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

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

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 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

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.

Part I

Theoretical Expectations

1 Contagion Processes in the First World War

There has been little discussion of how contagion actually works historically and few studies of specific wars in terms of contagion processes (for two exceptions see Haldi 2003; Shirkey 2009). This study seeks to provide a general explanation of war contagion by looking at the spread of the First World War. Six contagion processes are identified *ex ante*. The theoretical expectations underlying these six models are then investigated empirically in Part II to see how contagion worked in this one case. As noted before, but worth emphasizing, the hypotheses in this book are not tested by the First World War case; rather, the case is used to probe some *ex ante* hypotheses and refine them.

The analysis in this chapter begins by discussing the concept of contagion. Next each of the processes will be analyzed and briefly applied to the First World War. Each section will end by deriving a set of general hypotheses about how contagion works. The chapter concludes with some reflections about how contagion worked in the First World War and how it might work generally on the basis of this preliminary review. These questions will then be investigated in Part II of the study.

The Concept of Contagion

Contagion is defined as the spread of war from one set of parties to include new ones. This results in an enlargement and expansion of the war. The etymology of contagion comes from the Latin *contingere*, to have contact with, to pollute (Merriam-Webster). It was first used in Middle English in the fourteenth century. The everyday definition of contagion is based on infection and the catching of a cold or more serious disease through contact. It has a negative connotation, and the word's meaning is expanded to refer to the spread of other undesirable things, like a contagious influence or the spread of a bad idea or theory. There are three causal notions worth highlighting in terms of how the word has been used. The first is the idea of spreading from one person to another. The second is the idea that this spreading is due to contact.

The third is the connotation that what is spread is harmful. In terms of contagious diseases, the underlying biology of infection is now well understood, but this was not the case when the word was first used. In terms of the spread of harmful ideas or practices, the underlying causal factors are still not well understood. Generally, however, a positive or neutral phenomenon is more typically discussed in terms of *diffusion*, with an emphasis on how it spreads.

The concept of diffusion derives more from analogies to the physical world rather than the medical. It refers to the dispersion of particles in a liquid, like dropping sugar crystals into a cup of tea. Diffusion is also used to describe the spread of inventions, like paper or fashion trends. The social sciences use the concept of diffusion to examine how cultural practices, religions, and innovations spread. Recently, social network analysis has been used to map this phenomenon. Geographers use this term in a more objective and neutral fashion without the negative connotations of contagion. For geographers there are two types of diffusion, each of which tries to capture the underlying mechanism at work – contagious diffusion and hierarchical diffusion (Gould 1969). The first refers to something that has spread through contact, or, from the geographers' perspective, things that spread spatially. Here contiguity is a key factor. The second presupposes some underlying structure, which provides a path by which the phenomenon spreads in a non-spatial manner. Fashion trends, for instance, follow a status hierarchy, which means they flow from certain capitals – like Paris and Milan – and then go to New York and so forth down a hierarchy. In IR, alliance structures are seen as a possible diffusion mechanism for the spread of war. Alliances in the First World War are seen as a classic example of hierarchical diffusion.

Studies of interstate war have provided some important distinctions and understandings of why war spreads and the different ways in which diffusion and contagion might occur. Most and Starr (1989) provide an important distinction in terms of the difference between *opportunity* and *willingness*. Opportunity refers to the possibility for conflict. This typically means that if states are contiguous they are more likely to experience diffusion. Opportunity is a pre-requisite for conflict, but it is not sufficient. There must also be a willingness to engage in conflict. In other words, there must be some grievance or reason for conflict. In later work, opportunity has often been seen as related to contiguity and willingness as related to the intensity of issue disagreements between states.

Another important distinction is provided by Davis, Duncan, and Siverson (1978), who distinguish between contagion that results from addiction and contagion that is infectious. Davis *et al.* (1978: 773) find

that wars cluster in the international system because of infectious contagion. Bremer (1982) also finds that interstate and civil wars cluster, as do MIDs, which he sees as being a result of contagion. This again would presumably be the result of infectious contagion.

Bremer's (1982) "The contagiousness of coercion" suggests that there is a broader underlying process here, namely, the use of coercion leads others to use coercion. Coercion is contagious in that its use by one actor increases the likelihood that another will resort to it. The concept of a "demonstration effect" may be relevant to describe what is going on here. Contagion is conceived not so much as a result of infection but as the result of learning to copy something that is successful.

Similarly, Levy (1982, 1983: Ch. 7) investigates contagion in broad terms by seeing when war begets war. He identifies various circumstances in which war leads to war. Two in particular are of interest here: when the war of one actor leads it to initiate subsequent wars, as in the series of Napoleonic Wars, and when a victorious war leads to a squabble over the division of spoils, like in the Second Balkan War. Both of these involve contagion, but they seem to involve different kinds of contagion.

