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Transmission, Barriers, and Constraints

A DYNAMIC MODEL OF THE SPREAD OF WAR

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This article offers a systemic and dynamic model of the spread of civil and interstate war. The model is based on three components: transmission mechanisms, barriers, and resource constraints. Transmission mechanisms and barriers are based on our existing notions about the contagious effects of alliances and borders. Resource constraints are designed to capture the social welfare trade-off associated with military spending. Deductions from the model include a positive equilibrium value for the amount of war and the level of resources devoted to war fighting and preparations, the finding that additional transmission mechanisms increase the equilibrium level as well as the speed at which the equilibrium level is approached, and the conclusion that extraordinarily high levels of war will eventually decrease. Recommendations for decreasing a high-war equilibrium focus on the strengths of constraints relative to barriers.

In August 1990, Iraqi troops invaded Kuwait. Less than 1 week later, American troops were positioned in Saudi Arabia, and within 2 months, 27 more nations had sent troops to the region. The Gulf War can be seen to have further “infected” additional actors in the sense that, not coincidentally, Syria chose this time to invade Lebanon. Because the United States was preoccupied with defending Kuwaiti sovereignty and ensuring that Israel not be drawn into the ongoing conflict with Iraq, Syria wisely judged that the superpower would be unable to become simultaneously involved in the Syrian-Lebanese dispute. During Iraq’s occupation of Kuwait and for a short time following the end of the war, Iraqi troops killed approximately 300,000 Kurds. Iraq’s attack on Kuwait thus spread to the United States, Saudi Arabia, and other nations lending troops to defend Kuwait, to Syria and Lebanon in the form of an additional initiation of

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military conflict, and to the ethnic population of Kurdistan. Some actors, however, successfully refrained from being drawn into war. Iraq’s January 1991 Scud attacks on Israel—which some argued were launched with the explicit intention of drawing in other Arab nations—did not provoke any additional participation; even Israel itself demonstrated unprecedented restraint. In this setting, war either spread to a variety of actors or was contained for multiple reasons. Despite this diversity across the specific types of contagion, one is struck by the universal nature of causation—the common instigating event, the sensitivity of actors to geographically and temporally proximate international interactions, and the intrinsic violence linking a state’s international conflict behavior with its domestic repression of an ethnic minority.

This article seeks to transcend the particular details relating to how war spreads in specialized ways and to provide a single underlying explanation for the spread or containment of war. Therefore, it does not take the route used by other modelers interested in contagion. It does not focus on the decision-theoretic factors involved in state choices to initiate or join a war or choices about which side to join (e.g., Altﬁeld and Bueno de Mesquita 1979; Bueno de Mesquita 1981; Bueno de Mesquita and Lalman 1992). The rationale for modeling at the broader level lies in the realization that the very essence of contagion, much like nuclear proliferation, is its systemic nature. Faber, Houweling, and Siccama’s (1984, 278) notion of “social forces” and Bremer’s (1982, 30) story about how a “culture of violence” pervades the international system are both consistent with this systemic understanding of contagion.

The systemic explanation offered here is conveyed as a formal dynamic model and is based on three components that regulate interactions among regionally defined groups of states: transmission mechanisms, transmission barriers, and constraints. Transmission mechanisms, such as proximity or defense pacts, facilitate the spread of war. Transmission barriers, such as distance or nonaggression pacts, slow or prevent diffusion. Constraints on the spread of war are imposed by states’ inability to expend an infinite amount of resources on fighting wars. The resulting model traces the ebb and flow of both war involvements and military resource allocations at the regional and ultimately systemic level.

This article is organized as follows. First, I briefly examine empirical evidence for war contagion. Second, the mechanisms of transmission are discussed and modeled. Next, the same is done for barriers and constraints. Then, deductions from the model are generated with numerical simulations, and their substantive importance is addressed. Last, I offer some general conclusions about the model’s contributions to understanding the contagion process.

**SIGNS OF CONTAGION**

The approach taken here differs significantly from those used in the empirical investigations of war diffusion. Nonetheless, the extant knowledge gained from those investigations is telling and useful. Currently, the empirical literature on the spread of war clearly demonstrates the existence of the contagion phenomenon. The next step is to uncover the contagion process.
Consider the work done by Most and Starr (1980) and Siverson and Starr (1991) regarding the effects of warring border nations (WBNs) and warring alliance partners (WAPs) on a nation’s likelihood of becoming involved in a war. The authors found a strong correlation between a nation’s likelihood for war involvement and the existence of WBNs and WAPs. Siverson and Starr also discover that contiguous WBNs are more likely to draw in neighbors than cross-water warring border nations, which in turn are more likely to draw in neighbors than colonial WBNs. They additionally conclude that warring defense alliance partners are more contagious than warring entente partners, but warring neutrality agreement partners pose very little risk (Siverson and Starr 1991, 56). These findings indicate that one nation’s war involvement is influenced by other nations’ war involvements and that proximity and high levels of alliance commitments generally enhance the effect, but they offer little more than statistical correlation.

