

Systemic toxicological safety of oral 5-deazaflavin TND1128 used in anti-aging practice: a secondary assessment of publicly disclosed hepatic signals

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Research Article

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Abstract

Background: Oral agents promoted or used for anti-aging and aesthetic purposes create a toxicological question that extends beyond local cutaneous effects because systemic exposure can produce off-target organ injury. TND1128, a synthetic 5-deazaflavin derivative investigated as a mitochondrial modulator, has been used orally in Japanese anti-aging practice.

Objective: To characterize the toxicological evidence base for TND1128 and assess publicly disclosed hepatic abnormalities as a pharmacovigilance signal rather than as proof of causality.

Methods: We conducted a targeted secondary toxicological evidence assessment through 27 September 2026 using PubMed-indexed studies, official Japanese clinical-study registry records, society conference material, and first-party clinical disclosures. Evidence was mapped across exposure characterization, hazard-oriented endpoints, target-organ findings, dechallenge, dose-response information, denominator quality, and data required for drug-induced liver injury (DILI) adjudication.

Results: Three PubMed-indexed TND1128 papers were identified; all were preclinical studies centered on neuronal or mitochondrial efficacy-related endpoints, and none provided peer-reviewed human safety outcomes. A 2024 Japanese Society of Anti-Aging Medicine abstract reported oral TND1128 at 100 mg/day, more than 1,000 cumulative prescriptions, and two cases described as drug-induced liver dysfunction with AST/ALT elevation. The treating institution subsequently disclosed additional hepatic cases and reported improvement after cessation. Exact aminotransferase values, upper limits of normal, alkaline phosphatase, bilirubin, INR, latency, concomitant exposures, alternative-cause evaluation, and a verified unique-patient denominator were unavailable. A 2026 registered observational study (target n=10) remains recruiting without posted results.

Conclusions: The available evidence supports a publicly disclosed hepatic safety signal that warrants verification, but not a causal diagnosis or incidence estimate. Continued human use should be accompanied by structured hepatobiliary safety monitoring, transparent patient-level reporting, and dedicated systemic toxicology; pancreatic enzymes may be assessed where clinically indicated.

Introduction

Oral supplements and systemic drugs are increasingly discussed in the management of skin aging and aesthetic concerns. Dermatology reviews have emphasized that evidence for many systemic anti-aging approaches remains heterogeneous and that oral agents are not intrinsically free of adverse effects [1, 2]. From a toxicological perspective, this creates an important distinction: an intervention may be selected for an aesthetic or skin-aging objective while its clinically relevant toxicity emerges in an extra-cutaneous organ. Safety assessment therefore requires separation of intended use, systemic exposure, biological plausibility, and target-organ harm.

TND1128 (10-ethyl-3-methylpyrimido[4,5-b]quinoline-2,4(3H,10H)-dione) is a synthetic 5-deazaflavin derivative described in the peer-reviewed literature as a mitochondria-modulating compound. A 2021 study examined cultured mouse hippocampal neurons [3]. A 2023 study evaluated calcium dynamics in brain slices from mice pretreated subcutaneously with TND1128 [4], and a 2024 study evaluated mitochondrial membrane potential and ATP-related indices after intraperitoneal pretreatment in mice [5]. These studies provide mechanistic and pharmacodynamic hypotheses, but their indexed reports are not designed as dedicated systemic toxicology studies and do not establish human safety.

Public Japanese clinical materials indicate that TND1128 has nevertheless been administered orally to humans in anti-aging practice. A 2024 conference abstract reported more than 1,000 cumulative prescriptions at 100 mg once daily and two cases described as drug-induced liver dysfunction with AST/ALT elevation [6]. A current first-party clinical disclosure reports subsequent additional cases and states that the abnormalities improved after cessation [7]. Under contemporary pharmacovigilance principles, a safety signal is information that suggests a potentially causal association of sufficient plausibility to justify further evaluation; the presence of a signal does not itself establish causality [8].

The toxicological question is therefore narrower than whether TND1128 is effective for aging-related outcomes. It is whether the available evidence is sufficient to identify a target-organ safety signal, what can and cannot be inferred from that signal, and which data are missing for formal risk characterization. We addressed these questions using a source-hierarchical secondary evidence assessment with prespecified emphasis on hepatic toxicology and DILI adjudication.

