October 2006

Genetic Configurations of Political Phenomena: New Theories, New Methods

Ira H. Carmen
University of Illinois

Genetic Configurations of Political Phenomena:

New Theories, New Methods

Ira H. Carmen

Department of Political Science

Institute for Genomic Biology

University of Illinois

Paper prepared for delivery, Hendricks Conference on Political Behavior,

Lincoln, Nebraska, October 13-14, 2006
Genetic Configurations of Political Phenomena:

New Theories, New Methods

Abstract

Recent research by E.O. Wilson, James Q. Wilson, Simon, Alford-Hibbing, Carmen and others indicates that the competing social science paradigms of behavioralism and rational choice are in their last throes. Their salient weakness is insensitivity, bordering on ignorance, to politics as a biologically-orchestrated phenomenon. More specifically, political scientists know precious little about either genetics or evolutionary dynamics.

In this paper, I present a new theory--sociogenomics--to replace the shopworn conceptions of yesterday's political science. I then demonstrate how social scientists can employ the tools of molecular biology to flesh out the genes coding for baseline political attitudes and behaviors. The theory and methods of sociogenomics will serve to synthesize the social sciences with the natural sciences in a broader consilient framework, so that the laboratory of Darwinian investigation can become the laboratory of Aristotelian investigation.

Political science is a discipline in need of a paradigm. What else is new?

Fifty years ago, our predecessors could not even agree on what to call their academic domiciles: did we live in departments of government, of politics, or of political science? Were we institutionalists or behavioralists? Did we believe in natural laws or natural rights? If we were in the business of theory construction, could "theory" also mean normative theory?

Today, the waters are as murky as ever. The empiricism of behavioralism -- steeped in the premises and biases of social psychology and attitudinal inference -- has long since come under challenge from the deductivism of rational choice -- steeped in the premises and biases of the economic marketplace and Rawlsian philosophy. Those who hold dear to the preachments of the Enlightenment must fend off the rapacious deconstructionists who argue not only against scripture but against science.

This paper will not engage those who do not believe in science; it will not even engage those who do not believe in political science. As E.O. Wilson (1999: 269) has implied: in each and every bona fide competition between the theory and practice of science and the theory and practice of some other calling, science has won out. This paper will engage the central question at issue for political science in our time: how can we construct an overarching paradigm for the grand purpose of at long last ending the internecine squabbles among those of us who believe in the scientific pursuit of things political?

The principle "squabble" continues to be between behavioralists and rational choicers (Alford and Hibbing, 2004: 1, hereinafter A-H), with each side elegantly dissecting the weaknesses of the opposition. Behavioralists emphasize their commitment

to investigating the attitudes and actions of real people jousting for influence and power; rational actor models, they claim, are effective only in illuminating the politics of "unreal" people wallowing in acultural preferences. Social choice advocates counter with proofs that humans are fully capable of knowing what's good for them politically and acting accordingly; they scoff at the behavioralists' reliance upon socialization and group identification as result-oriented conditioning agencies. Moreover, both sides find themselves under fire from New Institutionalists who contend that politics cannot be ripped from the contexts of formal and informal decision-making trappings.

The core thesis of this paper is simply that these competing paradigms fail because they are not scientific enough. Put succinctly, they eschew a study of the human political species as a species, which means they have nothing whatever to say about the driving force of all other species' attitudinal and behavioral repertoires: genetics. The year 2005 has seen the dawn of a new subfield, a new focus for political science: "genetics and politics." In that year, Alford and Hibbing published two salient reports developing the genetics/politics nexus, while Carmen published a monograph staking out the broader lineaments of the new subfield. It would be a gross oversimplification, however, to believe that the emerging emphasis on human DNA in these writings sprang from nowhere or, as Greek mythology would have it, from Zeus's brow. A decent respect for history requires a brief acknowledgement of intellectual debts proudly incurred. The survey to follow emphasizes the writings of various political science disciplinary leaders and is designed to show their natural affinity with a "genetics and politics" perspective had they only had the benefit of today's molecular biology.

I. The Genetic "Presence" in Political Science

Our profession should exult in knowing that Aristotle -- loosely speaking, a disciplinary founding father extraordinaire -- was in a very real sense the discoverer of the DNA principle. He observed, in the highest spirit of empirical insight, that chickens came from eggs and that oak trees came from acorns; there was some plan or process that inevitably caused A to become B (Ridley, 2000: 12-13). And so was born the first law of genetics: the biological sciences, at root, are the study of information and its transmission. Are we to believe, haughtily, that the social sciences are immune or impervious to that law?

Certainly the founders of the American Political Science Association did not think so. One hundred years ago, John Burgess argued that politics was indeed a science and that students of politics should study political phenomena in the same fashion as a biologist studied life forms. His colleagues, Bryce and Lowell, talked of "political organisms" and "the Physiology of Politics" (emphases added). In short, integral to a scholarly investigation of Homo sapiens -- our species' patterns of thought and action -- was an investigation of Homo politicus (Somit and Tanenhaus, 1967: 19, 24, 33, 71-72, 75). To be sure, their followers declined to be their disciples; they concentrated their attention on congressional power structures and voting behavior inter alia, oblivious to the natural sciences. This did not discourage deeper disciplinary thinkers from pondering what could be. To quote Charles Merriam: "Is it not possible that the real relationship of students of politics is with biology or neurology rather than with psychology?" (1925, 3rd

ed. 1970: 171). Today, we could (and should) include along with psychology our current reliance on economics.

The problem was that political scientists and biological scientists back then had precious little to talk about. Darwin had long since startled the Western World with his grand theory of evolution, synthesizing the twin principles of natural selection and reproductive success, and eventually biologists would fold into their thinking the tenets of Mendelian genetics. But the Darwinian argument presented even these scholars with a raft of unanswered questions such as "survival of the fittest what?" and "if organisms are forever competing, why would they ever cooperate even within a species?" In the 1960s, evolutionists made significant progress. They developed a new terminology: "inclusive fitness," "coefficients of relatedness," "reciprocal altruism." What it all meant was that fitness was measured by genetic parameters, that genetics could explain the commonest forms of cooperation, and that altruism among nonrelateds was largely a function of contractual expectation forged in the crucible of mutual reproductive (read: genetic) success (Hamilton, 1964, Trivers, 1971).

From this literature came a new paradigm and a new guru. The paradigm was called sociobiology; the guru was E.O. Wilson. Essentially, sociobiologists contended that those social behaviors possessing the greatest survival capacity were precisely the behaviors best suited to carry forward the participating organism's relevant genetic characteristics. So human culture became, in their hands, the dependent variable of a population's DNA. "(T)he ...social sciences ... are the last branches of biology waiting to be included in the [Darwinian conception]," Wilson wrote (1975: 4). Ultimately, sociobiology in unvarnished form fell short of its aim. It smacked too much of genetic

determinism. And yet the study of human behavior received a well-justified jolt: No longer would the Freuds, Skinners, Meads, and Marxes corner the market in providing architecture for the human psyche.

Appreciating the weaknesses in his initial effort, Wilson, assisted by Charles J. Lumsden, a physicist, embarked on a major revision. They called their contribution the "gene-culture coevolutionary cycle." It works as follows: genes provide a context for neurological structure and function; the mind perceives and discriminates among cultural phenomena, expanding in power accordingly; human behavior will, in the long run, adapt to those cultural conditions that favor species survival; genotypic probabilities, therefore, are subject to evolution in the light of these conditions just as the conditions themselves are selected for by the relevant cranial processes. So genes affect minds that affect cultures that affect genes (Lumsden and Wilson, 1981). "The challenge is to link the genes and their products into functional pathways, circuits, and networks" (Loomis and Sternberg, 1995: 649). Taken together, these lines of analysis have given birth to a new field of study: evolutionary psychology. With the exception of the hardy breed of political scientists who founded and nurtured the Association for Politics and the Life Sciences 40 years ago (for examples of their work, see Schubert, 1981, Masters, 1989), sociobiology and Wilsonian thinking had barely made a dent among our troops. Now, with the merger of Darwinian principle and the cognitive sciences, a few noteworthy members of the political science discipline began to enter the conversational lists.

A good place to start is the work of Herbert Simon. For Simon, the subject matter of cognitive psychology, which investigates what "[happens] inside the head" of political actors, is the light and the way of today's scholarly mission. Cognitive psychology, for

him, goes hand in hand with his own formulation, bounded rationality. People are decidedly limited in their abstract reasoning capacities, and they search very heuristically, incompletely, and sometimes inadequately through partial sets of information, their algorithms of choice often veiled in ignorance and yielding, therefore, hopefully satisfactory, rarely optimal solutions. Displaying a deeper appreciation of public law concepts than many public law specialists, Simon likens "bounded rationality" to procedural due process of law, where the "choosing organism ... uses methods of choice that are as effective as its decision-making and problem solving means permit .... [This is] behavior that is adaptive within the constraints imposed both by the external situation and by the capacities of the decision maker" (emphases added in first two instances). With procedural due process and bounded rationality, the process of decision is rational if reasonable. Social scientists must "flesh out by a myriad of facts ... our aspirations for ... [general laws, using as a model] the complexities of molecular biology (emphasis added) (Simon, 1985: 294, 301).

