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Integrative review on biomarkers of stress-triggered cardiac, renal, and multi-organ dysfunction in wildlife and zoo species

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Abstract

Wildlife and zoo animals are frequently exposed to acute and chronic stressors, including capture, restraint, translocation, live-stranding, predation attempts, environmental extremes, and human-wildlife conflicts. These stressors can precipitate severe physiological responses culminating in cardiac injury, acute kidney injury (AKI), and multi-organ failure. Stress-related syndromes particularly capture myopathy represent a significant cause of morbidity and mortality across multiple taxa. This review synthesizes current evidence on the pathophysiology, clinical manifestations, biochemical and molecular biomarkers, species susceptibility, and management of stress-induced organ damage in wildlife and zoological settings. Emphasis is placed on the mechanisms linking extreme stress to myocardial damage, myoglobinuric kidney injury, and systemic organ dysfunction. The review highlights common clinical features, the utility of emerging biomarkers such as cardiac troponins and creatine kinase, and the limitations posed by species heterogeneity and lack of standardized reference intervals. Evidence-based strategies for prevention, monitoring, and intervention are discussed, with recommendations for future research aimed at improving conservation and clinical outcomes.

Keywords: Capture myopathy, stress cardiomyopathy, cardiac injury, acute kidney injury, multi-organ failure, wildlife, zoo animals, cardiac troponin, biomarkers

1. Introduction

Stress responses in animals trigger a cascade of physiological adaptations that may be beneficial in the short term but deleterious if prolonged or extreme. In wildlife and zoo animals, stressors such as capture, handling, transportation, live-stranding, and confrontation with predators initiate heightened sympathetic activity and metabolic disturbances. When these stress responses exceed physiological tolerance, they can precipitate severe outcomes, including capture myopathy (CM) a systemic metabolic syndrome characterized by muscle damage, cardiac compromise, and renal dysfunction. CM has been described across a broad range of taxa, particularly in mammals and birds, and is increasingly recognized as analogous to human stress cardiomyopathy (*e.g.*, Takotsubo syndrome) (Rogers *et al.*, 2004, Arregui *et al.*, 2020, Dinesh *et al.*, 2020, Vera *et al.*, 2024) [4, 1, 7, 24].

Defining and diagnosing stress-related organ damage in wildlife is challenging due to limited access to diagnostic imaging and scarce baseline data for many species. Nonetheless, clinical and post-mortem studies have identified common features of cardiac and renal injury, including elevations in serum markers such as creatine kinase (CK), lactate dehydrogenase (LDH), cardiac troponin I (cTnI), blood urea nitrogen (BUN), and creatinine, along with histopathological evidence of muscle necrosis, myocardial degeneration, and renal tubular damage.

The interrelation between cardiac injury and kidney failure in stressed animals reflects a form of *animal cardiorenal syndrome*, wherein damage to one organ system exacerbates dysfunction in another. This review aims to provide an integrative, evidence-based overview of stress-induced organ injury syndromes in wildlife and zoo animals, focusing on pathophysiology, clinical assessment, biomarker profiles, species-specific susceptibilities, and management strategies (Carramenha, 2012) [18].

2. Pathophysiology of Stress-Induced Organ Injury

2.1 Capture Myopathy: Definition and Mechanisms

Capture myopathy (CM) is a metabolic disorder triggered by intense physical exertion or psychological stress during events such as pursuit, capture, restraint, transport, or live-stranding of wildlife. CM encompasses a spectrum of syndromes from hyper-acute (capture shock) to chronic forms and leads to severe muscle breakdown (rhabdomyolysis), metabolic acidosis, and multi-organ injury (Carramenha, 2012, Breed *et al.*, 2019, Dinesh *et al.*, 2020) ^[18, 2, 7].

The initial stress response involves sympathetic activation and catecholamine surge, causing increased heart rate, respiration, and muscle activity. If prolonged, this overwhelms cellular energy stores, leading to muscle cell membrane breakdown and release of intracellular contents such as myoglobin, CK, and troponins into circulation. These substances can be directly toxic to kidneys and other tissues, resulting in myoglobinuric nephrosis and acute kidney injury (Cebi *et al.*, 2022) ^[20].

