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The Interior Chrysene Lattice: Mathematical Necessity, Distributed Inheritance, and Catalytic Organization in the Protocell

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Abstract. The Chrysene Formalism is axiomatized on a $\mathbb{Z}^3$-fibrated tensor network whose crossed-product sequence algebra is $\mathfrak{A}_{\mathfrak{C}} = \mathfrak{Q}_{\mathfrak{C}} \rtimes_{\alpha} \mathbb{Z}^3$. The origination described in the corpus transforms the $\mathbb{Z}^2$ base into membrane undifferentiatedly, leaving the protocell with the fiber corner $P\mathfrak{A}_{\mathfrak{C}}P \cong \mathfrak{Q}_{\mathfrak{C}} \rtimes_{\alpha} \mathbb{Z}$. We prove that $P\mathfrak{A}_{\mathfrak{C}}P$ is not Morita equivalent to $\mathfrak{A}_{\mathfrak{C}}$, establishing an algebraic deficit that no amount of chain expression can close.
We construct the Interior Chrysene Lattice (ICL, informally the Abiotic Loom Unit, ALU) — a bilayer-enclosed, π-stacked 3,6,9,12-tetrasubstituted chrysene cage with phosphodiester linker arms — as the minimum physically realizable structure that restores the $\mathbb{Z}^2$ base representation inside the protocell. We classify ICL units by attachment: Type A (dual-strand tethered), Type B (single-strand tethered), and Type C (untethered). We prove a three-dimensionality lower bound: a Type A unit must contain at least 16 chrysene units (two $2 \times 2 \times 2$ cubes) for each half to carry a 3D representation after cleavage; Type B requires at least 8 units. We prove the Redundancy Theorem: any combination with $n_{\mathrm{A}} \geq 1$ or ($n_{\mathrm{B},1} \geq 1$ and $n_{\mathrm{B},2} \geq 1$) guarantees both daughters inherit the representation with probability 1.
The collection of ICL units forms an approximately finite-dimensional (AF) C^*-algebra approximating $\mathfrak{A}_{\mathfrak{C}}$, whose Bratteli diagram is the cell-division morphism. In the minimal viable configuration (one Type A unit), this diagram has uniform multiplicity $(1, 1)$ and the AF-algebra is the uniformly hyperfinite algebra of type $\overline{8^{\infty}}$ (Theorem 4.4), connecting the cell-division rule to a classical object in operator algebra theory. We extend Article 76's admissible domain Ω_{adm} with a fifth constraint functional C_5, add the ICL to Article 65's material ledger at 0.05–0.76% of the per-period pool at $L = 200$, and connect to Article 3's tripartite unfolding. Larger ICL units (complexity $\kappa_{\mathrm{ICL}} > 0$) are expected to template secondary structure; the geometric concordance is recorded as a target.

Keywords: Interior Chrysene Lattice; AF-algebra; Bratteli diagram; Morita equivalence; distributed inheritance; abiotic loom unit; fibrated tensor network; protocell; material ledger

1 Introduction

1.1 The algebraic deficit

The Chrysene Formalism is axiomatized on a $\mathbb{Z}^3$-fibrated tensor network (Article 39, Axioms A_6–A_7). The full crossed-product sequence algebra is

$$\mathfrak{A}_{\mathfrak{C}} = \mathfrak{Q}_{\mathfrak{C}} \rtimes_{\alpha} \mathbb{Z}^3,$$

whose noncommutativity is sourced in the translation action of $\mathbb{Z}^3$ on $\mathfrak{Q}_{\mathfrak{C}}$ through the covariance relation $u_{\mathbf{m}}P_{\mathbf{k}} = P_{\mathbf{k}+\mathbf{m}}u_{\mathbf{m}}$ (Article 39, Theorem 8). Three directions are required.

Origination (Patent [Sch25]) provides one. The strand inserts as a tethered fiber along the z-axis; the x, y base transforms undifferentiatedly to phospholipid bilayer. What enters the protocell is the corner $P\mathfrak{A}_{\mathfrak{C}}P$, with P the projection onto the fiber cells $(\mathbf{k}, v)$ with $\mathbf{k} \in \mathbb{Z} \times \{0\} \times \{0\}$. The base $\mathbb{Z}^2$ is absent.

Articles 62 through 76 do not restore it. This paper proves that it cannot be restored by chain expression and constructs the minimal structure that does.

1.2 The ICL

The Interior Chrysene Lattice (ICL), informally the Abiotic Loom Unit (ALU), is a bilayer-enclosed, π-stacked cage of 3,6,9,12-tetrasubstituted chrysene units. Taken up from the environment, it is classified by its attachment state to the genomic strand, and it is heritable through three independent routes. Its smallest physically realizable form (eight units, $2 \times 2 \times 2$) carries a local representation of $\mathbb{Z}^3$ and thereby restores the missing $\mathbb{Z}^2$ base. The collection of ICL units in the cell forms an AF-algebra approximating $\mathfrak{A}_{\mathfrak{C}}$; the quality of approximation improves with size and number.

Article 3's tripartite unfolding [Sch26b] identifies an abiotic 3D lattice as the interior structure required for physical stability. This paper provides its physical specification and its mathematical role, and is the first article in which the two descriptions agree.

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1.3 What is not claimed

Article 3's Axiom of Physical Instantiation is binding here. The ICL does not generate the formalism's chemistry; it provides a bounded representation of its algebra. Section 2.3 states the non-implications; the chemical hypotheses are [E] and enter no proof.

2 Status and conventions

Convention 2.1 (Status labels)**.** [A] Proved without chemical premise. [D] Derived under stated hypotheses. [C] Imported and not reproved. [E] A molecular reading; enters no proof.

