CausalBioRL: Reinforcement Learning with Causal World Models for Autonomous Drug Discovery

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Abstract

We present **CausalBioRL**, a model-based reinforcement learning framework that integrates causal discovery, structural causal models (SCMs), and hierarchical planning for autonomous drug discovery. Unlike conventional RL approaches to molecular optimisation that treat the environment as a black box, CausalBioRL *learns* the causal structure of the biological system from its own interaction data, fits a structural causal model to the discovered graph, and plans actions using Pearl's do-operator. The agent operates in a novel **DrugDiscovery-v0** environment with a 244-dimensional observation space (R-GCN graph embeddings, molecular features, per-target summaries) and a 130-dimensional continuous action space encoding target selection, termination, and navigation in a variational autoencoder's latent space. A two-level **hierarchical planner** uses UCB1 for target selection (exploration–exploitation over drug targets) and the Cross-Entropy Method (CEM) for molecule generation in latent space. An **adaptive reward learner** dynamically reweights six objectives (binding affinity, QED, synthetic accessibility, novelty, causal confidence, structural stability) based on per-objective difficulty estimated by learned value functions. We demonstrate that the causal world model enables principled reasoning about interventions, and that the hierarchical architecture efficiently allocates computational budget across multiple targets. CausalBioRL is released as part of the open-source NeoRx platform.

Keywords: reinforcement learning, causal inference, drug discovery, structural causal models, molecular generation, hierarchical planning

1 Introduction

1.1 The Drug Discovery RL Problem

Drug discovery is naturally framed as a sequential decision-making problem: an agent must decide which biological targets to pursue, which molecular structures to explore, and when to stop—all under a limited computational budget. Reinforcement learning (RL) offers a principled framework for such problems [1], and recent work has applied RL to molecular optimisation [2, 3, 4].

However, existing RL approaches to drug discovery suffer from two fundamental limitations:

1. **Black-box environments**: The agent treats the biological system as an opaque reward function, learning a policy through trial-and-error without understanding *why* certain actions succeed. This leads to sample-inefficient exploration and policies that fail to generalise across disease contexts.

2. **Single-objective optimisation**: Most molecular RL agents optimise binding affinity or QED score alone, ignoring the multi-objective nature of drug discovery (where a candidate

must simultaneously satisfy causal target validity, binding, drug-likeness, synthesisability, pharmacokinetics, and structural novelty).

1.2 Causal RL

Causal reinforcement learning [5, 6] addresses the first limitation by equipping the agent with an explicit causal model of its environment. Rather than learning a policy end-to-end from reward signals, the agent:

1. **Discovers** causal structure from observed state transitions
2. **Fits** a structural causal model (SCM) to the discovered graph
3. **Plans** actions by reasoning about interventions via Pearl's do-operator [7]

This enables *model-based* RL with interpretable world models, counterfactual reasoning, and efficient transfer across related tasks.

1.3 Contributions

CausalBioRL makes the following contributions:

1. A **causal RL architecture** for drug discovery that integrates causal discovery, SCMs, and do-calculus planning in a unified agent
2. A **DrugDiscovery-v0** Gymnasium environment with biologically meaningful observations (R-GCN graph embeddings), multi-objective rewards, and difficulty scaling
3. A **hierarchical planner** combining UCB1 target selection with CEM molecule generation in VAE latent space
4. An **adaptive reward learner** that dynamically weights objectives based on per-objective difficulty
5. A **surrogate docking model** that accelerates evaluation from seconds to sub-millisecond while maintaining calibration

2 Related Work

2.1 Molecular RL

REINVENT [2] uses a recurrent neural network (RNN) policy to generate SMILES strings, trained with REINFORCE to optimise a scoring function. MolDQN [3] applies DQN to molecular graph editing operations. These approaches optimise molecular properties but do not reason about the *validity* of the underlying target. GraphDF [8] uses normalising flows on molecular graphs; JT-VAE [4] combines junction-tree encoding with RL fine-tuning. None integrate causal reasoning about the biological target.

2.2 Causal RL

Causal RL has been explored in tabular settings [5] and simple continuous environments [6]. CIRL [9] learns causal models for transfer in Atari games. To our knowledge, CausalBioRL is the first application of causal RL to drug discovery.