Simon was no student of behavioral genetics or even physiological psychology. But he helped set the stage for today's discussions. Consider his observation that species fitness is much enhanced by social learning or "docility," the ability to "accept well the instruction society provides." Because human rationality is bounded, docility carries big payoffs; people simply cannot optimize their capacity to select what they need to learn. Over time, docility becomes favored by natural selection, as it is at least reasonable to learn what society seems to think we should learn. If the social arbiters teach altruism, then altruism ought to spread eventually throughout the population, thus enhancing the

survival and genetic success of the altruists. Docility, then, is bestowed by the genes (Simon, 1990).

Very much in the spirit of Simon's work in this area are James Q. Wilson's contributions. Wilson says humans possess a "moral sense" arising from an "attachment response" or an "affiliate trait" that has been "selected for" by evolutionary processes. These sentiments "constitute the fundamental glue of society," and the "founding sentiment" is the "parent-child relationship" (Wilson, 1993: 1, 7). Wilson supported this assertion by citing evidence from anthropology and social psychology. Children are every bit as indulged by the precivilized on the Kalahari as they are by the suburbanites in the megalopolis; infanticide is practiced only under extreme environmental stresses that could occur anywhere; youngsters develop by assuming eventually the mantle of responsibility for the well-being of their aging forebears, though they gain little in tangible reward for so doing; the brain of the newborn is not a blank sheet of paper, for children prefer human sounds to other sounds, prefer their mothers' sounds above all other human sounds, and express a broad array of nonverbal facial displays before anyone can teach them anything. Wilson summed up these data as follows: "bonding is driven by powerful biological forces" (Ibid.: 4).

Wilson is a full-blooded Aristotelian. So when Aristotle talked of a "natural striving to leave behind another that is like oneself," remarked that a "parent would seem to have a natural friendship for a child, and a child for a parent," and found that "in the household" one sees exhibited the rudiments of "political organization," which by nature tends toward (that is, evolves over time into) the city state, Wilson pinpointed justification for each link in this chain of reasoning in "modern science." If the

fundamentals of political order are subsumed in nature, then perhaps the fundamentals of political rights are also subsumed in nature. For Wilson, the evidence is overriding. Social psychologists have shown that "fair play" -- that is, procedural due process -- "is a necessary condition for the child to satisfy its natural sociability." Children also reject "equality in results" when their peers don't deserve what they receive (Ibid.: 5, 8-9). In the light of all this, he has some justification for chiding evolutionary biologists because they "ordinarily do not specify the psychological mechanism by which a trait that has been selected for governs behavior in particular cases" (Ibid.: 7). If only they all -- including Wilson himself -- had what I shall presently call the new paradigm of sociogenomics staring them in the face.

Simon and the Wilsons were vitally concerned with the workings of the human mind, yet they fail to provide us with a serviceable model. "Men think in terms of models" (Deutsch, 1951: 230), so we need to ask: Which model of species decision theory best comports with the Darwinian perspective?

The models most favored in the social science literature are drawn from the theory of games, and the most often employed of these models is Prisoner's Dilemma (PD). Political scientists are partial to PD because it yields an uncommonly parsimonious solution to a central inquiry in our discipline: Should humans cooperate or should they defect? The leading figure studying the biobehavioral implications of PD is Robert Axelrod.

In an attempt to provide a solution to PD, Axelrod joined forces with W.D. Hamilton, one of the most brilliant geneticists of the past half century. They began by drawing a correspondence between "single-play" PD among non-kin and "single-play"

interactions of unrelated organisms in nature: defection is always the preferred strategy for maximizing survival and fecundity. Even when the number of interactions is multiple but known, defection rules the day. But when the game is an iterated PD and the number of plays is both unknown and multiple, other strategies become competitive. A computer tournament pitting these various algorithms against one another produced a surprise winner: TIT FOR TAT. The lesson is: it pays to cooperate provided defectors are punished immediately but never excessively. Empirical research shows TIT FOR TAT is an evolutionarily stable strategy among vampire bats and vervet monkeys, while experimental simulations demonstrate its robustness for tree swallows and stickleback fish (Axelrod and Hamilton, 1981, Axelrod and Dion, 1988).

Axelrod (1987) has also tested the fitness value of TIT FOR TAT by using the genetic algorithm format. He converted various PD strategies into chromosomal form featuring a 2-bit DNA code: C for cooperate, D for defect. After one all-play-all round, the most robust chromosomes were permitted to mate with one another; the less robust were dropped. Critical to the play of this game is that two well-documented processes of genetic adaptation, crossover and mutation, are here employed to order the DNA sequences of chromosomal progeny. Eventually, the high-scoring routines come to resemble in large degree TIT FOR TAT. What we have here is another proof predicated on genetic theory and methods to show the evolutionary potential of cooperation-reciprocity choice making as the most viable political strategy.

Perhaps the skeptic will argue: computer simulations are not the real world. Research on “green beards” provides a good test case. A “green beard” (the term was coined by geneticist Hamilton) is a simple gene for altruism that is species adaptive in

candidates where organisms display green beards, recognize one another, and respond preferentially. How, biologists wondered, could "green beards" possibly trigger complex social behaviors? Well, green beards do exist, and the proof of principle is found in the slime mold. When in need of food, two cells, each bearing the csA allele, will adhere to one another at their joint gp80 protein sites and coalesce into a larger mass consisting of reproductive spores and nonreproductive stalk cells. The stalk cells will then sacrifice themselves to enhance the reproductive cells' food-gathering opportunities. In laboratory tests, molds in which this gene was knocked out emerge as "cheaters" when "playing the game"; they would have the capacity to latch onto the proliferating mass while giving nothing in return, so green bearded alleles repulse them (Crespi and Springer, 2003). Enter Axelrod and colleagues. Their computer simulations demonstrate that cooperation through donation (classic altruism) will show itself a fit strategy over time as measured by rates of offspring proliferation in game theoretic contexts where green beards take the form of "tags" or display markings of some sort. Note: in each case, the "players" had never before "seen" one another (Riolo et al., 2001). Note also: our "table setting" peers are moving ever closer to the domain of "genetics and politics."

When would political scientists ever move into the biological sciences laboratory and conduct experiments in search of phenomena central to our discipline? That step was taken 20 years ago by Douglas Madsen. He linked whole blood serotonin to power seeking in humans. His research did not purport to show causation but did purport to show -- and, indeed, did show -- association, if one accepts how he defined power seekers. In that study, power seekers were self-defined dominance seekers, whose responses to written questionnaires demonstrated they were of the type A personality

configuration (Madsen, 1985). In subsequent work, Madsen provided two improvements. First, he substituted for questionnaire responses an actual competitive environment, and, second, he compared reactions to that environment among clusters of high WBS subjects, average WBS subjects, and low WBS subjects. Again, a strong association between power seeking as defined and WBS prominence emerged (Madsen, 1986). At this point, Madsen might well have pondered: "If only I could test for the genetic precursors for serotonin accrual in competitive situations. I would then be well on the road to operationalizing E.O. Wilson's coevolutionary cycle in the context of a salient political formulation." The answer to his musing would have been a brick wall. Not only did we lack knowledge of "genetic precursors," we seemingly had no way to find them if they indeed existed. And that is essentially why even those few political scientists who studied matters biological have written so little about DNA pure and simple. Today, it's a whole new ballgame.

II. Twin Studies

Social psychologists have been gathering voluminous data on identical and fraternal twins for at least 30 years both in the US and abroad. These findings have been seized upon only recently by political scientists, and they constitute the first building block for sociogenomics investigation. Identical twins are virtually congruent genetically as respects their nuclear DNA, while fraternals are no more genetically related than any other two brothers or sisters sharing the same biological parents. Twin study specialists

have published detailed reports documenting the heritability quotients arising from comparisons between the two cohorts. By "heritability quotient" (HQ) is meant the variation between them which can be ascribed to genetic differences. Of especial concern to us are heritabilities in the context of behavioral propensity. "Behavioral" is defined broadly to include personality and ideology. The conventional wisdom is that when comparing identicals (monozygotes) and fraternals (dizygotes), the heritability score for an array of behavioral traits is about .50 (Robins, 2005). This means that, on average, the variation between cluster members is .50, i.e., 50% of these behavioral differences can be attributed to genetics (for the computational method, see Wilson, 1999: 151). The other 50% is largely a function of unshared experiences. (For a study implicitly challenging the high HQs recited here, see Hughes, 2005). Even political scientists who eschew an overt "genetics and politics" commitment have spotted the implications of these data for their own scholarship. Take the public law research agenda. In his early work on jurimetrics, Schubert relied on "attitudes" as the key independent variable, and by attitudes he meant what the authors of The American Voter meant -- social psychological affinities (Schubert, 1965). Today's attitudinalists sound a somewhat different note.

> Although genetic explanations of behavior may not be 'politically correct' ... the evidence from the ... studies of identical twins reared apart is compelling: About half of the variance in personality traits, including several that tie closely to political attitudes, can be attributed to genetic diversity (Segal and Spaeth, 1993: 234).

The most oft-cited twin study data are drawn from repositories in Minnesota and Virginia. Table I reports the major findings of relevance to political science. By a "politically relevant" repertoire or variable as these terms are employed in Tables I-V is meant a behavioral propensity which social scientists have associated through their conventional scholarship with some political dynamic. The extent to which particular genes singly or collectively play a causal role in determining the expression of such propensities is the overriding long-term question before us.