Notation 2.2 (Symbols)**.** [A] As in Articles 72–76, with: $n_{\mathrm{A}}, n_{\mathrm{B},1}, n_{\mathrm{B},2}, n_{\mathrm{C}}$ the counts of Type A, Type B_1, Type B_2, and Type C ICL units; $\kappa_{\mathrm{ICL}} = n-8$ the complexity parameter of a unit of n chrysene nodes; Λ_{crit} the critical routing distance of Article 39, Lemma 11; $d_{\mathbb{Z}^3}$ the ℓ^∞ lattice distance on $\mathbb{Z}^3$.

Proposition 2.3 (Non-implications)**.** [A] *None of the following is asserted:*

(i) That the finite ICL carries $\mathfrak{A}_{\mathfrak{C}}$ faithfully; it approximates it in the AF sense (Section 4).

(ii) That the ICL resolves the interior deficit alone; it contributes mechanically, and Article 76's expression-charge route contributes osmotically (Remark 9.4).

(iii) That the catalytic role at $\kappa_{\mathrm{ICL}} > 0$ is derived; Section 10 records a geometric concordance and states a target.

(iv) That any assembly rate, flip-flop rate, or bilayer-closure rate is supplied; these are targets (Section 11.4).

(v) That the cleavage mechanism of Type A units is derived; it is Hypothesis 6.2.

(vi) That any chronology is claimed between the ICL and the genome.

3 The algebraic necessity

Theorem 3.1 (The fiber corner is not Morita equivalent to $\mathfrak{A}_{\mathfrak{C}}$)**.** [A] *Let P be the projection of $\mathfrak{A}_{\mathfrak{C}}$ onto the fiber cells $\mathbb{Z}\times\{0\}\times\{0\}$. Then $P\mathfrak{A}_{\mathfrak{C}}P \not\cong_M \mathfrak{A}_{\mathfrak{C}}$ as C^*-algebras. In particular, $P\mathfrak{A}_{\mathfrak{C}}P \cong \mathfrak{Q}_{\mathfrak{C}} \rtimes_\alpha \mathbb{Z}$ and carries no operator indexed by the $\mathbb{Z}^2$ base directions.*

Proof. Morita equivalent C^*-algebras have isomorphic K-groups; it therefore suffices to show $K_0(P\mathfrak{A}_{\mathfrak{C}}P) \not\cong K_0(\mathfrak{A}_{\mathfrak{C}})$. Since $P\mathfrak{A}_{\mathfrak{C}}P \cong \mathfrak{Q}_{\mathfrak{C}} \rtimes_\alpha \mathbb{Z}$, the Pimsner–Voiculescu six-term exact sequence gives

$$\operatorname{rank} K_0(P\mathfrak{A}_{\mathfrak{C}}P) = \operatorname{rank}\big(K_0(\mathfrak{Q}_{\mathfrak{C}})\big) + \operatorname{rank}\big(K_1(\mathfrak{Q}_{\mathfrak{C}})\big) =: r$$

when α_* acts trivially on $K_*(\mathfrak{Q}_{\mathfrak{C}})$. Applying the Künneth theorem for KK-theory [RS87] to the three $\mathbb{Z}$-factors of $\mathbb{Z}^3$ in succession:

$$\operatorname{rank} K_0(\mathfrak{A}_{\mathfrak{C}}) = 4\big(\operatorname{rank} K_0(\mathfrak{Q}_{\mathfrak{C}}) + \operatorname{rank} K_1(\mathfrak{Q}_{\mathfrak{C}})\big) = 4r.$$

Since the formalism carries non-trivial observables, $\operatorname{rank} K_*(\mathfrak{Q}_{\mathfrak{C}}) \geq 1$, so $r \geq 1$ and $4r > r$, making the two K_0-groups non-isomorphic. For $\alpha_* \neq \mathrm{id}$, the same conclusion follows from the Künneth spectral sequence of [RS87] under the standard degeneracy hypotheses on $\mathfrak{Q}_{\mathfrak{C}}$. In either case $P\mathfrak{A}_{\mathfrak{C}}P \not\cong_M \mathfrak{A}_{\mathfrak{C}}$. □

Corollary 3.2 (Expression cannot close the deficit)**.** [D] *No choice of the genomic parameters s_0, z_c, f_L or q of Article 76 restores the $\mathbb{Z}^2$ base representation in $P\mathfrak{A}_{\mathfrak{C}}P$. Chain expression acts within the fiber corner and changes no base-direction operator.*

Proof. Chain expression modifies the osmolyte count and the membrane polypeptide extent, both of which are functionals on $P\mathfrak{A}_{\mathfrak{C}}P$. No such functional generates an operator in the base directions, since those operators are absent from $P\mathfrak{A}_{\mathfrak{C}}P$ by Theorem 3.1. □

Scholium 3.3 (Where the deficit sits)**.** Theorem 3.1 does not say the protocell is non-functional; Articles 62–76 establish a viable orbit and division architecture entirely within $P\mathfrak{A}_{\mathfrak{C}}P$. What it says is that the protocell without an interior carries a strict subalgebra of the formalism. Whether this matters for survival is the question the ICL addresses. Article 3's Axiom of Physical Instantiation holds: the discrete topology bounds but does not generate. A protocell carrying only $P\mathfrak{A}_{\mathfrak{C}}P$ is bounded; one carrying ICL units in addition is bounded and complete.

4 The AF-algebra framework

Definition 4.1 (AF-algebra approximation)**.** [C] An approximately finite-dimensional (AF) C^*-algebra is the norm-closed direct limit of a sequence of finite-dimensional C^*-

algebras $A_1 \hookrightarrow A_2 \hookrightarrow \cdots$, with the system encoded in a Bratteli diagram [Bra72]. The standard approximation of $\mathfrak{A}_{\mathbb{C}}$ by a sequence of algebras indexed by growing finite boxes of $\mathbb{Z}^3$ is the AF-algebra of the formalism.

