Telocyte Newsletter
Q1 2017

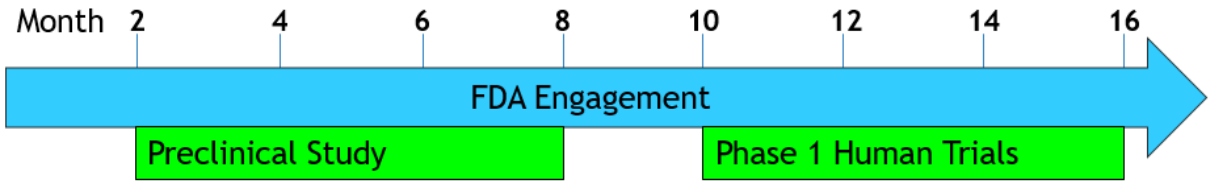
TELOCYTE
A FUTURE BEYOND ALZHEIMER'S

As we enter 2017, we find ourselves beginning new collaborations, new endeavors, new prospects, and new hopes for Alzheimer's. Telocyte has now completed our contractual arrangement for our FDA animal toxicity trial and, after working with our regulatory consultants and our collaborators, the protocol for that trial is in final form. Our financing is likewise entering a new phase, as a global investment group negotiates to provide funding for that same FDA toxicity trial.

Yet while we move ahead, the reason we do so remains unchanged: Alzheimer's disease continues to haunt all of us. The Alzheimer's Association estimates that one in three of us will be diagnosed with AD. Alzheimer's has become the leading cause of death in some nations, while the global incidence climbs in every nation as our average lifespans lengthen. Wherever we live, whatever our economy, however our health care is delivered, not only do the human costs of AD grow, but the economic costs (currently one quarter of a trillion dollars annually in the US alone) grow larger as well. At Telocyte, we believe that we can erase those costs, erase those fears, and erase the disease itself.

We hope that a year from now we can begin to take our vision from an old scientific hope to a new clinical reality, as we begin our first human trial.

What are we doing at *Telocyte*?



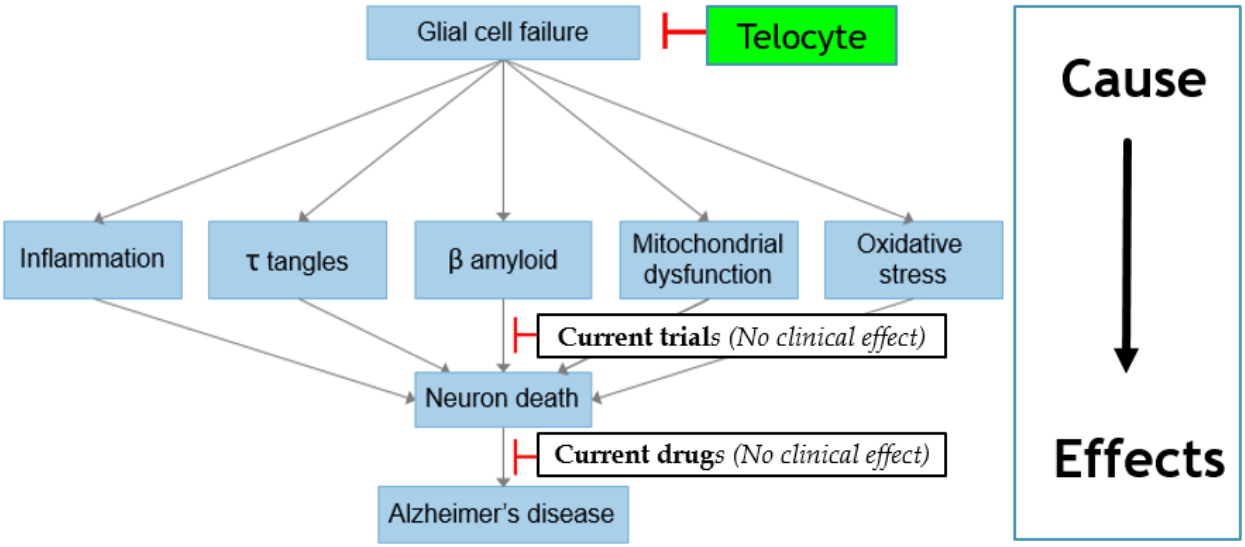
Getting to the clinics takes financing, hard work, intelligence, and careful planning. Over the next year or so, we have two goals:

1) To complete our FDA animal toxicity study (the "preclinical study") and
2) To complete our FDA Phase 1 human trial.

The first step, our preclinical study, will begin in the next few months and last a minimum of six months. The primary aim is to provide the animal data that the FDA requires, preliminary to our beginning an FDA-sanctioned human trial. This study includes both young and old mice, as well as human cell studies in the laboratory of our collaborator, CNIO, in Madrid. As that study moves ahead, we will be in discussion with the FDA, to ensure that we have the data they need to grant

us an IND (Investigational New Drug) status, so we can begin our phase 1 human trial. The second step, to begin in early 2018, will also last six months. We will treat 12 volunteers, each with confirmed moderate Alzheimer's disease. We anticipate clear cognitive improvements in these dozen human volunteers.

