

# Explaining Variance; Or, Stuck in a Moment We Can't Get Out Of

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Political scientists overwhelmingly seek evidence of causation by measuring changes in the mean of the distribution of the dependent variable. This article points out that some causal relationships produce changes in the *variance*, not the mean, of that distribution. It makes the case for variance-altering causation by demonstrating its empirical relevance to political scientists. The article also lays out an array of causal mechanisms—under the general headings of aggregation, contagion, and constraint—in order to demonstrate the logical coherence of variance-altering causation and the many ways in which it can arise. The discussion highlights the often-stringent empirical and logical requirements that must be met if the researcher hopes to make concrete predictions about changes in variance.

## 1 Introduction

Political scientists have long puzzled over the meaning of causation. From reliance on Aristotle's distinction among formal, material, efficient, and final causes to current discussions of probabilistic and counterfactual causation, causal complexity, manipulation, and intervention, we have struggled to put into uncontroversial language the nature of the relationship between the phenomena that we study and the things that bring them about.

At present, most empirically oriented political scientists have settled on a definition of a causal relationship that is “mean-centric” and at least implicitly counterfactual. These two aspects of a causal relationship are central to our understanding of politics. They drive research design in books, dissertations, and articles, and they establish essential criteria by which such works are judged: few are accepted for publication without having demonstrated some sort of causal effect, defined in these terms.

Nevertheless, an important and little-understood phenomenon that is likely to have a substantial impact on political science research has emerged in recent years: we have

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*Author's note:* I wish to thank, without implicating, Lars-Erik Cederman, Wendy Tam Cho, Kevin Clarke, Sarah Croco, Margarita Estevez-Abe, John Gerring, Gary Goertz, Yoshiko Herrera, Michael Hiscox, Andrew Kydd, Evan Lieberman, Lisa Martin, Beth Simmons, Hillel Soifer, Monica Duffy Toft, the participants in the Political Participation and Behavior workshop at Harvard University, and three anonymous reviewers for comments on earlier drafts of this article, and Jasjeet Sekhon and William Zimmerman for thought-provoking discussions relevant to it.

begun to notice that some causes produce changes in the *variance*, rather than the mean, of the distribution of a dependent variable. This form of relationship, though understood in fields as diverse as biology and baseball,<sup>1</sup> is undertheorized in political science. To the extent that we think about changes in variance, we tend to treat them as a nuisance to be compensated for (with, for example, robust standard errors) rather than as a substantively interesting phenomenon to be studied.<sup>2</sup> This situation is due to a sort of catch-22: in order to be recognized as causes, variance-altering causes must be made explicit, but in order to be made explicit they must first be recognized as causes.

Toward that end, this article accomplishes three goals. First, it discusses the nature of variance-altering causes and makes the case for an alternative understanding of a causal effect, one based on changes in variance. Second, to demonstrate the relevance of the phenomenon it offers a range of examples that span the subfields of political science. Finally, it elaborates three different classes of causal mechanisms—aggregation, contagion, and constraint—that can produce changes in variance and explores their implications.

The conclusion is a strong if qualified endorsement. Thinking theoretically about variance-altering causes can increase a researcher's theoretical leverage by bringing to light hypotheses that might not have been recognized, and studying variance-altering causes empirically can provide researchers with a more accurate and thorough description of the social and political world than they would otherwise possess. At the same time, the causal mechanisms that produce changes in variance are typically complex, and they rarely produce unequivocal predictions about changes in variance in the absence of detailed knowledge about the context in which they are embedded.

## 2 Causes and Effects

Understanding the nature of causation has been a growth industry for centuries. Distinctions have been made between agent causation, which is purposive, and event causation, which is purely material. Causation has been understood as a “constant conjunction” between events (i.e., as necessary and sufficient conditions), as straightforward logical entailment, as conditions that are insufficient but necessary parts of a condition that is unnecessary but sufficient, as probabilistic relationships involving simple covariance, as a logical “do” operator, as complexity (in which either two causes produce an effect in conjunction or multiple, redundant causes exist for a single event), as a form of manipulation, and as a counterfactual relationship (in which the effect would not have occurred in the absence of the cause)—to name just a few characterizations.<sup>3</sup>

Most empirical studies in political science that examine cause-effect relationships conceive of them as being mean-centric: a causal relationship is defined as one that

<sup>1</sup>See Gould (1996).

<sup>2</sup>Downs and Roche (1979), *inter alios*, make this point in the specific case of heteroskedasticity in bivariate regression. The authors argue against statistical “quick fixes” to eliminate the effects of heteroskedasticity and argue that we should instead seek substantive meaning in the difference in variance.

<sup>3</sup>See Hume (2000/1739); Goertz and Starr (2002), Mill (1865) and Hempel (1962); Mackie (1965) and Kim (1965); Suppes (1970), Eells (1991), and Salmon (1998); Pearl (2000); Ragin (1987, 2000), Jervis (1997), and Braumoeller (2003); von Wright (1971), Rubin (1974), and Rosenbaum (2002); and Lewis (1973) and Tetlock and Belkin (1996), respectively. The interested reader will find substantial additional background material on these subjects in Sosa and Tooley (2001) and Ruben (1993).

produces an effect, conceived of as a change in the mean of the distribution of the dependent variable, conditional upon a change in the value of the independent, or causal, variable. The relationship between variables matters because the conclusions drawn from it are counterfactual: in the absence of the cause, all else being equal, the effect would not have occurred. Such a causal argument draws its empirical strength from the regularity of the empirical relationship in question and its explanatory power from the counterfactual nature of the hypothesis.

This is, virtually without exception, the concept underlying the definition of causation in every social science methodology text that was surveyed for this article—at least, those that are empirically oriented: see, e.g., Brady and Collier (2004, *passim*), Campbell and Stanley (1963, 64), Cook and Campbell (1979, 33), Fay (1996, 122), Frankfort-Nachmias and Nachmias (1996, 103–4), Gerring (2001), King, Keohane, and Verba (1994), Little (1991, 19–25), Nicholson (1983, 26), Pratt (1978, 70–71), and Stinchcombe (1968, 31). The contributions in Brady and Collier (2004) and the chapters in Little (1991) do explore an unusually wide range of understandings of causation, though most are compatible with this one (if only, as with deterministic causation, as a limiting case). Studies utilizing a manipulative account of causation (Rubin 1974) typically compare means between matched samples, though in principle nothing precludes the comparison of variances (see Sekhon 2004, 8–9).

The discussion by King, Keohane, and Verba (1994) provides perhaps the clearest illustration. In their example, the causal effect of incumbency on the fraction of the vote received by a candidate for office is at issue, and the “mean causal effect” ( $\beta$ ) is defined as the difference between the means of two distributions—one a distribution of vote fractions across multiple hypothetical elections with an incumbent candidate, and the other a distribution of vote fractions across multiple hypothetical elections with a nonincumbent candidate (76–81). Put more succinctly,  $\beta = \mu_i^I - \mu_i^N$ , where  $\mu_i^X$  denotes the mean of the distribution of the dependent variable under condition  $X$  and the superscripts  $I$  and  $N$  stand for “Incumbent” and “Non-incumbent.”

Focusing on the *distributions* of variables rather than the values of particular observations was a major advance in the philosophy of science. It permitted researchers to analyze the causes of stochastic phenomena—those whose behavior can be analyzed statistically but not predicted precisely.<sup>4</sup> That said, there is no reason to limit our understanding of causal effects to changes in the means of such distributions. Subject to the same counterfactual interpretation, causal relationships may produce changes in variance as well.