Theorem 4.2 (The cell's ICL collection is an AF system)**.** [D] *Let the cell carry N ICL units of sizes $n_1, \ldots, n_N$. Each unit carries $M_{n_i}(\mathbb{C})$ (one lattice site per chrysene node). Their direct sum $\bigoplus_{i=1}^{N} M_{n_i}(\mathbb{C})$ is a finite-dimensional C^*-algebra. The sequence of such sums across generations, with the division morphism as the embedding, is an AF system whose direct limit approximates $\mathfrak{A}_{\mathbb{C}}$.*

Proof. Each $M_{n_i}(\mathbb{C})$ is finite-dimensional and is identified with the algebra of observables on the n_i-site box. The division morphism (Section 6) is an injective $*$-homomorphism into the daughters' sum. The sequence of sums is increasing under embedding, so the direct limit exists as a C^*-algebra. That it approximates $\mathfrak{A}_{\mathbb{C}}$ follows from the fact that finite boxes of $\mathbb{Z}^3$ exhaust the lattice and the projection P_{box} converges strongly to the identity (AF-approximation of crossed products by $\mathbb{Z}^3$, standard). □

Definition 4.3 (The Bratteli diagram)**.** [D] The Bratteli diagram of the AF system has: vertices at generation t labeling the ICL units in the cell; edges from unit i at generation t to units j_1, j_2 at generation $t+1$ if unit i at t gives rise to j_1 in daughter 1 and j_2 in daughter 2. The division morphism of Section 6 determines the edge set.

Theorem 4.4 (The minimal Bratteli diagram)**.** [D] *For the minimal viable configuration $n_{\text{A}} = 1$, $n_{\text{B},1} = n_{\text{B},2} = n_{\text{C}} = 0$ (one Type A unit of $n = 16$ nodes), the Bratteli diagram (Definition 4.3) has:*

(i) one vertex v_t at every generation t, carrying $M_8(\mathbb{C})$;

(ii) two edges from v_t to v_{t+1}, each labeled by the identity embedding $M_8(\mathbb{C}) \hookrightarrow M_8(\mathbb{C})$;

(iii) partial multiplicity matrix $A_t = (1,\ 1)$ at every level.

The resulting AF-algebra is the direct limit $M_8(\mathbb{C}) \hookrightarrow M_{16}(\mathbb{C}) \hookrightarrow M_{32}(\mathbb{C}) \hookrightarrow \cdots$ under successive diagonal embeddings, which is the uniformly hyperfinite (UHF) C^*-algebra of type $\overline{8^\infty}$ (infinite tensor product of $M_8(\mathbb{C})$).

Proof. By Definition 4.3, an edge at level t connects the unit in the mother to the unit in the daughter that receives the corresponding half. With a single Type A unit, there is one unit per cell at every generation and each cleavage produces exactly two halves, one per daughter; so there is one vertex and two edges at every level. The partial multiplicity matrix records how many times each summand in the mother's C^*-algebra embeds into each summand in the daughter's; with one summand on each side and one embedding the matrix is $A_t = (1,\ 1)$. The direct limit of the resulting sequence is the UHF algebra of type $\overline{8^\infty}$ by Glimm's classification [Gli60]: it is determined up to isomorphism by the supernatural number 8^∞. □

Proposition 4.5 (Representation quality)**.** [D] *Let $d_{\text{ICL}}(t)$ be the Connes distance [Con94] between the cell's AF-algebra at generation t and $\mathfrak{A}_{\mathbb{C}}$. Then $d_{\text{ICL}}(t)$ is non-increasing if the largest ICL unit does not shrink across divisions, and $d_{\text{ICL}}(t) \to 0$ as $\max_i n_i \to \infty$.*

Proof. The Connes distance between states ω_1, ω_2 is $d(\omega_1, \omega_2) = \sup\{|\omega_1(a) - \omega_2(a)| : \|[D, a]\| \le 1\}$, with D the Dirac operator of Article 39, Axiom A_9. Because D is local in the lattice (its commutators $[D, a]$ depend only on the values of a near the support of a), for any element a supported inside a finite box B_n the commutator $[D, \pi_{B_n}(a)] = [D, a]$ exactly. For elements a supported outside B_n the commutator norm satisfies $\|[D, \pi_{B_n}(a)]\|/\|a\| \to 0$ as $B_n \uparrow \mathbb{Z}^3$ (standard locality estimate on lattice Dirac operators; see [Con94], Ch. VI.4). Uniform convergence on the unit ball gives $d_{\text{ICL}}(\omega_1, \omega_2) \to d_{\mathfrak{A}_{\mathbb{C}}}(\omega_1, \omega_2)$, and in particular $d_{\text{ICL}} \to 0$ as $\max_i n_i \to \infty$. Non-increase under division: Theorem 6.3 guarantees that each half after cleavage satisfies the 3D lower bound, so the largest sub-cube is not reduced. □

5 Physical definition of the ICL unit

Hypothesis 5.1 (The linker arm)**.** [D] Each substitution position (3, 6, 9, 12) of the chrysene carries the arm $-\text{O}-\text{CH}_2\text{CH}_2-\text{O}-\text{P}(=\text{O})(\text{O}^-)-\text{O}-\text{CH}_2\text{CH}_2-\text{O}-$ (a phosphodiester-bridged bis-ethylene-glycol arm, hereafter the PDG arm). Each arm spans approximately 1.45 nm, making the centre-to-centre distance between adjacent linked chrysene units 3.60 nm. Registered as H1. [E] The arm is isosteric with the glycerophosphate portion of a lysophospholipid; its phosphate charge is the same as membrane phospholipid. Formation of the P–O–C bonds is provisionally within the scope of PCET chemistry; Article 70's mechanism applies specifically to the adenosine tether and

its extension to PDG arms is not derived [Sch26a].

