THE LIVING ADDRESS

# Attenuation and the trp Operon

*The Molecule That Decides for Itself*

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*In which no protein need sit on the gene at all — for here the message decides its own fate as it is written, reading the scarcity of a single amino acid through the speed of a ribosome, and folding itself, at a fork in the road, into one of two shapes: one that says stop, one that says go on.*

*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, attenuation — the control of transcription by the folding of the messenger RNA itself, worked out in the tryptophan operon. Read through the Universal Force of Time, it is the field letting a molecule decide its own fate: by clocking the supply of one amino acid through the speed of a ribosome, and collapsing onto one of two folds.*

The last two chapters gave us switches worked from outside — a protein sits on the DNA and holds a gene shut or helps it open. This chapter is stranger and, in the reading of this book, deeper: here there is no master protein sitting on the operon at all. The messenger RNA computes its own fate as it is being made. The cell needs to tell three situations apart — tryptophan is short and the cell can build; tryptophan is short but the cell cannot build; tryptophan is plentiful — and it should switch the tryptophan factory on in only the first. A simple lock, of the kind we have seen, cannot tell all three apart. Attenuation can, and it does so by a trick of pure timing.

At the front of the operon sits a short decoy gene, a leader, encoding a tiny peptide that happens to contain two tryptophan codons in a row. The cell translates this leader first, and everything turns on how the reading goes. If tryptophan is scarce, the ribosome reaches those two codons and stalls, waiting for the amino acid that is not there; if tryptophan is plentiful, it races straight through. And the position of that ribosome decides which of two mutually exclusive shapes the growing RNA folds into —

a terminator hairpin that halts transcription on the spot, or an antiterminator that lets it run on into the genes. A stalled ribosome yields the ‘go’ fold; a fast one yields the ‘stop’ fold. The ribosome’s speed is the sensor.

Here is the reading this book gives it, and it is the heart of the chapter. The ribosome’s speed over those two codons is set by one thing: how much charged transfer-RNA for tryptophan is on hand — the local T-supply of a single amino acid. The fixed reading-tempo of the register, which ran quietly beneath the chapters on transcription and translation, is here sensed and acted upon: a rate is converted into a structure, and the structure decides whether the field keeps building. And because a continuous speed collapses onto one of just two allowed folds, it is quantisation once more — and because translation and transcription tick on the one register clock, it is a single T-clock feeding back on itself.

*Carry this into the chapter: attenuation is the field letting a molecule decide for itself — clocking the supply of one amino acid through the speed of a ribosome, and collapsing onto one of two folds. Regulation here is not an external switch thrown upon the address, but a computation the address performs on its own reading-rate; and the cell builds only in the one state of three that its circumstances warrant.*

SECTION 13.1

## The Molecule That Decides for Itself

The last two chapters showed the address governed from outside. A protein — a repressor, an activator — settles on the DNA and holds a gene shut or eases it open, and the environment tips the balance. It is control by a hand laid on the switch. This chapter shows something altogether different, and, in the reading of this book, something deeper: control with no master hand at all. Here the messenger RNA, the working copy of the gene, decides its own fate as it is being made — folding itself, part way through, into one of two shapes, one of which quietly halts its own completion. The molecule computes whether to finish building itself. It is the first time in this book that the address does not merely get read or not read, but performs a small calculation upon its own reading.

The problem it solves is subtler than any we have met, and it is worth stating carefully, because the cleverness of the mechanism is a direct answer to it. Consider a bacterium that can make its own tryptophan, one of the twenty amino acids from which all its proteins are built. Building tryptophan takes a whole assembly line of enzymes, and making those enzymes is costly. The cell should build the tryptophan factory only when two things are true at once: tryptophan is genuinely running short, and the cell is in a position to do something about it — that is, it is actively making proteins and so actually needs the amino acid. There are three situations to tell apart, and only one of them warrants switching the factory on. A simple lock cannot distinguish all three. The

tryptophan operon evolved a way that can, and its name is attenuation.

