Abstract. The notion of building blocks can be related to the structure of the offspring probability distribution: loci of which variability is strongly correlated constitute a building block. We call this correlated exploration. With this background we analyze the structure of the offspring probability distribution, or exploration distribution, for a GA with mutation only, a crossover GA, and an Estimation-Of-Distribution Algorithm (EDA). The results allow a precise characterization of the structure of the crossover exploration distribution. Essentially, the crossover operator destroys mutual information between loci by transforming it into entropy; it does the inverse of correlated exploration. In contrast, the objective of EDAs is to model the mutual information between loci in the fitness distribution and thereby they induce correlated exploration.

1 Introduction

In the realm of evolutionary computation the notion of building blocks of evolution has been developed in Holland’s original works (Holland 1975; Holland 2000) to describe the effect of crossover. In that respect, building blocks are composed of genes with more or less linkage between them. This is one to one with the notion of schemata and eventually lead to the schema theories which describe the evolution of these building blocks.

In the biology literature though, the notion of building blocks has quite a different connotation. As a paradigm I choose the empirical findings of Halder, Callaerts, & Gehring (1995): The experimenters forced the mutation of a single gene, called “eyeless gene”, in early ontogenesis of a Drosophila Melanogaster fly. This rather subtle genotypic variation results in a severe phenotypic variation: an additional whole, functionally complete eye module grows at some place it was not supposed to. Here, the notion of a building block refers to the eye as a functional module which can be grown phenotypically by triggering a single gene. In other words, a single (and thus non-correlated) mutation of a gene leads to a highly complex, in terms of physiological cell variables highly correlated phenotypic variation. Such properties of the genotype-phenotype mapping are considered as the basis of complex adaptation (Wagner & Altenberg 1996). A theory on the evolution of complex phenotypic
Besides the discussion of crossover in GAs and that of functional modularity in natural evolution, there is a third field of research that relates to the discussion of building blocks: Estimation-of-Distribution Algorithms (EDAs, Pelikan, Goldberg, & Lobo 1999). These algorithms are a direct implementation of the idea of correlated exploration in the framework of heuristic search algorithms. They explicitly encode the search distribution (i.e., offspring probability distribution) by means of a product of marginals (PBIL, Baluja 1994), factorized distributions (FDA, Mühlenbein, Mahnig, & Rodriguez 1999), dependency trees (Baluja & Davies 1997), or, most generally, a Bayesian network (BOA, Pelikan, Goldberg, & Cantú-Paz 2000). To my point of view, the key of these algorithms is that they are capable to induce the same notion of building blocks as we introduced it in the context of natural evolution. For instance, consider a dependency tree where the leaves encode the phenotypic variables. Offsprings are generated by sampling this probabilistic model, i.e., by first sampling the root variable of the tree, then, according to the dependencies encoded on the links, sampling the root’s successor nodes, etc. Now, if we assume that the dependencies are very strong, say, deterministic, it follows that a single variation at the root leads to a completely correlated variation of all leaves. Hence, we may define a set of leaves which, due to their dependencies, always vary in high correlation as a functional phenotypic module in the same sense as for the eyeless paradigm.

Several discussions in the EC community though contradict this point of view: Some argue that the essence of EDAs is that they can model the evolution of crossover building blocks (schemata) by explicitly encoding the linkage correlations that are implicit in the offspring distribution of crossover GAs (Shapiro 2003, Introduction). In that sense, EDAs are “only” faster versions of crossover GAs; faster because EDAs actively analyze correlations in the selection distribution whereas crossover masks would have to self-adapt (see section 6). In this paper we want to point out that, certainly, crossover induces a correlation in the search distribution that can be modeled by graphical models, but the concept of graphical models is far more general than that of linkage correlations. Hence, EDAs and non-trivial gene interaction models (non-trivial genotype-phenotype mappings, Toussaint 2003b) can introduce correlational structures in the search distribution that go qualitatively beyond simple crossover GAs.

Most important of all: EDAs and gene interaction models can account for correlated innovation. Here, innovation means that some phenotypic variable changes its value and some other phenotypic variables change their values in high dependence of this change, such that the constellation of this set of variables is really new, has not been present in the parent population. In contrast, crossover can only preserve certain (by the crossover mask determined) linkage correlations that have been present in the parent population and never explores new correlated constellations in the sense of correlated innovation.