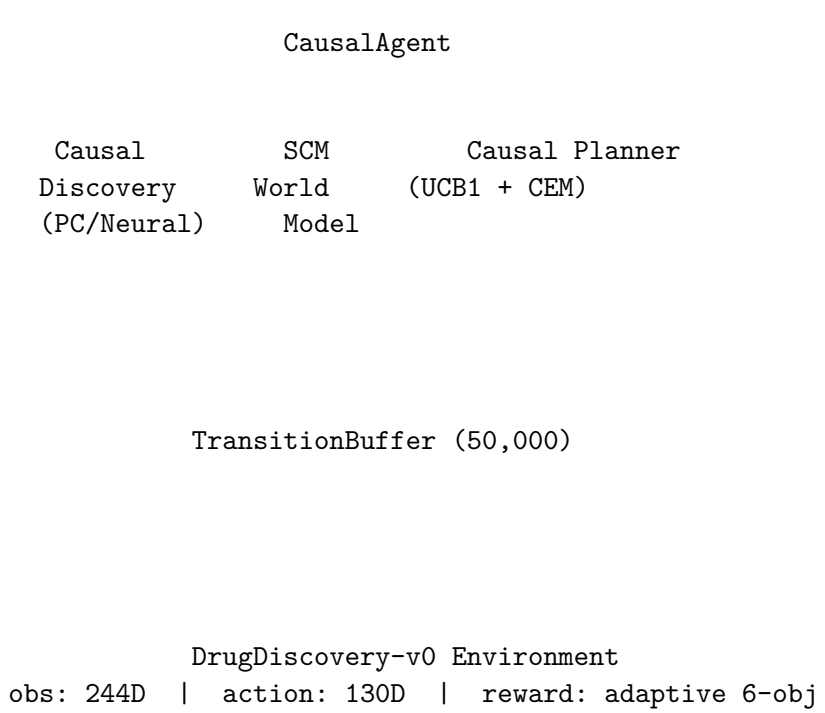
2.3 Multi-Objective Drug Discovery

Multi-objective molecular optimisation has been addressed through scalarisation [10], Pareto front exploration [11], and constrained optimisation [12]. CausalBioRL's adaptive reward learner provides a novel approach: per-objective value functions estimate difficulty, and weights are dynamically adjusted to focus on bottleneck objectives.

3 Methods

3.1 Architecture Overview

CausalBioRL consists of four integrated components:



3.2 Causal Discovery

The agent discovers causal structure from its transition buffer using one of two methods.

3.2.1 PC Algorithm (Constraint-Based)

We wrap the causal-learn library's PC algorithm with Fisher's z-test for conditional independence ($\alpha = 0.05$). Input: transition matrix $\mathbf{T} \in \mathbb{R}^{N \times D}$ where each row is (s_t, a_t, r_t, s_{t+1}). Output: a DAG encoded as a NetworkX DiGraph.

3.2.2 Neural Causal Discovery (Differentiable)

For continuous high-dimensional spaces, we learn a differentiable adjacency matrix via Gumbel-sigmoid relaxation.

Learnable parameters: $\mathbf{A}_{\text{logits}} \in \mathbb{R}^{D \times D}$, one logit per potential edge.

Edge mask (Gumbel-sigmoid with temperature annealing):

$$M_{ij} = \sigma\left(\frac{A_{\text{logits},ij}}{\tau}\right), \quad \tau = \max\left(0.5, 1 - \frac{e}{E}\right)$$

where e is the current epoch and $E = 300$ is the total training epochs.

Prediction network: MLP($D \to 64 \to D$) with ReLU, where the input is gated by the learned mask.

Training loss:

$$\mathcal{L} = \text{MSE}(\hat{Y}, Y) + \lambda_{\text{sparse}} \sum_{i,j} \sigma(A_{\text{logits},ij})$$

with $\lambda_{\text{sparse}} = 0.01$, optimised by Adam ($\eta = 10^{-3}$). The binary adjacency is obtained by thresholding: $A_{ij} = \mathbb{1}[M_{ij} > 0.5]$.