A single example illustrates this point. The most famous assertion of the economist John Maynard Keynes (1936) was that the economic booms and busts produced by unfettered laissez-faire capitalism could be smoothed out via government management. By using fiscal and monetary policy to put the brakes on the economy when it was growing too quickly and spending to revive it when it was heading into a slump, Keynesians believed, government could produce relatively steady growth with only minimal deviations. The average rate of growth may well be unaffected; only the variation around that mean would change.

If, in fact, government activity were to alter the variability of economic growth without having an impact on the mean, then the causal effect, as currently understood, of Keynesian intervention would be zero. If we use “I” to denote “intervention,” “NI” to denote “no intervention,” and  $\mu_g$  to denote the average growth rate, the fact that  $\mu_g^I = \mu_g^{NI}$

<sup>4</sup>I am indebted to the *Oxford English Dictionary* (2nd ed.) for this succinct formulation.

would imply no causal effect at all ( $\mu_g^I - \mu_g^{NI} = 0$ ) and hence no causal relationship. As the destitute citizens from previous Depressions would no doubt attest, such an understanding of causation is impoverished.

The obvious remedy is to enrich it. Unlike the means, the variances of the pre- and post-Keynesian distributions of growth rates would differ. Therefore, it makes sense to create another, distinct definition that captures this different understanding of a causal relationship:

A causal effect  $\gamma$  is equal to the difference between the variances of two distributions.

In this case,  $\gamma = \sigma_g^{2(I)} - \sigma_g^{2(NI)}$ .

Not one of the social science methodology texts surveyed for this article discusses such effects or offers a definition of causation that is compatible with them.<sup>5</sup> Nevertheless, as the remainder of this article demonstrates, the causes of changing variance can be modeled in ways that dramatically enrich our understanding of phenomena that interest us.

Happily, few if any new methodologies need be created to accommodate such a program: a diverse, if underutilized, array of equality-of-variance tests, alone or in conjuncture with equality-of-mean tests, already exists. What is lacking, rather, is widespread awareness of the fact that causes, however conceived, can produce changes in the variance rather than in the mean of the distribution of a dependent variable. For that reason, moreover, little effort has been devoted to understanding *how* causes produce changes in variance, i.e., to the general causal mechanisms<sup>6</sup> underlying variance-altering causal relationships. As the following pages demonstrate, absent a clear understanding of these mechanisms the empirical implications of variance-altering causes are difficult if not impossible to derive.

The larger potential implications of this argument—which, unfortunately, exceed the scope of the present article—are, however, considerably more radical. In embracing the distributions of variables rather than single observations as *explananda*, the literature on causation has addressed the need to understand stochastic phenomena, but in so doing it has opened up the notion of a cause to include, potentially, anything that produces a change in any aspect of the shape of a distribution—its skew, kurtosis, number of modes, etc. Given the variety of potential causal effects, it could become very difficult to specify a priori what a causal effect should look like absent strong and well-specified theory. This ontological agnosticism could have profound implications for the philosophy of science literature, which for decades has endeavored to produce a single, all-encompassing understanding of causation. It could also, in the long run, provide a formidable epistemological challenge for many of the more atheoretical, inductive statistical methodologies that are currently in vogue: simply looking for causal effects in the data and sifting them from the artifacts of chance becomes an immensely more daunting task (if not an impossible one) if such effects are conceived of as highly mutable and inherently theory-driven.

<sup>5</sup>King, Keohane, and Verba (1994) do make explicit mention of the variance of the causal effect on page 82; doing so is not the same as asserting, as I will, that in some cases a change in variance rather than a change in mean *is* the causal effect of interest. The fact that King (1989a, 139–40) recognizes changes in variance to be substantively interesting in a practical application underscores the invisibility of this phenomenon.

<sup>6</sup>On mechanisms see Elster (1989) and Hedström and Swedberg (1998), and especially Elster (1998).

### 3 Politics and Variance

Thinking in terms of variance-altering causation opens up a new theoretical dimension, one that has been largely neglected.<sup>7</sup> At present, the concept is absent from discussions of research design and social causation, and only a very small minority of practitioners seem to have utilized it in their work. Those few instances in which practitioners do recognize changes in variance as a substantively interesting phenomenon stand out as isolated flashes of insight.

A full account of variance-altering causation must provide an explication of its logical foundations. Accordingly, in this section I discuss three families of causal mechanisms that can produce changes in variance and highlight their empirical implications. Because many of these mechanisms are central to the study of politics, understanding variance-altering causation as one of the results of their operation is an issue of central importance. In the context of this discussion, I also provide a broad array of examples from across the sub-fields in political science, both to demonstrate the relevance of variance-altering causation and to illustrate the practical possibilities for (and limits of) inference.

#### 3.1 Aggregation

Aggregation permeates politics. Indeed, by some accounts politics *is* aggregation: the substantial literature on public choice, for example, focuses on the thorny question of how political institutions aggregate preferences. Legislative outcomes are modeled as the aggregate of individual actions. Politicians win or lose elections as the result of the aggregated behavior of many voters. Condorcet's famous Jury Theorem relies on aggregation for its result that the majority of a group is more likely to choose the better of two answers than is any one member of the group.

Aggregation often produces distributions; changes in the process of aggregation can produce changes in the variances of those distributions. In the most straightforward case, a distribution of a quantity of interest is, as the result of some policy, augmented by a second distribution, and the variance of the distribution that results is the sum of the variances of the individual distributions, plus twice their covariance. The covariance term implies that even this simple form of aggregation can produce a wide range of effects on variance. A tax rebate could broaden the distribution of income, for example, if everyone received a nonzero dollar amount drawn at random from a distribution with nonzero variance; It could have a narrowing effect if tax rebates are strongly and negatively correlated with income;<sup>8</sup> or it could have no effect at all on the variance of the income distribution, as in the case in which everyone receives the same amount.

The example of income distributions is one with clear implications, as the distributions in question are unbounded. The implications of aggregation become a bit more complex when boundaries, which by their nature limit variance, are introduced. Imagine, for

<sup>7</sup>Although a detailed discussion would stray too far from the central theme of this article, it is nevertheless worth noting in passing that changes in variance are sometimes seen as interesting *independent* variables as well. For example, Alesina and Spolaore (1997) posit that increased heterogeneity of preferences will decrease the optimal size of political units in a region, Aron (1966, 100) argues that homogeneous international systems (those in which states "belong to the same type" and "obey the same conception of policy") are more stable and less violent than heterogeneous systems, and students of interstate and intrastate violence have also examined the hypothesis that inequality of wealth (Muller and Seligson 1987; Sigelman and Simpson 1977) or landholdings (Midlarsky 1988) can be a source of conflict. Stinchcombe (1968, chap. 6) contains a plethora of similar examples. To the extent that understanding the origins of such changes in variance is important, such studies reinforce the argument that a better understanding of variance-altering causation is needed.

<sup>8</sup>More specifically, in cases in which twice the covariance of the income and rebate distributions is greater than the variance of the rebate distribution.

example, that the “currency” in which we are interested is individuals’ evaluations of presidential performance, as measured on a 1–7 scale, and that adding a distribution of positive or negative “messages” (in the form of news reports or campaign advertisements) alters the distribution of evaluation scores. To take the simplest case, if we start off with any realistic prior distribution and flood the individuals with so many positive messages that even the president’s fiercest detractors are converted into ardent supporters, we will have *reduced* the variance of the distribution to zero—a spike at 7—even if the message does not covary with individuals’ initial positions, and even if the variation of the distribution of messages is zero.

