Taxis Equations from the Diffusion Limit of Transport Equations

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There are two major approaches used to describe the motion of biological organisms: (i) a space-jump process in which individuals move by random jumps in space, and (ii) a velocity-jump process in which discontinuous changes in the speed or direction of an individual are generated by a stochastic process. The former leads to a renewal equation in which the kernel governs the waiting time between jumps and the redistribution after a jump, and these determine the type of partial differential equation that describes the asymptotic behavior of the evolution. In this talk we discuss velocity-jump processes, and in particular, the diffusion approximation to the transport equation

$$\frac{\partial}{\partial t}p(x,v,t) + v \cdot \nabla p(x,v,t) = -\lambda p(x,v,t) + \lambda \int_V T(v,v')p(x,v',t)dv' \qquad (1)$$

describing such a process. Here $p(x,v,t)$ denotes the density of particles at spatial position $x \in \Omega \subset \mathbb{R}^n$, moving with velocity $v \in V \subset \mathbb{R}^n$ at time $t \geq 0$. Here λ is the (constant) turning rate and $1/\lambda$ is a measure of the mean run length between velocity jumps. In general λ may be space dependent and depend on internal and external variables as well. The turning kernel $T(v,v')$ gives the probability of a velocity jump from v' to v if a jump occurs, and implicit in the above formulation is the assumption that the choice of a new velocity is independent of the run length. The turning kernel may also be space dependent. When applied to the bacterium *E. coli*, the kernel T includes a bias, and the turning frequency must depend on the extracellular signal, as transduced through the signal transduction and motor control system. When applied to the amoeboid cell *Dictyostelium discoideum*, which uses both run length control and taxis, both the turning kernel and the turning rate must depend indirectly on the extracellular distribution of the signaling substance. The backward equation that corresponds to (1) has been derived from the underlying stochastic velocity-jump process by Stroock to describe the motion of bacteria, and in a more general framework by Papanicolaou. In this talk we discuss the general assumptions on the turning kernel T which ensure that the turning operator defined by (1) is positive in an appropriate sense, and the positivity in turn guarantees that a diffusion limit of the jump process exists. We introduce the parabolic scaling and formally derive the parabolic limit equation. Since the parabolic limit is the outer solution in singular perturbations terms, these higher approximations depend only on the initial values for the parabolic limit problem. We derive several equivalent conditions on the turn angle distribution under which the diffusion matrix is a scalar multiple of the identity, and show that the diffusion constant depends on the second eigenvalue of the turning operator. We shall also discuss signal-dependent turning rates and redistribution kernels, and give an example of an order one, anisotropic perturbation of the redistribution kernel that nonetheless leads to a scalar diffusion matrix. We introduce several different classes of $\mathcal{O}(\epsilon)$ perturbations of the turning kernel and turning rate and show how the chemotactic velocity and sensitivity are obtained from more fundamental and measurable properties of the motion. This leads to a variety of different types of signal dependence of turning rates and kernels for which the jump process is asymptotically described by the Patlak–Keller–Segel–Alt chemotaxis equation. We also discuss an open problem connected with the incorporation of internal dynamics that describe the signal transduction process, and in particular give examples in which diffusion limits do not exist. Finally, we shall discuss some new computational techniques that couple the direct use of Monte Carlo methods with a large-time-step algorithm to produce an efficient scheme that can be used when the macroscopic equations are not known.