

Introduction

Overview

There has been much discussion about contagion and diffusion in interstate war, but there has been little discussion of how contagion works. This book seeks to provide a general explanation of the processes that make interstate war spread by looking at how contagion actually worked in the First World War. The logic of the analysis is to begin with some *ex ante* theoretical expectations, refine those on the basis of a general knowledge of the First World War into a set of hypotheses, investigate them empirically, and then infer from the investigation a set of generalizations about how and why wars spread. These generalizations must be seen as a set of untested hypotheses that need to be investigated on other cases before they are accepted.

The book is an exercise in the logic of discovery and not the logic of confirmation (see Scheffler 1967; Freyberg-Inan *et al.* 2016). From a philosophy-of-science point of view it is perfectly fine to derive hypotheses from one case as a way of discovering how processes might work, so long as these hypotheses are tested on a different data sample or set of cases as a way of trying to confirm or falsify them. The discovery of patterns and the formulation of hypotheses may involve a good deal of induction. Confirmation, however, is primarily a deductive exercise with strict rules for testing. The two have different logics, but both are legitimate aspects of the scientific path to knowledge (see Vasquez 1993: 4–5). The logic of discovery simply refers to the process by which hypotheses and theories are constructed. They can have a variety of sources, including derivation from paradigms, reflection on personal experience, hard thinking, counter-factual analysis, and so on and so forth. In fact to call all these sources a “logic” is to stretch the word. There are in fact few criteria that are accepted, although any one source may have criteria. The point is that there are a variety of sources, and Freyberg-Inan *et al.* (2016: 173) call for a social pluralism and inclusiveness of approaches in the realm of discovery, but more exclusiveness

2 Introduction

with regard to the logic of confirmation. With the latter they argue in favor of some kind of neopositivism. The relevant point here is that the conclusions and inferences of this study, while they may tell us something about why the First World War spread, will only provide some knowledge about war contagion as a general phenomenon once the derived hypotheses have been tested on other cases or data using criteria based on the logic of confirmation.

The cases included in this study are listed in Table I.1. These include all the major dyads that declared war, with the exception of several Latin American and miscellaneous states that had limited involvement and are listed in Table 5.1 in Chapter 5.

Six contagion processes are identified and their underlying logic elucidated. Each of the different kinds of contagion is used to explain how contagion worked to bring in the countries that went to war between 1914 and 1917. The six contagion processes are

1. Alliances
 - Contagion through alliances due to the failure of the coercion game
 - Contagion through alliances as a logical afterthought – valence balancing
2. Contiguity
3. Territorial rivalry
4. Opportunity
 - Opportunity due to changes in a rival's power
 - Opportunity due to the breakdown in the political order
5. Economic dependence
6. Brute force

To study these contagion processes each of the key pairs of states (dyads) that entered the First World War is analyzed in depth. These dyad analyses are presented in chronological order of their declaration of war. The study begins with the Austro-Hungarian–Serbian local war and then discusses how and why Germany–Russia joined that war and so forth until Greece, the last main dyad entry, joined in 1917. These dyad analyses combine both historical case analyses and Correlates of War data on major factors associated with war onset – the number of militarized interstate disputes (MIDs), alliance memberships, territorial claims, rivalry scores, and arms races. Within the dyad analysis, attempts will be made to separate what is unique to the case and what is generalizable. The last part of the book culminates in a set of testable hypotheses on how contagion works.

Table I.1. *Belligerents in the First World War by date*

Date	Country	Country
July 28, 1914	Austria-Hungary	Serbia
August 1, 1914	Germany	Russia
August 3, 1914	Germany	France
August 4, 1914	Germany	Belgium
August 4, 1914	Britain	Germany
August 5, 1914	Montenegro	Austria-Hungary
August 6, 1914	Austria-Hungary	Russia
August 6, 1914	Serbia	Germany
August 8, 1914	Montenegro	Germany
August 12, 1914	France	Austria-Hungary
August 12, 1914	Britain	Austria-Hungary
August 23, 1914	Japan	Germany
August 25, 1914	Japan	Austria-Hungary
August 28, 1914	Austria-Hungary	Belgium
November 2, 1914	Russia	Ottoman Empire
November 2, 1914	Serbia	Ottoman Empire
November 5, 1914	France	Ottoman Empire
November 5, 1914	Britain	Ottoman Empire
May 23, 1915	Italy	Austria-Hungary
August 21, 1915	Italy	Ottoman Empire
October 14, 1915	Bulgaria	Serbia
October 15, 1915	Britain	Bulgaria
October 15, 1915	Montenegro	Bulgaria
October 16, 1915	France	Bulgaria
October 19, 1915	Russia	Bulgaria
October 19, 1915	Italy	Bulgaria
March 9, 1916	Germany	Portugal
March 15, 1916	Austria-Hungary	Portugal
August 27, 1916	Romania	Austria-Hungary
August 28, 1916	Germany	Romania
August 28, 1916	Italy	Germany
August 30, 1916	Ottoman Empire	Romania
September 1, 1916	Bulgaria	Romania
April 6, 1917	United States	Germany
June 27, 1917	Greece	Austria-Hungary
June 27, 1917	Greece	Germany
June 27, 1917	Greece	Bulgaria
June 27, 1917	Greece	Ottoman Empire
July 22, 1917	Siam	Germany
July 22, 1917	Siam	Austria-Hungary
August 14, 1917	China	Germany
August 14, 1917	China	Austria-Hungary
October 26, 1917	Brazil	Germany
December 7, 1917	United States	Austria-Hungary