The main goal of this paper is to prove and formalize the claims that have been made above. After we define crossover in the next section, section 3 and 4 will present some theorems on the ‘structure’ of the search distribution after mutation and crossover. With structure we mean the correlational structure that we measure by means of mutual information. Many arguments are based on the increase and decrease of mutual information in relation to increase or decrease of entropy in the search distribution. Section 5 finally defines the notion of correlated exploration and thereby pinpoints the difference between linkage correlations and correlations in EDAs or gene interaction models. Figure 2 already explains the key idea.

2 Formalism

The Simple GA. We represent a population as a distribution p over genotype space Ω. In this paper we assume that a genotype is composed of a fixed number of genes, Ω = Ω₁ × · · · × Ωₙ, where the space Ωᵢ of alleles of the ith gene is arbitrary. We represent also finite populations as a distribution p ∈ ΛΩ, namely, if the population is given as a multiset A = {x₁, ..., xₙ} we (isomorphically) represent it as the finite distribution given by p = ¹⁄ₙ ∑ᵢ=₁ⁿ δₓᵢ, where δₓ is the delta distribution at x, i.e., p(x) = |A\{x}⟩ |A⟩ = multiplicity of x in A. Crossover and mutation are represented as operators ΛΩ → ΛΩ that map a parental (finite or infinite) population to an offspring distribution. Given some operator u : ΛΩ → ΛΩ we will use the notation ∆ₜB = B(ulp) − B(p) to
denote the difference of a quantity $B : \Lambda^\Omega \to \mathbb{R}$ under transition, e.g., the quantity may be the entropy $H(p)$ of a distribution.

In that framework we may write the evolution equation of a crossover GA as

$$p^{(t+1)} = S^\mu \mathcal{F}^{(t)} S^\lambda M \mathcal{C} p^{(t)},$$

with crossover $\mathcal{C}$, mutation $M$, offspring sampling $S^\lambda$, fitness $\mathcal{F}$, and parent sampling $S^\mu$. A sampling operator $S^\mu : \Lambda^\Omega \to \Lambda^\Omega$ draws $n$ independent samples from a distribution and maps this multiset of samples to the respective finite distribution; note that $\lim_{n \to \infty} S^n = \text{id}$. Fitness $\mathcal{F}^{(t)} : \Lambda^\Omega \to \Lambda^\Omega$ rescales a distribution proportional to some functional $f^{(t)}$, $(\mathcal{F}^{(t)} p)(x) = \frac{f^{(t)}(x)p(x)}{\sum_x f^{(t)}(x')p(x')}$. We define mutation and crossover more precisely as follows:

**Definition 2.1 (Mutation).** We define mutation as an operator $M : \Lambda^\Omega \to \Lambda^\Omega$ defined by the conditional probability $M(y|x)$ of mutating from $x \in \Omega$ to $y \in \Omega$:

$$M_p = \sum_x M(|x) p(x).$$

A typical mutation operator fulfills the constraints of symmetry and component-wise independence:

a) $M(y|x) = M(x|y)$

b) $\Omega = \Omega^1 \times \cdots \times \Omega^N \Rightarrow M(x|y) = \prod_{i=1}^N M_i(x^i|y^i)$

In the following we will refer to the the simple mutation operator for which all component-wise mutation operators are such that the probability of mutating from $x$ to $y$ is constant for $x \neq y$:

$$\forall i : M_i = M^* , \quad \forall x \neq y \in \Omega^* : M^*(x|y) = \frac{\alpha}{n}, \quad \forall x \in \Omega^* : M^*(x|x) = 1 - \frac{\alpha(n-1)}{n},$$

where $n = |\Omega^*|$ and $0 \leq \alpha \leq 1$ denotes the mutation rate parameter.

