THE LIVING ADDRESS

Induction and Repression: The araBAD Operon

The Switch That Reverses

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In which a second bacterial switch breaks the rule the first one taught — for here a single protein both closes the genes and opens them, its direction reversed by the sugar it senses; and to do the closing it reaches across two hundred letters of DNA by folding the address so the far becomes near.

Tau (T) is the living fabric of time itself — the sole substance of which all physical reality is composed. Every particle, force, wavelength, and conscious experience is a structured configuration of T-flow. There is no gravity, no electromagnetic force, no strong nuclear force as separate entities: all are registers of the single T-field operating across dimensional levels. The conservation law $d\Sigma T=0$ governs all change: T is never created or destroyed, only redistributed.

Introduction — Through the Force of Time

The chapter that follows is, in the conventional telling, the operon that overturned a dogma — the arabinose system, run by a protein that turns genes on, and by the same protein that turns them off. Read through the Universal Force of Time, it is a T-gate whose very polarity is reversed by the state of the field, and whose reach is made non-local by folding the address.

Chapter Eleven showed one address held between two hands — a protein that closes it and a different protein that opens it. This chapter brings a deeper surprise, and it once met real hostility, because it broke the assumption that all regulation was repression. Here a single protein, AraC, does both jobs. Without the sugar arabinose it shuts the genes; with arabinose it opens them. The sugar does not pull one hand away and bring in another. It flips the same hand. In this book that is a single T-gate whose sign is set by the environmental condition — a switch that reverses its own direction according to the local state of T. Regulation, it turns out, need not be a fixed lock; it can be a coupling whose very polarity the field's state decides.

The way it shuts the genes is the chapter's second marvel. AraC grips two sites on the DNA that lie some two hundred letters apart, and by holding both at once it bends the intervening stretch into a loop — so that a protein bound far from the promoter reaches

across and closes it. Biologists named this ‘action at a distance,’ and it is now known to be a general trick of the address. In the reading of this book it is non-local control within one coordinate: the address is not a line read strictly left to right, but a foldable object whose distant regions can be brought into contact to gate one another. When arabinose arrives, the loop breaks; the same protein, now settled at the near site, helps the reading begin. The conserved topology of Chapter Two — the address’s power to fold — is here the very substance of the switch.

And a smaller detail carries the same spirit. Arabinose is drawn into the cell by two separate pumps — one that grips it tightly, for catching the faintest trace, and one that grips it loosely, for a rich supply. The cell reads how much of the resource is present — its T-density — and deploys the receiver tuned to that amount, exactly as, elsewhere in this book, a pigment is tuned to the wavelength it is built to receive. Reading the world finely, and answering it finely, runs all the way down.

Carry this into the chapter: the arabinose operon is a T-gate whose polarity the field’s own state reverses, and whose reach is made non-local by folding the address so a distant point acts locally. Regulation is not merely open or shut — it is reconfigurable, foldable control over a coordinate.

SECTION 12.1

The Switch That Reverses

When the lactose switch of the last chapter was first worked out, biologists drew a natural conclusion: this is how genes are controlled — a repressor sits on the DNA and holds the genes shut until a signal lifts it. Control meant repression, and repression meant a lock removed. So sure was the field of this that when evidence arrived, from a second sugar, of a protein that did the opposite — that turned genes on rather than off — it was met not with delight but with resistance. Surely nature would not do the same job in two different ways. But it does, and in far more than two; and the switch that first taught this lesson is one of the most elegant in all of biology, because it does not merely add a new kind of control. It shows that a single switch can run in both directions.

The sugar is arabinose, and its genes are governed by a protein called AraC. Take arabinose away, and AraC holds the genes shut. Add arabinose, and the very same AraC throws them open. One protein, one binding on the DNA, and yet two opposite outcomes — repression or activation — decided entirely by whether the sugar is present. In this book that is a striking thing: not two hands, one to close and one to open, as we saw with lactose, but one hand whose direction the environment reverses. It is the first switch we meet whose very polarity is set by the state of the world, and understanding how it manages that reversal is the work of this chapter.