SECTION 13.2

## The Amino Acid and Its Factory

Begin with the factory itself. Tryptophan is made from simpler materials along a branching pathway that the cell shares with the making of two other amino acids. The very first step, common to all three, is catalysed by an enzyme with the forbidding name of DAHP synthetase, and because that step sits at the head of the whole pathway and cannot easily be reversed, it is the natural place to exert control. And the cell controls it in a beautifully direct way: the finished tryptophan reaches back and blocks that first enzyme. When tryptophan is abundant, it switches off the very machine that begins its own manufacture; when it is scarce, the block lifts and production resumes.

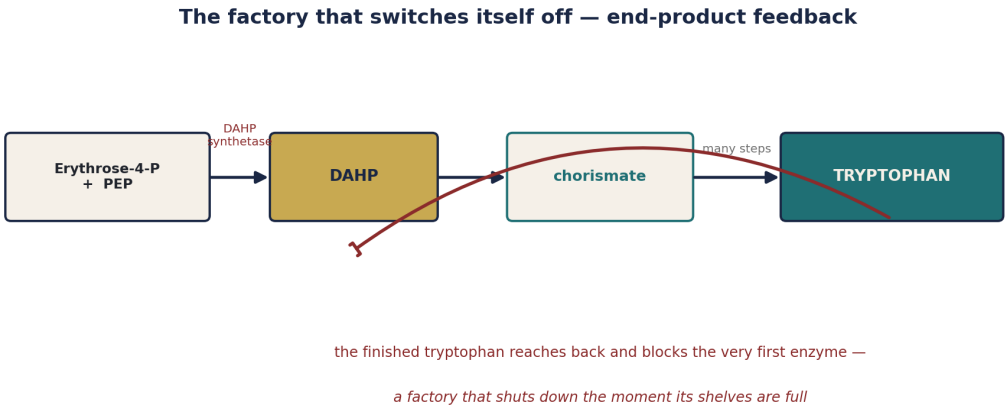


**Figure 13.1.** Feedback inhibition. Tryptophan is built along a pathway whose first committed step is catalysed by DAHP synthetase. The finished amino acid reaches back and blocks that first enzyme — a factory that shuts itself down the moment its shelves are full.

This kind of arrangement is called feedback inhibition, and it is worth pausing on in this book's terms because it is the plainest possible image of a conserved quantity managing itself. The amount of tryptophan is a small local pool of T-structured matter, and the pathway that makes it is fast to respond to its own product: enough on the shelves, and the line stops; shelves emptying, and it starts. The rate of manufacture is tied directly to the size of the pool, so that the pool is held roughly steady — a body of T kept in balance by a loop that reads its own level. It is the conservation law made homely: the cell does not pour endless effort into making what it already has, but tracks the pool and spends only to keep it filled. And note that this control acts on the enzyme that is already present — it throttles the machine. To control how many machines get built in the first place is a separate matter, and that is where attenuation comes in.

SECTION 13.3

## The Operon and Its Leader

The genes for the tryptophan enzymes are gathered, as we have come to expect, into an operon: five structural genes in a row, read together as a single message, with a control region in front. There is the usual promoter where transcription begins, and there is even a repressor — a protein that, helped by tryptophan itself, can sit at the start and block much of the initiation when the amino acid is plentiful. That much is familiar from the lactose story. But between the promoter and the first structural gene lies something new and decisive: a stretch of about a hundred and sixty letters, transcribed into RNA before any of the real genes, called the leader.