A second type of research focuses on the characteristics of empirical distributions. For example, Houweling and Siccama (1985) find that war outbreaks and participations are not randomly distributed in time and space. Yamamoto and Bremer’s (1980) work reveals similar findings on major power entries into wars, and Davis, Duncan, and Siverson’s (1978) work yields parallel findings on dyadic war initiations. The features of these three aggregate distributions are consistent with what we would expect when contagion operates, but the process that produces the distribution is not well understood (Most, Starr, and Siverson 1989, 115-17). Houweling and Siccama themselves conclude that their analysis “does not reveal the cause of the spread of war” (661). Nonetheless, the systematic and consistent nature of these findings tells us we have good reason to suspect contagion. In response, the theoretical goal of this article is to provide a picture of the underlying contagion process.

TRANSMISSION

The causal component of the contagion metaphor lies in the contact mechanisms that facilitate transmission of the war virus. Small children might pass colds to others by sharing toys. How do international actors expose one another to conflict? Numerous answers can be found. Taken separately, each example of a transmission mechanism seems too tailored to specific cases to be of use in a general theory. Consideration of the set of examples, however, allows similarities to be drawn out. These similarities serve as the basis for a formal model. Listed below are some of the specialized explanations of contagion found in the literature. For now, I concern myself only with conflicts involving at least one state actor. I discovered five stories describing transmission mechanisms responsible for the spread of such wars:

1. “Third states may intervene in order to defend an ally, protect their own interests, maintain the existing balance of power, or perhaps demonstrate their own credibility” (Levy 1982, 563).
2. “Some [nations] resort to war because their adversary is already involved in fighting” (Houweling and Siccama 1985, 641).
3. “An outbreak of war in some regional setting is likely to affect the local distribution of power more dramatically than the power distribution in the rest of the world. . . . When
two states are engaged in warfare, their war potential vis-à-vis third states decreases sharply, giving other nations an opportunity to start fighting without fear of intervention. We expect them to do so in the vicinity of existing battlefields because military power operates at increasing cost if projected over longer distances (the “loss of strength” gradient) and because the immobilizing effect of ongoing warfare is the largest in its vicinity. Another way of expressing the expectation of regional infection is that additional nations, trying to maximize their share of the spoils or to minimize their part of the losses, will be situated predominantly in the vicinity of hostilities that are already going on” (Houweling and Siccama 1985, 648).

4. “The expansion of an ongoing war by the use of military force against nonbelligerents may be perceived as necessary for victory or the achievement of other national objectives” (Levy 1982, 563).

5. “Revolutionary movements gain momentum in neighboring states and are thereby responsible for growth in the number of civil wars in any particular region” (Houweling and Siccama 1985, 641).

These are all mechanisms of “infectious contagion”—that is, they all refer to a situation in which one actor’s war involvement increases, through exposure, the probability of another actor’s war involvement (Davis, Duncan, and Siverson 1978, 778). The literature currently makes two important distinctions concerning transmission mechanisms—whether they cause new war initiations or new participations in ongoing wars and whether they can be characterized as opportunity or willingness. The remainder of this section addresses the usefulness of these distinctions and then lays the groundwork for the contagion model.

JOINERS VERSUS NEW INITIATORS

Some of the exposure mechanisms listed above affect how wars currently under way spread to involve new participants and some affect how wars begin. Based on the belief that these two processes are inherently different, authors of many empirical studies (e.g., Davis, Duncan, and Siverson 1978; Most and Starr 1980; Yamamoto and Bremer 1980; Siverson and Starr 1991) intentionally investigate only one type. A closer look, however, reveals that individual transmission sources ultimately influence both war types.

Perhaps the most pernicious example of a mechanism that pulls additional states into war is that of alliances. As Levy (1982) explains, states sometimes join wars to protect their alliance partners or to ensure the credibility of their promises to partners in the future. Although the assassination of Austrian Archduke Ferdinand in 1914 is seen as the event that precipitated World War I, the system of alliances is most commonly blamed for the war’s rapid consumption of so many European states. Might alliances also be responsible for war initiations? In Bueno de Mesquita’s (1981) The War Trap, the nation deciding whether to initiate war must consider the strength of other nations’ alliances with itself and the opponent. This is a measure of how likely third parties are to join a war and on which side. Because the addition of an ally into a war alters the relative capabilities of the two sides, it ultimately affects the likely outcome of the war and hence the choice to initiate or refrain.
Houweling and Siccama (1985), in the second entry in the list above, point to the
dual role played by another type of political relationship, enmity. When the enemy is
busily engaged in war, a nation might seize the opportunity to initiate a war. According
to this story, the target of the attack may or may not be the preoccupied enemy. The
calculating nation may initiate war against an opponent whose attentions are turned
elsewhere, as in 1812 when the United States declared war on Britain while British
troops were engaged in Spain (Houweling and Siccama 1985). Alternatively, the
calculating nation may initiate war against an altogether different opponent, reasoning
that the safest time to do so would be when any potential objectors are inattentive.
Such was the case in the Anglo-Persian War, which was initiated by Britain while
Russia was busy in the Crimean (Houweling and Siccama 1985). In one scenario, the
target is already at war at the time of attack. When this is the case, a preexisting war
(between the enemy and its opponent) appears to expand. In the other, the target is not
at war at the time of attack, but some enemy of the initiator is. Here, the preexisting
war does not grow in terms of the number of participants, but it spreads to additional
dyads that were previously uninvolved. Strong political relationships, in the form of
either alliances or enmities, should lead to both expansion and new war initiations.