SECTION 12.2

A Sugar From the Salad

First the sugar itself, because it is more familiar than its name suggests. Arabinose is one of the simple sugars locked up in the walls of plant cells — it is, quite literally, part of the crunch of a raw vegetable. Human beings cannot digest it; we lack the enzymes. But the bacteria that live in our gut can, and so when you eat a salad, the arabinose in it arrives in the intestine as a free meal for the microbes that live there. It is a small, homely fact, and it is the whole reason the arabinose genes exist: they are the toolkit a gut bacterium switches on to make a meal of the plant matter we cannot use ourselves.

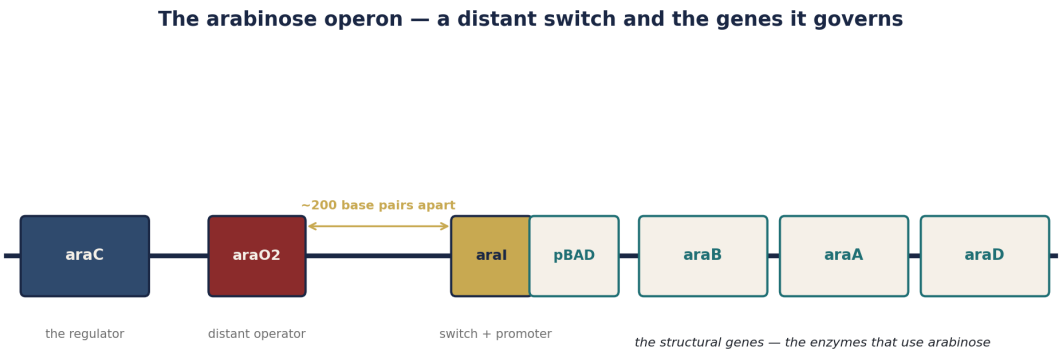

Figure 12.1. The arabinose operon. A regulator gene, araC, makes the AraC protein. A distant operator site, araO2, lies about two hundred letters upstream of the switch-and-promoter region (araI and pBAD); after them come the structural genes araB, araA, and araD, the enzymes that use arabinose.

The genes for using it are arranged, as in the last chapter, as an operon — a run of structural genes read together, with a control region in front. But the control region here has an unusual feature that will matter greatly: besides the switch that sits right at the promoter, there is a second control site set far away, some two hundred letters upstream. Hold that distance in mind. Two hundred letters is a long way along the DNA, far too far, you would think, for a protein bound there to have any say over a promoter so distant. And yet it does — and how it does is the heart of the chapter.

SECTION 12.3

The Protein of Two Minds

Consider first the ordinary state of affairs, when the bacterium sits in arabinose. AraC, with the sugar bound to it, settles at the site right beside the promoter and does a favour to the reading machine: it helps the RNA polymerase take hold and begin. This is positive control — not the mere absence of a block, but an active hand extended to help. The genes are read strongly, the enzymes are built, the meal is

used. This was the shock to the early biologists: a regulator whose job, in the presence of its signal, is to say yes.

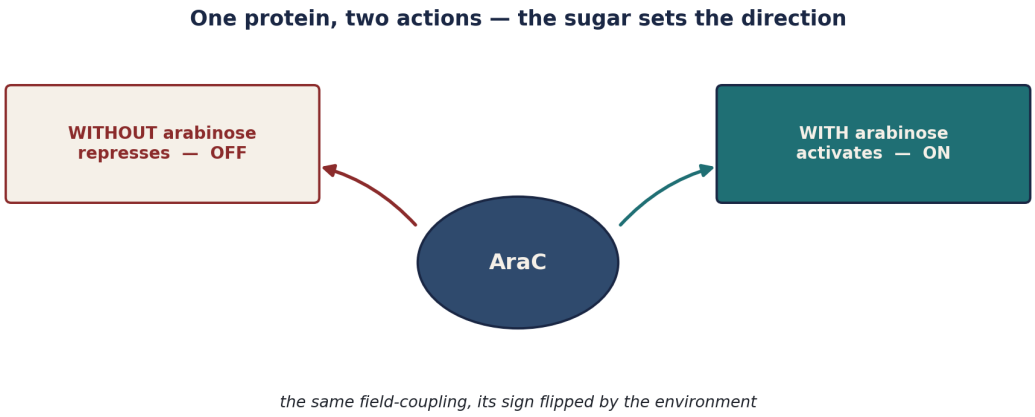

Figure 12.2. One protein, two actions. With no arabinose, AraC represses the genes; with arabinose, the very same protein activates them. The sugar does not swap one regulator for another — it flips the direction of a single one.