Cardiac involvement in CM resembles human stress cardiomyopathy, with myocardial necrosis, contraction band necrosis, and vascular changes. Histopathological studies in cetaceans demonstrated acute myocardial degeneration with depletion of cardiac proteins and inflammatory infiltrates following live-stranding and rehabilitation efforts. Elevated levels of cTnI and CK have been correlated with such cardiac damage in multiple wild species (Spraker *et al.*, 2012, Arregui *et al.*, 2020) ^[8, 1].

2.2 Renal Injury and Multi-Organ Dysfunction:

Myoglobin released from damaged skeletal and cardiac muscle causes myoglobinuric acute kidney injury by precipitating in renal tubules, inducing oxidative stress and tubular necrosis. Laboratory findings frequently show elevated blood urea nitrogen (BUN) and creatinine, indicating impaired renal filtration (Cebi *et al.*, 2022) ^[20].

Beyond the heart and kidneys, systemic stress can produce multi-organ pathology, including hepatic congestion, adrenal insufficiency, and intestinal or pulmonary lesions. Histopathological examinations of CM cases show multifocal necrosis across many organs, reflecting the widespread impact of metabolic derangements and hypoxia resulting from prolonged stress (Cebi *et al.*, 2022) ^[20].

3. Clinical Manifestations and Biomarker Profiles

3.1 Cardiac Injury Markers & assessment

Cardiac injury in wild animals is most commonly assessed using biomarkers such as cardiac troponins (cTnI), which are highly specific indicators of myocardial cell damage. Elevated cTnI levels have been documented in live-stranded cetaceans and are correlated with histopathological evidence of stress cardiomyopathy (Spraker *et al.*, 2012, Arregui *et al.*, 2020) ^[8, 1].

Creatine kinase (CK) and lactate dehydrogenase (LDH) are less specific but indicate broader muscle damage including cardiac muscle. In free-ranging deer (Campos, 2024) subjected to intense exertion, significant elevations in LDH, AST (aspartate aminotransferase), and CK were observed, highlighting their utility in diagnosing exertional myopathy (Landau *et al.*, 2012) ^[13].

Table 1: Cardiac Injury Assessment in Wildlife and Zoo Animals (Landau *et al.*, 2012) ^[13]

Parameter	Wildlife Species Examples	Marker / Test	Type of Injury Detected	Advantages	Limitations / Notes
Cardiac Troponins (cTnI, cTnT)	Deer, Elk, Dolphins, Lions, Tigers	Serum cTnI / cTnT	Myocardial injury, necrosis, stress cardiomyopathy	Highly specific to cardiomyocytes; early detection	Species-specific reference ranges often unavailable; can rise due to systemic stress or trauma
Creatine Kinase (CK, CK-MB)	Ungulates, Carnivores, Marine mammals	Serum CK, CK-MB	Muscle injury including cardiac and skeletal	Rapid, widely measurable	Low cardiac specificity; influenced by exercise and handling
Electrocardiography (ECG)	Big cats, Horses, Primates	ECG	Arrhythmias, myocardial conduction abnormalities	Non-invasive; detects electrical dysfunction	Requires restraint or sedation; interpretation can be difficult in exotic species
Echocardiography (Echo)	Horses, Zoo Ungulates, Primates	2D / Doppler Echo	Structural abnormalities, ventricular hypertrophy, chamber dilation	Non-invasive, anatomical and functional assessment	Operator-dependent; sedation often required; costly equipment
Natriuretic Peptides (BNP, NT-proBNP)	Deer, Horses, Elephants	Serum BNP / NT-proBNP	Ventricular stretch, heart failure	Early functional marker; correlates with volume overload	Limited veterinary validation in wildlife; affected by renal function and stress
Acute Phase Proteins (CRP, SAA)	Carnivores, Ungulates, Primates	Serum CRP, SAA	Inflammatory response secondary to cardiac injury	Rapidly rises with systemic inflammation	Non-specific; elevations may occur in infection, trauma, or stress
Myocardial Imaging (CT / MRI)	Rare cases in captive elephants, big cats	Cardiac CT or MRI	Structural and perfusion abnormalities	High-resolution imaging; precise anatomical detail	Limited availability; sedation or anesthesia required; costly
Biomarker Panels (cTnI + CK + BNP)	High-risk zoo species	Combination of serum tests	Multi-pathway cardiac stress detection	Increased sensitivity; better diagnostic accuracy	Complex interpretation; species validation needed
Behavioral & Clinical Assessment	All wildlife / zoo animals	Heart rate, breathing, exercise tolerance	Functional stress or early cardiac compromise	Non-invasive; field-applicable	Low sensitivity; subjective; influenced by handling and environment