Even in the case of simple additive aggregation of distributions, therefore, the hypothesis that aggregation produces an increase in variance is contingent upon both the covariance and the boundaries of the distributions. To see the implications of this point, consider Alvarez and Brehm’s (1995) argument that cognitive dissonance increases variance on survey responses. More specifically, Alvarez and Brehm hypothesize that “individuals who possess strong attachments to both of the underlying core principles” (of women’s rights and the sanctity of life) “should have a harder time making a decision about abortion; hence they should have a greater error variance” (1065). The key to this mechanism is a description of how the variances of distributions cumulate. Here, the assertion is that they do so additively because human beings derive their beliefs, when possible, in a manner consistent with the premises upon which those beliefs are based. The authors support their claim by showing that conflicting core beliefs about the role of women and the sanctity of life produce higher variability in responses to survey questions about abortion than do nonconflicting core beliefs.<sup>9</sup>

The hypothesis of increased variance follows if there is positive (or insufficiently negative) covariation between the two “prior” distributions of opinion—i.e., the contribution of belief in women’s rights to opinions about abortion and the contribution of belief in the sanctity of life to opinions about abortion do not cancel each other out and produce overwhelming moderation—and if the two “treatments” do not pull individuals toward the same (bounded) end of the distribution. The latter condition seems likely to be met in this case, while the former may or may not be: if belief in women’s rights and belief in the sanctity of life pull respondents in opposite directions and the former is a strong negative predictor of the latter (both of which are quite plausible conditionals), the implications for variance would be indeterminate a priori.

Aggregation may also proceed in more convoluted ways. Take, for example, the literature on war as a “costly lottery.”<sup>10</sup> Understanding war in this light sometimes requires modeling warfare explicitly;<sup>11</sup> perhaps the most venerable formal models in this tradition were produced by Frederick William Lanchester.<sup>12</sup> The most straightforward of these implies the so-called Square Law, which posits that the changes in the fighting strengths of two sides (call them Red and Blue) will follow a simple pattern:  $\frac{dB}{dt} = -rR$  and

<sup>9</sup>They do so utilizing heteroskedastic ordered probit. See also Alvarez and Brehm (1997, 1998) and Alvarez, Brehm, and Wilson (2003). For research that explores the inferential utility of the statistical techniques used to test these assertions, see Glasgow (2005). External events, too, can prompt changes in variance of beliefs within a group: Matthew Gabel (1998, 11) hypothesized that context-specific factors like differential press coverage could introduce unequal error variance across the respondents in his multiple-country, multiple-year study of levels of support for membership in the European Union, and Yuchtman-Yaar and Hermann (1998) found that the assassination of Yitzhak Rabin produced a marked and lasting decrease in variance in those attitudes of Israelis regarding the legitimacy of various forms of protest.

<sup>10</sup>See, e.g., Fearon (1995) and Wagner (2000).

<sup>11</sup>See, e.g., Smith and Stam (2004) and Grigorian (2006).

<sup>12</sup>See Taylor (1983) for exhaustive details.

$\frac{dR}{dt} = -bB$ , where  $R$  [ $B$ ] represents the strength of Red's [Blue's] forces at a given time and  $r$  [ $b$ ] represents the effectiveness of Red's [Blue's] fire on Blue [Red]. In this form, the Lanchester equations are deterministic: Red will defeat Blue if and only if  $rR^2 > bB^2$  at time 0.

It is possible, however, to add a stochastic element by modeling  $r$  and  $b$  not as constants but as distributions. Pursuing this line of thought, Lepingwell (1987, 130) has noted that increasing the number of combatants on a given side should decrease the variance of the distribution of the effectiveness. Variation in the distribution of effectiveness of the Red army alters the rate at which the Blue army's forces succumb to attrition in battle (and vice versa), but the distribution of battle outcomes depends in a complex and nonlinear way on the variances of those distributions as well as on their means and on the initial strengths of the two armies. The upshot is that once relative size and (average) effectiveness have been controlled for, smaller armies should experience greater variation in combat outcomes than larger ones.<sup>13</sup> Here, the mechanism by which aggregation takes place is precisely elaborated: individual soldiers shooting at one another, with varying degrees of success, produce victory or defeat in the aggregate.

Theories of aggregation can be based on more complex assumptions about human behavior, as, for example, in the public choice literature.<sup>14</sup> Much of the discussion of voting in multiple dimensions centers on the question of the effects of various assumptions on behavior. For example, the argument that strategic legislators will choose positions that can either defeat an opponent's position directly in majority voting or defeat some third position that can in turn beat the opponent's position in majority voting has led researchers to focus on the collection of positions that fulfill this criterion: the "uncovered set," a relatively small region near the multidimensional median (Miller 1980). McKelvey (1986) has proved that the uncovered set will always lie within the "yolk," a circle of radius  $4r$ , where  $r$  is the radius of the smallest circle that intersects all lines that divide the issue space in such a way that no more than half of the ideal points are on either side (the "median lines"). The size of the yolk, and therefore the variance of the set of potential outcomes, varies with the shape of the distribution of ideal points: typically, the similarity of the most centrist voters (that is, the proximity of the ideal points of those voters adjacent to the median along each dimension) has the greatest impact on the size of the yolk.

### 3.1.1 Redistribution

In a dynamic setting—that is, one in which the distributions of interest are the result of flows of goods and political actors can exert some control over the direction and magnitude of those flows—changes in the rate of aggregation, rather than aggregation per se, become the phenomenon of interest. These changes generally fall under the heading of "redistribution."

<sup>13</sup>Lepingwell does not explain precisely how variance in efficacy translates into variance in success, but the proof is relatively simple. Lanchester's Square Law states that Red will defeat Blue if and only if  $rR^2 > bB^2$  at time 0. When all of the parameters are constants, the probability of victory is either zero or one. If, in any given battle, instead of a constant  $r$  we have  $r$  plus a draw from a standard Gaussian error term, i.e., a Gaussian probability density function with mean  $r$  and variance  $\sigma^2$ , then the probability that Red will lose becomes the probability that  $(r + \varepsilon)R^2(0) > bB^2(0)$ , or equivalently that  $(r + \varepsilon) > \frac{bB^2(0)}{R^2(0)}$ , which equals  $\int_{-\infty}^{\frac{bB^2(0)}{R^2(0)}} \frac{1}{\sigma\sqrt{2\pi}} e^{-\frac{1}{2}(\frac{r+\varepsilon}{\sigma})^2} dx$ . If for convenience we refer to this probability as  $p$ , the distribution of wins and losses (for Red) in  $n$  battles has an expected variance of  $np(1-p)$ , which is maximized at  $p = 0.5$ . Intuitively, increasing  $\sigma$  pushes  $p$  away from 0 (or 1) and toward 0.5, thereby increasing  $np(1-p)$ .

<sup>14</sup>See Mueller (1997) for a review.

Redistribution is one of the fundamental functions of any political system; indeed, it is a necessary part of the social contract.<sup>15</sup> The term “redistribution” implies both that a distribution (of something) exists and that action is taken to alter it. The result is typically a change in one or more of the moments of the distribution, possibly including the variance. In the case examined by Hall (1986), for example, the advent of Conservative rule brought to power a group of people whose preference was less for social leveling and more for *laissez faire* than was that of their predecessors. As a result, the rich became richer, the poor became poorer, and the variance of the income distribution increased.