Now take the arabinose away. You might expect the helper simply to fall idle, leaving the genes at some quiet resting level. That is not what happens. Without arabinose, AraC does not merely stop helping — it actively represses, clamping the genes more firmly shut than their idle state would be. The same protein that says yes with the sugar says a definite no without it. It is a regulator of two minds, and which mind it is in is decided by one thing only: whether the arabinose is there to be sensed. In the reading of this book, AraC is a single T-gate whose sign — opening or closing — is set by the environmental condition. The field-coupling does not change; its polarity does.

The evidence that forced this on a reluctant field was beautifully simple. If AraC were only a repressor, like the lactose one, then destroying it ought to leave the genes stuck open — with the lock gone, they would be read freely. But when researchers knocked the araC gene out, they found exactly the opposite: the arabinose enzymes vanished. No AraC meant no reading at all. A missing lock does not silence a gene; a missing helper does. The only way to read the result was that AraC’s normal job, in the presence of arabinose, is to switch the genes on — and so the first hard proof of a positive regulator came not from watching the protein work but from removing it and seeing the genes fall silent.

SECTION 12.4

Action at a Distance

How can one protein clamp the genes shut? Here the far-off control site earns its keep. Without arabinose, AraC grips two places on the DNA at once — the near site,

beside the promoter, and the distant one, two hundred letters upstream. A single protein cannot span such a gap along a straight molecule. So it does not keep the molecule straight. Holding both sites, it draws them together, and the long stretch of DNA between them is forced to bow out into a loop. With the loop closed, the promoter is caught up in a knot of protein and folded DNA, and the reading machine cannot work. The genes are shut — by a protein bound two hundred letters away.

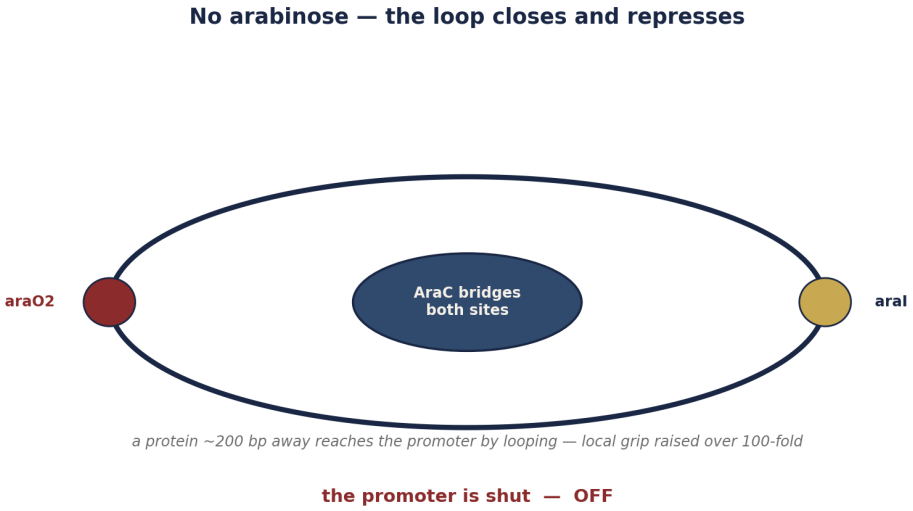

Figure 12.3. Action at a distance. With no arabinose, AraC grips the near site and the distant one at once, bending the intervening DNA into a loop. A protein bound far from the promoter thereby reaches it — and by folding, its local grip on the far site is raised more than a hundredfold.