**Definition 2.2 (Crossover).** We define crossover as an operator $\Lambda^\Omega \to \Lambda^\Omega$ parameterized by a mask distribution $c \in \Lambda^{(0,1)^N}$, where $N$ is the number of loci (or genes) of a genome in $\Omega$:

$$\mathcal{C} : \Lambda^\Omega \to \Lambda^\Omega : p \mapsto \mathcal{C} p = \sum_{x_0,x_1 \in \Omega} \mathcal{C}(|x_0,x_1) p(x_0)p(x_1),$$

$$\mathcal{C}(x|x_0,x_1) = \sum_{m \in \{0,1\}^N} c(m)[x = x_0 \ominus m x_1],$$

where the $i$th allele of the $m$-crossover-product $x_0 \ominus m x_1$ is the $i$th allele of the parent $x_m$, i.e., $(x_0 \ominus m x_1)^i = (x_m)^i$. We only consider symmetric crossover, where $c(m) = c(\bar{m})$.

In the case of bit strings, $\Omega = \{0,1\}^N$, it holds $x_0 \ominus m x_1 = x_0 \ominus m \oplus \bar{m} \oplus x_1$, where $\oplus$ denotes the XOR and $\ominus$ the AND. It follows that (Vose 1999, Theorem 4.4)

$$\forall x_0,x_1 \in \Omega : \mathcal{C}(|x_0,x_1) = \mathcal{C}(|x_1,x_0), \quad \mathcal{C}(x|x_0,x_1) = \mathcal{C}(0|x_0 \oplus x, x_1 \oplus x).$$

**Estimation-Of-Distribution Algorithms.** Concerning EDAs, we write their dynamics as

$$y^{(t+1)} = \mathcal{H}(\mathcal{F} S^\lambda \Phi y^{(t)}, y^{(t)}),$$

where, instead of a parent population, some other parameters $y^{(t)}$ (e.g., a Bayesian graph or dependency tree) determine the offspring distribution $\Phi y^{(t)}$, which is sampled, evaluated, and, instead of a simple parent sampling,
mapped back on new parameters $y^{(t+1)}$ by some update operator $\mathcal{H}$. The operator $\mathcal{H}$ is called heuristic rule and, in the case of Estimation-of-Distribution Algorithms, is such that the new search distribution $\Phi y^{(t+1)}$ approximates the experienced fitness distribution $\mathcal{F} S^\lambda \Phi y^{(t)}$. The generic implementation of this idea is

$$y^{(t+1)} = y^* , \quad y^* = \arg\min_{y \in Y} D(\mathcal{F} S^\lambda \Phi y^{(t)} \parallel \Phi y) ,$$

where $Y$ is the space of feasible parameters $y$ and $D(\cdot \parallel \cdot)$ denotes the Kullback-Leibler distance (see Toussaint (2003a) for a discussion of generic heuristic search and evolution). In fact, the BOA algorithm (Pelikan, Goldberg, & Cantú-Paz 2000), which uses Bayesian networks to parameterize the search distribution, realizes exactly this scheme. Other algorithms (Baluja & Davies 1997; Mühlenbein, Mahnig, & Rodriguez 1999; Baluja 1994) differ in some details, e.g., they use distance measures other than the Kullback-Leibler divergence or realize a gradual adaptation of continuous parameters $y$ of the style $y^{(t+1)} = a y^* + (1 - a) y^{(t)}$. See (Toussaint 2003b) for a survey on the relation between EDAs and the evolution of genetic representations ($\sigma$-evolution) in the context of non-trivial genotype-phenotype mappings.

### 3 The structure of the mutation distribution

This section derives a theorem that simply states that mutation increases entropy and decreases mutual information. It is surprising how non-trivial it is to prove this intuitively trivial statement.

**Lemma 3.1 (Component-wise mutation).** Consider the component-wise simple mutation operator $\mathcal{M}^*$ as given in definition 2.1. It follows that

$$a) \quad \mathcal{M}^* p(x) = (1 - \alpha) p(x) - \alpha \frac{1}{n} ,$$

which is a linear mixture between $p$ and the uniform distribution (“$\frac{1}{n}$”) with mixture parameter $\alpha$.

$$b) \quad \text{For every non-uniform population } p, \text{ the entropy of } \mathcal{M}^* p \text{ is greater than the entropy of } p ,$$

$$H(\mathcal{M}^* p) > H(p) .$$

**Proof.**

$$a) \quad \mathcal{M}^* p(x) = \sum_y \mathcal{M}^* (x|y) p(y) = \sum_y \frac{\alpha}{n} p(y) + \left[ \left( 1 - \frac{\alpha (n-1)}{n} \right) - \frac{\alpha}{n} \right] p(x) = \frac{\alpha}{n} + (1 - \alpha) p(x) .$$

$$b) \quad \text{We generally show that the entropy increases if you mix a distribution with the uniform distribution.}$$