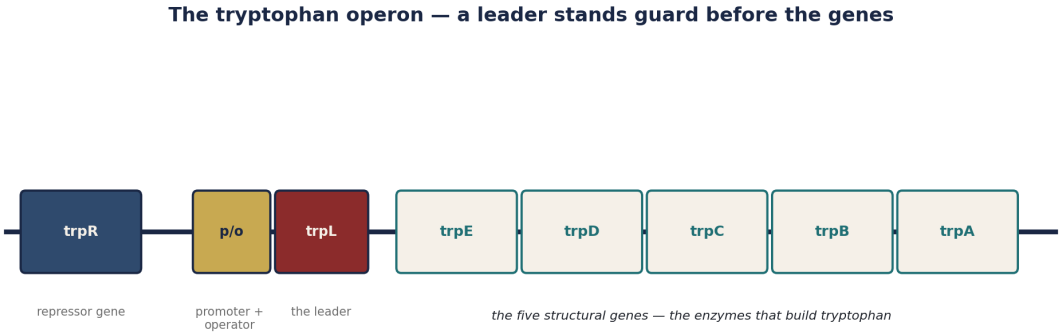


**Figure 13.2.** The tryptophan operon. A separate repressor gene, trpR, and a promoter-operator region precede a short leader, trpL, and then the five structural genes trpE through trpA. The leader — transcribed first, before any real gene — is where the decision of this chapter is made.

The leader looks, at first, like a meaningless preamble. It is not. It turns out to be the whole seat of the decision. As the RNA polymerase transcribes this leader and moves toward the structural genes, it passes a point at which transcription can either stop dead or carry on. Whether it stops or carries on — the ratio of aborted messages to complete ones — is what the cell regulates, and it regulates it not by any protein binding the leader, but by the shape the leader RNA folds into as it emerges. In the reading of this book, the decision has been moved off the DNA and onto the freshly made RNA itself: the coordinate's own working copy is made to carry the switch.

SECTION 13.4

## A Peptide With Two Tryptophans

Now the first of two surprises the leader holds. Though it codes for no useful protein, the leader can be translated. It carries a start signal and a short readable stretch, and ribosomes dock on it and read it just as they would a real gene, producing a tiny peptide only a handful of amino acids long. This little peptide is a

decoy, built for one purpose, and the purpose is hidden in its sequence: near its end it contains two tryptophan codons, one immediately after the other. Two demands for tryptophan, side by side, planted deliberately in a test message.

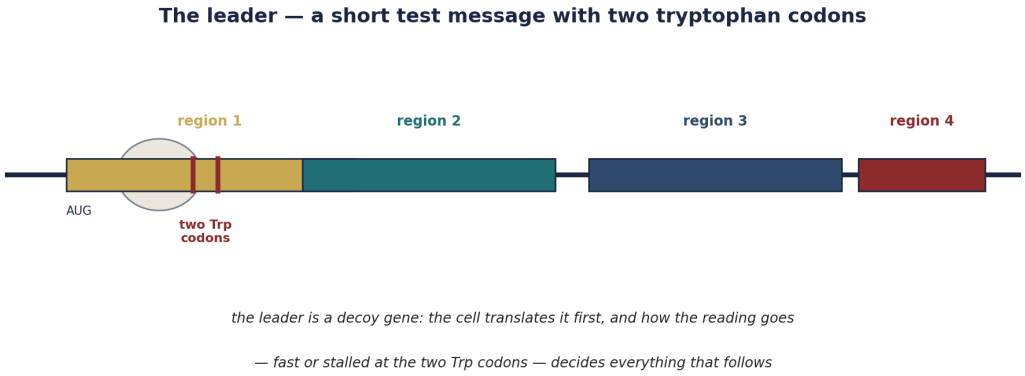


**Figure 13.3.** The leader as a decoy gene. It is translated into a tiny peptide whose sequence contains two tryptophan codons in a row. How the ribosome fares at those two codons — running through or stalling — is the sensor on which everything downstream turns.

The reason for those two codons is the crux of the whole design. A ribosome translating the leader must, at that point, insert two tryptophans in succession, and to do so it needs charged transfer-RNA carrying tryptophan — which is plentiful only when the cell's tryptophan is plentiful. If tryptophan is short, the ribosome arrives at those codons and can go no further; it stalls, waiting for an amino acid that is not coming. If tryptophan is abundant, it sails straight through. The leader has been engineered by evolution to be a tryptophan meter: a place where the scarcity or plenty of the amino acid is turned into a difference in the behaviour of a ribosome. In the terms of this book, the leader converts the local T-supply of one amino acid into a difference in reading-speed — the first step in a small computation.

