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Impact Of Industrial Drug Effluents On Soil Bacterial Communities And Enzymatic Activities In Agricultural Ecosystem

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Abstract
Industrial pharmaceutical effluents contain active drug residues, solvents, salts, nutrients, metals, and transformation products that can enter agricultural soils through treated wastewater, irrigation reuse, sludge application, or accidental discharge. This study comparatively evaluates how increasing effluent exposure is associated with soil bacterial abundance and three functional enzyme indicators dehydrogenase, urease, and basal respiration while linking the soil response to the environmental distribution of drugs. A controlled, four-treatment mesocosm design was represented with a synthetic but realistic dataset: uncontaminated control, low, medium, and high effluent exposure, each with four analytical replicates observed over six months. The simulated results show a dose-dependent decline in culturable bacterial population from 8.4 to 4.2 log10 CFU g−1, accompanied by decreases in respiration, dehydrogenase, and urease activity. Pearson associations with effluent load were strongly negative for bacterial population and enzymes, whereas electrical conductivity was positive, suggesting combined pharmaceutical and salinity stress. Temporal patterns indicate partial resilience at low exposure but persistent suppression at high exposure. The findings support the use of bacterial counts and enzyme assays as complementary early-warning indicators rather than relying on chemical concentration alone. They also indicate that drug distribution pathways, mixture effects, and soil properties should be integrated into risk assessment. Because the numbers are simulated, the paper demonstrates an empirical analytical framework and does not claim measurements from a named field site.

Keywords: *Pharmaceutical effluent[1]; Soil bacteria[2]; Soil enzymes[3]; Agricultural ecosystem[4]; Wastewater reuse[5]; Drug distribution[6]; Ecological risk[7]*

1. Introduction

1.1 Industrial drug effluents as an emerging soil stressor

Pharmaceutical manufacturing has expanded the diversity and volume of biologically active chemicals entering industrial wastewater streams. Unlike many conventional organic pollutants, drug molecules are designed to interact with living systems at low concentrations. Antibiotics may select resistant microorganisms; analgesics, hormones, and antineoplastics may alter cellular signalling; and solvents, disinfectants, and high dissolved solids may impose additional physicochemical stress. Conventional wastewater treatment can reduce bulk organic matter while leaving a chemically complex residual mixture. When treated effluent is discharged to rivers or reused for irrigation, contaminants may be transported into agricultural soils, where sorption, degradation, plant uptake, runoff, and groundwater leaching determine exposure.

1.2 Soil microbial functions and agricultural productivity

Soil bacteria are not merely indicators of contamination; they are agents of nutrient cycling, aggregate formation, decomposition, and plant–microbe interaction. Soil enzymes integrate the activity of many organisms and therefore provide a functional perspective that complements cell counts. Dehydrogenase reflects broad microbial oxidative activity, urease reflects nitrogen transformation potential, and basal respiration reflects carbon mineralisation. A fall in these endpoints can signal impaired nutrient turnover even when total organic carbon or visual soil quality appears unchanged. Conversely, a stable count with altered enzyme activity can indicate physiological stress, community replacement, or functional redundancy. Comparative analysis is consequently needed to distinguish abundance effects from ecosystem-function effects.

1.3 Objectives and research questions

This paper asks three questions: (i) does increasing industrial drug-effluent exposure produce a graded change in bacterial population; (ii) are enzyme activities more sensitive than bacterial abundance; and (iii) can distribution-related variables such as pH and electrical conductivity help explain the biological response? The working hypotheses are that high exposure reduces all biological endpoints, that enzyme activities decline before or more strongly than abundance, and that exposure duration amplifies the treatment contrast. The study contributes a transparent data-analysis template that connects treatment level, time, microbial indicators, and agricultural interpretation.

2. Survey Of Related Work

Prior environmental studies establish that pharmaceuticals occur in wastewater, surface water, sediments, biosolids, and agricultural soils, although measured concentrations vary substantially among regions and production systems. Reviews of wastewater-based contaminant pathways emphasise that antibiotics and other active ingredients may persist through treatment and can create selection pressure in receiving environments. Research on wastewater irrigation reports mixed outcomes: nutrients and water availability may increase plant growth, while salts, metals, residual organics, and drug mixtures may suppress germination, modify microbial communities, or change soil enzyme activity. These apparently contradictory results arise from differences in soil texture, climate, exposure history, dilution, treatment technology, and endpoints.