The redistribution of wealth, either directly via taxation or indirectly via provision of education, unemployment insurance, welfare benefits, “pork-barrel” politics, and so on, is a useful illustration of redistribution both because of its concreteness and because of the attention that it has received in recent scholarship. Acemoglu and Robinson (2000) argue that the history of political regime formation has important implications for economic redistribution: narrower European distributions of wealth reflect the fact that the institutions of today’s European democracies were forged in an ongoing struggle between rich and poor to a much greater degree than those of the United States. Another explanation, offered by Persson, Roland, and Tabellini (2000, 1126), is that form of government matters: proportional representation provides politicians with fewer incentives for thrift, and one result is a greater degree of redistribution. Alesina, Glaeser, and Sacerdote (2001) disagree with both conclusions, arguing instead that Americans reject greater redistribution because it would disproportionately benefit racial minorities.<sup>16</sup> Bénabou (2000) favors a more elaborate model of growth in which the conditions of capital markets, insurance markets, and credit constraints combine to form either a situation in which support for redistribution increases with equality or a situation in which lower redistribution produces higher inequality. Finally, the preferences of the citizens may play a critical role in redistributive politics: although most of the theories listed above are grounded on the assumption that the poor themselves would rationally favor a policy of economic equality, this assumption has been called into question both in detailed interviews<sup>17</sup> and in experimental settings.<sup>18</sup>

These examples illustrate the fact that redistribution is typically the result of the interaction of multiple actors with different distributional preferences and different abilities to bring about or prevent change, often acting within the context of preexisting and historically contingent political institutions. Changes in any part of this complex mechanism could bring about changes in the variance of the distribution in question.

### 3.2 *Contagion or Diffusion*

The general argument behind contagion (sometimes called diffusion) is that instances of a given phenomenon—whether they be wars, riots, currency crises, or even the presence

<sup>15</sup>For discussions of the origins of social contract theory see Morris (1999); for the most noteworthy recent discussion see Rawls (1971).

<sup>16</sup>Gilens (2000) makes a similar argument but emphasizes the media’s proclivity for “racializing” discussions of welfare.

<sup>17</sup>Hochschild (1981) demonstrates that, while the poor respondents in her sample support egalitarian norms in the social and political sphere, they largely do not seek equality in the economic sphere. Some see redistribution as antithetical to the liberal ideal of competition, others consistently support inegalitarian norms, and still others either cannot imagine that substantial redistribution would actually take place or are indifferent toward it.

<sup>18</sup>Rawls’s assertion that, under conditions of high uncertainty about the future, people would choose to maximize the welfare of the least well-off in society (whose ranks they might soon populate) has been questioned in experimental studies. Both Frohlich, Oppenheimer, and Eavey (1987) and Lissowski, Tyszka, and Okrasa (1991) find that maximizing average income with a floor constraint, rather than maximizing the welfare of the least well-off, is the preferred choice, while Scott et al. (2001) find considerable evidence for complex norm-driven behavior.

of female candidates on party ballots<sup>19</sup>—have a way of sparking other instances of the same phenomenon. The phenomenon in question therefore appears to be spreading like a disease: later instances are made more probable by the occurrence of earlier ones. Negative contagion, by contrast, occurs when later instances are made *less* probable by the occurrence of earlier ones.

While the specific causal mechanisms that produce political contagion are remarkable for their variety, the general mechanism of contagion is clear: actors respond to some compulsion, opportunity, or incentive to become more (or less) similar to one another along at least one dimension. Simmons and Elkins (2004) point to two mechanisms that create contagion in states' adoption of liberal economic practices: an action by or in one state at time  $t$  can either alter other states' payoffs or convey information to them in such a way that they become more (or less) likely to take a similar action at time  $t + 1$ . These mechanisms generalize to within-state contagion as well: Matland and Studlar (1996), for example, argue that the spread of female candidates in PR systems occurs because experience will prove that forwarding female candidates increasingly results in electoral rewards rather than penalties (information) and because forwarding female candidates makes other parties vulnerable to charges of inequity in representation (payoffs). Seeking a larger array of more specific mechanisms, Levy (1982) describes no fewer than thirteen very specific causal mechanisms—desire to regain lost territory or goods; opportunistic attacks on a protégé when its defender is embroiled in conflict elsewhere; defense of an ally; war weariness; and so on—that historians have implicated in specific instances of war contagion.

Despite the straightforward nature of the general mechanism of contagion, deriving its implications may be quite difficult. The aggregate empirical implication of simple contagion in the case of count variables is straightforward: the accumulation over set intervals of time of events that occur purely by chance—accidents per month, for example—should give rise to a Poisson distribution, in which the mean is equal to the variance, but if the events are contagious (if some people are more accident prone than others), the observations toward the upper end of the distribution will take on even greater values than they would by chance, and the distribution will be overdispersed. Similarly, negative contagion (accidents put people in hospitals, where they are for a time less likely to have accidents) produces underdispersion.<sup>20</sup> These implications were utilized in the rather extensive war-contagion literature to demonstrate that, in the aggregate, outbreaks of new wars do not exhibit symptoms of contagion but the spread of existing wars does.<sup>21</sup>

<sup>19</sup>See Most and Starr (1980) and Kadera (1998) for discussions of the logic of war diffusion. On riots, see Govea and West (1981); Midlarsky (1978). The literature on currency crises is immense, especially since the Asian financial crisis; see Agnor et al. (1999) and Glick and Rose (1999) for examples. And on the presence of female candidates, see Matland and Studlar (1996).

<sup>20</sup>In cases of overdispersion, negative binomial regression has been used, which permits the degree of overdispersion to be estimated; generalized negative binomial regression, a.k.a. a negative binomial variance function model, permits the degree of overdispersion to be modeled as a function of a vector of independent variables, as in heteroskedastic regression (see, e.g., Martin [1994]). See also the generalized event count model designed by King (1989b), as well as the symposium on the GEC in Freeman (1996). Explicitly modeling spatial independence at the level of the individual state is mathematically more difficult due to the interdependence of the observations; see Ward and Gleditsch (2002) for a recent application in which the authors solve the problem of analytic intractability using Markov chain Monte Carlo techniques.

<sup>21</sup>See Simowitz (1998) for a thorough summary and assessment of this literature. More elaborate models are necessary for multivariate count data, such as vote counts; Katz and King (1999), Jackson (2002), and Tomz, Tucker, and Wittenberg (2002) introduce vote-count models that permit overdispersion, and Mebane and Sekhon (2004) offer a robust estimator, the hyperbolic tangent estimator, that provides accurate parameter estimates even in the face of very substantial over- or underdispersion.

With categorical data, however, the implications of contagion are more complex. Even the simplest model of contagion—the so-called susceptible-infected-retired (SIR) model, in which a population is exposed to an infectious agent to which some of its members succumb<sup>22</sup>—has a nuanced impact on variance. In the short run, variance increases as the population moves to diverse categories, but in the long run, variance decreases as the last of the uninfected succumb. Political examples that conform to this logic might include Simmons and Elkins's (2004) discussion of liberalization: if the liberal model is successful, economic orientation might become more varied in the short run but less varied in the long run. The implications for variance depend on the shape of the initial distribution, that is, on how many countries have already been “infected” at the first time period under study.