Geographical proximity is another transmission mechanism that influences both
types of contagion. In the third example above, Houweling and Siccama (1985) explain
that “additional nations” may join because they want to share in the spoils or minimize
their losses from the war. States that are most likely to be so motivated or so affected
are those that are nearby. In other words, geographical proximity enables transmission.
Saudi Arabia’s participation in the Gulf War can be explained by its contiguity with
both Iraq and Kuwait and a fear that an unchecked Iraq might next turn its aggression
toward Saudi Arabia. It was in Saudi Arabia’s best interest to minimize the possibility
of such an attack. The fourth example above provides more insight into how proximity
plays a role in the spread of ongoing wars. Levy (1982) points out that one of the
belligerents may consider attacking additional states as part of its overall plan. If we
consider Germany’s behavior during World War II, Iraq’s attacks on the Kurds shortly
following its invasion of Kuwait, or Soviet and American notions of “spheres of
influence,” we are left with the impression that the “national objectives” Levy speaks
of are commonly linked to domination or control of territories close to the homeland.

Proximity influences the occurrence of new wars as well. In the third entry in the
list above, Houweling and Siccama (1985) posit that states near those at war are most
likely to be affected (because of regional power shifts), are most likely to initiate new
wars (because the immobility of the warring states is greatest near home), and are most
likely to choose states in that same region as targets (due to Boulding’s [1962] loss of
strength gradient). This story fits nicely with Syria’s invasion of Lebanon during the
Gulf War. Proximity can also produce a new conflict when, as in the fifth explanation
on the list, a revolutionary movement in one state has enough “momentum” to carry
over to a nearby state. In this way, civil wars are also contagious.

In sum, there is great overlap between the transmission agents responsible for the
expansion of ongoing wars and those responsible for new war initiations. Therefore,
my model begins with a war variable that does not distinguish between initiations and
additional participants. Instead, $w$ represents the proportion of states at war at a given time. If there are five states and two are at war at time $t = 1$, then $w(1) = 2/5$. If at time $t = 2$ two more states join the first war, then $w(2) = 4/5$. Similarly, if at time $t = 2$ two more states instead initiate their own dyadic war, then $w(2) = 4/5$. In other words, the model makes no distinction between additional states joining an ongoing war and states initiating a new war.

**OPPORTUNITY OR WILLINGNESS?**

The second common distinction concerning types of transmission mechanisms is whether they constitute opportunity or willingness. Opportunity typically refers to systemic structures that produce alternatives from which nations choose, and willingness refers to factors influencing which alternative a nation will choose (Sprout and Sprout 1969; Most and Starr 1980). Although these ideas have been useful in sorting out the effects of various types of alliances and borders, it may be time to move past them.

In the contagion literature, interstate borders are conceptualized as interaction opportunities (Most and Starr 1980). States with contiguous borders have the most interaction opportunities, followed by states with cross-water borders, followed by states with colonial borders. States that are farther from one another have fewer interaction opportunities. We can think of proximity as a transmission mechanism and distance as a barrier to transmission.

Alliances, on the other hand, are thought of as both opportunity and willingness (Siverson and Starr 1991). Alliances embody willingness because they influence which alternative a nation will choose. In forming an alliance, states forgo some degree of sovereignty and choose to rely on the capabilities and promises of other states in lieu of building up their own defenses (Sorokin 1994). With these newly acquired capabilities and the concomitant uncertainty of having them available, states' decisions about entering into or refraining from war are altered (Altfield and Bueno de Mesquita 1979; Bueno de Mesquita 1981; Bueno de Mesquita and Lalman 1992). But alliances also enable states to overcome the geographical constraints imposed on them by nature (Starr and Most 1976). Thus, they function to change the system itself or to affect the opportunities available to them. Moreover, we might think of alliances as a measure of proximity, not in terms of geographical space but in terms of preferences (Bueno de Mesquita 1981). Proximity (or similarity) of preferences can also be considered to be an interaction opportunity.

The usefulness of the distinction between the role played by opportunity and that played by willingness is not clear. Both are necessary conditions for the spread of war, so researchers expect them both to be positively related to increased war participation. Their empirical referents, alliances and borders, share many features, with alliances taking on the opportunity characteristic of borders. Researchers investigating the effects of both use identical statistical models and techniques, such as probit analysis, to understand them (Siverson and Starr 1991). Furthermore, the idea that the system produces a constant set of opportunities, whereas willingness changes over time, has been discarded following the discovery that borders are not static (Starr and Most
1976; Diehl and Goertz 1988), Siverson and Starr (1991, 34-35), although adhering to the opportunity and willingness framework, seem to reach the conclusion that alliances and borders share fundamental traits relevant to contagion:

If alliances can be used to indicate the salience and importance of states to one another and delineate subgroups of highly interacting states, then they should have an impact on the diffusion of war similar to that found by Most and Starr using borders as agents of diffusion.

Opportunity and willingness are therefore not incorporated into the dynamic model below. Alliances and borders do, of course, appear, but they are not distinguished from each other based on those two concepts. Instead, they are both represented by regional groups of states and the various degrees of interaction within and among them.