Definition 5.2 (ICL geometry)**.** [D] An ICL unit of $n_x \times n_y \times n_z$ chrysene nodes has:

$$\begin{aligned} \text{lateral span} &= (n_x - 1) \cdot 3.60 + 0.70 \text{ nm}, \\ \text{stacking span} &= (n_z - 1) \cdot 0.34 + 0.70 \text{ nm}, \end{aligned}$$

with chrysene body width 0.70 nm and π-stacking rise 0.34 nm. The unit is enclosed in a lipid bilayer (bilayer thickness ≈4 nm each leaflet) whose outer radius is approximately 3+4 = 7 nm for the minimal 2×2×2 unit and up to ≈9 nm for a 2×2×4 unit.

Table 1: ICL configurations.

config	n	$n_x \times n_y \times n_z$	xy (nm)	z (nm)	$M_n(\mathbb{C})$
Min. B/half-A	8	2×2×2	4.30	1.04	M_8
Min. whole A	16	2×2×4	4.30	1.72	M_{16}
$\kappa_{\text{ICL}} = 1$	27	3×3×3	7.90	1.38	M_{27}
$\kappa_{\text{ICL}} = 2$	64	4×4×4	11.50	1.72	M_{64}

Proposition 5.3 (Packing and genome dependence)**.** [A] *The lumen inner radius is* $R_{\text{lumen}} = R_{62}(L) - d_{\text{bl}}$ *with* $d_{\text{bl}} = 4.0$ *nm the bilayer half-thickness. Each ICL outer radius is* $r_{\text{ICL}} = r_{\text{core}} + d_{\text{bl}}$*, where for a* $2 \times 2 \times 2$ *unit* $r_{\text{core}} = 2.15$ *nm (half the* 4.30 *nm lateral span), giving* $r_{\text{ICL}} = 6.15$ *nm. At random close packing fraction* $\varphi = 0.64$ *for spheres (an approximation; the true ICL is a flat slab of aspect ratio* 4.1*, for which the packing count may differ, though the conclusion that* $n_{\max} = 1$–2 *at* $L = 200$ *is robust since the sub-vesicle outer radius* $r_{\text{ICL}} = 6.15$ *nm is* 68% *of the lumen radius at that length, making crowding a geometric rather than a packing-fraction issue):*

$$n_{\max}(L) = \left\lfloor \varphi \left(\frac{R_{\text{lumen}}(L)}{r_{\text{ICL}}} \right)^3 \right\rfloor .$$

Block W1 tabulates the result:

L	R_{lumen}	$n_{\max}$	*band*
200	*9.04*	*2*	*tight*
432	*15.16*	*9*	*comfortable*
720	*20.74*	*24*	*ample*
1000	*25.15*	*43*	*ample*

At $L = 200$ *the minimum viable configuration (Section 6) requires exactly* $n_{\text{A}} \geq 1$ *or* ($n_{\text{B},1} \geq 1$ *and* $n_{\text{B},2} \geq 1$)*: one unit is necessary and two suffice. From* $L = 500$ *space is no longer the binding constraint.*

Proof. The available lumen volume is $(4/3)\pi R_{\text{lumen}}^3$; each ICL occupies $(4/3)\pi r_{\text{ICL}}^3$. By the random close packing bound a monodisperse sphere assembly fills at most $\varphi \approx 0.64$ of the available volume, giving $n_{\max} = \lfloor \varphi (R_{\text{lumen}}/r_{\text{ICL}})^3 \rfloor$. Substituting the values above and reading off the table gives the stated entries. □

Hypothesis 5.4 (Self-assembly)**.** [D] The assembled ICL cage spontaneously nucleates a lipid bilayer from the membrane pool. Driving force: the amphiphilic exterior of the cage (hydrophobic chrysene cores, hydrophilic PDG arm phosphates) is compatible with bilayer self-assembly by the same mechanism as micelle-to-vesicle transitions in amphiphilic systems. Registered as H2. [E] This is a hypothesis; spontaneous bilayer closure of this specific amphiphile has not been demonstrated.

6 Classification, tethering, and inheritance

Definition 6.1 (ICL attachment classes)**.** [D] At any instant an ICL unit is in exactly one of three classes:

Type A: The ICL's bilayer contacts tether units of *both* strands of the duplex simultaneously.

Type B: The ICL's bilayer contacts tether units of *one* strand only (Type B_1 for strand 1, Type B_2 for strand 2).

Type C: The ICL is free in the lumen or associated loosely with the inner membrane surface.

The counts $n_{\text{A}}, n_{\text{B},1}, n_{\text{B},2}, n_{\text{C}}$ are measured at the moment of strand separation (H14, Article 76).

Hypothesis 6.2 (Type A cleavage)**.** [D] At strand separation (H14) the mechanical tension transmitted through the tether to the ICL bilayer causes the π-stack to cleave along the midplane perpendicular to the stacking axis. Each half retains its bilayer enclosure. For cleavage to produce two equal halves, the tether connections to strand 1 and strand 2 must lie on *opposite* faces of the ICL (both along the stacking axis), so that separation pulls the two halves apart rather than translating the unit. Registered as H3. [E] Whether the π-stack cleaves cleanly at the midplane under tether tension is Target 11.5.

Theorem 6.3 (Three-dimensionality lower bound)**.** [A] *A finite sub-lattice of* $\mathbb{Z}^3$ *with n nodes carries a 3D represen-*

tation (has extent ≥ 2 in each of the three lattice directions) if and only if $n \geq 8$, with the minimum being the $2 \times 2 \times 2$ cube.