Biologists gave this a suitably dramatic name — action at a distance — and it was first understood right here, in the arabinose system, before being found across the living world as a general trick. The looping is not a curiosity; it is a genuine amplifier. By bending the address so the far site is held near, the effective concentration of AraC at that distant place is raised more than a hundredfold, which is why so weak and remote a grip can nonetheless slam the switch. In the reading of this book, this is non-local control within a single coordinate. The address is not a tape read strictly from one end to the other, with each point mattering only to its neighbours. It is a foldable object, and two places far apart along its length can be brought into contact and made to govern each other. Distance along the DNA is not distance in the field’s bookkeeping; what is far can be folded near.

How can anyone be sure a molecule is truly looping, and not doing something simpler? The proof is elegant, and it turns on the twist of the double helix. Researchers changed the spacing between the two sites, adding or removing letters a few at a time, and watched whether the repression survived. Because the helix turns through a full circle about every ten or eleven letters, adding half that many puts the far site on the opposite face of the DNA — pointing away from its partner — while adding a whole turn’s worth brings it back into line. Repression came and went with this rhythm: strong

when the two sites faced the same way and could be brought together, lost when they faced apart and no loop could form. Only a loop behaves like that. The switch was reading the geometry of the helix itself, and so revealed the fold by the way it failed.

SECTION 12.5

The Ligand Sets the Polarity

Now watch the reversal happen. Arabinose enters the cell and binds to AraC, and that binding changes the protein's shape — and with it, its behaviour. The grip that held the two distant sites together loosens; the loop springs open. Freed from the loop, AraC settles instead at the near site alone, in the pose that helps rather than hinders, and the reading machine is welcomed in. The switch has not been replaced. The same protein, on the same DNA, has been turned from a jailer into a doorman by the arrival of a single small molecule.

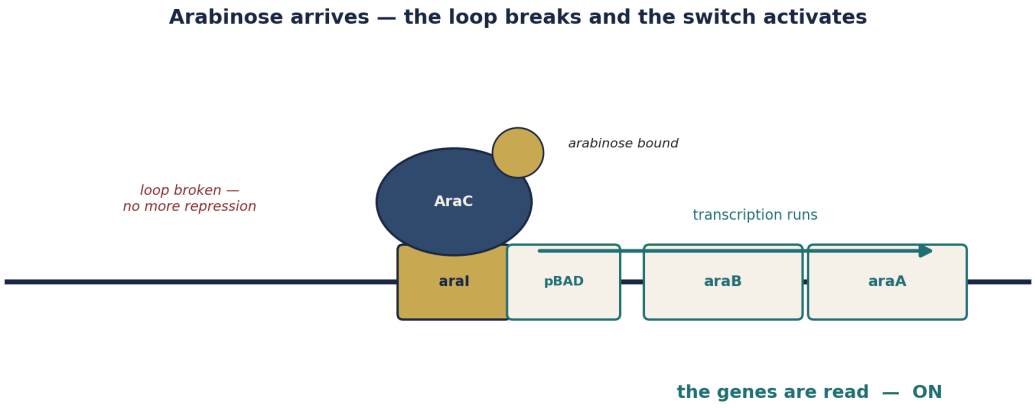

Figure 12.4. The reversal. Arabinose binds AraC and breaks the loop; the protein, now settled at the near site, helps the reading machine begin. One small molecule has turned the same regulator from a repressor into an activator.

This is the chapter's deepest lesson, and in this book it is worth stating plainly. In the lactose switch, on and off were two different proteins — a closing hand and an opening hand — and the environment chose between them. Here there is one hand, and the environment reverses it. The sugar is not merely permission to read; it is the thing that sets the sign of the gate. A T-gate, in the reading of the Universal Force of Time, need not have a fixed direction built into it. Its polarity can itself be a variable, set moment to moment by the local state of T — here, by the presence or absence of one sugar. Regulation is not only a matter of which switches are thrown; it can be a matter of which way a switch is currently wired, and the world does the wiring.

KEY IDEA

AraC is one protein with two opposite actions. Without arabinose it grips a near site and a distant one at once, looping the DNA to shut the promoter — action at a distance, its far grip raised over a hundredfold by the fold. With arabinose it changes shape, the loop breaks, and the same protein helps the reading begin. In this book, AraC is a single T-gate whose polarity — open or shut — is set by the environment, and whose reach is made non-local by folding the address.