Alford, Funk, and Hibbing were provided access to Eaves's Virginia data set, and included here are the salient heritabilities they have published (2005, hereinafter A-F-H). All are statistically significant at the .01 level. Note their substance: they essentially track respondents' support for issue positions, and virtually all significant policy items on the contemporary agenda are represented. The highest quotient (.51) is for the death penalty (cited in A-H), and the lowest (.27) is for segregation. Taken at face value and given our discipline's bias in favor of environmental/cultural determinants, these results should send shock waves through our ranks. But we need to dig deeper. For the Virginia sample, "socialism" elicits a .36 HQ, yet it could do no better than a .14 in an Australian study. Does the term mean such different things in these two universes? More importantly, the aggregate score for "conservatism" is approximately .38; however, the term "liberalism" achieves an underwhelming .18. We would certainly expect the concepts of "conservatism" and "liberalism" to trigger about the same level of acceptance, if we have defined them in the same way so that each is the obverse of the other. As I shall show in a moment, this was not the case here.

Needless to say, there is no "death penalty gene." Responses to Table I policy positions or culturally loaded words and phrases (e.g., "Moral Majority"), to the extent they implicate heritability, must link to some larger attitudinal/behavioral configuration(s) which is/are genetically influenced. There is an on-going, noteworthy debate as to the parameters of this larger phenomenon. Some commentators argue for a strong relationship between political ideology and psychological profiles (Tetlock, 1983, 1984, Jost et al., 2003); others disagree (Greenberg and Jonas, 2003, Alford and Hibbing, 2006). Table I presents several of these personal characteristics and their HQs. Most of the data come from the Minnesota studies and were cited in Carmen (2004). Others are included for the first time in the political science literature with this writing**.** They range from baseline happiness (.80) to anxiety (.32). Both of these have been tied directly to US presidential behavior (Barber, 1972); so also has novelty-seeking (.40) (Hamer and Copeland, 1998). Political scientists who study cooperation/defection would certainly want to know that the HQ for altruism is .50, and those who investigate rationality ought to benefit from the knowledge that the HQ for general intelligence is .52. Table I also reports robust heritability scores for radicalism and right-wing authoritarianism. Taken together, the statistically significant HQs for issue positions and theoretically relevant psychological indices would seem to establish a prima facie case of linkage.

The presumption, at this stage of the dialogue, is rebuttable. The heritability quotient for the Big Five (neuroticism, extraversion, conscientiousness, agreeableness, and openness) may be .50; however, taken as a whole, the set is unrelated to political ideology (Alford and Hibbing, 2006). And so, the argument runs, "the political realm may have unique biological substrates (and perhaps genetic markers)"; in any event,

much work needs to be done to flesh out the heritabilities not only of personal temperament but also of social temperament (responses triggered by "small-scale social situations") and political temperament ("preferences for the structure and conduct of group life") (Ibid., 6). It is the latter constellation of values which returns us to consideration of the conservative-liberal dimension.

The conservative-liberal dimension is the most commonly probed ideological affinity in our literature. Untutored in that literature, twin study specialists not only show little interest in developing standardized tests to flesh out its dynamics but sometimes provide precious little guidance for respondents. Both the Virginia and Australia research studies included the following instruction: "Here is a list of various topics. Please indicate whether or not you agree with each topic by circling Yes or No as appropriate. If you are uncertain, please circle ?" (italics in text). And yet Table I shows fairly consistent and high heritability quotients for conservatism: .43, .32, .62, .31, .55, .45. What we need are two uniform questionnaire rosters, one administered to conservatives and the other to liberals. These clusters would be identified via pretests. The items should deemphasize policy preferences; they should be predicated on perceptions and dispositions for and against political change and inequality which theory informs us (Jost et al., 2003) are the critical underlying variables. The Survey Research Center (Ann Arbor) has developed inquiries of this kind, though none have been employed of late and require fine-tuning. Rossiter (1962: 16-17, 74, 168) argues that conservative legions are divided between "traditionalists," who are constitutionalists, and "pure traditionalists," who are "enemies of change as well as reform [and who] live in a state of acute cultural schizophrenia." Liberals, presumably, display a corresponding

dichotomy. In fact, many observers years ago noted that a substantial number of ex-communists sat on the editorial board of the National Review. Can there be genetic antecedents linking hard-core conservatism and hard-core liberalism, wherein the particular ideological predisposition is the .40-.65 cultural contribution? Applying properly constructed sets of questionnaire items to identicals and fraternals would separate out various types of conservatives and liberals and then penetrate to the heritability dimension. That study is now underway.

III. Genetic Procedures, Genetic Precursors

Presumably skeptics are now prepared to give the notion of "genetics and politics" half a chance: twin study data may have pock marks but their central message is too clear simply to ignore. The fallback position for those remaining unconvinced is not difficult to imagine: Show us the genes. Scientists have been here before. Twenty years ago, when entomologists and lower-order mammalian experimentalists began the quest for behavioral genetic antecendents, "doubting Thomases" expressed the same reservations. They have been proven wrong.

Table II displays a sampling of genes in these nonhuman species which, if isolated in Homo sapiens, would provide important information as to our political behavior repertoires. Researchers have determined the structure and function of each gene. Table II captures this information. It specifies the particular piece of DNA, the

precise form that the DNA assumes in the species of interest, the protein artifact arising from this allelic configuration, the behavioral routine spawned by the action of the protein, and the appropriate published sources. Out of 30-odd instances of success in tracking down these genes, Table II shows only the most recent -- and, in many ways, the most impressive -- of these accomplishments. Our colleagues are learning more each year about how to shed light on the sociality of life forms, and our knowledge of insect, mouse, and primate politics has grown enormously. It is common knowledge among molecular biologists that the fundamental genes responsible for human action are conserved up and down the phylogenetic tree. Each and every gene cited in Table II -- genes influencing stress vulnerability in rats, foraging in honey bees, social feeding in nematodes, aggressiveness in Old World monkeys, and faithfulness in male prairie voles -- has its counterpart in the human. All are either orthologs or homologs of DNA found on every double helix of every cell in our bodies. The exact workings of these genes in our species is a front-and-center concern of our biological science peers.

Table III provides insight into some of the methodological tools available for analyzing genes. The idea is either to insert a gene of interest into the DNA of some other species and assess its impact in behavioral contexts or delete a gene of interest from a species' DNA to assess how that organism will manage in behavioral contexts without it. Again, precision is the order of the day: experimentalists must be able to chart which allele (version) is to be manipulated (genes come in different versions as with blue/brown eyes), what protein will be triggered by that allele, and, of course, the hypothesized, and later proved, behavioral repertoire resulting therefrom. Again, the genes employed have human counterparts, and the action patterns resulting from their expression have clear

political implications if the terms “power” and “influence” mean anything in the context of life form scrutiny. To illustrate: geneticists bred a “fierce” strain of mice and then inserted into its embryos a human gene matching the missing sociality rodent gene. Viola! The super-aggressive mice returned to normal. Putting human DNA into lower-order creatures will be commonplace in years to come.

Those who believe that investigating behavioral genes in lower-order organisms is one thing but that investigating them in the human constitutes some qualitative, unbridgeable leap are in for a rude awakening. While research reports are widely scattered and, incredibly, have never been appropriately organized, collated, and contrasted, Table IV attempts to convey to a political science audience the basics of what needs to be said. Proceeding in chronological order of discovery, one notes 15 gene-related sequences with clear political implications. Some of these fall in a twilight zone area between disease genes and personality genes. How does one classify clinical depression, bipolarism, and attention-deficit disorder? There are thousands of Americans and many thousands of non-Americans who exercise free speech, who vote, who contribute money to political causes, and who “play political games” each and every day who could slide conveniently into one of these behavioral categories. People who are clinically depressed handle stress far less satisfactorily than others, and there ought to be a high correlation between stress overload and various strains of political orientation and participation (Carmen, 2004). It used to be that scholars bandied about such wastebasket terms as “psychopathic personality” and “manic depression.” Genetics has rendered those terms obsolete and will render at least some of the terminology we employ today as

obsolete. The more refined the distinction, the better the opportunity to link one of these configurations to some political mind-set or action-set.

As one glances down the Table IV list, it is easy to spot the importance of the neurotransmitters serotonin and dopamine in influencing sociality. Consider the 5-HTT promoter on chromosome 17. The gene in question controls the serotonin transporter function. Initially, scientists were unable to associate that gene with a whole battery of behavioral characteristics. What they eventually did discover was that a critical difference lay not in the gene coding sequences but in the promoter regions located "upstream" on the double helix. This system can display a "long" version and a "short" version. The long or normal system (a mere 32% of the general population) effectively clears serotonin deposits; the short mutational system (68%), which is dominant, permits serotonin accrual (the percentages provided in Carmen, 2004: 178 are incorrect). (Note: most deleterious alleles are recessive as with, say, the cystic fibrosis mutation; others, such as the Huntington's chorea killer, are dominant.) What is the genetic disparity? The normal version contains 16 sequence repeats approximately 20 base pairs per repeat (a base pair is either A-C or T-G), while the shorter version contains 14 of these repeats, a difference, then, of 44 nucleotide sequences. There is a high correlation between the "short" version regime and neuroticism, and further tests showed that three particular manifestations of neuroticism -- anxiety, angry hostility, and impulsiveness -- are significantly related to the short version. Yet another high correlation was reported for harm avoidance, especially worry, pessimism, fear of uncertainty, and fatigability. Table I notes that neuroticism has an HQ of .50, and geneticists have concluded that the short version allele (whether in homozygous or heterozygous form) accounts for as much as

50% of that differential. Should political scientists synthesize twin study data on neuroticism and its sub-traits with 5-HTT promoter configurations of subject leaders-followers or winners-losers, the discipline would be taking a bold step forward in defining political behavior.