The effects of contagion or diffusion on variance are also a function of the correlation, if any, between the strength of the contagious phenomenon and the state's location in the distribution. Here is where the caveats introduced by Martin and Simmons (1998) and Moravcsik (1995) on the relationship between institutionalization and variance become relevant. While one might, in general, expect institutionalization to produce a decrease in variance (in, say, compliance) across states, there is no guarantee that this outcome will come to pass. If high-compliance states converge to a still-higher standard while low-compliance states do not, for example, the effect of institutions might be an increase rather than a decrease in variance. Also, the shape of the distribution prior to the initiation of the effect matters: some curious distributions—for example, one with hypercompliant states that would have to *decrease* their degree of compliance to comport with the institutional norm, or a bimodal distribution of perfectly compliant and perfectly noncompliant states with one or two states on the noncompliant side of the mean—could conceivably produce a net decrease in variance. Without an explicit model of compliance and an understanding of its impact on the prior distribution of cases, the implications for variance are impossible to derive.

In time series models, contagion has even more convoluted implications. In the case of currency crises, for example, intuition would suggest that contagion implies a decrease in the variance of the distribution of changes in the value of national currencies: rather than fluctuating normally, all currencies would plunge together. However, periods of extreme volatility imply larger changes, *ceteris paribus*, than periods of lesser volatility, so the variance-narrowing effects of contagion might be swamped by the variance-widening effects of volatility.<sup>23</sup>

All in all, as this discussion suggests, researchers hoping to derive the implications of contagion or diffusion for variance must take into account the period of time under examination (e.g., contagion may produce an initial increase in variance followed by a decrease), the shape of the initial distribution, the relative position of the point toward which actors are drawn, and the magnitude of the change produced by contagion (large changes, especially toward points outside of the initial distribution, could increase rather than decrease variance) if they are to offer concrete predictions.

<sup>22</sup>The model was originally described in Kermack and McKendrick (1927), though the mnemonic name was applied later. The model is as follows:  $\frac{dS}{dt} = -IS\kappa$ ,  $\frac{dI}{dt} = IS\kappa - I\lambda$ , and  $\frac{dR}{dt} = I\lambda$ , where  $\kappa$  is the parameter that captures the virulence of the “disease” and  $\lambda$  captures the rate of removal (i.e., recovery—and hence immunity—or death).

<sup>23</sup>Teasing out the implications of contagion for time series models is, at this writing, a growth industry, and no single test has been established. Rigobon (2001) reviews and proposes alternatives; Bekaert, Harvey, and Ng (2003) suggest examining correlations among idiosyncratic shocks in a time series model with three error terms.

### 3.2.1 Tipping

One possible result of contagion or diffusion is *tipping*, a phenomenon that occurs when an outcome—typically something with two possible conditions, such as war or peace, passage or failure of a law, etc.—is influenced or reversed by changes in the preferences or beliefs of those capable of influencing it. Tipping explanations must involve explanations of how these changes came about and the aggregation mechanism that is “tipped” when they do change.

The literature on revolution in repressive societies provides a nice example of how such an explanation can unfold over time. Kuran (1991) examines the shocking phenomenon of the East European revolutions of 1989 and concludes that a process of “preference falsification,” whereby East Europeans feigned acceptance of their political systems to such a degree that no one was capable of accurately gauging the preferences of other citizens, was responsible for the unexpected eruption of support for liberalization, once the spark had been lit. Lohmann (1994) discusses a model of “informational cascades” in which revolution occurs because dissatisfied citizens protest and other citizens gain information about the quality of the regime from the size of the protests. Nonparticipants subsequently decide whether or not to take part, and if a sufficient number of protesters do so, the regime “tips,” or fails to maintain sufficient support to survive. Her examination of the Monday demonstrations in Leipzig, East Germany, suggests just such a process. Finally, Ginkel and Smith (1999) offer a game-theoretic formulation that incorporates these insights and models regime action in a more strategic fashion.

### 3.3 Constraint

Another family of causal mechanisms that can produce changes in variance has to do with constraints. In the simplest case, theorists argue that constraints on political behavior exist and can change under certain circumstances. Such theories typically lack any explanation of how variance in behavior is produced in the first place, and as a consequence it is difficult to assert with confidence that a change in constraints will produce a change in the variance of the distribution of outcomes. Nevertheless, such predictions can typically be made if a small number of ancillary assumptions are likely to hold, and such assumptions are often inoffensive.

Still, a better solution would be to specify the mechanism that produces variation: the two other categories examined here—optimization subject to constraint and evolution—do just that and are therefore more able to produce concrete predictions.

#### 3.3.1 Simple Constraint

Theories of political behavior occasionally focus not on the sources of variation in the behavior in question but rather on factors that constrain variation. The exigencies of life in the international system, according to Waltz (1979, 73–74) place limits on the behavior of states; the internal structure of the state, according to Snyder (1991, 54, 311) constrains democratic leaders but leaves dictators unfettered.

Constrained behavior is typically anticipatory behavior. States are constrained from total disarmament for fear that an opportunistic adversary will take advantage. Chiefs of government are constrained from concluding agreements that their legislatures will not ratify. Democratic politicians are constrained from unpopular behavior because of the impact it would have on them and their successors. Rebels will prefer to rise up against the state when it is weak, not when it is strong Skocpol (1979). And so on.

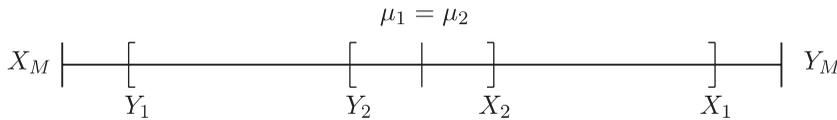


Fig. 1 Hypothetical two-level game.

From the point of view of causation, then, the interesting question becomes, what sorts of anticipated outcomes constrain behavior? The easiest answer is “suboptimal ones”—but it is also the worst answer, at least from the point of view of analytic utility. If we permit it, optimization subject to constraint becomes optimization subject to optimization, and “constraint” becomes a meaningless construct: I choose chocolate because I am constrained (by my preferences) from choosing vanilla.<sup>24</sup>

It is more useful, instead, to view constraints as circumstances external to the actor that rule out options that might otherwise have been taken. Resource constraints are a good example, often used in models of household spending but applicable to some of the answers given above (rebels are constrained from outright rebellion by their relative lack of resources vis-à-vis the state). Another constraint, well known to game theorists, is that created by the preferences of another player and the structure of their interaction: if your dominant strategy in a one-shot two-by-two game is to defect, I will never achieve any of the payoffs that I would obtain if you were to cooperate, and I am therefore constrained to choose between the remainders.

The two-level games literature, going back to Putnam’s (1988) seminal article, provides an ideal example of simple constraint in which the permissiveness of domestic coalitions within two states determines the range of possible bargaining outcomes between chiefs of government at the international level. Figure 1 displays two scenarios. In the first, both chiefs of government are afforded substantial leeway by their domestic constituencies, perhaps as the result of domestic fragmentation or indifference. This situation is depicted as a large gap between the two countries’ maximum, or most preferred, outcomes ( $X_M$ ,  $Y_M$ ) and the minimum outcomes that could be ratified ( $X_1$ ,  $Y_1$ ). The distribution of possible bargaining outcomes is therefore bounded by  $X_1$  and  $Y_1$ . In the second scenario, each country’s constituency has restricted its set of acceptable outcomes considerably; as a result, only agreements between  $X_M$  and  $X_2$  would be ratified in state X and only those between  $Y_M$  and  $Y_2$  would be ratified in state Y. This new set of constraints would produce a distribution of possible bargaining outcomes bounded by  $X_2$  and  $Y_2$ .

