Introductory remarks

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Models based on the interaction between diffusion (driven by concentration gradients) and reaction (at rates dependent on local concentrations) arise in a wide variety of applications in both biology and chemistry. Such models are also of considerable theoretical interest in the general area of nonlinear partial differential equations. Fisher [1] and Kolmogorov et al. [2] were the first to demonstrate that reaction–diffusion systems can sustain propagating structures of a permanent form—travelling waves or reaction fronts. These reaction fronts are usually based on some autocatalytic or positive feedback mechanism and are fundamental to many of the applications as they allow reactants to spread at relatively high concentrations and at rates usually much greater than would be the case through diffusion alone. Reaction–diffusion travelling waves have been much studied theoretically, see [3–5] for example, as well as observed experimentally. The differential diffusion of the reacting species can, in a reaction–diffusion system also, lead to the formation of patterns (steady, spatially non-uniform structures). The theoretical possibility of this occurring was demonstrated originally by Turing [6], but it took about another 30 years before patterns were seen experimentally.

The aim of this special issue is to illustrate a few of the many different areas where reaction–diffusion systems can arise, as well as to indicate some aspects of how reaction–diffusion systems may be treated theoretically. Enzymecatalysed reactions are an important feature of many biological processes and can form the basis for extended models of cell glycolysis. The paper by Boswell and Davidson examines a model, though relatively simple in character in the biological context, that is fundamental to enzyme reactions. They allow for the input and diffusive spread of the substrate in their model. These new and additional features enable their basic model to be embedded within much larger networks of coupled enzyme reactions, thus making it much more relevant to understanding the nature of the enzyme reactions that arise in vivo. An important feature of their modified model is that it allows the formation of product and enzyme-substrate complex to be sustained indefinitely, which is an important requirement for in vivo modelling.

A drawback to purely reaction–diffusion models is that they are based on Fickian diffusion, which means they can have the physically unrealistic property that concentrations diffuse to very large distances in infinitesimally small times. One way to overcome this problem is to include a small additional term into the model changing the system from parabolic to hyperbolic. This is the situation discussed in the paper by Leach and Needham, in which they also use the logistic kinetics proposed originally in [1,2]. One of their main conclusions, after a thorough and detailed analysis of their model, is that the reaction waves develop sharp fronts, thus allowing the concentrations

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to spread from the initiation site at a finite rate. The production of reactant through some autocatalytic process is essential for the formation of reaction-diffusion waves. However, this can be offset by the chemical decay of the autocatalyst, which can limit the parameter ranges over which travelling waves exist. This aspect is treated in the paper by Merkin which exploits the asymptotics available for large powers of the autocatalyst concentration. The main conclusion to emerge from this paper is that, if the decay step is relatively weak, then there is no limit on the range of existence for reaction fronts. For stronger decay steps, there is a limit on the range of existence with there then being two solution branches.

The interaction between diffusion and reaction of a solute and the flow of the solvent can have significant effects on both the flow and the concentration distribution. For example, this interaction can lead to changes in the local fluid density setting up convective flows, or can change the surface tension locally, thus affecting any surface wave propagation—the Marangoni effect. The paper by Pereira et al. examines how an excitable reaction, based on Fitz-Hugh–Nagumo kinetics, modifies the waves on the surface of a thin horizontal fluid layer through changing the surface tension. They show that an otherwise stable free surface can be made unstable by the Marangoni effect, resulting in cellular patterns, and that stable reaction–diffusion travelling waves lead in the flow to stable propagating pulses.

This theme is continued in the paper by Rongy and De Wit in which they consider the effects that changes in the surface tension, resulting from the concentration changes across a reaction–diffusion front, can have on that front and on the flow in a thin fluid layer. This work builds on their previous study [7] by examining how the depth of the fluid layer influences the complex dynamics arising from the interaction of the Marangoni effects with the basic reaction–diffusion mechanism. They find that the reaction front becomes more deformed, takes longer to reach a steady state and more vigorous flows are set up as the depth of the fluid layer increases.

An important requirement of reaction–diffusion waves is that they are temporally stable. Although reaction fronts can lose stability through flow-driven instabilities, as mentioned above, they can be intrinsically unstable through diffusion-driven instabilities. This can result in a longitudinal instability but, perhaps more significantly, they can become transversely unstable if the diffusion coefficient of the autocatalyst is sufficiently less than that of the substrate. This latter aspect is examined in the paper by Virányi et al. Their reaction–diffusion model includes the transport of electrically charged ions within an applied electric field and considers several reaction mechanisms. A linear stability analysis of the planar reaction–diffusion fronts that can arise in their model shows that the electric field can destabilize an otherwise stable front. The electric field strength required for this as well as the strength of the instability is found to depend on the particular reaction mechanism.

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