Methods

Study design and toxicological framework

We conducted a targeted secondary toxicological evidence assessment using publicly accessible records. The analysis was not designed to estimate treatment efficacy and was not a meta-analysis. Evidence was organized across five toxicology domains: (i) exposure characterization (route, dose, duration and denominator); (ii) preclinical hazard-oriented evidence; (iii) human target-organ observations; (iv) reversibility or dechallenge information; and (v) data sufficiency for formal causal and severity assessment. The search cut-off was 27 September 2026.

Source hierarchy and search strategy

The source hierarchy was prespecified as: peer-reviewed journal articles and official clinical-study registries; scientific-society conference abstracts or proceedings; government records; and first-party clinical disclosures from the treating institution. Third-party commentary, social-media posts, commercial summaries and opinion pieces were excluded as primary evidence. PubMed was searched using the exact term “TND1128” and combinations of “5-deazaflavin” with TND1128. Japanese equivalents (5-エチル-3-メチルピリミド[4,5-b]キノリン-2,4(3H,10H)-ジオン and TND1128) were used to locate Japanese society records and official clinical-

study registry material. The Japan Registry of Clinical Trials (JRCT) was searched for human TND1128 studies. First-party clinical webpages were included only when they contained explicit exposure or safety information.

Eligibility and data extraction

Records were eligible if they specifically evaluated or reported TND1128 exposure, pharmacology, toxicology, clinical use, or adverse events. For each source we extracted publication type, biological model or clinical population, dose and route when available, primary endpoint orientation, whether systemic or organ-specific toxicology was evaluated, human exposure denominator, hepatic laboratory findings, discontinuation information, peer-review status, and availability of patient-level data. Reports plausibly describing the same clinical experience were treated as overlapping disclosures and were not numerically pooled.

Signal identification and interpretation

We used “safety signal” in the pharmacovigilance sense of information from one or more sources that suggests a potentially causal association and justifies verificatory action [8]. We did not use the regulatory term “validated signal,” because no formal regulatory signal-validation process was undertaken. Repetition across public records, biologic target-organ plausibility, temporal information, and dechallenge were treated as signal-strengthening features; absence of a stable denominator, dose-response information, rechallenge, alternative-cause evaluation, or independent adjudication limited inference.

Hepatic toxicology and DILI assessment

Public hepatic observations were compared with the American Association for the Study of Liver Diseases (AASLD) practice guidance for drug-, herbal-, and dietary supplement-induced liver injury [9]. Clinically significant DILI is commonly defined using specified elevations of AST/ALT, alkaline phosphatase (ALP), bilirubin, or INR; DILI phenotype is classified with the R value, $R = (ALT/ULN)/(ALP/ULN)$, and diagnosis requires temporal exposure information and exclusion of alternative causes [9]. We also assessed whether publicly available data were sufficient for the revised electronic causality assessment method (RECAM) [10]. Because source data did not contain the required variables, no formal DILI score was calculated.

Denominator and quantitative analysis

The term “cumulative prescriptions” was retained exactly as reported. It was not interpreted as a count of unique exposed individuals and was not used to calculate incidence. No pooled event rate was

calculated because the denominator applicable to later disclosed cases was undefined and follow-up completeness was unknown. The analysis was therefore qualitative and evidence-mapping in nature.

Ethics

This study used only publicly accessible, non-identifiable published, registered, aggregate, and first-party disclosed information. The authors had no contact with patients and no access to private medical records. No attempt at re-identification was made. Ethics approval and participant consent were therefore not applicable to this document-based secondary analysis, subject to confirmation against any applicable institutional or local requirements before submission.

Results

Peer-reviewed TND1128 evidence is mechanistic rather than toxicological

Three PubMed-indexed peer-reviewed TND1128 articles were identified. The 2021 study used cultured mouse hippocampal neurons and reported changes in neuronal morphology and excitatory synapses [3]. The 2023 study used mice pretreated with TND1128 at 0.01–1 mg/kg subcutaneously before brain-slice calcium imaging [4]. The 2024 study used 0.1–10 mg/kg intraperitoneal pretreatment and evaluated mitochondrial membrane potential and an ATP-related index in mouse brain slices [5]. Across these indexed reports, the primary objectives were mechanistic or pharmacodynamic. No PubMed-indexed human TND1128 clinical safety article was identified, and no paper identified by the targeted search had dedicated repeated-dose systemic toxicology or human target-organ safety as its primary objective.