Here, chiefs of government are constrained from arriving at agreements that their legislatures would vote down but are free to arrive at any agreement outside of that region. As the constraints imposed by the legislatures relax, the distribution of bargaining outcomes is free to expand. Whether it does so or not, however, is indeterminate, because nothing in the theory describes how the characteristics of the chief of government (or the legislature or the state) will have an impact on where within those constraints the outcome will be located. For this reason, the essays on two-level games in Evans, Jacobson, and Putnam (1993) typically examine only the issue of whether agreements were possible or impossible in light of domestic constraints; more detailed predictions are not to be had.

<sup>24</sup>That is not to say that constraints cannot be *brought about* by optimizing behavior: actors may choose to constrain themselves because doing so may preclude suboptimal options. This sort of “hand tying” is evident, for example, in the decision of Italy, which has historically had high rates of inflation, to enter the European Monetary Union, an international institution that constrains inflation rates (Martin and Simmons 1998). The possible causal connection should not, however, blur the analytical distinction.

These mechanisms demonstrate that constraint in and of itself is underdetermining absent some form of theory about how actors behave within the limits of their constraints. Household spending models and game-theoretic models posit *optimization* subject to constraint, a mechanism that will be taken up in the next section. Evolutionary models posit some manner of stochastic behavior within the boundaries set by constraints, a mechanism that will be taken up in the section after that. But absent some theory at the level of the agent, constraint tells us very little.

Nevertheless, we *could* introduce a few simple assumptions in lieu of a detailed theory. Determinate predictions about variance would follow as long as the assumptions were consistent with whatever process actually produces variation. For example, the following set of assumptions ensures that increased constraint implies a decrease in the variance of the distribution of possible outcomes:

1. In at least one case, interaction under looser constraints would have produced an agreement outside of the range bounded by the tighter constraints.
2. In all cases described by assumption 1, the outcomes either are nonevents (i.e., no agreement in a two-level game) or are closer to the mean of the distribution of observed events, on average, than the original outcomes would have been.
3. In all cases not described by assumption 1, the presence of constraints does not alter the outcome.

The first requirement ensures that the tightened constraints are relevant in some cases: if, for example, chiefs of government were always to arrive at an agreement at  $\mu$  in Fig. 1, the variance—zero—would be the same regardless of the tightness of constraints. The second requirement describes what happens to positions or agreements that are precluded by tighter constraints. Imagine, for example, that the constraints in Fig. 1 were  $Y_2$  and  $X_2$ , but the vast majority of outcomes fell in the middle of the spectrum, between  $Y_2$  and  $X_2$ . This condition states that two chiefs of government who would have preferred an agreement just to the left of  $Y_2$  cannot instead choose an agreement at  $X_1$ . If their behavior is perverse enough to permit such an outcome, no narrowing of variance can be ensured. Finally, the third condition simply ensures that the rest of the distribution of outcomes—those that fall within the tight boundaries—remains the same regardless of whether boundaries are loose or tight.

These assumptions are, as assumptions go, relatively inoffensive. Nevertheless, a better solution would be to model the sources of variation within constraints. The approaches described in the next two sections do just this.

### 3.3.2 Optimization Subject to Constraint

When agents are assumed to choose the best from among the available set of options, the result is optimization subject to constraint. Here, the indeterminacy of simple models of constraint is resolved by positing a second mechanism that specifies how the actors in question will behave given the constrained set of options available to them.

How do agents select the “best” option? By far the most common mechanism posited as a source of optimizing behavior is rationality: an individual, group, or state faced with multiple actions will choose the one with the highest expected utility.<sup>25</sup> There is no reason, however, that other mechanisms could not provide criteria by which an action

<sup>25</sup>Given the recent prominence of rational choice theory, I need spill little ink in discussing this mechanism further; see Friedman (1996) for a discussion of sundry strengths and weaknesses.

would be deemed optimal: maximization of a function, like minimization of the sum of squared vertical distances to a regression line, lies in the realm of methods, not theory. One option of a set might be deemed optimal not because of its consequences but because of its appropriateness in a particular context, or because of its appeal given the actor's normative predispositions, or because it most closely comports with the actor's identity.<sup>26</sup> In practice, however, the theoretical literature that utilizes maximization subject to constraints to derive results is overwhelmingly rationalist.

How might optimization make implications for variance more clear in the example described above? Posit that chiefs of government engage in a standard Rubinstein bargaining game of alternating offers (Rubinstein 1982) within the limits imposed by their legislatures. The main result of the Rubinstein model, in short, is that patience pays: the higher one's discount factor (and the lower one's opponent's), the closer the outcome will be to one's ideal point.<sup>27</sup> A two-level game model in which each of the chiefs of government uses the other's legislature's worst permissible outcome as an ideal point<sup>28</sup> before engaging in bargaining would produce a straightforward hypothesis about variance across multiple cases: as long as the ratio of A's discount factor to B's varies across cases, a given set of bargaining situations would produce a distribution of outcomes with a variance that increases as the bargaining space widens.<sup>29</sup>

George Tsebelis (1999) offers another interesting example of optimization subject to constraint: preference aggregation in legislatures. Tsebelis examines the effects of large coalition governments on policy stability, conceptualized as the ability of the government to pass "significant" laws. His theory implies that a small ideological range among the most extreme parties is necessary but not sufficient for the passage of a large number of such laws: governments with little ideological distance among extreme parties may pass quite a few or none at all, but the number passed by highly polarized governments will be vanishingly small. Hence his second hypothesis—that the "ideological range of a coalition negatively affects the variance in the number of significant laws."<sup>30</sup> This argument, too, relies on constraint for its power, though in this case constraint is endogenous to the theory: increasing polarization among veto players puts ever-lower limits on the amount of cooperation that can emerge from the process. Here, constraint is coupled with an aggregation mechanism, a variant on the spatial model of voting (Enelow and Hinich 1984), with the distinguishing feature that some players can, if sufficiently dissatisfied with a proposed outcome, ensure that no outcome will result.

<sup>26</sup>For good recent reviews of the literatures on rational choice theory, the logics of consequences and appropriateness, norms, and constructivism, all in one convenient package, see Milner (1998), March and Olsen (1998), Finnemore and Sikkink (1998), and Ruggie (1998), respectively.

<sup>27</sup>The terminology is a bit confusing: a discount factor is the constant  $\delta$  ( $0 \leq \delta \leq 1$ ) by which one's payoff is discounted from one period to the next. So a high discount factor does *not* mean that one's payoff is discounted dramatically from one period to the next—quite the opposite. In the Rubinstein game, if A's discount factor is  $\delta_A$  and B's is  $\delta_B$ , the equilibrium will be a point  $\frac{(1-\delta_A) \times 100}{1-\delta_B}$  % of the distance from A's ideal point to B's.

<sup>28</sup>In terms of Fig. 1, the COG of X would choose  $Y_1$  or  $Y_2$ .

<sup>29</sup>An alternative approach, with similar implications, would be to incorporate both uncertainty and utility maximization into the game; see Iida (1993).