We prove this by considering the first two derivatives of the entropy functional with respect to the mixture parameter $\alpha$. Let

$$q(x) = (1 - \alpha) p(x) + \frac{\alpha}{n} ,$$

and recall $H(q) = -\sum_x q(x) \ln q(x)$ and $(X \ln X)' = X'((\ln X) + 1)$. It follows

$$\frac{\partial}{\partial \alpha} H(q) = -\sum_x \left[ -p(x) + \frac{1}{n} \right] \ln q(x) + 1 = \sum_x \left[ p(x) - \frac{1}{n} \right] \ln q(x) ,$$

$$\frac{\partial}{\partial \alpha} H(q) \bigg|_{\alpha=1} = \sum_x \left[ p(x) - \frac{1}{n} \right] \ln \frac{1}{n} = 0 ,$$

$$\frac{\partial^2}{\partial \alpha^2} H(q) = -\sum_x \frac{(p(x) - \frac{1}{n})^2}{q(x)} < 0 \quad \text{if } p \text{ is non-uniform.}$$
What we found is that (1.) the entropy is maximal for the extreme case \( \alpha = 1 \) since its derivative w.r.t. \( \alpha \) at this point vanishes (of course, this corresponds to the trivial case where \( q \) becomes the uniform distribution) and (2.) the second derivative is always negative if \( p \) is non-uniform. Hence, the plot of \( H \) versus \( \alpha \) is comparable to an upside-down parabola with maximum at \( \alpha = 1 \). It follows that for all \( \alpha < 1 \) (to the left of the maximum) the derivative \( \frac{\partial}{\partial \alpha} H(q) \) is positive. Entropy continuously increases with \( \alpha \). And hence, for every \( 0 \leq \alpha \leq 1 \) and every non-uniform population \( p \), \( H(M^*p) > H(p) \).

**Theorem 3.2.** Consider the simple mutation operator \( M(x|y) = \prod_i M^i(x^i|y^i) \) as given in definition 2.1. If \( p \in A^i \) is non-uniform it follows that entropy increases, \( H(Mp) > H(p) \), and mutual information decreases, \( I(Mp) < I(p) \).

**Proof.** We first prove that the cross entropy decreases. Assuming only two genes, the compound mutation distributions reads

\[
M p(x, y) = (1 - \alpha)^2 p(x, y) + (1 - \alpha) \alpha p(x) \frac{1}{n} + (1 - \alpha) \alpha \frac{1}{n} p(y) + \alpha^2 \frac{1}{n} \frac{1}{n}
\]

\[
= (1 - \alpha) \left[ (1 - \alpha) p(x, y) + \alpha \frac{1}{n} p(x) \right] + \alpha \frac{1}{n} \left[ (1 - \alpha) p(y) + \alpha \frac{1}{n} \right]
\]

\[
= (1 - \alpha) q(x, y) + \frac{1}{n} q(y),
\]

where \( q(x, y) = (1 - \alpha) p(x, y) + \alpha p(x) \frac{1}{n} \), \( q(x) = p(x) \), \( q(y) = (1 - \alpha) p(y) + \frac{\alpha}{n} \).

We call \( q \) a one-component \( \alpha \)-mixture since only in one component the uniform distribution was mixed to \( p \). This shows that the compound distribution \( Mp \) for two genes is a one-component \( \alpha \)-mixture of a distribution \( q \), which is itself a one-component \( \alpha \)-mixture. For compound distributions with more than two genes this will be recursively the case and generally the mutation operator can be expresses as concatenation of one-component \( \alpha \)-mixtures. Hence, it suffices when we prove that the mutual information decreases for one such step of one-component \( \alpha \)-mixing.