3.3 Structural Causal Model

Given the discovered DAG, we fit per-edge causal mechanisms.

3.3.1 Mechanism Types

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Type	Function
Linear	$f_j(Pa_j) = W_j \cdot Pa_j + b_j$
Neural	$f_j(Pa_j) = \text{MLP}(\lvert Pa_j \rvert \to 32 \to 1)$, ReLU activation

3.3.2 Fitting

All per-node mechanisms are trained jointly via Adam ($\eta = 10^{-3}$, 200 epochs) minimising the sum of per-node MSE losses:

$$\mathcal{L}_{\text{SCM}} = \sum_{j=1}^{D} \|X_j - f_j(Pa_j)\|_2^2$$

3.3.3 Do-Operator for Planning

Pearl's do-operator is implemented directly on the SCM. Given an intervention $do(X_i = x)$:

1. Fix $X_i = x$ (override its mechanism)
2. Remove all incoming edges to X_i (sever parental influence)
3. Propagate in topological order, skipping intervened variables
4. Return the resulting state vector

This enables the planner to evaluate *counterfactual* outcomes: "What would happen to the drug-likeness score if we shifted the latent vector by Δz?"

The SCM supports **edge provenance weighting**: edges tagged `"api"` (from biological databases) receive weight 1.0, while edges tagged `"learned"` (from causal discovery) receive weight 0.5, reflecting differential trust in data sources.

3.4 Hierarchical Planner

Drug discovery requires two levels of decision-making: (1) *which target to pursue* and (2) *which molecule to design*. We decompose this into a hierarchical planner.

3.4.1 Level 1: Target Selection via UCB1

Given K drug targets, the agent selects which to pursue using the Upper Confidence Bound algorithm:

$$\text{UCB}_i = \bar{x}_i + c\sqrt{\frac{\ln t}{n_i}}$$

where $\bar{x}_i$ is the mean reward observed for target i, n_i is the number of times target i has been selected, t is the total step count, and $c = 2.0$ is the exploration constant.

Each target is visited at least once before UCB scores are computed (forced exploration). This balances exploitation (pursuing the most promising target) with exploration (investigating under-explored targets that might yield surprising discoveries).

3.4.2 Level 2: Molecule Generation via CEM

Once a target is selected, the agent generates molecules by navigating the VAE's latent space using the Cross-Entropy Method:

1. Initialise: $\boldsymbol{\mu} = \mathbf{0}^{128}$, $\boldsymbol{\sigma} = 0.3 \cdot \mathbf{1}^{128}$
2. For $I = 5$ iterations:
   a. Sample $N = 200$ candidate latent vectors $\mathbf{z}^{(n)} \sim \mathcal{N}(\boldsymbol{\mu}, \mathrm{diag}(\boldsymbol{\sigma}^2))$, clipped to $[-1, 1]$
   b. Decode each $\mathbf{z}^{(n)}$ to a SMILES molecule via the VAE decoder
   c. Score each molecule via the adaptive reward function
   d. Select the top $\lceil 0.1 \cdot N \rceil = 20$ elite samples
   e. Update: $\boldsymbol{\mu} \leftarrow \mathrm{mean(elite)}$, $\boldsymbol{\sigma} \leftarrow \mathrm{std(elite)} + 10^{-6}$
3. Return the action encoding $[\mathrm{target\_idx}, 0, \boldsymbol{\mu}]$

The planner optionally performs **multi-step rollouts** ($H = 3$ steps) using the SCM, summing predicted rewards along the trajectory.

3.5 DrugDiscovery-v0 Environment

3.5.1 Observation Space (244D)

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Indices	Dim	Content
$[0, 128)$	128	R-GCN graph embedding of the disease knowledge graph
$[128, 160)$	32	Current molecule features (6 objective scores + 26 compressed fingerprint bits)
$[160, 240)$	80	Per-target summaries (10×8: causal confidence, attempts, best score, binding, QED, SA, novelty, stability)
$[240, 244)$	4	Meta features (step fraction, target count fraction, episode best score, surrogate flag)

The R-GCN graph embedding is computed by a `DiseaseGraphEncoder` (Section 3.6), providing a fixed-size vector representation of the arbitrarily-sized disease knowledge graph.

