Supplemental Material 1: Sender and receiver gene duplication. Sender genes may undergo duplication. Here, the unit of selection is the sender gene, but the selected phenotype is the signal. Likewise, receiver gene duplication could be viewed as involving action duplication and subsequent action neofunctionalization. Different models of gene duplication and acquisition of novel function, can therefore be framed as models of novel signal and action genesis (as in a Polya's Urn Model for generating synonymous signals in an expanding message alphabet [110]). For example, Ohno's model of gene duplication involves gene duplication followed by a period of drift in one gene duplicate, which may then eventually acquire a novel function by random mutation, or dysfunctionalization as a pseudogene [111]. This process can be viewed as sender gene duplication followed by signal drift by one gene duplicate, leading to the possible genesis of a novel signal by the sender (gene) duplicate.

In contrast, the innovation, amplification, divergence (IAD) model of gene duplication proposes that the ancestral gene is bifunctional with a major function, and a minor side function [112], [113], [22]. In response to an environmental change, the ancestral gene's amplification is favored in order to increase the minor functional activity, as a gene dosage effect. Then one of the gene duplicates may mutate to increase the activity of the minor function (usually at the expense of the major function). The signaling games framework would propose that an ancestral sender gene sends two signals, a major and a minor signal, each with its own receiver(s). After sender gene amplification, one gene duplicate may mutate so that the minor signal increases in intensity, at the expense of the major signal. There may be some relevance here regarding the evolution of human signals; Gambetta for instance proposes that novel signals originate as signs that are then sequestered for signaling purposes [114], a model consistent with genes that originate from so called 'junk' DNA [115], and also the occurence of ohnologs, which are paralogs that arise from whole genome duplications, such as p53/p63/p73 [116] and hemoglobin/myoglobin [117].

Supplemental Material 2: Defining Deception. To understand why such undesirable outcomes arise in the form of deception, we call upon a theory of information-asymmetric signaling games to unify many of the adversarial use cases under a single framework, in particular when adversarial actions may be viewed mathematically as rational (i.e., utility-optimizing agents possessing common knowledge of rationality).

The simplest model of signaling games involves two players. They are asymmetric in information and are called S, sender (informed), and R, receiver (uninformed). A key notion in this game is that of *type*, a random variable whose support is given by T (known to sender S). Also, we use $\pi_T(\cdot)$ to denote probability distribution over T as a prior belief of R about the sender's type. A round of game proceeds as follows: Player S learns $t \in T$; S sends to R a signal $s \in M$; and R takes an action $a \in A$. Their payoff/utility functions are known and depend on the type, signal, and action:

$$u^i : T \times M \times A \to \mathbb{R} : i \in \{S, R\}. \tag{1}$$

In this structure, the players' behavior strategies can be described by the following two sets of probability distributions: (1) $\mu(\cdot|t), t \in T$, on M and (2) $\alpha(\cdot|s), s \in M$, on A. For S, the sender strategy μ is a probability distribution on signals given types; namely, $\mu(s|t)$ describes the probability that S with type t sends signal s. For R, the receiver strategy α is a probability distribution on actions given signals; namely, $\alpha(a|s)$ describes the probability that R takes action a following signal s. A pair of strategies μ and α is in Nash equilibrium if (and only if) they are mutually best responses (i.e., if each maximizes the expected utility given the other):

$$\begin{aligned} &\sum_{t\in T, s\in M, a\in A} u^S(t,s,a)\pi_T(t)\mu^*(s|t)\alpha(a|s) \\ &\geq \sum_{t\in T, s\in M, a\in A} u^S(t,s,a)\pi_T(t)\mu(s|t)\alpha(a|s) \end{aligned} \tag{2}$$

and

$$\begin{aligned} &\sum_{t\in T, s\in M, a\in A} u^R(t,s,a)\pi_T(t)\mu(s|t)\alpha^*(a|s) \\ &\geq \sum_{t\in T, s\in M, a\in A} u^R(t,s,a)\pi_T(t)\mu(s|t)\alpha(a|s) \end{aligned} \tag{3}$$

for any μ, α. It is straightforward to show that such a strategy profile (α^*, μ^*) exists. We conjecture that the natural models for sender-receiver utility functions could be based on functions that combine information rates with distortion, as in rate distortion theory (RDT). For instance, assume that there are certain natural connections between the types and actions, as modeled by the functions f_S and f_R for the sender and receiver respectively:

$$f_S : T \to A; \quad f_R : A \to T. \tag{4}$$

Then the utility functions for each consist of two weighted-additive terms, one measuring the mutual information with respect to the signals and the other measuring the undesirable distortion, where the weights are suitably chosen Lagrange constants

$$\begin{aligned} u^S &= I(T,M) - \lambda_S d^S(f_S(t), a), \quad \& \\ u^R &= I(A,M) - \lambda_R d^R(t, f_R(a)), \end{aligned} \tag{5}$$

where I denotes information and d^R, d^S denote measures of distortion.