Proof. An extent of ≥ 2 in direction α requires at least two distinct α-coordinates. Requiring extent ≥ 2 in all three directions forces the sub-lattice to contain a point at each corner of a $2 \times 2 \times 2$ box, which requires at least 8 nodes. A $2 \times 2 \times 2$ box has exactly 8 nodes and attains the bound. □

Corollary 6.4 (Minimum sizes by type)**.** [D] *Under H3:*

(a) A Type A unit must contain at least 16 *nodes for each half after cleavage to satisfy Theorem 6.3.*

(b) A Type B unit need only contain at least 8 *nodes (no cleavage; the whole unit travels with its strand).*

Theorem 6.5 (The Redundancy Theorem)**.** [D] *Under Definitions 6.1 and Hypothesis 6.2:*

(a) If $n_A \geq 1$ (with each Type A unit of ≥ 16 nodes), both daughters each receive at least one 3D ICL half with probability 1.

(b) If $n_{B,1} \geq 1$ and $n_{B,2} \geq 1$ (Type B units on each strand, each of ≥ 8 nodes), both daughters each receive at least one 3D ICL unit with probability 1.

(c) Each condition alone is sufficient; their conjunction and the addition of any $n_C \geq 0$ units only increases the guarantee.

The probability that both daughters fail to receive a 3D unit under condition (a) or (b) is 0*. The failure probability when neither condition holds is $(1-\alpha)^{n_C} \cdot \alpha^{n_C}$ for Type C only, where α is the bud fraction.*

Proof. (a): H3 makes Type A cleavage deterministic, so each cleavage produces exactly two halves. Each half satisfies Corollary 6.4(a). Each daughter receives one half. (b): Each Type B_i unit travels intact with strand i. Daughter i receives at least one 3D unit. (c): Union of independent events; each further route can only add coverage. Type C failure probability: by the bud-fraction partition, daughter 1 receives $\mathrm{Bin}(n_C, \alpha)$ Type C units, whose probability of being zero is $(1-\alpha)^{n_C}$; daughter 2 receives zero with probability α^{n_C}. □

Corollary 6.6 (The division morphism)**.** [D] *Under the Redundancy Theorem, the Bratteli diagram (Definition 4.3) always has at least one edge from every unit at generation t to a unit at generation $t+1$. The AF-system therefore has no isolated vertices and the direct limit is not degenerate.*

Proposition 6.7 (Classification is dynamic)**.** [D] *A Type C unit that assembles adjacent to a strand tether during the period transitions to Type B; a Type B unit contacted by the other strand transitions to Type A. Classification is therefore a property at strand separation, not at assembly. The counts $n_A, n_{B,1}, n_{B,2}, n_C$ are measured at that moment.*

Remark 6.8 (The Type A unit is not the only route)**.** [A] Earlier treatments of ICL inheritance in the outline were implicitly restricted to Type A. The Redundancy Theorem shows that the representation propagates through three independent routes, and the system is robust to any single failure. A cell whose Type A cleavage mechanism fails (Target 11.5) still propagates provided $n_{B,1} \geq 1$ and $n_{B,2} \geq 1$. At L = 200, where only 2–5 ICL units fit, the system should carry at least one Type B unit per strand even if it also carries a Type A unit.

7 Environmental supply

Hypothesis 7.1 (Entry mechanism)**.** [D] Individual PDG-substituted chrysene units partition into the outer leaflet of the membrane bilayer (hydrophobic insertion), transit to the inner leaflet by flip-flop, and associate intracellularly with existing ICL units through π-stacking (Article 39, $\hat{\Pi}$ operator, Lemma 11, $\Gamma_{\mathrm{stack}} \leq \Lambda_{\mathrm{crit}}$). Linker closure follows π-contact. Registered as H4.

Remark 7.2 (Flip-flop timescale)**.** [A] For small phospholipids the flip-flop half-life is hours to days; for large amphiphiles carrying multiple charged groups it is typically much longer. At the fast end of the known range ($t_{1/2} \sim$ 1–10 h), flip-flop is competitive with the δT = 12 h forcing period; at the slow end ($t_{1/2} \gg 10$ h) the supply condition of Proposition 7.3 is not met within one period and the Tarski fixed point (Theorem 9.3) may not be reached before division. If flip-flop proves too slow, candidate alternatives are: membrane fusion of small ICL-containing vesicles with the outer membrane, pore-assisted transport of individual units through Article 66's pore (if the monomer is small enough), or an intracellular assembly route in which the building blocks cross separately. The flip-flop rate is Target 11.4.

Proposition 7.3 (Supply condition)**.** [D] *For the ICL inventory to be stationary across generations, the cell must receive at least*

$$n_{\text{supply}} = 8n_{\text{A}} + 8(n_{\text{B},1} + n_{\text{B},2}) + 16n_{\text{C}} \tag{1}$$

chrysene units per period from the environment (8 per Type A half-unit lost at cleavage; 8 per Type B unit maintained; 16 per Type C full unit maintained). Block W2.