Now consider dopamine. Hamer has investigated the dynamics of what he calls the "novelty" or thrill-seeking syndrome. High scorers on questionnaire items enjoy the play of new ideas; they are predisposed to openness in thought and often action. Low scorers are cautious and conventional, prudent and orderly. Table I tells us that novelty-seeking is .40 heritable. Hamer did not inquire -- we need to inquire -- if high scorers correlate with liberalism and if low scorers correlate with conservatism when we square off monozygotes against dizygotes. The misnamed "novelty gene," actually the D4DR gene, is located on chromosome 11. This gene makes a dopamine receptor protein. Rather like serotonin, dopamine is one of those brain chemicals that needs to be at equilibrium in the typical case, or personality problems and worse arise. Dopamine overload correlates with highly risky behavior: too much gambling, too much sex, too much drinking. What about too much politics? How would one define "too much politics"? The D4DR gene contains a series of 48 letter repeats. The average number of repeats runs from 4 to 7; those with 2 or 3 are extraordinarily effective in clearing dopamine, whereas those with 8 or more (the ceiling is 11) are not very effective at all. If a subject has two "longs" (one from the father, one from the mother) or a "long" and a "short," the correlation with novelty-seeking is far greater than for a subject exhibiting two "shorts." That is, people with less acute pleasure centers have a genetic impetus to develop compensating behavioral propensities. Hamer concludes modestly that the

D4DR gene accounts for only 4% of the .40 HQ (Hamer and Copeland, 1998, Ridley, 2000). Of course, it would be wrong to assume that "pure types" are forever gene-driven, that they can do nothing to counterbalance their firmly established mindsets. We take action contrary to our genetic pulls and pushes all the time. Still, these efforts are corollary to the fundamental role of genetics in politics.

Table IV highlights further instances of dopamine circulation as politically relevant antecedent. Note that in these cases either the critical gene or its allelic configuration varies. For example, the D4DR precursor displays a strong association with obsessive compulsiveness. Here, the critical genetic structure is not a question of "long" vs. "short" occurrence; it is the absence of allele 2 in the 48-letter polymorphism located in exon 3 of this gene (coding regions are called exons; DNA sequences that do not code for proteins are called introns). In another instance, the DAT1 gene on chromosome 5, the only dopamine transporter, enhances in children -- adults have yet to be tested -- what is called "generalized anxiety." The key parameter is the frequency of a 40-base pair repeat, viz., where the governing allele features 10 as opposed to 9 copies. And where subjects are homozygous for the 10-repeat allele in DAT1, attention-deficit disorders emerge; again, the line is not bright between personality and hygienic properties. Finally, the COMT gene on chromosome 22 (see Table IV) metabolizes released dopamine. It exhibits a diallelic polymorphism: <u>met</u> and <u>val</u>. Executive cognition, as demonstrated through Wisconsin Card Sorting Test facility, is enhanced significantly in subjects displaying two <u>mets</u>; the contrary occurs in subjects displaying two <u>vals</u>. In other words, increased dopamine circulation detracts from mental acuity.

As the genes coding for intellectual gifts are deciphered, the keys to political perspicacity should become ever clearer.

Perhaps the oddest, and certainly one of the most controversial, DNA sequences itemized in Table IV is Hamer's inaptly dubbed "God gene." Questionnaire returns have indicated that some people display a greater sense of "self-transcendence" than others, what Hamer calls a sense of spirituality. Spirituality, he says, "provides a numerical measure of people's capacity to reach out beyond themselves." Australian twin-study findings had pegged self-transcendence as .48 heritable. Could spirituality be, in considerable component, a genetic artifact? Hamer attempted to correlate self-transcendence with the D4DR. Result: negative. He then tried to correlate self-transcendence with serotonin DNA precursors. Result: negative. But Hamer hit the jackpot when he sought association between self-transcendence and the VMAT2 gene on chromosome 10. This is a less specialized gene than the others responsible for neurotransmitter function. Its protein packages all of the many monoamines into secretory vector units, bundles the brain uses to store signalling molecules. A certain polymorphism can present two alleles, one in which a key letter is an A and a second in which that letter is a C. If subjects carry a C on either of the two inherited 10s, then they will score much higher on a spirituality index than those carrying two As. The C configuration apparently occurs in about 28% of our species. Query: Is this a gene highly indicative as well of altruism? To what extent do spirituality and cooperation overlap? Are there ethnic differences in these DNA carriages?

Several paragraphs ago, there was discussion regarding the need to link "genes and their products into functional pathways, circuits, and networks." A proliferating

literature in political science stresses the need to address decision making as a neuroscientific phenomenon (McDermott, 2004). Table IV addresses part of the challenge -- but only part -- by pinpointing protein reactions and behavioral repertoires. Table V presents data on the missing connection: the neurophysiological rules and processes that capture the brain's several structures as action systems during the play of political contests. Virtually all these data were recruited by employing the functional magnetic resonance imaging technology as subjects participated in a wide variety of these contests. Table V takes note of the format or game being played, the identities of the competitors, the object or result of the contest, and the winners and losers broadly defined.

Of optimal value would be experiments conjoining genetic and cranial parameters. Only two Table V investigations qualify, unfortunately. One should keep in mind that this is a very new field; all Table V studies are post-2000. Table IV reported a strong correlation between those carrying the "short" version of the 5-HTT promoter and anxiety. This finding received a powerful boost when researchers demonstrated a statistically significant correlation between subjects carrying at least one copy of the "short" serotonin transporter promoter mechanism and elevated activity in their right amygdalas following exposure to anxiety-producing pictures. Control groups made up of "long-long" individuals recorded significantly lower levels of response. Acting instinctively in reaction to messages received from the thalamus, the amygdala sets up the first line of defense against perceived dangers. The linkages with the serotonin carriage system shows that when subjects are particularly fearful, often for genetic reasons, the right amygdala overreacts. Table IV also reported a high correlation between participants

bearing the met version of the COMT gene, dopamine catabolism, and high executive cognition. When MRI tests were then utilized to measure prefrontal cortical efficiency in met vs. val working memory task performers, the met allele individuals scored much higher.

The amygdala is a complex socially relevant information processing center, citations to which are just now beginning to appear in the political science literature. When the typical American white sees a picture of the typical American black, the subject's amygdala fires off an emotional response, a loose translation of which might be: here is someone different. We carry around the genomic baggage of prehistory when facial recognition developed as a key monitoring device for sorting in-groupers and out-groupers. Environmental conditions can alter the equation. If the typical American white sees a picture of Tiger Woods or Michael Jordan, then chances are that that individual's amygdala won't respond: the subject will have unconsciously coded them as insiders (Carmen, 2004: 189-190). Table V displays useful findings. We now know through functional MRI amygdalar screenings that inhibited infants grow up generally to be "avoidance" adults, while uninhibited infants grow up generally to be novelty-seeking adults (the Schwartz study). These tests have also detected differential roles played by the left amygdala and the right amygdala in reacting to "anger faces" and "fear faces." The left amygdala shows a high degree of sensitivity as to whether the stimulus gaze is frontal or averted whereas the right amygdala is unresponsive to such nuances (the Adams study). And when individuals were well apprised of cooperators, having identified them through the play of Prisoner's Dilemma, subsequent facial assessments triggered the left amygdala reacting in concert with such other reward centers as the

striatum (the Singer, 2004, study) (cf. the quite different cranial responses to cooperators during the actual playing of the PD game, at least among women) (the Rilling study). Finally, the amygdala along with the orbitofrontal cortex becomes much more active when players are challenged to accept ambiguous as against risky options, that is, individuals much prefer the latter to the former even when the expected payoffs are equal (the Hsu study). To say that we need to appreciate the genetic mainsprings of amygdalar function is a gross understatement, though we have learned recently that the stathmin gene is highly expressed in the lateral nucleus of the mouse amygdala, and when this gene is knocked out, subjects do not respond either to learned or innate fear (Shumyatsky et al., 2005).

Applications to more overtly political choice making are inevitable. U.C.L.A. researchers essayed a tentative first step when they used the MRI technology to measure partisan reactions to facial images of Mr. Bush and Mr. Kerry. They found an intriguing dance between the emotional centers and the cognitive centers, as both Republicans and Democrats fought to convince themselves of their candidates' manifest superiority. That these cranial processes can be captured by the tools of biological science -- therefore adding a further layer of richness to our understanding of a key political event -- is most informative. From these solid empirical findings, the investigators proceeded to spoil the party by jumping to the conclusion that the Red State/Blue State divide is a fictional artifact of our own self-deception. Equally unwarranted is the assertion that the divide is in fact an expression of genetically-driven "gut" affinities (A-F-H, 2005). We have a long way to go before we can demonstrate neurophysiological causation for any cultural cleavage among nation state electoral camps.

IV. From Genetics to Genomics

Thus far, our tale has been a commentary on what might be called "sociogenetics." The theory, methods, and data reflect the good science of 20 years ago brought up to the present moment by recent discoveries. The orientation is wedded to the structure and function of specific genes acting alone to orchestrate social behaviors in sundry species. Even today, sociogenetics comprises, along with the policy implications arising therefrom, the paradigmatic stuff of a viable "genetics and politics" subfield for our discipline. And yet, as we speak, the term sociogenetics is yielding to the term "sociogenomics." What is its genesis and what does it mean?

