Artificial Death for Attaining System Longevity

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Self-monitoring systems have the desired property of surviving damages. Many biological systems are self-monitoring to some extent, and can hint at possible ways of attaining this property. Inspired by biology, we propose a self-developing and self-monitoring system in which agents present the possibility of mutating their genes. The agents can repair genes to some extent but if they acquired un-repaired mutations and are no longer aiding the environment they are considered *aberrant* and should therefore die. This gives rise to an artificial creature made of multiple agents in which local death prevents the unhealthy agents from destroying the system. We call this system HADES (Healing and Agent Death Encouraging Stability).

The main application of the study is in distributed systems such as sensor networks, where agents communicate to accomplish the system's goal(s). If an agent becomes injured it should first try to self-repair [1]. If these injuries are not repairable, they may escalate so that it is beneficial that the agent kills itself. This improves on the robustness of previous systems.

HADES may shed light also on tumor formation and suppression. Current understanding of how cancer evolves requires a multi-stage model [3]. Our system also requires multiple steps of mutation in a specific order for a tumor to develop, otherwise the individual agent will either correct its own problems or kill itself to preserve the system's health. Using our system we will address the question of how a system that was in a healthy steady state can lose agents and become weak. We will compare protocols of possible apoptosis, and propose "dynamical-treatments." We will check the new idea of neighboring cells inducing awareness and death.

The System

We assume five gene types inspired by biology. Oncogenes control cell splitting and determine if an agent will replicate. The tumor suppressor genes ensure that the oncogenes do not cause excessive splitting. The repair genes attempt to repair any damage that has occurred to other genes. There are also apoptosis genes that enable death unless they are damaged. Blood supply genes ensure that the agents do not develop more than their nutrition supply allows.

Fig. 1: Genes in the order of their necessary mutations

HADES begins replication from a stem agent. The stem agent and blast agents replicate with a high probability whenever they detect a lack of agents in the system. A mature agent replicates with some fixed probability if there is room around it for the daughter to exist. Boundaries stop healthy cells from further replication.

Death of an agent is inspired by cell apoptosis. Apoptosis of an agent mainly occurs whenever an agent detects through self-monitoring that its genes are damaged and can not be repaired. However, if the genes controlling death are damaged the agent may not realize it should die and will continue to replicate, spreading its damaged genes to its daughters. Eventually the surrounding healthy agents will be pushed out of the way by this cluster of troubled agents. By a new mechanism we introduce, the healthy agents that were pushed aside will start sending local signals

toward the pushing agents to make them aware that they are not functioning properly. Any agent that receives the signal will interpret it as a "kill" command, although it is the agent's own prerogative to decide to kill itself. An agent is convinced to die by sensing the strength of the "kill" signal in its area, implying that multiple agents are sending it.

Results

We differentiated four cases of damage and response. The first case was the benchmark, with mutations occurring in random order. The second case had no functioning repair, the third had no functioning apoptosis, and the last had ordered mutations set to be the worst case so that they are the best to create a tumor. In the worse case, the first mutation is at the repair gene, and the second one is the apoptosis gene; this way we stop the agent's ability to repair and to die. The third mutation must be the tumor suppressor gene, so it would not keep the replication genes from expressing themselves. The fourth gene is then the replication gene, with the blood supply gene being the last one. If these orders are not followed, then a tumor is not formed.

The number of agents in the system during equilibrium in all cases was around 2250 until 25000 iterations. All cases were run for 1705810 iterations. Splitting occurred with probability 0.0025 and natural death not due to apoptosis was set for 0.0024, based on breast cancer literature [2]. The probability for mutation at each split was 0.00099 for each type of gene, based on having 10^6 base pairs per each of 5 genes that we modeled, and 10^9 other base pairs.

We first recorded efforts of cells to die. If the worst case ordering is forced, agents try 1525 times to die. However, if repair never works the agents have 517 attempts to die. If apoptosis never works the agents try 1134676 times to die, but of course every attempt fails. If the mutations are randomly ordered then only 375 attempts to die are made. Each attempt to die was fixed to have a 50% chance of succeeding. Therefore, a system that begins healthy does not need as much death as a system that is unhealthy. Death is an important factor in sustaining health, as it increases with the number of aberrant agents.

Death, however, can have a negative side effect on the health of the system over time. While the equilibrium size was 2250 for long time, around the 25000^{th} iteration the number of cells in the organisms went down in steady state to about 1000. This is probably since the values for the splitting probabilities and the natural death probabilities were chosen to be .0025 and .0024 respectively. When apoptosis is allowed this splitting probability can not match the death rate, so the size decreases. This gives an interesting view of some body areas that have fewer cells later in life. It is interesting to see how the required ratio between these two probabilities must be interpreted to enable stable equilibrium for different life spans.

A tumor can still be created, though with little probability, when the mutations occur in the particular worst case order. After one aberrant cell forms, a tumor will grow exponentially fast since aberrant cells have mutation in their splitting gene which makes them try to split frequently, and since tumor cells are blind to the distance maintained between healthy cells.

We are currently incorporating an additional mechanism to attain longevity where death signals are sent by cells which are being pushed by others. The assumption is that only healthy cells keep respectable distance. Preliminary results demonstrate the strength of this mechanism.

References

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