SECTION 12.6

Two Nets for One Fish

One more detail of the arabinose system carries the same spirit of finely reading the world, and it concerns not the genes but the getting of the sugar in the first place. Before arabinose can be used, it must be pulled into the cell from outside, and the bacterium keeps not one pump for this but two. One of them grips arabinose loosely; it works well only when the sugar is abundant, scooping up a rich supply. The other grips it tightly; it can catch arabinose even when it is present only in the faintest trace, at concentrations around a ten-millionth of a mole in a litre. Two nets for one fish — a coarse net for a shoal, a fine net for a single swimmer.

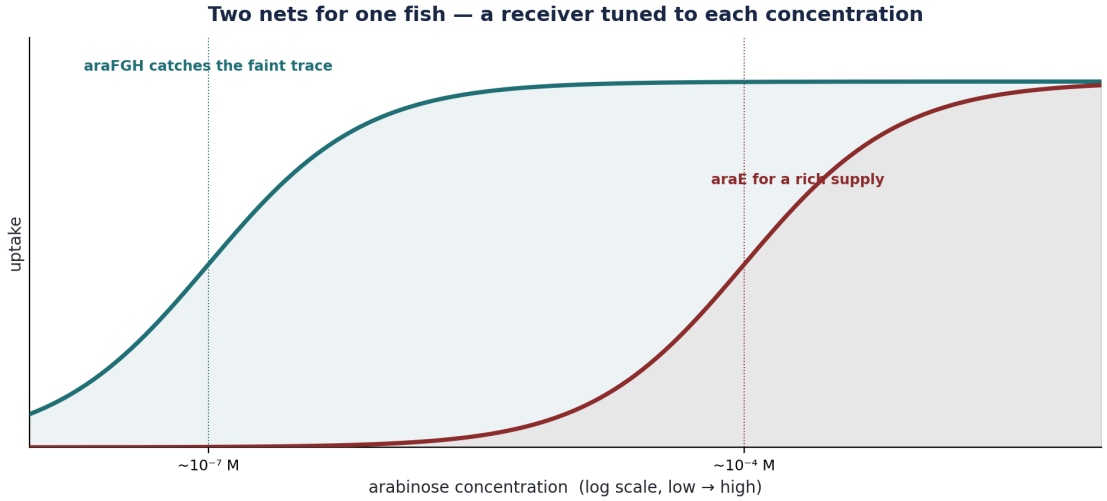

Figure 12.5. Two nets for one fish. A high-affinity pump (araFGH) draws arabinose in even at the faintest trace, around 10^{-7} moles per litre; a low-affinity pump (araE) serves when the sugar is plentiful. Each is tuned to a different range of concentration.

In the reading of this book, this is the node matching its intake to the T-density of the resource. The cell does not have one all-purpose receiver; it reads how much of the sugar the world is offering and deploys the catcher tuned to that amount — the tight grip for scarcity, the loose grip for plenty. It is the same principle we meet elsewhere in this work, where a pigment is built to receive a particular wavelength and a particular register of the field: reception is tuned to what is to be received. A living thing reads its

world not coarsely but at graded resolution, and answers each level with the instrument built for it. Even the humble business of taking in a sugar is a matter of reading the field's density and replying in kind.

There is a further nicety, easy to miss. Arabinose in solution is not one rigid shape but flickers between two ring forms, turning from one into the other and back with a half-time of about ten minutes; and it is thought that the two pumps may favour different forms. If so, the cell is reading its resource more finely still — not only how much sugar is present, but in which of its shifting shapes — and matching a receiver to each. It is a small thing, and not fully settled, but it points the same way as everything else in this chapter: a living node reads the world at fine grain, down to the very conformation of a molecule, and answers each detail with an instrument tuned to it.

SECTION 12.7

The Foldable Address

Step back and let the two great features of this switch settle together, because between them they change how we should picture the address itself. The first is that a single regulator can be reversed — that the sign of a control is not fixed but set by the world. The second is that control can act at a distance, a protein reaching a promoter two hundred letters away by folding the DNA between. Neither fits the simple picture of a tape read left to right, each site speaking only to its neighbours. Both say that the address is something richer: a reconfigurable, foldable object.