Human oral exposure and hepatic observations are publicly documented

The 2024 Japanese Society of Anti-Aging Medicine abstract described an ethics-approved observational clinical experience in which a 100-mg TND1128-containing capsule was administered once daily. It reported more than 1,000 cumulative prescriptions and two cases described by the treating investigator as drug-induced liver dysfunction with AST/ALT elevation [6]. The current treating-institution webpage likewise reports more than 1,000 cumulative administration cases, reiterates the two AST/ALT cases, states that additional cases described as drug-induced liver dysfunction subsequently occurred, and reports improvement after cessation in all disclosed cases [7]. These later disclosures were not added numerically to the 2024 cases because case overlap cannot be excluded.

The hepatic information constitutes a signal, not a causal diagnosis

Repeated disclosure of aminotransferase abnormalities during human exposure, together with reported improvement after discontinuation, provides qualitative information compatible with dechallenge and is sufficient to justify targeted verification. However, the public record does not provide exact AST or ALT values, laboratory ULNs, ALP, gamma-glutamyl transferase, total/direct bilirubin, INR, symptom status, hospitalization, latency from exposure to onset, resolution time, concomitant drugs or supplements, alcohol exposure, viral testing, autoimmune testing, imaging, or other competing-cause evaluation. Accordingly, clinically significant DILI thresholds cannot be tested, the R value cannot be calculated, Hy's-law-type severity assessment cannot be performed, and RECAM cannot be applied. The overall toxicological evidence pathway is shown in Fig. 1, and the evidence required for stronger inference is summarized in Table 2.

Incidence and dose-response cannot be estimated

The available denominator is “cumulative prescriptions,” not a verified number of unique individuals with complete safety follow-up. Duration of exposure, refill structure, laboratory-monitoring frequency, loss to follow-up, and the denominator applicable to subsequently disclosed cases are not publicly specified. Dividing two cases by 1,000 prescriptions would therefore create an unsupported incidence estimate. Likewise, no patient-level dose-response or exposure-response relationship can be evaluated.

Formal prospective evidence remains pending

jRCT1030260019 was first publicly registered on 3 April 2026 as an observational, open, single-arm study evaluating safety and efficacy of oral TND1128 in adults with age-related symptoms [11]. The registry lists a target sample size of 10 and a recruitment period extending to March 2030. At the search cut-off it was recruiting and no outcome or safety results were posted. The registry therefore confirms ongoing formal human investigation but does not yet resolve the hepatic signal. The TND1128-specific evidence identified in this assessment is summarized in Table 1.

Table 1
Toxicological evidence matrix for TND1128 through 27 September 2026

Source	Exposure / model	Primary endpoint orientation	Safety-relevant information	Toxicological interpretation
Katsurabayashi et al., 2021 [3]	Cultured mouse hippocampal neurons	Neuronal morphology / synapses	No human exposure; indexed report not designed for systemic safety	Mechanistic evidence only
Takahashi et al., 2023 [4]	Mouse pretreatment, 0.01–1 mg/kg s.c.; brain slices	Ca ²⁺ dynamics / mitochondrial function	No peer-reviewed human safety outcomes	Mechanistic / pharmacodynamic evidence
Takahashi et al., 2024 [5]	Mouse pretreatment, 0.1–10 mg/kg i.p.; brain slices	Mitochondrial membrane potential / ATP-related index	No peer-reviewed human safety outcomes	Mechanistic / pharmacodynamic evidence
JAAM abstract, 2024 [6]	100 mg orally once daily; >1,000 cumulative prescriptions	Clinical observational disclosure	2 cases described as drug-induced liver dysfunction with AST/ALT elevation	Core archival hepatic signal
Treating-institution disclosure [7]	> 1,000 cumulative administration cases	Clinical use / adverse-event disclosure	2 AST/ALT cases; later additional hepatic cases; improvement after cessation reported	Signal strengthening + qualitative dechallenge; no independent adjudication
jRCT1030260019, 2026 [11]	Oral TND1128; target n = 10; recruiting	Safety and efficacy observational study	No posted results at cut-off	Prospective evidence pending