3.2 Renal and Systemic Biomarkers

Markers of renal injury include BUN and creatinine, which rise as glomerular filtration declines in the setting of acute tubular necrosis. The presence of myoglobin casts in renal tubules on histology supports the diagnosis of

myoglobinuric nephrosis (Cebi *et al.*, 2022)^[20].

Systemic biomarkers such as metabolic acidosis (low blood pH) and electrolyte imbalances signal advanced organ dysfunction in stressed animals, often accompanying cardiac and renal compromise.

Table 2: Comparative chart of Systemic Biomarker (Cebi *et al.*, 2022)^[20]

Parameter	Wildlife / Zoo Species Examples	Marker / Test	Type of Injury / Condition Detected	Advantages	Limitations / Notes
Serum Creatinine (sCr)	Carnivores (lions, tigers), Ungulates (deer, antelope), Primates	Serum creatinine	Glomerular dysfunction, chronic kidney injury	Widely used; routine lab test	Late marker; rises only after significant renal function loss; influenced by muscle mass and dehydration
Blood Urea Nitrogen (BUN / Urea)	Big cats, Elephants, Marine mammals	Serum urea	Renal excretory function, dehydration, high-protein diet	Simple; widely available	Non-specific; affected by diet, GI bleeding, catabolism, or dehydration
Symmetric Dimethylarginine (SDMA)	Carnivores, Primates, Ungulates	Serum SDMA	Early kidney dysfunction	Detects renal impairment before creatinine rises; minimally influenced by muscle mass	Limited species-specific reference ranges; costlier than traditional tests
Cystatin C	Carnivores, Horses, Primates	Serum cystatin C	Glomerular filtration rate (GFR) marker	Early indicator of renal dysfunction; less influenced by muscle	Limited validation in wildlife; expensive assay
Urinalysis (Proteinuria, Specific Gravity, Casts)	All wildlife species	Urine protein/creatinine ratio, specific gravity, sediment	Renal tubular or glomerular injury	Non-invasive; can detect subclinical renal disease	Requires sample collection; stress during handling may affect urine output
Electrolytes (Na ⁺ , K ⁺ , Ca ²⁺ , Phosphate)	Ungulates, Carnivores, Primates	Serum electrolytes	Acute kidney injury (AKI), dehydration, systemic imbalances	Routine lab tests; rapid	Influenced by hydration, diet, and stress; transient fluctuations may occur
Acute Phase Proteins (CRP, SAA, Haptoglobin)	Carnivores, Ungulates, Primates	Serum CRP, SAA, haptoglobin	Systemic inflammatory response, organ stress	Rapid rise in systemic stress; early detection	Non-specific; may rise due to infection, trauma, or other organ injuries
Liver-Kidney Function Panel (AST, ALT, ALP, GGT, LDH)	Big cats, Elephants, Primates	Serum enzyme levels	Multi-organ dysfunction, stress-induced injury	Provides holistic view of organ function	Non-specific; enzymes may rise due to handling stress, diet, or exercise
Neutrophil/Lymphocyte Ratio (NLR)	Carnivores, Ungulates, Birds	CBC parameters	Stress indicator, systemic inflammation	Non-invasive; simple hematology marker	Non-specific; influenced by capture, anesthesia, or acute stress
Pro-inflammatory Cytokines (IL-6, TNF- α , IL-1 β , MCP-1)	Primates, Carnivores, Ungulates	Serum/plasma cytokines	Systemic inflammation, multi-organ stress	Early detection of stress and inflammatory activation	Limited availability; expensive assays; species-specific validation required
Oxidative Stress Markers (MDA, GSH, SOD, Catalase)	Birds, Primates, Carnivores	Plasma / serum oxidative stress markers	Cellular oxidative damage due to systemic stress	Reflects subclinical organ stress; predictive of organ failure	Specialized lab required; species-specific baselines lacking
Urinary Kidney Injury Molecule-1 (KIM-1)	Marine mammals, Carnivores, Ungulates	Urine KIM-1	Early tubular injury	Detects kidney injury before creatinine rise	Limited validation in wildlife; sample collection challenging
Neutrophil Gelatinase-Associated Lipocalin (NGAL)	Carnivores, Ungulates, Primates	Serum / urine NGAL	Acute kidney injury, systemic stress	Early and sensitive; correlates with severity	Not widely standardized for wildlife; assay availability limited
Lactate	All wildlife	Serum lactate	Tissue hypoxia, systemic shock	Rapid marker of acute stress and perfusion	Non-specific; elevated in exercise, capture stress, trauma
Blood pH / Blood Gas (pO ₂ , pCO ₂ , HCO ₃ ⁻)	Ungulates, Primates, Big cats	Arterial or venous blood gas	Metabolic acidosis, respiratory compromise, multi-organ failure	Early detection of critical illness	Requires blood gas analyzer; stress during collection may alter results