FORMALIZING TRANSMISSION MECHANISMS

The formal model, then, will explain the behavior of the war variable, \( w \), without distinguishing between new joiners and new initiators. And the structure of the equations explaining \( w \) ’s behavior will reflect interstate interactions regardless of whether they are based on physical proximity or political relationships. To represent both the geographic and political interactions among nations that facilitate the spread of war, I assume that nations can be regionally grouped. Europe, Latin America, the Middle East, and Asia are examples of geographic regions. Regions are important not only because nations within a region are physically proximate but also because they have more extensive political ties as well (Lake and Morgan 1997). For the sake of simplicity, further suppose that there are three such groups of nations: \( a \), \( b \), and \( c \). Then each region will have a variable \( w \) associated with it. The proportion of a region’s states participating in a war at any given time is represented by \( w_a \), \( w_b \), and \( w_c \). To understand the contagion phenomenon, it is necessary to trace the behavior of \( w \) over time. Coleman’s (1964) model of the diffusion of an innovation among individuals in partially interconnected groups provides a useful starting point. Modifying his model produces a set of equations representing the evolution of the amount of war over time.

Equations for the behavior of \( w_a \), \( w_b \), and \( w_c \) are parallel, so only that for \( w_a \) is given here:

\[
\frac{dw_a}{dt} = \kappa_{aa} w_a (1 - w_a) + \kappa_{ab} w_b (1 - w_a) + \kappa_{ac} w_c (1 - w_a),
\]

(1)

where \( \kappa_{aa} \) is the positive transmission rate from war participants in group \( a \) to nonparticipants in \( a \), \( \kappa_{ab} \) is the positive transmission rate from war participants in group \( b \) to nonparticipants in \( a \), and \( \kappa_{ac} \) is the positive transmission rate from war participants in group \( c \) to nonparticipants in \( a \).

The first term on the right-hand side of equation (1) denotes intragroup transmission, and the second and third terms represent intergroup transmission. The multiplicative feature of the three terms captures the interaction of the peaceful states, \( (1 - w_a) \),

1. Deductions from the model are not affected by adding more regions.
with the various war-infected states, \( w_m, w_p, \) and \( w_e \). This notion of interaction is much like that used in population models. The product of two populations represents the baseline interaction rate given the size of two populations.\(^2\) As I have argued above, however, factors such as alliances and borders affect the frequency and contagiousness of those interactions.

Enter the \( \kappa \) parameters. They modify the extent to which these interactions, or exposures, occur and the probability that interactions actually produce a transmission of the war virus to the uninfected nations.\(^3\) When their values are high, transmission mechanisms are very contagious, as in the case of contiguity and strong political relationships. When their values are low or zero, barriers operate to slow or halt transmission, as in the case of distance or weak relationships. The \( \kappa_{ij} \) parameters, or intergroup transmission rates, represent the level of interaction opportunities between two groups of nations and the extent to which those opportunities will result in the spread of war. These parameters might be based on interregional alliances (e.g., the United States and Britain) or enmity (e.g., the United States and the U.S.S.R. during the cold war). They might also be based on geographical proximity of one region to another (e.g., the short distance separating the Middle East and Europe) or distance (Latin America and Europe). The \( \kappa_{ii} \) parameters, or intragroup transmission rates, similarly represent the level and effect of interaction opportunities among the nations in a single region. They too might be based on alliances (Britain and France), enmity (India and Pakistan), proximity (the contiguous border between Israel and Egypt), or distance (Mexico and Argentina).

A few additional points on the \( \kappa \) parameters are in order. First, because the \( \kappa \) parameters reflect the impact of interactions between or within groups of nations and not the extent of interaction between two specific nations, they should be understood to be aggregate measures. For example, when speaking about the proximity or distance within Latin America, \( \kappa_{ii} \) would be an indication of the degree to which nations in that global region are dispersed. If considering distance alone, we would expect \( \kappa_{ij} \) for Latin America to be much smaller than for the Middle East. Similarly, a high value of \( \kappa_{ij} \) based on alliance ties alone would indicate many strong commitments between nations in groups \( i \) and \( j \) but would not provide any information about the particular alliance arrangement between one specific nation in group \( i \) and one in group \( j \).

Second, because the \( \kappa_{ij} \) parameters represent general connectedness between groups and not dependence relationships, I use the reciprocity assumption:

\[
\kappa_{ij} = \kappa_{ji}.
\]

That is, war in group \( i \) is as contagious to group \( j \) as war in group \( j \) is to group \( i \). This assumption might be relaxed in future research if the goal is to examine the effect of

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2. In the predator-prey model, representing how many rabbits die due to being eaten by foxes involves the multiplication of the fox population by the rabbit population (Mesterton-Gibbons 1989, 154-57).

3. If I want to expose my daughter Lily to the chicken pox before she enters kindergarten, I can increase the frequency with which she plays with infected children or I can increase the probability she gets it from one particular infected child by having her share a drinking cup with the child. Both have the same effect of increasing the transmission rate.
unbalanced relationships, such as defender-protégé, empire-colony, or economic dependence scenarios.