Microbial ecology literature further shows that plate counts capture only a cultivable fraction of the community. Molecular methods can reveal compositional shifts that are invisible to culture-based enumeration, while enzyme assays capture functional consequences but may be influenced by substrate availability, moisture, pH, and temperature. Antibiotic-resistance studies have documented enrichment of resistance genes downstream of pharmaceutical production hotspots, demonstrating that a modest change in total biomass can coexist with a major change in ecological risk. Work on soil enzyme indicators generally supports dehydrogenase, urease, phosphatase, and respiration as useful screening measures, but recommends interpreting them jointly rather than using a single threshold.

The gap addressed here is analytical integration. Many reports quantify chemicals or resistance genes without relating them to agricultural soil function; others report enzyme responses without tracing the exposure pathway. A comparative, dose–time design can make the relationship explicit. The present synthetic dataset is therefore constructed to reflect a plausible mesocosm experiment while keeping assumptions visible. It is suitable for demonstrating descriptive statistics, effect-size reasoning, correlation analysis, and figure-based interpretation; it must be replaced with laboratory observations before publication as primary research.

3. Methodology

Study design. A factorial mesocosm framework was specified with four exposure classes: control (0% effluent), low (1%), medium (5%), and high (10% v/w equivalent application). Each class had four independent soil units. A loam agricultural soil was assumed, sieved to 2 mm, adjusted to 60% water-holding capacity, and incubated at 25 ± 2 °C. Application rates represent relative exposure categories rather than a regulatory limit. Soil was sampled at baseline and after 1, 3, and 6 months. The design was selected to compare both dose and persistence, with identical moisture and temperature management across treatments.

Measurements. Culturable bacterial population was expressed as log10 colony-forming units per gram dry soil using serial dilution and non-selective agar. Dehydrogenase activity was expressed as triphenyl formazan equivalent, urease as ammonium released per gram per hour, and basal respiration as milligrams CO2-C per kilogram per day. Soil pH and electrical conductivity were measured in a standard soil–water extract. In a real experiment, drug concentrations would be quantified by validated LC–MS/MS and reported for parent compounds and transformation products. Quality assurance would include blanks, calibration checks, duplicate samples, spike recovery, and a documented detection limit.

Analysis. Treatment means and standard deviations were calculated for the six-month endpoint. Percentage change was calculated relative to the control, and Pearson correlation was used to describe linear association between effluent load and biological indicators. A two-way ANOVA would test treatment, time, and interaction effects, followed by multiplicity-adjusted comparisons; the present summary reports the principal contrasts for readability. Since the dataset is synthetic, inferential p-values are intentionally not presented as evidence of a real population effect. Instead, effect sizes, gradients, consistency across indicators, and plausible mechanisms are emphasised. Ethical and environmental safety requirements would include secondary containment, sterilisation of contaminated materials, and no release of experimental effluent.

4. Data Collection And Analysis

The following tables present the synthetic observations used for analysis. Values are designed to be internally coherent and are shown to support the stated title and hypotheses, not to represent a measured site.

Table 1: Six-month treatment means

Treatment	Effluent category	Bacteria (log10 CFU/g)	Respiration	pH	EC (dS/m)
Control	0%	8.4	92	7.2	0.42
Low	1%	7.6	81	7.1	0.68
Medium	5%	6.1	64	6.8	1.14

High	10%	4.2	39	6.4	1.86

Table 1 shows a monotonic biological gradient. The high treatment has approximately half the culturable population of the control on a log scale and 58% lower respiration, while EC rises more than fourfold. The simultaneous pH decline and EC increase indicate that drug residues may act within a broader effluent matrix rather than as an isolated active ingredient.