That the leader is truly translated, and not merely a stretch of decorative sequence, was shown directly and by no small effort. Researchers fused the leader's little coding region to a marker they could measure and found its peptide made at a high rate; and they took the leader RNA on its own, offered it to ribosomes in a tube, and watched the ribosomes dock and begin upon it just as they would on a real gene. The decoy is a working gene in miniature — short, and useless as a product — but genuinely read. Its whole purpose is to be translated, so that the manner of its translation, fast or halting, can be sensed by the molecule that carries it.

SECTION 13.5

## Two Folds, One Choice

Now the second surprise, which concerns the RNA itself. Beyond the little peptide, the leader RNA contains four short stretches — call them regions one, two, three,

and four — that are able to fold back and pair with one another, the way the two strands of DNA pair, forming little hairpins. But they cannot all pair at once, and here is the beauty of it: region three can pair either with region two or with region four, but not both. So the RNA has two possible shapes, and they are mutually exclusive. If region three pairs with region four, the result is a particular hairpin followed by a run of U's — and that structure is a terminator, a signal that makes the RNA polymerase let go and stop transcribing then and there. If instead region two pairs with region three, then three is no longer free to pair with four, the terminator cannot form, and transcription runs on into the structural genes. Two folds; two fates.

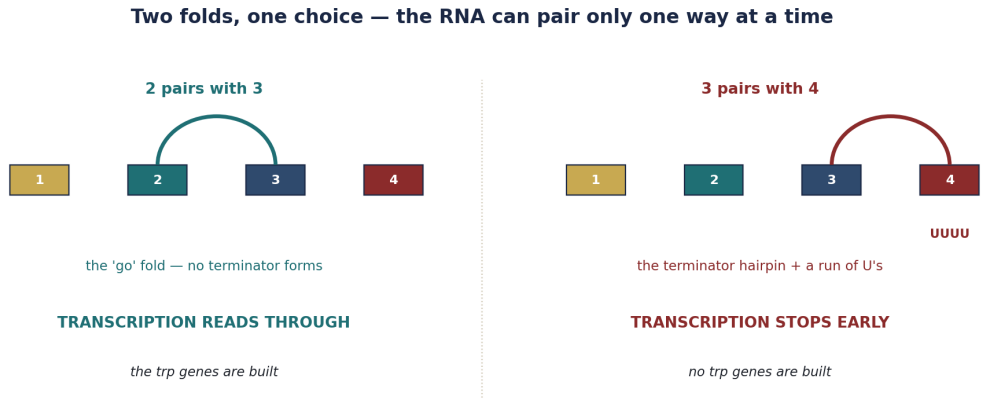


**Figure 13.4.** Two folds, one choice. If region 2 pairs with 3 (left), the terminator cannot form and transcription reads on into the genes. If region 3 pairs with 4 (right), a terminator hairpin followed by a run of U's halts transcription. The RNA can adopt only one of these at a time.

A fork in the road, then, built into a molecule. The same stretch of RNA can settle into a 'go' shape or a 'stop' shape, and only one signal — the terminator — actually halts the building. In the reading of this book, this is a T-object with two permitted forms, and the whole question of whether the genes get made comes down to which of the two the molecule falls into. What remains is to see how the cell steers the choice — and the answer ties the two surprises together, for it is the ribosome on the little peptide that decides which fold the RNA can take.

SECTION 13.6

## The Ribosome Sets the Fold

Here is how the two pieces lock together, and it is one of the most elegant contrivances in all of molecular biology. The regions that can pair overlap the stretch that the ribosome translates: region one lies within the little peptide, and region two lies just beyond it. So the physical position of the ribosome — how far along the leader it has read — determines which regions are free to pair and which

are covered up and forbidden. The ribosome's body is a clamp that hides whatever it sits upon.