This definition also captures the notion of *deception* as follows. Note that the distribution of signals received by R is given by the probability distribution π_M, where

$$\pi_M(s) = \sum_{t \in T} \pi_T(t)\mu(s|t), \tag{6}$$

and the distribution of actions produced by R is given by the probability distribution π_A, where

$$\pi_A(a) = \sum_{s \in M} \pi_M(s)\alpha(a|s). \tag{7}$$

Clearly π_T and π_A are probability distributions on T and A respectively. If $\hat{\pi}_T$ is the probability distribution on T induced by π_A under the function f_R, then

$$\hat{\pi}_T(\cdot) := \pi_A(f_R^{-1}(\cdot)). \tag{8}$$

A natural choice of measure for deception is given by the relative entropy between the probability distributions π_T and $\hat{\pi}_T$:

$$\begin{aligned} &\text{Deception} := \text{Rel. Entropy}(\hat{\pi}_T|\pi_T) \\ &= \sum_{t \in T} \hat{\pi}_T(t) \log_2 \frac{\hat{\pi}_T(t)}{\pi_T(t)}. \end{aligned} \tag{9}$$

This definition describes deception from the point of view of the receiver. To get the notion of deception from the point of view of the sender, one needs to play the game several rounds. The equation implies that deception can be both defined as the sending of misleading information, or the witholding of information, both in order to manipulate the receiver. The Shapley value describes the distribution of utility to different players in a cooperative game. In a signaling game where deception occurs the value is skewed towards the sender.

Supplemental Material 3.

Table S1: Examples of molecular deception by selfish elements

Selfish element	Type of Deception
DNA transposons	Evidence that the ALP1 gene in plants was a transposon gene that initially evolved as a means of evading transposon silencing Insertion close to ORFs may be a mechanism to evade transposon silencing [39], [40] MITE elements may avoid transposon silencing due to their short length [118] [40], [119] Demethylation provides a mechanism to evade transposon transcriptional silencing [39]. The rice CACTA transposon produces a micro RNA that binds to the mRNA of a DNA methylation silencing gene [120], thus intercepting the host defensive signal. Arabidopsis thaliana Hi encodes VANC, which promotes DNA demethylation [121]. Maize Spm transposon encodes TrpA protein which catalyzes DNA demethylation [122]. Trans-duplication, the acquisition of host gene fragment by a mobile element, may be a mechanism of camouflage to avoid silencing [39]. Examples include Pack-MULE [123], CACTA [69] and helitron-like [124] elements in plants. Frameshifting [125]
Retrotransposons	The Evad plant retrotransposon cloaks its RNA with small proteins in order to avoid the RNA interference host defence [126] Evidence of selection for evasion of transcriptional silencing of primate retrotransposons SVA and L1 [127] and for evasion of the fungal repeat induced point mutation anti-TE mechanism by Gypsy-like retrotransposons [128] Plant Cassandra elements use a 5S rRNA sequence fragment as a promoter, tricking RNA polymerase III to transcribe the element [129]. Human Alu SINE elements do something similar, using a promoter derived from 7SL RNA [130] Stop codon readthrough is utilized in retrotransposon expression [131], [132], [133], [134], [135], [136] Frameshifting is utilized in expression of retrotransposons [137](a survey) 60 % of human Alu SINE elements are present in introns [138]. Introns have lower levels of DNA methylation than exons [139], and so in this way Alu elements may avoid transcriptional silencing by DNA methylation
Bacterial insertion sequences	Stop codon readthrough [140] Frameshifting [141](a survey)
Homing endonucleases	Homing endonucleases hide within Group I introns, which are phenotypically silent [142] Homing endonucleases introduce a double stranded break into the host genome, which is (mis) recognized by the DNA repair system and repaired by recombination, in the process incorporating a copy of the gene [143]
Group I introns	Group I introns insert into intronless alleles, and excise themselves without affecting the mRNA. Preferentially target conserved regions of host proteins, decreasing chance of elimination [144], by conflating the identity of the conserved site
Group II introns	Double strand break repair recombination machinery sequestered in order to integrate intron cDNA into recipient allele [145]
Inteins	Inteins target conserved protein coding sequences apparently in order to evade host defences [144], commonly in essential proteins [146]
B chromosomes	B chromosomes mimic sex chromosomes [147]
Segregation distorters	Drosophila melanogaster Segregation Distorter (SD) is a modified form of RanGAP that deceives the intracellular transportation system, mislocalizing into the nucleus [148] where it promotes segregation distortion Maize knob repeat elements induce centromere-like neocentromeres leading to meiotic drive [149]
Plasmids	Plasmid encoded antirestriction ArdA proteins mimic DNA in order to inhibit host encoded restriction enzymes [150]

