

A particle P of mass 0.5 kg is moving under the action of a single force $(3\mathbf{i} - 2\mathbf{j})$ N.

(a) Show that the magnitude of the acceleration of P is $2\sqrt{13}$ m s $^{-2}$.

(4)

At time $t = 0$, the velocity of P is $(\mathbf{i} + 3\mathbf{j})$ m s $^{-1}$.

(b) Find the velocity of P at time $t = 2$ seconds.

(3)

Another particle Q moves with constant velocity $\mathbf{v} = (2\mathbf{i} - \mathbf{j})$ m s $^{-1}$.

(c) Find the distance moved by Q in 2 seconds.

(2)

(d) Show that at time $t = 3.5$ seconds both particles are moving in the same direction.

(3)

M1 June 2014 Mark Scheme

**Charles Dudley Mills, Laurel L
Lenz, Klaus Ley**

M1 June 2014 Mark Scheme:

M1/M2 Macrophages: The Arginine Fork in the Road to Health and Disease Charles Dudley Mills, Laurel L Lenz, Klaus Ley, 2015-03-23 Macrophages have unique and diverse functions necessary for survival. In humans and other species, they are the most abundant leukocytes in tissues. The innate functions of macrophages that are best known are their unusual ability to either kill or repair. Since killing is a destructive process and repair is a constructive process, it was stupefying how one cell could exhibit these 2 polar opposite functions. However, in the late 1980s, it was shown that macrophages have a unique ability to enzymatically metabolize Arginine to Nitric Oxide (NO), a gaseous non-specific killer molecule, or to Ornithine, a precursor of polyamines and collagen for repair. The dual Arginine metabolic capacity of macrophages provided a functional explanation for their ability to kill or repair. Macrophages predominantly producing NO are called M1, and those producing Ornithine are called M2. M1 and M2 dominant responses occur in lower vertebrates and in T cell-deficient vertebrates, being directly driven by Damage and Pathogen-Associated Molecular Patterns (DAMP and PAMP). Thus, M1 and M2 are innate responses that protect the host without Adaptive Immunity. In turn, M1/M2 is supplanting previous models in which T cells were necessary to activate or alternatively activate macrophages. The Th1/Th2 paradigm M1 and M2 macrophages were named such because of the additional key findings that these macrophages stimulate Th1 and Th2-like responses, respectively. So, in addition to their unique ability to kill or repair, macrophages also govern Adaptive Immunity. All of the foregoing would be less important if M1 or M2 dominant responses were not observed in disease. But they are. The best example to date is the predominance of M2 macrophages in human tumors, where they act like wound repair macrophages and actively promote growth. More generally, humans have become M2 dominant because sanitation, antibiotics, and vaccines have lessened M1 responses. And M2 dominance seems the cause of ever-increasing allergies in developed countries. Obesity represents a new and different circumstance. Surfeit energy, e.g., lipoproteins, causes monocytes to become M1 dominant in the vessel walls, causing plaques. Because M1 or M2 dominant responses are clearly causative in many modern diseases, there is great potential in developing the means to selectively stimulate or inhibit either M1 or M2 responses to kill or repair, or to stimulate Th1 or Th2 responses, depending on the circumstance. The contributions here are meant to describe diseases of M1 or M2 dominance and promising new methodologies to modulate the fungible metabolic machinery of macrophages for better health.

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Table of Contents M1 June 2014 Mark Scheme

1. Understanding the eBook M1 June 2014 Mark Scheme
 - The Rise of Digital Reading M1 June 2014 Mark Scheme
 - Advantages of eBooks Over Traditional Books
2. Identifying M1 June 2014 Mark Scheme
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an M1 June 2014 Mark Scheme
 - User-Friendly Interface
4. Exploring eBook Recommendations from M1 June 2014 Mark Scheme
 - Personalized Recommendations

- M1 June 2014 Mark Scheme User Reviews and Ratings
- M1 June 2014 Mark Scheme and Bestseller Lists

5. Accessing M1 June 2014 Mark Scheme Free and Paid eBooks

- M1 June 2014 Mark Scheme Public Domain eBooks
- M1 June 2014 Mark Scheme eBook Subscription Services
- M1 June 2014 Mark Scheme Budget-Friendly Options

6. Navigating M1 June 2014 Mark Scheme eBook Formats

- ePUB, PDF, MOBI, and More
- M1 June 2014 Mark Scheme Compatibility with Devices
- M1 June 2014 Mark Scheme Enhanced eBook Features

7. Enhancing Your Reading Experience

- Adjustable Fonts and Text Sizes of M1 June 2014 Mark Scheme
- Highlighting and Note-Taking M1 June 2014 Mark Scheme
- Interactive Elements M1 June 2014 Mark Scheme

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- Joining Online Reading Communities
- Participating in Virtual Book Clubs
- Following Authors and Publishers M1 June 2014 Mark Scheme

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- Benefits of a Digital Library
- Creating a Diverse Reading Collection M1 June 2014 Mark Scheme

10. Overcoming Reading Challenges

- Dealing with Digital Eye Strain
- Minimizing Distractions
- Managing Screen Time

11. Cultivating a Reading Routine M1 June 2014 Mark Scheme

- Setting Reading Goals M1 June 2014 Mark Scheme
- Carving Out Dedicated Reading Time

12. Sourcing Reliable Information of M1 June 2014 Mark Scheme

- Fact-Checking eBook Content of M1 June 2014 Mark Scheme

- Distinguishing Credible Sources
- 13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
- 14. Embracing eBook Trends
 - Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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