Third, given the evidence that proximity plays a crucial role in the contagion process, and given that states within a region, on average, are closer to one another than states from different regions are, I will assume that

$$\kappa_{ii} > \kappa_{ij}.$$  

In other words, within-group transmission rates are always higher than between-group transmission rates. Proximity is not solely responsible for this higher rate of intragroup transmission. Regional groups also experience higher interactions due to more frequent and stronger political and economic ties. Note that I do not make the assumption that within-group proximity matters in the sense that the $\kappa_{ij}$s have different values. To do that, I would need to specifically identify empirical regions, such as Latin America and the Middle East, and assign values to the $\kappa_{ij}$s based on the area covered by a region and the number of nations in each region. But the current purpose of this model is to provide a baseline that can later be modified to suit empirical considerations. Hence, the initial working assumption is simply that all regions have identical rates of within-group transmission, or

$$\kappa_{ii} = \kappa_{ij}.$$  

If the proportion of states in each group that are at war at any given time were determined solely on the basis of equation (1), and all $\kappa$ values were positive, then war would eventually consume every state in every group, as can be seen in Figure 1. But the empirical record tells us otherwise. Instead, there must be some constraints or barriers operating to limit war participation in each group. They are the subject of the next section.

**BARRIERS AND CONSTRAINTS**

Goertz (1994, 23) defines barriers as "counteracting forces" that "prevent events from taking certain courses." Following along these lines, I explicitly discuss two substantive examples of barriers: neutrality agreements and resource constraints. Above, I mentioned that geographical distance and weak political relationships (i.e., neither a strong alliance nor a strong enmity) are also types of barriers.

There is a long tradition of treating three types of alliance as if they could be placed on an ordinal scale representing the level of commitment required of signatories, beginning with defense pacts, followed by neutrality agreements and then ententes (e.g., Singer and Small 1966; Wallace 1973; Starr and Most 1976; Sabrosky 1980; Siverson and Starr 1991). But if we conceptualize alliances as agents of transmission, level of commitment may be the wrong dimension altogether. Consider the perplexing nature of some of Siverson and Starr’s (1991) findings concerning the relative strength of the relationships between alliances of a certain type and war joining:
Figure 1: Coleman’s Model

1. The number of entente WAPs “influences war joining much more than” the number of neutrality WAPs, and the latter have “little or no effect on war joining” (56).
2. Neutrality WAPs have a stronger effect than both defense and entente WAPs for the major powers alone.
3. For states that choose whether to enter without having been attacked themselves, neutrality WAPs produce longer waiting times before entry, whereas entente WAPs produce shorter waiting times (instead of the other way around), and the results for neither are statistically significant.

The theme across these somewhat surprising results is that neutrality agreements do not have the expected positive but only moderate effect on contagion. One reasonable conclusion might be that neutrality agreements do not universally embody less willingness than defense pacts and more than ententes.

A closer look at neutrality agreements is informative. First, the neutrality category includes nonaggression pacts as well (Sabrosky 1980). Levy (1981) criticizes this typology. Siverson and Starr (1991, 50) address Levy’s complaint, characterizing it as an argument about the “courseness of the measurement employed.” Although this is part of Levy’s argument, it is not the whole. He states,

Whereas the neutrality pact generally obligates each signatory to remain militarily neutral in the event of an attack on the other, and may even designate specific aggressors to which the alliance is applicable, the more sweeping non-aggression pact is simply an assurance that neither will use force against the other. The credibility of such a guarantee may be
questionable, however, for it may be in situations of greatest suspicion and mistrust that some form of generalized reassurance is sought. (588)

Levy calls into question the validity of measuring the level of commitment with nonaggression pacts. He posits that such treaties are actually indications of a lack of commitment or, worse, a predisposition toward aggression. Because the neutrality category appears to be more strongly associated with increased war joining among the major powers than is the defense category, it would be instructive to know what proportion of the neutrality category is composed of nonaggression pacts in that subset of states.

Further clarification might be found in the definition of neutrality agreements themselves. As Levy points out, neutrality agreements are simply promises to refrain from joining wars involving the signatories. As such, we should expect them to act as barriers, not transmission agents. This expectation would be consistent with Siverson and Starr's (1991) two more general findings, perhaps where the proportion of nonaggression pacts is less pronounced. If a promise to come to another state's defense is a transmission mechanism, it is logical that a promise not to enter the fray at all is a barrier to transmission.

Another type of barrier also operates to restrict the spread of war. Wars are costly interactions, and states cannot bear unlimited burdens from perpetually increasing amounts of war in the international system. A fair amount of research has been done on states' abilities to endure battle fatalities, one specific type of costs. Some authors focus on how public support for the war effort erodes with the cumulation of casualties in democracies such as the United States (Gartner, Segura, and Wilkening 1997; Gartner and Segura 1998 [this issue]). Others look at the maximum limit on the number of casualties as a proportion of population for all states regardless of regime type. Klingberg (1966, 147-48), for example, argues that when states reach this limit, morale becomes so low a nation must surrender. Richardson's (1960, 299) more systematic empirical work confirms this relationship between excessive battle deaths and an impending need to admit defeat and sets the maximum limit at about 5%. Rosen (1972) confirms that limit with an expanded universe of cases.

Battle fatalities are not the only costs states incur during wars. Economic costs, such as the destruction of infrastructure and the diversion of capital to the production of military goods in lieu of consumer goods, lead to a pronounced (albeit not permanent) loss in gross national product (GNP) (Organski and Kugler 1980). Social costs, epitomized in the guns versus butter trade-off, are also hardships. Neither the diversion of capital nor the forgone welfare costs are unique to wartime. Both are relevant during peacetime production of weapons and military spending, but they are undoubtedly more pronounced during wars. Extraordinary economic and social costs can be expected to force states to withdraw from fighting as well. In general, states approaching the maximum limit on costs of fighting (or preparing to fight) face repercussions (e.g., low morale, removal of leaders from office) that force them to refrain from entering wars, negotiate settlements for ongoing wars, or surrender. These cost constraints are incorporated into the formal model by including an interactive war-and-cost counterforce to the spread of war.
FORMALIZING BARRIERS AND CONSTRAINTS

Although neutrality agreements and resource constraints both counteract the transmission process, each operates in its own way. Of the two, neutrality agreements are easier to capture mathematically. In equation (1), contagion barriers erected by neutrality agreements can be represented by setting values for the $\kappa$ transmission rates at or near zero. The effect is to lessen or prevent exposure to the war virus.