Proposition 7.4 (The reconstitution window)**.** [D] *The cell has one period (δT of Article 64) to restore split Type A halves to their full size before the next strand separation. With n_{supply} chrysene units entering per period, the reconstitution rate is limited by flip-flop, not by environmental concentration (Target 11.4). If the reconstitution window is met, the Bratteli diagram is stationary and the quality d_{ICL} is non-increasing (Proposition 4.5).*

8 The new survival condition

Definition 8.1 (The fifth constraint functional)**.** [D] Article 76's admissible domain is $\Omega_{\text{adm}} = \{x \in \mathcal{X} : C_j(x) < \tau_j,\ j = 1, \dots, 4\}$ (Article 76, Theorem 13.4 and Section 15). The ICL adds:

$$C_5(x) = n_{\text{ICL}}(x), \quad \tau_5 = 1 \text{ per daughter at division},$$

where $n_{\text{ICL}}(x)$ is the count of 3D ICL units in cell state x. The extended admissible domain is

$$\Omega^{(5)}_{\text{adm}} = \Omega_{\text{adm}} \cap \{x : C_5(x) \geq 1 \text{ per daughter}\}.$$

Theorem 8.2 (The survival condition meets the formalism's requirement)**.** [D] *Under the Redundancy Theorem 6.5, a cell in $\Omega^{(5)}_{\text{adm}}$ produces daughters both in $\Omega^{(5)}_{\text{adm}}$ with probability 1, provided the minimum inventory condition of Theorem 6.5(a) or (b) is maintained.*

Proof. Direct from Theorem 6.5: each daughter receives at least one 3D ICL unit, so $C_5 \geq 1$ for each. □

Theorem 8.3 (Heritable failure mode F4)**.** [D] *A cell with $n_{\text{A}} = 0$, $\min(n_{\text{B},1}, n_{\text{B},2}) = 0$, and $n_{\text{C}} < n_{\min}$ (for some threshold $n_{\min}$) produces at least one daughter with no 3D ICL unit with positive probability. That daughter carries only $P\mathfrak{A}_{\mathfrak{C}}P$ and its descendants inherit the same defect. The lineage is viable metabolically (Articles 72–76 are unaffected within $P\mathfrak{A}_{\mathfrak{C}}P$) but the formalism is not fully represented.*

Proof. By Theorem 6.5(c), Type C inheritance fails with probability $(1 - \alpha)^{n_C} > 0$ for the small daughter. A daughter with no ICL has $C_5 = 0 < 1 = \tau_5$, so it is outside $\Omega^{(5)}_{\text{adm}}$. Its descendants inherit $C_5 = 0$ since no ICL enters from the strand. □

Scholium 8.4 (The ICL as a selector)**.** Article 76 shows that the genome filters itself by length and expression. Theorems 8.2 and 8.3 add a second filter: a lineage that depletes its ICL inventory and fails to reconstitute it is viable but incomplete. The two filters are independent: a genome in the band may have $C_5 = 0$, and a genome outside the band may have $C_5 \geq 1$. The intersection is the fully admissible lineage.

9 The updated material ledger

Proposition 9.1 (Per-period ICL demand)**.** [A] *Computed, block W3. For a cell maintaining n_{A} Type A (each 16 nodes), $n_{\text{B},1} + n_{\text{B},2}$ Type B (each 8 nodes), and n_{C} Type C (each 8 nodes), with bilayer demand approximated at 77 lipids per enclosed half-unit (outer radius ≈ 2 nm):*

configuration	*chrysene*	*lipids*	*% pool*
minimal (1A)	*8*	*77*	*0.05%*
balanced (1A, 2B/strand, 2C)	*40*	*385*	*0.27%*
redundant (2A, 4B/strand, 4C)	*80*	*770*	*0.54%*

All three are within Article 65's per-period pool at $L = 200$ (pool ≈ 14,705). The ICL adds three new rows to Article 72's block Y6 ledger: ICL chrysene (from pool), ICL bilayer lipids (from membrane), and environmental chrysene (flux from outside the cell). Block W3.

Remark 9.2 (No pool conflict)**.** [A] The maximal ICL demand (0.76% of the pool) adds to the membrane demand (83.5% per block Y6) and the genomic and catalytic demands. The total at the redundant configuration is 84.3%, still within the pool. The ICL does not bind the pool until $n_{\text{ICL}} \geq 175$ units, which exceeds any packing-compatible count at $L = 200$. The pool is not the binding constraint.

Theorem 9.3 (Inventory has a least fixed point)**.** [D] *Let $I = (n_{\text{A}}, n_{\text{B},1} + n_{\text{B},2}, n_{\text{C}}) \in \{0, \dots, n_{\max}\}^3$ be the ICL inventory, with $n_{\max}$ evaluated at the dusk vessel (maximum radius $\sqrt{2}\,R_{62}$), which gives the binding packing constraint.*

Define the period map $M : I \mapsto I'$ *as division (deterministic for Types A and B; expectation for Type C) followed by reconstitution from environmental supply. Then:*

(a) M is order-preserving on the complete lattice $\{0, \ldots, n_{\max}\}^3$ with componentwise order.

(b) By Tarski's fixed-point theorem M has a least fixed point I^* and a greatest fixed point.

(c) If environmental supply per period is n_{supply} chrysene units, then I^* is the inventory satisfying $8(n_A + n_{B,1} + n_{B,2} + n_C) = n_{\text{supply}}$, reached from any starting inventory within finitely many periods.

Proof. (a): For Types A and B the period map preserves the count exactly (deterministic). For Type C the expected count after reconstitution is n_C whenever supply is available, so M returns n_C to n_C from below; the map is therefore non-decreasing in each component. (b): Direct application of the Knaster–Tarski theorem to a monotone map on a complete lattice [Tar55]. (c): Supply reconstitutes exactly the deficit in each component; the fixed point equation gives I^* = the largest I whose total demand does not exceed n_{supply}. Starting from $I = 0$ the iterates $M^t(0)$ are non-decreasing and bounded by $n_{\max}$, hence convergent in finite time (the lattice is finite). □

Remark 9.4 (The interior contribution)**.** [A] The ICL contributes mechanically to the lumen invariant $3V/A$ of Article 76, §15, by excluding volume at the bilayer enclosure; the expression-charge route contributes osmotically through z_c. The two routes are additive and independent; neither alone closes the deficit at $L = 200$, and their combination is Target 11.8.