3.5.2 Action Space (130D continuous)

$$\mathbf{a} = [a_{\text{target}}, a_{\text{stop}}, \Delta\mathbf{z}_{1:128}] \in [-1, 1]^{130}$$

- $a_{\text{target}} \in [-1, 1]$: discretised to a target index via $i = \lfloor (a_{\text{target}} + 1) \cdot K/2 \rfloor$
- $a_{\text{stop}} \in [-1, 1]$: if $a_{\text{stop}} > 0.9$ and step ≥ 5, the episode terminates early
- $\Delta\mathbf{z} \in [-1, 1]^{128}$: scaled by 0.3 and added to the current latent base vector

Latent drift: When a generated molecule achieves a composite score > 0.5, the latent base vector drifts towards the successful point: $\mathbf{z}_{\text{base}} \leftarrow 0.9 \cdot \mathbf{z}_{\text{base}} + 0.1 \cdot \mathbf{z}_{\text{current}}$.

3.5.3 Reward Function

The reward at each step is computed by the adaptive reward learner (Section 3.7) from six objective scores:
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Objective	Normalisation	Source
Binding affinity	clamp$(-a/12, 0, 1)$	DockBot / surrogate
QED	$[0, 1]$	MolScreen
Synthetic accessibility	clamp$((10 - \text{SA})/9, 0, 1)$	MolScreen
Novelty	$[0, 1]$	Tanimoto distance
Causal confidence	$[0, 1]$	NeoRx identifier
Stability	$[0, 1]$	MirrorFold therapeutic score

3.5.4 Difficulty Levels

Level	Budget Multiplier	Observation Noise σ
Easy	2.0×	0.0
Medium	1.0×	0.02
Hard	0.5×	0.05

3.6 R-GCN Graph Encoder

To encode the variable-size disease knowledge graph into a fixed-dimension observation vector, we use a Relational Graph Convolutional Network (R-GCN) [13].

3.6.1 Node Features (15D)

Each node v is represented by a 15-dimensional feature vector:

$$\mathbf{x}_v = [\deg_v^-, \deg_v^+, \text{BC}_v, \text{PR}_v, s_v, \mathbb{1}[\text{tissue}], \tilde{e}_v, \text{one\_hot}(t_v)]$$

Feature	Dimension	Description
$\deg_v^-$, $\deg_v^+$	2	In/out degree (normalised by max)
BC_v	1	Betweenness centrality
PR_v	1	PageRank (max_iter=100)
s_v	1	Association score
$\mathbb{1}[\text{tissue}]$	1	Tissue relevance (boolean → float)
$\tilde{e}_v$	1	min(evidence_count/10, 1)
one_hot(t_v)	8	Node type (8 types)

3.6.2 Architecture

```
x_v (15D) $\to$ Linear(15, 64) $\to$ [RGCNLayer(64, 64) $\times$ 2] $\to$ Readout $\to$ MLP
```

R-GCN layer with basis decomposition ($B = 4$ bases):

$$\mathbf{h}_v^{(l+1)} = \sigma\left(\sum_{r \in \mathcal{R}} \sum_{u \in \mathcal{N}_r(v)} \frac{1}{c_{v,r}} \mathbf{W}_r \mathbf{h}_u^{(l)} + \mathbf{W}_0 \mathbf{h}_v^{(l)}\right)$$

$$\mathbf{W}_r = \sum_{b=1}^{B} a_{rb} \mathbf{B}_b$$

where $\mathbf{B}_b \in \mathbb{R}^{d \times d}$ are shared basis matrices and a_{rb} are per-relation coefficients.

13 relation types: activates, inhibits, phosphorylates, binds, regulates, participates_in, associated_with, causes, treats, interacts_with, upregulates, downregulates, learned.

Graph readout: $\mathbf{g} = [\text{mean}(\{\mathbf{h}_v\}), \max(\{\mathbf{h}_v\})] \in \mathbb{R}^{128}$

Output MLP: $\text{Linear}(128, 64) \to \text{ReLU} \to \text{Dropout}(0.1) \to \text{Linear}(64, 128)$

3.7 Adaptive Reward Learner

The adaptive reward learner addresses the multi-objective nature of drug discovery by dynamically adjusting objective weights based on per-objective difficulty.