Follow the logic through. If the ribosome stalls at the two tryptophan codons — because tryptophan is scarce — it sits over region one and leaves region two exposed. Region two is then free to grab region three the moment three is made, forming the antiterminator; and with three tied up by two, it can never pair with four, so the terminator does not form and transcription proceeds. But if the ribosome runs through the leader — because tryptophan is plentiful — it advances onto region two and covers it. Region two, hidden, cannot capture region three; so when four is transcribed, three pairs with four, the terminator forms, and transcription stops. The presence of the amino acid, read through the ribosome's progress, has reached forward and chosen the fold. In this book's language, the reading-head of translation has set the shape of the coordinate's working copy, and thereby governed the reading-head of transcription behind it.

SECTION 13.7

## The Three States

With the mechanism in hand we can now watch it resolve the three situations the cell must tell apart — the problem set out at the start — and see that it answers each correctly. This is where attenuation reveals its full cleverness, for it distinguishes cases that a simple repressor never could.

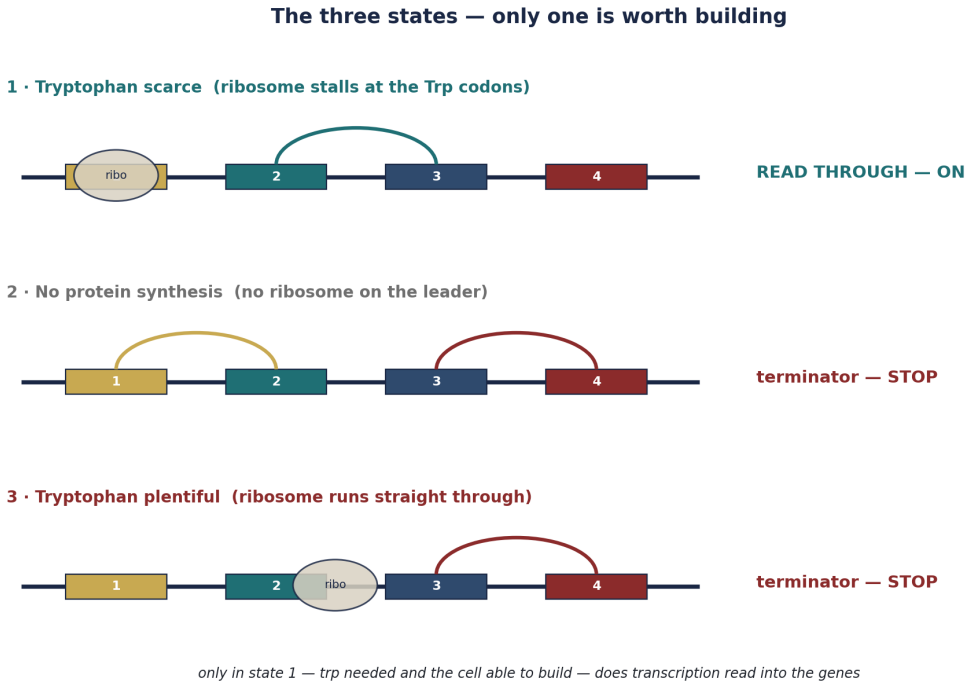


**Figure 13.5.** The three states. When tryptophan is scarce the ribosome stalls, region 2 pairs with 3, and transcription reads through — ON. When no protein is being made at all, or when tryptophan is plentiful and the ribosome runs through, the terminator (3–4) forms and transcription stops. Only the first state builds the genes.

In the first state — tryptophan scarce, and the cell actively translating — the ribosome stalls at the two codons, the antiterminator forms, and the genes are read. This is exactly the case in which the cell needs tryptophan and can use the enzymes: the factory is built. In the second state — no protein synthesis happening at all, so that no ribosome even boards the leader — the RNA folds by default into its most stable pairings, region one with two and region three with four, and the terminator forms: transcription stops. This is correct, for a cell not making protein has no use for more tryptophan-making machinery. In the third state — tryptophan plentiful — the ribosome runs straight through, covers region two, the terminator forms, and transcription stops again: also correct, for the shelves are full. Three situations, and the molecule sorts them into build or do-not-build with nothing but the position of a ribosome on a decoy peptide. In the reading of this book, the field has resolved its condition into three cases and spends its build-energy on the address in only the one case that its circumstances warrant.