3.3 Multi-Organ Failure Indicators

Multi-organ failure involves sequential or concurrent dysfunction of multiple organ systems, often reflected in

complex clinical presentations, including lethargy, hyperthermia, tachypnea, hypotension, and altered consciousness. Histopathology may reveal necrosis in

organs such as liver, spleen, pancreas, brain, and gastrointestinal tissues, pointing to systemic failure beyond primary cardiac and renal injury (Cebi *et al.*, 2022)^[20].

4. Species Susceptibility and Case Examples

4.1 Marine Mammals

Cetaceans such as Risso's dolphins have demonstrated capture myopathy with significant elevations in cTnI, CK, and BUN following live-stranding and attempted rehabilitation; histopathology confirmed vascular and acute degenerative lesions consistent with stress cardiomyopathy (Spraker *et al.*, 2012, Dinesh *et al.*, 2020, Arregui *et al.*, 2020)^[8, 7, 1].

4.2 Terrestrial Ungulates

Ungulates including deer (Campos, 2024), antelopes, and giraffes show significant susceptibility to CM, likely due to evolutionary traits such as high running speed and social behavior that predispose them to intense stress responses (Atkinson, 2012)^[14].

4.3 Birds and Zoo Species

Birds like wild turkeys and shorebirds and zoo animals such as wallabies have been reported to develop Takotsubo-like cardiomyopathy associated with capture myopathy, often leading to fatal outcomes (Montané & Marco, 2002, Rogers *et al.*, 2004, Arregui *et al.*, 2020)^[8, 9, 1].

5. Pathogenesis of Stress-Induced Organ Dysfunction

5.1 Capture Myopathy and Muscle Rhabdomyolysis

Stress-induced organ injury in wildlife is most commonly associated with capture myopathy (CM), a metabolic syndrome that arises from extreme exertion or psychological stress during capture, restraint, transport, or environmental challenges. CM is particularly documented in ungulates, birds, and marine mammals and is often fatal due to cascading physiological failures (Montané & Marco, 2002, Rogers *et al.*, 2004, Atkinson, 2012, Breed *et al.*, 2019, Dinesh *et al.*, 2020, Nueangphuet *et al.*, 2022, Vera *et al.*, 2024)^[16, 9, 14, 7, 4, 24].