The human genome, that is, all of our species' nuclear DNA, is made up of approximately 3 billion nucleotide sequences (base pairs). The "pairs" are a 4-bit code with A always binding to T and C always binding to G. If we knew every A, C, T, and G, then the search for the 25,000 genes buried throughout these sequences, which themselves make up the various chromosomes we carry, would be simplified exponentially. This major coup was largely achieved in the 1990s.

Access to the human genome, taken as a whole, provides parsimonious entrée to the investigation of complex traits of which human social behavior -- human political behavior -- is a prime example. Complex traits arise from a battery of genes acting together. According to Gene Robinson, a leading entomologist who coined the term and nurtured its growth into full-fledged paradigmatic status among biologists, "sociogenomics" refers to the social behavior of life forms as an outgrowth of global

determinants (that is, genomic patterns) employing a comparative species perspective. Sociogenomicists avail themselves of DNA data culled from the fruitfly, the yeast, the mouse, and the rat, among many other species, all of whose genomes have now been sequenced (Robinson, 2002). This cross-species approach adds a key evolutionary component to sociogenomics. Genes are no longer treated as static entities, frozen in time. Genes evolve as they negotiate the interspecies journey, and they evolve in humans as our species, forever seeking to maximize reproductive and survival opportunities, cope with exogenous stimuli. Lumsden and Wilson had argued that culture itself placed certain genes under selective pressure, thus spawning new alleles. There is evidence that the D4DR dopamine receptor has come under selective pressure, enhancing Homo's wanderlust (Olson, 2002). There isn't much genetics, much less genomics, in Lumsden-Wilson. Sociogenomics provides a corollary article to their canon of truths.

Until very recently, before the dawn of sociogenomics, behavioral traits could be traced down (not without difficulty) by utilizing four categories of inquiry: linkage analysis, allele-sharing technologies, association studies in humans, and model organism comparisons (Lander and Schork, 1994). An in-depth treatment of each is beyond the scope of this paper. In candor, political scientists cannot hope to perform sociogenetic -- not even to mention sociogenomic -- experiments without forming collaborations with scholars well versed in molecular biology. They still have a responsibility to appreciate the underlying logics and larger theoretical dimensions of the several procedures mentioned here. Suppose one wanted, ten years ago, to investigate the genetic antecedents of the conservatism-liberalism attitudinal complex. It would first have been necessary to establish objectively the phenotypic characteristics to be tested for and then

develop family tree blueprints which would serve as transmission models. After a putative gene such as those cited in Table IV had been mapped, the next step would evidently have been to show that allelic concordance occurred more often than expected by chance. One would also construct experimental and control groups in order to compare unrelated affecteds with unrelated unaffecteds. The question would be whether specific alleles crop up in those displaying a certain personality characteristic at a significantly higher frequency. Finally, as we have seen, some genes can be slipped into or out of animal models to pin down precise behavioral manifestations. Now that researchers can clone mice, sheep, cats, and pigs, we have at our disposal today uniform physiological environments for assessing the role of human DNA in a wide variety of animal contexts.

All of this should sound sufficiently daunting to explain why an empirically grounded subfield entitled “genetics and politics” has been considered pie in the sky. Capturing the human genome changes everything. At the lowest level of magnitude, the conventional linkage and association procedures mentioned above have generated impressive new insights. With the former, researchers, who have at their disposal genetic markers highly correlated with certain behavioral tendencies, scan the genome in search of precursor DNA chromosomal locations. The short arm of chromosome 6, the short arm of chromosome 8, and the long arms of chromosomes 13 and 22 seem to be prime locations for mutations implicated in various disorders. With the latter, comparing the presence of candidate genes in those displaying sundry antisocial patterns against those immune from these stresses in the context of genomic investigation has yielded virtually all of the probative data found in Table IV (Bouchard and McGue, 2003: 36).

At a higher level of magnitude, new theory also inspires new methods, and, in this case, the breakthrough procedure of choice is called the microarray gene expression technology. In an array experiment, genetic material from two sources of interest, for example species A and species B or perhaps a human experimental and a human control group, are laid on a glass substrate, and scientists measure the levels of expression occurring simultaneously. Subject clusters provide DNA samples, and putative genes of interest contained in these samples trigger expression responses from their counterparts in the microarray universe. Researchers can place billions of DNA sections on the chip, each one about 25 bases in length. In a notable study, honey bee specialists tested 5500 genes employing 72 microarray runs to demonstrate that age-related shifts by adults from hive tasks to foraging tasks could be linked to expression change in 39% of the sample; this finding eventually led to nearly perfect predictions of species behavior based on genetic expression profiles (Whitfield et al., 2003). Figure 1 provides an oft-cited display of the microarray system in action. After "competitive hybridication," in which single-stranded complimentary DNA from the two discrete samples interdigitate, a greater number of Gene X copies in sample A will lead to one color-coding (say, red) and a greater number of Gene X copies in sample B will inspire a different color-coding (say, green). Put more precisely, the expressed genes are detected by the presence of messenger RNA, which is converted back to complimentary DNA (genes without introns or "junk"). Figure 2 is an artist's reproduction of one of the 16 array boxes, created to highlight exactly what the scientist sees.

Microarrays have led to startling new genetic discoveries, examples of which can be found in Table II. To repeat, all these genes have human counterparts; assessing their

functions, however, requires the use of animal models. If we could apply the microarray procedure to humans as we do to honey bees, we could test directly for personality and ideological antecedents in the straightforward manner described above. As we cannot kill human subjects and rescue the messenger RNA expressed in their brains following, say, the play of some game, we must be content now with the rich harvest of behavioral genetic precursors emerging from the laboratories of entomology and related disciplines.

Microarrays become a truly robust vehicle for sociogenomics investigation when the experimental data take the form of human single nucleotide polymorphisms (SNPs). All of us share 99.9% of our DNA sequence. Polymorphisms are stretches of DNA we do not share, thus ensuring that we are not all clones. Genes that exhibit different alleles in different people hold the secret to phenotypic variation. That is, they hold the secret to political attitude and behavior heritabilities. Each one of these is called a SNP. By convention, more than 1% of the population must share the solitary letter substitution. The human genome, it is now estimated, contains 9 million SNPs; 400,000 of them reside in exons; SNPs responsible for amino acid composition shifts could number 200,000. Also to be accounted for are promoter region SNPs, which, as we have seen, can have a marked influence in gene expression levels. To repeat: Each SNP variation yields a unique allele. The Holy Grail in the now well-underway SNP race is the identification of all functional SNPs. This paper takes the position that the Holy Grail of Human Sociogenomics is the totality of functional SNPs coding for behavioral propensities. One of the keys to SNP discovery is the microarray procedure. That is to say, microarrays ferret out disparate gene expression levels in nonhuman subjects, the genes implicated in the sundry behavioral repertoires then provide clues to the identities of SNPs in human

subjects, following which individual variations are correlated with behavioral/attitudinal differences. Eventually, political scientists working with biological scientists can commence to control for the role of each gene one by one, folding in as well interspecies and pedigree data. The sociogenetic tasks referred to earlier as daunting will become manageable, though exceedingly challenging, sociogenomic tasks.

An alternative vision (Alford and Hibbing, 2006: 15) suggests a procedure in which individuals provide saliva specimens for genetic information after which statistical evaluation of correlations between candidate alleles and behavioral responses can be undertaken. The procedure certainly would permit us to compare SNP composition with phenotypic reaction; a drawback is that gene expression cannot be demonstrated in saliva (or blood for that matter) unless salivary genes were involved, because messenger RNA implicated in the tests of interest to political science would be tissue specific to the brain. It is hard to believe that the DNA relevant to ideology encodes proteins known to be present in saliva. And note, it is proteins, not genes, that would show up in saliva following, say, the play of game theoretic exercises. Even for microbiologists, getting from proteins to DNA is exceedingly difficult.

Working with molecularists to ascertain politically-relevant messenger RNA in the brain and working with molecularists to track politically-relevant proteins backwards in time to DNA is the new world of political science as a science, a reaffirmation of our founders' vision.

V. The Longer View

In 1998, E.O. Wilson endeavored to sketch a coming world view of scholarly, of intellectual inquiry. The term he used to describe the inevitable fusion of all human knowledge is "consilience." He envisioned social science achieving a heightened maturity as its practitioners labored cheek to jowl with natural scientists. Already, his ideas have mobilized enlightened souls working in the trenches of other fields to rethink their paradigmatic premises. So we see economists, psychologists, and neuroscientists converging into a single, unified discipline called neuroeconomics (Glimcher and Rustichini, 2004). These scholars have not even bothered to consider political scientists as allies. We are coded as either irrelevant or hopeless. Perhaps they are right. However, they have already committed a fatal oversight. Yet to be included in their paradigm is a sociogenomic component. Those few of us toiling in "genetics and politics" terrain can, this early in the game, claim a leg up on them. The question is whether our discipline as a whole can achieve a leg up on them. Should we choose to do so, we will make for ourselves a unique contribution in the drive towards consilience. Aristotle, who saw the end of the tunnel but no ways to reach that end, would enjoy, one fervently hopes, a burgeoning consilience of scientific inquiry in which the various tasks find practitioners in a laboratory of discovery faithful to the grand empirical enterprise.

References

Abrahams, B.S., et al. (2005). "Pathological Aggression in 'Fierce' Mice Corrected by Human Nuclear Receptor 2E1." 25 (27) Journal of Neuroscience 6263.

