A non-linear stability analysis using a multi-scale perturbation procedure is carried out on the practical Thomas reaction-diffusion mechanism which exhibits bifurcation to non-uniform states. The analytical results compare favourably with the numerical solutions. The sequential patterns generated by this model by variations in a parameter related to the reaction-diffusion domain indicate its capacity to represent certain key morphogenetic features required in a recent model by Kauffman for pattern formation in the *Drosophila* embryo.

1. Introduction. Understanding the development of biological pattern is a major problem in morphogenesis. In his seminal paper Turing (1952) proposed in essence a reaction-diffusion mechanism involving two morphogens as a model for pattern formation. Such models have been widely studied and it is now well known that they can generate a remarkably wide spectrum of patterns. Recently there have been several attempts to relate such models to genuine biological situations: see, for example, Gierer and Meinhardt (1972), Kauffman et al. (1978), Bunow et al. (1980), Catalano et al. (1981), Gierer (1981), Kauffman (1981) and Murray (1981a, b). There are, of course, limitations in the applicability of such a mechanism: see, for example, Bard and Lauder (1974), Lacalli and Harrison (1979) and Bunow et al. (1980).

There have been several papers on analytical procedures for finding the non-uniform steady-state solutions to specific reaction-diffusion systems, for example by Granero et al. (1977) and Haken and Olbrich (1978), who used synergetic techniques developed by Haken (1977). Here we analyse the Thomas (1975) model mechanism using a multi-scale perturbation method.

In Section 2 we briefly describe the model system and discuss the
bifurcation conditions we consider. These are motivated by the patterns observed in the early embryo of *Drosophila*. In Section 3 we give the results of the non-linear analysis. Finally, in Section 4 we discuss and compare our results with numerical computation and describe their biological relevance.

2. Model Reaction-Diffusion System and Linear Analysis. The Thomas (1975) reaction-diffusion mechanism can be represented by the dimensionless non-linear system (Kernevez et al., 1979)

\[ \begin{align*}
    u_t &= f(u, v) + \Delta u, \\
    v_t &= g(u, v) + \beta \Delta v,
\end{align*} \quad (1) \]

where

\[ \begin{align*}
    f(u, v) &= \gamma [\alpha (a_0 - u) - \rho F(u, v)] \\
    g(u, v) &= \gamma [s_0 - v - \rho F(u, v)] \\
    F(u, v) &= \frac{uv}{(1 + v + kv^2)}
\end{align*} \quad (2) \]

and \( \gamma, \alpha, a_0, s_0, k, \rho \) are positive constants, \( \beta \) is the ratio of diffusion coefficients and \( u, v \) represent the concentration of a co-substrate and substrate. The form of \( F \) and the kinetics (2) indicate a substrate inhibition mechanism. The parameter \( \gamma \) is directly proportional to the square of the domain size.

We consider (1) to have a unique positive uniform steady state \((\bar{u}, \bar{v})\) given by the solution of

\[ f(u, v) = 0 = g(u, v), \quad (3) \]

that is, the intersection of the kinetics nullclines; Figure 1 illustrates a typical form for these.

We consider one space dimension and zero flux boundary conditions. Perturbations from the uniform state are denoted by the vector \( W \). Equations (1) can then be written in the form

\[ W_t = L(\gamma, \beta)W + h(W, \gamma, \beta), \quad W = \left(\begin{array}{c}
    u - \bar{u} \\
    v - \bar{v}
\end{array}\right), \quad (4) \]