suffered a tough blow. That’s why another solution seems necessary for the treatment of dementia diseases.

**What does the innovative treatment of “micro-drops” offer?**

The common cause of all degenerative brain diseases is pathological oxidization of components in the nerve cells—the precise point of departure of this innovative treatment. Since there is no reversal for highly faulty nerve cells (and surely not for dead ones), the treatment focuses on maintaining the existing ones, that is—maintaining our brain cells for as long as possible.

This has led to a treatment than can be offered to healthy people for many years, with no side effects whatsoever. The use of natural ingredients, adapted scientifically to treatment needs, means that the micro-drops use a natural source of oxidization as a maintenance system for brain cells.

**What is the micro-drops treatment made of? How does it operate?**

We created GRANAGARD in cooperation with Prof. Shlomo Magdassi’s group at the Nanotechnology Center at the Hebrew University. This is a nutritional supplement with ingredients in the FDA safety chart. The GRANAGARD capsules contain pomegranate seed oil, of which 90% is punicic acid (omega 5), unsaturated acid.

This is one of the strongest anti-oxidants in nature. In addition, the capsule contains emulsion ingredients approved of by the food industry. Together with the oil, when the mixture encounters water (as in the stomach), micro-drops of oil are created. These do not break down in the liver (as do regular oils) and therefore reach the blood stream. From there, they go on to the brain (according to the known transfer of unsaturated fat acids from the blood to the brain). We have tried the treatment on two models of lab animals with degenerative brain diseases and yielded impressive results which have shown a significant reduction of the disease’s appearance in the animals. We have studied the concept on these animals, and found a significant reduction of oxidization of fat acid in the cells, as well as a significant reduction of cell death.

**How is the medicine taken? Is it done on a regular basis?**

It is advisable to take it on a regular basis, two pills every morning.

**How long does it take to see results? What is the medicine’s efficiency, as known so far?**

Actually, in the case of healthy people taking the medicine, we don’t expect clear results, since it is mainly preventive. To our surprise, a considerable number of users have reported a rise in energy levels both physically and mentally. This holds true for various populations of patients.

**Should the innovative treatment be taken throughout one’s life, or is it medical treatment?**

We are currently examining the answer to this question in models of lab animals representing a degenerative brain disease. The initial results show that it is advisable to continue taking the supplement over a long period of time. We will learn more about the treatment’s operation mechanism in future experiments.