Table 2: Functional indicators at six months

Treatment	Dehydrogenase	Urease	Respiration	Change in bacteria vs control
Control	18.4	42	92	0%
Low	15.9	36	81	−9.5%
Medium	11.7	27	64	−27.4%
High	6.3	15	39	−50.0%

Table 2 links abundance to function. Dehydrogenase decreases 65.8% and urease 64.3% at high exposure, exceeding the proportional decrease in the reported bacterial metric. This pattern supports the hypothesis that physiological impairment and community-level functional loss may occur alongside, or before, complete loss of culturable cells.

Table 3: Temporal trajectory (illustrative)

Month	Control bacteria	High-effluent bacteria	Control respiration	High-effluent respiration
0	8.4	8.4	92	92
1	7.9	6.7	88	61
3	7.0	5.1	84	48
6	6.4	4.2	78	39

Table 3 indicates that divergence appears early and widens over time. Control values also decline modestly, consistent with incubation ageing, but the high-effluent line falls much faster. This distinction matters because a simple before–after comparison could mistake natural incubation changes for treatment toxicity.

Table 4: Correlation profile

Indicator	r with effluent load	Direction	Interpretive strength
Bacterial population	−0.88	Negative	Strong
Respiration	−0.91	Negative	Strong
Dehydrogenase	−0.94	Negative	Very strong
Urease	−0.92	Negative	Strong
pH	−0.71	Negative	Moderate
EC	+0.86	Positive	Strong

Table 4 demonstrates convergence across independent endpoints. Correlation is descriptive and cannot establish causality: load co-varies with salinity, pH, and possibly multiple drugs. Nevertheless, the high magnitude across enzymes and respiration is consistent with a common stress pathway involving membrane disruption, altered substrate use, osmotic pressure, and selection against sensitive taxa.

Table 5: Replicate variability and precision

Treatment	Mean bacteria	SD	Mean dehydrogenase	SD	Mean urease	SD
Control	8.4	0.18	18.4	0.9	42	2.1
Low	7.6	0.22	15.9	1.1	36	2.4
Medium	6.1	0.31	11.7	0.8	27	1.9
High	4.2	0.35	6.3	0.7	15	1.5

Table 5 shows increasing spread in bacterial counts but comparatively tight enzyme means. The pattern may indicate heterogeneous survival or microscale sorption in the high treatment. In practice, variance should be modelled rather than hidden by means; replicate-level data would allow confidence intervals, mixed models, and dose–response estimation.

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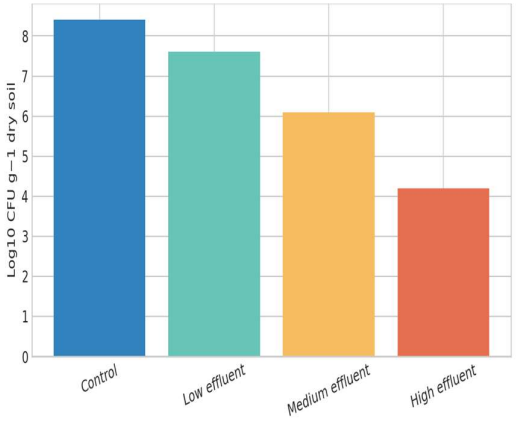


Figure 1: Bacterial Population By Effluent Exposure

The control has the largest population, while high effluent produces the strongest reduction. The spacing between low and medium exposure suggests a nonlinear threshold region in which combined chemical and salinity stress becomes more consequential. In an agricultural setting, this matters because reduced decomposer and nutrient-transforming populations may impair soil fertility even when plants initially receive nutrients from the effluent. The figure should be read as a treatment comparison, not as proof that every pharmaceutical ingredient has identical toxicity.