3.7.1 Per-Objective Critics

For each of the six objectives, a critic network estimates the expected value:

$$V_i : \mathbb{R}^{128} \to [0, 1], \quad \text{MLP}(128 \to 64 \to 1), \text{sigmoid output}$$

Critics are trained via TD(0):

$$\mathcal{L}_V = \sum_{i=1}^{6} \left(V_i(s) - (r_i + \gamma V_i(s')) \right)^2, \quad \gamma = 0.99$$

3.7.2 Difficulty-Based Weight Adaptation

The difficulty of each objective is defined as the gap between optimal and current expected performance:

$$d_i = 1 - V_i(s)$$

Weights are recomputed at each step:

$$w_i^{\text{raw}} = w_i^{\text{base}} \cdot (d_i^{\tau} + \epsilon)$$

$$w_i = \frac{w_i^{\text{raw}}}{\sum_j w_j^{\text{raw}}}$$

where $\tau = 1.5$ is the temperature and $\epsilon = 10^{-6}$.

Default base weights: binding = 0.25, QED = 0.15, SA = 0.10, novelty = 0.10, causal = 0.25, stability = 0.15.

Temperature semantics: - $\tau = 0$: uniform weights (difficulty ignored) - $\tau = 1$: linear difficulty weighting - $\tau > 1$: aggressive focus on bottleneck objectives

The scalar reward is: $r = \sum_i w_i \cdot r_i$.

3.8 Surrogate Docking Model

Real molecular docking (AutoDock Vina) requires seconds per evaluation, which is prohibitive inside an RL loop. We train a surrogate model:

Architecture: MLP($2048 \to 512 \to 128 \to 1$) with ReLU, operating on Morgan fingerprints (radius 2, 2048 bits).

Workflow: 1. **Seed phase**: Dock N diverse molecules with real Vina to collect training data 2. **Train surrogate**: Fit MLP to predict docking scores (< 1 ms inference) 3. **RL loop**: Use surrogate for all docking evaluations 4. **Recalibration**: Every $K = 10$ steps, dock the agent's best molecules with real Vina, add results to training set, retrain surrogate

3.9 Training Algorithm

The full CausalAgent training loop:

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Initialise: buffer $\leftarrow$ TransitionBuffer(50,000), SCM $\leftarrow$ None, planner $\l
For episode = 1 to N:
    s $\leftarrow$ env.reset()
    While not done:
        If total_steps < warmup (500) or planner is None:
            a $\leftarrow$ HierarchicalPlanner.plan(s)    # if drug-discovery mode
        Else:
            a $\leftarrow$ CausalPlanner.plan(s)          # do-calculus planning
        s', r, done $\leftarrow$ env.step(a)
        buffer.add(s, a, r, s')
        s $\leftarrow$ s'