Adams, R.B., Jr., et al. (2003). "Effects of Gaze on Amygdala Sensitivity to Anger and Fear Faces." 300 Science 1536.

Alford, J.R. and Hibbing, J.R. (2004). "The Origin of Politics: An Evolutionary Theory of Political Behavior." 2 Perspectives on Politics 707.

Alford, J.R. and Hibbing, J.R. (2006). "How Are Political Orientations Genetically Transmitted? A Research Agenda?" Paper Presented at the Midwest Political Science Association Annual Meeting, Chicago.

Alford, J.R., Funk, C.L., and Hibbing, J.R. (2005). "Are Political Orientations Genetically Transmitted?" 99 American Political Science Review 153.

Axelrod, R. (1987). "The Evolution of Strategies in the Iterated Prisoner's Dilemma." In Genetic Algorithms and Simulated Annealing, ed. L. Davis. London: Pitman.

Axelrod, R. and Hamilton, W.D. (1981). "The Evolution of Cooperation." 211 Science 1390.

Axelrod, R. and Dion, D. (1988). "The Further Evolution of Cooperation." 242 Science 1385.

Barber, J.D. (1972). The Presidential Character. Englewood Cliffs, N.J.: Prentice Hall.

Ben-Shahar, Y., et al. (2002). "Influence of Gene Action across Different Time Scales on Behavior." 296 Science 741.

Bouchard, T.J., Jr. and McGue, M. (2003). "Genetic and Environmental Influences on Human Psychological Differences." 54 Journal of Neurobiology 4.

Carmen, I.H. (2004). Politics in the Laboratory: The Constitution of Human Genomics. Madison: University of Wisconsin Press.

Caspi, A., et al. (2002). "Role of Genotype in the Cycle of Violence in Maltreated Children." 297 Science 851.

Caspi, A., et al. (2003). "Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene." 301 Science 386.

Crespi, B. and Springer, S. (2003). "Social Slime Molds Meet Their Match." 299 Science 56.

de Quervain, D., et al. (2004). "The Neural Basis of Altruistic Punishment." 305 Science 1254.

Deutsch, K.W. (1951). "Mechanism, Organism, and Society: Some Models in Natural and Social Science." 18 Philosophy of Science 230.

Eaves, L.J. and Eysenck, H.J. (1974). "Genetics and the Development of Social Attitudes." 249 Nature 288.

Eaves, L.J. et al. (1999). "Comparing the Biological and Cultural Inheritance of Personality and Social Attitudes in the Virginia 30,000 Study of Twins and Their Relatives." 2 Twin Research 62.

Egan, M.F., et al. (2001). "Effect of COMT Val 108/158 Met Genotype on Frontal Lobe Function and Risk for Schizophrenia." 98 Proceedings of the National Academy of Sciences 6917.

Francis, D., et al. (1999). "Nanogenomic Transmission Across Generations of Maternal Behavior and Stress Responses in the Rat." 286 Science 1155.

Freedman, J. (2005). "This is Your Brain on Politics." New York Times, Jan. 18: A 21.

Gibbons, A. (2004). "Tracking the Evolutionary History of a 'Warrior' Gene." 304 Science 818.

Glimcher, P.W. and Rustichini, A. (2004). "Neuroeconomics: The Consilience of Brain and Decision." 306 Science 447.

Greenberg, J. and Jonas E. (2003). "Psychological Motives and Political Orientation – The Left, the Right, and the Rigid: Comment on Jost et al. (2003)" 129 Psychological Bulletin 376.

Hamer, D. (2004). The God Gene. New York: Doubleday.

Hamer, D. and Copeland, P. (1998). Living with Our Genes. New York: Anchor Books.

Hamilton, W.D. (1964). "The Genetical Evolution of Social Behavior," parts 1 and 2. 7 Journal of Theoretical Biology 1.

Hammock, E.A.D. and Young, L.J. (2005). "Microsatellite Instability Generates Diversity in Brain and Sociobehavioral Traits." 308 Science 1630.

Hariri, A.R., et al. (2002). "Serotonin Transporter Genetic Variation and the Response of the Human Amygdala." 297 Science 400.

Holden, C. (1995). "More on Genes and Homosexuality." 268 Science 1571.

Holden, C. (1998). "The First Gene Marker for IQ?" 280 Science 681.

Hsu, M., et al. (2005). "Neural Systems Responding to Degrees of Uncertainty in Human Decision-Making." 310 Science 1680.

Hughes, C., et al. (2005). "Origins of Individual Differences in Theory of Mind: From Nature to Nurture?" 76 Child Development 356.

Jacobsen, L.K. et al. (2000). "Prediction of Dopamine Transporter Binding Availability by Genotype: A Preliminary Report." 157 American Journal of Psychiatry 1700.

Jost, J.T. et al. (2003). "Political Conservatism as Motivated Social Cognition." 129 Psychological Bulletin 339.

King-Casas, B., et al. (2005). "Getting to Know You: Reputation and Trust in a Two-Person Economic Exchange." 308 Science 78.

Lander, E.S. and Schork, N.J. (1994). "Genetic Dissection of Complex Traits." 265 Science 2037.

Lee, H-J., et al. (2003). "Allelic Variants Interaction of Dopamine Receptor D4 Polymorphism Correlate With Personality Traits in Young Korean Female Population." 118B American Journal of Medical Genetics Part B, 76.

Lemery, K.S. and Doelger, L. (2005). "Genetic Vulnerabilities to the Development of Psychopathology." In Development of Psychopathology, eds. B.L. Hankin and J.R.Z. Abela. Thousand Oaks: Sage.

Lesch, K-P., et al. (1996). "Association of Anxiety-Related Traits with a Polymorphism in the Serotonin Transporter Gene Regulatory System." 274 Science 1527.

Loomis, W.F. and Sternberg, P.W. (1995). "Genetic Networks." 269 Science 649.

Lumsden, C.J. and Wilson, E.O. (1981). Genes, Mind, and Culture. Cambridge: Harvard University Press.

Lykken, D. and Tellegen, A. (1996). "Happiness is a Stochastic Phenomenon." 7 Psychological Science 186.

Madsen, D. (1985). "A Biochemical Property Relating to Power Seeking in Humans." 79 American Political Science Review 448.

Madsen, D. (1986). "Power Seekers are Different: Further Biochemical Evidence." 80 American Political Science Review 261.

Martin, N.G. et al. (1986). "Transmission of Social Attitudes." 83 Proceedings of the National Academy of Sciences 4364.

Masters, R.D. (1989). The Nature of Politics. New Haven: Yale University Press.

McCourt, F. et al. (1999). "Authoritarianism Revisited: Genetic and Environmental Influence Examined in Twins Reared Apart and Together." 27 Personality of Individual Differences 985.

McDermott, R. (2004). "The Feeling of Rationality: The Meaning of Neuroscientific Advances for Political Science." 2 Perspectives on Politics 691.

Meng, J., et al. (2005). "DCDC2 is Associated with Reading Disability and Modulates Neuronal Development in the Brain." 102 Proceedings of the National Academy of Sciences 17053.

Merriam, C.E. (1970). New Aspects of Politics, 3rd ed. Chicago: University of Chicago Press.

Millet, B., et al. (2003). "Association Between the Dopamine Receptor D4 (DRD4) Gene and Obsessive-Compulsive Disorder." 116B American Journal of Medical Genetics 55.

Olson, S. (2002). "Seeking the Signs of Selection." 298 Science 1324.

Plomin, R., et al. (1994). "The Genetic Basis of Complex Human Behaviors." 264 Science 1733.

Ridley, M. (2000). Genome. New York: Perennial.

Rilling, J.K., et al. (2002). "A Neural Basis for Social Cooperation." 35 Neuron 395.

Riolo, R.L., et al. (2001). "Evolution of Cooperation without Reciprocity." 414 Nature 441.

Robins, R.W. (2005). "The Nature of Personality: Genes, Culture, and National Character." 310 Science 62.

Robinson, G. (2002). "Sociogenomics Takes Flight." 297 Science 204.

Rossiter, C. (1962). Conservatism in America, 2nd ed. New York: Vintage Books.

Rowe, D.C., et al. (1998). "The Relation of the Dopamine Transporter Gene (DAT1) to Symptoms of Internalizing Disorders in Children." 28 Behavior Genetics 215.

Rushton, J.P., Littlefield, C.H., and Lumsden, C.J. (1986). "Gene-Culture Coevolution of Complex Social Behavior: Human Altruism and Mate Choice." 83 Proceedings of the National Academy of Sciences 7340.

Sanfey, A.G., et al. (2003). "The Neural Basis of Economic Decision-Making in the Ultimate Game." 300 Science 1755.

Schubert, G. (1965). The Judicial Mind. Evanston: Northwestern University Press.

Schubert, G. (1981). "The Sociobiology of Political Behavior." In Sociobiology and Human Politics, ed. E. White. Lexington, Mass.: Heath.

Schwartz, C.E., et al. (2003). "Inhibited and Uninhibited Infants 'Grown Up': Adult Amygdalar Response to Novelty." 300 Science 1952.

Segal, J.A. and Spaeth, H.J. (1993). The Supreme Court and the Attitudinal Model. Cambridge: Cambridge University Press.

Shaw, S.H., et al. (2003). "Linkage of a Bipolar Disorder Susceptibility Locus to Human Chromosome 13q32 in a New Pedigree Series." 8 Molecular Psychiatry 558.