We use the same technique of calculating derivatives with respect to the mixture parameter to proof decreasing cross entropy. To simplify the notation we use the abbreviations:

\[
A = q(x, y), \quad A|_{\alpha=1} = \frac{\alpha p(x)}{n}, \quad A' = \frac{\partial}{\partial \alpha} A = -p(x, y) + \frac{p(x)}{n}, \quad A'' = 0, \quad A'' = 0,
\]

\[
B = q(x) q(y) = p(x) \left[ (1 - \alpha) p(y) + \frac{\alpha}{n} \right], \quad B|_{\alpha=1} = A|_{\alpha=1}, \quad B' = p(x) (-p(y) + \frac{1}{n}), \quad B'' = 0.
\]

With these abbreviations (keeping the dependencies on \( x, y \), and \( \alpha \) in mind) we can write:

\[
I(q) = \sum_{x,y} A \ln \frac{A}{B}
\]

\[
\frac{\partial}{\partial \alpha} I(q) = \sum_{x,y} \left[ A' \ln \frac{A}{B} + A' - \frac{A B'}{B} \right]
\]

\[
\frac{\partial^2}{\partial \alpha^2} I(q)|_{\alpha=0} = \sum_{x,y} A'|_{\alpha=1} \ln \frac{A|_{\alpha=1}}{A|_{\alpha=1}} + \sum_{x,y} \left[ -p(x, y) + \frac{p(x)}{n} \right] - \sum_{x,y} \frac{A|_{\alpha=1}}{A|_{\alpha=1}} p(x) (-p(y) + \frac{1}{n}) = 0
\]

\[
\frac{\partial^2}{\partial \alpha^2} I(q) = \sum_{x,y} \left[ A' \frac{B}{B} - \frac{A B'}{B^2} \right] + 0 - \frac{A' B'}{B} + A \left( \frac{B'}{B} \right)^2
\]

\[
= \sum_{x,y} \frac{(A')^2}{A} - 2 \frac{A' B'}{B} + A \left( \frac{B'}{B} \right)^2 = \sum_{x,y} \frac{(B A' - AB')^2}{A B^2} > 0
\]

So, what we found is that (1.) for \( \alpha = 1 \) the cross entropy is minimal since its derivative w.r.t. \( \alpha \) at this point vanishes (of course, this corresponds to the trivial case where \( q(x, y) = p(x) \frac{1}{n} \)) and (2.) for all other points the
second derivative is positive. The plot of $I$ versus $\alpha$ is comparable to an upwards parabola with minimum at $\alpha = 1$. It follows that for $\alpha < 1$ (to the left of the minimum) the derivative $\frac{d}{d\alpha} I(q)$ is negative and thus the cross entropy continuously decreases with increasing $\alpha$.

Concerning increasing entropy, it is obvious that the marginals of the mutation distribution $\mathcal{M}p$ are simply

$$(\mathcal{M}p)^i = \mathcal{M}^*p^i .$$

For the component-wise mutation operators we proved that entropy increases (for non-zero $\alpha$ and non-uniform $p$) and thus $\Delta_H I^i > 0$. Consequently,

$$\Delta_M H = \sum_i \Delta_H I^i - \Delta_M I > 0 .$$

\[\Box\]

## 4 The structure of the crossover distribution

What is the structure of the crossover search distribution $\mathcal{C}p$, given $p \in \Lambda^\Omega$ and $c \in \Lambda^{\{0,1\}^N}$? The first theorem can directly be derived from our definition of the crossover operator. It captures the most basic properties of the crossover operator with respect to the correlations it destroys in the search distribution:

**Theorem 4.1.** Let $H(p)$, $\alpha^i$, $H^i(p)$, and $I(p) = \sum_i H^i(p) - H(p)$ denote the entropy, the $i$th marginal distribution, the marginal entropies, and the mutual information of a distribution $p$. For any crossover operator $\mathcal{C}$ and any population $p$ it holds

a) $\forall i : \ (\mathcal{C}p)^i = p^i$, $\Delta_\mathcal{C}H^i = 0$, i.e., the marginals and hence their entropies do not change,

b) $\Delta_\mathcal{C}I = -\Delta_\mathcal{C}H \leq 0$, i.e., the increase of entropy is equal to the decrease of mutual information.