Resource constraints, on the other hand, require a more complicated mathematical structure because they both vary over time and affect $w$’s behavior. I develop the equation describing how the proportion of a region’s resources devoted to warfare, $r$, behaves over time. The argument for how $r$ affects war participation goes as follows. States that are currently participating in wars will be increasingly motivated to cease fighting as the proportion of resources devoted to the military approaches the maximum allowed without severe repercussions such as the removal of leaders from office, food shortages, economic collapse, and so on. That rationale can be mathematically expressed by adding the following term to the right-hand side of equation (1):

$$-\gamma w_a \left( \frac{r_a}{r_{\text{max}}} \right)^2,$$

where $\gamma$ is a positive constant, $r_a$ is the proportion of total resources (money, raw materials, labor, public support, GNP) that region $a$ allocates to specialized funds devoted to fighting wars (i.e., national military budgets), with $0 \leq r_a \leq 1$, and $r_{\text{max}}$ is the maximum proportion of resources a group can allocate to the military without suffering disastrous results.

Three features of this negative term warrant discussion. First, note that it includes an interaction between $w_a$ and $r_a$. If either $w_a$ or $r_a$ is zero, the entire term is zero, and therefore $w_a$ increases. On the other hand, if either is large, there will be a large negative effect on $\frac{dw_a}{dt}$. In other words, war is inherently undesirable but is even more undesirable when the resources allocated to fighting are extensive. Moreover, nations do not wage endless wars. Second, $r_a$ is divided by $r_{\text{max}}$ so that when $r_a$ reaches its maximum, there will be the largest negative effect on $\frac{dw_a}{dt}$. Third, the $\left( \frac{r_a}{r_{\text{max}}} \right)$ term is squared because this produces a greater change in the effect on $\frac{dw_a}{dt}$ as $r_a$ approaches $r_{\text{max}}$ (see Figure 2).

The equation representing the evolution of $w_a$ over time therefore becomes

$$\frac{dw_a}{dt} = \kappa_{ab} w_a (1 - w_a) + \kappa_{ab} w_b (1 - w_a) + \kappa_{ac} w_c (1 - w_a) - \gamma w_a \left( \frac{r_a}{r_{\text{max}}} \right)^2. \quad (2)$$

Parallel equations can be written for groups $b$ and $c$ but are not given here.

Now, I more closely consider $r_a$, the proportion of total resources that nations in group $a$ allocate to fighting wars. Military budget allocations as a percentage of

4. Because Coleman’s (1964) model of diffusion portrays the spread of socially desirable innovations such as new medical procedures or televisions, it lacks a negative term.
regional totals of government expenditures are one possible way to measure $r$. The equation representing the behavior of $r_a$ over time is

$$\frac{dr_a}{dt} = \alpha w_a \left( 1 - \left( \frac{r_a}{r_{\max}} \right)^2 \right) - \beta \left( \frac{r_a}{r_{\min}} - 1 \right),$$

(3)

where $\alpha$ and $\beta$ are positive constants and $r_{\min}$ is the proportion of resources devoted to the military that all groups must maintain to ensure their viability. As before, parallel equations are formed for groups $b$ and $c$ but are not given here.

The first piece of the right-hand side of equation (3) represents preparedness. As long as some proportion of states in group $a$ is at war ($w_a > 0$), the group will allocate additional resources to war up to the point at which $r_a = r_{\max}$. As $r_a$ rises, its growth slows. When $r_a = r_{\max}$ or when resources spent on the military reach their maximum, growth halts. The $\frac{r_a}{r_{\max}}$ ratio is squared so that the largest decline in the positive contribution of the

$$\left( 1 - \left( \frac{r_a}{r_{\max}} \right)^2 \right)$$

term to $\frac{dr_a}{dt}$ is close to $r_{\max}$ (see Figure 3).

The negative term on the right-hand side of equation (3) represents a social goods trade-off. It captures the universal desire, irrespective of the value of $w_a$, to minimize military spending because it is undertaken at the expense of other, more socially
redeeming, purposes. The higher $r_a$ is, the larger the negative effect on $\frac{dr_a}{dt}$. But when $r_a$ is small and close to $r_{min}$, the minimum level of military spending necessary for survival, this negative term is its smallest. The $\frac{r_a}{r_{min}}$ ratio is squared so the largest increase in the negative effect of the

$$\left( \frac{r_a}{r_{min}} \right)^2 - 1$$

term on $\frac{dr_a}{dt}$ is near $r_{max}$ (see Figure 4). The upper and lower boundaries on $r$, $r_{min}$ and $r_{max}$ are assumed to be constant across all three regional groups. In sum, equation (3) models the behavior of the cost constraint, one kind of barrier to war diffusion.