Table 2
Toxicological signal assessment: available versus required evidence

Domain	Evidence needed for stronger inference	Publicly available TND1128 information	Inference
Signal detection	Repeated credible observations from one or more sources	AST/ALT abnormalities reported in conference and first-party clinical sources	Signal present; verification warranted
Exposure definition	Unique patient count, formulation, dose, duration, cumulative exposure	100 mg/day reported in 2024 abstract; cumulative prescriptions reported	Exposure magnitude incompletely characterized
Magnitude of hepatic injury	Exact AST/ALT, ALP, bilirubin, ULNs	AST/ALT elevation only; exact values absent	DILI thresholds cannot be tested
Injury phenotype	ALT/ULN and ALP/ULN	ALP and ULNs absent	R value cannot be calculated
Severity	Bilirubin, INR, symptoms, hospitalization, outcome	Not reported in aggregate disclosure	Severity and Hy's-law-type assessment unavailable
Temporality / dechallenge	Start, onset, stop, serial laboratory recovery	Improvement after cessation reported; timing absent	Qualitative dechallenge information only
Dose-response / rechallenge	Individual exposure-response or recurrence on re-exposure	Not reported	No dose-response or rechallenge inference
Alternative causes	Concomitant drugs/supplements, alcohol, viral, autoimmune, metabolic evaluation	Not available	Independent causal attribution not possible
Incidence	Unique exposed persons with complete follow-up	"Cumulative prescriptions"; later denominator undefined	No valid incidence estimate
Formal causality	Complete RECAM/RUCAM variables	Incomplete	No formal causality score

Discussion

This secondary assessment identifies an evidence asymmetry that is toxicologically important: TND1128 has peer-reviewed mechanistic studies and documented human oral use, but peer-reviewed human safety data have not kept pace with exposure. The central result is not a diagnosis of TND1128 hepatotoxicity. It is the presence of a publicly disclosed hepatic safety signal in a setting where the data required for verification and risk characterization remain incomplete.

The distinction between signal detection and causal attribution is fundamental. Pharmacovigilance signals are intended to trigger further assessment rather than to establish that an exposure caused an event [8]. Here, repeated AST/ALT abnormalities across an archival conference abstract and a later first-party disclosure, together with reported improvement after cessation, are sufficient to justify structured follow-up. At the same time, aminotransferase elevations are nonspecific, and alternative causes may include metabolic liver disease, alcohol, infection, muscle injury, concomitant prescription drugs, and other supplements. Without patient-level laboratory trajectories and competing-cause evaluation, the correct toxicological classification remains “signal requiring verification,” not “confirmed DILI.”

The dechallenge information increases the relevance of the signal but remains incomplete. AASLD guidance emphasizes start and stop dates, dose history, serial liver biochemistries, and the clinical course after withdrawal when evaluating suspected DILI [9]. The public disclosure reports improvement after cessation, but not latency, time to recovery, magnitude of improvement, or whether other exposures changed simultaneously. These missing variables preclude formal RECAM assessment [10] and prevent estimation of how much evidentiary weight should be assigned to dechallenge in individual cases.

The preclinical evidence base also has a toxicological gap. The three PubMed-indexed studies identified here focused on neuronal morphology, calcium dynamics, mitochondrial membrane potential, and ATP-related effects [3–5]. Those endpoints may support mechanistic hypotheses but do not substitute for a dedicated systemic safety package. Because TND1128 is administered orally in humans, future nonclinical work would be strengthened by repeated-dose studies using clinically relevant exposure ranges, toxicokinetic characterization, clinical chemistry, hematology, organ weights, and histopathology, with explicit attention to liver findings. Given the compound’s proposed mitochondrial activity, hepatic mitochondrial function and metabolite characterization may also be relevant hypothesis-generating endpoints. These are research priorities rather than a claim that any specific regulatory testing framework necessarily applies to the current product.

The relevance to aesthetic and skin-aging practice is practical. An agent can be used with the intention of modifying aging-related appearance or physiology while producing systemic exposure and extra-cutaneous toxicity. Dermatologic safety reasoning should therefore follow route of exposure rather than assume that the target organ of intended benefit will also be the target organ of toxicity. Reviews of oral skin-aging interventions already emphasize that systemic treatments and supplements can have adverse effects and that evidence quality is variable [1, 2]. TND1128 illustrates why safety evidence should be developed in parallel with mechanistic and efficacy claims.