**Proof.** Let us first calculate the marginals after crossover. Let $a$ be an allele of the $i$th gene.

$$\mathcal{C}p^i(a) = \sum_{x_0,x_1} \sum_c c(m) \ [a = (x_m)^i] p(x_0) p(x_1) ,$$

$$= \sum_{x_0,x_1} \sum_{m,m_i=0} c(m) \ [a = (x_0)^i] p(x_0) p(x_1) + \sum_{x_0,x_1} \sum_{m,m_i=1} c(m) \ [a = (x_1)^i] p(x_0) p(x_1) ,$$

$$= p^i(a) \left( \sum_{m,m_i=0} c(m) \right) + p^i(a) \left( \sum_{m,m_i=1} c(m) \right) = p^i(a) .$$

Since the marginals are not changed by crossover, the marginal entropies do not change either. Statement b) follows from the definition of the mutual information:

$$\Delta_\mathcal{C}H + \Delta_\mathcal{C}I = H(\mathcal{C}p) - H(p) + I(\mathcal{C}p) - I(p)$$

$$= H(\mathcal{C}p) - H(p) + \sum_i H^i(\mathcal{C}p) - H(\mathcal{C}p) - \left[ \sum_i H^i(p) - H(p) \right]$$

$$= \sum_i H^i(\mathcal{C}p) - \sum_i H^i(p) = 0 .$$

\[\Box\]

The following theorem makes this more concrete when focusing on two specific genes of a genome of arbitrary length. We calculate the mutual information between these two genes in the search distribution $\mathcal{C}p$—which is a measure for the linkage between them. Let it be the $i$th and $j$th gene. We use $a$ and $b$ as alleles; $p^{ij}(a,b) = \sum_{x \in \Omega} [x^i = a] [x^j = b] p(x)$ denotes the probability that the $i$th gene has allele $a$ and the $j$th gene allele $b$. Analogously, let $c^{ij}$ be the marginal of the crossover mask distribution with respect to the two genes, i.e., $c^{ij} = \sum_{m \in \{0,1\}^N} [m^i = 0] [m^j = 1] c(m)$.

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Theorem 4.2. For any crossover operator $\mathcal{C}$ and any population $p$ it holds:

a) The compound distribution of two genes after crossover is given by

\[
(\mathcal{C}p)^{ij}(a,b) = 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b),
\]

i.e., a linear combination of the original compound distribution $p^{ij}(a,b)$ and the decorrelated product distribution $p^i(a)p^j(b)$.

b) The mutual information $I(\mathcal{C}p)^{ij}$ in the compound distribution of two specific genes is

\[
I(\mathcal{C}p)^{ij} = \sum_{a,b} \left( 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right) \ln \left( \frac{2c^{ij}(00)p^{ij}(a,b)}{p^i(a)p^j(b)} + 2c^{ij}(01) \right),
\]

c) and we have

\[
0 \leq 2c^{ij}(00) \left( I(p)^{ij} + \ln(2c^{ij}(00)) \right) \leq I(\mathcal{C}p)^{ij} \leq I(p)^{ij}.
\]

The two left $\leq$ are exact for complete crossover, $c^{ij}(00) = 0$, $c^{ij}(01) = \frac{1}{2}$, the right $\leq$ is exact for no crossover, $c^{ij}(00) = \frac{1}{2}$, $c^{ij}(01) = 0$.

Proof. a)

\[
\mathcal{C}p^{ij}(a,b) = \sum_{x_0,x_1} \sum_{m} c(m) [(x_{m_0})^0 = a][x_{m_1}]^1 = b] p(x_0) p(x_1)
\]

\[
= \sum_{x_0,x_1} \left( c^{ij}(00) [(x_0)^0 = a][(x_0)^1 = b] + c^{ij}(01) [(x_0)^0 = a][(x_1)^1 = b] \right) p(x_0) p(x_1)
\]

\[
= 2 \sum_{x_0} c^{ij}(00) [(x_0)^0 = a][(x_0)^1 = b] p(x_0) + 2 \sum_{x_0,x_1} c^{ij}(01) [(x_0)^0 = a][(x_1)^1 = b] p(x_0) p(x_1)
\]

\[
= 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b).
\]

b&c)

\[
I(\mathcal{C}p)^{ij} = H(\mathcal{C}p^i) + H(\mathcal{C}p^j) - H(\mathcal{C}p) = H(p^i) + H(p^j) - H(p)
\]

\[
\leq H(p^i) + H(p^j) - H(p) = I(p)^{ij}
\]

\[
H(\mathcal{C}p) = -\sum_{a,b} \left( 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right) \ln \left( 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right)
\]

