Conclusion

The conclusion must not merely repeat that discussion in the earlier results chapters but extend the discussion so that it is a broader argument in support not only of your answers to the research questions, but also of the contributions to the research field. The second key aspect addressed in the conclusion is an evaluation of the practical and/ or theoretical contributions or significance of your research. This second aspect usually includes an acknowledgement of limitations of your research as well as a discussion of key research questions for future research. As you address these two aspects in your conclusion, you may include elements or types of information which serve the following functions:

1) **Restate the aim(s)** of your study (RE-AIM)
2) **Review key results** obtained in your study (RE-RES),relating them to how your overall aim has been achieved
3) **Relate** your work **to broader research areas** to show your contribution to the field by:
    a) **Comparing** your results/work with previous results/work(CP)
    b) **Explaining** key results(EXP)
    c) **Generalizing** on the basis of key results(GEN)
4) **Evaluate the significance** of your results/study(SIG)
5) Acknowledge **limitations** of your study(LIM)
6) **Recommend** future research or application(REC)

Conclusions in papers seldom contain all these types of information, but conclusions in these typically contain most, if not all of them. Broadly speaking, ideas in a conclusion tend to move from the 'specific' (to the study) to the 'general' (beyond the study) in a cyclical pattern, as in the case of Sample 6.1, which is the conclusion section at the end of a Results chapter of a thesis.

§ 1 This chapter examined the effect of the canonical epidemiological structure-SIS dynamics- in the canonical growth model. §2 The detailed modeling of disease dynamics has revealed how SIS dynamics may affect the dynamics of economic variables. For the SIS dynamics of disease transmission, depending on the infectivity of the disease, it was found that there exists a disease-free steady state in which case there are no long run effects on the economy. The disease could be endemic and reduce the steady state capital and consumption or if the disease is infectious enough, cycles and chaos emerge. §3 This is because the prevalence of diseases affects the labor supply in the model, and hence the dynamics of capital, which in turn affects output and consumption. §4 It was also found that the emergence of chaos and cycles does not depend on the parameters of the economy but on the disease dynamics, §5 thus showing that non-linear dynamics are possible for a wider range of models and parameters. The modeling

results have shown that changes in the epidemiological parameters of disease transmission can have major implications for the resulting equilibrium dynamics. §6 The model has taken the first step in integrating epidemiology models from the mathematics-biology literatures into dynamic economic models. By using the epidemiological structure of disease transmission we can look into the blackbox: the details of disease transmission and the capital accumulation process that crucial in understanding their interaction, and for the formulation of public policy. By incorporating disease dynamics into a standard neoclassical growth model, we are able to generate endogenous fluctuations which do not depend on the stability properties of the capital accumulation process. This is important since it means we can interpret the disease as a 'real productivity shock'. §7 The only difference between the model here and the classical real business cycle model is that here the disease is not a random shock but a purely deterministic one. §8 This also points to the possibility of stabilizing the disease shock because only the chaos generated by a deterministic dynamic system can be stabilized by using the method of Ott, Grebogi and Yorke[21]. §9 In the model, this method which perturbs the parameters of disease transmission can be interpreted as either a low efficacy vaccination programme or isolation of infective individuals. §10 The interpretation of what the real shocks are has major implications for potential stabilization policies because if we only think the real shock is random, there is no way we could stabilize it. §11 It should be noted that the model here has two limitations. First, it does not take into account the fact that a major impact of many diseases is that they cause increased mortality amongst the infected population. Taking a first step in integrating epidemiology models, the model does not incorporate mortality as it would complicate the model. The model only considers the case where infection incapacitates the individual from participating in the labor force but does not lead to increased mortality. Secondly, in this chapter, the disease transmission is exogenous in that the parameters do not depend on economic variables. As such, the model could be interpreted as a developing economy with no public health policies in place. §12 Further research is needed to extend the simple model to incorporate disease related mortality and more complex epidemiological process, and to embed these into an endogenous growth framework.