Shumyatsky, G.P., et al. (2005). "Stathmin, a Gene Enriched in the Amygdala, Controls Both Learned and Innate Fear." 123 Cell 697.

Simon, H.A. (1985). "Human Nature in Politics: The Dialogue of Psychology with Political Science." 79 American Political Science Review 293.

Simon, H.A. (1990). "A Mechanism for Social Selection and Successful Altruism." 250 Science 1665.

Singer, T., et al. (2004). "Brain Responses to the Acquired Moral Status of Faces." 41 Neuron 653.

Singer, T., et al. (2006). "Empathetic Neural Responses are Modulated by the Perceived Fairness of Others." 439 Nature 466.

Smoller, J.W., et al. (2003). "Association of a Genetic Marker at the Corticotropin-Releasing Hormone Locus with Behavioral Inhibition." 54 Biological Psychiatry 1376.

Sokolowski, M.B. (2002). "Social Eating for Stress." 419 Nature 893.

Somit, A. and Tanenhaus, J. (1967). The Development of Political Science. Boston: Allyn and Bacon.

Stenzel-Poore, M.P., et al. (1994). "Overproduction of Corticotropin-releasing Factor in Transgenic Mice: A Genetic Model of Anxiogenic Behavior." 14 (5) Journal of Neuroscience 2579.

Tellegen, A., et al. (1988). "Personality Similarity in Twins Reared Apart and Together." 54 Journal of Personality and Social Psychology 1031.

Tetlock, P.E. (1983). "Cognitive Style and Political Ideology." 45 Journal of Personality and Social Psychology 118.

Tetlock, P.E. (1984). "Cognitive Style and Political Belief Systems in the British House of Commons." 46 Journal of Personality and Social Psychology 365.

Trivers, R.L. (1971). "The Evolution of Reciprocal Altruism." 46 Quarterly Review of Biology 35.

Whitfield, C.W., et al. (2003). "Gene Expression Profiles in the Brain Predict Behavior in Individual Honey Bees." 302 Science 296.

Wilson, E.O. (1975). Sociobiology: The New Synthesis. Cambridge: Harvard University Press.

Wilson, E.O. (1999). Consilience. New York: Vintage Books.

Wilson, J.Q. (1993). "The Moral Sense." 87 American Political Science Review 1.

Zhang, X., et al. (2005). "Loss-of-Function Mutation in Tryptophan Hydroxylase-2 Identified in Unipolar Major Depression." 45 Neuron 11.

FIGURE I

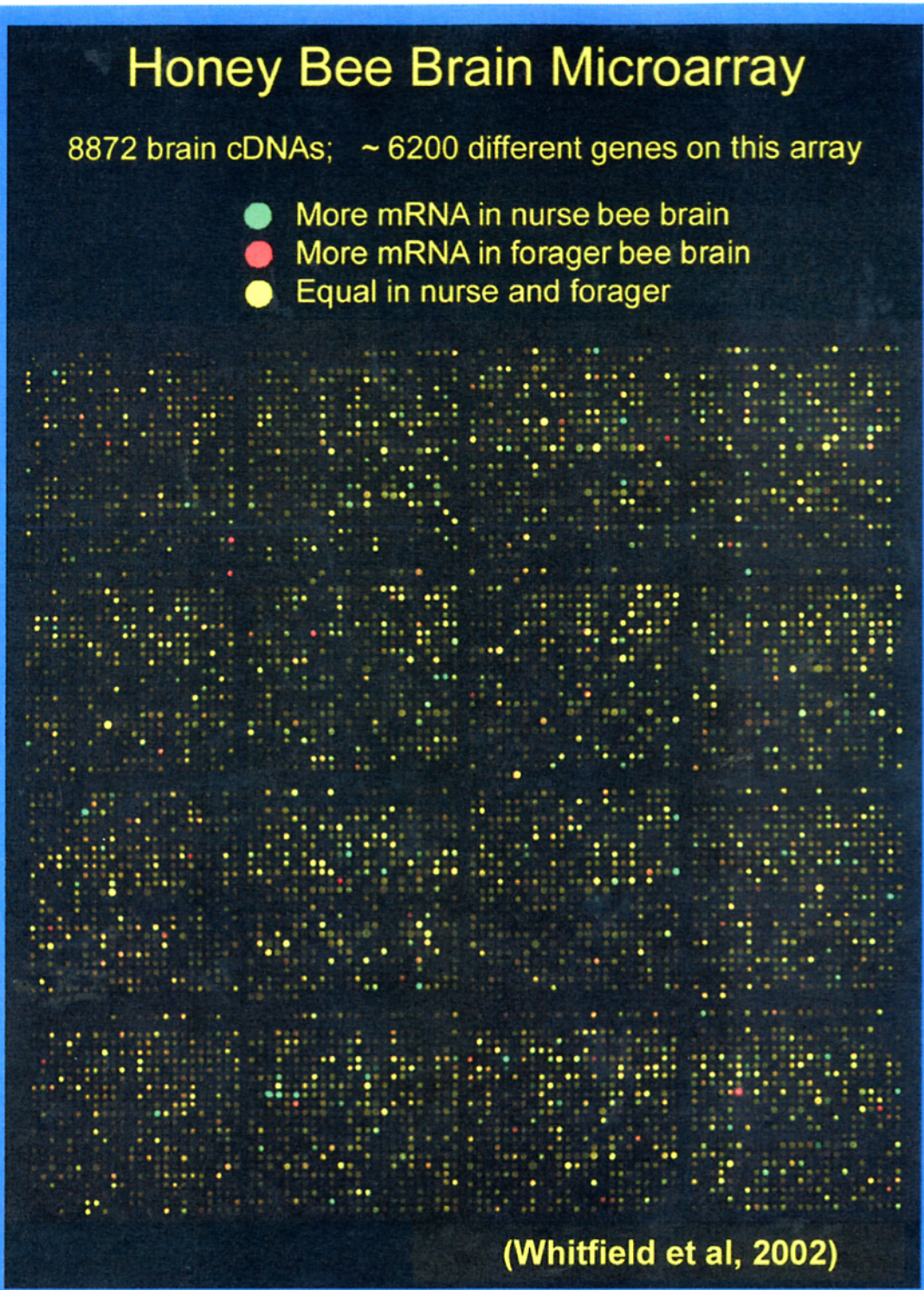
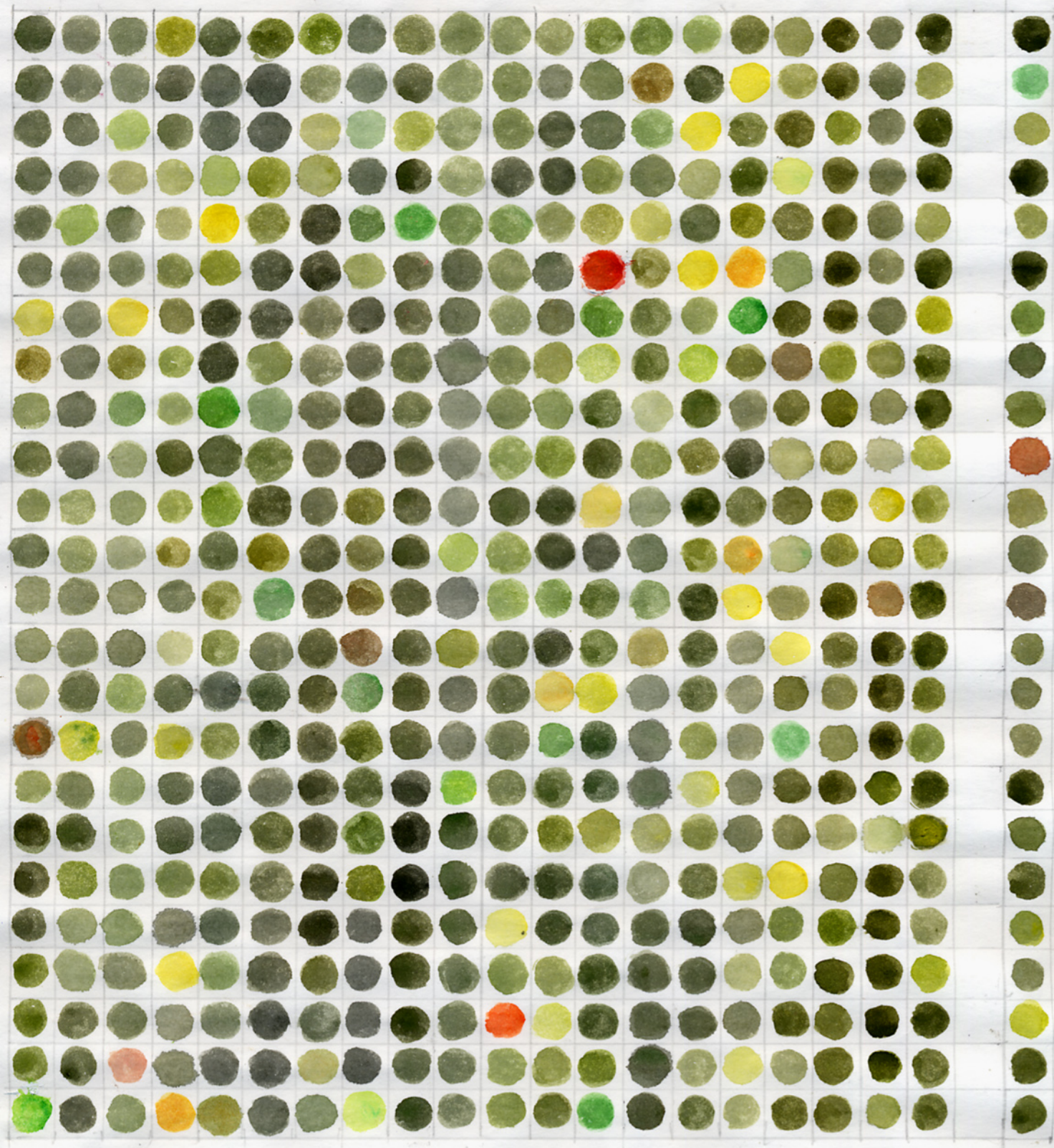