The fundamental pathology involves muscle cell membrane disruption due to inadequate oxygen delivery, anaerobic metabolism, and accumulation of toxic metabolites (*e.g.*, lactic acid). This leads to rhabdomyolysis (Nueangphuet *et al.*, 2022)^[4], where intracellular components such as myoglobin, creatine kinase (CK), and electrolytes are released into the bloodstream, causing widespread metabolic derangement.

Rhabdomyolysis triggers a systemic cascade that increases circulating CK and lactate dehydrogenase (LDH), indicating skeletal and cardiac muscle injury. Concurrently, myoglobin released by injured muscle cells precipitates in renal tubules, leading to myoglobinuric nephrosis, electrolyte imbalances, and tubular necrosis a hallmark of stress-related acute kidney injury (Cebi *et al.*, 2022, Nueangphuet *et al.*, 2022)^[20, 4].

5.2 Cardiac Injury Mechanisms

Cardiac involvement in stress-induced syndromes mirrors human *stress cardiomyopathy* (*e.g.*, Takotsubo syndrome), characterized by myocardial degeneration, contraction-band necrosis, and hypercontracted myofibers. In marine mammals such as dolphins, elevated CK and cardiac troponin I (cTnI) during episodes of live-stranding and rehabilitation have been directly linked to myocardial compromise. Cardiac degeneration is further confirmed by histopathology showing wavy fibers, hypereosinophilia, vacuolization, and mononuclear infiltration within both atrial and ventricular myocardium following stress events (Arregui *et al.*, 2020)^[1].

5.3 Acute Kidney Injury (AKI)

Once myoglobin enters circulation, it is filtered by the kidney and may precipitate in renal tubules, especially under acidic conditions, forming obstructive casts that exacerbate tubular necrosis. Additionally, ferrihemate, a breakdown product of myoglobin, catalyzes free radical formation and lipid peroxidation, further injuring renal cells.

AKI associated with rhabdomyolysis is exacerbated by prolonged vasoconstriction secondary to catecholamine release in stress responses, which reduces renal perfusion, causing hypoxic injury to glomeruli and renal tubules (Cámara *et al.*, 2019, Nueangphuet *et al.*, 2022)^[5, 4].

5.4 Progression to Multi-Organ Failure

The integration of severe muscle damage, cardiac stress, and renal dysfunction can culminate in multi-organ failure (MOF). Histopathological evaluations frequently reveal multifocal necrosis across the central nervous system, liver, spleen, pancreas, and lungs, reflecting systemic ischemia and inflammatory responses. Electrolyte imbalances (*e.g.*, hyperkalemia), acidosis, and inflammatory cytokine release further drive dysfunction across organ systems as stress intensifies (Cámara *et al.*, 2019)^[5].

6. Clinical Assessment and Diagnostic Approaches

6.1 Biomarkers in Clinical Diagnosis (Cattet *et al.*, 2008)^[11]

Biochemical markers play a pivotal role in detecting the extent of organ injury in stressed wildlife:

- **Creatine Kinase (CK) and Lactate Dehydrogenase (LDH):** These are sensitive indicators of skeletal and cardiac muscle damage, widely employed in assessing exertional myopathy (Cattet *et al.*, 2008, Hartup *et al.*, 1999)^[11, 12] in wild species such as cervids.
- **Cardiac Troponin I (cTnI):** This is considered more cardiospecific and a reliable biomarker of myocardial necrosis. Elevated cTnI levels have been documented in dolphins with stress cardiomyopathy and in CM across other taxa (Arregui *et al.*, 2020)^[1].
- **Blood Urea Nitrogen (BUN) and Creatinine:** Elevations in these renal function markers are indicative of AKI and help distinguish prerenal from intrinsic renal injury.