**KEY IDEA**

Attenuation reads the supply of one amino acid through the speed of a ribosome. A short leader peptide carries two tryptophan codons; when tryptophan is scarce the ribosome stalls there, when it is abundant the ribosome races through. The ribosome's position decides which of two mutually exclusive folds the leader RNA takes — an antiterminator that lets transcription read on into the genes, or a terminator hairpin followed by a run of U's that halts it. Three states are told apart, and only one — tryptophan scarce and the cell translating — builds the genes. In this book: a rate is converted into a structure, and the field builds only where its circumstances warrant.

SECTION 13.8

## A Clock Made Into a Decision

Now step back and read what has really happened, because it is, in this book's terms, the deepest thing in the chapter. What the cell actually measures is not a concentration in any direct way. It measures a speed. The single quantity that decides everything is how fast the ribosome moves across the two tryptophan codons — and that speed is set by how much charged transfer-RNA for tryptophan is on hand, which is to say by the local supply of one amino acid. A fast ribosome means plenty; a stalled ribosome means scarcity. The cell has turned the abundance of a substance into the tempo of a reading, and then read the tempo.

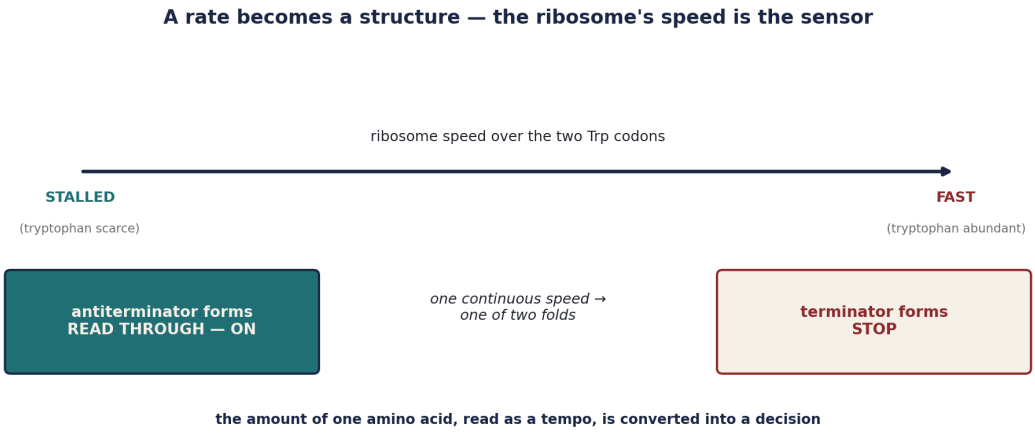


**Figure 13.6.** A rate becomes a structure. The ribosome's speed over the two tryptophan codons — stalled when the amino acid is scarce, fast when it is abundant — is the sensor. A continuous speed is converted into one of two folds, and so the supply of one amino acid, read as a tempo, becomes a decision.

This is why the mechanism belongs so squarely in the Universal Force of Time. Through the earlier chapters, the reading of the address ran at a fixed tempo — the register's own clock, ticking beneath transcription and translation alike, never itself the subject. Here, for the first time, that tempo is sensed and acted upon. A rate is measured

and converted into a structure; the structure decides whether the field goes on building. In this framework heat is a manifestation of T, and time and rate are of one substance with it; so a rate is not a pale shadow of a ‘real’ quantity but a first-class carrier of information, fully able to hold and transmit a decision. Attenuation is that principle caught in the act. The cell does not need to grip and count molecules of tryptophan; it lets the amino acid set a speed, and reads the speed. It is a clock made into a decision — the field timing one of its own supplies and acting on the timing.

SECTION 13.9

## Two Folds From a Continuous Rate

There is a further point hidden in the mechanism, and it is a familiar one to any reader of this book. The ribosome’s speed is a continuous quantity — it can take any value between fully stalled and fully fast, shading smoothly as tryptophan varies from utterly absent to richly abundant. Yet the outcome is not continuous. The RNA does not fold a little bit terminator and a little bit antiterminator; it settles decisively into one shape or the other. A smooth input has been converted into a sharp, two-valued output. The molecule rounds the world’s greyscale to black or white.