Honey Bee Brain Microarray
8872 brain cDNAs; ~ 6200 different genes on this array
More mRNA in nurse bee brain
More mRNA in forager bee brain
Equal in nurse and forager
(Whitfield et al, 2002)

FIGURE 2



HONEY BEE BRAIN MICROARRAY BLOWUP

Table I: Selected Twin Study Reports of Political Variable Heritabilities (Above .25 and Statistically Significant)
(Ranked by HQ Within Studies)

Phenotypic Characteristic	Heritability Quotient	Primary Lit. Source	Pol. Sci. Lit. Source
Death Penalty	.51	Martin et al. (1986) (Australia-Brit.)	A-H (2004)
Censorship	.41	Ibid.	Ibid.
White Superiority	.40	Ibid.	Ibid.
Disarmament	.38	Ibid.	Ibid.
School Prayer	.41	A-F-H (2005) (Virginia)	A-F-H (2005)
Moral Majority	.40	Ibid.	Ibid.
Capitalism	.39	Ibid.	Ibid.
Pacifism	.38	Ibid.	Ibid.
Republicans	.36	Ibid.	Ibid.
Socialism	.36	Ibid.	Ibid.
Women's Liberation	.33	Ibid.	Ibid.
Death Penalty	.32	Ibid.	Ibid.
Censorship	.30	Ibid.	Ibid.
Gay Rights	.28	Ibid.	Ibid.
Segregation	.27	Ibid.	Ibid.
Democrats	.26	Ibid.	Ibid.
[Liberalism]	.18	Ibid.	Ibid.
Conservatism (Pearson's Correl. Coeffic. of the Above)	.43	Ibid.	Ibid.
Conservatism (Mean of the Above)	.32	Ibid.	Ibid.
Death Penalty	.48	Martin (1986) (Australia)	Ibid.
Censorship	.39	Ibid.	Ibid.
Disarmament	.37	Ibid.	Ibid.
Abortion	.26	Ibid.	Ibid.
[Socialism]	.14	Ibid.	Ibid.

Table I (Cont.) Twin Study Data

Phenotypic Characteristic	Heritability Quotient	Primary Lit. Source	Pol. Sci. Lit. Source
Conservatism (Mean of the Above)	.31	A-F-H (2005)	Ibid.
Baseline Happiness	.80	Lykken & Tellegen (1996)	Carmen (2004)
Behavioral Inhibition to the Unfamiliar	.41-.70	Smoller et al. (2003)	This Paper (2006)
Shyness-Boldness	.55	Hamer & Copeland (1998)	Carmen (2004)
General Intelligence	.52	Plomin et al. (1994)	Ibid.
Neuroticism	.50	Plomin et al. (1994)	Ibid.
Male Homosexuality	.50	Bailey (1995) (cited in Holden 1995)	Ibid.
Altruism	.50	Rushton-Lumsden (1986)	A-F-H (2005)
Self-Transcendence (Spirituality)	.48	Hamer (2005)	This Paper (2006)
Conservatism	.45	Tellegen et al. (1988)	Carmen (2004)
Novelty-Seeking	.40	Hamer & Copeland (1998)	Ibid.
Anxiety	.32	Lemery & Doelger (2005)	This Paper (2006)

Table II: Lower-Order Genetic Precursors of Politically-Relevant Repertoires
(Abridged List of 30 Conserved Genes)

Gene	Species	Allelic Configuration	Protein Reaction	Behavioral Propensity	Primary Lit. Source	Pol. Sci. Lit. Source
glucocorticoid receptor gene	rats	number of receptors	expression depletion	stress vulnerability	Francis et al. (1999)	This Paper (2006)
FOR	honey bees	none	expression elevation	roving, foraging	Ben-Shahar et al. (2002)	Carmen (2004)
NPR-1	nematodes	presence of phenylalanine	expression triggered by envir. stress	social feeding	Sokolowski (2002)	Ibid.
MAOA promoter	apes; Old World monkeys	shorter versions of 30 or 18 base pair repeats	neurotransmitter deficit	aggressiveness; violence	Newman (2004) (cited in Gibbons)	This Paper (2006)
V1aR-associated microsatellites	male prairie voles	long version (19 repeats of 2 BPs)	increase in vasopressin receptors	faithfulness toward partners & offspring	Hammock & Young (2005)	Ibid.

Table III: Examples of Lower-Order Genetic Engineering of Politically-Relevant Repertoires

A. Gene Insertion Studies

Gene	Species	Allelic Configuration	Protein Reaction	Behavioral Propensity	Primary Lit. Source	Pol. Sci. Lit. Source
CRF	mice	rat CRF gene and human growth hormone inserted	hormonal overexpression	inhibition to unfamiliarity; proneness to anxiety in novel situations	Stenzel-Poore et al. (1994)	This Paper (2006)
V1a receptor gene promoter	male voles	transfer of prairie vole promoter to montane vole	vasopressin receptor influence on brain structure	love-making	Young et al. (1999)	Carmen (2004)
NR2E1	"fierce" mice embryos	human gene on chrom. 6 inserted	abnormal neuronal proliferation abated	normal behavior	Abrahams et al. (2005)	This Paper (2006)

B. Gene "Knockout" Study

Gene	Species	Allelic Configuration	Protein Reaction	Behavioral Propensity	Primary Lit. Source	Pol. Sci. Lit. Source
stathmin	mice	the entire gene	amygdalar deprivation of stathmin protein	increase in daring; decrease in caution	Shumyatsky et al. (2005)	Ibid.

Table V: Game Theoretic or Other Neurophysiological Models of Political Behaviors

Format (Game)	Players	Object or Result	Winners	Losers	Primary Lit. Source	Pol. Sci. Lit. Source
working memory tasks (MRI)	met vs. val COMT allele cohorts	prefrontal cortical efficiency	met allele	val allele	Egan et al. (2001)	This Paper (2006)
anxiety-producing pictures (MRI)	long vs. short 5-HTT cohorts	right amygdala overreaction	long allelic regime	one/two short allelic sequences	Hariri et al. (2002)	Carmen (2004)
Prisoner's Dilemma (MRI)	female bargainers	reward system regions (nucleus accumbens, caudate nucleus, etc.) are activated.	mutual cooperators	defectors	Rilling et al. (2002)	This Paper (2006)
Ultimatum Game (MRI)	unfair offerers vs. buyers	bilateral anterior insula reaction vs. dorsolateral prefrontal cortex reaction in buyers	passion rules: offer rejected	reason rules: punishment temporized	Sanfey et al. (2003)	Carmen (2004)
anger and fear face gaze (MRI)	human subject responses to anger/fear, direct/averted 4-fold table	anger/averted and fear/direct gazes differentially more threatening as measured by amygdala response	left amygdala involvement in threat-related activity	right amygdala noninvolvement in threat-related activity	Adams et al. (2003)	Ibid.

Table V (Cont.) Game Theoretic or Other Neurophysiological Models of Political Behaviors

Format (Game)	Players	Object or Result	Winners	Losers	Primary Lit. Source	Pol. Sci. Lit. Source
amygdalar response to novel vs. familiar faces (MRI)	adults coded as inhibited or uninhibited as kids	inhibited subjects exhibited stronger amygdalar responses to novel faces than uninhibited subjects	uninhibited kids/adults	inhibited kids/adults	Schwartz et al. (2003)	Ibid.
sequential Prisoner's Dilemma plus facial assessment (MRI)	bargainers	recognizing cooperators activates left amygdala, striatum and other reward centers	cooperators	defectors	Singer et al. (2004)	This Paper (2006)
monetary payoffs among males (PET)	givers vs. recipient cheaters	subcortical striatum activation (revenge centers). same as Rilling: anticipation of a preferred social outcome	giver's passion	giver's reason	De Quervain et al. (2004)	Ibid.
partisan images (MRI)	Dems. and Reps.	VPC: affinity (limbic) DPC: alienation (reason) ACC: conflict (mediation)	admiration	antipathy	Freedman (2005)	Ibid.

Table V (Cont.) Game Theoretic or Other Neurophysiological Models of Political Behaviors

Format (Game)	Players	Object or Result	Winners	Losers	Primary Lit. Source	Pol. Sci. Lit. Source
10-round Trust Game (MRI)	investors vs. trustees	caudate nucleus activation linked to benevolent reciprocity in trustees	altruists	cheaters	King-Casas et al. (2005)	Ibid.
playing card selection from two decks with known and unknown properties (MRI)	A) normal subjects B) normals vs. OFC defectives	amygdala and OFC modulate striatum	risk probabilities	ambiguity probabilities	Hsu et al. (2005)	Ibid.
cheaters and noncheaters	male and female observers	reduction in empathy-related cortices among men when cheaters are punished	NA	NA	Singer et al. (2006)	Ibid.