How does Alzheimer's happen?



Alzheimer's is not only the loss of our memory, but also the loss of independence, the loss of our ability to reason, and even – some would say – the loss of our souls. Stepping back from the human cost, however, what causes Alzheimer's in the first place?

For more than a century, we have known that the clearest change in the brains of those with AD is the death of our nerve cells. Neurons die and the results are obvious in autopsy. But going "upstream" a bit, what makes our neurons die? For several decades now, we have seen a fairly good (although not perfect) correlation between neuron death and two findings that are seen under a microscope: deposits of amyloid protein (plaques) and deposits of tau protein (tangles). Reasonably enough, researchers have tried valiantly to prevent or decrease both the plaques and the tangles. The frustrating outcome, however, is that no matter what we do to the plaques or tangles, neurons continue to die and our cognitive decline continues downhill. So far, not a single AD trial has succeeded.

Many researchers have begun to think that the reason is that there is something that lies further "upstream", something that causes not only plaques and tangles, but all of the many other, perhaps less obvious pathological findings. They reason that if you only attack amyloid or only attack tau proteins, the "upstream" problem continues causing all of the other problems. If you only attack one part of the problem, such as amyloid, then the other problems continue, as does neuronal death and cognitive decline. Put differently, no matter what you do to amyloid alone, you haven't changed the overall disease process, so the cognitive decline continues. This is exactly what the human trials show: if all you do is attack one of the many problems, the result is futility and frustration.

The result is Alzheimer's.

Over the past decade or so, however, there has been a growing consensus that AD – and all of those various "upstream" problems such as plaques and tangles – are the result of changes

in the glial cells and that aging itself (particularly cell aging) plays a key role. A growing consensus has begun to look not only at targeting the glial cells, but at targeting the way these cells age. This would be merely an interesting theory, except for two facts:

1) All of the data supports the role of the glial cells and
2) We are now capable of reversing glial cell aging in human patients.

Which is exactly what Telocyte is about to do.

Hello everyone,


Peter Rayson
Founder and CEO of Telocyte

I would first like to thank all Telocyte Associates who have supported and helped Michael and I throughout 2016. Your dedication and commitment has been an inspiration, as we pursue our goal of developing telomerase therapy to treat Alzheimer's Disease.

If you have read Michael's latest book, *The Telomerase Revolution*, you will have seen his description of the Parabolic Cliff. That parabola describes the trajectory of our health as we age, the initially flat curve tightens ever quicker as we progress in time and finally pass over the event horizon of this reality.

My core driving inspiration for Telocyte is Iris, my mother who sadly was diagnosed initially with vascular dementia and subsequently also Alzheimer's Disease in 2011. My family and I had the privilege of walking with her along her Parabolic Cliff as she bravely coped and battled with her dementia.

In those early years she asked me to look for a treatment or cure, her career as a nurse had given her a deep faith in science and medicine that sat well along aside her Christian beliefs. On the 23rd November, 2016 she sadly slipped over the edge of her cliff.

She had been delighted to know that Michael and I had teamed up and founded Telocyte. Global awareness of the disease called dementia has escalated rapidly during the 5 years of her illness. This is transforming the understanding of the socio-economic impact of dementia on patients and their families and friends.

There are now more Dementia care and support organizations and charities becoming established, Also funds being raised and committed by governments for research into the causes and finding treatments. The problem for Iris and others like her is that all this well-meaning effort and focus assumes that treatments and cures are 20 years in the future. So we have a generation of people who created the 20th century for us now sliding over a precipice with no hope.

Humanity has been here before, facing plague, smallpox, polio and most recently Ebola. Our success against these diseases has derived from deep insights into their pathology and biology. It is the same for dementia and other diseases of age. The

discovery of Telomerase and the subsequent laboratory research in the last century has lain dormant. Much as has happened in previous centuries when novel phenomena have been identified but not applied or have not been rediscovered until times of crisis change the dynamic and prevailing social agenda.

Telocyte has been founded to rapidly bring Telomerase Therapy to first clinical human trials for Alzheimer's Disease, working closely with our world class science collaborator CNIO and in compliance with FDA regulations.

This year we have made great progress with Amarex, our regulatory consultants, in detailing the program of work required by the FDA. We have also gained the support of key investors to take us through the required first phase of our program.

Telocyte is rapidly accelerating, our strategic objectives are clear:

- We will measure our success by curing patients.
- We will provide proven clinical interventions.
- We will abolish age related diseases

Michael and I are contrarians, swimming against a consensus view, but we have good reasons, good data, and a clear view of the real point of intervention in the cascade of pathology that is age-related disease.

Our focus is on the generation now suffering with Alzheimer's Disease, not 20 years away. We are looking forward to an exciting 2107 for us and for them!

Peter