For future clinical use or research, a structured safety dataset would materially improve interpretability. At minimum, prospective records should capture exact product identity and formulation; dose; start and stop dates; adherence; baseline and follow-up ALT, AST, ALP, gamma-glutamyl transferase, total and direct bilirubin; symptoms; concomitant medications and supplements; alcohol exposure; and prespecified evaluation of competing hepatic causes. INR should be measured when clinically indicated or when injury severity is a concern. If abdominal symptoms or other findings raise concern for

pancreatic involvement, lipase, with or without amylase, may reasonably be added. Current evidence does not identify pancreatic toxicity as a TND1128 signal, and pancreatic testing should not be presented as if such a signal had already been observed. The proposed minimum data fields for prospective toxicological surveillance are summarized in Table 3.

The denominator should also be redesigned. “Cumulative prescriptions” is useful as evidence that human exposure is not trivial, but it is unsuitable for incidence estimation. A pharmacovigilance-quality denominator requires unique exposed individuals, duration of exposure, cumulative dose, a defined laboratory-monitoring schedule, and ascertainment of patients lost to follow-up. With those data, event frequency, exposure-response patterns, and subgroup susceptibility could be estimated without conflating prescriptions with persons.

This study has limitations. The human safety evidence is partly based on conference material and first-party disclosure rather than independently adjudicated patient-level records. Public webpages are mutable. Cases may overlap across sources. The search was targeted to TND1128 and was not a broad systematic review of all 5-deazaflavin chemistry, metabolites, or analogues. Absence of a PubMed-indexed human safety paper does not exclude unpublished clinical data, non-indexed reports, or future publications. Finally, the present assessment cannot determine incidence, severity distribution, or causality. These limitations are central to the conclusion rather than incidental to it: they define the evidence gap that makes verification necessary.

Table 3
Proposed minimum data fields for prospective toxicological surveillance of oral TND1128

Domain	Proposed data fields	Purpose
Exposure	Product/formulation, batch where available, dose, frequency, start/stop dates, cumulative duration, adherence	Defines exposure and supports temporality/exposure-response assessment
Hepatobiliary laboratory panel	ALT, AST, ALP, GGT, total/direct bilirubin; baseline and serial follow-up	Quantifies injury magnitude and phenotype
Severity	Symptoms, INR when indicated, hospitalization, outcome	Distinguishes mild biochemical abnormality from clinically important injury
Co-exposures	Prescription drugs, OTC agents, supplements, alcohol	Supports alternative-cause and interaction assessment
Competing causes	Clinically appropriate viral, autoimmune, metabolic and imaging evaluation	Supports DILI adjudication
Dechallenge / rechallenge	Serial laboratory trajectory after withdrawal; any re-exposure only if it occurs clinically	Clarifies temporal association; deliberate rechallenge is not proposed
Pancreatic assessment	Lipase $\pm$ amylase when clinically indicated	Broadens evaluation only when symptoms or findings justify it
Denominator quality	Unique persons, monitoring completeness, loss to follow-up	Permits defensible event-frequency estimates

Conclusion

As of 27 September 2026, the peer-reviewed TND1128 literature identified in PubMed remains preclinical and mechanistically oriented, whereas public Japanese clinical sources document oral human exposure and repeated hepatic laboratory abnormalities. The available record supports a publicly disclosed hepatic safety signal and reported improvement after cessation, but does not establish event incidence, DILI phenotype, severity, or causal attribution. The toxicological priority is therefore verification: patient-level laboratory trajectories, exposure characterization, competing-cause assessment, reliable denominators, and dedicated systemic safety studies. Taken together, the current evidence indicates the need for structured hepatobiliary monitoring during continued human use of TND1128 and, where clinically appropriate, pancreatic-enzyme assessment, while avoiding inference beyond the available data.

Declarations

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Disclosure statement: The authors report no conflicts of interest.

Ethics approval: Ethics approval was not applicable to this analysis of publicly accessible, non-identifiable sources; this wording should be confirmed against applicable institutional and local requirements before submission.