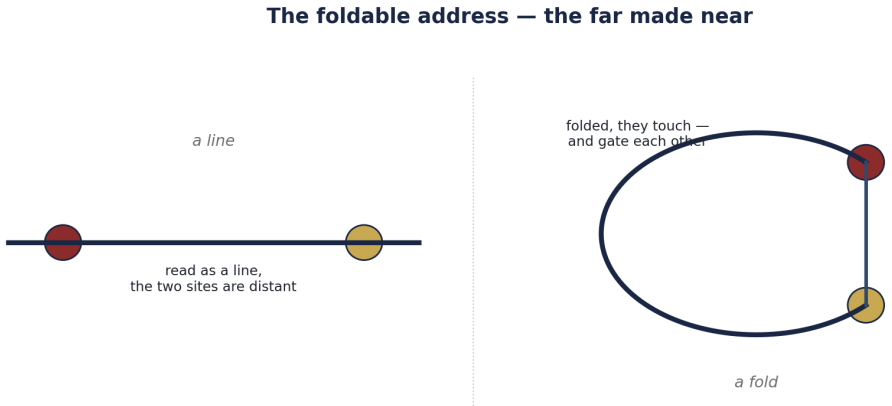

Figure 12.6. The foldable address. Read as a line, two control sites are distant and unrelated. Folded, they are brought into contact and gate one another. Distance along the DNA is not distance in the field's reckoning.

We first met the folding of DNA, chapters ago, as topology — the mathematics of how a molecule twists and coils, its total linkage conserved through every rearrangement. That looked like bookkeeping, a housekeeping fact about how a long thread is stored. Here, for the second time, we find the cell putting that same foldability to work as a

device for control — and in the arabinose switch more boldly than anywhere yet, reaching across two hundred letters to govern a promoter. In the terms of the Universal Force of Time, a coordinate in the field is not a line but a form. Its distant regions can be curved into contact, so that what is far along its length becomes near in its action, and points that never touch on a map can gate each other in the flesh. The address is read, but it is also folded; and the folding is not incidental to the reading — it is one of the ways the reading is governed.

SECTION 12.8

Why This Should Matter to You

It is easy to think of a gene as a fixed instruction — a line in a book, either read or not. The arabinose switch shows how far from that the truth is. A control can run forwards or backwards depending on a single molecule in the surroundings; a protein can govern a gene it sits nowhere near, by folding the intervening DNA into a loop. The address of life is not a passive list. It is a responsive, foldable thing, whose reading is shaped by the world outside and by the shape the molecule takes in the moment. Your own genes are governed by exactly such devices — regulators that reverse, controls that reach across great stretches of folded DNA — multiplied and elaborated far beyond anything a bacterium needs.

And there is a reason this matters beyond the marvel of it. When this book turns, later, to health and disease, the switches of these chapters are where much of the story will be told — because a control reversed at the wrong moment, or a fold that brings the wrong two places together, is exactly the kind of small misreading that turns an orderly cell disorderly. To see that a switch can be reversed, and that the address can be folded to make the far act near, is to see two of the ways the reading of a coordinate can go right — and, if disturbed, wrong. In the next chapters the controls grow more layered still, coupling the reading of a gene to the very machinery that translates it, and weaving single switches into circuits; but the arabinose operon has already shown us the two ideas that matter most — that a gate can reverse, and that an address can fold.

The Numbers at a Glance

The parts of the switch, and the reading the Force of Time gives each. Every biological fact is left exactly as measured.

Part of the switch	What it does	The Force of Time reading
AraC + arabinose	activates the genes (positive)	a T-gate set to open by the field's state
AraC without arabinose	represses the genes (negative)	the same gate, its polarity reversed
The distant operator	~200 base pairs from the promoter	a far point folded near to act locally
The DNA loop	raises the far grip >100-fold	the address's foldability used as a switch
Two transporters	araFGH tight, araE loose	intake tuned to the resource's T-density
High-affinity uptake	catches arabinose near 10^{-7} M	reception tuned to the faintest trace

References

[1] S. Daubney, The Universal Force of Time — Master Compendium v5, The Daubney Foundation (2026).

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[3] S. Daubney, The Force of Time — Where It Departs From Current Science, The Daubney Foundation (2026).