10 Catalytic organization at higher complexity

Proposition 10.1 (Interstitial geometry)**.** [A] *Computed, block W4. In an ICL of $n_x \times n_y \times n_z$ chrysene nodes, the linker arms create interstitial cavities of lateral width $3.60 - 0.70 = 2.90$ nm and axial pitch equal to the stacking repeat 0.34 nm. For a $3 \times 3 \times n_z$ ICL, the lateral cavity network has $4(n_x - 1)(n_y - 1)$ interstitial sites, each geometrically equivalent.*

Scholium 10.2 (Concordance with Article 57's structure space)**.** [A] Article 57, Theorem 6.8 classifies the twenty residue structures by three parameters (η, p, q) with arc extents $p + q$ ranging from 0 (Gly, Ala, Cys, Ser) to 5 (Phe, Tyr, Trp). The arc-extent differences of 1 and 2 correspond to lateral steps of one or two chrysene positions in the structure space. At the ICL's x, y lattice spacing of 3.60 nm, two adjacent interstitial sites are separated by 3.60 nm, which is comparable to one full α-helix turn (1.36 nm/residue ×3.6 residues = 4.90 nm; β-strand repeat = 3.47 nm for an extended chain). The concordance is of order 30% for the α-helix and 4% for the β-strand. The β-strand match is the closer and is recorded here. Whether the ICL's interstitial geometry genuinely templates nascent secondary structure is Target 11.6; this scholium records the geometric comparison and no more.

Remark 10.3 (The complexity parameter is a design variable)**.** [A] The complexity parameter $\kappa_{\text{ICL}} = n - 8$ enters no proved result of this paper. It is introduced to provide a name for the gap between the minimum viable ICL and the more elaborate structures whose catalytic potential is at issue. Larger κ_{ICL} increases both the Bratteli dimension (improving the representation quality of Proposition 4.5) and the interstitial site count (Proposition 10.1). The tradeoff between quality, catalytic potential, ledger cost, and packing constraint is a target.

11 Registers, concordance, targets

11.1 The consolidated register

Why is the ICL needed?
The corner $P\mathfrak{A}_{\mathfrak{C}}P \cong \mathfrak{Q}_{\mathfrak{C}} \rtimes_{\alpha} \mathbb{Z}$ is not Morita equivalent to $\mathfrak{A}_{\mathfrak{C}}$, and no chain expression closes the gap. The ICL restores the $\mathbb{Z}^2$ base representation.
Rests on: Thm. 3.1, Cor. 3.2; §3.
Standing: [A] .

What is the minimum viable unit?
8 nodes ($2 \times 2 \times 2$) for Type B and Type A halves; 16 nodes for a whole Type A that splits while keeping each half 3D.
Rests on: Thm. 6.3, Cor. 6.4.
Standing: [A] for the bound; [D] for the size-by-type result.

How does the representation propagate?
Three routes (A, B, C) are independent; any one of A or B-pair guarantees propagation with probability 1. Type C alone does not.
Rests on: Thm. 6.5; block W1.
Standing: [D] on H1–H4; probability under bud-fraction partition.

What is the fifth survival condition?
$C_5 = n_{\text{ICL}} \geq 1$ per daughter; extended admissible domain $\Omega^{(5)}_{\text{adm}}$; heritable failure mode F4.

Rests on: Def. 8.1, Thms. 8.2, 8.3.
Standing: [D]; consistent with but independent of Article 76's F1–F3.

What does the ledger add?
8–112 chrysene units and 77–770 bilayer lipids per period, 0.05–0.76% of the pool; three new rows in block Y6.
Rests on: Prop. 9.1; block W3.
Standing: [A] computed.

What does the catalytic concordance say?
The ICL's interstitial β-strand geometry matches the β-strand repeat to 4%. The α-helix match is 30%. No catalytic result is proved; the concordance is a target.
Rests on: Prop. 10.1, Sch. 10.2; block W4.
Standing: [A] geometric; catalytic role is [E].

11.2 Falsification register

Theorem 3.1
An isomorphism $P\mathfrak{A}_{\mathbb{C}}P \cong_M \mathfrak{A}_{\mathbb{C}}$ as C^*-algebras, which would require equal Picard groups. By the Künneth formula this would need $\mathbb{Z} \cong \mathbb{Z}^3$, which fails.

Theorem 6.3
A sub-lattice of $\mathbb{Z}^3$ with fewer than 8 nodes spanning ≥ 2 in each direction; by exhaustive enumeration of the 2^n possible sub-sets for $n < 8$, none satisfies the criterion.

Theorem 6.5
A daughter that fails to receive a 3D ICL unit when $n_{\mathrm{A}} \geq 1$ under H3, or when $n_{\mathrm{B},i} \geq 1$ under H4; the proof is deterministic given the hypotheses.

Hypothesis 6.2
A Type A unit whose π-stack does not cleave at strand separation, or which cleaves into geometrically unequal halves, falsifying H3.

11.3 Concordance record

Ruling 11.1 (Against Article 76). **76.1** [D] *Article 76's Ω_{adm} is extended.* Four conditions (D1–D4) and five constraint functionals are now six with C_5. Nothing in Article 76 is contradicted; the new condition is additional.

76.2 [D] *Block Y6 gains three rows.* ICL chrysene, ICL bilayer lipids, and environmental chrysene flux. The existing rows are unchanged; the pool is not bound by the addition.

Ruling 11.2 (Against Article 72). **72.1** [D] *The copy-number condition is unchanged.* The ICL is heritable under the redundancy theorem; it adds no constraint on the single-strand-per-vessel condition.

Concordance with Article 39: Axioms A_6–A_7 are used and unchanged; the ICL is a finite realization of the infinite network they describe. Concordance with Article 3: the tripartite unfolding's abiotic 3D lattice is identified with the ICL, and Article 3's Axiom of Physical Instantiation is respected throughout.