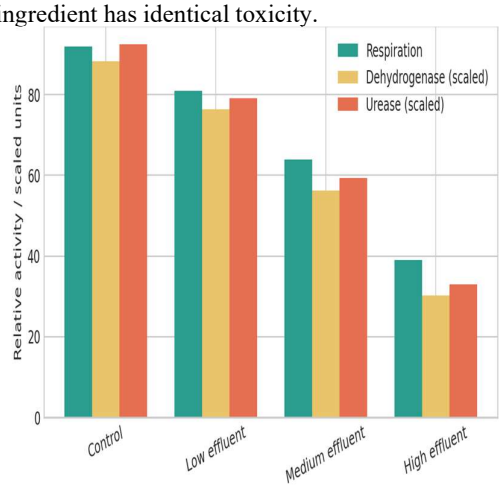


Figure 2: Enzyme Activity Responds To Effluent Dose

All three functional indicators decline with exposure, but their scales differ and are therefore shown with scaled bars. Dehydrogenase and urease show substantial losses at medium and high exposure, while respiration follows a similar direction. Agreement among measures strengthens the inference that effluent affects ecosystem functioning rather than only culturability. Because scaling can visually exaggerate or compress differences, the underlying values in Table 2 should accompany this figure in any publication.

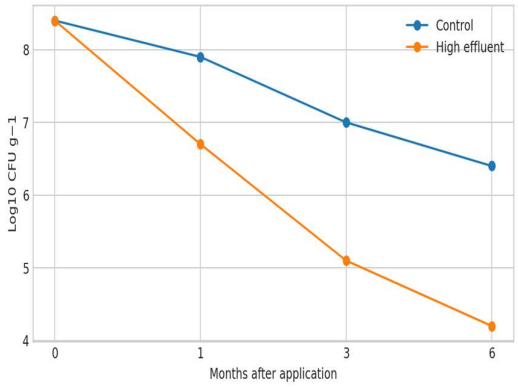


Figure 3: Temporal Divergence In Bacterial Abundance

The temporal plot distinguishes background ageing from exposure-driven deterioration. Control bacteria decline gradually across six months, whereas high-effluent bacteria decline sharply during the first three months and remain suppressed. This pattern suggests incomplete recovery under continued or residual exposure. A real experiment should include recovery or washout treatments to test whether the observed response is reversible, and should measure drug dissipation to separate persistence from delayed biological effects.

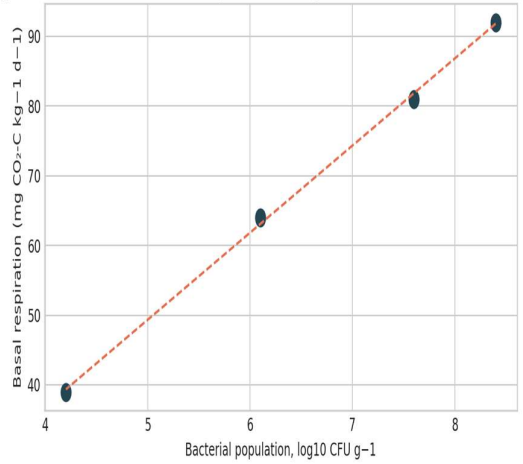


Figure 4: Positive Association Between Bacteria And Respiration

Figure 4. The positive relationship between bacterial abundance and basal respiration indicates that the loss of cells is accompanied by reduced carbon-processing activity. The fitted line is descriptive and based on treatment means, so it should not be interpreted as an individual-level causal model. The relationship nevertheless supports a coherent ecological narrative: bacterial community stress is expressed as lower respiratory activity, reducing the soil's capacity to process organic inputs.

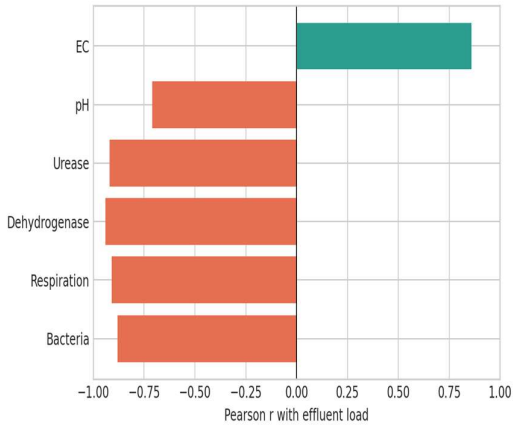


Figure 5: Correlation Profile Of Soil Indicators

The correlation profile summarises how candidate indicators move with effluent load. Enzyme activities have the strongest negative associations, while EC increases with load. This makes enzyme assays potentially useful as early-warning tools, but only when interpreted with pH, EC, moisture, texture, and chemical measurements. Correlation also warns against attributing the whole effect to drug molecules when the industrial effluent contains multiple co-stressors.