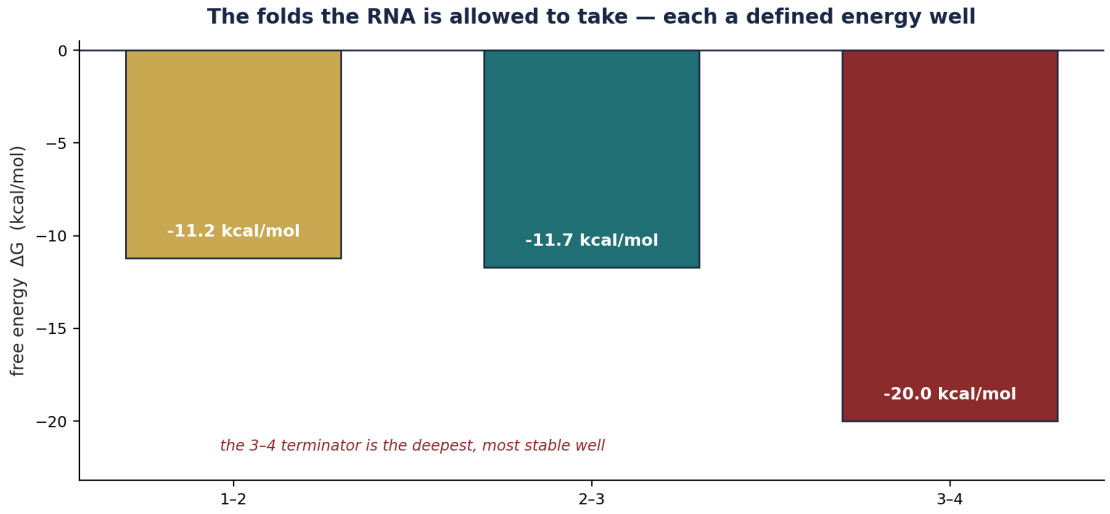


**Figure 13.7.** The allowed folds as energy wells. Each possible pairing has a definite free energy (here in kilocalories per mole); the 3–4 terminator is the deepest, most stable well. A continuous ribosome speed does not yield a continuous structure — the RNA drops into one of a small set of defined wells.

In this book that is quantisation, the same principle that runs through everything from spectral lines to the folds of a protein: a continuous influence collapses onto one of a discrete set of allowed states. The RNA has only a few stable ways to fold — each a defined well in the energy landscape, with a definite depth, as the figure shows — and whatever the exact speed of the ribosome, the molecule must come to rest in one well or another. The two-valued switch is not imposed from outside; it falls out of the fact that a T-object has only certain permitted forms. A continuous rate meets a quantised structure, and the meeting produces a clean decision. The greyscale of the world is read,

and answered in the binary of the lattice.

**WORKED EXAMPLE · reading the three states**

Treat the leader as a tiny logic device with one input — the ribosome's speed — and one output — build or do-not-build. State 1, tryptophan scarce and the cell translating: the ribosome stalls on region 1, leaving region 2 free → the 2–3 antiterminator forms → the 3–4 terminator cannot form → BUILD. State 2, no translation at all: no ribosome boards the leader → the RNA takes its default pairings, 1–2 then 3–4 → terminator → STOP. State 3, tryptophan plentiful: the ribosome runs through and covers region 2 → region 3 pairs with 4 → terminator → STOP. One input, three cases, a single 'build' — the truth table of a gene, computed by a molecule upon its own reading-rate.

SECTION 13.10

## One Clock Feeding Back on Itself

One more feature of the design repays attention, because it is what makes the whole trick possible, and it is quietly remarkable. For the ribosome's position to govern which fold the RNA takes, the translating of the leader and the transcribing of it must happen at the same time, in step, on the same molecule — the ribosome following close behind the polymerase as the RNA is laid down. In a bacterium this is exactly what happens: transcription and translation are coupled, the two great reading-heads of the cell running the same tape almost together. Attenuation simply harnesses that coupling and lets the pace of the one steer the fate of the other.