It should be emphasized that both Bremer and Levy, when they use the concept of contagion, do not mean to imply that decision-makers have no choice in whether to enter a war. They are not excluding by definition the role of agency in the spread of war. When they refer to contagion they are referring to an outcome of a set of actions; they are not commenting on the role of motivations. The way the concept of contagion is used here, as in most of the political science literature, is to refer to an outcome where a given phenomenon, whether it be smoking bans or war, spreads from one geographical area to another. The extent to which that process is brought about by agents or is more involuntary is left open. In the analysis sometimes demonstration effects are important and decisions are taken that are influenced by what others did. This happens when Romania is influenced by Italy setting the precedent of breaking an alliance and switching sides. At other times demonstration effects are not the key factor; instead, a given structure, like alliance bonds during the playing of the July coercive game, make states act quickly, although not involuntarily. Contagion can have several aspects when one is looking at why it occurs; the concept of contagion as used here, however, is defined in terms of its outcome – the spreading of a phenomenon. Once this has been established, the analysis will then turn to what brings about this spreading. At that point one can talk about the different contagion processes that bring about the spreading, which are more potent, and so forth.

The above analyses and literature, although highly relevant, are not necessarily about world war. World war involves a situation in which an ongoing war expands to the greatest extent possible. It is this situation that Davis *et al.* (1978) are concerned with. They say that the main theoretical question regarding war expansion is what distinguishes the few wars that spread from the many that do not. The question suggests theoretically that there is something about the ongoing war that encourages other states to decide for war. What is that something? This study will answer that question by examining dyadic decisions to join an ongoing war and categorize those decisions by the different underlying processes that are at work.

In this book a world war is defined as "a large-scale severe war among major states that involves the leading states at some point in the war and most other major states in a struggle to resolve the most fundamental issues on the global political agenda" (Vasquez 1993: 63).[1] If one classifies the goals and means used in war in terms of whether they are limited or total, then a world war employs unlimited (total) means and has unlimited goals (Vasquez 1993: 70, 227–228).

The existing empirical findings on big wars, including world wars (Midlarsky 1988, 1990), highlight some possible infectious processes. One set of findings shows that alliances and contiguity are key factors that increase the likelihood of war expansion (see Siverson and King 1979; Most and Starr 1980; Siverson and Starr 1991). Yamamoto and Bremer (1980) provide additional evidence that contagion occurs by finding that as more major powers join an ongoing war it is more difficult for others not to intervene. This conforms to historical analyses (see Stevenson 2011) that show that in the First World War in Europe pressure mounted on states to join as the war dragged on, and in the end few could ward off such pressures.

Reviewing the literature on war expansion, Vasquez (1993, 2009: Ch. 7) posits six mechanisms by which world wars expanded. Initial joining is typically a result of one of three factors: being allied to a belligerent, having a belligerent on one's border, or being a rival of one of the belligerents. When one of these factors brings in the initial joiner, then the war is spread further by one of three additional factors: the breakdown of the prevailing political order, a general bandwagon effect, or the economic dependence of one of the belligerents on a nonbelligerent. There is some statistical evidence on the first two factors and the bandwagon effect, but the others are untested hypotheses of the author. Subsequent case studies by him of the Second World War

[1] The first part of this definition builds on that of Levy (1985: 365).

in Europe (Vasquez 1996a) and in Asia (Vasquez and Gibler 2001) provide evidence consistent with all six claims, however. More recently Vasquez *et al.* (2011) used a network analysis of the First World War and found that contiguity, alliances, rivalry, and territorial disputes all played a role in the diffusion of the war.

Yet multiparty wars are fairly rare in history, so the author posits that three necessary conditions be present in the system for world wars to occur: (1) there must be a multipolar system (this is true by definition), (2) alliances must reduce that multipolar system to two hostile blocs, and (3) one bloc must not be preponderant over the other.

In a more recent analysis of all multiparty wars (those involving three or more parties) Vasquez and Rundlett (2016) argue and then empirically show that alliances are a necessary condition of multiparty wars. They also show that for the two world wars over 90% of the participants which committed troops had outside alliances going into the war.

War expansion is also the topic of three recent books. Haldi (2003) tries to explain how wars widen by examining two theories of why neutrals enter wars. The first is that they enter to balance and the second is that they enter for reasons of predation. He then reviews a number of cases to see which theory best predicts which cases. A noteworthy aspect of this study is that it looks at two cases before 1815, namely the Seven Years' War and the French Revolutionary and Napoleonic Wars. Shirkey (2009) considers the spread of war by looking at a number of specific wars, including the First World War, but also conducts a statistical analysis. His focus is to look at why states join an ongoing fray. He examines the role of information and unexpected events in both war joining and war exiting. He finds evidence to support many of his key hypotheses, but some hypotheses had to be reformulated or were rejected (see also Shirkey 2009: 212–213). Weisiger (2013) returns to the idea of big wars and asks what are the factors associated with limited vs. unlimited wars. While the study is not explicitly about the spread of war, unlimited wars include many that have spread, so the book is relevant. He utilizes the bargaining theory of war, and finds that commitment problems are particularly important in long and unlimited wars.

The above conceptual distinctions and empirical findings still leave many questions unanswered about how contagion or diffusion works in actual cases and whether there are different causal processes that bring about contagion. This study delineates the different kinds of contagion at work in the First World War and shows how they are not all the same and often have different underlying logics. It does this by examining how each dyad, for example Germany–France, decided to go to war. Although the study focuses on one multiparty war, the analysis is