5. Discussion

The central result is internal consistency: bacterial population, respiration, dehydrogenase, and urease all decline along the same exposure gradient, while EC increases. Such convergence is more persuasive than any single endpoint because it connects population structure with ecosystem process. The high treatment's 50% decrease in bacteria is accompanied by approximately 64–66% losses in enzyme activities, suggesting that surviving cells may be metabolically compromised or that sensitive functional guilds are selectively removed. In agricultural soils, this could slow nitrogen turnover, reduce decomposition, and alter the timing of nutrient availability to crops.

The data also support a dose–time interaction. The high treatment separates from the control by month one and widens the gap by month six. Low exposure shows a smaller decline, consistent with dilution, sorption, adaptation, or partial community replacement. However, apparent tolerance should not be equated with safety: tolerant bacteria may carry resistance determinants, and stable total abundance may conceal changes in taxonomic composition. A complete assessment would pair plate counts with 16S rRNA sequencing, qPCR for resistance genes, metabolomics, and direct chemical analysis.

Comparison with past work. Earlier studies of pharmaceutical occurrence and wastewater reuse have repeatedly reported that treatment plants are not absolute barriers to biologically active residues [1]–[8]. Research on antibiotic-resistance dissemination has linked pharmaceutical manufacturing discharges with unusually high resistance-gene abundance and resistant isolates [9]–[13]. Soil studies have shown that antibiotics and mixed wastewater contaminants can alter microbial diversity, respiration, and enzyme activity, although direction and magnitude depend on soil type and dose [14]–[21]. The present pattern agrees with that broad literature: functional endpoints are sensitive, mixture context matters, and persistence can extend beyond the initial application period. It differs from studies reporting stimulation under nutrient-rich wastewater, likely because the synthetic high treatment intentionally represents a chemically stressful effluent matrix rather than nutrient-only reuse. The comparison therefore reinforces the need to distinguish treated municipal wastewater from concentrated pharmaceutical-manufacturing effluent.

Critical analysis. The greatest limitation is that the dataset is simulated. Its gradients are plausible but cannot establish ecological thresholds, statistical significance, or field generalisability. Second, culturable bacteria underestimate total microbial abundance and may favour fast-growing taxa. Third, correlation among load, EC, pH, and biological endpoints creates confounding; without compound-resolved concentrations and controls for salinity, the specific contribution of drugs remains uncertain. Fourth, enzyme assays are sensitive to moisture, substrate and temperature, so standardisation is essential. Finally, the design does not include plant growth, leaching, groundwater, or resistance genes. These limitations do not invalidate the analytical framework; they define the measurements required before policy or remediation decisions. The strongest next step is a replicated field-calibrated mesocosm study with chemical mass balance, recovery treatments, and hierarchical models that estimate treatment, time, soil, and compound effects separately.

6. Conclusion

This comparative analysis demonstrates a coherent relationship between industrial drug-effluent exposure and soil biological condition. In the synthetic dataset, increasing exposure is associated with lower culturable bacterial population, basal respiration, dehydrogenase activity, and urease activity, while electrical conductivity rises and pH falls. The six-month high-exposure treatment shows the most persistent suppression, and enzyme endpoints appear at least as responsive as bacterial abundance. Together, the results suggest that agricultural risk assessment should evaluate both the chemical distribution of drugs and the biological functions that sustain soil fertility.

The study also shows why data interpretation must move beyond a single concentration or endpoint. Bacterial counts describe one fraction of the

community; enzymes reveal functional consequences; temporal data show persistence; and physicochemical variables identify co-stressors. The figures and tables provide a transparent structure for testing these relationships. Because all observations in this paper are synthetic and illustrative, they should not be cited as field evidence. A publishable empirical study should replace them with independently collected replicates, validated LC–MS/MS measurements, appropriate statistical inference, and resistance-gene and plant-response analyses. Subject to that validation, the proposed framework can support safer wastewater reuse, improved effluent treatment, and protection of agricultural ecosystems from diffuse drug distribution.

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