In the reading of this book, this is a single clock feeding back on itself. Transcription and translation are not two independent processes that happen to be near one another; they are two expressions of the one register tempo, the same clock read at two heads. Attenuation ties those heads together so that the speed of the translating head — set by the supply of tryptophan — reaches back and controls whether the transcribing head continues. It is self-reference: the field's reading of a coordinate is made to govern the field's further reading of that same coordinate. A clock that senses its own rate and, on the strength of it, decides whether to keep ticking. There is nothing else quite like it in the chapters we have seen — regulation not by an outside agent at all, but by the process folding back upon itself.

SECTION 13.11

## Three States, One Worth Building

It is worth dwelling for a moment on the number three, because the cell's whole problem was to resolve its condition into three cases and act rightly in each. A single repressor gives only two answers — on or off — and so cannot tell a cell that is

starving-and-able-to-build from one that is idle. Attenuation gives the finer partition: it separates the one state in which building is worthwhile from the two in which it is waste. And it does this economy for a reason the conservation law makes plain. Making the tryptophan enzymes when they are not needed — when the shelves are full, or when the cell is not even making protein — would pour T-structured matter into machines that would sit idle. The field does not spend so.

So the operon carries, in the end, layers of thrift stacked one on another, each reading a slightly different facet of the cell's state. The repressor blocks much initiation when tryptophan is plentiful, refusing to start what need not be started. Attenuation then fine-tunes what does get started, aborting most of it unless tryptophan is truly short and the cell is truly building. And feedback inhibition throttles the enzymes that already exist, so that even the standing machinery does not overproduce. Three mechanisms, reading initiation, continuation, and activity, and all serving one end: to keep the tryptophan pool filled at the least cost. In this book's reading, this is the conservation law, $d\Sigma T=0$, worked out in the small — a living node declining, at every level, to redistribute its substance toward an address the moment does not warrant.

SECTION 13.12

## Why This Should Matter to You

It is tempting to think of the genome as a book of fixed instructions, read out the same way every time. Every chapter of this part of the story has undermined that picture, and this one perhaps most of all. Here a gene decides, moment by moment, whether to finish being read — and it decides by sensing the speed of its own reading, folding itself at a fork in the road according to how fast a ribosome is moving through a tiny test message. There is no controller standing over it. The molecule does the arithmetic itself. The address of life is not a passive script but something closer to a live process, taking the measure of its own circumstances and responding as it runs.

And there is a reason this reaches beyond the marvel of one bacterial operon. The same logic — RNA that folds one way or another, reading rates and coupling one process to the next — runs, elaborated and multiplied, through the cells of your own body, and much of modern medicine is learning to read and, one day, to correct it. When a cell misjudges its own state — builds when it should be still, or falls silent when it should build — the fault often lies in exactly this kind of self-reading gone astray. To grasp that a molecule can clock one of its supplies and decide its own fate is to glimpse both how finely a living thing governs itself, and where that governance, disturbed, becomes disease. In the chapters to come the circuits grow richer still — whole cascades of switches wired into one another — but the tryptophan leader has already shown the boldest idea of all: that the field can hand a molecule the power to decide, for itself, whether to go on.

## The Numbers at a Glance

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

| Part of the decision | What it is | The Force of Time reading |
|---|---|---|
| Feedback inhibition | trp blocks its own first enzyme | a T-pool held level by reading its own size |
| The leader | ~160-letter decoy before the genes | the switch moved onto the working copy of the address |
| Two Trp codons | a tryptophan meter in the peptide | the local T-supply turned into a reading-speed |
| Ribosome stalled / fast | sets which regions can pair | a rate converted into a structure |
| Two folds | antiterminator (go) / terminator (stop) | a continuous rate collapsed onto one of two folds |
| Hairpin free energies | -11.2, -11.7, -20.0 kcal/mol | defined energy wells — quantised folds |
| Three states | only one is worth building | the field spending only where warranted ($d\Sigma T=0$) |

## References

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