<sup>30</sup>This sort of relationship is more complex than an equality-of-variance test envisions, but it can be modeled with what Harvey (1976) calls a *multiplicative heteroskedasticity* model, which can be adapted to a wide range of circumstances. Just as the mean is typically modeled in standard regression,  $Y = \beta X + \varepsilon$ , where  $\beta$  is a vector of parameters to be estimated and  $X$  is a vector of independent variables, so we can model the variance:  $\sigma^2 = e^{\gamma Z}$ , where  $Z$  is the vector of independent variables that cause a change in the variance of  $Y$  and  $\gamma$  is the vector of parameters to be estimated. One might also envision quantile regression for such an application (Buchinsky 1998), given that the variances of some distributions are not particularly well behaved. Alternatively, in a standard regression model in which the variance is thought to be a function of a single independent variable, simple weighted least squares could be utilized (Greene 2000, 512–13).

### 3.3.3 Evolutionary Dynamics

In contrast to optimization subject to constraint, evolution might be called “randomization subject to constraint.” Spruyt (2001, 110) lists a number of examples:

Some of the most influential literature in political science suggests that international competition will lead to high degrees of uniformity in institutions and similarity of policies. Rivalry in security affairs will weed out those states that are too feeble to defend themselves or that engage in policies that jeopardize their own security. Globalization and economic competition will relegate inefficient nations to secondary status. . . . Less competitive forms of government and less successful economic policies will be selected out. The result will be institutional convergence.<sup>31</sup>

Evolutionary mechanisms typically comprise two defining processes. The first of these is internal: over time, the distribution of one characteristic of a group has a tendency to spread, typically as a result of some internal trait of the entities that comprise the group (random genetic variation being the exemplar). The most straightforward version of a process of this sort might be a simple random walk. The result of this process, if unimpeded, is a general increase in variance over time. MacKuen, Erikson, and Stimson (1989) (both initially and in response to Green, Palmquist, and Schickler [1998], in Erikson, MacKuen, and Stimson [1998]) argue that macropartisanship in the United States follows just such a pattern—and that, as a result, the cross-sectional variance in party identification increases with the age of the cohort.<sup>32</sup>

The second process is typically environmental rather than internal, and processes that fall into this category mitigate (and sometimes reverse) the increased variance that results from the first. These constraining processes may involve selection, socialization, learning, or any number of phenomena that mimic these effects. The effects, simply, are to alter the characteristics of individuals by subjecting them to conditions under which only a certain range of realizations of those characteristics—long or short beaks for finches, sooty coloration for peppered moths, or realpolitik behavior for states—is desirable, viable, or possible.

Regardless of the source of change over time, environmental processes take on a few characteristic forms. One is a *drift*, a situation in which selection pressures push the individuals in question farther along the given dimension over time. Another environmental element that can be relevant is a *barrier*, an upper or lower limit to variation.<sup>33</sup> It is also possible that a combination of optimization and randomization is at work: individuals in later time periods or generations, driven by one of the optimization mechanisms described

<sup>31</sup>Spruyt’s list is by no means complete. One of the more interesting literatures involving the role of evolution in politics is one to which Spruyt himself has contributed: that on the process by which variation in forms of governance since the Middle Ages decreased until the modern nation-state emerged as the political unit of choice. Spruyt (1994) posits three evolutionary engines—economic, sociological, and competitive—as the sources of change. Other prominent accounts in the same body of literature include Tilly’s (1990), which emphasizes military competition, and the complex adaptive systems approach of Cederman (1997), who focuses on the role of the offense-defense balance and the proportion of non-status-quo political entities in the system. Cederman and Gleditsch (2004) formalize an evolutionary model of regime change with a transition rule based on the regime types of states in the immediate vicinity; the result is an increase in the variation of regime type over time.

<sup>32</sup>Indeed, it is well known that econometric work relating to the question of whether a process has a unit root (i.e., follows a random walk such as the one just described) reflects a fundamental concern with the statistical properties of a process with ever-increasing variance, such as the one at the top of Fig. 2. What is less well understood is that the nature of such processes has substantial implications for our understanding of causation itself: Hoover (2003), for example, uses the fact that statistical tests misrepresent dependencies among non-stationary data to undermine a criticism of Reichenbach’s principle of the common cause.

<sup>33</sup>Gould (1996, 54–55) refers to these as “walls.” See also Goertz (1994).

above, are drawn toward some *optimal point*, while randomization continues to occur. These processes are illustrated in Fig. 2.<sup>34</sup>

The result of the combination of internal and environmental processes may be convergence *or* divergence (implying an increase or a decrease in variance), depending on which prevails.<sup>35</sup> It may even, as with the case of a drift toward a barrier illustrated in Fig. 2, produce an increase in variance followed by a decrease.

Evolutionary examples from the political science literature have a tendency to focus on the environmental process rather than on the adaptive or randomizing one. Doing so only tells half of the story.<sup>36</sup> Without understanding the sources of divergence in states' abilities to defend themselves, or in economic efficiency, or in economic policies and forms of government, we cannot predict whether evolutionary dynamics should lead to a decrease or an increase in variance—or to no change at all. These examples demonstrate that the theorist must often be quite precise about the internal process, the environmental process, and how far the system has already “evolved” before anything concrete can be said about the effects of evolutionary processes on variance.

### 3.3.4 Punctuated Equilibrium

The concept of punctuated equilibrium has on occasion been borrowed from the literature on paleobiology and applied to political science. The analogy is applied to situations in which a variable measuring some dimension of the phenomenon of interest, after changing little for a long period of time, experiences an abrupt and lasting shift to a different value or range of values. This shift is typically accompanied by an increase in variance as the value of the variable seeks its new level, followed by a decrease in variance once it finds it.