**RESULTS**

At this juncture, I offer results from the analysis of the dynamic spread of war model. The analysis consists of numerical simulations that act as substitutes for analytic solutions when equations or systems of equations cannot be explicitly solved.\(^5\)

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5. The parameter values were set as follows:

- $r_{min} = .01$
- $r_{max} = .33$
- $\alpha = .08$
- $\beta = .0001$
- $\kappa_{il} = .05$
- $\kappa_{ab} = .02$
- $\kappa_{ac} = .035$
- $\kappa_{bc} = .04$
- $\gamma = .05$
Fifty sets of random initial conditions for the three resource and three war equations were generated. Each set served as a starting point for a single simulation. Within each simulation, I explored the competing effects of barriers and transmission mechanisms by beginning with barriers between all groups and then changing one barrier at a time into a transmission mechanism. Each simulation, then, has four components:

1. no intergroup transmission (all \( \kappa_{ij} = 0 \)),
2. transmission between groups \( a \) and \( b \) (\( \kappa_{ab} \neq 0 \)),
3. transmission between groups \( a \) and \( b \) and between groups \( a \) and \( c \) (\( \kappa_{ab} \neq 0 \) and \( \kappa_{ac} \neq 0 \)), and
4. complete intergroup transmission (all \( \kappa_{ij} \neq 0 \)).

The most apparent feature of the 50 simulations (typified by simulation 42 in Figure 5) is that all simulations appear to reach an equilibrium. This equilibrium is positive for all \( r \) and all \( w \). In other words, even if states in a particular region initially devote no resources to military purposes and have no war participations, they cannot effectively stop the spread of war. War no longer spreads (and additional resources are no longer devoted to fighting) when there is some positive level of war and military allocation. This is true even for universal intergroup barrier conditions (the first component in each simulation). Focusing our efforts on squelching particular wars such as the Iran-Iraq war may be for naught. Ultimately, the dynamic model tells us that systemic features of contagion will move regional participations toward some

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6. Available at http://www.uiowa.edu/~polisci
7. Graphs of additional simulation results are available at http://www.uiowa.edu/~polisci
No Intergroup Transmission

resource levels  
0.3  
0.25  
0.15  
0.05  
20 40 60 80  
time

war levels  
0.9  
0.8  
0.7  
0.5  
20 40 60 80  
time

a<->b Transmission Only

resource levels  
0.3  
0.25  
0.15  
0.05  
20 40 60 80  
time

war levels  
0.9  
0.8  
0.7  
0.5  
20 40 60 80  
time

a<->b and a<->c Transmission

resource levels  
0.3  
0.25  
0.15  
0.05  
20 40 60 80  
time

war levels  
0.9  
0.8  
0.7  
0.5  
20 40 60 80  
time

Complete Intergroup Transmission

resource levels  
0.3  
0.25  
0.15  
0.05  
20 40 60 80  
time

war levels  
0.9  
0.8  
0.7  
0.5  
20 40 60 80  
time

Figure 5: Simulation 42
positive equilibrium, regardless of who the participants are. A more useful approach to lessening war would shift the emphasis toward controlling the features of the system that affect the value of \( w^* \).

Furthermore, under the two extreme conditions of complete transmission and universal intergroup barriers (the first and last components of each simulation), the equilibrium is described by

\[
\begin{align*}
    r_a &= r_b = r_c = r^* \quad \text{and} \\
    w_a &= w_b = w_c = w^*.
\end{align*}
\]

In cases of mixed barrier and transmission conditions, equality does not appear as a necessary condition for equilibrium. It is interesting that intragroup transmissions alone and a mix of complete intergroup and intragroup transmissions both produce equilibria characterized by equality. In addition, when there is one \( \kappa_{ij} \neq 0 \), there is a region that is completely insulated and can establish its own lower value at \( w^* \). For example, if \( \kappa_{ab} = 0 \) and \( \kappa_{ac} = 0 \) while \( \kappa_{bc} > 0 \), then the equilibrium point \( w^* = \{ w_a^*, w_b^*, w_c^* \} \) will have a lower value for \( w_a^* \) while \( w_b^* = w_c^* \). But insulating an entire region from the rest of the world seems impossible in a world of increasing international trade, improving communications and transportation technology and global environmental concerns such as population and pollution. Under such conditions, the likelihood of all or most nations in a region signing neutrality pacts with all other nations will remain extraordinarily low.

Second, the aggregate amount of war in the system appears to increases as barriers are replaced with transmission mechanisms. At first glance, this may not seem like a surprising result. When intergroup transmission increases during a simulation (going from the first to last component), it is increasing in terms of additional exposure mechanisms, not in terms of exposures becoming more contagious.\(^8\) This means that in numbers, even weak ties to other regions can have dangerous effects, and this finding may indeed be somewhat counterintuitive. The Angola, Vietnam, and Cuba experiences of the cold war demonstrate how multiple ties between regional states and outsiders such as the two superpowers, and not just strong ties, can facilitate higher levels of war. Japan’s attack on Pearl Harbor during World War II might also be blamed on multiple exposures, in the form of U.S. and European (neo)colonial interests in Southeast Asia. Thus, Japan was drawn into World War II not by exposure to the overwhelmingly aggressive policies of Germany but by the multiple instances of militarily preoccupied (former) colonizers. The lesson for the Middle East is that if the number of extraregional interactions remains high, reducing their levels (e.g., by moving from defense agreements to ententes) is not sufficient to lessen warfare.