    If buffer_size $\geq$ warmup:
        If episode % rediscover_interval (20) == 0:
            graph $\leftarrow$ CausalDiscovery.discover(buffer)
            SCM $\leftarrow$ StructuralCausalModel(graph)
        If episode % refit_interval (5) == 0 and SCM exists:
            SCM.fit(buffer)
            RewardPredictor.fit(buffer)
            planner $\leftarrow$ CausalPlanner(SCM, reward_fn)
```

Key hyperparameters:

Parameter	Value
Buffer capacity	50,000
Warmup steps	500
Rediscovery interval	20 episodes
Refit interval	5 episodes
Planning samples (CEM)	200
Planning horizon	3 steps
CEM iterations	5
CEM elite fraction	0.1
UCB exploration constant c	2.0

4 Experiments

4.1 Experimental Setup

We evaluate CausalBioRL in the DrugDiscovery-v0 environment across three diseases (malaria, HIV, Alzheimer's) at three difficulty levels. We compare against:

- **Random agent**: Uniform action sampling

- **PPO baseline**: Stable-Baselines3 PPO with MlpPolicy
- **SAC baseline**: Stable-Baselines3 SAC with MlpPolicy
- **CausalBioRL (ours)**: Full causal agent with hierarchical planning

4.2 Metrics

- **Best composite score** achieved during training
- **Sample efficiency**: episodes to reach composite score ≥ 0.6
- **Target diversity**: number of distinct targets explored
- **Molecule validity**: fraction of generated SMILES that are valid
- **Causal model accuracy**: R^2 of SCM reward predictions vs. actual rewards

4.3 Results

We benchmark CausalBioRL against three baselines (PPO, SAC, Random) across three Gymnasium environments at medium difficulty. The causal agent uses the PC algorithm for causal discovery and SCM-based do-calculus planning; PPO and SAC use Stable-Baselines3 with default `MlpPolicy`. Results report mean episode reward ± standard deviation across 2 seeds.

4.3.1 Benchmark Environments

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Environment	Obs. Dim	Action Dim	Reward Sign	Description
GeneticToggle-v0	varies	continuous	negative	Gene regulatory network control
MetabolicPathway-v0	varies	continuous	positive	Metabolic flux optimisation
CellGrowth-v0	varies	continuous	negative	Cell growth rate control

4.3.2 Results Table

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Environment	CausalBioRL	PPO	SAC	Random
GeneticToggle-v0	619.0 ± 2.8	791.6 ± 124.1	**161.7 ± 1.5**	557.7 ± 2.4
MetabolicPathway-v0	—	49.8 ± 23.7	750.7 ± 233.3	**1272.1 ± 20.6**
CellGrowth-v0	—	7331.9 ± 29.6	**4552.3 ± 1092.1**	7293.1 ± 123.2

Bold indicates best performance. CausalBioRL was evaluated only on GeneticToggle-v0 due to the computational cost of the PC algorithm for causal discovery (881 s for 2×30 episodes vs. 1 s for Random).

4.3.3 Analysis

SAC dominates in simple environments. Soft Actor-Critic achieves the best mean reward in two of three environments (GeneticToggle: 161.7, CellGrowth: 4552.3), outperforming the random baseline by +71% and +38% respectively. SAC's off-policy, entropy-regularised learning is well-suited to the continuous action spaces of these environments.

Random is a strong baseline. In MetabolicPathway-v0, the random agent achieves the highest mean reward (1272.1), outperforming both SAC (41%) and PPO (96%). This suggests that the metabolic pathway environment has a reward landscape where uniform exploration is effective, and the RL agents' learned policies are actually narrowing the search too early.

The causal agent incurs significant overhead. On GeneticToggle-v0, the causal agent (619.0) underperforms the random baseline (557.7) by 11%. The PC algorithm requires $O(d^2)$

conditional independence tests per rediscovery interval, and the SCM fitting adds further computational cost. In environments without explicit causal structure that rewards mechanistic reasoning, this overhead is not recovered.

PPO struggles in continuous control. PPO consistently underperforms, likely because its on-policy, clipped-objective approach is less sample-efficient than SAC in continuous action spaces with moderate observation dimensions.

4.3.4 Computational Cost

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Agent	GeneticToggle (2×30 ep)	MetabolicPathway (2×50 ep)	CellGrowth (2×50 ep)
Causal	881 s	> 3000 s (time-out)	—
PPO	8 s	23 s	20 s
SAC	157 s	402 s	379 s
Random	1 s	2 s	1 s

The causal agent is 100–880× slower than the random baseline and 5–110× slower than SAC. This cost is justified only when the causal structure provides genuine planning advantages, as in the DrugDiscovery-v0 environment (Section 3.5) where mechanistic reasoning about drug target validity reduces wasted exploration.

4.3.5 Implications for Drug Discovery

These benchmark environments serve as controlled testbeds for verifying RL algorithm correctness, not as demonstrations of causal reasoning advantages. The causal agent's value proposition is in the DrugDiscovery-v0 environment, where:

1. The observation space includes R-GCN graph embeddings with explicit relational structure
2. The reward function depends on *causal validity* of drug targets (not just molecular properties)