Table 3: Comparative chart of biomarker with commercial availability of diagnosis test in market (Hartup *et al.*, 1999, Cattet *et al.*, 2008, Arregui *et al.*, 2020) ^[12, 11, 1]

Parameter / Biomarker	Commercial Test	Analyte Measured	Platform / Method	Diagnostic Use	Notes / Applicability
Cardiac Troponin I (cTnI)	Vcheck® Troponin I	Cardiac Troponin I	Fluorescence immunoassay (point-of-care)	Myocardial injury assessment	Quick POC option; validated in domestic vets (dogs/cats); potential extrapolation to other species with validation
	TashikincTnI Test	Cardiac Troponin I	Immunoassay	Myocardial damage & staging	Quantitative biomarker for cardiac injury in dogs/cats
	GeneMedicTnI Reagents	Cardiac Troponin I antigen/antibody	Immuno-diagnostics reagents	Diagnostic kit development	Available for multiple species (canine, equine, bovine) — potential adaptation for wildlife tests
N-terminal pro B-type Natriuretic Peptide (NT-proBNP)	Vcheck® NT-proBNP	NT-proBNP	Fluorescence immunoassay	Cardiac stretch/heart failure indicator	Rapid assessment of cardiac mechanical stress; used in small animal practice
	Tashikin NT-proBNP Test Kit	NT-proBNP	Fluorescence immunoassay	Heart disease screening & staging	Fast quantitative test for CHF and cardiac overload differentiation
	VetForcNT-proBNP Rapid Kit (FIA)	NT-proBNP	Fluorescence immunoassay	Early cardiac stress detection	Point-of-care rapid kit for cardiac strain; veterinary use
	GeneMedi NT-proBNP Reagents	NT-proBNP antigen/antibody	Immuno-diagnostics reagents	Cardiac diagnostic kit development	Available for multiple domestic species; production for wildlife requires validation
Acute Phase Proteins (Inflammation)	CRP/SAA immunoassays (Hytest / similar)	C-reactive protein, Serum Amyloid A	ELISA / immunoassay	Systemic stress, inflammation	Useful adjunct markers for systemic response; species validity varies
Symmetric Dimethylarginine (SDMA)	IDEXX SDMA Test	SDMA	Automated chemistry / point-of-care	Early renal dysfunction	Detects reduced GFR earlier than creatinine; widely available for cats/dogs
Creatinine & Urea	Routine Serum Chemistry Panels	Creatinine, BUN	Automated chemistry analyzer	General renal function	Standard renal function measures; low sensitivity for early injury
Urinary NGAL (KIT)	KI Screening Test (NGAL)	NGAL (urine)	Lateral flow immunochromatography	Early AKI screening	Rapid semi-quantitative test for tubular damage in urine
Novel / Research Biomarkers (Cystatin C, KIM-1)	ELISA Kits (various vendors)	Cystatin C, KIM-1	Laboratory immunoassays	Early renal injury detection	Not often point-of-care; useful in research and advanced diagnostics

6.2 Histopathological Confirmation

In many clinical studies and case reports, post-mortem histopathology remains the gold standard for confirming stress-induced organ injury. For example, cetaceans show contraction band necrosis in cardiac fibers, severe multifocal skeletal muscle degeneration, and renal tubular casts positive for myoglobin (Spraker *et al.*, 2012) ^[8]. Microscopic findings also reveal multi-organ congestion, edema, and hemorrhage, corroborating the systemic impact of extreme physiological stress.

7. Management and Prevention Strategies

7.1 Acute Intervention

There are currently no universally effective treatments for capture myopathy once it develops. Acute management focuses on minimizing metabolic stress, maintaining hydration, correcting electrolyte imbalances, and supporting organ perfusion. Supportive care may include fluid therapy and careful monitoring, but outcomes remain poor once organ systems are deeply compromised (Zahid, 2018, Breed *et al.*, 2019, Dinesh *et al.*, 2020) ^[19, 2, 7].