Supplemental Material 4: Allelic exclusion, identity and conflict. Allelic exclusion typically involves the assignment of identity, and consequently may become a focal point in genetic conflicts. In trypanosomes, it provides a mechanism to shift expression of antigenic cell surface proteins from time to time [151]. This periodic identity switching is a way of deceiving the host immune system into classifying the parasite as self. Systems for assigning identity at the molecular level, such as the immune system, appear to constitute a form of kin recognition, which proposes that identity tags are used to promote altruistic behavior amongst related individuals [152]. In multicellular organisms this behavior would promote cooperation between cells, and guard against non-self infiltrators. However, cancer cells are technically 'kin,' but when detected by the immune system are recognized as non-self. This mechanism would suggest an analogy with human social mechanisms that assign identity to cooperating in-groups, and non-cooperating out-groups.

In the Brassicaceae higher plants, allelic exclusion also functions in identity, in the mechanism of self-incompatibility at the *S*-locus. Self-incompatibility is a mechanism of preventing self-fertilization in hermaphroditic plants. Here, the *SP11* allele of dominant *S*-haplotypes is expressed, while that of recessive *S*-haplotypes is repressed by a small RNA expressed from the dominant *S*-haplotype locus, which acts in trans to induce methylation of the recessive *S*-haplotype locus [153]. This strategy implies that only the SP11 protein encoded by the dominant *S*-haplotype is incorporated into the pollen coat. A corresponding receptor encoded by the same *S*-haplotype in the pistil results in incompatibility. A dominant-recessive system results in a reduction in the numbers of potential mates that are rejected, compared to a co-dominant system [154], [155]. Genetic conflict would be expected to occur within this system between the sporophyte and gametophyte due to differing reproductive needs [156], and so we hypothesize that some forms of molecular deception should be present.

Supplemental Material 5.
Figure 2 Image information
(a) shows images (obtained from Encyclopedia of Life, eol.org, creative commons license CC BY-NC-SA 2.0) of a solitary wasp (*Eumenes subpomiformis*, photo by Valter Jacinto), social wasp (*Vespula vulgaris*, photo by Sean McCann), honey bee (*Apis mellifera*, photo by John Baker), hornet (*Vespa crabo*, photo by Biopix), bumblebee (*Bombus lucorum*, photo by BioLib.cz), carpenter bee (*Xylocopa virginica*, photo by Kent McFarland) and drone fly (*Eristalis tenax*, photo by Denis Doucet). (b) shows three dimensional Protein Data Bank (PDB) images of *Escherichia coli* phe-tRNA (3L0U), *Streptococcus mutans* release factor 1 (1ZBT), *E.coli* release factor 2 (1GQE), *Thermus thermophilus* ribosome recycling factor (1EH1), *Acinetobacter baumannii* elongation factor P (5J3B), TYMV tRNA-like structure (4P5J) and the *E.coli* 70S ribosome (4V4A).

Supplemental Material 6: Cancer as a signaling game. Cancers appear to be initiated and maintained by a small population of tumor stem cells [157]. In normal tissue there is a feedback loop where healthy differentiated cells send signals to the stem cells, inhibiting their replication, until there is a need for proliferation and subsequent differentiation [158]. This mechanism represents a signaling convention between the differentiated cells (sender) and stem cells (receiver), justified by common interest between the two. However, mutations can cause the stem cells to escape this feedback loop by altering their response (i.e., action) to the inhibitory signals. In this scenario, the signaling convention is subverted and leads to the (short term) benefit of the stem cells through enhanced replication rates. However, eventually the convention breakdown will lead to punishment of both sender and receiver, through death of the animal. Punishment is a feature of social contracts [159], and likewise the differentiated cells and stem cells can be considered bound by a contract, which entails sanctioning when a player breaks the contract. There are further features of cancer which can be understood in terms of signaling games. Cell apoptosis is a mechanism to destroy aberrant cells and can be regarded as a means to remove a player from a molecular signaling game, if it is likely to break the signaling convention. Cancer cells have acquired the ability to escape this enforced retirement from the game, by a variety of mechanisms [160]. In addition, necroptosis is where an entire region of tissue undergoes programmed cell death, and can be seen as a way of removing cancer cells from the game, but at the cost of the removal of neighboring cells from the game as well, which assumes they are both in direct communication (to be players in a signaling game they must be communicating).