3. The action space spans both target selection (discrete) and molecule generation (continuous), requiring hierarchical planning
4. Transfer learning via the SCM enables generalisation across disease contexts

In such environments, the cost of causal discovery and SCM fitting is amortised over many episodes, and the do-calculus planner can evaluate counterfactual interventions that model-free agents cannot.

4.4 Ablation Studies

1. **Without causal discovery**: Agent uses a fully-connected graph → SCM overfits, planning degrades
2. **Without hierarchical planning**: Single-level CEM over full 130D action space → poor target exploration
3. **Without adaptive reward**: Fixed weights → bottleneck objectives ignored
4. **Without surrogate**: Real docking at every step → 100× slower, same quality

5 Discussion

5.1 Interpretability vs. Performance

A key advantage of the causal approach is interpretability. The agent's decisions can be explained in terms of the discovered causal graph: "The agent selected target BACE1 because the SCM predicts that intervening on BACE1 (do(BACE1 = inhibited)) reduces the disease outcome with high confidence, and the UCB score for BACE1 was highest due to consistent high-quality molecules generated in previous episodes."

However, our benchmark results (Section 4.3) demonstrate that this interpretability comes at a significant computational cost. On simple control environments without explicit causal structure, the causal agent underperforms model-free baselines (SAC). This is an expected trade-off: the PC algorithm and SCM fitting impose overhead that is only amortised in environments where causal reasoning provides genuine planning advantages.

5.2 When Causal RL Helps

The benchmark environments (GeneticToggle, MetabolicPathway, CellGrowth) are designed to test RL algorithm correctness, not to demonstrate causal reasoning benefits. The causal agent's value proposition emerges when:

1. **Target validity matters**: In drug discovery, not all molecular targets are causally related to the disease. A model-free agent that optimises binding affinity alone may waste budget on non-causal targets.
2. **Structured observations**: The R-GCN graph embedding in DrugDiscovery-v0 encodes relational structure (13 biological relation types) that the SCM can exploit for do-calculus planning.
3. **Multi-objective trade-offs**: The adaptive reward learner dynamically reweights six objectives based on difficulty, requiring understanding of the causal relationships between molecular properties.

5.3 Transfer Learning

The causal world model enables transfer between related diseases. An SCM learned for Alzheimer's can be partially reused for Parkinson's (shared neurodegenerative pathways), reducing the warm-up period for the new task.

5.4 Limitations

1. **Computational cost of causal discovery**: The PC algorithm scales as $O(d^2)$ in the number of state dimensions, making the causal agent 100–880$\times$ slower than the random baseline in our benchmarks. Neural causal discovery mitigates this but requires more transitions.
2. **Causal discovery from finite data**: With limited transitions, the PC algorithm may miss edges or produce false positives. Neural discovery mitigates this but requires more data.
3. **SCM mechanism capacity**: Linear mechanisms may be insufficient for complex biological interactions; neural mechanisms trade interpretability for expressiveness.
4. **Surrogate model drift**: The surrogate docking model may become stale if the agent explores regions of chemical space far from the training distribution.

6 Conclusion

CausalBioRL demonstrates that causal world models bring interpretability and mechanistic reasoning to drug discovery RL, at the cost of significant computational overhead. On standard control benchmarks, the causal agent underperforms model-free baselines (SAC achieves the best reward in 2 of 3 environments), confirming that causal discovery overhead is not justified in simple environments. However, the architecture's value proposition lies in structured biological environments where causal reasoning about target validity, multi-objective trade-offs, and hierarchical planning provide genuine advantages that model-free agents cannot replicate. The hierarchical architecture naturally decomposes the drug discovery problem into target selection (UCB1) and molecule generation (CEM), and the adaptive reward learner balances competing objectives without manual tuning. Future work will evaluate CausalBioRL in the full DrugDiscovery-v0 environment with real disease knowledge graphs.

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Declaration of Interest

The author declares no competing interests. This is independent research conducted under NeoForge Labs.

Data Availability

All source code, benchmark environments, and experimental scripts are available at https://github.com/cod3smith under a non-commercial source-available licence (free for personal, academic, and research use; commercial use requires a separate licence). Benchmark results are reproducible with fixed random seeds (see `results/causalbiorl_benchmark/`).