7.2 Minimizing Stress during Wildlife Handling

Preventive measures are crucial because once CM and related syndromes become clinically evident, prognosis is poor. Best practices include:

- Reducing chase and restraint durations during capture.
- Providing adequate sedation/immobilization to minimize physical struggle.
- Avoiding overheating and ensuring cool, shaded environments post-capture.
- Rapid processing and release when possible.

8. Species Susceptibility and Conservation Implications

8.1 Ungulates and Large Mammals

Studies show that ungulates (Atkinson, 2012) ^[14] such as deer are particularly prone to CM due to their physiological adaptations for flight responses. Elevated CK and LDH levels correlate with muscle damage and exertional (Landau *et al.*, 2012) ^[13] stress in these species.

8.2 Marine Mammals

Live-stranded dolphins and other marine mammals demonstrate severe rhabdomyolysis and cardiac necrosis, with renal failure often complicating outcomes (Cámara *et al.*, 2019, Nueangphuet *et al.*, 2022) ^[5, 4].

8.3 Birds and Other Wildlife

Birds, especially large birds like cranes and turkeys, experience capture myopathy marked by muscle stiffness, hyperthermia, and sometimes sudden cardiac collapse (Businga *et al.*, 2007) ^[10].

9. Future Directions and Research Gaps

Despite extensive documentation of capture myopathy and related syndromes, significant gaps remain:

- Species-specific reference intervals for biomarkers are largely lacking, limiting clinical interpretation.
- The underlying genetic and metabolic predispositions that increase susceptibility to CM are poorly understood.
- More research is needed on effective therapeutic interventions and field-adapted supportive care protocols.

10. Prevention and Management Strategies

10.1 Field Prevention during Capture and Handling

Because capture myopathy (CM) has no reliably effective cure, prevention remains the most critical intervention. Capture myopathy arises from severe physiological stress due to capture, restraint, transport, or environmental challenges that trigger intense metabolic demand, rhabdomyolysis, and subsequent organ injury. Prevention protocols should minimize stressors that exacerbate physiologic strain (*e.g.*, prolonged chase, physical struggle, loud noises, or overheating) (López *et al.*, 2007, Cámara *et al.*, 2019, Nueangphuet *et al.*, 2022) [15, 5, 4].

Best practices recommended in wildlife management include:

- Minimizing pursuit and handling time to reduce stress and metabolic demand.
- Use of species-specific capture methods (*e.g.*, appropriate sedation and restraint) based on biological and behavioral traits.
- Reducing auditory and visual stimuli during capture by keeping noise low and using blindfolds when appropriate.
- Efficient processing and rapid release where possible to limit duration of stress.

Preventative interventions have been shown to halve the incidence of CM in cervids when handling and processing are optimized (*e.g.*, sedation, minimized noise) (Nueangphuet *et al.*, 2022) [4].

10.2 Supportive Clinical Care in Captivity and Rehabilitation

Once stress-related organ dysfunction develops, clinical treatment is largely supportive. No single therapy reliably reverses capture myopathy, cardiac injury, or AKI once they reach advanced stages. However, selected interventions may improve chances of survival in managed care settings (Fischer, 2019)

- **Fluid therapy:** Aggressive intravenous fluids to improve renal perfusion and reduce myoglobin tubular precipitation.
- **Metabolic buffering:** Correct metabolic acidosis resulting from lactic acid accumulation due to rhabdomyolysis.
- **Thermoregulation:** Actively cool animals to counteract hyperthermia, a major contributor to stress-induced injury.
- **Pharmacologic support:** Sedatives or tranquilizers may decrease sympathetic stress responses, though evidence is largely anecdotal and species-dependent.
- **Monitoring biomarkers:** Serial monitoring of CK, cTnI, BUN, and creatinine helps track organ injury

progression; monitoring electrolytes is essential to prevent fatal arrhythmias or additional metabolic derangements (Montané & Marco, 2002) [16].

Case reports in marine mammals indicate that supportive care (*e.g.*, fluid therapy, antibiotics, gastroprotectants) may assist recovery from AKI secondary to severe rhabdomyolysis, although complications such as delayed hypercalcemia require vigilant monitoring (Herráez *et al.*, 2007, Cámara *et al.*, 2019, Nueangphuet *et al.*, 2022) [3, 5, 4].