\[
= -\sum_{a,b} \left( 2c^{ij}(00)p^i(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right) \left[ \ln \left( \frac{2c^{ij}(00)p^{ij}(a,b)}{p^i(a)p^j(b)} + 2c^{ij}(01) \right) - \ln p^i(a) - \ln p^j(b) \right]
\]

\[
= -\sum_{a,b} \left( 2c^{ij}(00)p^i(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right) \left[ \ln \left( \frac{2c^{ij}(00)p^{ij}(a,b)}{p^i(a)p^j(b)} + 2c^{ij}(01) \right) \right] + H(p^i) + H(p^j)
\]

\[
I(\mathcal{C}p)^{ij} = \sum_{a,b} \left( 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right) \ln \left( 2c^{ij}(00)p^{ij}(a,b) + 2c^{ij}(01)p^i(a)p^j(b) \right)
\]

\[
\geq \sum_{a,b} \left( 2c^{ij}(00)p^{ij}(a,b) \right) \ln \left( 2c^{ij}(00)p^{ij}(a,b) \right) = 2c^{ij}(00) \left( I(p)^{ij} + \ln(2c^{ij}(00)) \right)
\]
Correlated exploration

What crossover and EDAs share is that both introduce a non-trivial correlational structure in the search distribution. The crucial difference is that Estimation-of-Distribution Algorithms try to “carry over” the correlations in the population of selected to the search distribution whereas crossover destroys correlations. Carrying over correlations is non-trivial if the search distribution is to be explorative, i.e., of more entropy: Typically mutation operators add entropy to the distribution by adding independent noise to each marginal, but this reduces the mutual information between genes (see Lemma 3.2).

Consider illustration 2. In a finite population of 3 individuals, marked by crosses, the values at the 1st and 2nd loci are correlated (here illustrated by plotting them on the bisecting line). The crossed population $C_p$ comprises at most 9 different individuals; in the special cases $c^{ij}(01) = 0$ and $c^{ij}(01) = \frac{1}{2}$ the population is even finite and comprises 3 respectively 9 equally weighted individuals marked by circles. Mutation adds independent noise, illustrated by the gray shading, to the alleles of each individual. The two illustrations for the GA demonstrate that crossover destroys correlations between the alleles in the initial population instead...
of carrying it over to the search distribution: the gray shading is not focused on the bisecting line. Instead, an EDA would first estimate the distribution of the individuals in \( p \). Depending on what probabilistic model is used, this model can capture the correlations between the alleles; in the illustration the model could be a Gaussian parameterized by the mean and covariance matrix (just as for the CMA evolution strategy (Hansen & Ostermeier 2001)) and the estimation of the covariance in \( p \) leads to the highly structured search distribution in which the entropy of each marginal is increased without destroying the correlations between them. We capture this difference in the following definition:

**Definition 5.1 (Correlated exploration).** Let \( \mathcal{U} : \Lambda^\Omega \rightarrow \Lambda^\Omega \) be an operator. The following conditions need to hold for almost all \( p \in \Omega \) which means: for all the space \( \Omega \) except for a subspace of measure zero. We define

- \( \mathcal{U} \) is explorative \( \iff \Delta_{\mathcal{U}} H > 0 \) for almost all \( p \in \Omega \),
- \( \mathcal{U} \) is marginally explorative \( \iff \mathcal{U} \) is explorative and \( \exists i : \Delta_{\mathcal{U}} H^i > 0 \) for almost all \( p \in \Omega \),
- \( \mathcal{U} \) is correlated explorative \( \iff \mathcal{U} \) is explorative and \( \Delta_{\mathcal{U}} I > 0 \), or equivalently \( 0 < \Delta_{\mathcal{U}} H < \sum_i \Delta_{\mathcal{U}} H^i \), for almost all \( p \in \Omega \).

