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Towards Self-Healing Swarm Robotic Systems Inspired by Granuloma Formation

Amelia Ritahani Ismail
Department of Computer Science
University of York
Heslington, York, YO10 5DD, United Kingdom
Email: ritahani@cs.york.ac.uk

Jon Timmis
Department of Computer Science and
Department of Electronics
University of York
Heslington, York, YO10 5DD, United Kingdom
Email: jtimmis@cs.york.ac.uk

***Abstract*—Granuloma is a medical term for a ball-like collection of immune cells that attempts to remove foreign substances from a host organism. This response is a special type of inflammatory reaction common to a wide variety of diseases. Granulomas are an organised collection of macrophages, whose formation involves the stimulation of macrophages as well as T-Cells. Fault tolerance in swarm robotic systems is essential to the continued operation of swarm robotic systems. Under certain conditions, a failing robot can have a detrimental effect on the overall swarm behaviour, causing stagnation in the swarm and affecting its ability to undertake its task. Our study is concerned specifically with modelling the trafficking of macrophages and T-cells in the development of granuloma formation, and using that as a basis to create a self-healing swarm robotic system, in the context of power system failure.**

I. Introduction

Robustness, self-organisation and adaptation are some of the key properties that have been a source of inspiration for research in swarm robotics. Robustness is a fundamental characteristic of biological systems. As reported in [5], numerous articles have been published on how robustness is involved in various biological processes and on mechanisms that give rise to robustness in living systems [2], [7]. In addition to robustness, other biological properties that have inspired research in swarm robotics are self-organisation and adaptation. Self-organisation is widespread in biological systems, including cells, organisms, and groups that possesses a large number of subunits. These subunits lack either the communicational abilities or the computational abilities, or both, that are needed to implement centralised control [3]. The main contribution of this paper is to demonstrate how modelling the development of granuloma formation can provide useful insight into the understanding of their properties leading to the instantiation of these properties to swarm robots aggregation tasks.

II. Granuloma Formation

Adams [1] defined granuloma as :

> *'a compact (organised) collection of mature mononuclear phagocytes (macrophages and/or epitheliod cells) which may or may not accompanied by accessory features such as necrosis of the infiltration of other inflammatory leukocytes'*

From this definition, a simplified version of this terminology is also provided by [1] as an organised collection of macrophages. [1] further explained that granulomas evolve conceptually in three stages. First, is the development of an infiltrate of young mononuclear phagocytes, followed by the maturation and aggregation of these cells (phagocytes) into a mature granuloma, and finally the further maturation of mature granuloma into epithelioid granuloma.

Sneller identifies some of the cells involved in granuloma formation [6]. The cells' interactions start when they are exposed to an antigen. Within seconds, or minutes, of the exposure to the antigen, pre-stored tumor necrosis factor (TNF) released by mast cells recruits neutrophils, which in turn signal to, and circulate, monocytes: this leads to the granulomatous inflammation. Interferon-gamma produced by local natural killer (NK) and T cells further activates resident tissues' histiocytes and dendritic cells. These, in turn, release a large amount of chemokines and TNF that alter the local microcirculatory environment and facilitate cellular trafficking into tissues. Following the accumulation and activation of macrophages, the inflammatory lesion (region that has suffered damage) begins to take on a granulomatous form. With the arrival of antigen-specific T cells, the lesion transforms into a mature granuloma where activation of macrophages by interferon-gamma and tumor necrosis factor (TNF) results in inhibition (slowing or prevention) of microbial growth. Eventually, the granuloma becomes encapsulated by a fibrotic rim and the center becomes necrotic (death). These tissue reactions function to protect the host by promoting microbial containment (under control) and reducing the nutrient supply to the pathogen [6].

III. The Simplified Agent-Based Model of the Development of Granuloma Formation

Agent-based modelling is a useful simulation technique that is used in many different fields, from social sciences to computer science. The effectiveness of applying Agent-Based Modelling (ABM) to immunology have been discussed in [4]. ABM is highly applicable in modelling granuloma formation, given that we wish to model populations of different cells circulating through the environment. We have chosen to

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implement the simplified model of granuloma formation using NetLogo [9].

Our simplified model of granuloma formation contains the following agents: uninfected macrophages, infected macrophages, T cells and cytokines. Cytokine agents act as a proxy for cytokines expressed by infected macrophages and T cells. They direct T cells and uninfected macrophages towards the site of infection. The infections themselves are not physically represented except by the presence of an infected macrophage in the model. We use the cytokines gradients to direct the trafficking of the cells. To reduce complexity in the model, we do not include many aspects of the adaptive immune response, as the scope of this model is to understand cell trafficking and the interaction between cells during the development of granuloma formation. Suitable assumptions are therefore employed. Agents move on a 2D grid in discrete time-steps. Movement of any agent is probabilistically determined by levels of cytokines at a location. Using the model, we are able to capture the interactions between cells in the development of granuloma formation. We begin by introducing an infected macrophage to the model. The infected macrophage will secrete cytokines and attract uninfected macrophages. These will form a "wall" that, in effect, isolates the infected cell from the uninfected cells. In the meantime, the infected macrophage will begin to infect other uninfected macrophages and spread the infection. Using our model, we observe the trafficking of T cells to the site of infection leading to the communication between T cells and infected macrophages.

Using this simplified model, we are able to extract abstract behaviour of cells during the development of granuloma formation such as the the trafficking of uninfected macrophages and T cells to the site of infection leading to the formation of granuloma. Using a principled approach, [8] we use of the simplified model to guide the development of an algorithm in the context of swarm aggregation.

IV. Swarm Aggregation and Self-Healing

As discussed in [10], robots in swarms make use of local communication for collectively navigating across an arena. If there are one or two failing robots, there is a little impact on the rest of the swarm . However, the failure of many robots will have a serious impact on the rest of the swarm, and in certain cases act as an "anchor point" for the swarm, and potentially lead to swarm stagnation. In the context of specific types of failure, we take inspiration from granuloma formation to physically contain faulty robots and initiate a self-repairing function, which is of course subject to certain abilities of the robotic unit. Employing this idea allows us to perform two related tasks. First, it allows us to isolate faulty robots. The faulty robots will then emit certain visual signals so that other functional robots in the swarm can recognise and then be attracted towards them. This is similar with the case of T cells been attracted by cytokines emitted by the infected macrophages as outlined in the previous section. The few functional robots that isolate the faulty robots to minimise the impact on the remaining swarm, similar to T cells surrounding infected macrophages, as outlined above. Such isolating of a robot, will allow for potential repair of the robot, and reduce the potential for swarm stagnation. For example, in the case of a major power drain on a robotic unit, such a drain could be identified, the faulty robot would then stop moving, signal other robots to initiate an "artificial granuloma", in which charged units might power share in order to re-charge the faulty unit, and once repairs are complete, allow the unit to continue operation, thus minimising impact on the whole swarm.

V. Conclusion

Using a simplified model of granuloma formation, we are able to abstract the behaviour of cells during the development of a granuloma, specifically, the trafficking of uninfected macrophages and T cells to the site of infection. This model has been used to derive a distributed algorithm that mimics the properties of granuloma formation and aggregate robots to initiate self-repair in the context of a swarm robotic system.

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