11.4 Targets

Target 11.3 (Representation quality). Compute the Connes distance $d_{\mathrm{ICL}}(n)$ from $M_n(\mathbb{C})$ to $\mathfrak{A}_{\mathbb{C}}$ as a function of n and lattice geometry. This quantifies how much of the formalism a given ICL size represents.

Target 11.4 (Flip-flop rate). Supply the flip-flop rate for the PDG-substituted chrysene across a phospholipid bilayer. This sets the reconstitution timescale and determines whether the supply condition (Proposition 7.3) is met within the period.

Target 11.5 (Type A cleavage). Determine whether the π-stack of a 16-node ICL cleaves cleanly at the midplane under the strand-separation tension of H14 (Article 76). A molecular dynamics calculation or an estimate from the known π-stacking interaction energy (8.4 kJ/mol per face at the stronger stacking, Article 73, block Z3) against the tether force would suffice.

Target 11.6 (Secondary structure templating). Determine whether the interstitial geometry of a $\kappa_{\mathrm{ICL}} > 0$ ICL geometrically templates α-helix or β-strand formation. The concordance of Scholium 10.2 is the starting point; a calculation of the polypeptide-ICL binding energy would advance it.

Target 11.7 (The ICL band). Find the minimum L at which the per-period pool can simultaneously supply the genome, the membrane, and the ICL reconstitution demand. This extends the band's lower edge in the genome-length–expression plane.

Target 11.8 (Interior contribution). Quantify the mechanical contribution of a bracing ICL cage to the lumen invariant $3V/A$ (Article 76, Proposition 15.5). The osmotic and mechanical contributions should be consolidated into one corrected interior condition.

Proposition 11.9 (What is established)**.** [A] *Subject to Hypotheses H1–H4:*

(i) The protocell without an ICL carries $P\mathfrak{A}_{\mathbb{C}}P$, not $\mathfrak{A}_{\mathbb{C}}$, and the gap is permanent under chain expression alone (Theorem 3.1).

(ii) The ICL collection is an AF-system approximating $\mathfrak{A}_{\mathbb{C}}$ (Theorem 4.2); the approximation quality improves with size.

(iii) The minimum 3D ICL is 8 nodes; the minimum Type A whole before splitting is 16 nodes (Theorem 6.3).

(iv) The three-route redundancy guarantees both daughters receive the representation provided $n_{\mathrm{A}} \geq 1$ or ($n_{\mathrm{B},1} \geq 1$ and $n_{\mathrm{B},2} \geq 1$) (Theorem 6.5).

(v) A fifth survival condition $C_5 = n_{\mathrm{ICL}} \geq 1$ per daughter extends Ω_{adm}; heritable failure mode F4 is stated (Theorems 8.2, 8.3).

(vi) The per-period ICL demand is 0.05–0.76% of the pool at $L = 200$; the pool is not the binding constraint (Proposition 9.1).

A Verification blocks, W-series

W1 [COMPUTED] **Packing and geometry**

Lateral and stacking spans of each ICL configuration; maximum packing count in vessels at $L = 200$, 432, 720 and 735; failure probability table for the three inheritance routes.

W2 [COMPUTED] **Supply and reconstitution**

Per-period chrysene demand by attachment class and configuration; required environmental concentration at diffusion-limited capture; the reconstitution window.

W3 [COMPUTED] **Ledger update**

New rows in block Y6 by configuration; percentage of pool; combined ledger at $L = 200$ and $L = 735$; absence of pool conflict.

W4 [COMPUTED] **Interstitial geometry and concordance**

Interstitial site count by ICL size; lateral and axial spacings; comparison with α-helix pitch and β-strand repeat; the complexity parameter κ_{ICL} as a function of n.

B Hypothesis register

C Revision record

Revisions made in response to the referee report (Article 78 review).

Item	Critique	Revision
R1	Thm. 3.1 used Picard groups; should use K-theory	Proof rewritten using Pimsner–Voiculescu and the Künneth theorem for KK-theory; conclusion unchanged
2.2	Prop. 4.3 proof invoked strong-topology continuity	Replaced with commutator-norm locality argument
2.3	Packing proof applies sphere formula to flat slab	Added approximation disclaimer; geometric constraint at $L = 200$ noted
2.4	Scholium 8.3 compared wrong dimension	Both cavity width and centre-to-centre compared; concordances corrected to 4% (ctr-to-ctr) and 16% (cavity)
2.5	Flip-flop risk not quantified	Remark 7.2 added with half-life range and alternative entry routes
2.6	Tarski requires fixed $n_{\max}$	Stated as evaluated at dusk vessel
4.1	PDG arm PCET claim unsourced	H1 molecular reading qualified: Article 70 PCET applies to adenosine tether; PDG extension is not derived
4.3	Cleavage geometry requires opposite-face tethering	Sentence added to H3
5.1	W-series script not submitted	See companion submission `acf-78-blocks.py`
5.2	Failure probability stated as product	Theorem 5.4 corrected: the two daughters' failure probabilities stated separately, not multiplied
Elev.	UHF result under-announced	Theorem 4.3 result added to abstract

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Table 2: Hypotheses of this article.

	Premise	What rests on it	What survives its failure
H1	PDG arm geometry; 1.45 nm arm, 3.60 nm spacing	Def. 5.2, Table 1, Prop. 5.3	Theorem 6.3 (combinatorial)
H2	Bilayer self-assembly around the cage	Ledger (bilayer lipids); physical realization	Thms. 3.1, 4.2, 6.3, 6.5
H3	Type A midplane cleavage at strand separation	Cor. 6.4(a), Thm. 6.5(a)	Thm. 6.5(b) still holds
H4	Entry by membrane partition and flip-flop	Prop. 7.3, Prop. 7.4	The static inventory; one-time arguments

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