**Corollary 5.1.** From this definition it follows that

a) If and only if there exist two loci \( i \) and \( j \) such that the marginal crossover mask distribution \( c_{ij}(01) \) for these two loci is non-vanishing, \( c_{ij}(01) = c_{ij}(10) > 0 \), then crossover \( \mathcal{C} \) is explorative. For every mask distribution \( c \in \Lambda^{[0,1]^N} \), crossover \( \mathcal{C} \) is neither marginally nor correlated explorative.

b) Mutation \( \mathcal{M} \) is marginally but not correlated explorative.

c) Mutation and Crossover \( \mathcal{M} \circ \mathcal{C} \) are marginally but not correlated explorative.

d) In the case of a non-trivial genotype-phenotype mapping mutation as well as crossover can be phenotypically correlated explorative.

**Proof.** a) That \( \mathcal{C} \) is neither marginally nor correlated explorative follows directly from Theorem 4.1a, which says that for every \( c \in \Lambda^{[0,1]^N} \) and any population \( p \in \Lambda^\Omega \) the marginals of the population do not change under crossover, \( \Delta_{\mathcal{C}} H^i = 0 \). But under which conditions is \( \mathcal{C} \) explorative?

If, for two loci \( i \) and \( j \), \( c_{ij}(01) \) is non-vanishing, it follows that \( \mathcal{C} \) reduces the mutual information between these two loci (Theorem 4.2c). The subspace of populations \( p \) that do not have any mutual information \( I_{ij} \)
between these two loci is of measure zero. Hence, for almost all \( p \), \( \Delta_C^{ij} < 0 \) and, following Theorem 4.1b this automatically leads to an increase of entropy \( \Delta_C^{H^{ij}} > 0 \) in the compound distribution of the two loci and, since \( \Delta_C H \geq \Delta_C^{H^{ij}} \), also of the total entropy.

The other way around, if, for every two loci \( i \) and \( j \), \( e^{ij}(01) \) vanishes it follows that there is no crossover, i.e., on the all-0s and all-1s crossover masks have non-vanishing probability. Hence, \( C = \text{id} \) and is not explorative.

b) In lemma 3.2 we prove that for every non-uniform population \( p \Delta_M H > 0 \), \( \Delta_M H > 0 \), and \( \Delta_M I < 0 \).

c) Since both mutation and crossover are not correlative, it follows that their composition is also not correlative:

\[
\Delta_C I \leq 0, \ \Delta_M I \leq 0 \quad \Rightarrow \quad \Delta_M C I \leq 0.
\]

d) What is different in the case of a non-trivial genotype-phenotype mapping? The assumptions we made about the mutation operator (component-wise independence) hold only on the genotype space, not anymore on the phenotype space: On genotype space mutation kernels are product distributions and mutative exploration is marginally explorative but not correlated; projected on phenotype space, the mutation kernels are in general not anymore product distributions and hence phenotypic mutative exploration can correlated.

6 Conclusion

There are three main points to conclude:

- First, we point out that crossover does the inverse of correlated exploration. It destroys correlations in the exploration distribution by transforming them into entropy. In an information theoretic sense, the exploration distribution after crossover is less complex (carrying less mutual information) that before crossover. To me it seems just “countersensible” to base the notion of building blocks on a discussion of crossover. The most natural building blocks are individuals carrying the mutual information between genes within the exploration distributions. Crossover is splitting these building blocks in smaller ones.

- Of course, the crossover exploration distribution can be modeled by graphical models since graphical models can model any distribution. In that respect, one could certainly design search algorithms based on probabilistic models of the search distribution (instead of a population) that model crossover GAs—the PBIL is a candidate. However, I would challenge to call such an algorithm an Estimation-of-Distribution algorithm because its objective is not to really estimate the distribution of selected and in particular the correlations within this distribution (with the exception of PBIL who’s objective is to only estimate the marginals which coincides with modeling crossover). In general, EDAs go beyond modeling crossover since they introduce a quality which is not a quality of crossover: correlated exploration.

- Finally, there is a crucial difference between EDAs and (crossover) GAs with respect to the self-adaptation of the exploration distribution. EDAs always adapt their search distribution (including correlations) according to the distribution of previously selected solutions. In contrast, the crossover mask distribution, that determines where correlations are destroyed or preserved, is usually not self-adaptive. However, if considering a phenotypic level (i.e., if we consider an indirect encoding) then both, mutational exploration and crossover exploration can be correlated and self-adaptive on the phenotypic level (see the theory on \( \sigma \)-evolution in (Toussaint 2003b), realizing that both, the definition of mutation and the definition of crossover do not commute with phenotypic equivalence).

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