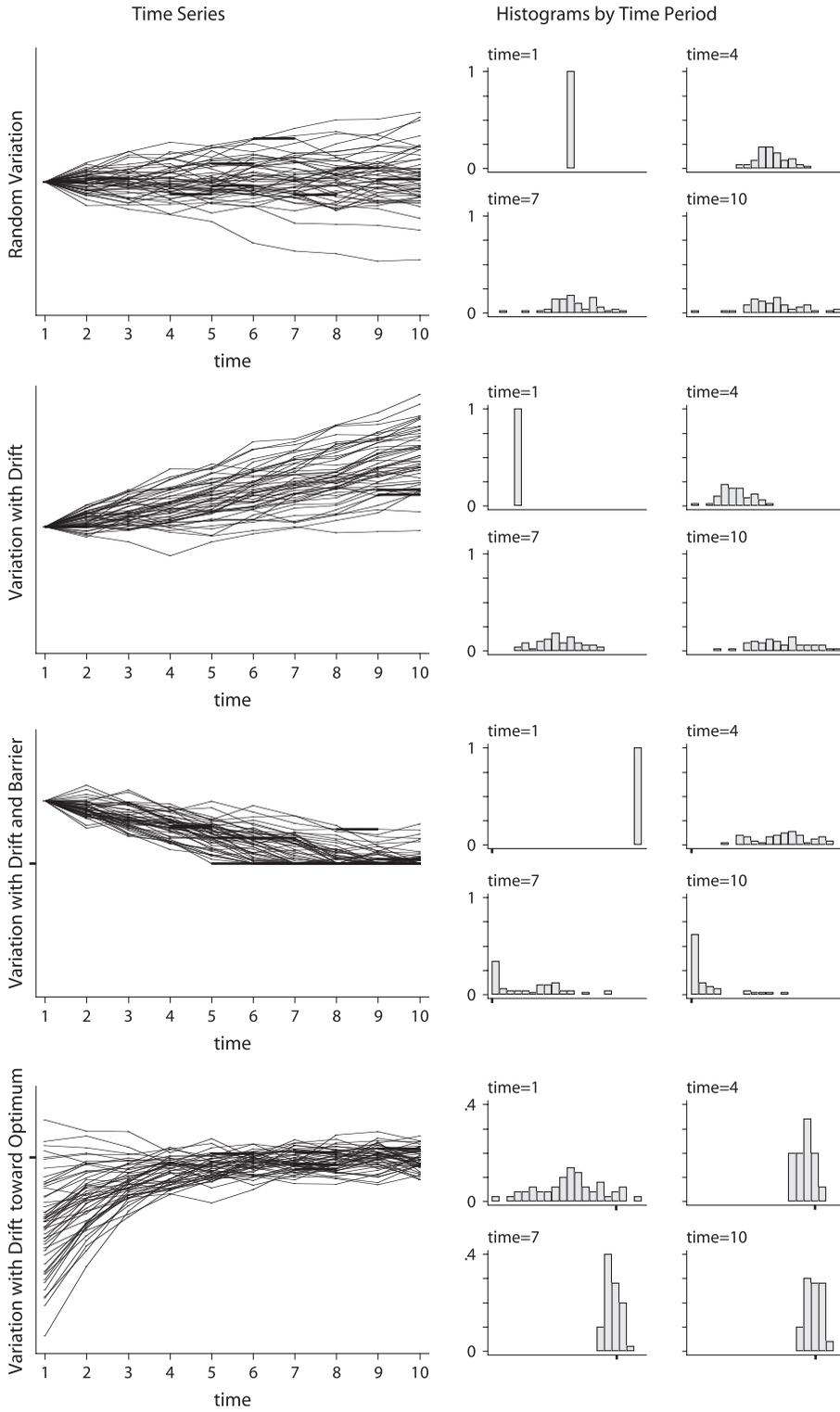
At least two mechanisms produce such an outcome. In paleobiology, it is argued, phenotypical variation occurs first in small, isolated groups. Only after those groups have substantially evolved to take advantage of their local circumstances do they spread elsewhere and prosper. This process creates the illusion that new species appear abruptly, *sui generis*, in the fossil record without any indication of a transition from the old species (Eldredge and Gould 1972).

Although this mechanism might translate into the political realm in some interesting ways, the mechanism most utilized in political science is rather different. In political science, “punctuated equilibrium” typically describes a situation in which structural variables shift to create a new optimum, and people must scramble to adjust to it. This is essentially a case of optimization subject to constraint, with changing constraints. For the prediction of increasing and then decreasing variance to obtain, the theory must also explain why the individuals in question fail to optimize immediately; typically, some form

<sup>34</sup>Random variation is modeled quite simply as  $Y_{t+1} = Y_t + \varepsilon$ ,  $\varepsilon \sim N(0, 1)$ . Variation with drift is modeled by adding a constant,  $c$ , at each time period:  $Y_{t+1} = Y_t + c + \varepsilon$ ,  $\varepsilon \sim N(0, 1)$ ,  $c = 1$ . A lower bound or wall  $Y_w$  is incorporated so:  $Y_{t+1} = Y_t + c + \max(\varepsilon, (Y_w - Y_t))$ ,  $c = -1$ ,  $\varepsilon \sim N(0, 1)$ ,  $Y_w = 0$ . Note that the resulting distribution is skewed. If  $c$  were positive and the drift were therefore toward an upper bound,  $\min(\varepsilon, (Y_w - Y_t))$  would be used instead. Drift toward an optimal point  $Y_p$  with fractional rate of adjustment  $\beta$  is modeled as  $Y_{t+1} = Y_t + \beta(Y_p - Y_t) + \varepsilon$ ,  $\varepsilon \sim N(0, 1)$ ,  $\beta = 0.4$ ,  $Y_o = 7.5$ . In the lower two series, the barrier and the optimum, respectively, are denoted by horizontal lines.

<sup>35</sup>It may, in fact, be quite difficult to determine which trend is prevalent. Movement of a distribution along a given dimension could be driven by a drift or produced by purely random variation in proximity to a natural lower bound. McShea (1993, 1994) describes and illustrates three tests useful in such situations. The first is based on the behavior of the minimum of the distribution, the second involves a longitudinal study of the direction of variation of offspring from parents, and the third involves examining the skew of the distribution of a random subsample of cases. The latter test evaluates the only hypothesis of which I am aware that is based on the *third* moment of a distribution.

<sup>36</sup>Spruyt (2001).



**Fig. 2** Changes in variance as a result of evolutionary dynamics.

of partial randomization, costliness, feedback, or learning under conditions of incomplete information are incorporated, at least implicitly.

Though examples abound, two political science literatures are especially noteworthy for having relied on punctuated-equilibrium explanations. The first is the literature on enduring rivalries. One of the earliest works on this subject, by Goertz and Diehl (1995), suggests that environmental “shocks”—world wars, territorial changes, and changes in the balance of power, for example—are very nearly necessary (though not sufficient) conditions for the termination of a rivalry. The analogy was made more explicit by Cioffi-Revilla (1998), who demonstrated that rivalries typically experience three distinct phases, which he calls lock-in, maturation, and termination, and that variability in the hazard rate, or probability of termination, increases significantly from one phase to the next.<sup>37</sup>

Theories of the state also make use of the punctuated equilibrium analogy. For example, Jones, Baumgartner, and True (1998) demonstrate that postwar congressional budget authority over discretionary domestic spending can be broken into three phases, with breaks at 1956 (the beginning of the Eisenhower “peace dividend” period) and 1976 (the first impact of the Congressional Budget and Impoundment Control Act of 1974). Their data demonstrate both that the beginning of each period is marked by high variance in the annual percentage change in the discretionary budget and that that variance decreases over time.<sup>38</sup>

#### 4 Conclusion

This article has attempted to make the case for the importance of an underappreciated phenomenon: the variance-altering cause, one that produces a change in the variance rather than the mean of the distribution of the dependent variable. Such a relationship is completely alien to discussions of causation and research design. This article has provided an array of examples of variance-altering causation from the political science literature and has described three broad classes of causal mechanisms that can bring them about.

The idea of variance-altering causation is not conceptually problematic, as the examples cited and the causal mechanisms described herein demonstrate, and it offers substantial “value added.” Thinking about the variability of variables rather than just their central tendencies, and by extension thinking about the conditions under which that variability might change, opens up a new dimension to discussions of politics. In purely statistical terms, it offers a more detailed summary of a distribution of interest than does the mean alone. In the realm of description, accordingly, it offers researchers the opportunity to provide a more detailed depiction of the phenomena that we study. In many cases, the variance represents a quantity that is substantively as interesting as, if not more interesting than, the mean. Thinking about variance and how to test hypotheses related to variance also holds out the promise of enhanced leverage in theory testing. In short, the inclusion of variance-altering causation in the collective lexicon of political science should both widen the range of hypotheses that can be tested and substantially enrich our ability to understand phenomena of interest to us.

Nevertheless, if the catalog of causal mechanisms described herein points to any common caveat, it is the danger of underspecification. Each mechanism can have implications

<sup>37</sup>For discussion and development see Goertz and Regan (1997), Diehl and Goertz (2000), and Stinnett and Diehl (2001).

<sup>38</sup>See also Baumgartner and Jones (1993, 2005, chap. 9), as well as Krasner (1984) and Cortell and Peterson (1999).

for variance that are both complex and nonobvious; each requires both theoretical and empirical specificity if unambiguous predictions are to be derived.

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