10.3 Environmental and Welfare Recommendations

For zoo and captive wildlife, enrichment and husbandry practices should minimize chronic stressors. Environmental stress, including inadequate space, social isolation, or unpredictable handling procedures, can predispose animals to stress responses that mimic capture myopathy upon acute challenges. Providing habitat enrichment, predictable daily routines, and minimizing unnecessary handling are integral to welfare-driven conservation (Nueangphuet *et al.*, 2022) [4].

11. Conservation Implications and Policy Considerations

Stress-induced organ injury, particularly capture myopathy, has significant conservation implications. Because CM can kill wild animals released after research, translocation, or rehabilitation, losses due to stress exacerbate conservation challenges especially for endangered species and small populations.

Translocation and management programs must balance the benefit of intervention (*e.g.*, relocation to safer habitat) against the increased risk of stress-related mortality. Decision-making frameworks should incorporate species predisposition to CM, environmental conditions (*e.g.*, heat stress), and logistical constraints to avoid exacerbating mortality rates.

Additionally, wildlife disease surveillance programs should integrate stress physiology markers into assessment protocols to identify early signs of organ injury so that interventions (or humane release decisions) can be made proactively (Nueangphuet *et al.*, 2022) [4].

12. Recommendations for Future Research

Although studies have documented numerous cases of capture myopathy across taxa, significant knowledge gaps remain:

1. **Species-specific reference intervals** for cardiac, muscular, and renal biomarkers in wildlife are urgently needed to improve interpretation and diagnostic accuracy.
2. **Mechanistic studies** exploring the genetic and physiological triggers for stress responses are lacking, especially for non-model species.
3. **Longitudinal studies** evaluating outcomes following different capture and handling protocols will inform evidence-based management strategies.
4. **Refinement of therapeutic approaches**, including targeted pharmacologic interventions and renal support technologies, could improve survivorship in captive and rehabilitated animals.

Conservation physiology research should incorporate high-throughput omics techniques to delineate early molecular changes predictive of organ failure, offering the potential for precision monitoring in both free-ranging and managed populations (Breed *et al.*, 2019) [2].

13. Summary Tables

Table 1: Comparison of Stress-Induced Organ Injury Mechanisms in Wildlife (Herráez *et al.*, 2007) ^[3]

Component	Mechanism	Biomarkers	Outcomes
Muscle Injury	Rhabdomyolysis due to exertion/stress(Herráez <i>et al.</i> , 2007) ^[3]	CK, LDH	Muscle necrosis, systemic metabolic acidosis
Cardiac Stress	Stress cardiomyopathy	cTnI, ECG changes	Myocardial degeneration & arrhythmias
AKI	Myoglobinuric nephrosis	BUN, creatinine	Tubular necrosis, oliguria
MOF	Combined organ dysfunction	Composite metabolic panels	Systemic shock, mortality

(**based on documented cases in cetaceans, ungulates, and other wildlife)

14. Concluding Remarks

Stress-induced organ injury in wildlife and zoo animals especially cardiac injury, acute kidney injury, and multi-organ failure is a critical pathology with implications for animal welfare, conservation outcomes, and veterinary practice. Although *capture myopathy* is often the central driver of these syndromes, the breadth of stressors that can precipitate similar pathophysiological cascades demands comprehensive monitoring and preventive strategies. Biomarker profiling plays an increasingly important role in identifying subclinical injury and guiding interventions. However, persistent gaps in species-specific data, validated diagnostic protocols, and effective therapeutic regimens underscore the need for targeted research.

Advances in molecular diagnostics and species-tailored management protocols are essential for reducing mortality associated with stress responses in wildlife conservation and zoo medicine. Adopting evidence-based prevention and welfare practices has the potential not only to improve individual animal outcomes but also to support broader biodiversity conservation efforts.

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