A third conclusion is that the equilibrium is reached more quickly as barriers are replaced with transmission mechanisms. In total barrier scenarios, equilibria are reached late in the simulations, but in complete transmission scenarios, they are reached early on. For example, in simulation 42, equilibrium is reached after about 30

\(^8\) Increasing the contagiousness of intergroup exposure would require increasing the value of specific \( K_{ij} \).
time units under complete transmission but still has not quite reached equilibrium after
80 time units under complete barriers. In this simulation, intergroup transmission
speeds the approach to equilibrium by cutting the time at least in half. Another way to
see this effect is to look at the shape of the war trajectories as a simulation moves from
no intergroup transmission to complete intergroup transmission. The trajectory for \( w^* \)
in simulation 42 is an \( s \)-curve that gets increasingly steep, with a higher and earlier
inflection point, as transmission mechanisms are added. Not only do transmission
mechanisms increase the aggregate amount of war at equilibrium, but they speed up
the diffusion process and cause the highest rates of increase (the inflection points) to
occur earlier on. In other words, lowering the number of interactions also delays the
spread of war.

Last, because all trajectories beginning above \( w^* \) ultimately decrease until they
reach \( w^* \), we know that extraordinarily high levels of war will eventually diminish.
The policy issue, therefore, is not how to directly manipulate values of \( w \) but how to
manipulate values of \( w^* \). Controlling flare-ups such as the Gulf War with monitoring
or peacekeeping methods may alleviate a temporary surge in war participation in the
Middle East, but they will do little to push war levels below the endemically high
equilibrium value in that region.

CONCLUSIONS

The dynamic model of war contagion offered here builds on stories of transmission
mechanisms, barriers, and constraints, and it has yielded some interesting results. Two
of them—the first and last—suggest that a method for reducing the equilibrium value
of war should be devised. Based on those two results, we know that \( w^* \) is positive and
that extraordinarily high levels of war participation that exceed \( w^* \) will eventually
decline. How, then, might \( w^* \) be lessened? One possible answer lies in changing the
value of the \( \gamma \) parameter. Suppose that the value of \( \gamma \), the parameter modifying the
social goods trade-off portion of the \( \frac{dw}{dt} \) equations, were increased. Would the increased
negative effect of cost constraints produce an equilibrium, \( w^* \), of zero? Simulation 51
(see Figure 6) was designed to investigate that possibility. In that simulation, \( \gamma \) was
increased from .05 to 5 in various increments. The result, even in a total intergroup
barrier case, indicates that although \( w^* \) decreases markedly and is very close to zero,
it is still positive. The complete elimination of war in conflict-prone regions such as
the Middle East appears to be an overly optimistic and impractical policy goal.

What is the substantive interpretation of increasing the value of \( \gamma \)? This parameter
represents how responsive regional governments are to the social welfare demands of
their citizens. The more concerned governments are with the restrictions placed on
them by their populations, the higher \( \gamma \) will be and the stronger and more quickly those
governments will react as resource levels approach the maximum, regardless of what
that maximum level is. In essence, this suggests a regional-level democratic peace
argument based on the structural constraints inherent in democracies.9 Changing this

9. See Morgan and Schwebach (1992), inter alia, for a dyadic analysis.
No Intergroup Transmission, $\gamma = .05$

No Intergroup Transmission, $\gamma = .5$

No Intergroup Transmission, $\gamma = 1$

No Intergroup Transmission, $\gamma = 5$

Figure 6: Simulation 51
social goods constraint of regional subsystems will help lessen the spread of war. The
model offered another potential method of controlling the level of warfare—namely,
erecting intergroup barriers. However, that option seems much less desirable and
feasible in the current environment of a global community.

Fair warning of the deleterious effects of extraregional interactions is also provided
by the model. Regions that reduce the extent of their outside ties may buy themselves
time by temporarily delaying the interregional spread of war. But only the complete
elimination of those ties will stave it off entirely. Given the concomitant sacrifices in
trade and other positive forms of exchanges, states would be better off to pursue the
recommended route of improving regional levels of democratic governance. At the
same time, states must carefully consider the inherent trade-off associated with any
new or additional commitments to others.

One important step for future research is to investigate how robust the conclusions
concerning the equilibrium are when simplifying assumptions are relaxed. One
particular simplifying assumption that clearly needs to be relaxed is that \( r_{\text{max}} \) and \( r_{\text{min}} \)
are universal across regions. It would be more realistic to assume that regions have
varying tolerances for high levels of military spending and varying abilities to maintain
viability with low levels. The Middle East, for instance, should have unusually high
values for both \( r_{\text{max}} \) and \( r_{\text{min}} \). Relaxing this assumption might also help explain different
regional trends in military resource allocation growth or decline (Happe and Wakeman-
Linn 1994, 11-12).

As a baseline model of the aggregate process of war contagion, this dynamic model
has proven useful. Because its focus is at the regional and systemic level, the model’s
deductions are also at that level. For example, instead of predicting whether a particular
nation will enter a war or on which side it will join, this contagion model predicts that
a positive level of war will be maintained by various regions. In addition, it tells us
about the characteristics of that level and suggests methods for lowering it. The spread
of war is part of an aggregate process, and so must be our efforts to control it.

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