4 Introduction

Organization

The book is divided into three parts. Part I of the book presents the theoretical expectations of the book and its research design. Chapter 1 provides a summary of the theoretical perspective that is taken in the book and lists the hypotheses that will be probed. It reviews the concept of contagion in the international relations (IR) literature, and defines each of the six processes with illustrations from the First World War based on a general knowledge of the war. These were specified *ex ante* before the research in the second part of the book was conducted. Chapter 2 provides a research design for the book. This study is a qualitative analysis, and the chapter outlines how that analysis will be conducted as well as providing an overview of the quantitative data used to supplement each case study. One of the unique features of the book is the utilization of existing Correlates of War data in combination with an historical analysis to write a case study of each pair of states that declared war, with emphasis on those that fought extensively and meet the Correlates of War threshold of being a participant.¹ The data, which have been collected by others over a period of over three decades, are carefully examined using multiple measures for what *ex ante* are regarded as the key factors making for war involvement.

Part II is the main part of the book, with a case study of each dyad that entered the war. In fact, readers interested primarily in the First World War and not so much in models and research design can start the book here and go back to Part I after reading Part III. Part II presents narratives of each of the main cases. The approach is a **dyadic** analysis, which has been found in quantitative IR to be a more fruitful approach than a systems or country-specific approach (see Rummel 1972). A dyadic analysis looks at conflict by examining pairs of states and their interactions with each other. It is assumed that war arises because of what states do to each other. While a dyadic analysis can be misleading in a multiparty event such as the First World War (see Poast 2010), outside parties can be brought into the analysis by examining their impact on the decision-making that takes place between the principal members of the dyad that is being studied. In analyzing

¹ To be considered a participant in a war in Correlates of War data a state must have had either 100 battle deaths or a minimum of 1,000 troops in active combat (Sarkees and Wayman 2010: 61). All countries in dyads that met this threshold are included in this analysis; most others that declared war are also included, if they are of historical interest, e.g. Brazil (see Chapter 2).

the war, this will often be done in the dyadic analyses by examining the role of “third parties.” Likewise, an emphasis on dyads in the book does not mean that certain structural factors are of little causal import; it is only to say that these structural factors often work through how states behave toward each other. The book centers on a dyadic analysis by examining each and every dyad that legally entered the war, with emphasis on those that did the bulk of the fighting. It also includes a chapter in Part III on those that did not join and why. While dyadic analysis is familiar among political scientists, it is less common among historians. Even among IR scholars the scope of dyadic analysis presented here in terms of case analyses coupled with data is unusual.

Each dyad in the First World War is examined individually, with an emphasis on those that have had intense conflict and subsequent war. Data have been collected on all dyads that have resorted to at least once to the threat or use of force during the period 1816–1929. Technically this means that they have had at least one militarized interstate dispute. The analysis of each dyad will include both a quantitative and a qualitative element. A histogram of a dyad’s MIDs is presented for all dyads that have had at least three MIDs. Data that are thought to be theoretically relevant for explaining this pattern of conflict and whether a MID escalates to war will also be presented. These include data on rivalry, the dominant issue under contention, the presence of territorial claims, the allies each side has, and whether they have arms races. The heart of each dyad analysis consists of a narrative that explains why war between the two states occurred, if it did.

Part III, the Conclusion, consists of two chapters. The first, Chapter 6, is an analysis of the neutrals. This chapter compares those that entered the war with those that did not. The neutrals serve as a benchmark or comparison group for evaluating the explanation of contagion presented in Parts I and II. If that explanation is correct, then the neutrals should lack most if not all of the contagion factors that brought in the dyads that went to war. Chapter 6 tests whether some of the factors that are thought to have spread the First World War can actually distinguish the states that joined the war from those that remained neutral.

Chapter 7 is the culmination of the qualitative analysis to see how contagion actually worked. It draws upon the individual dyad case studies to present a set of hypotheses on contagion meant to apply to all multiparty wars, past and future. It begins by systematically examining what factors brought each of the main dyads into the war – summarizing the primary, secondary and other contagion processes at work. It reviews

6 Introduction

what new insights were learned from each case and, where relevant, formulates new supplemental hypotheses on contagion. Next, it provides a set of general conclusions on the role of alliances, why deterrence failed in 1914, and possible causal mechanisms at work that make for contagion, among other topics. The chapter concludes by looking at future research and its relevance to policy and theory.