Consent to participate: Not applicable.

Consent for publication: Not applicable.

Data availability: All data used in this study derive from publicly accessible sources cited in the manuscript. The evidence matrix in Tables 1–3 contains the extracted data used for the present analysis.

Author contributions: Author 1: conceptualization, methodology, evidence synthesis, toxicological interpretation, and manuscript drafting. Author 2: critical review, interpretation, and manuscript revision. Contributions should be confirmed by both authors before submission.

Generative artificial intelligence statement: OpenAI ChatGPT (GPT-5.6 Sol; accessed September 2026) was used to assist literature discovery, evidence organization, and language drafting. The human authors are responsible for verifying the cited sources, interpretations, and final manuscript.

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Figures

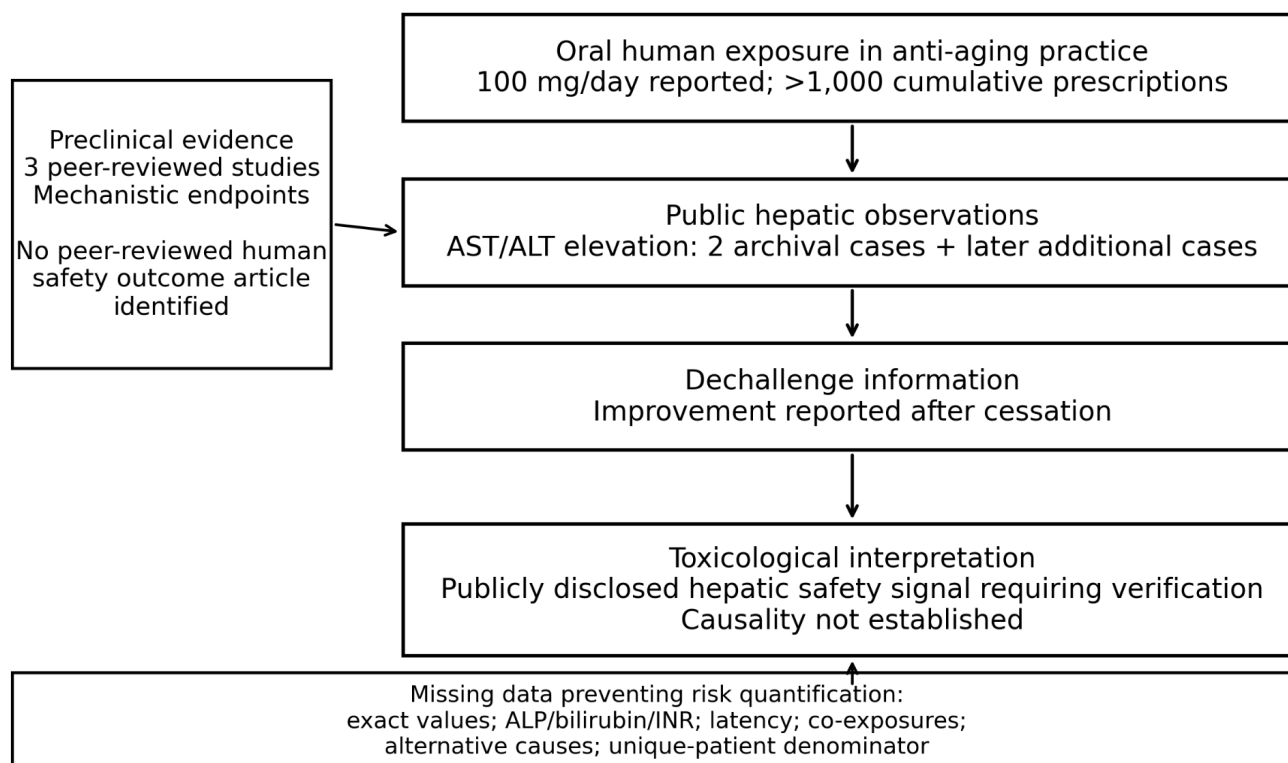


Figure 1

Toxicological evidence pathway for TND1128. Publicly disclosed hepatic abnormalities and reported improvement after cessation support signal detection, while missing laboratory magnitude, phenotype,

timing, alternative-cause, and denominator data prevent incidence estimation or causal adjudication.